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Original Article

Coexistence of Medullary and Papillary Thyroid Carcinomas Detected Incidentally

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Abstract

Papillary thyroid carcinoma (PTC) and medullary thyroid carcinoma (MTC) are extremely rare and constitute less than 0.5% of all thyroid malignancies. In this study, the prevalence and characteristics of 8 patients with simultaneous PTC and MTC diagnoses were evaluated.

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Keywords: *papillary thyroid carcinoma, medullary thyroid carcinoma, thyroid cancer, germline RET mutation*

Introduction

The incidence of thyroid cancer, the most common cancer of the endocrine system, has increased over the years. Thyroid cancer is the 12th most common cancer with 2.9% of all new cancer cases in the U.S.[1] Papillary thyroid carcinoma (PTC), originating from thyroid follicular epithelial cells, is the most common form of thyroid cancer which accounts for about 70% of thyroid malignancies.[2] Medullary thyroid carcinoma (MTC) develops from parafollicular cells that express calcitonin, and accounts for 1-2% of all thyroid cancers.[3] Coexistence of PTC and MTC is extremely rare, accounts for less than 0.5% of all thyroid malignancies.[4] Here, we report 8 cases of co-existing papillary and medullary thyroid carcinomas with histopathological features, imaging and laboratory findings, outcomes and impact of treatment with the data in the literature.

Material and Methods

We retrospectively analyzed data of 31 patients with medullary thyroid carcinoma between 2012 and 2020 at our institution. The information collected included age, gender, type of surgery, histopathological findings (tumor

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localization, maximal diameter, capsule invasion, lymphovascular invasion, lymph node involvement and number, tumor stage), presence of RET protooncogene, time since diagnosis of thyroid cancer, previous and current sonographic findings, treatments received for thyroid malignancies, and related comorbidities. Calcitonin, thyroglobulin and anti-thyroglobulin levels were measured with local methods and commercial kits. The staging of thyroid cancers were re-evaluated based on Tumor–Node–Metastasis (TNM) cancer staging system by the new, 8th editions of the relevant Union for International Cancer Control (UICC) and American Joint Committee on Cancer.

Results

The mean age of all patients (MTC and MTC/PTC) was 53.7±12.01 years at the time of diagnosis. The mean age of only MTC patients (23/31) was 50.6±10.6 years. Eight of 31 patients (25%) with MTC had PTC and MTC simultaneously. Overall, median age was 64.5 years (range, 41-83) in MTC/PTC group, and seven of eight participants were female, five patients among 8 were treated in Uludag University hospital. Median follow-up for these patients was 25 months, with a range of 16-104 months. In 7 of the 8 patients, the maximal diameter of PTC was 10 mm or less. In 7 patients the largest tumor diameter was 23.5±18.5 mm for MTC and 6.75±4.6 mm for PTC. 3 patients had lymph node involvement. Calcitonin, CEA and thyroglobulin were respectively 380 pg/mL (range: 2-16072), 24.6 mcg/L (range: 1-551.6), and 9.2 mcg/L (range: 0.19-25) preoperatively. Same blood tests were performed after surgery and measurements of serum calcitonin, CEA and thyroglobulin were 2 pg/mL (range: 2-10071), 2.5 mcg/L (range: 0.9-419.3), and 0.27 mcg/L (range: 0.13-1.4), respectively. Fifty percent of patients (50%, 4/8) had received adjuvant RAI treatment and one patient received tyrosine kinase inhibitor (TKI) therapy.

Discussion

Three previous studies have reported that mean age of only MTC and MTC/PTC patients were respectively 48.2±16.9, 44.5±12.6, median age 44.3 (range: 43-45.7) years for only MTC and 49.9±13.9, 53.5±6.5, median age 50.2 (range: 44.6-55.8) years for MTC/PTC patients.[5-7] In these studies, MTC/PTC patients were older than only MTC patients at the time of diagnosis. The median age in our study was also higher. Despite low numbers, similar to previous studies reported, the median age of patients with MTC/PTC was higher than that of patients with only MTC in our study.

Several studies have shown different frequencies of coexistence of MTC and PTC between 3.6% and 19%. The higher frequency (25%) in our study can be explained by the fact that the patients have been diagnosed in recent years and the frequency of PTC has increased significantly in the last 2 decades.[8]

In this study, the mean follow-up period of the patients was 25 months. In the previous studies, follow-up time was 32 months (range: 0-261) and 49.1±33.4 months, respectively. The medical records were reviewed from 1996 to 2006 and from 1992 to 2014 by these studies.[5,9] Follow-up period was shorter in this study because most of our patients diagnosed lately. For instance, six of 8 patients with MTC/PTC were diagnosed in 2018 and later.

PTC diameter of seven patients was equal or less than 1 cm. In Limh et al.'s study[8], the largest tumor diameter was equal or less than 1 cm in 32.5% of patients with PTC. The fact that mPTC was detected in most of the patients in our study can be explained by the advanced age of the patients. In addition, the reason for this high rate may be related to the fact that fine needle aspiration biopsy is not performed for nodules smaller than 1 cm detected by thyroid ultrasound before surgery. In our study, only 2 patients were diagnosed with PTC preoperatively by FNAB. Similarly, in the Korean study, 9 of 10 patients had mPTC.[6]

In conclusion, although our results support the coincidental existence of MTC/PTC, physicians should be aware of the coexistence of these thyroid malignancies to avoid possible misdiagnosis.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Short Communication

A meta-analysis on the role of IL-6 associated JAK/STAT3 signaling pathway modulation in the inflammatory bowel disease complicated colonic cancer development

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Keywords: *Inflammatory bowel disease, colon cancer, IL-6, JAK/STAT3*

The signaling pathway of Janus kinase (JAK)/signal transducer and activator of transcription 3 (STAT3) is suggested to be involved in various pathophysiological processes, including immune function, cell growth, differentiation, hematopoiesis and more importantly oncogenesis of distinct tumoral conditions. Interleukin (IL) 6 is a proinflammatory cytokine produced by antigen-presenting cells and non-hematopoietic cells in response to external stimuli and considered to be a key player in the development of the microenvironment of malignancy by promoting tumor growth and metastasis by acting as a bridge between chronic inflammation and cancerous tissue. In tumor cells, JAK/STAT3 hyperactivation can occur as a result of elevated IL-6 levels in the serum and/or in the tumor microenvironment, owing to signals from other growth factors and/or their receptors, activation by non-receptor tyrosine kinases, or loss-of-function mutations affecting negative regulators of STAT3. Ulcerative colitis (UC) and Crohn's disease (CD) are subtypes of inflammatory bowel disease (IBD) in which abnormal reactions of the immune system cause inflammation and ulcers on the distinct segments of the gastrointestinal system with a significant risk of colorectal cancer development. Recent studies suggest that aberrant interleukin IL6/JAK/STAT3 signaling pathway exists in both IBD and inflammation-related gastrointestinal cancers. In the present meta-analysis, we aimed to analyze the relationship between IL-6/JAK/STAT3 and IBD associated colorectal carcinogenesis and the effect of the inhibition of this system on disease follow-up and management.

A systematic literature review was carried up to January 2021 to identify all primary studies examining the role of IL-6 associated JAK/STAT3 system in IBD associated colorectal carcinogenesis. Studies related with the rest of other interleukins other than IL-6 was excluded.

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Table 1. Studies evaluating IL6 associated JAK/STAT3 pathway activation in colitis associated cancer

Reference	Result	Proposal
Li et al. (2009)	In dysplasia and cancer, epithelial cells of UC patients expressed significantly more IL6 and p-STAT3 compared with controls	This study demonstrated the importance of IL6/p-STAT3 in patients with inflammation-induced colorectal cancer. Moreover, cytokine signaling 3 may be involved in UC pathogenesis and the absence of cytokine signaling 3 seems critical for colorectal cancer progression.
Grivennikov et al. (2010)	IL-6 is found to be a critical tumor promoter during early colitis associated cancer tumorigenesis. In addition to enhancing proliferation of tumor initiating cells, IL-6 produced by lamina propria myeloid cells protects normal and pre-malignant intestinal epithelial cells (IEC) from apoptosis.	The proliferative and survival effects of IL-6 are largely mediated by transcription factor STAT3, whose IEC-specific ablation has profound impact on colitis associated tumorigenesis. Thus, the NF-κB-IL-6-STAT3 cascade is an important regulator of the proliferation and survival of tumor initiating IEC.
Yang et al. (2013)	The IL-6/STAT3 signaling pathway was attenuated in oroxylin A-treated mice. Oroxylin A effectively inhibited IL-6/STAT3 pathway in human HCT-116 cells.	The result of this study demonstrated that oroxylin A inhibits colitis-associated cCRC via modulating IL-6/STAT3 pathway in AOM/dextran sodium sulfate mouse model and in HCT-116 cells.
Chakilam et al. (2013)	It was found that death-associated protein kinase (DAPK)-induced conformational changes in the STAT3 dimer masked its nuclear localization signal. Alternatively, pharmacological inactivation of STAT3 resulted with an increase in DAPK mRNA and protein levels.	This study revealed that DAPK as a negative regulator of STAT3 emerges as therapeutic option in the treatment of UC and UC associated CRC.
Kim et al. (2013)	It has been found that shRNA-mediated galectin-4 silencing increases cell proliferation and, concomitantly, activates NF-κB and STAT3 signaling along with IL-6 up-regulation in CRC patients.	Authors proposes that abrogation of galectin-4 expression promotes cancer cell proliferation and provide s evidence that down-regulation of galectin-4 elicits tumor promotion in vitro and in vivo through activation of IL-6/NF-κB/STAT3 signaling.
Dai et al. (2014)	Embelin suppressed colonic IL-6 expression and secretion, and subsequently STAT3 activation in vivo. Moreover, embelin protected mice from AOM/DSS induced colitis before tumor development.	Embelin suppresses colitis-associated cancer, and its antitumor effect is partly mediated by limiting IL-6/STAT3 activation and Th17 immune response. It may be a potential agent in the prevention and treatment of colitis- associated cancer
Saadatdous t et al. (2015	Cocoa significantly decreased the tumor incidence and size in colitis-associated colorectal cancer in a rat IBD model of azoxymethane/dextran sulfate sodium. Moreover, cocoa suppressed colonic IL-6 expression and resulted in activation of STAT3.	This study demonstrated that cocoa may be a potential agent in the prevention and treatment of colitis-associated colorectal cancer by suppressing IL-6 secretion
Chen et al. (2015)	In UC model + empty vector group, IL6 and STAT3 expression was increased as lesion degree increased ($P < 0.05$). The expression of cytokine signaling 3 was weakened and the degree of activation decreased ($P < 0.05$)	The expression and activation of IL6 and STAT3 expression were enhanced in ulcerative colitis carcinogenesis, and their expression increased with the lesion degree increased, reflecting the disease progression to a certain extent
Do et al. (2016)	The total numbers of tumors in the Balsalazide and probiotic agent VSL#3 groups were significantly low compared with the colitis-associated carcinogenesis group	The results of this study demonstrated that Balsalazide and probiotic agent VSL#3 have chemo preventive effects against colitis-associated carcinogenesis through IL- 6/STAT3 suppression. Balsalazide and VSL#3 could be suitable options for chemoprevention of colorectal cancer.
Zhang et al. (2018)	IL-6 treated cells stimulated inflammatory microenvironment and found that glucose uptake, lactate production and lactate dehydrogenase activity elevated dramatically.	This study revealed that metabolic disruptions triggered by inflammatory signaling are associated with tumorigenesis via the STAT3/c-Myc axis in rat model of DSS induced colitis.
Ye at al. (2019)	Retinoid X receptor-alpha (XRα) is abnormally cleaved in tumor cells and tissues, producing a truncated RXRα (tRXRα) and transgenic expression of tRXRα in mice accelerates the development of colitis-associated colon cancer. The tumorigenic effect of tRXRα is primarily dependent on its expression in myeloid cells, which results in IL-6 induction and STAT3 activation.	Results of this study provides new insight into tRXRα action and identify a promising tRXRα ligand for treating colitis associated cancer.
Huangfu et al (2020)	Transcriptomic sequencing indicated that modified Pulsatillae decoction treatment downregulated the IL-6/STAT3 signaling pathway, and reduced the levels of p-NF-κB, IL-1β and NLRP3, which were confirmed by western blot.	This study propose that modified Pulsatillae decoction could efficiently relieve clinical signs and inflammatory mediators of UC, providing evidence of the anti-colitis effect of modified Pulsatillae decoction, which might provide novel strategies for therapeutic intervention in UC, which may be applied to the prevention of IBD-related colorectal cancer

All articles were critically appraised with regard to methodological quality and risk of bias. Twelve clinical trials that fulfilled the inclusion criteria were further pooled into a meta-analysis.

Twenty-two studies met initial selection criteria but only 12 were eligible for inclusion in the meta-analysis. The majority of studies demonstrated a significant role of IL6 associated JAK/STAT3 in the pathophysiology of UC related carcinogenesis. Table 1 summarizes the clinical trials that shows the potential role of IL6/JAK/STAT3 pathway in UC associated colorectal cancer development.

In light of the small number of studies able to be included in the meta-analysis, evidence strongly proposed that JAK/STAT3 signaling especially via the IL-6/STAT3 axis is involved in the transition of inflammatory lesions to tumoral diseases and leading to UC associated colorectal cancer. For this reason, based on the evidence presented in this meta-analysis it is reasonable to suggest that targeting components of the IL-6/JAK/STAT3 signaling pathway can inhibit tumor cell growth and relieve immunosuppression in the UC associated colonic tumoral microenvironment.

Conflict of Interests

Authors declare that there are none.

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This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Short Report

Relationship between Vitamin D level and Insulin Resistance According to Obesity Level

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Keywords: *insulin resistance, obesity, vitamin D deficiency, metabolic syndrome*

Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health. Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of his height in meters (kg/m^2). In adults, WHO defines overweight as a BMI greater than or equal to 25 kg/m^2; and obesity as a BMI greater than or equal to 30 kg/m^2. BMI over 40 kg/m^2 is classified as morbid obesity, and over 50 kg/m^2 as super obesity. BMI is the same for both sexes and for all ages of adults. Overall, about 13% of the world's adult population (11% of men and 15% of women) were obese in 2016.[1]

Insulin resistance (IR) is defined as a defective metabolic response of insulin to stimulate glucose uptake into skeletal muscle and adipose tissue and/or to suppress hepatic gluconeogenesis and glucose release into circulation.[2,3] The metabolic response of IR and subsequent hyperinsulinemia is attributed to the development of serious health consequences such as for overweight, hypertension, hyperlipidemia, cardiovascular disease, and type 2 diabetes. IR is considered a common mechanism underlying derangements associated with the syndrome.[4] Metabolic syndrome (MetS) is a cluster of metabolic disorders and diagnosed on the following criteria and if the individual is positive for three or more of the following measurements5: Abdominal obesity/waist circumference (≥94-102 cm in men or ≥80-88 cm in women), high blood pressure (≥130/85 mmHg), abnormal fasting glucose (≥100 mg/dL), elevated triglycerides (TG: ≥150 mg/dL) and low HDL (males <40 mg/dL, female <50 mg/dL). These metabolic disorders collectively or independently increase the risk of an individual developing cardiovascular disease (CVD), diabetes mellitus, and vascular or neurological complications.

It has been proposed 25-OH-vitamin D concentration affects insulin sensitivity and beta-cell function.[6,7] Numerous clinical studies showed that vitamin D supplementation reduces the level of metabolic parameters such as total cholesterol (T-chol), low-density lipoprotein (LDL), TG, glycated hemoglobin (HbA1c), as well as decreases IR indicator (HOMA-IR) in type 2 diabetic patients.[8-10] However, the underlying mechanism not fully understood how vitamin

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D may reduce the risk of metabolic disorders development. Besides, the effect of vitamin D on beta-cell dysfunction is more apparent in patients without metabolic syndrome or obvious beta-cell dysfunction.[11,12] In this study, we aimed to investigate the relationship between vitamin D level and IR in obese patients without diabetes mellitus, hypertension, and hyperlipidemia in the light of previous studies.

The study enrolled 95 obese adult patients without a history of hypertension, diabetes mellitus, and hyperlipidemia who applied to our clinic were included in the study. None of the patients were using antihypertensive drugs, oral antidiabetic drugs and/or insulin, statin and/or fibrate derivatives, calcium and/or vitamin D supplements. Insulin, vitamin D, parathyroid hormone, total calcium, ionized calcium, T-chol, HDL, LDL, TG levels were measured from venous blood samples taken after 12 hours of fasting. IR was calculated using fasting glucose (FBG) and insulin levels (HOMA-IR = FBG x insulin/405). IR was considered as HOMA-IR level above 2.5. 25-hydroxyvitamin D level was measured, vitamin D level above 20 ng/mL was normal, and below it was accepted as a deficiency. Patients were classified according to BMI level, between 30-39.9 kg/m^2 were classified as mild-moderate obese, 40-49.9 kg/m^2 as morbid obese, and 50 kg/m^2 and above as super obesity. The diagnosis of the MetS was made when three or more were present according to NCEP/ATP III criteria.

Seventeen (17.9%) of the patients were male and 78 (82.1%) were female. The mean age of the patients was 40.4 (18-75) years. The mean BMI of all patients was 43.42±7.74 kg/m². 39 (41.1%) of all patients had MetS. The baseline demographic characteristics and laboratory findings of the

Table 1. Demographic characteristics and laboratory findings of patients (n=95)

Age (years)	40.44±12.54 (18-75)
Sex (female)	78 (82.1%)
BMI (kg/m²)	43.42±7.74 (30-62)
Fasting blood glucose (mg/dL)	89.82±13.07 (65-125)
Insulin (mg/dL)	18.17±12.19 (2.6-80)
HOMA-IR	3.99±2.68 (0.45-15.8)
Total cholesterol (mg/dL)	200.62±36.92 (104-275)
HDL (mg/dL)	41.49±10.16 (23-88)
LDL (mg/dL)	127.61±32.33 (58-209)
Triglyceride (mg/dL)	163.93±91.41 (56-512)
Vitamin D (ng/mL)	14.89±11.9 (4-54.9)
Calcium (mg/dL)	9.56±0.41 (8.5-10.5)
Parathormone	6.11±2.27 (1.9-14.88)
Metabolic syndrome (n, %)	39 (41.1%)

Table 2. Characteristics of patients according to vitamin D levels

Vitamin D level	**<20 (n=75)**	**≥20 (n=20)**	**p value**
Sex Female	64 (82.1%)	14 (17.9%)	0.185
Male	11 (64.7%)	6 (35.3%)	
Age (years)	37	37.5	0.862
BMI (kg/m²)	43	40.5	0.156
HOMA-IR	3.57	2.39	0.029

Table 3. Relation between Vitamin D level and insulin resistance

Vitamin D level		**<20 (n=75)**	**≥20 (n=20)**	**p value**
HOMA-IR	<2.5	16 (59.3%)	11 (40.7%)	0.005
	≥2.5	59 (86.8%)	9 (20%)	

Table 4. Characteristics of patients according to insulin resistance

HOMA-IR	<2.5 (n=27)	≥2.5 (n=68)	p value
Age (years)	47	36	0.003
Sex Female	24 (30.8%)	54 (69.2%)	0.277
Male	3 (17.6%)	14 (82.4%)	
BMI (kg/m²)	40.4	43.5	0.202
Vitamin D	8.7	11.6	0.461
Metabolic syndrome (n, %)	6 (15.4%)	33 (84.6%)	0.034
Insulin	8	18.7	<0.001
Parathormone	5.1	6	0.328

Frequency n (%), mean ± standard deviation, median (Q1-Q3).

patients are shown in Table 1. The average HOMA-IR level of women was 3.84 and 4.68 for men. There was no statistically significant difference in terms of HOMA-IR levels between both sexes (p=0.283). Vitamin D deficiency was found in 77.9% of the patients. When the groups with and without vitamin D deficiency were compared, no difference was found in terms of age, gender, and BMI. The median HOMA-IR level of the group with vitamin D deficiency (<20 ng/mL) was 3.57, and the HOMA-IR median level of the group without vitamin deficiency (>20 ng/mL) was 2.39. The median HOMA-IR level of the vitamin D deficiency group was statistically significantly higher than the group without vitamin D deficiency (p=0.029) (Table 2). Vitamin D deficiency was statistically significantly more common in the group with IR (p=0.005) (Table 3). The mean age of the group with IR (HOMA-IR >2.5) was statistically significantly lower than the group without IR (HOMA-IR <2.5) (p=0.003). There was no significant difference between the groups according to gender, BMI, and vitamin D levels (Table 4).

The patients were divided into 3 groups according to their BMI (obese, morbid obese, and super-obese). No difference was found between these groups in terms of age, gender, and vitamin D level (Table 5). However, while insulin level and IR level are significantly higher in morbid obese patients compared to obese patients, this relationship could not be demonstrated in super-obese patients (p=0.03 and p=0.019, respectively).

This study is the first study investigating the relationship between vitamin D-HOMA-IR in morbid and super-obese patients. No statistically significant linear correlation was found between vitamin D and HOMA-IR level, but vitamin D deficiency was more common in the group with IR. Karatas et al.[13] demonstrated that vitamin D deficiency was more common in overweight/obese patients regardless of the presence of MetS.

Table 5. Characteristics of patients according to obesity level

BMI level (kg/m²)	30-39.9 (n=33)	40-49.9 (n=44)	≥50 (n=18)	p value
Age (years)	37	37	39.5	0.894
Sex Female	27 (34.6%)	36 (46.2%)	15 (19.2%)	0.989
Male	6 (36.3%)	8 (47.1%)	3 (17.6%)	
HOMA-IR	2.79	3.65	3.89	0.019
Metabolic syndrome	11 (28.2%)	23 (59%)	5 (12.8%)	0.110
Vitamin D	13.4	10.45	11.45	0.278
Glucose (mg/dL)	86	90	92	0.175
Insulin	12	17	16.3	0.030

Frequency n (%), median (Q1-Q3).

Clemente-Postigo et al. and Ferreira et al. showed a negative correlation between vitamin D levels and IR.[14] There is increasing evidence that vitamin D level is inversely related to BMI and IR. It is thought that factors such as improper food intake, decreased sun exposure due to lack of mobility, and decreased bioavailability of the vitamin. However, the effect of vitamin D supplementation on IR is limited and insufficient. Additional studies are needed to explain the relationship between the level of obesity and the effect of vitamin D on IR.

Conflict of Interests

Authors declare that there are none.

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TURKISH JOURNAL OF INTERNAL MEDICINE
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Short Report

3-year Follow-up Results of Abant Izzet Baysal University Training and Research Hospital Patients with Pulmonary Hypertension

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Pulmonary hypertension (PH) is defined as the mean pulmonary artery pressure (mPAB) measured by right heart catheterization (RHC) 25 mmHg or higher at rest. Different hemodynamic PH definitions were made according to the combinations of pulmonary artery pressure measured by right heart catheterization, pulmonary artery end pressure (PCWP), cardiac output (CO), diastolic pressure gradient, and pulmonary vascular resistance (PVR).[1] The differential diagnosis of PH is at least as important as its treatment, as it is a condition that can cause many diseases. Symptoms and signs include shortness of breath, lightheadedness, syncope, tiredness, chest pain, swelling of the legs, poor appetite, chest pain, right-sided abdominal pain, palpitations, cyanosis and rarely non-productive cough, exercise-induced nausea, and vomiting. We herein present 3-year follow-up results of patients with PH.

In symptomatic patients with suspected PH, those with the possibility of PH echocardiographically were evaluated in our clinic with a multidisciplinary approach. Left heart diseases and lung diseases were excluded as a result of the evaluations of Chest Diseases, Internal medicine, and Rheumatology departments. RHC was performed in our clinic between January 2018 and January 2021 to confirm the diagnosis of group 1 PH in 30 patients whose chronic thromboembolic PH (CTEPH) diagnosis was excluded by negative ventilation-perfusion scintigraphy and/or pulmonary CT angiography. Considering the PH etiology of the patients; Eisenmenger syndrome due to congenital diseases in 2 patients; Group 4 PH in 7 patients; Group 2 PH in 3 patients; idiopathic PH diagnosis in 5 patients, and 5 patients were diagnosed with PH secondary to connective tissue diseases, and thus, pulmonary arterial hypertension (PAH)-specific treatment was initiated in a total of 22 patients (Table 1). In our clinic, vasoreactivity test is performed with adenosine. There was no vasoreactivity response in any of the patients who underwent the vasoreactivity test.

PAH was followed-up before specific treatment

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Table 1. Clinical classification of 22 patients with pulmonary hypertension

PH subtypes	Patient number
Group 1 PAH	Idiopathic PAH (n=5)
	Connective tissue disease (n=5)
	Congenital heart disease (n=2)
Group 2 PH	PH due to left heart disease (n=3)
Group 4 PH (CTEPH and other pulmonary artery obstructions)	CTEPH (n=3)
	Operated due to CTEPH and residue PH (n=2)
	Operated due to hydatid cyst and residue PH (n=1)
	Arteritis (n=1)

PAH: pulmonary arterial hypertension, PH: pulmonary hypertension, CTEPH: chronic thromboembolic pulmonary hypertension.

was initiated because of isolated postcapillary PH or the average PAP <25 mmHg in 8 patients according to RHC findings. When the treatments received by the patients were examined, 5 patients were referred to as single PH specific therapy; 14 patients dual combination therapy; 3 patients received triple combination therapy (Table 2). Since there was no vasodilator response in patients who underwent vasoreactivity test during right heart catheterization, no patient was followed up with high-dose calcium channel blocker. During their follow-up, 9 of our patients were followed up in the NHYA 1, 10 in the NHYA 2, 1 in the NHYA 3, and 2 in the NHYA 4 clinic, and one of these patients started subcutaneous prostanoid treatment in an external center; 1 patient in group 2 with precapillary and postcapillary PHT died in our hospital.

A diagnosis of PH requires clinical suspicion based on symptoms and physical examination. A series of examinations are required to determine the compliance of this suspicion with hemodynamic criteria, the etiology of the disease, and the functional and hemodynamic severity. These examinations should at least be interpreted by a multidisciplinary team of Cardiology, Rheumatology, Radiology, and Chest Diseases specialists. In our daily practice, it is necessary to raise awareness for this patient group, whose diagnosis is delayed, and the specialist referral centers should be determined and the referral chain should be operated.

Table 2. Pharmacological treatments given to patients

Treatment	Patient number	Side effect	Combination therapy	Drug monotherapy
Endothelin receptor antagonists				
Bosentan	8	2	7	1
Macitentan	5	-	4	1
Ambrisentan	3	-	3	-
Phosphodiesterase type 5 inhibitors				
Sildenafil	9	-	9	-
Tadalafil	5	-	5	-
Guanylate cyclase stimulators				
Riociguat	7	-	4	3
Prostacyclin analogues				
Iliprost (inhaled)	3	1	3	-
Subkutan PG	1	-	1	-
IP receptor agonists				
Selexipag	1	-	1	-

Conflict of Interests

Authors declare that there are none.

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Short Report

The retrospective analysis of urinary tract infection in renal transplant recipients

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Infection is the most common reason for admission to the emergency department in the early period after transplantation. Urinary tract infections (UTIs) in adult kidney transplant patients are common. Their incidence is significantly higher than in the general population.[1] The majority of sepsis cases in this period are composed of UTIs.[2] While mild UTIs generally do not affect graft function, acute pyelonephritis observed in the post-transplant period causes a decrease in graft function and an increase in mortality.[3] In the literature, a small amount of data has been published regarding the incidence, epidemiological features, and risk factors of UTIs in kidney transplant recipients. In this retrospective study, we evaluated the data of 550 patients who underwent kidney transplantation between January 2006 and May 2019 at our center and analyzed UTIs' characteristics.

Among 550 recipients, 633 episodes were detected in 200 patients (36.4%). Recurrent infection was encountered in 74 (37%) of 200 patients. We determined 1 episode in 63 (31.5%), 2 episodes in 39 (19.5%), 3 episodes in 31 (15.5%), 4 episodes in 14 (7%), 5 episodes in 10 (5%), 6 episodes in 8 (4%), and 7 or more episodes in 35 (17.5%). While 48.3% (n=306) of 633 episodes were asymptomatic bacteriuria, 51.6% (n=327) was symptomatic. The risk factors for UTI were female gender, the advanced age of transplant and advanced donor age, long-term dialysis period before transplant, prolonged urinary catheterization and hospitalization time after transplant, cytomegalovirus infection, vesicoureteral reflux, and neurogenic bladder history. At the survival analysis, a transplant from living donor, female gender, use of tacrolimus, mycophenolate mofetil and corticosteroid combination as maintenance immunosuppressive therapy, preemptive transplant compared to

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patients receiving peritoneal dialysis was found to be associated with longer survival; advanced age of transplant, obesity, delayed graft function, acute rejection, diabetes mellitus and a history of cytomegalovirus infection was also associated with a shorter life span. Escherichia coli (64.9%) and Klebsiella pneumonia (51.6%) were the most common causative microorganisms, and ESBL (Expanded Spectrum Beta-Lactamase) was positive in 19.9% and 67.5% of them, respectively.

Similarly, female gender, the advanced age of transplant, prolonged catheterization time, history of vesicoureteral reflux, neurogenic bladder, acute rejection, and deceased donor were dependent risk factors for developing of UTI in transplant recipients in other studies,[4-6] but not body mass index, history of diabetes mellitus, dialysis type and duration, primary kidney disease, donor type, delayed graft function, and history of acute rejection in another study.[7] UTI frequency in the female gender increases due to anatomical differences such as the shorter urethra compared to men and its relative proximity to the perianal region and vulva, as in the general population. Age-related changes in the urinary tract and existing additional diseases are important factors that predispose to bacterial colonization. Prolonged urinary catheterization and hospitalization durations facilitate pathogen entry into sterile body parts in the post-operative period. Immunosuppression, frequent hospitalizations, and surgical interventions increase the risk of nosocomial infections in dialysis patients. A history of vesicoureteral reflux facilitating bacterial invasion and a neurogenic bladder causing urinary stasis poses a UTI risk.

Hospitalizations for septicemia are most commonly associated with UTI.[8] In our cohort, antibiotics in the carbapenem group, started in more than half of the episodes. There is an increase in trimethoprim/sulfamethoxazole, ciprofloxacin and ceftazidime resistance in Klebsiella species.[9] Although the microorganism spectrum in the studies is similar all world, ESBL positivity is noticeably higher than the average in UTI episodes is an observation in parallel with the increasing antibiotic resistance both in our country and in the world in recent years. This observation was considered as a cautionary finding for the review of antibiotic selection preferences in our clinic. Finally, UTIs being a threat to graft and patient survival in the post-transplant period should be treated effectively by carefully evaluating risk factors.

Conflict of Interests

Authors declare that there are none.

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Experience with FLT3 Inhibitor Midostaurin in Newly Diagnosed Acute Myeloid Leukemia Patients

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Keywords: *AML, FLT3 mutation, Midostaurin*

FMS-like tyrosine kinase 3 (FLT3) gene is located on the 13q12 chromosome. It is a member of the class III receptor tyrosine kinase (RTK) family that plays an important role in the proliferation and differentiation of hematopoietic stem cells.[1] FLT3 mutation is seen in approximately 30% of newly diagnosed acute myeloid leukemia (AML) patients.[2,3] FLT-internal tandem duplication (ITD) mutation was first reported in AML in 1996 by Nakao et al.[4] These mutations disrupt the autoinhibitory function of the juxtamembrane of the receptor and result in the autophosphorylation of FLT3. ITD and tyrosine kinase mutation can be encountered in two different ways. FLT3 mutation is an important marker showing relapse and resistance to standard therapies in patients with AML. Patients with FLT3 ITD mutation or high allele burden of FLT3 ITD mutation; have lower mean survival rates compared to patients with negative FLT3 mutation.[5] With the introduction of FLT3 inhibitors in recent years, the presence of FLT3 mutation in AML patients newly diagnosed has become more important. In the RATIFY study for which midostaurin, one of the FLT3 inhibitors, was approved by the FDA, it was observed that the addition of midostaurin to the remission induction and consolidation treatment provided a 22% reduction in mortality risk in patients with FLT3 mutation.[6] In light of the above information, we aimed to share our experience and experience regarding the use of midostaurin in our newly diagnosed AML patients.

The data of 20 patients diagnosed with AML between April 2020 and November 2020 in the Hematology Department of Bursa City Hospital and who were eligible to receive standard remission induction therapy were evaluated retrospectively. Patients diagnosed with acute promyelocytic leukemia or who could not receive standard remission induction therapy because

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of comorbidities and unfitness were excluded from the study. Standard remission induction chemotherapy consisting of a combination of cytosine arabinoside (ARA-C) and an anthracycline was applied to patients diagnosed with AML. While consolidation treatment with high dose ARA-C was applied to patients who were in remission; midostaurin 100 mg daily was added to the patients with FLT3 mutation together with high-dose ARA-C between the 8th and 21st days in each cycle. Patients' characteristics, demographic information, and response status were retrospectively scanned from their files and hospital information system. Response status of patients was evaluated with bone marrow biopsies performed after remission induction chemotherapy. Standard criteria were used for response definitions.[7] Complete response neutrophil count over 1,000x10^9/L, platelet count over 100,000x10^9/L, Independence from erythrocyte suspension transfusion, blast rate below 5%, and complete maturation of all series (granulocyte, erythrocyte, and megakaryocyte) in bone marrow examination.[7]

Of the 20 patients included in the study, 14 were male and 6 were female. There were 6 patients with the FLT3 mutation. The frequency of FLT3 mutation among our patients was consistent with the current literature information. Five of the six patients with FLT3 mutation were male and one was female. The mean age of the patients is 53.8; The mean age of the patients with FLT 3 mutation was 49.8 years. The mean hemoglobin value of the patients at the time of diagnosis was 8.49 mg/dL, the mean leukocyte count was 53,000 x10^9/L, and the mean platelet count was 56,650 x10^9/L. While two of our 6 patients with FLT 3 mutation died due to sepsis during remission induction treatment; in the other 4 patients, complete response was obtained with remission induction therapy. Four patients had been given midostaurin with high dose ARA-C and referred for bone marrow transplantation.

Risk classification in AML plays an important role in the planning of treatment. Molecular markers and mutations are included in the risk classification. It is known that AML patients with FLT3 mutations have shorter mean survival and progression-free survival rates.[3,8] The use of FLT3 inhibitors together with chemotherapeutic agents in the treatment of AML patients with FLT3 mutation is considered as the standard approach. It is recommended to add FLT3 inhibitors along with standard remission induction therapy in patients with FLT3 mutation.[9] However, it takes a certain amount of time for the FLT3 mutation to result. For this reason, midostaurin treatment could be added not during remission induction but consolidation. In addition to all these, when patients with FLT3 mutation enter remission, it is recommended to continue treatment with allogeneic hematopoietic stem cell transplantation.[6,9] We directed our patients who were in remission and had suitable donors for allogeneic hematopoietic stem cell transplantation. Although our study included a limited number of patients, it shows the significance of midostaurin treatment in newly diagnosed AML patients with FLT3 mutation. FLT3 inhibitors will show their value as real-world data where targeted therapies are getting more and more important.

Conflict of Interests

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Short Report

Prognostic Biomarkers in Lung Cancer Patients in terms of Long-term Survival

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Keywords: *Lung cancer, M30 and M65 antigen, neoadjuvant therapy, long-term prognosis*

Lung cancer is the leading cause of death associated with cancer worldwide. The most common lung cancer is non-small-cell lung cancer (NSCLC). Drugs used in the treatment of NSCLC, such as paclitaxel, induce microtubule stabilization in cancer cells, resulting in mitotic arrest in a subset of cancer cells accompanied by apoptotic cell death.[1] Circulating fragments of cytokeratin 18 are M30 released during apoptosis and M65 released during necrosis and are known to be important markers for evaluating the chemotherapy response, especially in epithelial cancers.[2] In this study, we aimed to investigate the predictive effect of serum M30 and M65 antigens on long-term prognosis and in patients with advanced stage lung cancer before and after the first dose of chemotherapy.

Forty-eight patients with advanced stage lung cancer were included in the study. Demographic data and histopathological characteristics of the patients were recorded. Serum levels of M30 and M65 were studied in 48 patients before chemotherapy, and in 43 patients both before and 48 hours after chemotherapy. Long-term survival was evaluated. The median value of M30 and M65 of all patients was selected as the cut-off point for the distinction of M30 and M65 -high patients from M30 and M65 -low patients. The effect of high or low M30, M65 levels and M30/M65 ratio on long-term survival was investigated. Statistical analyses were performed using SPSS 22.0 (SPSS Inc., Chicago, IL, USA) software. The mean serum M30 and M65 values which were calculated before and after chemotherapy were compared using the paired sample t-test. According to the Kaplan-Meier process, survival analysis and curves have

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been developed and compared by the log-rank test.

The mean age of the patients at the time of diagnosis was 57.52±9.38 years. Forty-six of the 48 patients were men. 47.9% of the patients were stage IIIA, 39.6% stage IIIB and 12.5% stage IV. The most dominant histopathological type was squamous cell carcinoma, accounting for 87.5% of the patients. The remaining 8.3% patients had adenocarcinoma and 4.2% patients had large cell carcinoma.

While M30 value before chemotherapy was 163.23±112.30 U/L; It was measured as 249.74±266.67 U/L 48 hours after chemotherapy ($p<0.001$). While M65 value before chemotherapy was 415.97±214.63 U/L; It was 656.65±394.15 U/L 48 hours after chemotherapy ($p<0.001$). Median survival time was calculated as 17 (2-142) months. It was found that the long-term survival of the group with a low M30/M65 ratio before chemotherapy was statistically significantly longer than the group with a high M30/M65 ratio (Figure 1). It was observed that the M30/M65 ratio, or M30 or M65 alone, measured 48 hours after chemotherapy had no predictive value for long-term survival. Chemotherapeutic agents such as paclitaxel is the mainstay of the treatment in advanced lung cancer. It is crucial to identify best candidates for the treatment.[1]

M30 and M65 are two distinctive fragments of cytokeratin 18. While M30 is released during apoptosis; M65 is released during necrosis.2 M30 was used for determination of apoptosis in many cancers including head and neck tumors, pancreatic cancer, colorectal carcinoma, melanoma, nasopharyngeal carcinoma, breast cancer, etc.[3-9] The role of M30 and M65 also investigated in lung cancer.[10]

In our previous work, we demonstrated increased levels of both M30 and M65 in this patient cohort when compared to healthy individuals.[11] In this study we aimed to evaluate the predictive effect of these markers on long term prognosis. We performed Kaplan-Meier curves depending on lower and higher levels of M30, M65 and the ratio of M30/M65 before and after the chemotherapy. Only higher ratio of M30/M65 was found to be statistically significant for longer overall survival. We speculate the higher levels of baseline apoptotic activity is important for treatment response of taxane based chemotherapy. In their study, Chu et al.[12] compared the ratio of M30/M65 in advanced lung cancer who had administered paclitaxel therapy. Unlike our results, they found that lower rates of M30/M65 after chemotherapy was a significant predictor of long-term survival. They explained this link with reduced activation of necrotic cell death by chemotherapy with paclitaxel has a better effect on treatment with NSCLC. Also, according to their hypothesis this may be due to activation

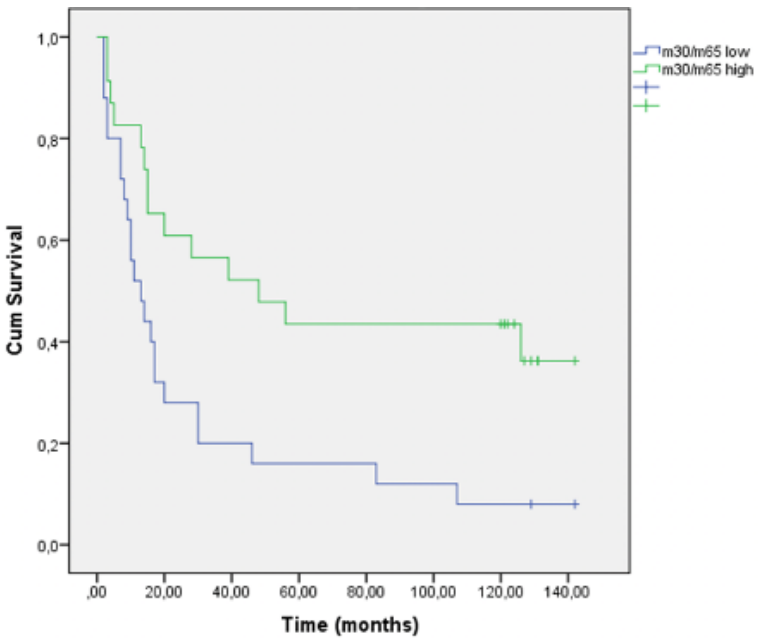

Figure 1. Comparison of survival rates regarding M30/M65 ratio before chemotherapy.

in NSCLC cells of the classic caspase-mediated apoptosis mechanism.[12]

In conclusion, the M30/M65 ratio before chemotherapy may be a prognostic factor for long-term survival in patients with advanced lung cancer. In order to better determine the importance of these prognostic markers, multi-center studies with a higher number of patients are needed.

Conflict of Interests

Authors declare that there are none.

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Short Report

The comparison of obese and non-obese persons in terms of food addiction

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Keywords: *Feeding and Eating Disorder, Addictive Behavior, Obesity*

Although the term 'addiction' was previously used only to describe excessive alcohol and substance abuse, it has recently been realized that some behaviors have a neurobiological basis similar to alcohol and substance addiction. Food addiction is one of these behaviors. In many studies, it has been suggested that especially obese and overeating people have food addiction.[1,2] The aim of our study was to compare the food addiction prevalence among obese and non-obese persons who applied to obesity and family medicine outpatient clinics.

The patients who applied to the Family Medicine outpatient clinic and obesity outpatient clinic between 15.01.2019-30.06.2019 were included in the study. The age, gender, weight, height, and smoking status of the patients were questioned. Yale Food Addiction Scale which was developed by Gearhardt et al.[1] and adapted to the Turkish language by Bayraktar et al.[2] was conducted on the individuals who volunteer to participate in the study.

195 patients without obesity and 403 patients with obesity were included in the study. The mean age and body mass index, the distribution of gender, and smoking status of the individuals were shown in Table 1. The food addiction prevalence in the obese group was 33.7% and 14% in the non-obese group. The prevalence of food addiction was significantly higher in obese individuals ($p<0.001$, OR: 3.13, 95% CI: 2.09-4.68).

The prevalence of food addiction varies between 1.60% and 24% in non-obese people and 7.7% and 56.8% in obese people in different populations.[1,3-7] Food addiction prevalence among obese and non-obese people detected in our study was in harmony with the literature. In a study comparing food addiction prevalence in obese and non-obese individuals, food addiction was more common among obese persons.[8] Contrarily, food addicted individuals did not differ in body mass index from non-addicted participants in another sample.[9] Food addiction is a discussed concept recently. Food addiction should be considered as a behavioral addiction and should be treated as an addiction. It is detected more common in obese

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Table 1. Comparison of sociodemographic features of obese and non-obese individuals

Variables	Non-obese Group n=403	Obese Group n=195
Age (years)	22.44±7.5	45.01±13.09
Gender		
Female (n, %)	264 (61.4)	166 (38.6)
Male (n, %)	179 (86.1)	29 (13.9)
BMI (kg/m^2)	22.49±2.8	35.56±4.6
Smoking		
Yes (n, %)	40 (9)	36 (47.4)
No (n, %)	403 (91)	159 (28.2)

mean±SD.

individuals than in those who are non-obese. Therefore, food addiction should be screened and treated in obese individuals.

Conflict of Interests

Authors declare that there are none.

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Short Report

Does Lymphocyte/CRP Ratio Predict Progression of Disease in COVID-19 Patients with Myocardial Injury?

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Keywords: *Covid-19, myocardial injury, lymphocyte/C-reactive protein ratio*

This study aims to reveal whether the lymphocyte-C-reactive protein ratio (LCR), a systemic inflammatory marker, predicts disease progression in COVID-19 patients with myocardial injury.

A total of 172 patients, 18 years and older, hospitalized due to COVID-19 between April 2020 and May 2020 in our hospital were included retrospectively. Hemoglobin, leukocyte, lymphocyte, neutrophil, thrombocyte, AST, ALT, creatinine, LDH, albumin, ferritin, triglyceride, procalcitonin, C-reactive protein, fibrinogen, D-dimer, troponin, LCR values of the patients were recorded. LRC ratios were amplified as x100. Patients were divided into two groups as with and without myocardial injury. IBM SPSS Statistics 21.0 program was used for statistical analysis. Statistically, $p<0.05$ was considered significant.

Study patients were divided into two groups as with and without myocardial injury. The patients' mean age was 68.56±13.43 years, 58.46±16.67 years, respectively ($p=0.002$). There was no difference between the groups in terms of the clinical severity of the disease, the severity of lung involvement on computed tomography, coronary artery disease, diabetes mellitus, chronic obstructive pulmonary disease, those with lymphocyte, C-reactive protein, LCR, ferritin, D-Dimer, and fibrinogen (all $p>0.05$). Hypertension (HT), chronic renal failure (CRF) were more common in the group with myocardial injury ($p<0.01$, $p<0.01$). Procalcitonin and creatinine levels were significantly higher in the group with myocardial injury ($p=0.002$, $p<0.001$). When we analyzed the correlation analysis of parameters associated with myocardial injury with troponin, it showed a good correlation with CRF (r: 0.484, $p<0.001$), moderate with procalcitonin (r: 0.274, $p<0.001$), and weak correlation with age (r: 0.180, $p=0.18$) and HT (r: 0.159, $p=0.37$). DM and D-dimer did not correlate (r: 0.055, $p=0.472$ and r : 0.072, $p=0.345$, respectively). In the multivariate regression analysis, only CRF (Odds ratio [OR]: 11.062 (95% confidence interval [CI]: 1.866-65.580) and reference procalcitonin levels (OR: 1.183, 95% CI: 1.014-1.379) predicted myocardial

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injury.

A recent meta-analysis evaluated laboratory test results of severe and non-severe COVID-19 cases at the time of admission. They found that lymphocytes, monocytes, eosinophils, hemoglobin, and platelet levels were significantly reduced. In contrast, high neutrophil counts were found among complete blood count indices in severe and non-severe patients. Inflammatory or infection markers (erythrocyte sedimentation rate, C-reactive protein, procalcitonin, lactate dehydrogenase, but not interleukin-6), coagulation function tests (fibrinogen, prothrombin time, and D-dimer), and glucose were positively correlated with COVID-19 severity.[1] Our study found that LRC did not predict disease progression in COVID-19 patients with myocardial injury.

Conflict of Interests

Authors declare that there are none.

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Short Report

Evaluation of Patients on the Deceased Kidney Waiting List of Bursa Uludag University Medical Faculty according to Gender, Age, Blood Type and Renal Replacement Therapies

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Keywords: *EDC, Kidney Failure, Right to Health*

In Turkey, there are 22,953 end-stage kidney disease patients on the national deceased kidney waiting list.[1] According to the 2019 Registry Report of the Turkish Society of Nephrology, the number of patients receiving hemodialysis (HD) and peritoneal dialysis (PD) treatment is 64,633.[2] However, patients registered on the national kidney waiting list consist of preemptive, PD, and HD patients. Due to the increase in the average life expectancy of the elderly population in our country, chronic kidney disease and comorbid diseases are increasing day by day.

This retrospective study evaluated the patients' characteristics on the deceased kidney waiting list from our kidney transplant center. Our center's patient data were obtained from Transplantation Dialysis Monitoring System records. Turkey's general patient data were obtained from the Turkish Statistical Institute and the 2019 Registry Report of the Turkish Society of Nephrology.

In our center, the number of patients on the deceased donor kidney waiting list was 420 (HD 301, PD 24, preemptive 95). The number of female patients was 162 (38.6%), and the number of male patients was 258 (61.4%). The age distribution of the patients; 0-9: 1.1%, 10-19: 3%, 20-29: 4.4%, 30-39: 11.6%, 40-49: 20%, 50-59: 24.4%, 60-69: 26.8%, 70-79: 7.8%, and 80-89: 0.2%. The blood group distribution of patients was consistent with Turkey's general data [0 Rh (+): 26.7%, 0 Rh (-): 3.1%, A Rh (+) 43.3%, A Rh (-) 5.7%, B Rh (+) 11%, B Rh (-) 1.7%, AB Rh (+) 8.1%, and AB Rh (-) 0.5%]. The number of male patients was higher than female patients in other age groups except the

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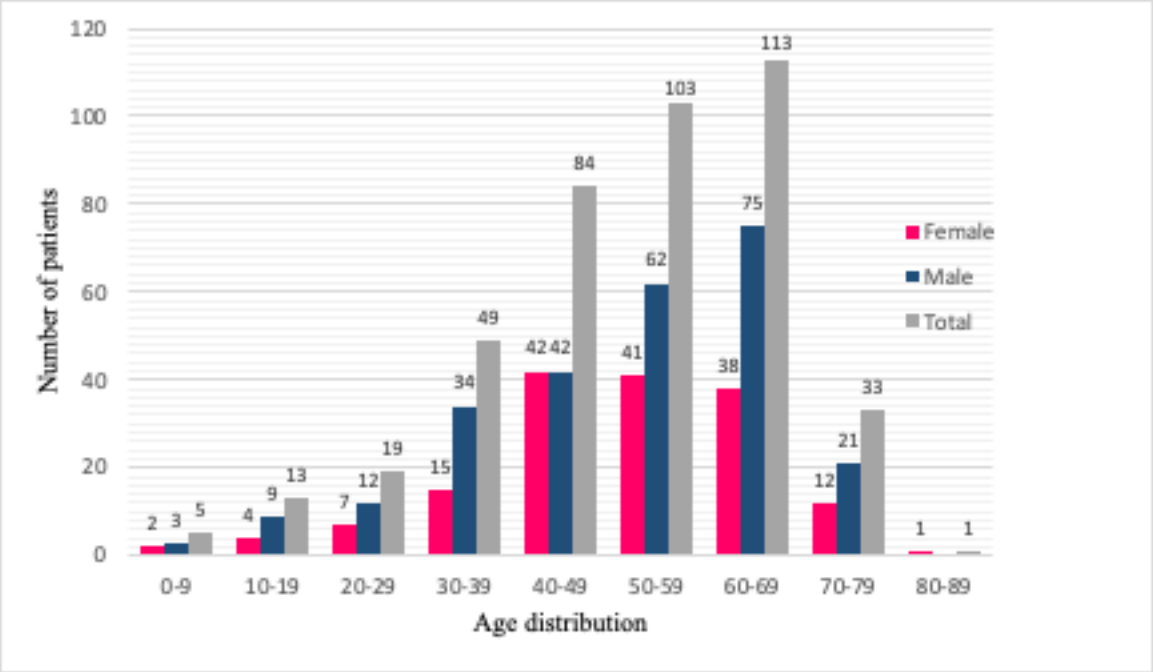

Figure 1. Gender and age distribution of the patients.

40-49 and 80-89 age range (Figure 1). Our patients' distribution by gender and kidney replacement therapies was in line with the 2019 Registry Report of the Turkish Society of Nephrology.

An increase in comorbid diseases and kidney failure in advanced age also increases health expenditures. The Republic of Turkey Ministry of Health aims to increase the awareness of kidney disease and the rate of early diagnosis with the "Turkey Kidney Diseases Prevention and Control Program.".[3] In our center, the highest number of patients on the deceased kidney waiting list was observed in the age range of 60-69, 50-59, and 40-49, respectively. Physical quality of life worsens as the dialysis period prolongs and the patient ages. Therefore, elderly HD patients need more social and mental support.[4] The age of the donor being 60 years and above is one of the extended donor criteria (EDC).[5] Using kidneys with advanced age and EDC criteria in candidates of similar age may increase both patient and graft survival and physical quality of life. For this reason, the authorized persons should be developed policies to ensure the transplantation of EDC grafts to patients who will benefit most.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Short Report

Evaluation of Clinical, Genetic and Treatment-Related Characteristics in FMF Patients by Gender Distribution

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Keywords: *FMF, Gender, MEFV*

Individuals with familial Mediterranean fever (FMF) may be exposed to stress due to gender-related differences and consequently the frequency of attacks may be different. For example, FMF attacks can be triggered in women during menstrual periods.[1,2] The aim of this study is to investigate the differences between males and females in clinical findings, hereditary characteristics, treatment responses and pathogen Mediterranean fever (MEFV) gene phenotype frequencies in FMF patients.

The charts of 213 patients who were followed up in the rheumatology outpatient clinic with a diagnosis of FMF were retrospectively reviewed. The data of 105 patients (70 females, 35 males) whose charts were available for all research data were evaluated. While evaluating the clinical findings; The age of attack onset, attack character (typical, atypical), dominant attack location (peritoneum, pleura, synovia, isolated fever), presence of recurrent fever, appendectomy history, family history (first degree, second degree) were evaluated. While evaluating the treatment response, the response (complete, partial, unresponsive) to colchicine treatment was questioned. The phenotype frequencies of the pathogen variations (M694V, V726A, M680I, E148Q) in the MEFV gene were determined. Findings were compared between groups.

The median age (minimum-maximum) in women and men was 37.5 (19-62) and 30.0 (19-59) years, respectively (p=0.148). Demographic characteristics, clinical findings, treatment responses of the participants are summarized in Table 1. The frequency of individuals with typical attack character was 71.4% in women and 82.9% in men. The frequency of the predominant attack localization with peritoneum was 90% in women and 88.6% in men. The frequency of recurrent fever in women and men was 67.1% and 65.7%, the frequency of appendectomy was 34.3% and 42.9%, and the presence of a family history was

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Table 1. Evaluation of clinical, hereditary and treatment-related characteristics of individuals with Familial Mediterranean fever disease by gender distribution

Variables	Female (n=70)	Male (n=35)	p value
Age (years)	37.5 (19-62)	30.0 (19-59)	0.148
Attack onset age (years)	15.5 (1-50)	16.0 (4-43)	0.965
Clinical, hereditary and treatment-related findings, n (%)			
Attack character			0.201
Typical	50 (71.4)	29 (82.9)	
Atypical	20 (28.6)	6 (17.1)	
Dominant attack localization			0.557
Peritoneum	63 (90.0)	31 (88.6)	
Pleura	1 (1.4)	0 (0)	
Joint	5 (7.1)	2 (5.7)	
Isolated fever	1 (1.2)	2 (5.7)	
Relapsing fever	47 (67.1)	23 (65.7)	0.884
Appendectomy	24 (34.3)	15 (42.9)	0.392
Family history	53 (75.7)	27 (76.1)	0.175
First degree	40 (57.1)	25 (71.4)	
Second degree	13 (18.6)	2 (5.7)	
Colchicine response			0.427
Good	57 (81.4)	25 (71.4)	
Limited	8 (11.4)	5 (14.3)	
No	5 (7.2)	5 (14.3)	

Table 2. Comparison of phenotypic frequencies of MEFV gene mutations according to gender distribution of individuals with Familial Mediterranean fever disease

Phenotype frequency (n, %)	Female (n=70)	Male (n=35)	p value	OR (CI)
M694V	42 (62.7)	31 (88.6)	0.006	4.613 (1.456-14.613)
V726A	15 (22.4)	5 (14.3)	0.328	0.578 (0.191-1.749)
M680I	14 (20.9)	8 (22.9)	0.819	1.122 (0.419-3.002)
E148Q	10 (14.9)	5 (14.3)	0.931	0.950 (0.298-3.033)

MEFV: Mediterranean fever, OR: Odds ratio, CI: confidence interval.

75.7% and 76.1%, respectively. The proportion of those with colchicine response was 92.8% and 85.7% in women and men, respectively. Clinical findings and colchicine response were not different between genders. The phenotype frequency of pathogen MEFV gene mutations are summarized in Table 2. The phenotype frequencies of pathogen MEFV gene mutations were 62.7% and 88.6% for M694V (p=0.006), 22.4% and 14.3% (p=0.328) for V726A, 20.9% and 22.9 for M680I (p=0.819) and 14.9% and 14.3% (p=0.931) for E148Q in women and men, respectively.

Clinical findings and treatment responses are not different in individuals with FMF disease. The frequency of the M694V mutation, which has high penetration and is associated with important complications such as amyloidosis, is higher in men. There is a need for studies to evaluate FMF activity according to gender distribution.

Conflict of Interests

Authors declare that there are none.

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Short Report

Retrospective Evaluation of The Cases with Malignant Pheochromocytoma: A Single Center Experience

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Keywords: *Malignant pheochromocytoma, surgery, metastasis, Lutetium-177, radiotherapy*

Pheochromocytomas are neuroendocrine tumors that arise from chromaffin cells of the adrenal medulla.[1] It is a rare disease with an estimated annual incidence of 0.8 per 100.000 person years.[2] Pheochromocytomas are most common in the fourth to fifth decade with equal distribution in men and women.[3] The disease is mostly sporadic but forty percent of the cases are part of the familial disorders such as von Hippel-Lindau (VHL) syndrome, multiple endocrine neoplasia type 2 (MEN2), and less commonly, neurofibromatosis type 1 (NF1).[4] Approximately 10 percent of pheochromocytomas are malignant (8.3% to 13%).[5] Local invasion into surrounding tissues and organs or distant metastases that may occur anytime can allow to make a distinction of benign from malignant type.[6,7] All pheochromocytomas have some metastatic potential according to World Health Organization (WHO) because metastasis can appear as many as 53 years after resection.[8] Metastasis may occur frequently in lymph nodes, bone, liver and lungs.[9]

The clinical picture is almost same as benign pheochromocytoma. The classic triad of symptoms in cases consists of episodic headache, sweating, and tachycardia.[10] Pheochromocytoma sometimes is diagnosed with imaging methods in patients with unrelated symptoms.[11] After the biochemical diagnosis, localization of the tumor is made by computed tomography (CT), magnetic resonance imaging (MRI) first. If the tumor is not find by abdominal and pelvic CT or MRI, metaiodobenzylguanidine (MIBG) scintigraphy, fludeoxyglucose-positron emission tomography (FDG-PET) and gallium 68 1, 4, 7, 10-tetraazacyclododecane-1,4,7,10-tetraacetic acid-octreotate-positron emission tomography (68-Ga DOTATATE PET) can be done.[12-14]

There is no curative treatment for malignant pheochromocytoma. If possible both primary and metastatic lesions should be resected which may improve symptoms and possibly survival.[15,16]

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Table 1. Clinical characteristics of the patients and some features of the tumors

Characteristics		Number (n)
Gender (female)		3
Age at presentation (years)		45
Type of presentation		
Hypertension		3
Asymptomatic		2
Macroscopic features		
Tumor localization	Right adrenal	2
	Left adrenal	2
	Bilateral	1
Tumor size (mm)		58
Microscopic features	Capsular invasion	1
	Vascular invasion	1
	Extension into periadrenal adipose tissue	2

External radiation therapy for bone metastasis, cryoablation, radiofrequency ablation or transcatheter arterial embolization for hepatic metastasis are other treatment modalities for malignant pheochromacytoma.[17] I123 MIBG treatment is another therapeutic option for palliation.[18] Chemotherapy may prolong the survival of the cases and play a role in the palliation.[17] Furthermore, medical control of symptoms with adrenergic blockage is important. The prognosis of malignant pheochromocytoma is variable; the overall 5-year survival that range widely from 12 to 84 percent.[19]

In this study, we evaluated the patients with malignant pheochromocytoma who were followed up by our clinic. We retrospectively evaluated the data of cases admitted to our department between 2013 and 2020 and diagnosed with malignant pheochromocytoma in this study. Three patients with a diagnosis of malignant pheochromocytoma were female and mean age at presentation was 45 years. The baseline characteristics of the patients, imaging and pathological features of the tumors were demonstrated in Table 1.

Among five cases, three were diagnosed with pheochromocytoma after hypertensive attack. Two cases were diagnosed after detection of adrenal mass in one with abdominal pain and one with elevated liver enzymes. Noradrenergic functionality was detected in all cases. Primary site of the tumor was the left side in two patients, the right side in two patients and bilateral in one patient. The average diameter of the adrenal mass of the cases was 58 mm. There was no familial pheochromocytoma syndrome in our cases. Transperitoneal surrenalectomy was performed in all cases. Extension to periadrenal adipose tissue was detected in two patients, vascular invasion in one patient and capsule invasion in 1 patient in the surgery-resected specimen. Intraabdominal lymph node metastasis was found in 1 patient and metastatic focus was seen in the liver in 1 patient at the time of the diagnosis. During follow-up, metastasis was detected in the perirenal region in 1 patient. Bone metastasis was seen in 3 patients, lymph node metastasis in 2 patients and lung metastasis in 1 patient. Surgery followed by Lutetium-177 treatment as an adjuvant therapy in two patients. Transperitoneal surgery was performed for a patient with metastasis in the perirenal area and resection of the mass was performed. Also, a patient with a liver metastasis at the time of the diagnosis was underwent surgery and the metastatic focus was resected. Two patients received radiotherapy for bone metastases. The mean follow-up period of the cases was 44 months. Three patients died from progression of the disease or acute complications and the other 2 patients are still under follow-up.

There is no curative therapy for malignant pheochromocytoma. After resection of primary tumor, metastatic foci should be resected entirely as possible. Multidisciplinary and individualized approach to treatment of patients with metastatic pheochromocytoma is warranted. Our study has retrospective design and the relatively low number

of patients. Better therapeutic approach can be established by future studies.

Conflict of Interests

Authors declare that there are none.

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Short Report

Evaluation of Death Rates of Patients on The Deceased Kidney Waiting List of Bursa Uludag University Faculty of Medicine According to Years by Gender, Age, Blood Group and Dialysis Types

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Keywords: *WHO, kidney failure, death*

There are 22,953 end-stage kidney disease patients on the national deceased kidney waiting list.[1] According to the 2019 Registry Report of the Turkish Society of Nephrology, the number of patients receiving dialysis is 64,633. The number of patients receiving hemodialysis (HD) and peritoneal dialysis (PD) is 61,341 and 3,292, respectively.[2] However, patients registered on the national kidney waiting list consist of preemptive, PD, and HD patients. Due to the increase in the average life expectancy of the elderly population in our country, chronic kidney disease and comorbid diseases are increasing day by day.

In this study, the characteristics of the patients who died on the deceased kidney waiting list of our kidney transplant center were evaluated retrospectively. Our center's patient data were obtained from Transplantation Dialysis Monitoring System records. Turkey's general patient data were obtained from the Turkish Statistical Institute and the 2019 Registry Report of the Turkish Society of Nephrology.

In our center, the number of patients who died on the kidney waiting list from deceased donors was 350 (HD: 273, PD: 73, and preemptive: 4). The number of female patients was 134 (38.2%), and the number of male patients was 216 (61.4%). Our patients' distribution by gender and kidney replacement therapies was in line with the 2019 Registry Report of the Turkish Society of Nephrology. The number of patients who died by year is shown in Figure 1.

In our study, deaths between the age range of 50-59 and 60-69 registered in our center and died

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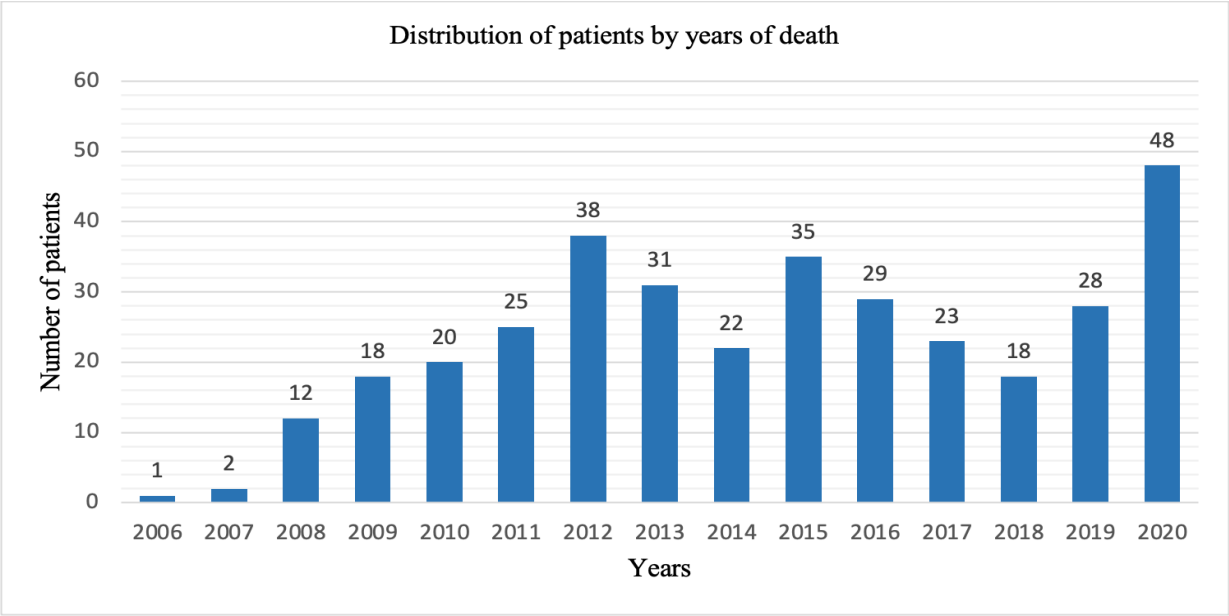

Figure 1. Distribution of the number of patients who died by years.

constitute 56% of the total deaths. Patients with chronic kidney failure have a high risk of death between the ages of 50 and 69. In one study, the course of COVID-19 disease caused by the SARS-CoV-2 virus is terrifying in elderly, co-morbid diseases, and male patients.[3] Also, COVID-19 is severe and fatal in chronic kidney disease. With the COVID-19 disease being a pandemic in the world in 2020, according to the data of the current World Health Organization (WHO) dated 07.02.2021, 105,394,301 cases were detected in the world 2,302,302 deaths occurred.[4] When we look at the distribution of cases by year, the death rate in 2020 is the highest. This situation may be related to COVID-19 disease. However, we could not be obtained the causes of death of these cases from the death notification system of the Ministry of Health, and the relationship with COVID-19 could not be clearly determined.

Due to the high mortality rates in the range of 50 to 69 years of age of patients in kidney transplant waiting lists, especially for patients with advanced age with chronic kidney disease "Turkey Kidney Diseases Prevention and Control Program"[5] to be controlled although work on the policy for new infectious diseases needs to be improved.

Conflict of Interests

Authors declare that there are none.

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Short Report

May the Neutrophil Lymphocyte Ratio be a New Marker for Uveitis Development in Ankylosing Spondylitis?

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Keywords: *Ankylosing spondylitis, disease activity, neutrophil lymphocyte ratio, uveitis*

Spondyloarthropathies (SpA) includes several rheumatic disorders that share clinical, genetic and radiographic features. Ankylosing Spondylitis (AS) is an autoimmune disease of unknown cause belonging to the group of spondyloarthritides associated with the human leucocyte antigen (HLA)-B27. It is a chronic, progressive, multi-system inflammatory disorder which primarily involves the sacroiliac (SI) joints and the axial skeleton. Peripheral joints and tendons can also be affected. Inflammatory low back pain is main symptom for diagnosis of AS. Although, the etiology is unknown, but there is evidence about genetic predisposition, HLA-B27 gene is commonly present. The prevalence of AS is variable, the disease is more common in Caucasians than in other races. Typically, the prevalence of AS in a population reflects the associated prevalence of HLA-B27 gene in that population.[1] A recent study reported a mean AS prevalence per 10,000 population as 23.8 cases in Europe, 16.7 in Asia, 31.9 in North America, 10.2 in Latin America and 7.4 in Africa.[2] Inflammation of the anterior uveal tract is known as anterior uveitis or iritis. Acute anterior uveitis (AAU) is the typical uveitis associated with SPA and is the most common extraarticular clinical feature of SPA. AAU occurs in about 25% to 40% of patients with AS[3] of whom approximately 90% are HLA-B27 positive.[4] Neutrophil-lymphocyte ratio (NLR) is a marker that has been researched in recent years to be used as a marker of inflammation. The aim of our study is to evaluate the NLR in AS patients with uveitis, which is an extraarticular involvement, while having uveitis and to compare it with the NLR at first admission.

Ninety patients with uveitis and diagnosed with AS according to the modified New York Criteria were included in the study. The files of the patients were analyzed retrospectively. Demographic data and laboratory parameters were recorded. NLR was calculated arithmetically using neutrophil and lymphocyte values in complete blood count. 53 (58.9%) of the patients were male, 37 (41.1%) were female. The mean age was 42.51±9.23 years in male, 45.84±9.78 years in female, the mean

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duration of diagnosis was 14.53±10.34 years in male, 10.84±6.01 years in female, and the mean body mass index (BMI) was 27.61±3.53 kg/m^2 in male and 25.82±3.12 kg/m^2 in female. There was no significant difference between the genders in terms of age, duration of diagnosis, and BMI (p=0.104, p=0.073, p=0.557, respectively). No significant difference was found between genders in terms of NLR values at first admission and at the time of uveitis (p=0.016). There was a significant difference between the NLR values (1.660±0.67 and 2.623±1.293, respectively) in both genders at first admission and at the time of uveitis (p<0.001).

In studies, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) are used as activation indicators. There are studies in the literature showing that NLR is an important marker for determining disease activity and inflammation in AS patients. Gokmen et al. compared the NLR of AS patients with healthy individuals in their study. Together with acute phase reactants such as CRP, it has been found to be a useful marker in showing inflammation.[5] In the meta-analysis of Shanshan Xu et al. including 10 studies, it was shown that NLR is a marker that can be used to show systemic inflammation in AS patients compared to healthy controls.[6] In the study of Kucuk et al.[7], NLR was found to be significantly higher in AS patients with severe disease activity than in AS patients with mild disease activity. In the study conducted by Coskun et al.[8], In AS patients with high disease activity, NLR was found to be significantly lower in the 3rd month of the treatment compared to the start of treatment with an anti-tumor necrosis factor drug (anti-TNF). Uveitis is an extraarticular manifestation seen in 25% to 40% of AS patients. It is correlated with disease activity. In the literature, there is no study about NLR comparison in AS patients with developing uveitis. In our study, we compared the NLR values of AS patients at the first admission and at the time of having uveitis. We found that NLR was significantly higher in both genders while having uveitis.

In conclusion NLR can be used as a marker for the development of uveitis in AS patients and correlates with disease activity.

Conflict of Interests

Authors declare that there are none.

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Short Report

General characteristics of the patients diagnosed with Enteropathic arthritis (ENA) whose treatment have been performed with biological agents

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Spondylarthritis (SpA) comprises multiple divergent forms of inflammatory arthritis. It has got extraarticular symptoms like uveitis, psoriasis and inflammatory bowel diseases (IBD). Enteropathic arthritis (ENA) (in another naming, arthritis due to IBD ranks in SpA group because of the similarity of clinical features and the common genetic susceptibility as well. The term of ENA is used for the patients who has ulcerative colitis or Chron's disease and developed inflammatory arthritis as well.[1] The most frequent extraintestinal symptom in the individuals with IBD is the articular ones. The frequency of SpA in IBD patients is 28.7%.[2] The male/female ratio among ENA patients' uniform. Peripherial joint involvement or axial spine contagion have been reported in 17-39% of the IBD patients. Sometimes arthritis might originate before the diagnosis of IBD.[2] Non-steroidal anti-inflammatory drugs (NSAID's), corticosteroids, sulfasalazine, anti-TNF agents can be used in the treatment of the patients with axial or peripheral involvement. In this study, we aimed to present the features of 33 ENA cases that have been being monitored in our center.

Demographical and clinical features, presentation forms, family histories, smoking habits, HLA-B27 positivity of the 33 ENA cases who sought medical service in our outpatient clinic and were diagnosed with ENA and treated with biological agents have been recorded. Their peripheric arthritis, dactylitis, enthesitis, uveitis, and psoriasis stories have been picked. It has been interrogated before biological agents treatment whether conventional DMARD's (disease-modifying antirheumatic drugs) were used.

Sixteen of the patients (48.4%) have been females, whereas 17 of them (51.6%) have been males. Eleven of the patients (33.3%) have been diagnosed with Chron's disease, whereas 22 of them (66.7%) with ulcerative colitis. Eighteen patients (54.5%) have presented with joint complaints, 13 of them (39.4%) with bowel complaints, and 1 of them (3%) has presented with simultaneous joint and bowel complaints. Families of the 2 of the 18 patients (6.1%) whose family stories could have

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been achieved had rheumatic disease background. Fourteen of the 26 patients (42.4%) whose smoking characteristics could have been identified, have been active smokers. HLA-B27 of the 12 patients (36.4%) have been positive. Sacroiliitis have been identified in the graphics of the 26 patients (78.8%). Thirty-four patients (93.9%) had axial skeleton involvement whereas 11 of them (24.2%) had joint involvement. Eight of the patients (24.2%) had peripheric arthritis, 2 of them (6.1%) had enthesitis, 6 of them (18.2%) had heel pain. The inquiry of the pre-treatments before the biological agents among the patients whose data could have been achieved presented that 6 of them (18.2%) used methotrexate, 16 of them (48.5%) used steroids, 16 of them (48.5%) used azathioprine, 16 of them (48.5%) salazopyrin, 23 of them (69.7%) used NSAID's. Seventeen of the patients (51.5%) have used NSAID's after IBD diagnosis whereas 4 patients (12.1%) had IBD inflammation symptoms as bleeding and diarrhea due to NSAID's use after IBD diagnosis.

Since there is a close link between SpA and ENA, patients should also be monitored in the aspect of bowel symptoms during the SpA course. Even if it is rare, the initial seeking way of the patients with IBD might be joint complaints. Hence joint complaints must be taken into consideration. Early diagnosis is substantial with regards to treatment options. It is needed to take into account the activation of intestinal symptoms as well, while deciding on the treatment of the patients with enteropathic arthritis.

In a systematic review and metanalysis in which 13 studies evaluating the effects of NSAID's use on intestinal findings in patients with IBD; It has been shown that NSAID's do not cause intestinal exacerbation in both CH and UC.[3] Anti-TNF agents; It has proven to be highly effective in the treatment of IBD patients who are steroid-dependent or refractory to conventional therapy and in extraintestinal findings. Anti-TNF agents are effective in both axial and peripheral joint involvement in patients with entropathic arthritis. Infliximab, adalimumab and certalizumab are indicated in the treatment of remission induction and remission maintenance in Crohn's disease.[4] In ulcerative colitis, infliximab, adalimumab and golimumab are indicated for remission induction and remission maintenance therapy.[5] Thus the choice and the configuration of the treatment are important for the course of the disease. The well-being can be achieved in both the articular and the intestinal symptoms of the patients thanks to the common medications to be used in the treatment.

Conflict of Interests

Authors declare that there are none.

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Short Report

Single Center Experience in Patients with A Diagnosis of Nodular Lymphocyte Predominant Hodgkin Lymphoma

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Keywords: *Nodular lymphocyte predominant Hodgkin lymphoma, Hodgkin lymphoma, prognosis*

Nodular Lymphocyte Predominant Hodgkin Lymphoma (NLPHL) is a rare hematological malignancy with an excellent prognosis. It is about 5% of Hodgkin lymphoma cases. The incidence of the disease is 8-9 per 10 million. In this study, we planned to evaluate the clinical features and prognosis of patients diagnosed with NLPHL in the last ten years followed in the hematology department.

In our study, the data of 10 patients who were diagnosed with NLPHL between January 2010 and December 2020 in Bursa Uludag University Faculty of Medicine, Department of Hematology, were evaluated retrospectively. 2.2% of 450 Hodgkin lymphoma-diagnosed patients were diagnosed with NLPHL, in 10 years. 80% of patients were male. Median age was found 36 years (28-60). At the time of diagnosis, all patients had ECOG score of 0 and 80% of the patients were at early stage. According to International Prognostic Score (IPS), patients were low risk. All patients who applied to the hospital, complained about palpable lymphadenopathy at first. None of the patients had B symptoms. Hemograms and biochemical parameters were in reference intervals at the time of diagnosis. Immunohistochemical staining of the biopsies showed that all specimens were positive for CD20, but just one of them positive for CD30. Eight patients were administered ABVD (doxorubicin, bleomycin, vinblastine, dacarbazine) chemotherapy as first-line therapy. On the other hand, two patients received coadministration of radiotherapy (RT) with ABVD. Two patients who were refractory first-line therapy, took DHAP (dexamethasone, cytarabine, cisplatin) as salvage chemotherapy regimen. Patients, completed remission after treatment, had autologous stem cell transplant (ASCT) after completion of BEAM (carmustine, etoposide, cytarabine, melphalan) conditioning regimen. One of our transplant patients recurred 5 months after the transplant. Gemcitabine chemotherapy was given to the patient who developed recurrence. He died due to sepsis in the third cycle of the treatment and in the 10th month of the transplant. Other patient is still alive and followed up in remission. Kaplan Meier

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survival analysis computed four-year survival rate 80%. Overall survival could not be estimated. Median survival time was 42.2 months.

NLPHL is a less common disease that differs from classical Hodgkin lymphoma in histology, course, and treatment. It is associated with a favorable prognosis. The most important problems with the management of the disease are relapses, transformation to non-Hodgkin lymphoma, and treatment-related toxicities. There are no randomized controlled trials with its treatment due to its rarity. All treatment options are based on retrospective studies involving a small number of patients. In early-stage disease, untreated follow-up, surgery, involved area radiotherapy, and single-agent rituximab are preferred. Chemo-immunotherapy may be preferred in early disease with high tumor and symptom burden. In advanced-stage disease, radiotherapy can be added to chemotherapy and chemoimmunotherapy. Due to the slow course of the disease, long-term side effects should be observed. Good responses can be achieved in relapsed disease. The risk of transformation is high. It is reasonable to obtain re-biopsies at relapses. Transformed disease and the primary disease have been shown to be clonally related in most cases. Prognosis of transformed disease which is treated with salvage chemo-immunotherapy followed by autologous stem cell transplantation is similar to that of de novo diffuse large B-cell lymphoma.[1-3] NLPHL patients diagnosed in our clinic are incompatible with the incidence rates stated in the literature. It is suitable to discuss and compare with difference between our data and other data, that has been reported from Turkey.

Conflict of Interests

Authors declare that there are none.

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Short Report

Venetoclax and Azacytidine in Relapsed/Refractory Acute Myeloid Leukemia and High Risk Myelodysplastic Syndrome: Bursa Uludag University Experience

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Keywords: *Venetoclax, azacitidine, acute myeloid leukemia, myelodysplastic syndrome*

Acute myeloid leukemia (AML) is the deadliest leukemia in adults, with a mean age of 67 years at the time of diagnosis.[1,2] Approximately 20-50 percent of patients do not achieve a complete remission with intensive induction chemotherapy, and most patients' relapse.[3] Azacytidine, one of the hypomethylating agents, is commonly used to control the disease. Venetoclax is a BCL-2 inhibitor that is synergistic with azacytidine. The addition of venetoclax to the treatment is based on a randomized trial that improves survival and increases response rates.[4,5] This study aims to evaluate the efficacy and safety data of venetoclax+azacytidine combination, a new treatment modality in relapsed/refractory (R/R) AML and high-risk myelodysplastic syndrome (MDS).

R/R AML and high-risk MDS patients aged ≥18 years were included in the study. The off-label use of drugs for each patient was received permission from the Turkish Medicines and Medical Devices Agency. Venetoclax was given orally on day 1: 100 mg, day 2: 200 mg, next days: 400 mg/day. Azacytidine 1-7. days (every 28 days) 75 mg/m² administered subcutaneously (alternatively, 50 mg/m² as 5-2-5). All patients received prophylaxis for tumor lysis syndrome (TLS) for at least three days. In patients receiving antifungal therapy that inhibits CYP3A enzyme activity, the dose of venetoclax was reduced by 75%.

A total of 15 patients, 14 R/R AML and 1 MDS RAEB-1, followed in the Bursa Uludag University Hematology Department was included in the study. 60% (n=9) of these patients were female, and the median age was 65 years (32-84). 50% (n=7) of AML patients were de novo leukemia. Fourteen patients were between ECOG 0 and 2. Fourteen were in the medium, and one was in the high cytogenetic risk group. The treatment indication was salvage therapy in 13 patients and consolidation in 2 patients. Median 2 cycles (1-4) of venetoclax+azacytidine chemotherapy was

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applied to the patients. The median follow-up time was 1.2 months (0.1-6.5). The median time to first response with treatment was one month (0.5-5). The refractory disease was persistent in all four patients who were found to have a refractory disease at the end of the first cycle. The best responses obtained with venetoclax+azacytidine were complete remission (CR) in one patient, complete remission with incomplete hematologic recovery (CRi) in one patient, morphological leukemia-free state (MLFS) in 4 patients, and partial response (PR) in one patient. The remission duration in 2 patients with composite remission (CR + CRi) was 3.5 and 17 months. Minimal residual disease was negative in only one patient. Venetoclax+azacytidine was used as a bridge therapy to allogeneic transplant in one patient. With Venetoclax+azacytidine, erythrocyte and platelet replacement requirement decreased in only 13.3% (n=2) of the patients. Grade 3 and above hematological toxicity was observed in 14 patients. The most common non-hematologic toxicities were fatigue-anorexia (80%), pneumonia (25%), and associated with the gastrointestinal tract (40% nausea and vomiting, 13% diarrhea). 66.6% of the patients were complicated with infection. Two patients had COVID-19 infection and recovered with treatment. TLS was developed in 2 patients. One of these patients had TLS at the time of diagnosis. Venetoclax dose reduction or an interruption was required in 60% (n=9) patients. The mortality rate in the first 30 days was 20%.

The management of AML gradually turns into an individualized approach to applying targeted therapies. The time to enter early remission is critical. For this reason, deaths due to infection should be prevented. Although the toxicities of this combination therapy can be managed, close follow-up of the patients is mandatory. Venetoclax+azacytidine combination therapy is relatively well-tolerated, can improve blood counts, relieve symptoms, improve life quality, and prolong survival. Still, more studies are needed to assess long-term disease control.

Conflict of Interests

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Short Report

Polycystic Renal Disease Presented by Anuria and Nephrolithiasis Associated Progressive Kidney Damage: A Case Report

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Keywords: *Anuria, Nephrolithiasis, Polycystic renal disease*

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited kidney disease.[1] Kidney stones develop in 20-30% of patients with ADPKD and urinary stones are usually treated with conservative methods (urinary alkalinization, spontaneous stone passage, extracorporeal shock wave etc.).[2,3] Uric acid stones and calcium oxalate stones are the most frequently detected stones and seen in similar proportions.4 Hydronephrosis, which is the most valuable radiological finding in the diagnosis of stone-related postrenal insufficiency, may be difficult to differentiate from common cysts in ADPKD patients.[3] When kidney dysfunction develops in ADPKD patients, glomerular filtration rate (GFR) loss reaches an average of 4.4 to 5.9 mL/min per year.1 Faster deterioration in these patients requires investigation for prerenal factors such as dehydration that triggers acute kidney damage or stone-related postrenal factors.[2] In ADPKD, stone-related postrenal obstruction should be considered in rapid GFR losses despite negative ultrasound report for hydronephrosis as there is frequent occurrence of kidney stones and the difficulty in detecting hydronephrosis with ultrasonography (US) in these patients.[5] Here, we presented a case of ADPKD who developed stone-related renal dysfunction during chronic follow-up, and renal function improved after the intervention.

The 73-year-old patient, who was diagnosed with ADPKD and was in outpatient follow-up with basal creatinine level of 1.6 mg/dL and GFR: 62 mL/min, had a history of type 2 diabetes, hypertension and occlusive type of cerebrovascular events. During the follow-up, the creatinine level increased from 1.6 mg/dL to 3.6 mg/dL. In the evaluation, there was no new drug use or fluid loss suggesting a prerenal event. The patient, who had no signs of pain, bleeding, and urinary tract infection, was considered to have the accelerated natural course of ADPKD, considering the high creatinine level and accompanying diseases. In the evaluation one week later, the patient's general condition was worsened, the creatinine value increased to 7.1 mg/dL and GFR was 10 mL/min then patient has hospitalized for further investigation and treatment. No postrenal pathology was detected in US performed to detect hydronephrosis for the diagnosis of postrenal renal insufficiency in the patient who has been on emergency hemodialysis due to uremic symptoms. In order to explain the current situation, noncontrast computed tomography (CT) has been performed and it showed dilatation in the pelvicalyceal system of both kidneys and stones in the distal ureters on the both sides, which were 9.5x4.5 mm and 5.5x3 mm, respectively. In addition, right indirect inguinal hernia was observed simultaneously in the case. In the emergent bilateral ureterorenoscopy performed by the urology clinic, the stones causing complete

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obstruction at the bilateral distal ureter level were observed. Bilateral Double J stents were placed to the bilateral collecting system in the same session. After the procedure, urine output is observed, and the creatinine level decreased to 1.7 mg/dL, which is the basal level.

After development of the renal failure in ADPKD, progression is usually rapid and varies according to genetic and clinical risk factors.6 It should be kept in mind that stones are observed with high frequency in these patients and unexplained accelerated progression in deterioration may be caused by obstruction in the urinary system.[2,7] Cysts in both kidneys that disrupt the normal anatomy can easily be confused with pelvic enlargement due to obstruction. Therefore, in patients with a diagnosis of ADPKD who are investigated for postrenal kidney failure, pelvic enlargement may not be differentiated by US because of the abundant cysts. In US, which is inexpensive, practical, lack of radiation exposure and the first choice in the evaluation of postrenal pathologies, pelvicalyceal dilatation can easily be evaluated as a cyst in these patients. Obstruction due to stones in these patients may result in irreversible renal failure when not diagnosed in the early period.[5,8]

Stones may cause acute renal failure in ADPKD, usually due to infection or urosepsis, persistent pain, vomiting and rarely bilateral obstruction in the ureteropelvic region, and urgent intervention is required.[9] In the present, extracorporeal shock wave (ESWL), ureteroscopy, percutaneous nephrolithotomy (PCNL) and retrograde intrarenal surgery (RIRS) are the procedures commonly used in the treatment of urinary stone disease.[5,10]

The prevalence of stones in patients with ADPKD has been reported in variable rates in the literature, and the prevalence of urinary stones varies between 3% and 59%. In cases with urolithiasis, the rate of patients who undergo interventional treatment is between 1-8%, but there is no meaningful data on the rate of stone-related interventions in adults with ADPKD.[2,7] Stone-related pain in ADPKD can be confused with pain due to cyst rupture. However, it should be considered in patients with acute GFR losses under chronic follow-up, as in our case, and advanced imaging with low-dose non-contrast CT should definitely be performed in these patients if the diagnosis cannot be made by US.[3]

Imaging methods, especially US, may be insufficient to distinguish pelvicalyceal stones because some of the cysts may contain wall or septa calcification or calcium milk. Diagnostic uncertainty may occur in the detection of kidney stones. Therefore, the best imaging method is high-resolution CT that detects stones and calcifications. Studies suggest that non-contrast CT is the preferred imaging method for the diagnosis of suspected nephrolithiasis in ADPKD.[5,8]

We presented this case in order to explain that nephrolithiasis accompanying polycystic kidney disease may lead to clinical picture of postrenal acute renal failure, anuria and progressive kidney damage, and that it should be accurately diagnosed with appropriate imaging technique in order not to be overlooked, as this problem can be reversed with appropriate intervention.

Conflict of Interests

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TURKISH JOURNAL OF INTERNAL MEDICINE

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Short Report

Polypharmacy and Potential Inappropriate Drug Use in The Elderly Admitted to the General Internal Medicine Outpatient Clinic

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Keywords: *Polypharmacy, potential inappropriate drug, elderly*

The frequency of chronic diseases, the number of drugs used, the rates of polypharmacy and consequently, the risks of potentially inappropriate drug (PID), drug-drug interactions and drug adverse reactions have increased in the elderly.[1,2] In this study, we aimed to investigate the rates of polypharmacy and PIDs use in elderly patients who admitted to General Internal Medicine Outpatient Clinic of Uludag University Hospital. It was planned as a prospective cross-sectional study. The sociodemographic characteristics, diagnoses, concomitant chronic diseases and the drugs they used were recorded in detail through face-to-face interviews with the patients on the previously prepared questionnaire form. Then, the files of the patients were examined, and the diseases and drugs information were confirmed.

In this study, daily use of 5 drugs was accepted as polypharmacy.[3] After then, the drugs used by the patients were evaluated in terms of PIDs according to the 2015 Beers Criteria.[4] According to these criteria, PIDs are grouped as follows;

1. Table 2-related PIMs: Taking drugs that should be avoided in the elderly.
2. Table 3-related PIMs: Taking drugs that should be avoided due to drug-disease or drug-syndrome interactions in the elderly.
3. Table 4-related PIMs: Inappropriate use of drugs that should be used with caution in the elderly.
4. Table 5-related PIMs: Taking drugs that should be avoided due to clinically important drug-drug interactions in the elderly.
5. Table 6-related PIMs: Inappropriate use of drugs that should be avoided or reduced in dosage due to renal impairment in the elderly.

SPSS 21 package program was used for data analysis. Pearson Chi-Square and Fisher's exact Chi-Square tests were used for comparison of categorical variables, the Kruskal-Wallis test was used to compare more than two independent groups, and the Mann-Whitney U test was used to compare two independent groups.

In a 4-month period (from September 1 to

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Table 1. The distribution of the number of drugs used and potential inappropriate drugs by gender and geriatric age groups

	Gender		Total	Age Group	
	Female	Male		65-74 years	≥75 years
Number of drugs used					
No drug	6 (3.4%)	5 (4%)	11 (3.6%)	10 (4.1%)	1 (1.6%)
- 1-4 drugs	73 (40.8%)	59 (47.2%)	132 (43.4%)	105 (43.2%)	27 (44.3%)
- 5-9 drugs	86 (48%)	55 (44%)	141 (46.4%)	114 (46.9%)	27 (44.3%)
- ≥10 drugs	14 (7.8%)	6 (4.8%)	20 (6.6%)	14 (5.8%)	6 (10%)
Potential inappropriate medications					
Table 2-related PIDs	54 (30.7%)	32 (25.6%)	86 (28.3%)	53 (21.8%)	33 (54.1%) *
Table 3-related PIDs	7 (3.9%)	5 (4%)	12 (3.9%)	8 (3.3%)	4 (6.6%)
Table 4-related PIDs	6 (3.4%)	5 (4%)	11 (3.6%)	3 (1.2%)	8 (13.1%) *
Table 5-related PIDs	8 (4.5%)	4 (3.2%)	12 (3.9%)	8 (3.3%)	4 (6.6%)
Table 6-related PIDs	2 (1.1%)	1 (0.8%)	3 (9.9%)	2 (0.8%)	1 (1.6%)

* $p<0.001$

December 31, 2020), 304 (58.9% female, 41.1% male) patients who gave informed consent were included in the study. The mean age of the patients was 71.5±5.2. 95.8% had at least one concomitant chronic disease, the mean number of chronic diseases was 2.7±1.6 and the most common chronic diseases were hypertension (67.1%) and diabetes mellitus (39.8%). The daily mean number of drugs used by the patients was 4.9±3.3 and the rate of polypharmacy was 52.9%. A total of 124 PIDs were observed in 104 (34.2%) patients, and this rate was higher in patients with polypharmacy ($p<0.05$) (Table 1). Table 2- and Table 4-related PIDs were higher in the ≥75 age group. There was no difference between gender and age groups in terms of other categories. The most common PID was the use of drugs that should be avoided in the elderly (Table 2-related PIDs, 28.3%). On the basis of drugs, the most common of these was the inappropriate use of proton pump inhibitors (PPI) (n=30, 9.9%) and potent anticholinergics (n=23, 7.6%).

The rates of polypharmacy were reported as 47.6% in a study that patients admitted to a geriatric outpatient clinic in Turkey[5], 59.6% in a study that patients living in nursing homes in Turkey[6], 47% in a study that patients admitted to the hospital in Norway[7] and 51.9% in another study in Italy.[8] The rates of PIDs in studies using Beers 2015 criteria were reported as 60% in a study in two assisted living facilities in the USA[9] and 29.3% in another study at the time of admission to internal medicine and surgical services in Jordan, and in the latter study PPIs were the most common PIDs.[10] Similarly, in this study, we observed that the number of chronic diseases, polypharmacy and PIDs rates were high in the elderly, and there was a linear correlation between polypharmacy and PIDs, and PPIs were the most commonly used PIDs. Therefore, in order to reduce the possible risks that may occur in the elderly, patients should be examined by a comprehensive geriatric assessment, drugs should be prescribed according to rational drug use recommendations, the drugs used in terms of PIDs should be evaluated, and patients should be explained in detail how to use their drugs. Then, at each visit, they should be carefully questioned in terms of how they use the drugs and drug adverse effects.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

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Case Report

Heart Failure Related to Large Hiatus Hernia: a Case Report

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Abstract
Heart failure is a disease that may present with various presentations depending on different etiological factors. External pressure on the heart is one of the rare causes that can cause heart failure. Here, we presented a 73-year-old female patient with inoperable endometrial cancer who developed heart failure due to hiatal hernia compression.

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Keywords: *Hiatus hernia, heart failure, hypotension*

Introduction

Hypotension is defined as a blood pressure less than 90/60 mmHg. Its etiology includes anaphylaxis, cardiac tamponade, right heart failure, hemorrhage, dehydration, shock, adrenal insufficiency, life-threatening arrhythmias, tension pneumothorax, and pulmonary embolism. Mass, lymph node, or hiatal hernia, which can cause heart failure by external pressure on the heart, are less common causes. Hiatal hernia is the herniation of organs in the abdominal cavity to the supradiaphragmatic region through the esophagus's transition point in the diaphragm. It appears more frequently age-dependent as a result of abrasions due to phrenoesophageal membrane degeneration.[1]

Case Report

The 73-years-old patient, who was followed up in the palliative inpatient clinic with the diagnosis of inoperable endometrial cancer, was evaluated with low blood pressure despite fluid replacement and inotropic therapy. In the physical examination, the patient had a blood pressure of 80/50 mmHg, pulse 90 beats/min, respiratory rate 15/min, fever 97.3 F. Chest auscultation was compatible with decreased sounds in lung bases, heart sounds were normal, there were no pathological or additional sounds. Peripheral edema was not observed in the patient, and there was a small amount of hemorrhagic vaginal discharge that was observed intermittently. In the differential diagnosis, C-reactive protein level was 18 mg/L,

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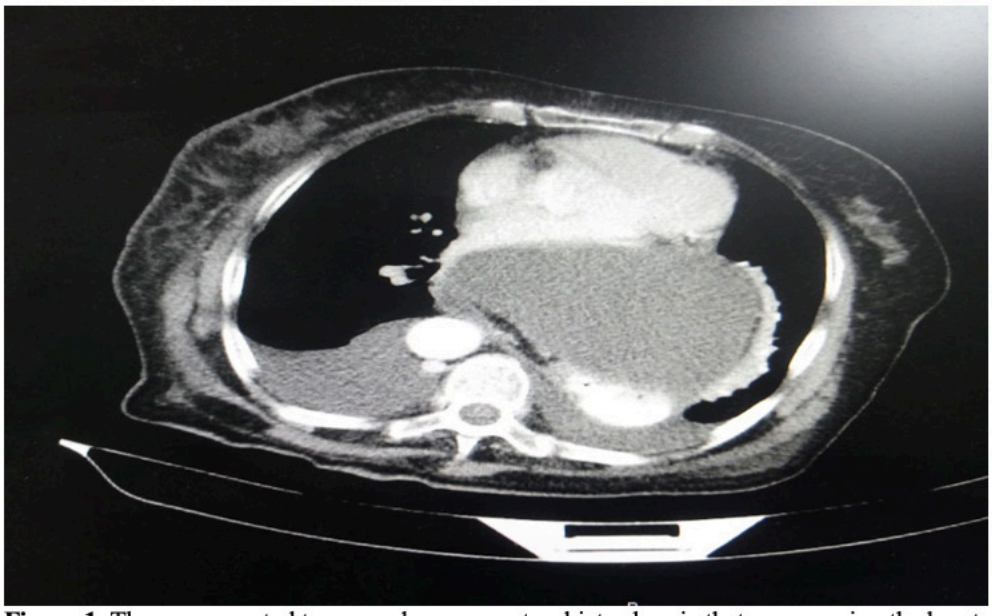

Figure 1. Thorax computed tomography represents a hiatus hernia that compressing the heart.

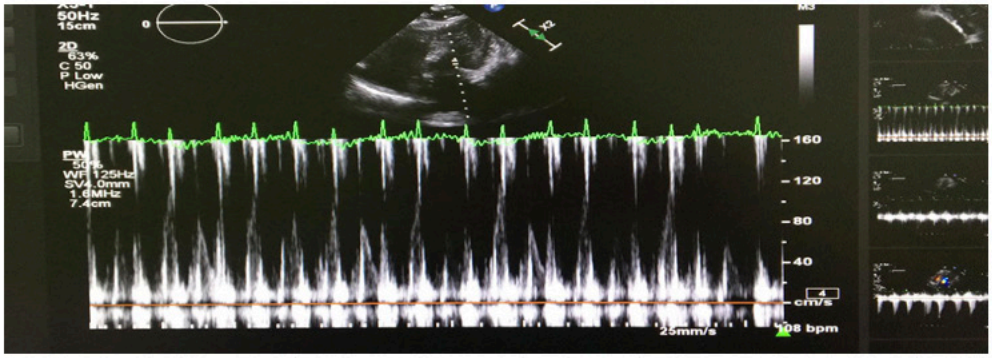

Figure 2. In the left ventricle inflow pulse wave doppler evaluation, a respiratory switch within physiological ranges was observed. Significant construction finding was not detected.

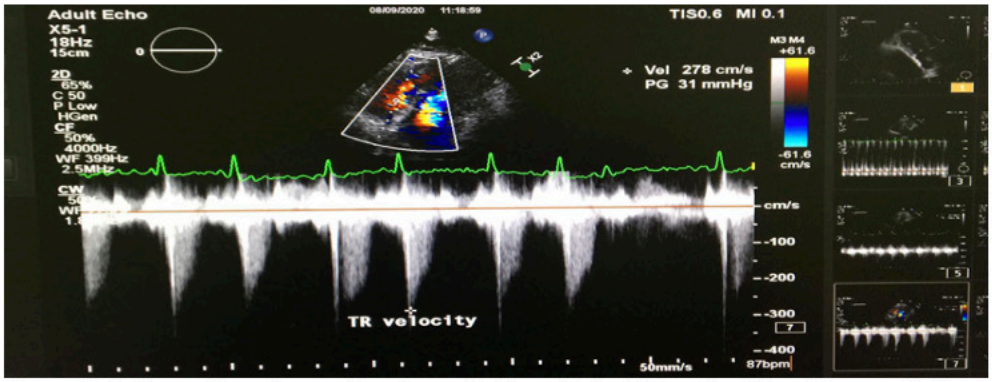

Figure 3. In the continued wave doppler evaluation obtained from the tricuspid regurgitation jet flow, the tricuspid regurgitation peak jet velocity was detected as 2.8 m/sec.

hemoglobin level was 12.4 g/dL , and no decrease was observed during the follow-up. In the examinations requested for adrenal insufficiency, cortisol, blood glucose, sodium, potassium levels were 34 mg/dL, 79 mg/dL, 141 mmol/L, and 3.5 mEq/L, respectively. Thyroid functions were within normal limits (fT3: 1.3 pg/mL and fT4: 0.7 ng/dL). A mass compressing the heart was observed with a transthoracic echocardiogram. A hiatus hernia compressing the heart was observed in thorax computed tomography (Figure 1). Nasogastric decompression was recommended for a general surgery consultation. After nasogastric decompression, the patient's inotropic therapy requirement was decreased and discontinued during the follow-up. The echocardiographic review

showed regressed compression findings, and intracardiac filling pressures were within normal limits (Figure 2 and 3). The patient's clinical findings improved, and improvement was confirmed by echocardiographic examination, and vital measurements reached normal values. Our patient, whose general well-being continued in the following days, was discharged with recommendations.

Discussion

The patient, who had treatment-resistant hypotension, was shown objectively by the hiatus hernia's compression by tomography scan and transthoracic echocardiographic evaluation and was successfully treated with nasogastric decompression. The definitive diagnosis of hiatal hernia, whose incidence increases with age, can be diagnosed with barium imaging as well as computed tomography. Although treatment is surgical, conservative approaches such as eating less, not sleeping on a full stomach, sleeping with a few pillows are also included. Even though they are generally asymptomatic, larger ones may present with different clinics.[2] Adrenal insufficiency was not considered in the differential diagnosis of treatment-resistant hypotension in our patient since concurrent hyperkalemia, hyponatremia, hypoglycemia was not present, and cortisol levels were within normal limits. Thyroid function tests were within normal limits. Despite a mild C-reactive protein elevation (18 mg/dL), sepsis was not considered because there were no accompanying findings such as leukocytosis/leukopenia, hyperthermia/hypothermia, and hypoxemia. In the follow-up of the patient, there was no hemoglobin decrease or any bleeding findings. No symptoms such as nausea, vomiting, diarrhea, or using antihypertensive or diuretics therapy suggest hypovolemia. There was no dyspnea or urticarial rash suggestive of anaphylaxis. In another case report[3], dyspnea at exertion was reported, but our patient had no effort dyspnea. Though in a published case[4], arrhythmia and acute heart failure was reported during the follow-up, our patient also did not represent any finding considering arrhythmia, but she had pyrosis and early satiety symptoms. In another case[5], ST-T changes were reported in ECG, which is not valid for our patient. Considering these rarely reported clinical findings in the literature, it is important to reveal the etiological factors to initiate effective treatment earlier.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Fundic Gland Polyps in Atypical View

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Abstract
Stomach polyps are pedunculated or sessile lesions that arise from the gastric epithelium or submucosa and project into the lumen. Most of them are detected incidentally during an endoscopy performed for another reason. Here we aimed to present an atypical case of the gastric polyp.

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Keywords: *Fundus gland polyps, endoscopy, patolgy*

Introduction

Gastric polyps are divided into three groups as epithelial polyps, hamartomatous polyps, and mesenchymal polyps. Epithelial polyps are the most common type. Fundic gland polyps and hyperplastic polyps are the most common epithelial polyps. Adenomatous polyps, NETs (carcinoids), ectopic pancreas, pyloric gland adenomas are less common epithelial polyps. Hamartamatous polyps, Peutz-Jeghers, juvenile polyps are polyps known as Cowden syndrome. On the other hand, mesenchymal polyps are rarely seen as inflammatory fibroid polyp, gastrointestinal stromal tumor, and leiomyoma. Fundic gland polyps (FGP) is the most common type of gastric polyp, with a rate of 47%. Its frequency has increased due to the widespread use of proton pump inhibitors, especially in western societies. FGPs are mostly multiple polyps smaller than 1 cm and are seen in the fundus and proximal corpus. They are flat, sessile, transparent, and round lesions.[1] The rate of malignancy is low. Its association with atrophic gastritis and H. pylori is rare. Histologically, dilatation in the auxintic glands and hyperplasia in the enterochromaffin cells are observed. There are three subtypes: sporadic type; one or several pieces are seen. It is common in people negative for H. pylori. The type associated with proton pump inhibitors (PPI) is seen frequently (about 4 times more) and in large numbers. Polyps regress after discontinuation of

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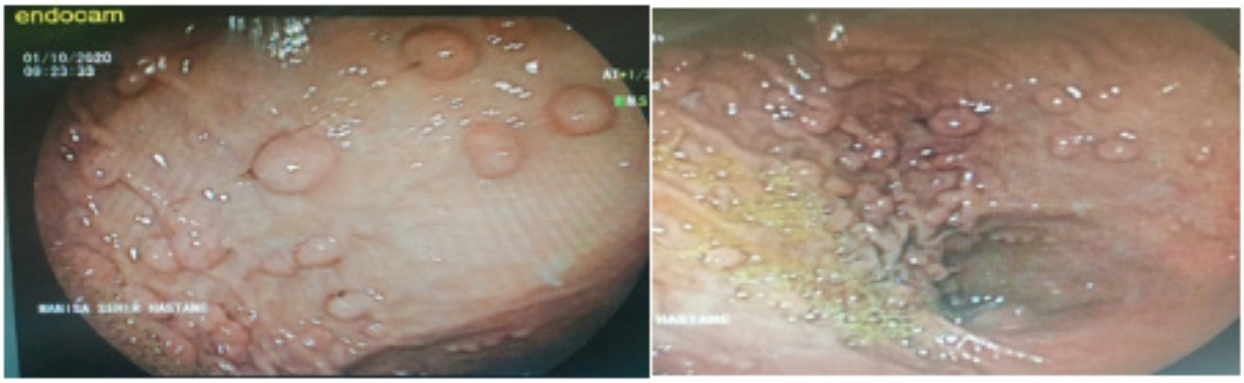

Figure 1. Endoscopic view of polyps.

the drug. The type has been seen together with FAP (familial polyposis). Familial polyposis syndrome should be questioned in the presence of sporadic FGP. APC gene mutation is an important cause in pathogenesis. It is not associated with H. pylori and atrophic gastritis. FGP is seen in 95% of patients with FAP. There is a risk of developing dysplasia in 25-40% of the FGPs seen in these patients.[2,3]

Case Report

The male patient, 69 years old, presented with nausea, swelling, and intermittent epigastric pain for about 6 months. The patient's medical and family history was unremarkable, except for the use of intermittent PPIs. Physical examination revealed blood pressure 120/70 mmHg, pulse 72 beats/min, and fever of 36.8 °C. There was no pathological finding in the systemic examination. In the laboratory findings, leukocyte 5,600 K/UI, hemoglobin 13 g/dL, ESR 10 mm/h, creatinine 0.6 mg/dL, total/direct bilirubin 1.1/0.6 mg/dL, AST/ALT 15/10 U/L, LDH 143 U/L, GGT 11 U/L, HBsAg (-), anti-HCV (-), anti-HBs (+), iron 117 µg/dL, ferritin 152 ng/mL, B12 vitamin 261, folic acid 3.5 ng/mL, and TSH 0.8 IU/mL. Tumor markers were within normal limits. There were multiple and different polyps in upper gastrointestinal endoscopy, with the largest 1 cm in the fundus and corpus. Polypectomy was performed on random large ones (Figure 1). Colonoscopy was normal. Whole abdominal tomography detected simple cysts of 2 and 4 cm in size in segments 6 and 8 of the liver. Other structures were usual. FGPs were pathologically diagnosed.

Discussion

FGPs are the most common stomach polyps. With the widespread use of PPIs, their incidence has increased. It is considered to regress after the cessation of PPIs. Its association with chronic gastritis or H. pylori gastritis is rare.[3] The risk of malignancy is low. During endoscopy, it is often not possible to directly distinguish between malignant and benign gastric polyps. Sometimes, endoscopic appearance and pathological diagnosis may not be compatible.[3,4] Therefore, pathological evaluation after polypectomy is diagnostic. In our rare and interesting case, diffuse gastric involvement and multiple polyps of different shapes and sizes suggested malignancy, but FGPs were pathologically diagnosed.

Conflict of Interests

Authors declare that there are none.

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Case Report

An Unforgettable Pre-Diagnosis for an Elderly Patient with Renal Failure and Back Pain: Multiple Myeloma

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Abstract

Multiple myeloma is a malignant disease that can cause kidney failure. Here, we filed a patient with back pain and renal dysfunction diagnosed as multiple myeloma.

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Keywords: *Fundus gland polyps, endoscopy, patolgy*

Introduction

Multiple myeloma is a malignant disease that can result in organ damage due to abnormal plasma cell growth and immunoglobulin or light chain overproduction in the bone marrow. It is the second most common hematologic cancer after lymphomas. It constitutes 1% of all cancers and 10% of hematological cancers.[1] At the time of diagnosis, 25% of patients with multiple myeloma have an increase of more than 2 mg/dL in creatinine. In other words, about half of the patients have renal failure.[2-7] Bone involvement is one of the most common organ damages in myeloma. Bone involvement is a feature that has diagnostic and prognostic value, shows tumor burden, organ damage, and affects the patient's quality of life. Any patient with myeloma suspicion must have a whole-body bone scan. Myeloma typically causes lytic lesions, osteopenia, and pathological fractures in the bones. Bone lesions are detected in 75% of myeloma cases. These lesions are most commonly found in the head bones, vertebra, ribs, sternum, proximal humerus and femur.[2-4]

Case Report

A 72-year-old male patient was admitted to the outpatient clinic due to low back pain and increased urea-creatinine. In the examinations of the patient without known chronic disease, urea 74

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mg/dL, creatinine 3.5 mg/dL, uric acid 10 mg/dL, hemoglobin 9.6 g/dL, MCV 102 fL, and 6,795 mg protein in 24-hour urine was found. Extensive examinations were planned for the patient. ANA: negative, ANCA: negative, anemia parameters were normal, hepatic markers were normal, serum protein electrophoresis was normal. In the patient, in whom kappa-lambda was sent in urine and rectal biopsy was planned for amyloidosis, immunoglobulins were low, kappa-lambda in urine was normal, and rectal biopsy was found to be normal. The patient whose kidney biopsy was considered non-diagnostic, but had severe low back pain, anemia, and high urea-creatinine, was considered multiple myeloma. In repeated examinations, bone marrow biopsy and peripheral smear were observed in the patient due to high lambda and low kappa in 24-hour product and immune fixation electrophoresis. Peripheral smear and bone marrow biopsy were consistent with multiple myeloma. The treatment of the patient was started.

Discussion

With the recent advances in multiple myeloma treatment, the survival of patients has increased dramatically. Although new agents contribute positively to overall survival and disease-free life span, they cannot prevent disease recurrence.[8] Therefore, it should be considered in patients with back pain, anemia, and kidney failure, and treatment delay should be avoided.

Conflict of Interests

Authors declare that there are none.

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TURKISH JOURNAL OF INTERNAL MEDICINE

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Case Report

A Case of Normocomplementemic Urticarial Vasculitis Triggered by Urinary Tract Infection

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Abstract

Urticarial vasculitis is a variant of cutaneous small-vessel vasculitis. Urticarial vasculitis is generally classified into two types: normocomplemanaemic urticarial vasculitis and hypocomplementemia urticarial vasculitis. Here, we presented a case with a diagnosis of normocomplementemic urticarial vasculitis.

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Keywords: *Urticaria, Vasculitis, Normocomplementemia*

Introduction

Urticarial vasculitis (UV) can be both classified under the heading of urticaria and considered a subtype of leukoclastic vasculitis, characterized by urticarial lesions. Histopathologically, it is distinguished from simple urticaria by the presence of vascular injury.1 Its typical findings include lesions lasting for more than 24 hours that do not blanch upon pressure; burning and pain being the predominant symptoms rather than itching; lesions that leave pigmentation when fading; a skin biopsy showing endothelial swelling and injury usually involving postcapillary venules, secondary red blood cell extravasation, fragmentation of leukocytes (leukocytoclasis), fibrin deposition in the vessel wall, and perivascular infiltration with a neutrophil predominance.[1] In addition, it is possible to show immunoglobulin, complement, or fibrin deposits in the perivascular area and/or dermo-epidermal junction by direct immunofluorescent (DIF) examination.[2] Although these findings are not typical of UV, their absence does not exclude the condition, either. The underlying cause is sometimes drugs, stress, malignancies, rheumatological disorders, autoimmune disorders; occasionally, an infection is the culprit trigger, as is the case in our patient.[3]

Case Report

A 39-year-old woman, who had no history of any disease other than urticaria and who had been occasionally using antihistaminic medications for

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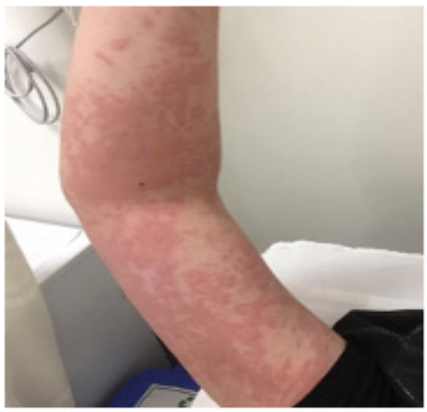

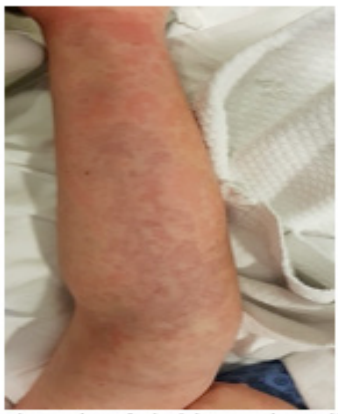

Figure 1 and 2. Widespread urticarial plaques and purple lesions that faded in patches all over the body

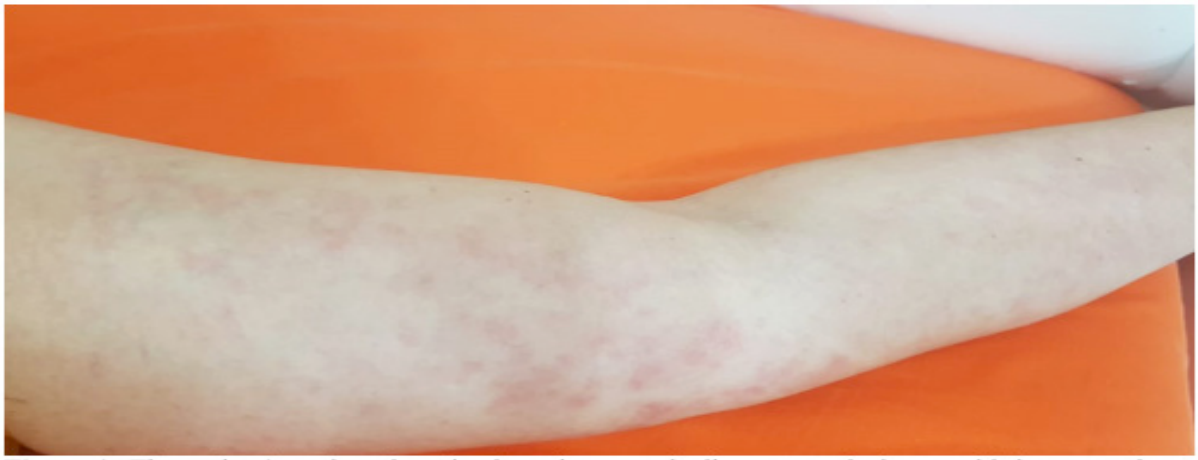

Figure 3. The patient's rash and angioedema have gradually regressed, the steroid dose was down titrated

itching, presented to our outpatient clinic with a diffuse rash all over the body, itching, and difficulty breathing. On physical examination, she had edema and redness of the entire face, which were more prominent on the eyelids and lips, as well as widespread urticarial plaques and purple lesions that faded in patches all over the body (Figure 1 and 2). She had no uvula edema or abnormal breath sounds. Her history was not notable for any medication except for an antihistaminic medication that she had taken in the last month; her complaints had started 5 days ago and not responded to the antihistaminic medications (rupatadine fumarate) that she had used. It was also learned that she had been admitted to the emergency service of an outside medical facility, where she had been treated with intravenous pheniramine, methylprednisolone, and dexamethasone; her complaints had regressed following the first three days of intravenous treatment but resumed thereafter. As she began to have a skin rash, itching, burning, and stinging sensation again on the fourth day, and as she remained unresponsive to intravenous treatment administered in the emergency service, she presented to our facility with resistant urticaria. Her laboratory tests revealed the following: urea 39 mg/dL, creatinine 0.81 mg/dL, total protein 76 g/L, albumin 45.5 g/L, uric acid 2.7 mg/dL, AST 13 IU/L, ALT 15 IU/L, LDH 258I U/L (N: 135-214), ALP 52I U/L, GGT 10 IU/L, total bilirubin 0.47 mg/dL, direct bilirubin 0.15 mg/dL, calcium 9.5 mg/dL, sodium 140 mmol/L, potassium 4 mmol/L, lekocyte 22.2x103 mcg/L, neutrophil 20.02x103 mcg/L, lymphocyte 0.95x103 mcg/L, monocyte 1.21x103 mcg/L, TSH 1.38 mlU/L, HBsAg, anti-HCV and anti-HIV were negative. Anti TPO and anti-thyroglobulin were negative. Sedimentation rate 10 mm/h, C3 1.15 g/L (N), C4 0.14 g/L (N), total IgE 296 mcg/L (N: 0-240), CRP 15 mg/L. In urinalysis, there were 7 erythrocytes and 7 leucocytes.

The patient was admitted to a regular ward with the diagnoses of resistant urticaria and urinary tract infection. Urine culture, PA chest X-Ray, ANA, and Anti-ds DNA were ordered. ANA and Anti-ds DNA tests returned negative.

There were no pulmonary infiltrates on PA chest X-Ray. The patient was begun on intravenous methylprednisolone at a dose of 1 mg/kg, pheniramine, and pantoprazole. Empirical intravenous ceftriaxone was also added for a possible urinary tract infection. As urticarial lesions persisted for more than 24 hours and they left pigmentation after fading and caused pain and tingling sensation in addition to itching, UV was primarily considered, which prompted us to take a punch biopsy of 4 mm thick from the lesions. As the patient's rash and angioedema have gradually regressed, the steroid dose was down titrated (Figure 3). Urine culture produced no bacterial proliferation. The pathology report of the biopsied lesions returned as the following: mild orthokeratosis in the epidermis; edema and vascular proliferation in the upper dermis; swollen endothelium; perivascular infiltration by lymphocytes, histiocytes, neutrophils, and eosinophils; and the presence of neutrophils and interstitial eosinophils on some vascular walls. Diagnosed with "Normocomplementemic UV" that was triggered by a urinary infection, the patient was discharged to receive an oral cephalosporin, an antihistaminic, prednisolone, and urticarial diet.

Discussion

Our patient was a 39-year-old female with chronic urticaria, who had been having occasional urticaria attacks and antihistaminic use, but no chronic medication uses between the attacks. After having dysuria not mentioned as a complaint as well as starting to have a rash and itchy lesion that had been considered as her previous attacks but remained unresponsive to treatment first with oral antihistaminic medications and then daily intravenous steroids and antihistaminic medications, the patient presented to our medical institution for further investigation and treatment. As the patient had lesions that had persisted longer than those of simple urticaria, that had a color close to violet, and that left noticeable brown pigmentation when they faded; and additionally, as she had urinary tract infection and a high CRP level, she was considered to have resistant urticaria caused by a urinary tract infection and UV as the preliminary diagnoses; therefore, C3 and C4 levels were studied and a skin biopsy was taken. C3 and C4 levels returned normal, and the skin biopsy was reported to be consistent with urticarial vasculitis. The patients were put on intravenous empirical antibiotic treatment after urine culture was taken; following the second antibiotic dose, the itching, burning, and stinging sensations began to subside, and the lesions began to fade. On the third day of treatment, the patient's lesions faded with mild residual pigmentation; itching and burning sensations completely disappeared; the CRP level returned to normal, and the urine culture revealed no bacterial proliferation. The patient was diagnosed with normocomplementemic urticarial vasculitis; subsequently, her treatment was arranged, and she was discharged with recommendations.

In patients with resistant urticarial symptoms who are unresponsive to classical urticaria treatment, underlying causes should be sought, and UV should be excluded, as was the case with our patient.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Vertebrobasilar Artery Dolicoectasia: A Case Report

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Abstract

Dolichoectasia is a dilative arteriopathy and often affects the intracranial vertebral and basilar arteries. Here, we present a hypertensive patient who presented with dizziness and balance disorder and was diagnosed with dolichoectasia.

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Keywords: *Dolicoectasia, Vertebrobasilar Artery, Headache*

Introduction

Dolichoectasia is a dilative arteriopathy characterized by the lengthening, widening and bending of an artery. Intracranial vertebral and basilar arteries are the most commonly affected vessels. The incidence of vertebrobasilar artery dolichoectasis (VBD) in the population has been reported 0.06-5.8% range. Most cases are asymptomatic and diagnosed by cranial imaging performed for other reasons. Risk factors are hypertension and age over 40 years. Symptomatic patients show cerebral ischemia, bleeding, or compression of the brainstem/third ventricle/cranial nerve roots. VBD is a rare cause of trigeminal neuralgia. One of the theories for pathogenesis of VBD; loss of elastic tissue due to early fragmentation or degeneration of internal elastic lamina; accompanying smooth muscle atrophy. Another theory is, it occurs as a complication of atherosclerosis. The extension of the artery lateral to the clivus or dorsum sella, and its bifurcation on the suprasellar cistern is considered to be elongated (dolicho). If the basilar artery diameter is over 4.5 mm, it is considered to be enlarged (ectasic).[1-3] In this case report, we aimed to present the detection of VBD in a hypertensive patient who presented with dizziness and balance disorder, since it is a rare condition.

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Case Report

67 y, female with type 2 diabetes mellitus, hypertension managed with medical treatment (metformin, clopidogrel, amlodipine, atorvastatin, ginkgo biloba). The patient had complaints of dizziness, tinnitus, and numbness in her hands and feet. Physical examination was normal. HbA1c: 6.4%, LDL: 166 mg/dL (60-130), 25-OH vitamin D: 8.51 ng/mL (30-80), LDH: 314 U/L (0-247). Sedimentation, CRP, TSH, count blood count, vitamin B12, AST, ALT, GGT, urea, creatinine, electrolytes, and urinalysis was normal. In bilateral carotid-vertebral artery doppler ultrasonography, partially calcified echogenic plaque with a stenosis rate -not exceeding 50%- was observed in the posterior wall of the right bulbus. Local diffuse enlargement was noted at the top of the basilar artery in cranial MRI. Six months later, contrast-enhanced cervical MR angiography showed normal traction, contour, flow signal patterns of the bilateral internal and external carotid arteries, and mild contour irregularities were observed in the right bifurcation region due to plaques, without significant stenosis. In cranial arterial MR angiography, local enlargement of approximately 6 mm in size in the anterior contour of the artery, which could not be clearly identified on the top of the basilar artery on MIP images, was noted in the raw images, and the findings were primarily thought to be aneurysm; therefore, it was recommended to be evaluated with conventional angiography. After this stage, the patient was consulted with neuroradiology in another center to clarify the need for conventional angiography. In time-of-flight (TOF) cranial MR angiography; it has been shown that the vertebrobasilar system and both internal carotid arteries (ICA) have an appearance compatible with dolichoectatic changes, it has been determined that there is no aneurysmatic condition and conventional angiography is not required. The patient will be followed clinically and angiographically.

Discussion

Dolichoectasis; describes pathological long, dilated, tortuous cerebral arteries.[4] Traditionally, the diagnosis of VBD was made by catheter angiography. However, VBD diagnosis can now be made by non-invasively with CT and MR angiography imaging. MR angiography is the most sensitive imaging method; high resolution and thin section T1 and T2 weighted spin echo sequences, three-dimensional TOF MR angiography, 3D CISS sequence are the most effective sequences in the evaluation of VBD. In addition, differential diagnosis of VBD from pathologies such as aneurysms and other vascular problems, demyelinating diseases, and space-occupying lesions can be easily made with MR imaging.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Osteosarcoma of Rib Treated with Regorafenib: A Case Report

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Abstract

Osteosarcoma is a common primary malignant bone tumor in young adults presenting with back and chest pain complaints. Here, we presented a rare case of osteosarcoma.

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Keywords: *Oncology, osteosarcoma of rib, regorafenib*

Introduction

Osteosarcoma is the most common primary malignant bone tumor among young adults.[1] It is more frequently seen in men than women. It usually originates from the metaphysis of long bones. Primary osteosarcoma rarely occurs in the rib. Modalities such as surgery, chemotherapy, and radiotherapy form the basis of the treatment, which should be planned in a multidisciplinary way. In this report, a case of osteosarcoma originating from the rib and spreading to the cervical and thoracic vertebrae is discussed.

Case Report

A 23-year-old female patient was admitted to our center with left-back pain for almost a year. In thoracic computed tomography (CT), a mass originating from bone with mixed density, causing erosion in the cortex, and covering the entire posterior and lateral arches and most of the anterior arch of the 1st left rib was detected (Figure 1). The mass at the level of T1 vertebra was compressing the left lung apex. The 1st rib, expanded by the mass, was completely removed by dissection. Resected material was reported as 8x8 cm osteoblastic type osteosarcoma and surgical margins were

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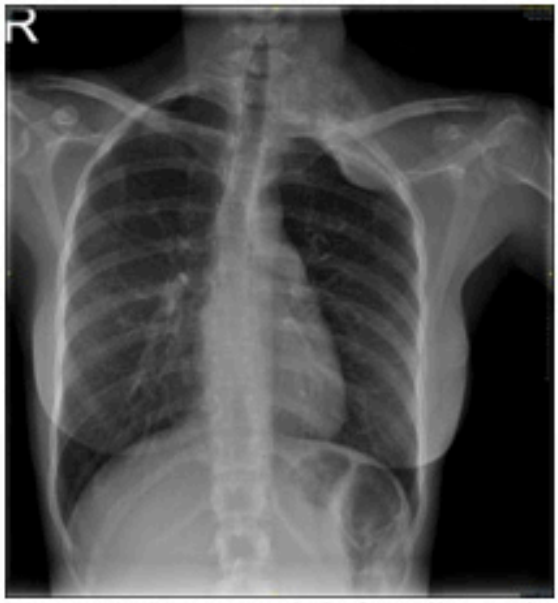

Figure 1. X-ray graph before diagnostic surgery.

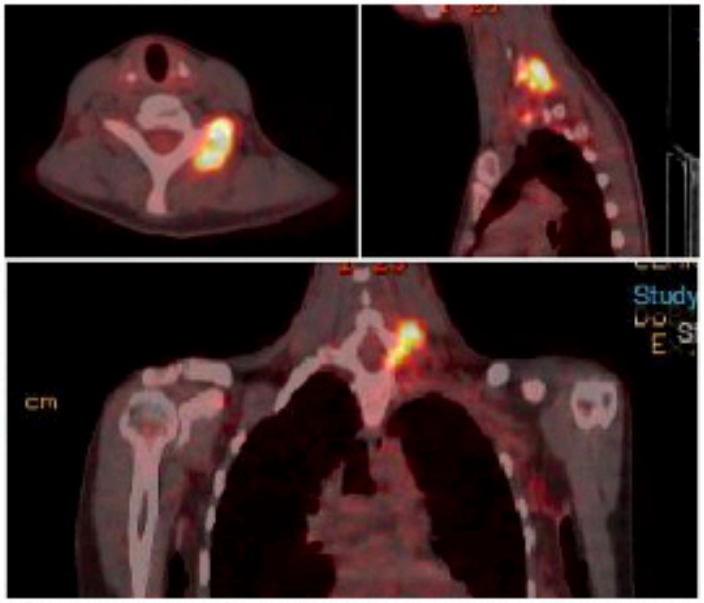

Figure 2. Systemic evaluation after diagnostic surgery.

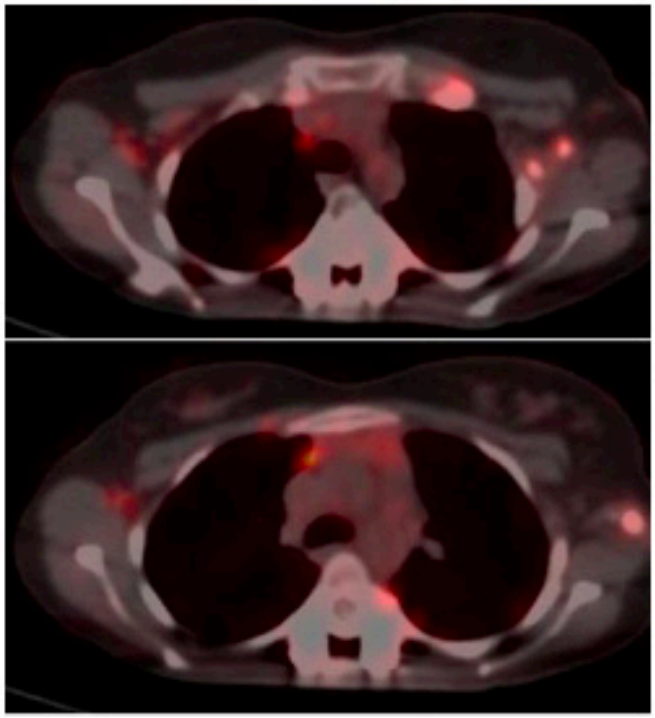

Figure 3. Recurrence at left axilla and left 1st costochondral junction in PET-CT.

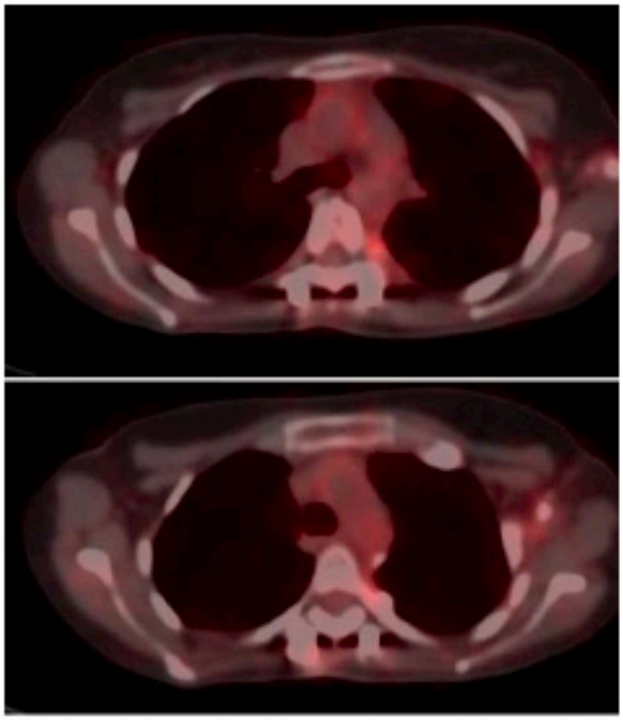

Figure 4. PET-CT images after local radiotherapy and regorafenib treatment.

positive. The PET-CT for systemic evaluation showed a hypermetabolic mass (Suv-max 12-16) of 27x28 mm in the left half of the T1 vertebra corpus and the left transverse process of the C7 vertebra (Figure 2). Thereon, the patient was treated with doxorubicin and cisplatin. The PET-CT following the third cycle of chemotherapy demonstrated a partial response, and the patient was evaluated as resectable. All of the C7, T1, and T2 vertebrae and the posterior arch of the 2nd left rib were removed with en bloc resection. The mass was reported as 6x4.5 cm osteosarcoma. Surgical margins were positive, and necrosis was 10%. Radiotherapy was planned for the patient since the margins were positive, with the low chemotherapy response, interpreted as grade 1. 45 Gy radiotherapy was

administered to the cervicodorsal region in 25 fractions. The chemotherapy regimen was changed, and docetaxel and gemcitabine were started. During the follow-up after adjuvant chemotherapy, a nodular lesion was detected in the left axilla on CT in the 4th month, and the biopsy was compatible with recurrence. PET-CT scan showed increased metabolic activities within nodular lesions in the left axilla and 1st left costochondral junction (Figure 3). Regorafenib was started after local radiotherapy. The patient continues to be followed up in the tenth month under regorafenib treatment (Figure 4).

Discussion

Osteosarcoma is the most common malignant tumor with the origin of bone in adults. Although malignant bone tumors located in the chest wall are most commonly originating from the ribs, primary osteosarcoma of the ribs is extremely rare, its prognosis is worse than osteosarcoma from long bones and there is a limited number of cases in the literature. Back and chest pain is the most important factors for clinical presentation. En bloc resection of the mass and the negative surgical margins are the main factors determining the prognosis of the disease. Survival is aimed to be increased with neoadjuvant and adjuvant chemotherapy. Radiotherapy may be considered in patients with positive surgical margins after resection of large masses. There are various studies showing the efficacy of cytotoxic therapy with regorafenib in recurrent cases.[2,3] In patients presented with back and chest pain, there may be delays during the diagnosis of osteosarcoma of the rib because there are other reasons to be considered first. In the patient discussed in this report, the tumor has already reached large dimensions at the time of diagnosis because the clinical finding was only pain. Upon surgery, postoperative surgical margins were positive, and she experienced progression despite cytotoxic chemotherapy and radiotherapy. With regorafenib which is started later, the disease was controlled with partial response.

In conclusion, osteosarcoma of the rib is rare, and recurrence is common. Survival can be prolonged with a multidisciplinary approach including surgery, systemic chemotherapy, targeted agents, and radiotherapy.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Primary Membranous Glomerulonephritis in a Young Patient with Proteinuria

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Abstract

Membranous nephropathy is a common form of glomerulonephritis that typically presents with nephrotic syndrome between the 3rd and 5th decades and one-third of patients experience spontaneous remission. Here, a patient with primary membranous nephropathy is presented.

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Keywords: *Dolicoectasia, Vertebrobasilar Artery, Headache*

Introduction

Under physiological conditions, daily protein excretion with urine is below 150 mg. Detecting protein excretion above this value in repeated measurements, i.e., proteinuria, should not be ignored and further evaluation should be made. Protein excretion above this level is generally an important indicator of underlying kidney damage. The commonly used method to evaluate whether the protein excretion in urine is within normal limits is the measurement of protein in 24-hour urine.[1]

Case Report

A 31-year-old patient without a known illness and no regular medication was admitted to the outpatient clinic with proteinuria. The patient did not have any complaints other than foaming in the urine and a new rash on the nasal wings. There was no history of a recent upper respiratory tract infection or arthritis. No pathology was found on physical examination. The test results of the patient are given below: urea: 24 mg/dL, creatinine: 0.7 mg/dL, albumin: 2.9 g/dL, sodium: 141 mmol/L, potassium: 4.4 mmol/L, LDL: 259, HDL: 40 mg/dL, TSH: 2, PTH: 57,

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hemoglobin: 15.2 g/dL, MCV: 82, IgM: 31, IgG: 644, IgA: 205, C4: 28, and C3: 129. The protein excretion was 9 g in 24-hour urine. RF, ANA, PR3 ANCA, MPO ANCA, HBsAg, anti-HIV, and anti-HCV were negative. ACE inhibitor and acetylsalicylic acid were initiated for the patient. The patient, who was found to have proteinuria at the nephrotic level, hyperlipidemia, and hypoalbuminemia, was referred to a kidney biopsy to investigate proteinuria's etiology. The pathology result was determined as IgG4 + membranous glomerulonephritis. During the controls, the patient, who was considered low risk, had a 24-hour urine protein of 1.5 g, was started on 10 mg atorvastatin. A dietician regulated the patient's diet, and 24-hour urine control was recommended. In the patient's follow-up, phospholipase A2 was positive, and it was followed up as idiopathic membranous glomerulonephritis.

Discussion

Membranous nephropathy is the leading cause of nephrotic syndrome in the adult population.[2-5] The disease is characterized by the deposition of immune complexes outside of the glomerular basement membrane. This accumulation causes the glomerular filtration barrier to lose its function and this results in proteinuria.[3-5] It is generally classified as membranous nephropathy, primary or secondary membranous nephropathy. There is no known etiology in 70-80% of cases. If a secondary cause cannot be determined, this group is classified as "Primary membranous nephropathy".[4,6,7] In 70% of adult patients, phospholipase A2 in podocytes has been shown to be the target antigen in primary membranous nephropathy.[8]

In conclusion, glomerulonephritis should be considered in every patient with proteinuria. Patients should be directed to biopsy before progressing to end-stage renal disease, and they should be followed up and treated.

Conflict of Interests

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TURKISH JOURNAL OF INTERNAL MEDICINE

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Case Report

Enteric adenocarcinoma arising from mediastinal teratoma in a man: A case report

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Abstract

Teratomas are a type of germ cell tumor with malignant potential, located gonadal or extragonadal. Here, we reported a patient with enteric adenocarcinoma based on teratoma presenting with a mediastinal mass.

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Keywords: *Mediastinal teratoma, enteric adenocarsinoma, metastasis*

Introduction

Teratomas are types of germ cell tumor which can arise from gonads or extragonadal tissues and can show malignant transformation. Adenocarcinoma is the most common histological subtype in malignant transformation. In this article, a patient with enteric adenocarcinoma on the basis of teratoma presenting with a mediastinal mass invading the sternum is presented.[1-3]

Case Report

A 54-year-old male patient presented with a swelling in the chest wall under the right neck that started 9 months ago and progressed (Figure 1). In the thoracic computed tomography of the patient, a mass causing lytic destructive changes with a lobulated contour of 68x57 mm in the right half of the mediastinum and extending to the manubrium sterni in the anterior was detected. The pathological evaluation of trucut biopsy from the mass was reported as adenocarcinoma (Cytokeratin 20 and CDX2 positive, Cytokeratin 7, PSAP, TTF1, Napsin A, Chromogranin, Synaptophysin negative), but the histomorphological and immunohistochemical features of the tumor were not characteristic for conventional type pulmonary adenocarcinoma. It was recommended to exclude the possibility of metastatic carcinoma of gastrointestinal origin firstly. Esophagogastric endoscopy and colonoscopy, which was planned for the gastrointestinal screening of the patient, were unremarkable. However, the patient did not continue with the examination and treatment. The patient was admitted to the hospital 1 month ago with lack of oral intake and dyspnea after 6 months his first evaluation and a mass -12x12 cm- diameter was revealed on the sternum and

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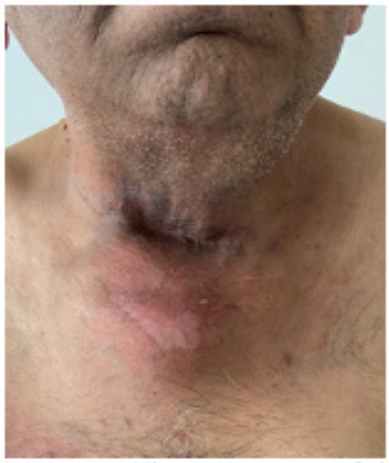

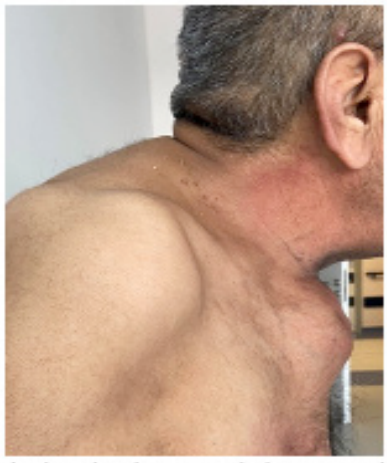

Figure 1. The appearance of the lesion in the manubrium sterni area of the patient

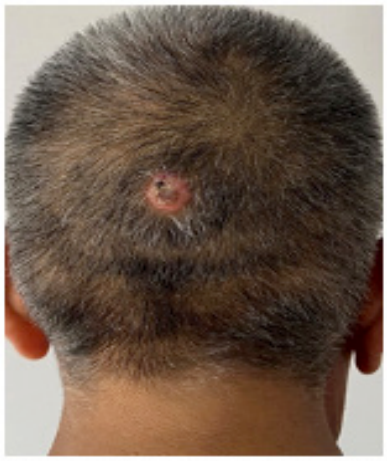

Figure 2. The appearance of the lesion on the patient's scalp.

two masses 3 cm diameter was detected in the scalp. The patient who was tachypneic had severe dyspnea at rest and exertion. PET CT of the patient revealed that increased F-18 FDG uptake in lymph nodes with a diameter of 28x31 mm in the right supraclavicular area (SUV-max 4.45) and increased F-18 FDG uptake in the non-metabolic/necrotic-looking mass lesion with heterogeneous density, 84x124x151 mm in size (suv-max 8.22). The central part of mass was nonmetabolic and protruded under the skin and destructed the sternum by filling the mediastinum almost completely, showed significant expansion in the anterior and deviated the trachea to the left. Biopsy was repeated from the lesion on the manubrium sterni, and pathology result was reported as adenocarcinoma infiltration/metastasis, metastatic enteric adenocarcinoma developed on the teratoma background (TTF1, Napsin A, Psa, Cytokeratin 7, Chromogranin, Synaptophysin, MUC 5AC negative, Cytokeratin 20, EMA, CEA, CDX2, MUC 1 positive). Pathological examination of the 2x3 cm lesion on the scalp from the patient was evaluated to be consistent with adenocarcinoma metastasis (Figure 2). Upon the development of VCSS in the patient, 20 Gy in 5 fractions of 9400cGy/day (in sitting position because the patient could not lie down) was given palliatively. First step palliative chemotherapy was planned for the patient. In the follow-up after the first cycle, it was observed that the patient's clinical findings started to regress. Follow-up and treatment of the patient is still ongoing.

Discussion

Although mediastinal teratomas show different clinical behaviors, they are associated with rapid progression and poor prognosis after malignant transformation. In cases where surgery is possible, survival is similar to patients without malignant transformation, but is incurable in metastatic patients.[2,4]

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Kappa light chain myeloma: A case report

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Abstract

Light chain myeloma nephropathy is the most common form of renal involvement in plasma cell dyscrasias. It usually causes tubulointerstitial renal damage. About one in five people with multiple myeloma produce only light chains. We report a case of lambda light chain deposition disease in a 61-year-old female who presented with acute renal failure. She is currently in partial remission following treatment with bortezomib, cyclophosphamide, and steroids. We present a case with rare kappa light chain myeloma with light chain deposition in renal tubules.

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Keywords: *Kappa myeloma, light chain, acute renal failure*

Introduction

Light chain myeloma nephropathy is the most common form of renal involvement in plasma cell dyscrasias. It usually causes tubulointerstitial renal damage. The diagnosis is made by showing the wide band structures in the tubule that cause obstruction with pathology. It should be considered in the differential diagnosis in patients with unknown cause of urea, elevated creatinine and anemia. Light chain only variant constitutes approximately 15% of patients with multiple myeloma.[1] Renal failure, bone disease, and systemic light chain AL amyloidosis appear to be more frequent in patients with light chain multiple myeloma (LCMM). LCMM has an earlier average age of onset and appears to have a poorer prognosis when compared to IgG or IgA variant.[2,3] We present a case with rare kappa light chain myeloma with light chain deposition in renal tubules.

Case Report

A 61-year-old female patient has no known chronic disease other than chronic bronchitis. She applied to an external hospital with complaints of headache, weakness and vomiting. She was referred to us due to the high urea and creatinine

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value in the laboratory tests. Hemoglobin 6.9 g/dL, creatinine 5.67 mg/dL, urea 97 mg/dL, sedimentation rate 52 mm/hour, IgG 3.14 g/L (7-16), IgA <0.28 g/L (0.7-4), an IgM 0.17 g/L (0.4-2.3). There was 10.8 g of proteinuria in 24-hour urine. Peripheral blood smear was normal. No monoclonal band was formed in serum protein and immune electrophoresis. Kappa and free kappa light chain bands were observed in urine immune electrophoresis. A bone marrow biopsy was then performed: a mild hypercellular imprint (LCMM) With atypical plasma cell infiltration (84%) was reported. In PET-CT, extensive metabolic activity increase was detected in many bone marrows. A kidney biopsy was performed to determine the cause of kidney failure. The pathology result of kidney biopsy was compatible with cast nephropathy. The patient was taken over by hematology. Chemotherapy treatment was started with cyclophosphamide + bortezomib + dexamethasone. There was a significant improvement in the clinic of the patient, who received 4 cycles of chemotherapy. Serum creatinine level significantly decreased from 6 mg/dL to 3.5 mg/dL.

Discussion

LCMM is a difficult disease to diagnose. As in our case, the majority of patients have renal failure and anemia at the time of diagnosis.[4] In patients with high urea and creatinine whose cause is unknown, in the presence of anemia, myeloma disease should be suspected, and urine immune electrophoresis and serum immune electrophoresis should be requested together with serum protein electrophoresis.[5] In our case, no significant findings were detected in peripheral blood smear, serum protein electrophoresis and serum immune electrophoresis. Bone marrow biopsy was performed in our patient as a result of the findings in urine immune electrophoresis. Subsequently, a kidney biopsy was performed to determine the cause of kidney failure. Although the importance of kidney biopsy in light chain myeloma nephropathy is discussed, its effect in determining the prognosis is great. The presence of numerous casts and diffuse tubular atrophy is associated with poor renal prognosis.[5] As in our case, the majority of patients who received cyclophosphamide + bortezomib + dexamethasone chemotherapy benefited from the treatment. Blood levels of urea and creatinine are decreased. Some of the patients who needed dialysis became independent from dialysis.[6-8]

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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TURKISH JOURNAL OF INTERNAL MEDICINE
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Case Report

Blastic Plasmacytoid Dendritic Cell Neoplasia: A Rare Case Report

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Abstract

Blastic plasmacytoid dendritic cell neoplasia is a rare hematological malignancy whose pathogenesis has not been clarified yet. Skin, bone marrow, and lymph node involvement can be seen and is usually seen in men and older adults. Immunohistochemistry features of skin and bone marrow biopsy are important in diagnosis. Here, we presented a case of blastic plasmacytoid dendritic cell neoplasia.

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DOI: 10.46310/tjim.876554

Keywords: *Blastic plasmacytoid dendritic cell neoplasia, cutaneous lymphoma, NK-cell lymphoma*

Introduction

BPDNC is a rare hematological malignancy that is considered to be caused by plasmacytoid dendritic cells and was included in the acute myeloid leukemia-associated precursor neoplasm subgroup by the World Health Organization (WHO) in 2008.[1] The patients frequently presented with common cutaneous lesions. Blastic cell infiltration can be seen in the peripheral smear. Lymphadenomegaly and pancytopenia can be detected in patients. Typically, CD4, CD56, CD123 expression is observed in the bone marrow and skin biopsy by flow cytometry/immunohistochemistry (IHC). The patients are generally men and older adults.[2] The median survival is 12-14 months from diagnosis. Advanced age and stage are thought to be associated with poor prognosis. Patients often respond to initial chemotherapy, but relapses are frequent. Multiagent chemotherapies such as CHOP, hyper-CVAD are frequently used. Stem cell transplantation can be an option in young and well-performing patients.

Case Report

The patient, 81 years old, known to have no comorbidities, was examined five months ago with

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newly developed swelling in the right arm and widespread rash on the whole body. The patient didn't have B symptom. On physical examination, there were multiple lymphadenomegalies in bilateral cervical, axillary, and inguinal regions, the largest of which was 2.5x2 cm in size. He had hepatomegaly. Splenomegaly wasn't detected. In laboratory tests, leukocyte: 3060/mm^3, neutrophil: 1,270/mm^3, lymphocyte: 1,620/mm^3, hemoglobin: 11.1 g/dL, platelet: 16,800/mm^3, ESR: 25 mm/h, LDH: 170 U/L, beta-2 microglobulin: 3.23 mg/dL. Platelet count in his peripheral smear was consistent with the hemogram. Aspiration and imprints were infiltrated with heterogeneous cells, some with lymphocyte morphology, some with narrow cytoplasm and large-small lymphoblasts without granules at bone marrow biopsy. Blast rate was evaluated as 51%. The immunophenotype was in flow cytometry CD3 (+), CD4 (+), CD5 (+), CD7 (+), CD8 (+), HLA-DR (+), CD20 (-), CD34 (-), and CD103 (-). IHC staining as CD4 (+), CD8 (+), CD38 (+), CD138 (+), CD20 (-), TDT (-), CD5 (-), and LCA (+) bone marrow biopsy interpreted CD4 expression as supporting T-cell neoplasia. The skin biopsy has been reported as punch biopsy showing atypical lymphoid infiltration. It resulted as IHC as CD3 (-), CD4 (+), CD7 (+), CD8 (-), CD20 (-), LCA (+), and MPO (-). Ki-67 index was reported as 50%. BPDCN was considered primarily supported by skin findings, pancytopenia, and flow. On PET-CT, diffuse increased metabolic activity was observed in the neck, right lung, mediastinum, bilateral axillae, subcutaneous soft tissue in the right humerus, right nipple, spleen, and all bone structures entering the imaging field. Based on the available information, the patient was considered as BPDCN. After four cycles of chemotherapy inter evaluation with the CVP protocol, the CVP protocol was planned to continue. The follow-up and treatment of the patient who has been applied cure CVP continues.

Discussion

In summary, BPDCN is a rare disease with poor prognosis.[3] Response to initial chemotherapy is good, but relapse is common. More studies are necessary to have a better understanding of the disease for proper management.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Intravascular Large B-Cell Lymphoma: A Rare Case Report

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Abstract

Diffuse large B-cell lymphoma is characterized by the infiltration of small vessels by lymphoid cells. These cells are not seen in the systemic circulation. Depending on the involvement location, it may present with different clinics such as central nervous system and skin involvement. Therefore, diagnosis may delay. Diagnosis is based on pathology. Herein we presented a rare patient who initially presented with cellulitis and was ultimately diagnosed with intravascular large B-cell lymphoma.

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DOI: 10.46310/tjim.876799

Keywords: *Diffuse large B-cell lymphoma, subcutaneous nodules, intravascular lymphoma*

Introduction

Diffuse large B-cell lymphoma is a common hematological malignancy that usually presents with signs of peripheral lymphadenopathy, hepatosplenomegaly, and bone marrow suppression, and/or symptoms such as fever, night sweats, and weight loss. Intravascular large B-cell lymphoma is an aggressive and rare tumor characterized by neoplastic cells' tendency to remain in the vessel. Most of the patients are in the middle or advanced age group. The symptoms are related to the lesions caused by the tumor cells' occlusion in the small vessels. Findings related to the central nervous system (convulsions, neurological deficits, progressive dementia) or skin involvement (subcutaneous nodules, plaques) are frequently observed. In patients with skin lesions, the prognosis is better than those with only central nervous system involvement, as early diagnosis can be made by skin biopsy.[1]

Case Report

A 71-year-old female patient with known hypertension was examined with complaints of redness, warmth, swelling, and pain in the legs for about 3 months. Systemic examination revealed

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redness, increased warmth, and edema in the lower extremities. Multiple lymph nodes, the largest of 2x1 cm, were palpated in the bilateral inguinal area. There was no organomegaly. Cellulite and deep vein thrombosis were excluded, skin punch biopsy was performed from the lower extremity. Its pathology resulted in "LCA positive Ki-67 blast lymphoid cell groups with high proliferative activity in subcutaneous vessel lumens". Leukocyte was 6,500/mm³, neutrophil 3,308/mm³, hemoglobin 8.6 g/dL, platelet 181,000/mm³, and sedimentation rate 71 mm/h. The peripheral blood smear was unremarkable. No infiltration was detected in the bone marrow biopsy. Intravascular large B-cell lymphoma Stage 3B was evaluated. R-CHOP chemotherapy was planned for the patient. Since hypermetabolic lymph nodes in the mediastinum and hiluses persisted after 8 cycles of R-CHOP chemotherapy, R-BENDA was planned as a second-line treatment. Lenalidomide+Rituximab was chosen as the treatment plan for the patient who was evaluated as stable disease by PET-CT after 4 cycles of R-BENDA. The follow-up and treatment of the patient continue in Bursa Uludag University Faculty of Medicine Hematology Department.

Discussion

Although diffuse large B-cell lymphoma is the most common subtype among non-hodgkin lymphomas, its variant, intravascular large B-cell lymphoma, is a scarce form.[2] The diagnosis can be easily missed due to nonspecific presentations, infective and vascular problems included in the differential diagnosis. The main diagnosis is based on pathology. Neoplastic cells are rarely seen in the bone marrow, and peripheral blood smears, so intravascular large B-cell lymphoma diagnosis is difficult. Most of the reported cases have been confirmed by autopsy or cutaneous biopsies.[3] In conclusion, we think it would be appropriate to evaluate the skin lesions that could not be diagnosed specifically in lymphoma infiltration.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Bone Marrow Metastasis of Rhabdomyosarcoma Mimicking Acute Leukemia: A Case Report

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Abstract

Although it is more common in childhood, rhabdomyosarcoma is an extremely rare soft tissue sarcoma for adults. It originates from mesenchymal cells which were differentiated into striated muscles. Their frequent locations are head-neck, genitourinary system and extremities, respectively. Here, we presented a case of rhabdomyosarcoma mimicking hematological malignancy.

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DOI: 10.46310/tjim.876918

Keywords: *Rhabdomyosarcoma, bone marrow metastasis, bone marrow infiltration*

Introduction

Rhabdomyosarcoma caused bone marrow metastasis cases are among the rarest cases reported in the literature. Similar to the cases reported in the literature, the clinical presentation of our patient mimicked acute leukemia, and the leukoerythroblastosis in the peripheral smear test and the cytopenia in the hemogram raised suspicion of hematological malignancy initially.[1,2] The diagnosis made by immunohistochemical staining in biopsy. The primary tumor field could not be detected in the radiological imaging, and it was determined by rhinoscopy evaluation performed upon patient's anosmia and epistaxis complaints. Here, we reported a case in which we were diagnosed with acute leukemia at admission but later diagnosed as a primary malignancy that metastasized.

Case Report

In December 2018, a 24-year-old female patient, who had not been diagnosed with a chronic disease before, applied our institution's emergency service with syncope, epistaxis and hypermenorrhea complaints. Lymphadenomegaly and organomegaly were not found on physical examination, but ecchymoses with different maturities were found spread on her body. The

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admission hemogram resulted with leukocyte: 5,460 K/µl, neutrophil: 1,520 K/µl, hemoglobin: 10.6 g/dL, and platelet: 13,540 K/µl. Patient's peripheral smear test resulted in slightly leukoerythroblastosis, blastic characterized non-fully differentiated cells and absolute thrombocytopenia. Bone marrow examination and flow cytometric study were decided to be performed with prediagnosis of acute leukemia. Flow cytometric study of bone marrow resulted in blast rate within normal range and non- hematopoietic cell infiltration. At gate CD45, blast rate was 0.4%. Within 87% of the cell group, CD56 expression was strongly positive and CD45 expression was weak. Cranial and thoracoabdominopelvic tomography were performed on patient with suspected bone marrow infiltration, but no pathology was found. In bone marrow biopsy result, neoplastic cells replacing hematopoietic cells in the intertrabecular space contained cells with narrow cytoplasm and mild pleomorphism. Positive staining with desmin, myoglobulin and MSA were observed. Widespread membranous staining with CD56 was observed. Based on these findings, metastatic rhabdomyosarcoma was considered. In the clinical follow-up of patient, whose primary focus was investigated, a mass was detected in the nasal root as a result of rhinoscopy evaluation, which was performed due to the presence of anosmia, gradually increasing nasal obstruction and intermittent epistaxis. Patient was diagnosed with rhabdomyosarcoma, the primary focus of which was accepted as nasal root and had diffused bone marrow metastases, which were shown on PET imaging. Then, patient was referred to oncology department for treatment plan and follow-up procedures.

Discussion

Bone marrow aspiration/biopsy examinations, which are referred to differentiate clinical entities such as anemia, leukopenia or thrombocytopenia, play an essential role in diagnosing, staging and managing the treatment of hematological malignancies and bone marrow metastasized solid tumors. In the initial evaluation, we have considered presence of a hematological malignancy in the foreground. But flow cytometric study provided us the first differential diagnosis. In our case, rhabdomyosarcoma metastasis was detected by bone marrow biopsy performed on patient, who came with thrombocytopenia initially. We aim to emphasize the fact rhabdomyosarcoma presents with bone marrow involvement and mimics lymphoma and leukemias. Still, it is mostly diagnosed by the absence of specific hematopoietic markers.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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TURKISH JOURNAL OF INTERNAL MEDICINE
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Case Report

HIV Related Primary Central Nervous System Lymphoma: A Case Report

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Abstract

Cranial imaging is vital in patients with complaints of sudden speech disorder, deterioration in performance status, urinary and fecal incontinence, and numbness in hands and feet. It provides an investigation of etiology and clarification of diagnosis. Hematological malignancies sometimes present with these symptoms and imaging may be the first method for diagnosis. In this case, we will present a patient diagnosed with primary central nervous system lymphoma, who was found to be HIV-positive during follow-up, and who progressed early and died despite treatment.

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DOI: 10.46310/tjim.876945

Keywords: *HIV, Lymphoma, Central Nervous System*

Introduction

Primary central nervous system (CNS) lymphoma is a rare variant of extranodal lymphoma with an isolated brain, leptomeningeal, eye, or spinal cord involvement. Iatrogenic immunosuppression, X-dependent immunodeficiency (Wiskott-Aldrich syndrome), and especially HIV are the main risk factors for immunosuppression. In this case report, we will present a patient diagnosed with primary central nervous system lymphoma, who was found to be HIV-positive during follow-up, and who progressed early and died despite treatment.[1]

Case Report

A 28-year-old male patient presented with complaints of sudden onset of speech disorder, urinary and fecal incontinence. On physical examination, he was conscious, oriented, and cooperative. The pupils were isochoric, direct/indirect light reflexes were bilaterally positive and there were no significant signs of lateralization. There was paresthesia at the level of L1-T12. The anal tone was normal. Deep tendon reflexes were normoactive, and no pathological reflex was observed. Scattered mass lesions in the right thalamus, right middle cerebral peduncle, and posterior fossa were observed on cranial MRI. These mass lesions created significant compression in bilateral caudate nuclei and frontal horns. The stereotactic biopsy was performed by the Neurosurgery. The biopsy result was found to be consistent with non-Hodgkin diffuse large B-cell lymphoma-CNS involvement. The patient was admitted to the clinic with a plan of high dose methotrexate (MTX) chemotherapy. In

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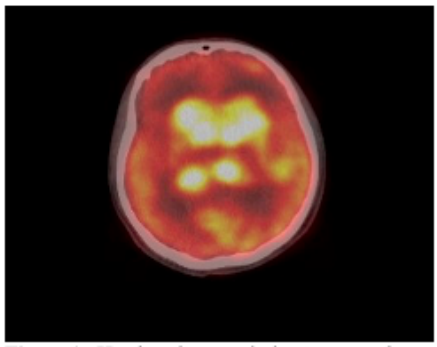

Figure 1. Head positron emission tomography (PET) before chemotherapy.

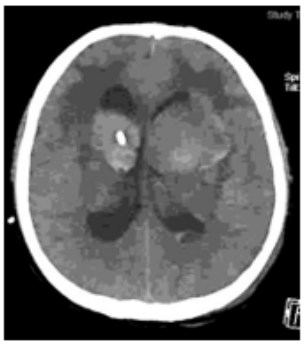

Figure 2. Head computed tomography (CT), hemorrhage findings in basal ganglia.

PET/CT, multiple hypermetabolic mass lesions (SUVmax: 15) were observed in the 4th ventricle of the brain, caudate nuclei, and right cerebellum. No involvement was observed in other parts of the body (Figure 1). The patient's hepatitis markers were negative, and HIV antigens and antibodies were found to be positive (CD4 count: 6 cells/mm3). Further questioning the patient's history, he was found to be a methamphetamine addict. The patient was started on bictagravir + emtricitabine + tenofovir alafenamide treatment. HIV-RNA was found to be positive (HIV-RNA: 66,576). After starting high dose MTX, the patient had an epileptic seizure on the second day of chemotherapy. On the second cycle of high dose MTX, Glasgow coma score dropped to three, and a CT scan of the brain showed a six cm mass lesion and hemorrhage into the ventricles (Figure 2). An external ventricular drainage system (EVDS) catheter was inserted. He was started to be followed intubated under mechanical ventilator support. The patient died on the 31st day of chemotherapy with signs of intracranial bleeding and sepsis.

Discussion

Although primary CNS lymphomas are rare malignancies, their incidence has increased due to the frequent use of diagnostic and immunosuppressive treatment methods. Its relationship with HIV is strong, and differential diagnosis can be made with lymphoma in HIV-positive individuals according to the CD4 count. In HIV-related cases, initial treatment with high dose MTX is recommended instead of radiotherapy alone or antiretroviral therapy (ART). Diagnosis should be made by stereotactic biopsy. In addition to tumor-directed therapy, ART should also be given to patients. Five factors that affect the prognosis of the disease have been reported: age, ECOG score, LDH level, CSF protein amount, and deep brain regions involvement.[2] 2-year survival rates are higher in patients who received high-dose MTX with or without radiation therapy.[3]

As a result, primary CNS lymphoma may present itself with focal or non-focal symptoms (lethargy, aphasia, confusion, etc.) due to edema or mass compression.[4] Especially white matter and deep regions are affected in the brain. Lower CD4 counts are related to a poor prognosis. The CD4 count of our patient was found to be low, consistent with the literature.[5] The prognosis is poor, and survival is shorter than one year in primary CNS lymphoma cases, especially in HIV-related groups. Improvement in survival can be observed with combined treatments.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Pectoral muscle hematoma as a complication of COVID-19 treatment: A Case Report

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Abstract

In COVID-19 disease, the activation of the coagulation system is increasing. Here we presented a case of COVID-19 who developed a hematoma in the pectoral muscle possibly associated with anticoagulation therapy.

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DOI: 10.46310/tjim.876970

Keywords: *COVID-19, bleeding, anticoagulation, coagulopathy*

Introduction

The newly identified coronavirus, SARS-CoV2, spread throughout the world rapidly and turned into a pandemic. The virus causes COVID-19 disease targeting the lungs leading to acute lung injury and respiratory failure if the disease progresses. It has been shown that one of the most important processes in the pathogenesis of the disease is the activation of the coagulation system. Due to the evidence, we order appropriate doses of anticoagulants to patients with COVID-19 according to their progress and test results.[1-3]

Case Report

A 70-year-old male patient with a positive COVID-19 RT-PCR test was admitted to our hospital with complaints of fatigue, fever and increasing shortness of breath. Before his admission to the hospital, he has completed 5 days of favipiravir treatment. He has a known history of hypertension and cardiac by-pass surgery. He is not a smoker. He uses captopril 25 mg, amlodipine 10 mg. On physical examination, diffuse crepitant rales were heard in the lungs. Heart sounds were normal. There was no rebound or defense in the abdominal examination; he had no edema. His

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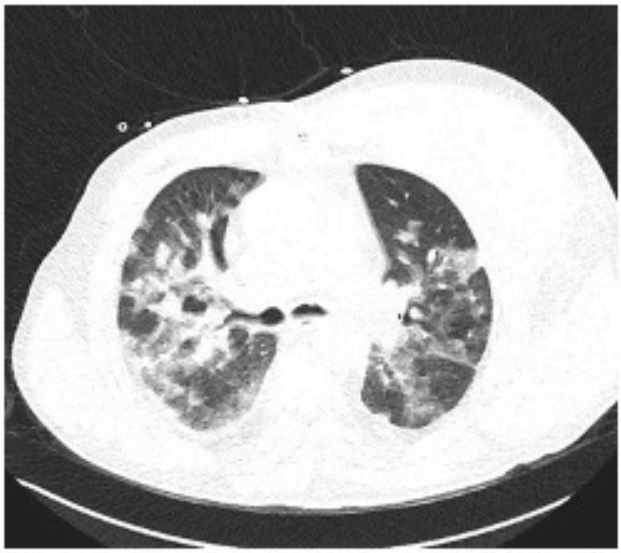

Figure 1. Extensive ground-glass consolidations in both lungs consistent with COVID-19 infection

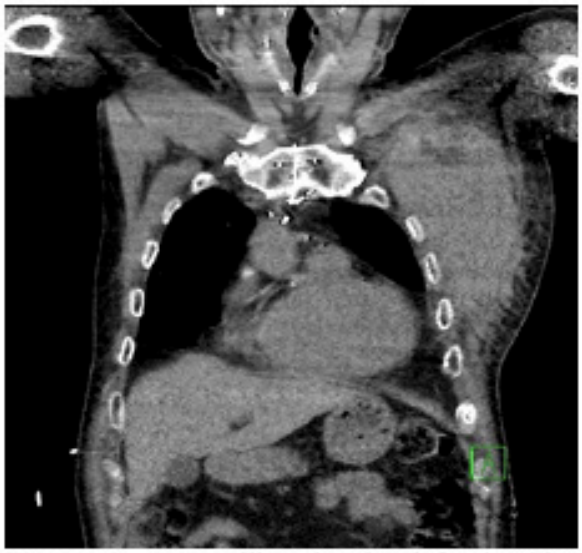

Figure 2. A swelling in the left shoulder because of 11x7 cm hematoma in the pectoralis muscle anterior to the left thorax wall

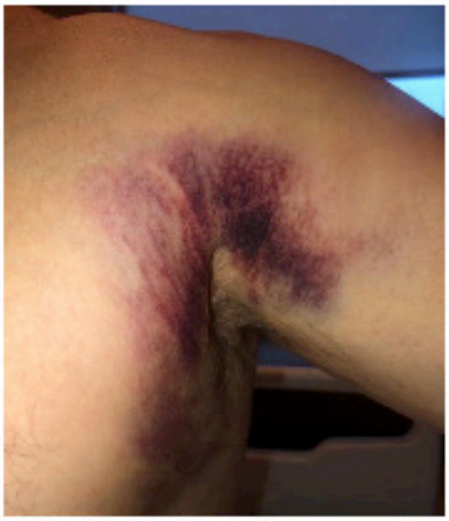

Figure 3. An ecchymosis appeared on the anterior axillary area in the second day

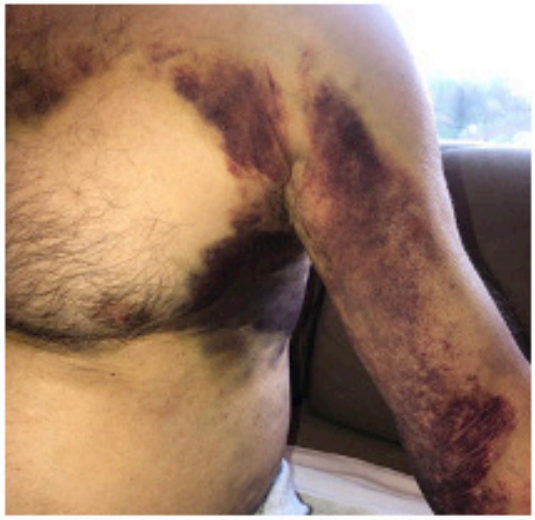

Figure 4. Ecchymosis spread to the inner surface of the arm causing stiffness in that area in the following days

weight was 78 kg. Vital findings: fever was 39.8°C, blood pressure was 128/67 mmHg, pulse was 96 beats/min, respiratory rate was 18 breathe/min. Oxygen saturation was 99% with 3 L/min oxygen support. The laboratory findings were as follows: D-dimer 4.22 ug/mL, erythrocyte sedimentation rate 67 mm/h, C-reactive protein (CRP) 18.96 mg/dL, ferritin 1544 ng/mL, fibrinogen 713 mg/dL, lactate dehydrogenase (LDH) 498 IU/L, albumin 3.10 g/dL, AST 106 IU/L, creatinine 1.92 mg/dL, glomerular filtration rate (GFR) 34.5 mL/min/1.73 m2, proBNP 1,158 pg/mL, and troponin I 0.151 ng/mL. In the CT, there were extensive ground-glass consolidations in both lungs consistent with COVID-19 infection (Figure 1). Treatment was started as favipiravir 600 mg 2x1, levofloxacin 500 mg 1x1, enoxoparin (Oksapar) 2x0.8 IU, dexamethasone 8 mg 1x1 and 1 L IV fluid. On 3rd day of his hospitalization, the patient described pain in his left chest and limitation of movement in his left arm. ECG was normal. The blood pressure was measured as 170/80 mmHg

and 50 mg of captopril was given. Perlinganit infusion was started when it was 220/130 mmHg in the follow-up. The laboratory tests were as follows: hemoglobin 8.7 g/dL, D-dimer 1.78 ug/mL, ferritin 1,109 ng/mL, fibrinogen 596 mg/dL, LDH 363 IU/L, CRP 4.47 mg/dL, AST 48 IU/L, creatinine 1.33 mg/dL, troponin I 0.060 ng/mL. All the parameters showed a decreasing trend. Only GFR increased to 53.8 mL/min/1.73 m2. In the follow-up, a swelling was recognized in the left shoulder. 11x7 cm hematoma was seen in the pectoralis muscle anterior to the left thorax wall in the thorax CT (Figure 2). Also, common ground-glass opacities and consolidated areas were evaluated as progressive. Oksapar was stopped on the 3rd day. 4 x 5 mL transamine was administered. 1 IU erythrocyte suspension was given. Codein 2x1 was added to the treatment because of hemoptysis. Two days later, an ecchymosis appeared on the anterior axillary area (Figure 3), and in the following days, it spread to the inner surface of the arm causing stiffness in that area (Figure 4). In the laboratory follow-ups, hemoglobin values were 8.0-9.0 g/dL. On the 5th day of treatment, his oxygen requirement decreased and his saturation in room air was 94%. The patient's treatment was extended to 9 days and he was discharged to come to the polyclinic control.

Discussion

Anticoagulant therapy that we are using in the treatment of COVID-19 has the adverse effect of bleeding. Therefore, the patients using anticoagulants need to be followed for a possible risk of bleeding. Especially if GFR level is low, checking anti-factor Xa levels regularly is advised. Also, creatine clearance value is very crucial in determining the correct dosage in the treatment algorithm.[3] In conclusion, anticoagulant doses should be adjusted according to anti-factor Xa and other coagulation markers. In this way, the morbidity and mortality can be decreased by reducing the risk of bleeding in patients.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Evaluation of Fever of Unknown Origin under the COVID-19 Pandemic Condition

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Abstract

HIV infection can manifest itself in many different clinical situations. In a pandemic such as COVID, when fever is of diagnostic importance and manifests itself with different clinical pictures, it is very difficult to distinguish the true cause of high fever. In this case report, we shared the process of diagnosing HIV infection in a patient with type 1 diabetes who presented with fever and neurological symptoms under pandemic conditions.

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Keywords: *HIV, COVID-19, fever of unknown origin, diabetic ketoacidosis*

Introduction

The increased diagnosis and treatment methods developed in the last 30 years improved the detection of human immunodeficiency virus (HIV) infected individuals at an earlier stage which increased the life expectancy and decreased the transmission rate of these patients.[1,2] Although patients diagnosed with HIV mostly present with infectious mononucleosis-like symptoms, some patients may present with unusual clinical presentations.3 In this case, we aimed to present a 23-year-old diabetic patient (type 1 diabetes mellitus: T1DM) admitted to hospital with generalized tonic clonic seizure and fever of unknown origin who finally diagnose with HIV infection and recovered after receiving anti-retroviral therapy.

Case Report

A 23-year-old male, who had been followed up with intensive insulin therapy for 12 years with the diagnosis of T1DM, admitted to the emergency department with complaints of high fever, nausea-vomiting and headache. Under the

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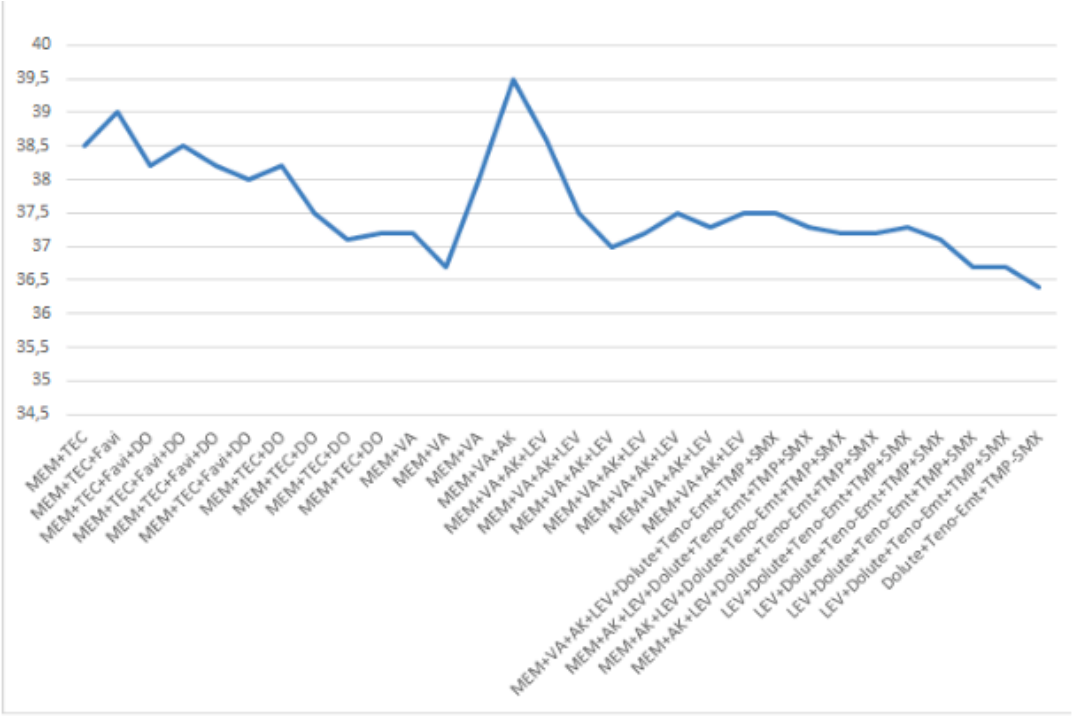

Figure 1. Fever response to antibiotics and antiviral treatments. MEM: Meropenem, TEC: Teicoplanine, Favi: Favipiravir, DO: Doxycycline, VA: Vancomisine, AK: Amikacin, LEV: Levofloxacine, Dolute: Dolutegravir, Teno-Emt: Tenofovir disoproxil + emtricitabine, TMP-SMX: Trimethoprime sulphamethaxasole

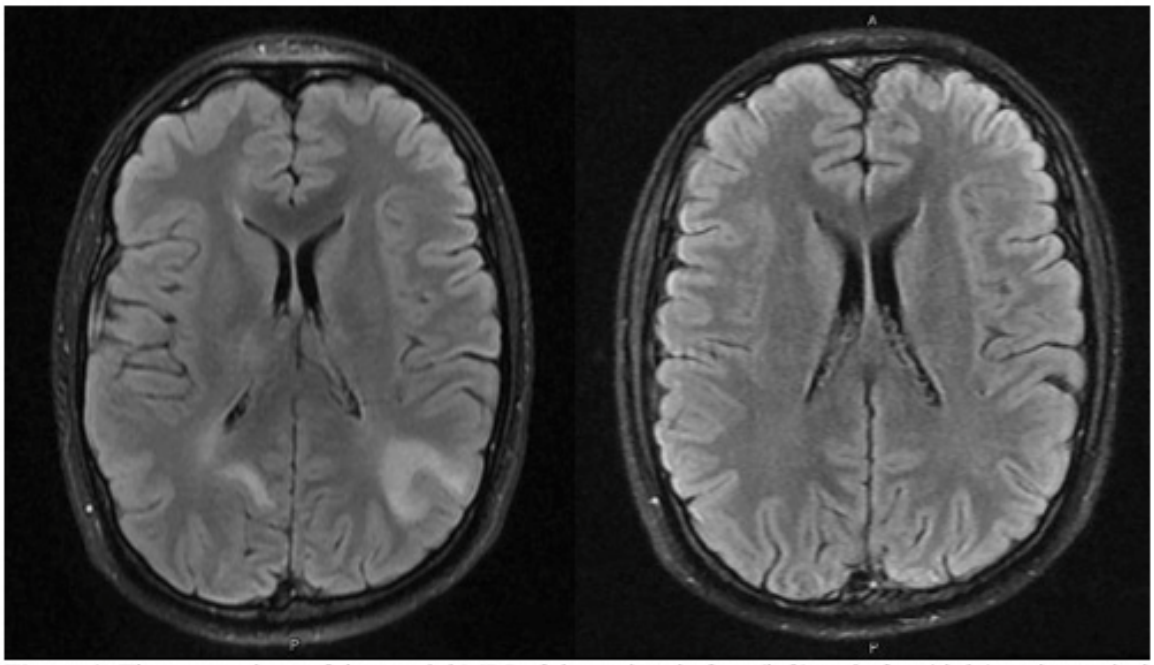

Figure 2. The comparison of the cranial MRI of the patient before (left) and after (right) antiretroviral treatment

pandemic condition, the patient was evaluated for COVID-19 and admitted to the intensive care unit after the observation of a generalized tonic clonic seizure in the emergency room. Although, he had his first generalized tonic clonic seizure six months ago he did not admit neurologist for accurate diagnosis and treatment. On physical examination, he had a fever (38.6 °C), tachycardia (pulse 110 beats/min). Chest auscultation showed decreased respiratory sounds at lower zones. Neurological examination revealed retrograde amnesia and a positive Babinski reflex on the left.

In laboratory examinations, plasma glucose level was 420 mg/dL, +3 ketone in the urine,

arterial pH: 7.18, ferritin >2,000, CRP: 109 mg/dL, and procalcitonin >50 pg/mL. Minimal pleural effusion in both hemithoraxes, and a light ground glass density with unclear borders in the right lung middle lobe lateral segment posterior peripheral area was observed in chest computed tomography (CT). In cranial magnetic resonance imaging (MRI), high signal areas and mild edema signals observed in the ventrals of both temporal lobes and temporoparietal lobes, subcortical areas in T2 and Flair were noted, and it was reported that those findings may be associated with metabolic events or encephalomyelitis.

Diabetic ketoacidosis treatment protocol was initiated. Meropenem and teicoplanin antibiotherapy was started empirically due to increased acute phase reactants including procalcitonin and fever. Favipiravir was started with the pre-diagnosis of COVID-19 pneumonia due to the ground-glass densities in chest CT. The patient, whose fever did not respond to treatment during the follow-up, was evaluated as pericarditis, and colchicine and ibuprofen treatment was initiated. The treatments and fever response of the patient are shown in Figure 1. Since the patient's headache complaint did not resolve and retrograde amnesia was observed in the repeated mini mental test the treatment was changed with the pre-diagnosis of encephalitis. The anti-HIV test requested from the patient, whose fever and neurological findings continued despite the treatment changes, was reported as positive. HIV-RNA was detected as 115,200 copies/mL, and the CD4/CD8 ratio of the patient was measured as 0.23. Lumbar puncture was performed on the patient to evaluate in terms of opportunistic infections. No cells were observed in the cell count of the sent cerebrospinal fluid (CSF) samples, glucose, protein and LDH levels were observed as 113 mg/dL (simultaneous blood glucose 139 mg/dL), 50 mg/dL, and 22 U/L, respectively. Bacteriological, mycological and mycobacteriological growth was not detected in CSF cultures. ARB and VDRL tests were negative in CSF, meningitis-encephalitis panel (CMV-DNA, VZV-DNA, HSV1-DNA, HSV2-DNA, HHV6-DNA, Enterovirus-RNA, H. influenzae, N. meningitidis, S. pneumoniae, C. neoformans) resulted as negative. Rose-Bengal test performed for brucellosis was also negative. After the initiation of trimethoprim-sulfamethoxazole and dolutegravir, tenofovir disoproxil+emtricitabine for opportunistic infection prophylaxis and anti-retroviral therapy, the patient's fever decreased. No pathology was observed in the control cranial MRI taken 1 week after the patient's neurological examination showed improvement. The comparison of the cranial MRI of the patient before and after antiretroviral treatment is given in Figure 2.

Discussion

Despite the multiple antibiotic treatments given for high fever, as seen in Figure 1, it was observed that the high fever and neurological findings of the patient continued. Patient's fever decrease just after the initiation of anti-retroviral treatment. Although there is no response to broad spectrum antibiotic treatments given for the fever symptom, which is often associated with opportunistic infections during the course of AIDS, it can be speculated that HIV itself may cause high fever, based on the regression of fever just after the initiation of antiretroviral treatment.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

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Case Report

A case report of euglycemic ketoacidosis due to dapagliflozin treatment

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Abstract

Diabetic ketoacidosis (DKA) is a leading cause of mortality and morbidity in type 2 diabetic patients. Sodium-glucose co-transporter (SGLT-2) inhibitors are a new antidiabetic treatment class that increases the renal excretion of glucose. The Food and Drug Administration issued a warning in May 2015 notifying that patients using this class of anti-diabetic drugs may develop DKA. Risk factors for DKA development among patients who take SGLT-2 inhibitors include carbohydrate intake/starvation or acute illness. In the current report, we aimed to present a case of euglycemic DKA using dapagliflozin treatment.

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Keywords: *dapagliflozin, sodium-glucose co-transporter 2 inhibitors, euglycemic ketoacidosis*

Introduction

Recently, new drugs have been developed that reduce plasma glucose levels by inhibiting sodium-glucose co-transporter 2 (SGLT-2) that provide glucose reabsorption in the ultrafiltrate. SGLT-2 inhibitors also called "gliflozins", reduce glucose reabsorption by causing SGLT2 inhibition in the proximal tubules in the kidneys and increase urinary glucose excretion.[1] Dapagliflozin was approved by Europe and the United States as the SGLT-2 inhibitor to be used in the treatment of diabetes. Euglycemic ketoacidosis (EKA) is a rare form of diabetic ketoacidosis that occurs without a severely elevated blood sugar level.[2] It has been reported that patients using SGLT-2 inhibitors have developed EKA as a side effect in the follow-up.[3–5] The mechanism by which SGLT-2 inhibitors cause the development of EKA is not fully elucidated. It is thought that the decrease in insulin secretion from the pancreas due to urinary glucose excretion may increase the glucagon/insulin ratio, and this situation predisposes to increased gluconeogenesis, ketogenesis, and ketoacidosis.[3,6] Herein, we aimed to present an EKA case that developed after the use of dapagliflozin treatment.

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Case Report

A 57-year-old male patient, who had diabetes for 20 years, was admitted to our center with complaints of nausea, dizziness, and weakness. There was no pathological finding in the physical examination of the patient. In venous blood gas, pH: 7.3, lactate: 2.6 mmol/L, and HCO3: 10 mmol/L was detected. In the biochemical analysis, fasting plasma glucose was 167 mg/dL, creatinine level 0.83 mg/dL, and HbA1c level was 9.5%. Urinalysis showed ++ ketone and ++++ glucose, despite serum glucose was below 200 mg/dL. It was learned that the patient used metformin 1000 mg twice daily, linagliptin 5 mg once daily, and dapagliflozin 10 mg once daily for the treatment of diabetes. The patient was diagnosed with EKA. Dapagliflozin treatment was stopped, and insulin infusion was started in addition to fluid therapy. The patient's acidosis status improved with urine ketone becoming negative after intravenous fluid therapy and insulin treatment. Diabetes treatment of the patient has arranged as insulin glargine 14 units in addition to vildagliptin 50 mg twice daily and metformin 1000 mg twice daily. The patient was discharged by recommending outpatient follow-up.

Discussion

If there is no contraindication, SGLT-2 inhibitors are recommended as antidiabetic drugs in patients with a history of atherosclerotic cardiovascular disease, a history of heart failure, or a high risk of atherosclerotic cardiovascular disease.7 Before giving SGLT-2 inhibitors in treatment, a detailed history should be taken, and factors that may predispose the patient to ketoacidosis should be evaluated. SGLT-2 inhibitors should be used with caution in patients with low insulin reserve, restricted oral intake, decreased carbohydrate intake, and cases where insulin needs increase due to acute medical illness or surgery.3,6,8 EKA should be kept in mind in the differential diagnosis, especially in patients using SGLT-2 inhibitors and in whom acidosis is found despite not having very elevated blood sugar levels.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

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Case Report

Recognizing the sarcoidosis behind the window of infection

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Abstract

Sarcoidosis is a chronic multisystemic inflammatory disease of unknown etiology, often associated with pulmonary involvement and characterized histopathologically by noncaseating granulomatous lesions. In this report, we aimed to present a 32-year-old patient who was hospitalized with a pre-diagnosis of cellulite, diagnosed with sarcoidosis based on skin lesions, and whose symptoms completely regressed with sarcoidosis treatment.

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Keywords: *sarcoidosis, cutaneous, erythema nodosum*

Introduction

Sarcoidosis is a chronic multisystemic inflammatory disease of unknown etiology, often associated with pulmonary involvement and characterized histopathologically by noncaseating granulomatous lesions. The most common clinic features are respiratory symptoms (cough, dyspnea, bronchial hyperreactivity), fatigue, night sweat, weight loss, and erythema nodosum. Fifty percent of the sarcoidosis patients are asymptomatic, and their diagnosis are usually made with the detection of the bilateral and/or unilateral, hilar and/or paratracheal fullness, seen in the chest X-ray taken for other reasons.[1] Cellulitis is a suddenly starting and rapidly spreading infection that affects the skin and subcutaneous adipose tissue. The responsible pathogen is usually Staphylococcus Aureus and Streptococcus Pyogenes. They are generally unilateral lesions in the lower extremities that do not have a sharp boundary with intact skin. It may present with local symptoms such as erythema, swelling, warmth, pain and tenderness or systemic symptoms such as fever, chill, shivering and malaise. These signs of cellulitis sometimes can be confused with erythema nodosum seen in the course of sarcoidosis.[2]

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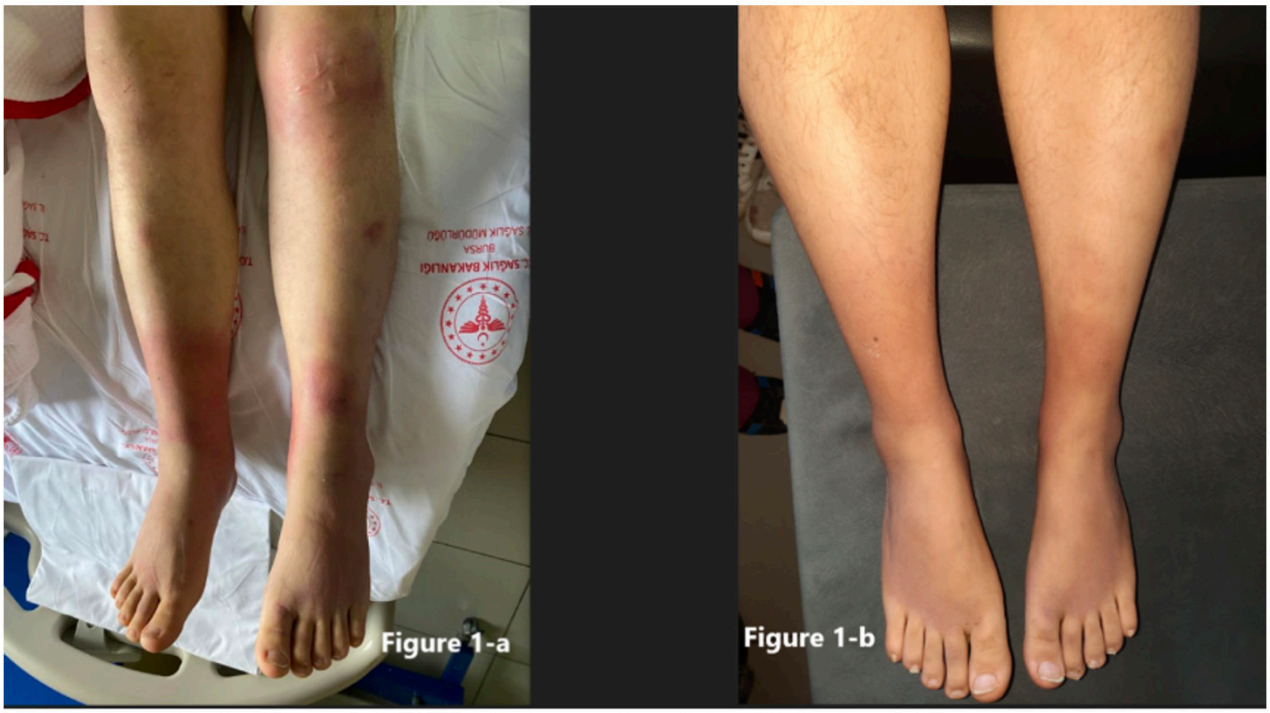

Figure 1. a: skin lesions on admission, b: regression of the skin lesions after treatment.

Case Report

A 32-year-old patient who have repetitive admission to several hospitals with the complaints of chills, shivering, fever, and redness of the legs in the last month. Various oral and parenteral antibiotics was prescribed with a pre-diagnosis of cellulitis. During the follow-up the patient's complaints did not relieve and the lesion which was initiated from the right tibial region spreaded to both lower and upper extremities (Figure 1a). The lower extremity superficial tissue ultrasonography was reported as cellulitis. He was hospitalized with the pre-diagnosis of multiple cellulitis for further examination. The history of the patient related to the lesions reveals that the lesion was first appeared three weeks ago and progressed despite the treatments given in the outpatient clinic. Clinical and laboratory findings did not improve despite antibiotic treatment. Erythema nodosum was considered as a differential diagnosis. Chest X-ray reveals bilateral hilar lymphadenopathy. Steroid and indomethacin treatment was initiated with the pre-diagnosis of sarcoidosis, and antibiotherapy was stopped. During follow-up, the skin lesions were completely resolved within 3 days (Figure 1b). Further evaluation for the other organ involvement of sarcoidosis was performed. No uveitis, cardiac arrythmia, hepatomegaly and splenomegaly were observed. The patient was discharged after the complete clinical and laboratory remission. Keeping the differential diagnoses on a large scale and making the true diagnosis among these differential diagnoses is the key to reach an effective treatment promptly. Although radiological and laboratory tests are powerful guides in diagnosis, the role of history taking, and physical examination is undeniable.

Discussion

Early diagnosis of the patient with detailed history, physical examination and accurate laboratory and imaging tests are important for starting an effective treatment as soon as possible. The early diagnosis of a systemic, chronic disease affects the prognosis of the disease positively. Early initiation of necessary treatments by early

recognition of multiple organ involvement and chronic injuries plays an active role in preventing complications related to disease-related chronicity. The most difficult patients are the patient who come with a pre-diagnosis. For this reason, we should approach each patient with an objective perspective and consider possible differential diagnoses, even if they come with a prediagnosis.

Conflict of Interests

Authors declare that there are none.

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Case Report

Thyrotoxic Hepatitis in a Patient with Subacute Thyroid: A Case Report

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Abstract

Subacute granulomatous thyroiditis (DeQuervain's disease) is a self-limiting inflammatory disease which is often diagnosed in the "temporary thyrotoxicosis period" during the initial phases of the disease. Liver enzyme abnormalities can be seen in the course of thyrotoxicosis. The definition of "thyrotoxic hepatitis" is used to describe the elevation of liver enzymes after excluding other causes. In this case report, a female patient who developed thyrotoxic hepatitis in the course of her subacute thyroiditis, is reported to remind the importance of evaluating liver functions in the follow-up of subacute thyroiditis.

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Keywords: *Thyrotoxic Hepatitis, Subacute Thyroiditis, Liver Enzyme Abnormalities*

Introduction

Subacute granulomatous thyroiditis (DeQuervain's disease) is a self-limiting inflammatory disease with a triphasic clinical course -hyperthyroidism, hypothyroidism and euthyroidism- frequently seen in young and middle-aged women during seasonal transition periods. In the etiology, viral (adenovirus, coxsackievirus, mumps, EBV, and influenza) infections and genetic predisposition (HLA-BW35) are blamed while autoimmune response is not. Weakness, fatigue, myalgia, mild/moderate fever and arthralgia are common features in patients with subacute thyroiditis. Severe neck pain in the thyroid gland region often radiating to the jaws or ears is observed. On physical examination, the thyroid gland is enlarged and sensitive. In laboratory tests ESR, CRP are elevated. The disease is often diagnosed in the early "temporary thyrotoxicosis period". In this phase, thyroid gland parenchyma destruction secondary to inflammation causes increase in blood fT3 and fT4 levels and suppression of TSH. Thyroid radioactive iodine131 uptake (RAIU) is low (<2% at the 24th hour). After stored colloid in the follicles is completely discharged, hypothyroidism phase initiates. During the recovery period, parenchyma cells regain hormone

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producing capacity in several weeks and the euthyroid period is entered. The hypothyroidism rarely becomes permanent.[1,2]

Liver enzyme abnormalities can be seen in the course of thyrotoxicosis. The diagnosis of "thyrotoxic hepatitis" can be made after excluding other causes of liver pathology. In the pathogenesis of thyrotoxic hepatitis, the inability to compensate for the increase in hepatic oxygen demand (perivenular hypoxemia), autoimmunity, thyrotoxicosis complications such as heart failure. etc are held responsible. Thyrotoxic hepatitis clinical picture ranges from asymptomatic enzyme elevation to severe liver damage but is generally self-limiting.[3-5] Here, we want to report a patient who had concurrent hepatitis in the thyrotoxic period of subacute thyroiditis and whose clinic improved with symptomatic treatment, to remind the importance of evaluating liver functions in the follow-up of subacute thyroiditis.

Case Report

A 35-year-old female patient without known chronic disease was admitted to our clinic with complaints of generalized body pain, weakness, hair loss, palpitations, dyspnea on exertion and sore throat for a month. Physical examination showed the thyroid gland was enlarged and tender to palpation. Lab studies showed TSH: 0.01 mU/L (0.34-5.60), fT4: 41.29 ng/L (6.1-11.2), anti-TPO: 0.3 kU/L (0-9), ALT: 110 U/L (0-50), AST: 118 U/L (<35), ALP: 254 U/L (33-98), GGT: 129 U/L (0-38), and CRP: 94.35 mg/L (<5). Hemogram, ferritin, vitamin B12, glucose and creatinine results were normal. Thyroid ultrasonography revealed thyroid parenchyma was hypoechoic-heterogeneous and the gland was diffusely enlarged. The RAIU test was found to be 4% (8-15) at the 2nd hour and 1% (15-35) at the 24th hour. The patient was started on 1mg single dose dexamethasone, propranolol 40 mg 2x1/2, (etodolac 400 mg 2x1, paracetamol 500 mg 2x1, when necessary). Tests performed 5 weeks post-treatment showed: TSH: 13.2 mU/L (0.34-5.60), fT4: 4.22 ng/L (6.1-11.2), ALT: 21 U/L (0-50), AST: 23 U/L (<35), ALP: 83 U/L (33-98), GGT: 30 U/L (0-38), and CRP: 1.12 mg/L (<5). The patient, who was treated with L-thyroxine during the hypothyroid period, was planned to be followed up with 4 weeks intervals.

Discussion

Since thyrotoxicosis occurs without hyperthyroidism in the course of subacute thyroiditis, there is no place for antithyroid drugs in the treatment. Beta blocker drugs are used to reduce the systemic effect of elevated thyroid hormones during the thyrotoxicosis phase while analgesics/NSAIDs and short-term steroid therapy can be used for fever and pain. Similar to the conditions in hyperthyroidism, the possibility of developing acute hepatitis during thyrotoxicosis of subacute thyroiditis should be kept in mind. The patient's liver functions should be monitored regularly. Likewise, thyroid functions should definitely be evaluated in the etiology of patients presenting with acute hepatitis.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

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Case Report

A Case of Resistant Hypocalcemia Treated with Teriparatide

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Abstract

Postsurgical chronic hypoparathyroidism is most commonly seen in adults. Synthetic recombinant human parathyroid hormone (rhPTH) 1-34 can be used to stabilize serum calcium levels in patients with resistant hypocalcemia in rare conditions. Here we report a 52-year-old woman with postsurgical uncontrolled hypocalcemia despite the usual therapy. Once-daily treatment with PTH 1-34 maintained serum calcium within the normal range and reduced the dose of previous medical therapies.

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Keywords: *Hypoparathyroidism, hypocalcemia, teriparatide*

Introduction

Parathyroid hormone (PTH) is the main hormone that regulates serum calcium levels by affecting kidneys, bones and gastrointestinal tract. Hypoparathyroidism is a rare disease accompanied by hypocalcemia that occurs in the absence or deficiency of PTH synthesis. Postsurgical chronic hypoparathyroidism is the most common cause in adults.[1] Second most common cause is autoimmune hypoparathyroidism.[2] Post-surgical transient hypocalcemia is more common and calcium replacement alone may be sufficient. The incidence of permanent hypocalcemia is 2% in the postsurgical patients operated for hyperparathyroidism.[3] Oral calcium and vitamin D supplementation is used for the initial management of patients with chronic hypoparathyroidism. Serum calcium levels cannot remain within normal ranges despite calcium and vitamin D supplementation in rare instances. In these situations, synthetic recombinant human parathyroid hormone (rhPTH) 1-34 can be used once or twice daily to stabilize serum calcium levels in patients with chronic hypoparathyroidism.[4] In this case report, we described a 52-year-old woman with postsurgical uncontrolled hypocalcemia despite the usual therapy with calcium, magnesium and calcitriol. Once-daily treatment with PTH 1-34 maintained serum calcium within the normal range and reduced the dose requirement of previous medical therapies.

Case Report

A 52-year-old woman with Graves' disease underwent a total thyroidectomy in the year 2000. She had persistent hypocalcemia after

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the surgery. She was treated with levothyroxine sodium, calcium and vitamin D supplements. She had cataract surgery 10 months ago. She had multiple emergency department admissions and hospitalization due to recurrent hypocalcemia symptoms and signs. When she was admitted to the endocrine clinic, the laboratory evaluation confirmed severe hypocalcemia due to postsurgical hypoparathyroidism: calcium was 5 mg/dL, albumin was 4.1 g/dL, phosphorus was 4.1 mg/dL, magnesium was 1.6 mg/dL, 25-OH vitamin D was 14.8 mcg/L and parathyroid hormone (PTH) was 9 ng/L. Celiac autoantibodies were undetectable. Bone mineral densitometry was normal. Teriparatide (recombinant human PTH) was begun with the approval of the Ministry of Health. There was no emergency department admission or hospitalization within 9 months after the initiation of teriparatide treatment. At the last control of the patient, the calcium level was increased to 7.4 mg/dL and phosphorus was decreased to 2.7 mg/dL. The 25-OH vitamin D level was markedly increased to 34.5 mcg/L. The treatment of the patient continues with calcium, magnesium, vitamin D supplements, and teriparatide.

Discussion

Most of the patients with hypoparathyroidism are treated with high-dose vitamin D (calcitriol, cholecalciferol or ergocalciferol) and calcium supplements.[5] As a result of these treatments some patients may have hyperphosphatemia and hypercalciuria that increase the risk of renal disease. In a randomized controlled study comparing once-daily treatment with rhPTH 1-34 and calcitriol, it was reported that serum calcium levels could be kept within the normal ranges and urinary calcium excretion could be reduced with rhPTH 1-34 therapy.[4] However, rhPTH 1-34 is not yet first line therapy, because of high cost and subcutaneous administration. In our case, rhPTH 1-34 treatment was initiated due to resistant hypocalcemia. Serum calcium levels were normal or close to normal during follow-up period.

In a study of 31 patients treated with rhPTH 1-34, it was reported that 16 patients developed new-onset or progressive nephrocalcinosis due to hypocitraturia.6 In a patient who was followed up for 3 years, severe joint pain was developed with the use of rhPTH and the pain decreased with the discontinuation of the drug.[7] Since our case has been followed up for almost 1 year, none of the mentioned side effects was observed.

In conclusion, the use of rhPTH 1-34 is a good option in patients with resistant hypoparathyroidism. However, long-term follow-up and studies involving a large number of patients are needed in terms of side effects and complications.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

A Case of very Severe Hypertriglyceridemia during Pregnancy

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Abstract

Severe hypertriglyceridemia is defined as serum triglyceride levels of 1000 mg/dL and more. Acute pancreatitis and fetal losses can be observed in association with severely high triglyceride levels during pregnancy. Here we report the management of a 28-year-old pregnant woman with very severe HTG with the data in the literature.

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Keywords: *Severe hypertriglyceridemia, pregnancy, pancreatitis*

Introduction

Hypertriglyceridemia (HTG) is defined as serum triglyceride levels of 150 mg/dL or more.[1] According to the Endocrine Society guidelines, serum triglyceride levels between 1000-1999 mg/dL are categorized as severe HTG and over 2000 as very severe HTG. In a recent analysis, more than 25% of all US adults (56.9 million individuals) have triglyceride (TG) levels ≥150 mg/dL.[2] The risk of developing acute pancreatitis at TG levels above 2000 mg/dL is approximately 10-20%.[3] During pregnancy, triglyceride and total cholesterol levels increase physiologically with the effect of estrogen.[4] Acute pancreatitis and fetal losses can be observed in association with severe triglyceride levels during pregnancy.[5] In this case, we evaluated the management of a pregnant patient with very severe HTG with the data in the literature.

Case Report

A 28-year-old female patient with a history of acute pancreatitis due to hypertriglyceridemia was admitted to our clinic at 18th week of her second pregnancy. She had hypertriglyceridemia-induced acute pancreatitis during her first pregnancy 2 years ago. She was treated with therapeutic apheresis to achieve a safe TG level for preventing the complications of acute

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pancreatitis. She subsequently delivered a baby with agenesis of the corpus callosum by natural spontaneous birth. She reported no follow-up after she was discharged. She was referred to our clinic by obstetrics and gynecology department due to high triglyceride levels. Serum lipid profile revealed following findings; TG: 3117 mg/dL; total cholesterol: 337 mg/dL; HDL cholesterol: 35 mg/dL. Her amylase and lipase levels were within normal limits. On examination, she had no signs and symptoms, blood pressure: 120/70 mmHg and pulse rate: 98/min, regular. Medical nutrition therapy, omega-3 supplementation at 1000 mg daily, and intravenous regular insulin infusion (0.05 IU/kg/h) were started. She was discharged at the third day with triglyceride concentration of 669 mg/dL and total cholesterol of 227 mg/dL.

Discussion

During pregnancy, VLDL and TG levels increase physiologically 2-3 times of nonpregnant levels. Hyperlipidemia is the most common cause of patients diagnosed with acute pancreatitis after gallstones and alcohol and is seen in 1-14% of cases.[6] The risk of preeclampsia, pancreatitis, hyperviscosity syndrome, preterm delivery and fetal death increases when TG levels greater than 2000 mg/dL.[7] Therefore, treatment should be started immediately. In the treatment, besides dietary regulation and lifestyle changes, intravenous insulin, omega-3 support, plasmapheresis and other medical treatments are used.[8] Inhibition of lipolysis in adipose tissue, a decrease in circulating free fatty acid levels and lipoprotein lipase activation may reduce serum TG levels by using insulin therapy. However, the guidelines do not recommend insulin infusion therapy in cases of severe hypertriglyceridemia without diabetes. They recommend the therapeutic apheresis approach in very severe HTG cases that do not respond to pharmacological treatments.[9] With the appropriate and timely multidisciplinary treatment approach, the risk of developing complications can be reduced..

Conflict of Interests

Authors declare that there are none.

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Case Report

Pituitary Stalk Interruption Syndrome: A Case Report

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Abstract

Panhypopituitarism occurs as a result of the insufficiency of all hormones produced in the anterior pituitary gland. Pituitary stalk interruption syndrome (PSIS) is a rare congenital syndrome leading to hypopituitarism. The anterior pituitary hormones should be evaluated in patients with the signs and symptoms of hormone insufficiency and magnetic resonance imaging of the pituitary should be performed for the etiology. Although PSIS is a rare cause of hypopituitarism, it must be treated with the replacement of the insufficient hormones. In this case report, we evaluated a PSIS case presented with panhypopituitarism.

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Keywords: *Pituitary stalk interruption syndrome, hypopituitarism, hormone replacement*

Introduction

Panhypopituitarism is the name given to the clinical situation that occurs as a result of the insufficiency of all hormones produced in the anterior pituitary gland. Pituitary stalk interruption syndrome (PSIS) is a rare congenital syndrome that is usually presented with the hypoplastic pituitary gland, undemonstrated pituitary stalk, and ectopic neurohypophysis. PSIS is an intrinsic pituitary cause of panhypopituitarism. Available data suggests that complex genetic patterns and environmental factors work together for PSIS.[1,2] Although the disease onset varies regarding age, it can typically be seen in pediatric ages as combined anterior pituitary hormone deficiencies and can progress to panhypopituitarism even in adulthood. Therefore, lifelong follow-up of patients is essential for sufficient management. In this case, we aimed to report a PSIS case presented with panhypopituitarism.

Case Report

A 22-year-old female patient, who was followed up with a diagnosis of panhypopituitarism, was admitted to our endocrinology outpatient clinic with complaints of anorexia, weakness, nausea, and vomiting. It was discovered that she had an involuntary weight loss of approximately 5% in the last 2 months and did not comply with levothyroxine sodium and fludrocortisone treatments. Also, she was amenorrheic. During the physical examination, her blood pressure was

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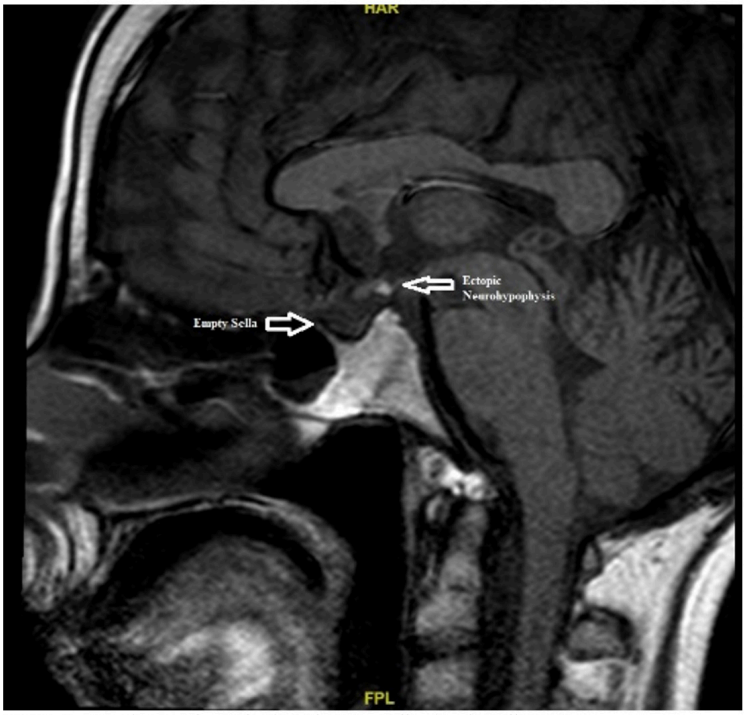

Figure 1. Ectopic neurohypophysis and empty Sella view in Sella MRI.

100/60 mmHg, pulse rate was 130 beats/min, rhythmic and body temperature was measured to be 36.4 °C. Cardiovascular assessments were normal. No pathological finding was found in other physical examinations. In the laboratory investigation during admission, her serum creatinine was 0.75 mg/dL, sodium 136 mmol/L, potassium 3.8 mmol/L, AST 21 U/L, and ALT 9 U/L. As a result of the clinical and laboratory evaluation, the patient was suspected to have adrenal insufficiency and administered hydrocortisone and hospitalized in the endocrinology clinic. Her follow-up and treatment were initiated. In the examinations performed during hospitalization, her anterior pituitary hormone levels were lower than normal; ACTH: 5 ng/L, TSH: 0.07 mU/L, free T3: 1.73 ng/L, free T4: 1.10 ng/dL, FSH: 0.24 IU/L, LH: <0.09 IU/L, GH <0.05 ug/L, somatomedin C (IGF-1) <15, cortisol 0.8 µg/dL, estradiol 10 ng/L, and progesterone <0.1 µg/L. Sella magnetic resonance imaging (MRI) showed an image compatible with ectopic neurohypophysis, empty Sella syndrome, and PSIS (Figure 1). Oral hydrocortisone and oral contraceptive replacement treatments were initiated in appropriate doses for the patient, and other hormonal treatments were arranged accordingly. After observation of clinical improvements, the patient was discharged and asked to attend regular outpatient follow-ups.

Discussion

As in our case, hypopituitarism may present with various clinical findings. The anterior pituitary panel should definitely be examined in patients who have signs of hormone insufficiency. After the diagnosis is made, magnetic resonance imaging of the pituitary gland should be performed for the etiology. Although PSIS is a rare cause of hypopituitarism, its treatment should include the replacement of the insufficient hormones like thyroxine, glucocorticoids, sex steroids and growth hormone when necessary. The prognosis of the disease is good under regular follow-ups and treatments.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Premenopausal Osteoporosis in A Patient with Autoimmune Polyglandular Syndrome: A Case Report

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Abstract

Osteoporosis is a skeletal disease characterized by low bone mass associated with decreased bone strength and increased risk of fractures. Low bone mass in premenopausal women is less common than in postmenopausal women, and bone loss in premenopausal women is usually due to secondary causes such as estrogen deficiency, glucocorticoid exposure, malabsorption, thyroid disorders, and hyperparathyroidism. In women with premenopausal osteoporosis, treatment should be planned according to the underlying secondary causes. In this case report, the importance of investigating the secondary causes leading to premenopausal osteoporosis and its treatment are discussed.

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Keywords: *Autoimmune polyglandular syndrome, diagnosis, etiology, premenopausal osteoporosis, treatment*

Introduction

Osteoporosis is a skeletal disease characterized by low bone mass associated with decreased bone strength and increased risk of fractures.[1] Low bone mass may be associated with insufficient peak bone mass acquisition and/or ongoing bone loss. Peak bone mass which is acquired 90% of its total adult value by the age of eighteen, occurs totally around the age of 30.[2] Most of peak bone mass formation is determined by family history, gender and race while nutrition and exercise are responsible for 25% of peak bone mass formation. Physiological bone loss that starts after the age of 35 in women accelerates with menopause. Osteoporosis is most commonly seen in postmenopausal women. Low bone mass is less common in young premenopausal women.[3,4] Bone loss in the premenopausal period may be due to secondary causes such as estrogen deficiency, glucocorticoid exposure, malabsorption, thyroid disorders, and hyperparathyroidism, as well as idiopathic factors. Bone mineral densitometry measurements alone should not be used to define premenopausal osteoporosis. Premenopausal osteoporosis in a young woman can be defined as low bone mineral density (BMD) for age (Z score ≤-2.0) together with the presence of fractures in

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Table 1. Secondary causes of osteoporosis in premenopausal women

Anorexia nervosa
Gastrointestinal malabsorption (celiac disease, postoperative states)
Diabetes mellitus
Vitamin D and/or calcium deficiency
Hyperthyroidism
Hyperparathyroidism
Cushing's syndrome
Hypogonadism
Hypercalciuria
Rheumatoid arthritis and other inflammatory conditions
Alcoholism
Renal disease
Liver disease
Homocystinuria
Hereditary hemochromatosis
Hematologic disorders (systemic mastocytosis, Gaucher disease, thalassemia major)
Medications (glucocorticoids, immunosuppressants,antiseizure medications [phenobarbital and phenytoin], GnRH agonists, heparin, chemotherapeutics, thiazolidinediones, depot medroxyprogesterone acetate, excess thyroid hormone, selective serotonin reuptake inhibitors, proton pump inhibitors

bones or presence of any risk factor for secondary osteoporosis (Table 1).

Case Report

Our case was a 21-year-old female patient who was diagnosed with autoimmune polyglandular syndrome due to presence of type 1 diabetes mellitus, autoimmune thyroiditis, autoimmune primary adrenal insufficiency, Sjögren's disease, fibromyalgia, primary biliary cholangitis and autoimmune hepatitis. After long-term steroid use, she applied to another clinic with the complaint of diffuse bone pain and was diagnosed with osteoporosis after clinical evaluation and bone mineral densitometric measurements. The patient was then admitted to our endocrinology outpatient clinic with complaints of recurrent bone pain and amenorrhea. In the current bone mineral density measurement, total Z score, T score and BMD were -3.1, -3.1, and 0.620 g/cm^2 for femur and -2.2, -2.5, and 0.892 g/cm^2 for L1-L4, respectively. The patient was scheduled for further diagnostic tests for secondary causes of premenopausal osteoporosis. Blood chemistry showed a calcium level of 8.2 mg/dL [normal range (NR): 8.4-10.2] with a phosphorus level of 2.9 mg/dL (NR: 2.4-4.4). 25-OH vitamin D level was 13.3 µg/L (NR: 20-50), while parathormone level was 186 ng/L (NR: 15-68.3) and ALP level was 88 U/L (NR: 40-150). Laboratory results for sex hormones included a FSH level of 0.73 IU/L (NR: 1.38- 5.47) with a LH level of 0.21 IU/L (NR: 0.56-14.0), an estrogen level of 24 ng/L (NR: 21- 312) and a progesterone level of 1 ng/mL (NR: 1- 4.5). Pertinent blood work for thyroid function resulted with normal TSH 2.17 mU/L (NR: 0.35-4.94) and free T4 levels 1.08 ng/dL (NR: 0.7-1.48) under replacement treatment. Further tests revealed high blood glucose level 133 mg/dL (NR: 70-100) and a HbA1c level of 7.5% (NR: 4-6%). AST level was 14 U/L (NR: 11-25), while ALT level was 17 U/L (NR: 7-28) and creatinine level was 0.67 mg/dL (NR: 0.56-0.85). Anti-tissue transglutaminase IgA level was detected to be 0.7 U/mL (NR: 0-10).

The patient was hospitalized and an esophagogastroduodenoscopy (EGD) was performed because she complained of nausea that did not regress with antiemetic therapy. Grade A esophagitis, laxity in the lower esophageal sphincter, food residues in the stomach and scalloped duodenal folds were detected during EGD. Gastroparesis secondary to diabetes mellitus was suspected in the patient who had food residues in the stomach during endoscopy after 12 hours of fasting. A biopsy was taken from the scalloped duodenal folds with a prediagnosis of celiac

disease, however no pathological finding other than chronic mucosal inflammation was reported. The patient was diagnosed with autoimmune polyglandular insufficiency. Combined estrogen and progesterone treatment was initiated for her amenorrhea. Vitamin D and calcium replacement therapy was given for vitamin D deficiency and secondary hyperparathyroidism. The dosage of steroid treatment for her autoimmune hepatitis and adrenal insufficiency was rearranged. Bisphosphonate treatment was not planned because she was in reproductive age. Thyroid hormone and insulin replacement therapies were continued.

Discussion

The diagnosis and treatment approaches for osteoporosis differ in premenopausal and postmenopausal women. The relationship between BMD and fracture risk in premenopausal women is not correlated, and the prevalence of fracture is much lower than in postmenopausal women. Since the relationship between bone mass and fracture risk in premenopausal women is not the same as postmenopausal women, guidelines for the diagnosis and treatment of postmenopausal osteoporosis do not apply to women diagnosed with premenopausal osteoporosis. In women diagnosed with premenopausal osteoporosis, treatment should primarily address the underlying cause. Lifestyle changes as well as a pharmacological treatment with calcium (1000 mg/day) and cholecalciferol (800-1500 IU/day) are recommended in premenopausal osteoporosis. Lifestyle modification includes regular load-bearing exercises (such as walking), cessation of smoking and alcohol consumption and limiting caffeine intake. Estrogen replacement has beneficial effects on bone mass in women with premenopausal osteoporosis secondary to hypogonadism.[5] Although there are publications showing that bisphosphonates are beneficial in women diagnosed with osteoporosis secondary to glucocorticoid usage, their use is not recommended in childbearing age women.[6] As in our case, the treatment plan in premenopausal osteoporosis should be tailored according to the patient, and if there is an underlying secondary cause, the treatment should be directed primarily to this etiology.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Hypertriglyceridemia Induced Recurrent Pancreatitis Case

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Abstract

Hypertriglyceridemia is a common cause of acute pancreatitis. Metabolic syndrome can lead to the development of hypertriglyceridemia. Here we presented a pancreatitis case with type 2 diabetes, obesity, and dyslipidemia.

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Keywords: *Hypertriglyceridemia, acute pancreatitis, etiology, treatment*

Introduction

The diagnosis of acute pancreatitis (AP) consists of the history and typical clinical criterias of the patient, serum amylase and lipase levels being 3 times the upper limit of the normal and imaging findings. The diagnosis is made based on the fact that two of these three criterias are positive. Severe AP is determined by commonly used APACHE II score 8 and Ranson score 3. Hypertriglyceridemia (HTG) is defined as a fasting serum value above 150 mg/dL. HTG is classified as mild (150-199 mg/dL), moderate (200-999 mg/ dL), severe (1,000-1,999 mg/dL) and very severe (>2,000 mg/ dL). When the triglyceride value is over 1,000 mg/ dL, it poses a risk for AP. While the risk for AP above 1,000 mg/dL is 5%, it is 10-20% when it is above 2000 mg/dL.1 Both the acute inflammatory response of the pancreatitis itself and the lipotoxicity caused by free fatty acids formed by the breakdown of triglycerides as a result of activation of the pancreatic lipase enzyme are effective in the severity of AP. HTG is the third most common cause of acute pancreatitis after gallstones and alcohol. AP develops due to HTG at a rate of 1-4%. HTG caused by disorders in lipoprotein metabolism can be due to primary (genetic) or secondary causes like alcohol use, diabetes

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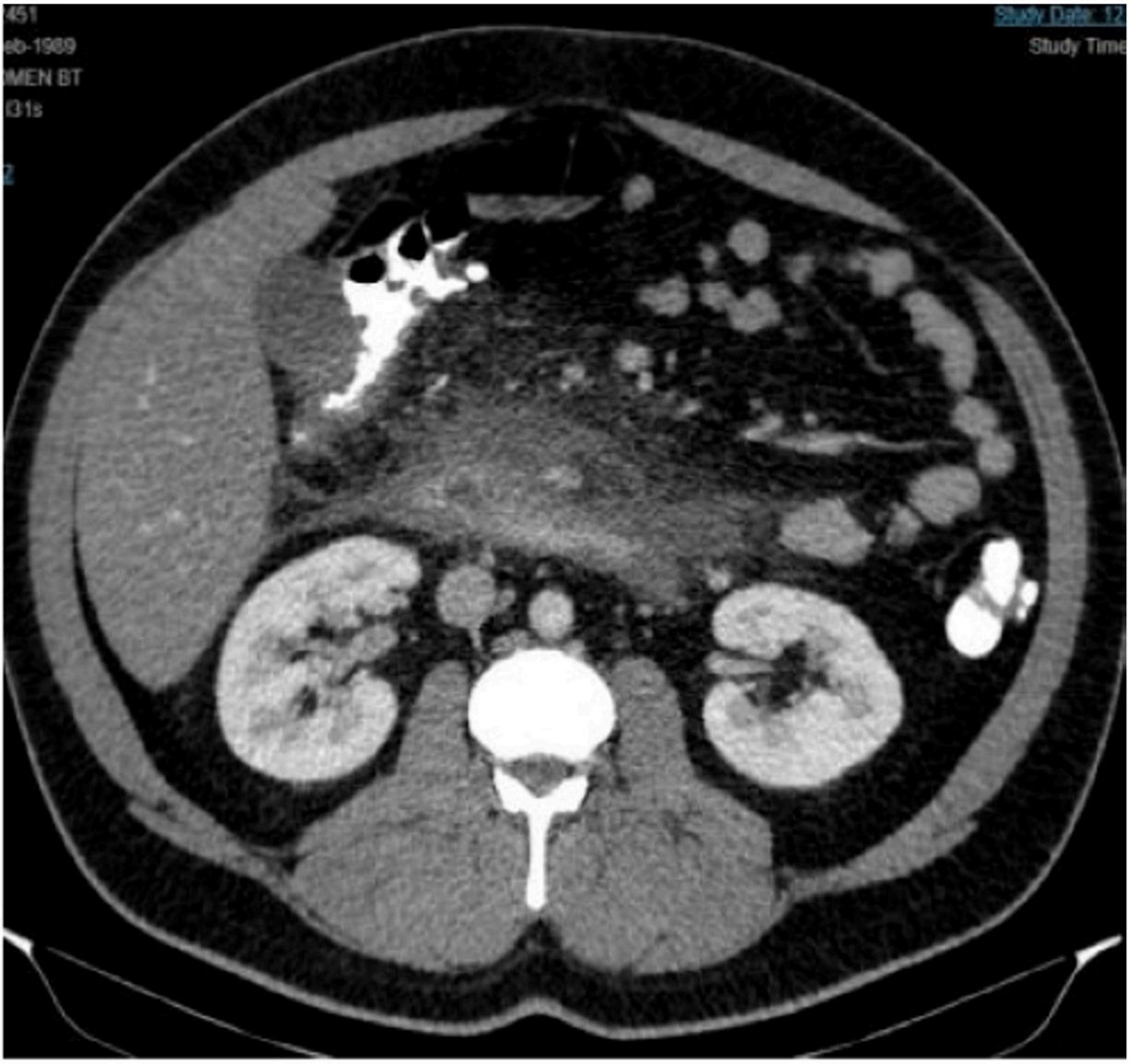

Figure 1. Patient's computed tomography indicating acute pancreatitis.

mellitus, pregnancy, obesity, hypothyroidism, nephrotic syndrome and some medications. Primary and secondary causes may coexist in patients. Metabolic syndrome is also an important factor contributing to the development of HTG.[1-3] In this report, a case of AP triggered by HTG with other metabolic syndrome components like type 2 diabetes mellitus and obesity was presented.

Case Report

A 31-year-old male patient admitted to the emergency service of our center due to acute onset epigastric pain radiating to the back. His medical history included type 2 diabetes mellitus which was diagnosed 1 year ago and a previous hospitalization due to AP seven months ago. He was using his insulin glargine, insulin Aspart and fenofibrate medications irregularly. He had no alcohol use but 14 package-years of smoking in his history. His height was 180 centimeters, weight was 122 kilograms and body mass index were 37 kg/m². His body temperature was 36.7 °C, blood pressure was 110/70 mmHg and pulse rate were 78 beats/minute on admission. In his physical examination, there was no pathologic feature except epigastric tenderness. He had a laboratory evaluation, in which total cholesterol was 340 mg/dL, triglyceride 3,117 mg/dL, LDL cholesterol 211 mg/dL, amylase 131 mg/dL, glucose 249 mg/dL, and HbA1c 10%, all being above the normal range. There was no acidosis in the arterial blood sample. His abdominal computed tomography revealed findings of AP (Figure 1). His Ranson criteria

was 1 point which was scored as mild pancreatitis. In his treatment 4-6 liters/day intravenous fluid mainly sodium chloride, 0.1 IU/kg/h insulin infusion and 5% dextrose infusion to avoid hypoglycemia were given. In the 3rd day of his admission, his oral nutrition and diabetes mellitus treatments were regulated. His subcutaneous insulin treatment was rearranged, and infusion was stopped. Rapid triglycerides decrease and clinical improvements were observed with insulin infusion without the need for plasmapheresis. In the 8th day of his admission, his triglyceride level was 642 mg/dL, total cholesterol was 227 mg/dL and serum glucose were 201 mg/dL in the laboratory tests.

Discussion

Fenofibrate and omega-3 were prescribed for HTG, a diet program was scheduled for obesity. Necessary lifestyle changes were advised. He was discharged from the hospital in the 8th day of his admission. The pathophysiology associated with pancreatitis, one of the life-threatening acute complications of HTG, is not fully understood. The primary treatment goal in these cases is to restore triglyceride levels within normal ranges. This situation can be achieved with fluid replacement, intravenous insulin infusion and/or plasmapheresis treatments. Plasmapheresis is a high cost and rarely used method when necessary with complications like infection and allergic reactions. In addition to these, analgesia, anticoagulation prophylaxis, lipid lowering agents (fibrates as the first choice) and supportive treatments according to the severity of pancreatitis (antibiotics, oxygen) are used. Medium chain fatty acids, omega-3-fatty acids, niacin, microsomal transport protein inhibitors and gene therapy can be used as adjuvants.[4] Strict diet restricted primarily from fat and simple sugars, exercise and providing weight control also contribute to HTG control.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Endogenous Obesity Associated with Cushing's Disease: A Case Report

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Abstract

Cushing's disease (CD) constitutes most common cases of adrenocorticotropic hormone (ACTH) dependent Cushing's syndrome (CS). CD more often occurs in women. Recent studies indicate increasing prevalence of CD amongst the obese people. Therefore, the possibility of underlying CD should be ruled out in obese people. Inferior petrosal sinus sampling (IPSS) is an important diagnostic method for diagnosing and localizing CD cases who can't be diagnosed by standard imaging methods. IPSS can increase the success of the surgeon's treatment with information about the location of the adenoma. Here, we presented a case of a 36-year-old female patient admitted to the hospital for bariatric surgery who was diagnosed having CD localized by IPSS.

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Keywords: *Cushing's disease, inferior petrosal venous sinus sampling, endogenous obesity*

Introduction

Cushing's syndrome (CS) is a rare disease characterized by hypercortisolemia. Endogenous CS is classified as adrenocorticotropic hormone (ACTH)-dependent or ACTH-independent.[1,2] ACTH-dependent CS accounts for 80-85% of the cases. Of these, 75-80% are due to ACTH production from a pituitary adenoma namely Cushing's disease (CD).[1] The prevalence of CD is of 40:1,000,000 people and more often occurs in women.[1,3] The most common sign of hypercortisolemia is central obesity.[4] Other findings in physical examination include moon face, buffalo hump, thinning of the skin, purple-colored cracks, atrophy and weakness in the proximal muscles, hirsutism, and hyperpigmentation.[1,4]

Case Report

A 36-year-old female patient without drug usage and comorbidity other than morbid obesity

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Table 1. IPSS results of our patient before and after CRH stimulation indicating a left sided Cushing's disease

	ACTH 5 minutes before CRH injection	ACTH before CRH injection	ACTH 2 minutes after CRH injection	ACTH 5 minutes after CRH injection	ACTH 10 minutes after CRH injection
Right inferior petrosal sinus sample	34 ng/L	34 ng/L	65 ng/L	81 ng/L	84 ng/L
Left inferior petrosal sinus sample	35 ng/L	40 ng/L	65 ng/L	94 ng/L	1035 ng/L
Peripheral venous blood sample	29 ng/L	29 ng/L	45 ng/L	68 ng/L	77 ng/L

Right and left inferior petrosal sinuses of the patient were accessed through the right common femoral vein. Blood sampling was performed from bilateral inferior petrosal sinuses and peripheral vein at the 5th minute before corticotropin releasing hormone (CRH) injection and at 0^{th}, 2^{nd}, 5^{th}, and 10^{th} minutes after intravenous injection of CRH.

was admitted for sleeve gastrectomy for the surgical treatment of obesity. The patient gained 35 kilograms in the last two years and had prominent central obesity. Although she had no menstrual irregularity, hirsutism, hyperpigmentation, plethora, moon face, purple striae and buffalo hump, CS was investigated for significant weight gain in 2-year time period. In the diagnosis of CS, 24-hour urinary free cortisol (UFC) level, serum or salivary cortisol measurements at night and 1 mg dexamethasone suppression test (DST) are used as screening tests as well as basal ACTH and cortisol measurements.3 In our case, the basal measurements suggest primarily ACTH-dependent CS with an ACTH level of 28 ng/dL and cortisol level of 11 mcg/dL. In our case serum cortisol level was not suppressed and measured as 11 mcg/dL (above 1.8 mcg/dL) at 1 mg DST. Serum cortisol level at night (23:00 h) was measured to be 13,2 mcg/dL with an ACTH level of 20 ng/L, indicating a disrupted diurnal rhythm. Her 24-hour UFC level was higher than normal. The next step in cases where CS is suspected includes performing a high-dose dexamethasone suppression test followed by localization of the lesion with imaging techniques. Since our patient who was not suppressed with 1 mg DST, a high dose 2 mg DST for 2 days was performed in which cortisol was not suppressed and measured to be 6.6 mcg/dL. After proving CD biochemically with increased cortisol levels, a sellar magnetic resonance imaging (MRI) was performed and a 2x2 mm isointense-slightly hypointense suspicious microadenoma was detected in the left side of the pituitary gland.

Discussion

On presentation, over 50% of the patients with CD have pituitary microadenoma with a diameter smaller than 5 mm.3 IPSS is the most important test, especially in patients who do not present with a definite pituitary lesion.2 Our patient had no suppression in both DST tests and a 2x2 mm suspicious microadenoma detected in pituitary MRI was decided to have an IPSS for making sure that the CS was caused by a pituitary adenoma. While the ratio of ACTH level in blood taken from inferior petrosal sinus to peripheral blood is almost always above 2 in CD, it is below 1.7 in ectopic ACTH syndromes.[4] Most of the time, sampling is done with a corticotropic releasing hormone (CRH) stimulation to strengthen the specificity of the test. In this test, central to peripheral ACTH ratio is expected to be above 3 for diagnosis to CD.[4] As a result of the IPSS the central to peripheral ACTH ratio was found to be high in our patient. The central to peripheral ACTH was 1.17 for the right side and 1.37 for the left side for our case. With CRH stimulation, this ratio was measured to be 1.09 for the right side and 13.44 for the left side at the 10th minute after CRH (Table 1). It was determined that 2x2 mm pituitary microadenoma caused CS in our patient whose ACTH ratio was above 3 on the left side after CRH stimulation. Upon these findings, the patient was referred to

neurosurgery department for pituitary surgery.

In conclusion, IPSS is an important test in distinguishing whether the cause is pituitary or ectopic in ACTH dependent CS patients. It can contribute to increasing the success of the treatment by increasing the rate of successful resection of pituitary adenomas by guiding the surgeon in terms of tumor localization.

Conflict of Interests

Authors declare that there are none.

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Case Report

Focal Segmental Glomerulosclerosis with Sjogren Syndrome: A Case Report

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Abstract

Cushing's disease (CD) constitutes most common cases of adrenocorticotropic hormone (ACTH) Sjögren's syndrome is a chronic lymphoproliferative disease. Sjögren's syndrome is rarely complicated by focal segmental glomerulosclerosis. Here, we presented a 39-year-old female patient with a history of Sjögren's syndrome for 5 years and diagnosed with focal segmental glomerulosclerosis.

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Keywords: *Sjogren syndrome, focal segmental glomerulosclerosis, nephrotic syndrome*

Introduction

Primary Sjögren's syndrome (pSS) is a chronic, slow-progressing, autoimmune and lymphoproliferative disease. The syndrome's main symptoms are xerostomia and keratoconjunctivitis sicca as a result of chronic inflammatory infiltration of the salivary and lacrimal glands. The exocrinopathy can be encountered alone (pSS or in association with other autoimmune disorders, the three most common ones being rheumatoid arthritis, systemic lupus erythematosus, and progressive systemic sclerosis (secondary SS).[1] Several systemic features have also been described; the presence of autoantibodies against the ubiquitously expressed ribonucleoprotein particles Ro (SS-related antigen A - SSA) and La (SSB) underline the systemic nature of SS. The original explanatory concept for the pathogenesis of pSS proposed a specific, self-perpetuating, immune-mediated loss of acinar and ductal cells as the principal cause of salivary gland hypofunction.[2] SS-associated renal disease usually consists of tubulointerstitial nephritis (75% of patients) and glomerulopathy, such as membranous proliferative glomerulonephritis.[3,4] Focal segmental glomerular sclerosis (FSGS) as a dominant pathological finding is found rarely in patients with pSS. Although rare, glomerulonephritis may accompany membranoproliferative glomerulonephritis,

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membranous nephropathy, and focal mesangioproliferative glomerulonephritis. In our case, FSGS, which is associated with pSS, was considered suitable for the presentation because of its rare occurrence.

Case Report

A 39-year-old woman presented with bilateral leg edema. Urinary examination revealed increased urinary protein levels, and blood tests revealed hypoalbuminemia; thus, she was diagnosed with nephrotic syndrome. She reported a history of pSS diagnosed 5 years earlier. Physical examination of the bilateral lower extremities revealed pitting edema. Urinary examination revealed urinary protein levels of 4700 mg/day, no hematuria. Blood tests revealed the following results: serum albumin 2.6 g/dL, blood urea nitrogen 20 mg/dL, creatinine 0.6 mg/dL, aspartate aminotransferase 22 U/L, alanine aminotransferase 13 U/L, and LDL cholesterol: 215 mg/dL. Anti-SS-A and anti-SS-B antibody test results were positive. Tests for antinuclear antibodies showed a positive result. A kidney biopsy was performed, and the specimen included 23 glomeruli, with 3 showing segmental glomerulosclerosis, detected. There were six glomeruli in the immunofluorescence specimen. Immunofluorescence staining showed no glomerular deposition of immunoglobulin (IgG, IgA, and IgM), C3, or fibrinogen. Based on these findings, she was diagnosed with FSGS. The current patients did not show any electrolyte disturbances in the blood. The blood pH level of the patient did not show acidemia. Hepatitis markers were negative. Histopathological findings in kidney biopsy compatible with FSGS. Steroid (oral prednisolone at a dose of 15 mg/day) and cyclosporine (2x100 mg/d) therapies were initiated. Four weeks after initiation of steroid and cyclosporine therapy, the creatinine levels increased; hence, cyclosporin was discontinued. After 2 months of treatment initiation, urinary protein levels decreased to 303 mg/day and creatinine 0.6 mg/dL, and her leg edema disappeared. Steroid treatment was continued with 7.5 mg/day oral prednisolone. Angiotensin-converting enzyme inhibitor (ramipril) was added to her regimen to decrease urinary protein levels.

Discussion

In the current case, the patient developed nephrotic syndrome secondary to FSGS. Typical renal complications associated with pSS are tubulointerstitial nephritis and renal tubular acidosis. Glomerular diseases manifested by nephrotic syndrome are infrequent in these patients. Although secondary FSGS could be attributed to several etiopathogenetic factors such as familial, viral, drug-induced, structural, and functional responses (nephron depletion and hemodynamic changes)[5], this patient showed no obvious findings that could have resulted in secondary FSGS. Therefore, we could not conclusively establish an association between pSS and FSGS. Goules et al.[6] and Maripuri et al.[7] reported only 5 cases of severe proteinuria or nephrotic interval out of 60 patients who underwent kidney biopsy in his studies. Unfortunately, Kurihara et al.[8] reported that there had been no investigations or case reports that concern the prognosis and clinical response of the cases with FSGS and pSS. Therefore, to discuss the appropriate treatment or prognosis, further accumulation of similar cases with current patients is necessary. Because there are few cases of glomerular diseases coexisting with pSS, it is difficult to describe the best treatment options for each glomerular disease.[8] Treatment for renal involvement primarily includes the administration of corticosteroid, and a few patients receive other immunosuppressants such as cyclosporine, cyclophosphamide, mycophenolate mofetil, and rituximab. Jasiek et al.[9] and Ren et al.[10] reported that 5 cases of 95 patients and 4 cases of 130 patients developed end-stage renal disease. In conclusion, limited data are available regarding renal involvement and prognosis in patients with pSS.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress,

6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

The Pigtail Stenting Treatment for the Biliary Leakage after the Liver Hydatid Cyst Operation: A Case Report

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Abstract

Echinococcus granulosus commonly involve the liver and are mostly seen in Turkey. One of the early complications following the surgery for liver hydatid cyst is biliary leakage. Endoscopic sphincterotomy and biliary stenting are usually successful in treating biliary leakage. In this case, I'm presenting endoscopic treatment for the biliary leakage after the liver hydatid cyst operation.

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DOI: 10.46310/tjim.876345

Keywords: *ERCP, hydatid cyst, biliary leakage, biliary stenting*

Introduction

The incidental hosts of Echinococcus granulosus are humans in which the definitive hosts are canines.[1] E. granulosus commonly involve the liver and are mostly seen in Turkey.[2,3] One of the early complications following the liver hydatid cyst surgery is biliary leakage, which's incidence is %4.5-26.[4,5] Endoscopic sphincterotomy and biliary stenting are usually successful in treating biliary leakage. Here, I presented endoscopic treatment for the biliary leakage after the liver hydatid cyst operation.

Case Report

The patient was a 43 years old man. He had upper abdominal pain for 4-5 months in September 2020. He had gone to the hospital for this complaint, and he was diagnosed with liver cyst hydatid. He had no comorbid disease or medications. In his abdominal magnetic resonance imaging (MRI), the cysts were involved the right lob anterior and left lob medial segments, which were nearly 175x12x148 mm complex and multiple (Figure 1). He had surgery for his liver hydatid cyst on 4 November 2020. The partial cystectomy and omentopexy were done and drained the cyst in the left lobe externally (Figure 2). The patient has consulted me because of the bile ooze from

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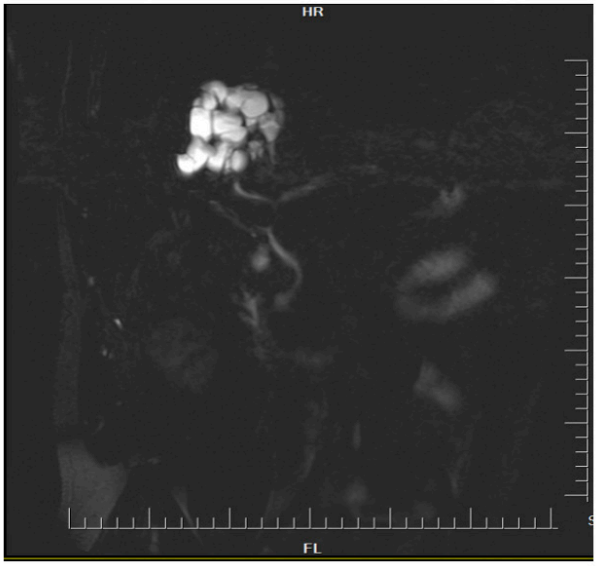

Figure 1. MRI of the patient before surgery

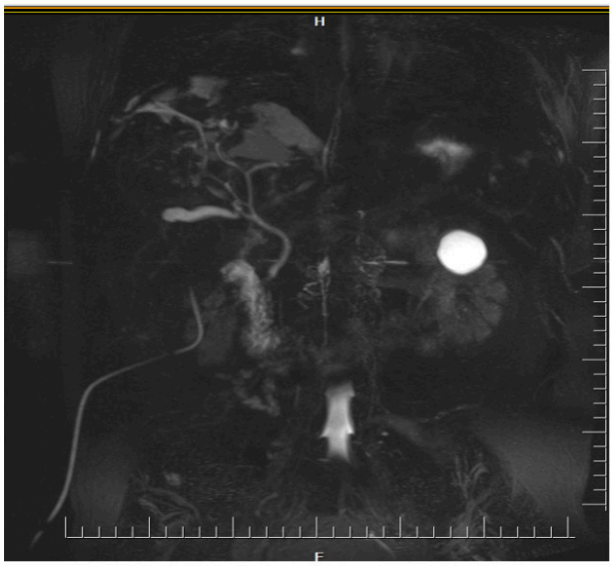

Figure 2. MRI of the patient after surgery

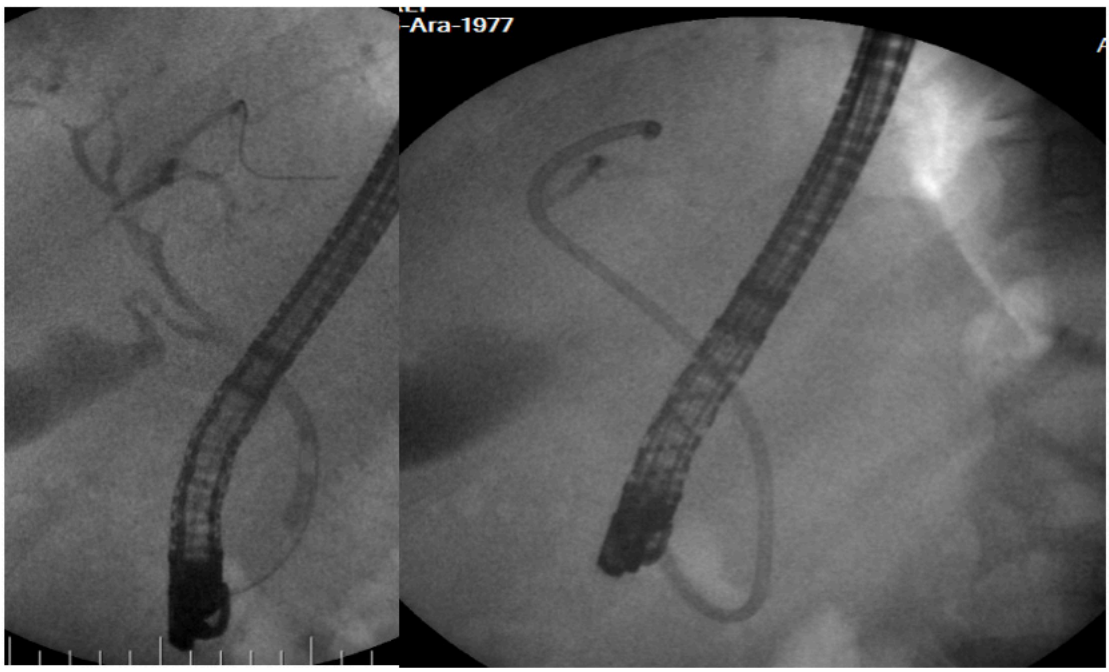

Figure 3. Biliary stent in ERCP imaging

the surgical drain on 24 November 2020. There was 500 cc bile from the drain. The abnormal values were direct bilirubin, alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT) in his laboratory finding. Direct bilirubin was 0.74 mg/dL (0-0.5 mg/dL). ALP was 173 U/L (NR: 40-150). GGT was 126 IU/L (NR: 12-64). The endoscopic retrograde cholangiopancreatography (ERCP) showed BL into the left lobe. Choledocus was normal. The ES was carried out. The pig tale stent 10 Fr 8 cm was placed to the cyst distal (Figure 3). He was treated with albendazole 400 mg twice a day for a month. After one week in his follow-up, there was 20-30 cc fluid without bile in his drain. The control of the ALP, GGT, and total bilirubin values was normal.

Discussion

Echinococcus granulosus commonly involves

the liver.[2,3] most of the disease symptoms are fever, pain, jaundice, and abdominal mass. As in the case, the patient had abdominal pain. Medical therapy is effective only with surgery. The principal mode of treatment is surgery. The biliary leakage, which develops because of communication between the residual cyst cavity and the biliary tree, could be manifested in the postoperation.[6] ERCP is a therapeutic method to treat biliary leakage. It is safe and effective. Endoscopic sphincterotomy and biliary stenting are usually successful in treating biliary leakage.[7] Endoscopic sphincterotomy and biliary stenting decrease the pressure in the biliary tract. The time for closure of the fistula is reduced.[8,9] The biliary stenting is more effective, especially in patients with a narrow choledochus and high fistula output.

Most of the postoperative hydatid cyst complication is biliary leakage. Biliary leakage occurs typically within two of four weeks of surgery. Endoscopic technics such as endoscopic sphincterotomy and biliary stenting are commonly effective in treating the biliary fistula. As in the case, the time from endoscopic sphincterotomy and biliary stenting to the biliary fistula's closure is reported to be 3-21 days.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

The Two Huge Pulmonary Hydatid Cyst in the One Lobe of Lung: A Case Report

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Abstract

The hydatid cysts disease is a parasitic cystic infection of the liver, lungs, and other organs caused by Echinococcus granulosus. It is mostly seen in the sheep and cattle farms, which have poor health conditions. It is endemic in the eastern and southwestern regions of Turkey. The big cysts seen in the lungs are called huge pulmonary cysts, and it is a particular clinical situation. The symptoms of the disease are cough, chest pain, dyspnoea, hemoptysis, or allergic reactions. The hemoptysis could be seen when these cysts rupture. The use of anthelmintic drugs to treat the pulmonary hydatid cysts could cause cyst rupture. The surgery must be the first choice of treatment. I reported two large pulmonary hydatid cysts in the left upper lobe of the lung that I treated with thoracotomy.

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Keywords: *The hydatid cysts, thoracotomy, cystotomy, quilting*

Introduction

The hydatid disease (Echinococcosus) is a common parasitic infection in agricultural communities, especially in the middle east and Mediterranean countries and the sheep and cattle farms.[1] It is endemic in the eastern and southwestern regions of Turkey.[1,2] Echinococcus granulosus mostly involves the liver and lungs. It is mainly seen in the liver (60-70%) and the lungs (20-30%). The extrapulmonary involvement in the thoracic cavity is infrequent.[3] The recurrence rate of hydatid cyst after surgery is 1.4 % in the lung and 11.3% in the liver. The cyst's growth rate varies depending on the host's immunity and the elasticity of the organ in which it is located.[4,5] While tissue resistance and hepatobiliary capsules in the liver limit the cyst's growth, the lung tissue's low tissue resistance is favorable for the rapid growth of hydatid cysts.[5] The immune response against the parasite and the symptoms caused by the patient's mass effect prevent the cyst's limitless growth. The disease is usually diagnosed in a patient living in an endemic area by radiological

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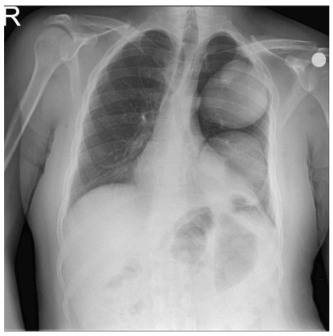

Figure 1. Preoperative posterior-anterior chest radiography

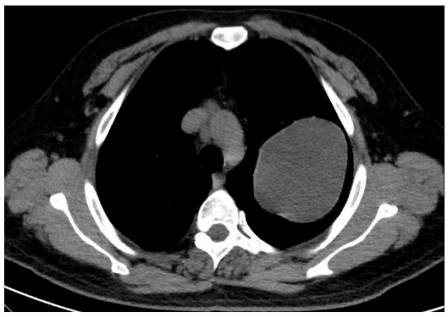

Figure 2. The thoracic CT image of the cyst in the left superior lobe

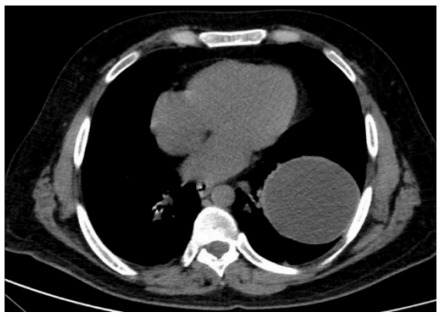

Figure 3. The thoracic CT image of the cyst in the left inferior lobe

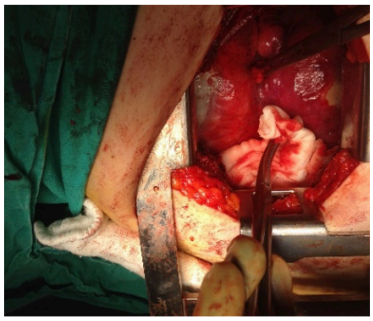

Figure 4. Photograph of cystic lesions in the left upper lobe after thoracotomy (superior cystic lesion on the left, inferior cystic lesion on the right)

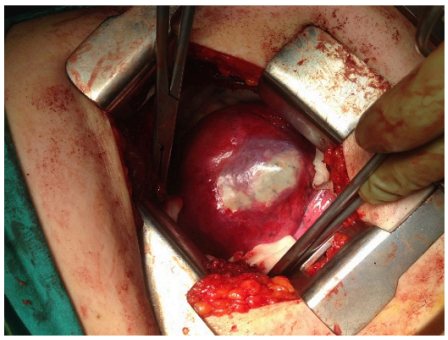

Figure 5. Photograph of the inferior cystic lesion after thoracotomy

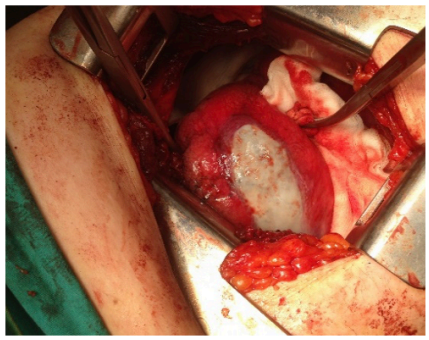

Figure 6. Photograph of superior cystic lesion after thoracotomy

examinations or radiological examinations taken for other reasons.[6,7] Cough, chest pain, dyspnea, hemoptysis, or allergic reactions may occur in symptomatic patients. Cysts rarely reach six centimeters in diameter.[8] Large cysts in the lungs are called giant pulmonary cysts. In our case, two cysts of 8 cm and 10 cm in diameter were accepted as giant pulmonary hydatid cysts. In this case, I discussed two large pulmonary hydatid cysts in the left upper lobe of the lung treated by thoracotomy.

Case Report

A 38-year-old mentally disabled male patient was admitted to the hospital with a hacking cough for three months. He was diagnosed with a left lung hydatid cyst by posterior-anterior chest radiography (PALG) (Figure 1). He had no comorbid disease or medication. In thoracic computed tomography imaging (CTI), there was a cyst involving approximately 10x7 cm of the left lobe of the lung, and another cyst of 8x8 cm inferior to this cyst (Figures 2 and 3). On his exploration, two lesions compatible with hydatid cysts in the left upper lobe of the lung, and total atelectasis in the lower lobe were observed (Figures 4-6). I entered the inferior cyst with the needle connected to the aspiration apparatus, and 500 mL of rock water was aspirated (Figure 7). The pericystic layer was cut with electrocautery. After the cystotomy, the germinative membrane was removed (Figure 8). Later, the same procedure was applied to the upper lesion. 600 mL of rock water was aspirated. Bronchial fistulas in both cystic cavities were closed with 3/0 prolene and quilted with 3/0 vicryl (Figures 9 and 10). When the left lung was ventilated, the upper lobe and atelectasis lower lobe were ventilated. A thoracic drain was inserted, and the patient was closed, and the lungs were expanded in the control PALG radiography (Figure 11). He was discharged on the 4th day. Pathology examinations confirmed the diagnosis

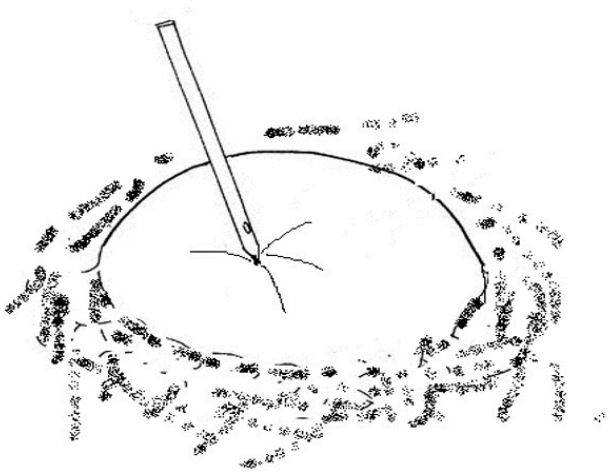

Figure 7. Intra-cyst rock water aspiration

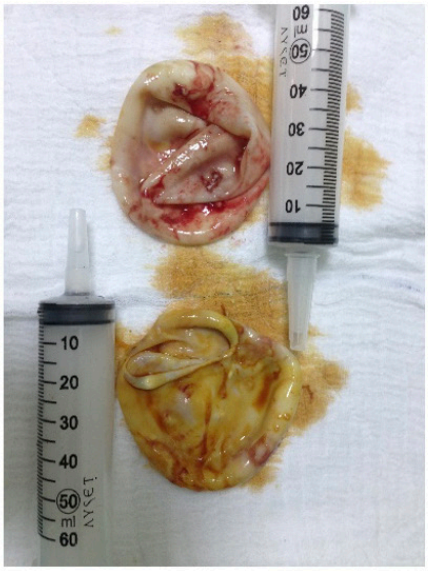

Figure 8. Germinative membrane

of a hydatid cyst. He received albendazole treatment to preventing relapse. There was no recurrence in his 6-month follow-up.

Discussion

PALG and thoracic CT are usually sufficient for the diagnosis of pulmonary hydatid cysts.[9-11] Pathological confirmation is required for the definitive diagnosis. Patients with hydatid cysts are usually asymptomatic. Symptomatic patients present with cough, chest pain, shortness of breath, hemoptysis, or allergic reactions. Cystic fluid, germinative membrane expectoration, and infection may be observed in ruptured cysts. The parasite's growth rate depends on the host's immune response and the tissue's properties in which the cyst is located. Its' large size can change the clinic, treatment, and postoperative course of pulmonary hydatid disease. In this disease, it is rare for the cyst to reach 6 cm in diameter. In our study, two cysts in one lobe with 10 cm and 8 cm diameter were accepted as giant hydatid lung cysts. In the treatment of pulmonary hydatid cyst, parenchyma sparing operations are preferred more frequently.[9,12,13] Lung resection should be avoided because the compressed lung parenchyma is generally healthy and expands in the postoperative period. Cystotomy and quilting procedure was applied to our patient. Treatment

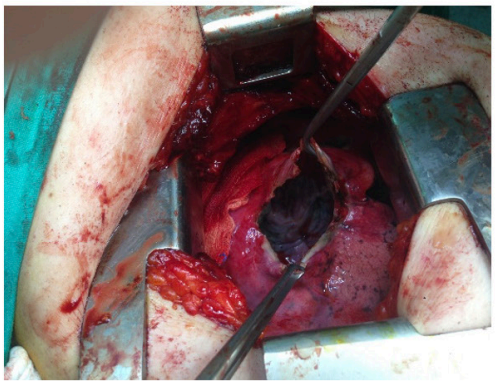

Figure 9. Photograph of the inferior cystic lesion after cystotomy and cystic membrane removal

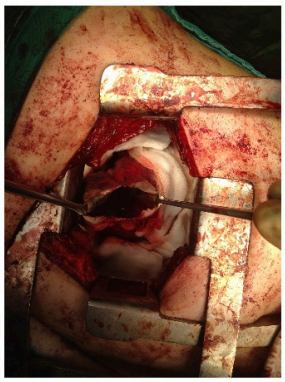

Figure 10. Photograph of superior cystic lesion after cystotomy and cystic membrane removal

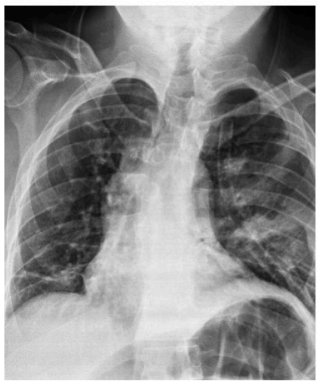

Figure 11. Post-operative PALG

with anthelmintics can cause cyst rupture. We recommend this treatment only in patients who cannot tolerate surgery and to prevent a recurrence.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

A Peripartum Cardiomyopathy Case Treated with Bromocriptine

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Abstract

The hydatid cysts disease is a parasitic cystic infection of the liver, lungs, and other organs caused by Echinococcus granulosus. It is mostly seen in the sheep and cattle farms, which have poor health conditions. It is endemic in the eastern and southwestern regions of Turkey. The big cysts seen in the lungs are called huge pulmonary cysts, and it is a particular clinical situation. The symptoms of the disease are cough, chest pain, dyspnoea, hemoptysis, or allergic reactions. The hemoptysis could be seen when these cysts rupture. The use of anthelmintic drugs to treat the pulmonary hydatid cysts could cause cyst rupture. The surgery must be the first choice of treatment. I reported two large pulmonary hydatid cysts in the left upper lobe of the lung that I treated with thoracotomy.

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Keywords: *Peripartum, heart, failure, bromocriptine, cardiomyopathy*

Introduction

Peripartum cardiomyopathy (PPCM) is a rare disease that is difficult to diagnose and treat. It can be mortal. Its etiopathogenesis is not fully understood. The symptoms are similar to those in the physiological course of pregnancy. Herein, we report the case of a 32- year-old woman diagnosed with PPCM and treated with bromocriptine.

Case Report

A 32-year-old woman presented with dyspnea, orthopnea and haemoptysis 4 days after first delivery. Although hemodynamically stable, she had signs of pulmonary congestion. She had no past medical history. On auscultation, grade 3/6 systolic murmur radiating from apex towards axilla was heard. Electrocardiography revealed sinus tachicardia. Chest X-ray showed cardiomegaly. Arterial blood gas analysis was hypoxic and hypocarbic (pH: 7.54, pCO2: 31.3 mmHg, pO2: 77.8 mmHg). Serum BNP level was measured 766.6 ng/L. Transthoracic echocardiography

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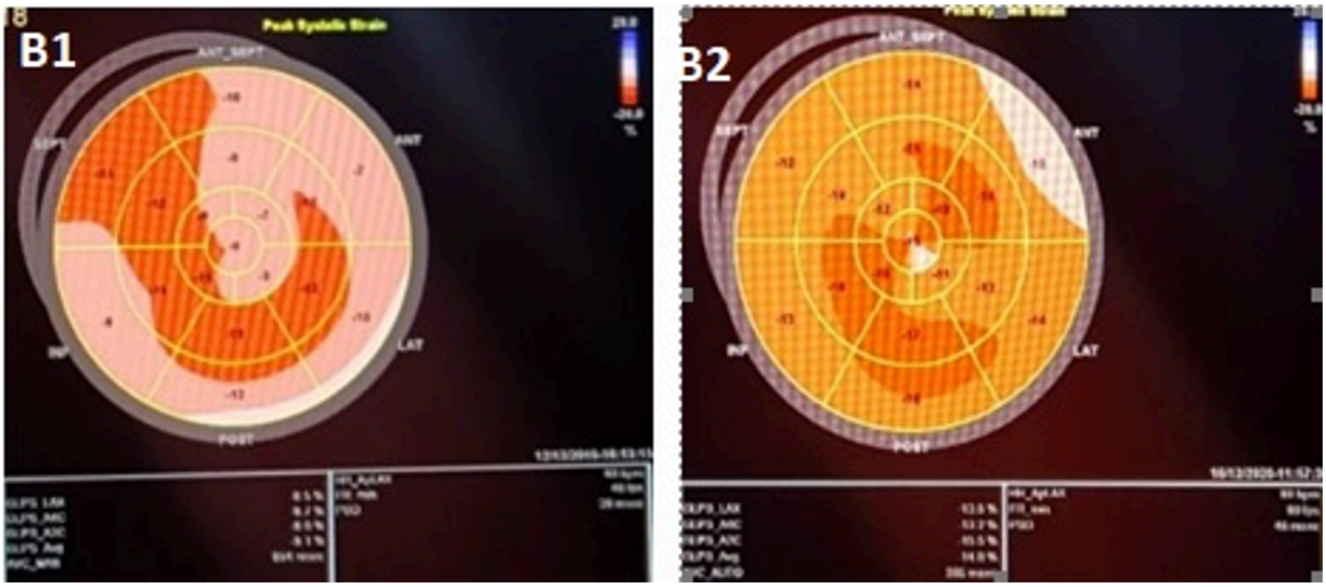

Figure 1. 2D Speckle strain echocardiography finding B1: at admission, B2: 6 months of follow-up

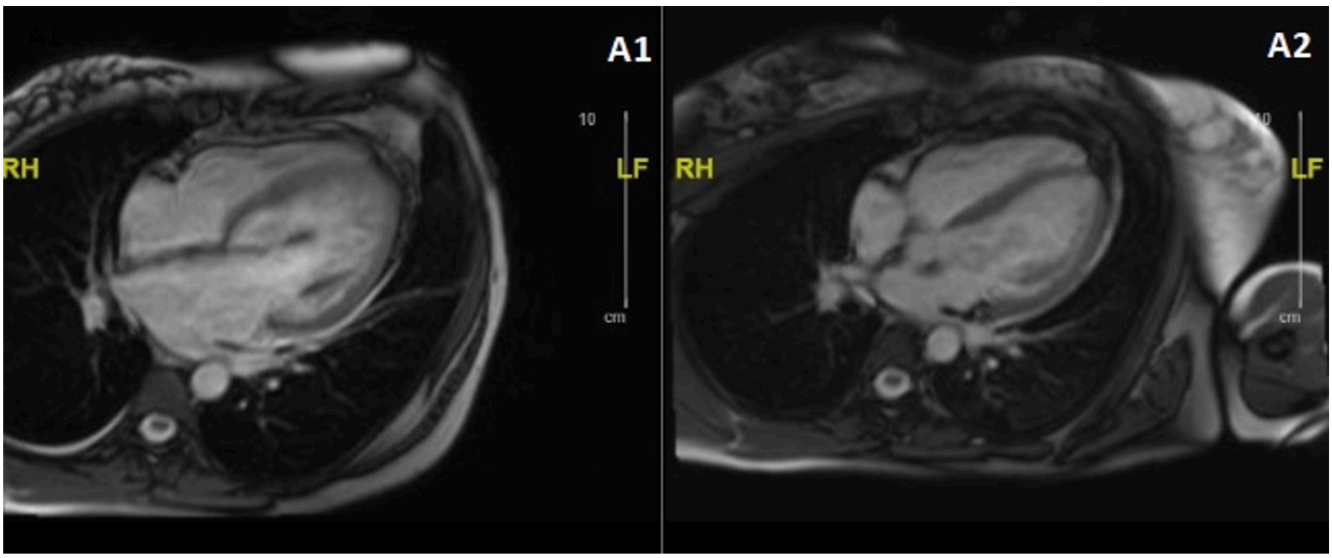

Figure 2. Cardiac magnetic resonance imaging findings show normalization in left heart size A1: at admission, A2: 6 months of follow-up

(TTE) indicated a dilated left atrium and a dilated left ventricle without hypertrophy, severe mitral regurgitation and ventricular dysfunction (left ventricular ejection fraction [LVEF], 30%) and general hypokinesia. The global longitudinal strain was –9.1% (Figure 1). Contrast-enhanced thorax computed tomography showed signs of effusion up to 3 cm thickness in the right pleural space, cardiomegaly, and pulmonary edema. There was no filling defect consistent with embolism in pulmonary arteries. Cardiac magnetic resonance imaging (MRI) was performed and revealed no pathological staining in the first pass images after the administration of contrast material. Mid-myocardial staining in patchy style was detected in late phase images taken at 10 minutes (Figure 2). Inhospital telemetry found no extrasystoles or arrhythmias. After treatment with diuretics, metoprolol, ramipril, ivabradine, spironolactone, enoxaparin and bromocriptine (planned as: 2.5 mg po twice a day for 15 days and then 2.5 mg po once a day for 15 days), she progressively improved and was discharged after 10 days. The clinical course was satisfactory; transthoracic echocardiography at first month revealed an improved LVEF (50%) and at 6 months, TTE revealed no dilatation in

cardiac chambers, also global longitudinal strain of –14% (Figure 1). Cardiac MRI indicated normal biventricular function and size, without delayed contrast enhancement (Figure 2) and serum BNP level was measured 15.6 ng/L.

Discussion

PPCM is an "idiopathic cardiomyopathy" and a rare form of congestive heart failure of unknown etiology. Demakis criteria and echocardiography findings are used in the diagnosis. The current diagnostic criteria for peripartum cardiomyopathy include: cardiac failure in a previously healthy woman in the last month of pregnancy or within 5 months of delivery, absence of etiology for heart failure. absence of a cardiac disease prior to last month of pregnancy, echocardiographic evidence of diminished left ventricular ejection fraction.[1]

Clinical course of PPCM may rapidly deteriorate, need for intensive care may develop, and it may even be mortal. In patients who do not progress mortally, heart failure may regress or become permanent. PPCM is not an indication for emergency delivery.

Increased pro-inflammatory cytokines and oxidative stress are thought to play role. Factors causing peripartum cardiomyopathy include cardiotrophic viruses, autoimmune diseases, toxins that cause immune system dysfunction, abnormal serum relaxin levels, selenium deficiency, presence of antibodies that respond abnormally to heart tissues and myocarditis.[2-6]

Some studies report the increase of 16 kDa prolactin hormone, which has antiangiogenic and proapoptotic properties.[3,7] It has been reported in the literature that bromocriptine can also be used with standard heart failure treatment in the treatment of these patients.[8] Although there is no consensus regarding the risk of recurrence of PPCM in subsequent pregnancies, Elkayam et al.[9] reported that 21% of the patients who regain normal ventricular function presented with heart failure in their next pregnancy.

PPCM should be considered in the differential diagnosis of patients presenting with symptoms of heart failure during the peripartum period and these patients may benefit from concomitant bromocriptine use with standard heart failure therapy.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

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Case Report

Acute Tubular Necrosis Associated with Autoimmune Hemolytic Anemia due to Acute Gastroenteritis

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Abstract

Autoimmune hemolytic anemia (AIHA) is a rare disease and defined as primary (idiopathic) or secondary depending on the presence or absence of accompanying disease. Here, we reported a case of AIHA due to acute gastroenteritis and acute tubular insufficiency.

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Keywords: *Acute Tubular Necrosis, Autoimmune Hemolytic Anemia, Acute Gastroenteritis*

Introduction

Autoimmune hemolytic anemia (AIHA) is a rare disease with a rate of 1-3 in 100,000 in adults.[1] AIHA encompasses a group of heterogeneous conditions mainly characterized by red blood cell (RBC) lysis due to autoantibodies against surface erythrocyte antigens. Based on the thermal characteristics of the autoantibody, AIHAs can be classified into warm forms, generally caused by IgG antibodies reacting at warm temperatures and able to fix complement in some cases; cold agglutinin disease (CAD), due to IgM antibodies that agglutinate RBCs at low temperatures and lyse them via the complement cascade activation, and mixed forms (coexistence of warm and cold autoantibodies).[2,3] AIHA are defined as primary (idiopathic) or secondary depending on the presence or absence of accompanying disease. Secondary causes include drugs, immunodeficiencies, infections, other autoimmune diseases, or malignancies.[4,5] Coexistence of autoimmune hemolytic anemia and acute renal failure has been observed in studies. Generally, this association has developed due to various drugs. In our case, the association of AIHA due to acute gastroenteritis and acute tubular insufficiency was considered suitable for the presentation because of its rare occurrence.

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Case Report

A 42-year-old female patient without chronic disease was applied to the emergency service with complaints of diarrhea, nausea-vomiting, fever, chills, and jaundice for two days. At admission, fever: 37.1 °C, blood pressure: 104/64 mmHg, pulse: 99 beats/minute, conjunctivae were pale, sclera, and skin were icteric. Leukocyte: 31,000/mm3, hemoglobin: 11 g/dL, platelet: 176,000/mm3, urea: 104 mg/dL, creatinine: 3.3 mg/dL, alanine aminotransferase: 48 IU/L, aspartate aminotransferase: 100IU/L, lactate dehydrogenase: 1,340 U/L, total bilirubin: 16 mg/dL, direct bilirubin: 3.2 mg/dL, reticulocyte: 3%, C-reactive protein: 197 mg/L, and procalcitonin: >100 µg/L. She was hospitalized with pre-diagnosis of acute kidney injury secondary to hemolysis. Direct Coombs test was positive. Methylprednisolone 40 mg/day was started considering AIHA. ANA was negative, and complements were normal. No pathology was found in the thorax, neck, and abdominal tomographies performed for malignancy screening. There was no history of drug use or broad beans consumption. Klebsiella and E.coli grew in the stool culture of the patient sent in terms of infection focus. Brucella agglutination test, rotavirus, adenovirus test, and hepatitis markers were negative. The patient's pathology result, who underwent renal biopsy, was consistent with acute tubular necrosis due to an anuric course and progressive creatinine values. She was on hemodialysis at regular intervals. On the 14th day, ceftriaxone was discontinued. LDH, bilirubin, and creatinine returned to the normal range. The patient's direct Coombs test was negative. The patient did not need hemodialysis treatment later. She was discharged with 32 mg/g methylprednisolone treatment.

Discussion

Determining the etiology of AIHA is important in terms of treatment. The coexistence of AIHA and acute tubular necrosis is common in the literature. However, infection-related AIHA cases are limited in the literature. Also, infections in AIHA are a known player in the pathogenesis of the autoimmune process. On the other hand, infections can also occur as a consequence of the disease and its treatments. There is increasing awareness of AIHA infections, as they can impact outcomes, including morbidity and fatality.[6] Infectious agents can trigger AIHA through various mechanisms, including modification of erythrocyte membrane antigens, polyclonal B cell activation, an innocent bystander, and molecular mimicry.[7,8] AIHA cases secondary to legionella and brucella infections are presented.[9,10] AIHA may complicate about 3% of infectious mononucleosis, with a typical onset within 1–2 weeks.[11] Regarding bacterial infections, Mycoplasma pneumonia may be accompanied by severe AIHA, mainly cold but even warm forms.[12] Most recently, about 20 cases of AIHAs (both cold and warm forms) secondary to COVID-19 infection have been reported, with only one fatality.[13] As a result, this case shows us that, although rare, acute tubular necrosis can be seen together with AIHA associated with acute gastroenteritis.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

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Case Report

Large Ganglioneuroma Case Mimicking as An Adrenal Adenoma

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Abstract

Ganglioneuroma (GN) is a rarely seen benign tumor originating from neural crest cells and consisting of ganglion and Schwann cells. Adrenal GNs occur most frequently in the fourth and fifth decades of life. They have an equal frequency in male and female patients and are usually found incidentally during imaging. It is not related to hormonal activity and is clinically asymptomatic. We aimed to present a 49-year-old female patient whose magnetic resonance image performed for abdominal pain was found a biochemically normal mass in the right adrenal gland and then was pathologically diagnosed as GN after right adrenalectomy.

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Keywords: *Adrenal ganglioneuroma, magnetic resonance imaging, adrenal adenoma, incidentaloma*

Introduction

Ganglioneuroma (GN) is a rare tumor with benign behavior mainly originating from neural crest cells.[1] Histologically, it consists of Schwann and ganglion cells.[2] The lesion is mostly located along the length of the sympathetic chain.[3] While they are usually located in the posterior mediastinum or retroperitoneal space, they rarely occur from adrenal medulla.[3] Retroperitoneum and posterior mediastinum GNs are usually diagnosed in children and young adults, while adrenal GNs occur most frequently in the fourth and fifth decades of life. In the case series, it has been determined that GNs are seen equally in males and females.[4,5] Adrenal GNs can be seen as single or together with other neuroendocrine tumors.[4] Since these lesions do not tend to be hormonally active, they are discovered incidentally during imaging techniques. The imaging features of adrenal GN are variable, and some may resemble adrenal tumors such as adrenocortical carcinoma and malignant pheochromocytoma.[4,5] For this reason, it is generally difficult to diagnose adrenal GN preoperatively precisely. In the current case, we aimed to present a 49-year-

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Table 1. Laboratory parameters of the patient at the admission

Variables	Results	Reference range
Glucose (mg/dL)	91	74-106
Creatine (mg/dL)	0.63	0.66-1.09
ALT (U/L)	9	3-35
Sodium (mEq/L)	139	136-146
Potassium (mEq/L)	4,11	3.5-5.1
Calcium (mg/dL)	10,51	8.8-10.6
LDH (U/L)	185	25-247
ACTH (pg/mL)	29.8	0-46
Cortisol (μg/dL)	15.99	6.7-22.6
Aldosteron (ng/dL)	13.73	7-30
Renin (ng/mL/saat)	4.06	0,98-4,18
DHEA-S (ug/dL)	180.3	56.2-282.9
Urine normetanephrine (ug/24 saat)	254.17	100-500
Urine netanephrine (ug/24 saat)	93.35	50-250
Urine ndrenaline (ug/d)	6.26	0-20
Urine noradrenaline (ug/d)	37.42	15-80
Urine nopamine (ug/d)	195.58	65-400
ESR (mm/h)	14	0-20

ALT: alanine aminotransferase, LDH: lactate dehydrogenase, ACTH: adrenocorticotropic hormone, DHEA-S: dehydroepiandrosterone sulfate, ESR: erythrocyte sedimentation rate.

old female patient pathologically diagnosed as GN after adrenalectomy, which was found incidentally mass in the right adrenal gland.

Case Report

A 49-year-old female patient was admitted to our center when a mass of 81x48x92 mm in the right adrenal gland was revealed in abdominal magnetic resonance imaging (MRI) performed for abdominal pain (Figure 1). She had hypertension as comorbidity, which was under control with 10 mg amlodipine. A physical examination was normal. The results of laboratory and hormonal analysis were all found to be within the normal ranges (Table 1). Endocrine investigation for excessive hormone secretion, including urine catecholamine and hormonal parameters, were normal. Cortisol was found as 1.1 ug/dL in the 1 mg dexamethasone suppression test. MRI revealed a mass of 81x48x92 mm, which was hypointense on axial T1-weighted images and heterogeneously hyperintense on T2-weighted images, relative to the paravertebral muscles (Figure 1). Right adrenalectomy was performed on the patient due to the size of the lesion after excluding pheochromocytoma. The surgical specimen was evaluated, and immunohistochemical examination showed positive staining of S-100, neuron-specific enolase (NSE), and synaptophysin. The tumor was diagnosed GN after right adrenalectomy.

Discussion

Although malignant cases have been reported, ganglioneuroma is a rare, generally benign tumor arising from primordial neural crest cells.[1,6] Although most retroperitoneum and posterior mediastinum GN cases are under the age of 20, adrenal GNs are diagnosed in the fourth and fifth decades.[4,5,7] GN have an equal frequency in male and female patients.[4] They are usually asymptomatic and found incidentally during imaging. Symptoms may develop due to the pressure from mass in large lesions. In a case series comprising 17 adrenal GN patients, eleven of them were diagnosed during controls incidentally, four of them were diagnosed after imaging studies for abdominal pain, and two of them were diagnosed after imaging studies for low back pain. None of these patients had hormonal secretion.[5] Similarly to previous reports, in the present case, a mass was

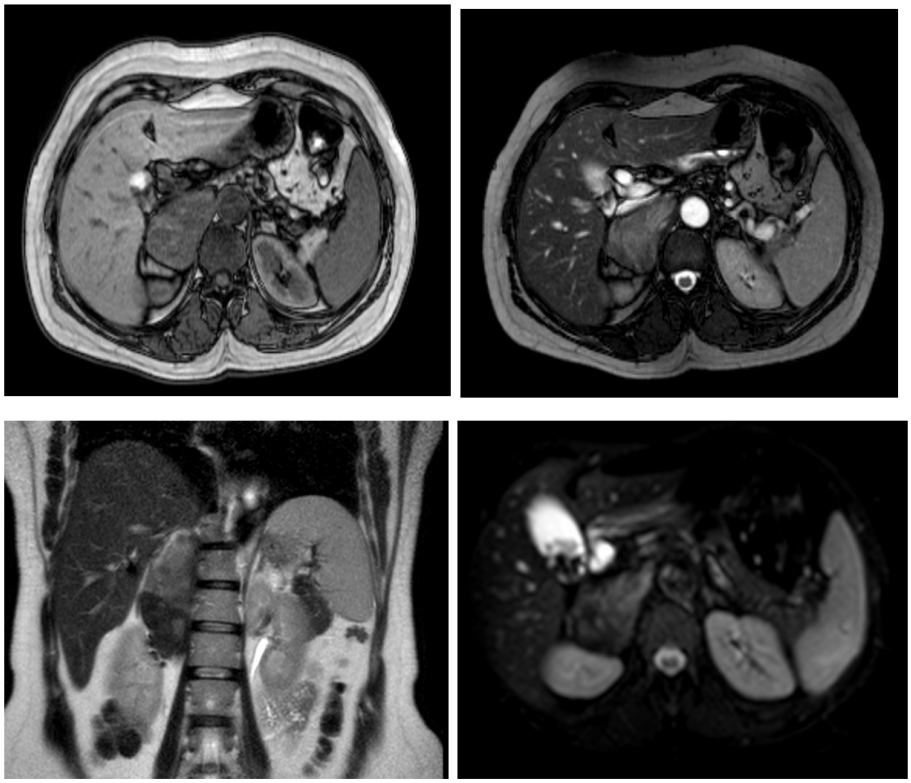

Figure 1. Mass lesion is in the right adrenal region on MR imaging

detected in the right adrenal gland on abdominal MRI performed for abdominal pain at 49-year-old female patient.

Most GN do not secrete catecholamines or steroid hormones, but it has been reported that up to 30% of patients may have elevated urinary catecholamine levels. Rarely ganglion cells can secrete vasoactive intestinal peptide (VIP) or produce steroid hormones. Diarrhea, hypertension, or hypokalemia may be observed due to VIP secretion.[3,6,8] Computed tomography (CT) findings are usually compatible with a well-defined, encapsulated, solid lesion. These tumors can been appeared punctuate calcifications, and nonenhanced attenuation of less than 40 Hounsfield Units on CT imaging. Punctate calcifications are observed at a frequency rate from 20% to 69% and are considered highly indicative of GNs. On MRI, T1-weighted images tend to have homogeneously low or intermediate signal, whereas T2-weighted images have heterogeneously intermediate or high signal.[4,5] Unfortunately, radiology findings are not pathognomonic of adrenal GNs, and preoperative misdiagnosis frequency of adrenal GNs based on MRI and CT findings has been observed to be 64.7%.[5] In the current case, a large mass in the right adrenal gland was located on MRI, which has not clinically significant hormone secretion, and the imaging findings could not eliminate the suspicion of malignancy.

The treatment of adrenal incidentalomas varies according to the functional status of the lesion and whether it is malignant. Regardless of size, functional adrenal lesions with clinically significant hormone excess should be surgically removed. Non-functional lesions <4 cm can be followed up, and surgery should be planned in case of progression (surgical resection if the lesion enlarges by more than 20% or at least a 5 mm increase in maximum diameter).[9] A clear approach has not been determined for asymptomatic adrenal lesions between 4-6 cm. However, surgery should be recommended for adrenal lesions larger than 6 cm. In a study conducted by Qing et al.[5], the mean pathologic size of the adrenal GNs was found at 6.3±3.1 cm. In the present case, performed right adrenalectomy due to the size of the mass and suspicion of malignancy. In pathological evaluation, adrenal GNs are composed of spindle-shaped cells and ganglion cells. They stain positively for S-100, vimentin, chromogranin A and synaptophysin.[4,10] In the

present case, immunohistochemical evaluation, positive staining for NSE and synaptophysin in ganglion cells and for S-100 in Schwann cells were observed. So, the patient was diagnosed as GN.

In conclusion, the detection of adrenal lesions has become easier nowadays due to the ease of access to imaging examinations. GNs are difficult to diagnose preoperatively because they resemble other tumors radiologically. Adrenal GN should be kept in mind in the differential diagnosis, especially in large asymptomatic masses that are not hormonally active.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

Cardiac Amyloidosis in a Patient Presenting with Symptoms of Heart Failure

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Abstract

Cardiac amyloidosis (CA); it can be referred to as a progressive cardiomyopathy that occurs as a result of the accumulation of endogenous proteins in the form of amyloid fibrils, whose folding is disrupted in the kidney, liver, gastrointestinal system, soft tissue and heart. The course of the disease depends on the involvement of the organs and treatment options depending on the source of the protein. Immunoglobulin light chain (AL) amyloidosis and transthyretin (TTR) amyloidosis are the most common CA types. While AL amyloidosis is more common in the heart and kidney, TTR amyloidosis is more common in the heart. Although CA is not considered a common disease, TTR amyloidosis is observed in approximately 15% of patients with heart failure with preserved ejection fraction and severe aortic stenosis. CA diagnosis: it can be placed by echocardiography, magnetic resonance or nuclear scintigraphy methods. At the same time, genetic analysis, biopsy and histopathological tests are also useful for early diagnosis. After the diagnosis, antiplasma treatment or stopping the produced protein constitute the main lines of the treatment.

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Keywords: *Amyloidosis, mitral valve regurgitation, heart failure*

Introduction

Cardiac amyloidosis is a restrictive form of cardiomyopathy that occurs as a result of improper accumulation of endogenous proteins with impaired folding. Although the diagnostic awareness of this disease is increasing, many cases are easily overlooked due to the need for endomyocardial biopsy and the introduction of genetic diagnosis procedures in the definitive diagnosis of the disease. Due to hypertrophy caused by cardiac infiltration; It is often confused with hypertensive heart disease or hypertrophic obstructive cardiomyopathy. The cases causing cardiac amyloidosis to occur due to two types of protein accumulation.[1] In immunoglobulin light chain (AL) amyloidosis, which occurs due to the accumulation of immunoglobulin light chain, 50% of the heart, 50% of the kidney involvement, 16% of the gastrointestinal system and 10%

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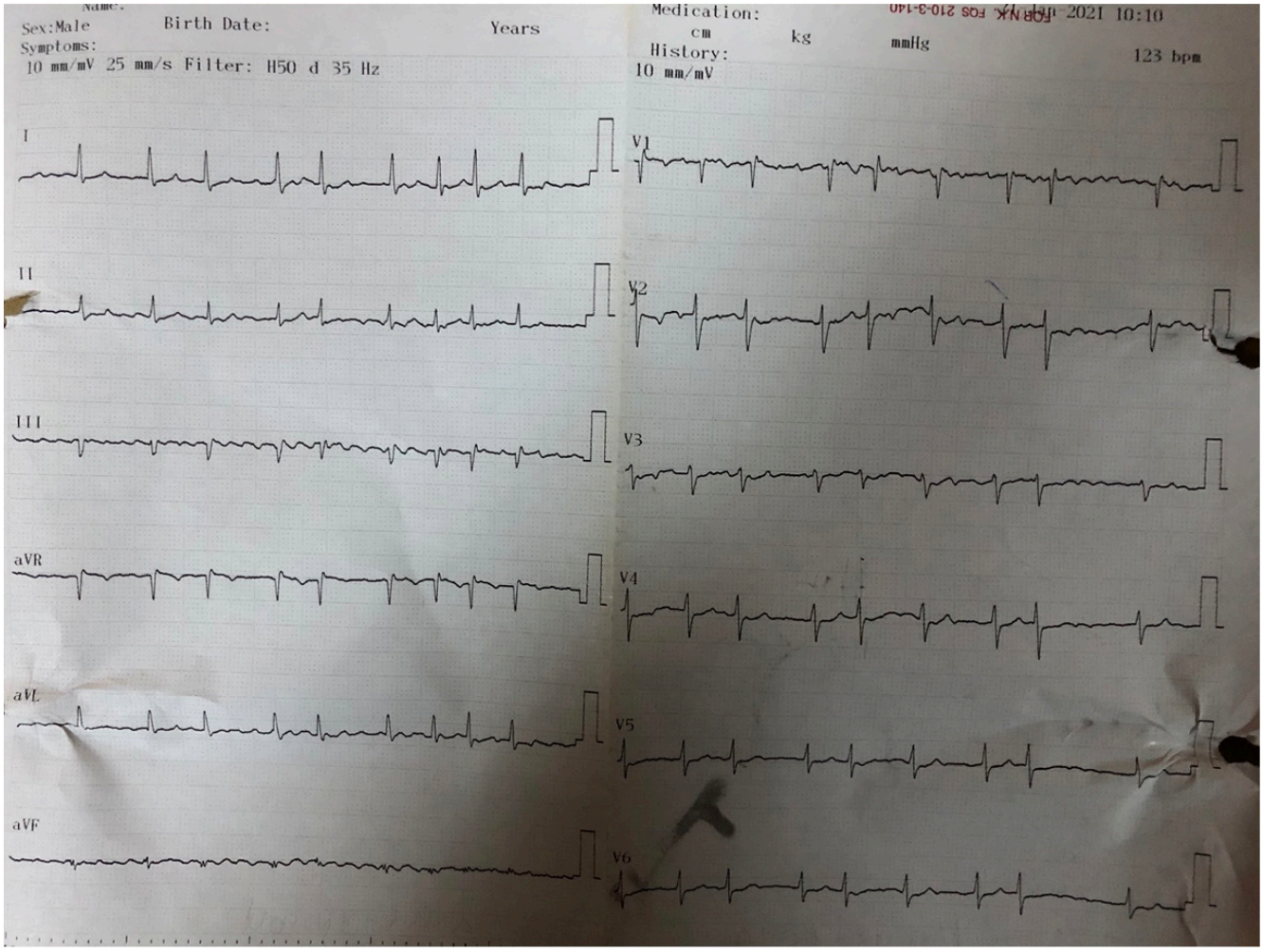

Figure 1. Voltage loss and atrial fibrillation were detected in the electrocardiography taken before the examination.

of neurological involvement occur.[2] Although transthyretin (TTR) amyloidosis, which occurs due to the accumulation of TTR produced in the liver, mostly involves the heart, accumulation occurs in soft tissues (carpal tunnel syndrome, etc.) and is called the wild type. In the inherited form of TTR amyloidosis, neurological system is affected, and it is inherited at a rate of 50%.[3] Low voltage[4] and conduction defects (e.g. atrial fibrillation) can be detected in electrocardiography, which is one of the non-invasive diagnostic methods. It occurs before low voltage heart failure findings occurring on electrocardiography (ECG) and hypertrophy on echocardiography (ECHO).[5] In the diagnosis of the disease, ventricular hypertrophy, left ventricular outflow stenosis, biatrial dilatation and hypertrophies in the atrioventricular valves can be detected on ECHO. Cardiac MRI, bone scintigraphy, genetic analysis can be performed for diagnosis, and the gold standard diagnostic method is endomyocardial biopsy. Treatment in amyloidosis is plasma therapy or the use of stabilizers to prevent accumulation. In this case report, we will be sharing a 61-year-old male patient with hereditary TTR amyloidosis.

Case Report

A 61-year-old male patient with persistent shortness of breath and headache was referred to our cardiology outpatient clinic by neurology. Hypotension was found in the detailed history of the patient. In the examination of the patient, his general condition was found to be moderate, heart rate 125 beats/min, arterial blood pressure 100/65 mmHg. In cardiac auscultation, a second-degree systolic murmur on the mitral valve and a pansystolic murmur on the tricuspid valve, and

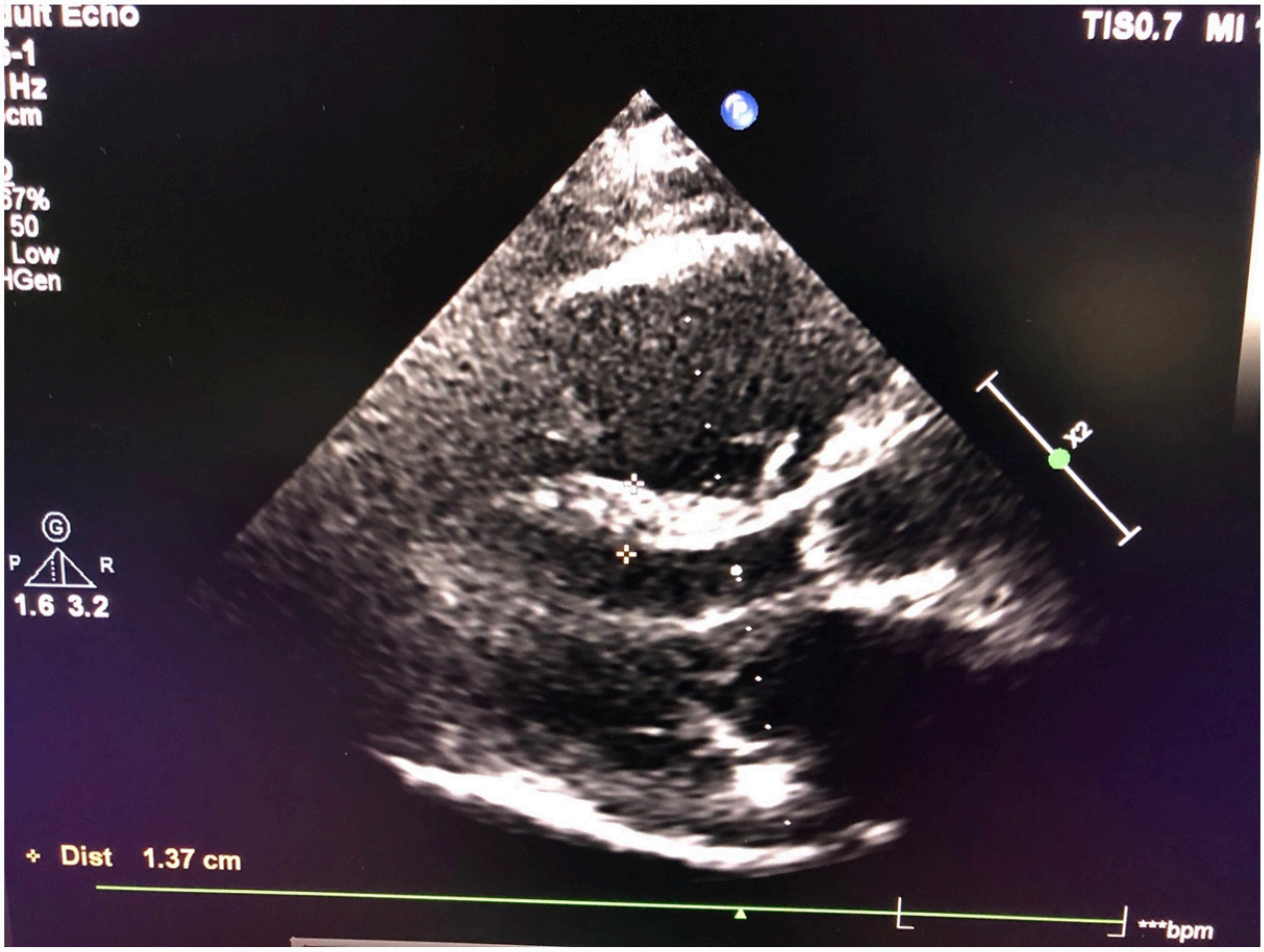

Figure 2. Echocardiography revealed left ventricular hypertrophy, left atrial dilation, and 2nd degree mitral regurgitation.

rales in the lower zones on lung examination were detected. The patient had +1/+1 pretibial edema. Voltage loss and atrial fibrillation (AF) were detected in the ECG taken before the examination (Figure 1). In the biochemical tests performed on the patient at that moment, creatinine 1.2 g/dL, potassium 5.5 mmol/L and NT-proBNP 1,250 were found. Abnormal values were not found in other biochemical parameters. ECHO revealed left ventricular hypertrophy, left atrial dilation, and 2nd degree mitral regurgitation (Figure 2). The patient had 1-2° tricuspid regurgitation in the right ventricular evaluation. With these findings, heart failure with preserved ejection fraction (HFPEF) was initially considered in the patient. On the other hand, cardiac amyloidosis was also suspected due to the presence of left ventricular hypertrophy in echocardiography and low voltage in the precordial leads inconsistent with left ventricular hypertrophy on the ECG. Internal medicine and neurology consultation was also requested to investigate neurological and gastrointestinal involvement. Diuretic therapy, diltiazem 2x90 mg and anticoagulation were initiated, and cardiology outpatient control was recommended to the patient. The patient was ultimately referred to the gastroenterology clinic after being evaluated by the internal medicine clinic, and as a result of rectal biopsy, primary amyloidosis was diagnosed.

Discussion

Amyloidosis: it is a disease caused by the accumulation of proteins called amyloid with incorrectly folded beta chains in tissues. There are 4 types of amyloidosis: primary, secondary, familial, and senile amyloidosis. Cardiac

amyloidosis occurs when amyloids are deposited in myocytes. Cardiac amyloidosis can be seen in both primary and secondary types. However, it is more common in primary amyloidosis. Although diffuse myocardial involvement mostly causes restrictive cardiomyopathy, it may rarely cause constrictive pericarditis by causing pericardial involvement in some cases. Much more rarely causes cardiac tamponade.[6] In ECHO, thickening of the left ventricular wall, non-dilated left ventricular cavity, diastolic dysfunction and enlargement of the atria are common features.[7] On the other hand, the most common abnormality in cardiac amyloidosis on ECG is low voltage.[8] Amyloidosis in cardiac involvement leads to restrictive cardiomyopathy, leading to a decrease in early diastolic filling and peripheral edema findings occur. For definitive diagnosis, only if abdominal fat biopsy is negative, endomyocardial biopsy can be considered.[9] Another clinical picture that can be caused by cardiac amyloidosis is congestive heart failure. More rarely, orthostatic hypotension can be seen due to nephrotic syndrome caused by vascular involvement and renal involvement.[10] It has also been reported that it may cause arrhythmias and sudden death by involving her purkinje, which is one of the cardiac conduction pathways.[11] Treatment in primary amyloidosis is chemotherapy and bone marrow transplantation. Many cases of amyloidosis are not suitable for cardiac transplantation due to significant non-cardiac amyloidosis. However, in selected cases, chemotherapy and transplantation after bone marrow transplantation can be applied in primary amyloidosis.[12]

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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Case Report

A Case of Hairy Cell Leukemia Diagnosed by Leukocyoclastic Vasculitis Symptoms

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Abstract

Hairy cell leukemia (HCL) is a rare chronic lymphoproliferative disease. LSV is characterized by inflammation of small vessels. Its association is rare in the literature and HCL can be seen in LSV etiology, although the relationship between the 2 diseases is far from clear. Here, we aimed to present a case with constitutional symptoms, palpable purpuric lesions on the body and diagnosed as HCL from bone marrow biopsy.

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Keywords: *Leucocytoclastic vasculitis, Hairy cell leukemia, skin rash*

Introduction

Hairy cell leukemia (HCL) is a rare chronic lymphoproliferative disease characterized by pancytopenia and splenomegaly, with 'hairy' cells seen in peripheral blood and bone marrow. The average age at diagnosis is between 50 and 55 years. The male/female ratio is 4/1. Common findings of HCL includes massive splenomegaly, pancytopenia, and lymphadenopathy; fever is a rare symptom. The frequency of several autoimmune diseases is increased in HCL, but rare cases of leukocytoclastic vasculitis (LV) have been rarely reported.[1,2] LV is characterized by inflammation of small vessels. The etiology of LV includes drugs, infections, malignancies, and systemic inflammatory diseases. Palpable purpura is typical, especially in the lower extremities. In this article, we aimed to present an HCL case with leukocytoclastic vasculitis symptoms.

Case Report

A 70-year-old male patient was admitted to the emergency department with complaints of palpable purpuric lesions, fever, night sweats, and weakness, which started on his extremities for 6 months and were more intense on his abdomen and back for

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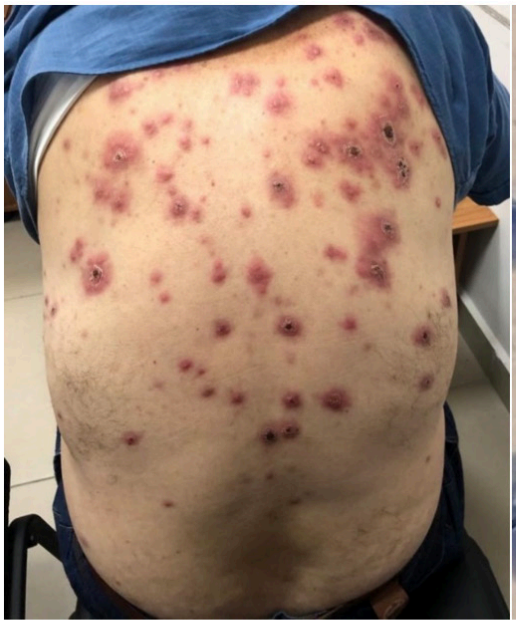

Figure 1. The appearance of pustular lesions on the erythematous ground in the patient's extremities and body.

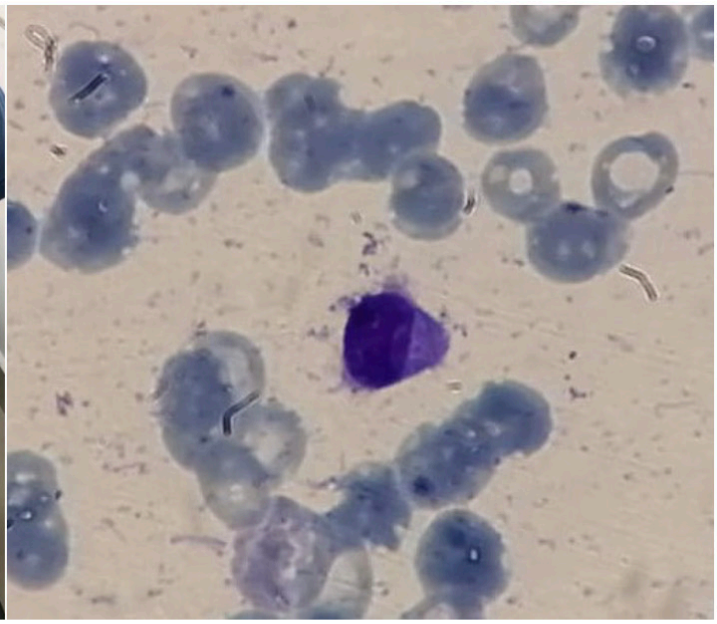

Figure 2. Bone marrow examination

the last 2 weeks. On physical examination, his general condition was normal, there were crusted, pustular lesions on the erythematous ground on his extremities and body (Figure 1). The patient did not report any history of a medical disease and his family history was normal. Complete blood count revealed: leukocyte: 1470/mm³, neutrophil: 831/mm³, lymphocyte: 601/mm³, hemoglobin: 7.9 gr/dL, platelet: 150,200/mm³, C-reactive protein: 123 mg/L, erythrocyte sedimentation rate: 140 mm/h. The serological examination for Brucella, hepatitis was negative, parvovirus, cytomegalovirus, Epstein-Barr virus, measles, chickenpox, mycoplasma, Lyme, rubella were also found to be negative. Anti-neutrophil cytoplasmic antibody, rheumatoid factor and anti-cyclic citrullinated peptide negative, C3 and C4 normal, antinuclear antibody (ANA) 1/100 end point positive, ANA profile negative. The patient was hospitalized and piperacillin/tazobactam 3x4.5 gr was administered. Punch biopsy pathology of the lesion in the extremity was reported as LV. The patient's constitutional symptoms also increased the probability of possible systemic disease. The patient's COVID-19 PCR test was negative, the absence of ARB in urine and sputum, normal level of immunoglobulins, no lymphadenomegaly in thoracic and abdominal computed tomography, spleen size at the border of 13 cm, and peripheral smear showed atypical lymphocytes. Bone marrow biopsy was performed to explain the cause of pancytopenia. In bone marrow aspiration and imprint examination, atypical lymphocytes were larger than normal, chromatin structure was thin, cells with large cytoplasm, and cytoplasmic protrusions (Figure 2). Bone marrow pathology; morphological features primarily suggest HCL. TRAP was poorly stained and immunohistochemical negative results were reported as other B-cell lymphoid neoplasms could be excluded, and the diagnosis of HCL was confirmed. The patient was transferred to the pandemic service due to respiratory distress in the follow-up and the received COVID-19 PCR test was positive. The treatment for COVID-19 was administered. In his follow-up, acute respiratory distress syndrome developed and died.

Discussion

HCL and LSV are rare diseases. Few cases are reported together in the literature, and the underlying mechanism is not clear. In its pathogenesis, infiltration into the vessel wall by

hairy cells is thought to be responsible. Local cytokines causing inflammatory tissue damage, antibody cross reactivation with the vessel wall of hairy cells may be responsible for further propagation of the process. LV can be seen at any stage of HCL.[1,2] Cutaneous symptoms also regress when treating the underlying HCL with interferon or purine analogs. In conclusion, HCL can be seen in histological LV etiology even though there is no clinical picture.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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