E-ISSN
e-ISSN 2149-9934
Volume: 11 **Issue:** 3 November 2020

Journal of Emergency Medicine Case Reports

EPAT
1999
Emergency Physicians Association of Turkey

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Editorial

Dear colleagues,

Nowadays, when we are going through extraordinary conditions, we are in a front-line struggle in a biological war. While the efforts made for the future of the country are being burdened on our shoulders in this difficult time, while advising everyone to stay at a distance we continue to strive to fulfill our profession with the difficulty of serving our patients face to face. The anonymous heroes of the health army, who show sacrifice far from being expressed by words, also continue to produce science and research. Our case report journal which is one of the 4 journals of EPAT, is scanned in many national and international indexes and within the scope of ESCI, continues its publication life despite all difficulties. Emergency medicine has an extremely wide spectrum. Very different cases can be applied almost every day and these interesting cases make important contributions to the medical literature. This prestigious journal, which is very popular in the international arena and is the first in our country in this regard, has come to this day with the important contributions of many scientists. Since this issue, there has been a flag assign and our journal will continue to move forward with a new, young and dynamic editorial team. We would like to thank all our stakeholders who have worked so far, and wish success to our new team.

Prof. Dr. Başar Cander

Değerli Meslektaşlarımız

Olağandışı şartlardan geçtiğimiz bugünlerde adeta biyolojik bir savaşın içinde ön cephede sürekli bir mücadele içindeyiz. Bu zor zamanda ülkenin geleceği için yapılan çabalar omuzlarımıza yüklenirken, herkese mesafeli olmalarını tavsiye ederken biz hastalarımızla burun buruna hizmet vermenin güçlüğüyle mesleğimizi icra etmeye gayret göstermeye devam ediyoruz. Bu kelimelerle ifade edilmekten uzak fedakârlığı gösteren sağlık ordusunun isimsiz kahramanları bir taraftan da bilim üretemeye, araştırma yapmaya devam etmekteler. ATUDER'in sürekli yayın yapan 4 dergisinden biri olan ulusal ve uluslararası birçok indekste taranan ESCI kapsamındaki case report dergimiz de yayın hayatına tüm zorluklara rağmen devam etmektedir. Acil tıp son derece geniş bir spektruma sahiptir. Hemen her gün çok farklı vakalar başvurabilmekte ve bu ilginç vakalar tıp literatürüne önemli katkılar sunmaktadırlar. Uluslararası arenada da çokça rağbet gören ve bu konuda ülkemizde ilk olan bu saygın dergi, birçok bilim insanının önemli katkılarıyla bu günlere gelmiştir. Bu sayımızdan itibaren bir bayrak devri olmuştur ve dergimiz genç dinamik yeni bir editör ekibiyle ileriye doğru yürümeye devam edecektir. Bugüne kadar emek sarf eden tüm paydaşlarımıza teşekkür eder yeni ekibimize başarılar dileriz.

Prof. Dr. Başar Cander

Contents

Case Report

Journal of Emergency Medicine Case Reports

Toxic Epidermal Necrolysis in a Patient with Allopurinol, Colchicine and Alcohol Use

Munise Daye[1], Selami Aykut Temiz[2], Şevket Arslan[3], Alper Yosunkaya[4], Selim Gümüş[1], Orkun Uyanık[5], Hayri Ahmet Burak Nurşen[5]

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Abstract

Introduction: Toxic epidermal necrolysis is a severe, acute, mucocutaneous, life-threatening hypersensitivity syndrome with high mortality and bullous lesions on the skin, eyes and mucous membranes. It often develops due to drugs. Sulfonamide group antibiotics and antiepileptic drugs are the most commonly responsible agents. Allopurinol is a common cause of toxic epidermal necrolysis as in most drug reactions. Colchicine is widely used in dermatology and rheumatology and is generally known as an agent with a broad safety profile.

Case report: Here we present a case of toxic epidermal necrolysis in our case with allopurinol, colchicine and alcohol use in order to draw attention to the increased risk of drug coexistence.

Conclusion: Again, we wanted to draw attention to the management of our case and the efficacy and safety of high-dose intravenous immunoglobulin therapy.

Keywords: Toxic epidermal necrolysis, allopurinol, colchicine, intravenous immunoglobulin.

Introduction

Toxic epidermal necrolysis (TEN) is a serious life-threatening hypersensitivity syndrome with high mortality and acute course of bullous lesions in the skin, eyes and mucous membranes[1]. The annual incidence of TEN, which often develops due to drugs, is approximately 0.4-1.2 per million[1]. Sulfonamide group antibiotics and antiepileptic drugs are the most frequently held responsible agents[2]. In some epidemiological studies, allopurinol has been held responsible for approximately 10% of all cases[3]. Allopurinol is a xanthine oxidase inhibitor, especially used in the treatment of gout and hyperuricemia. It causes cutaneous drug reactions quite frequently. Colchicine is widely used in gouty arthritis, familial mediterranean fever, Behçet's disease, erythema nodosum and neutrophilic dermatoses[4]. Here we wanted to present our case of toxic epidermal necrolysis and treatment management in the case of allopurinol, colchicine and alcohol use.

Case Report

A 33-year-old male patient was diagnosed with gouty arthritis from internal medicine department and treatment was started with allopurinol and colchicine 15 days ago. On the fifteenth day of the treatment, he was referred to our hospital with complaints of widespread redness, mouth ulcers and stinging eyes. The patient, who was evaluated in the emergency department, had a history of daily alcohol use in his medical background.

On his physical examination, the blood pressure was 140 / 80 mmHg, pulse was 128 / min, and body temperature was 36.7 °C. In dermatological examination there was erythematous maculopapular eruption in approximately 80% of his body which tended to merge bullous lesions and erode areas in more than 30% of his body (Figure 1). There was eye, oral and genital mucosa involvement (Figure 2).

Laboratory analysis revealed C-reactive protein (CRP) 165 mg / L, serum bicarbonate 19 mmol / L. The SCORe

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Received: 06.02.2020 • **Accepted:** 03.10.2020

DOI: 10.33706/jemcr.684184

DOI: 10.33706/jemcr.684184
Journal of Emergency Medicine Case Reports 2020; 11(3): 65-67

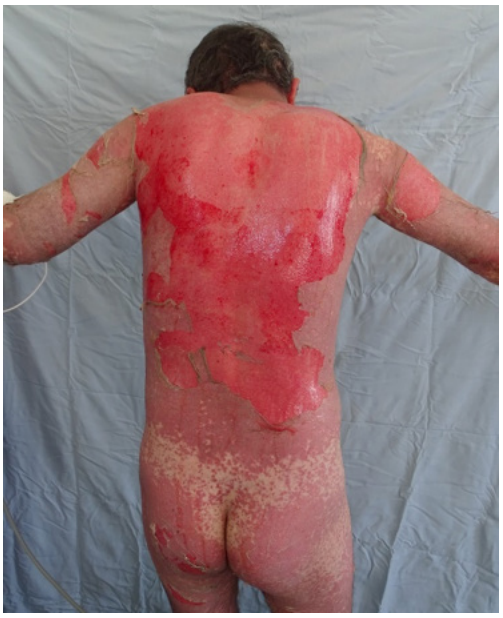

Figure 1: Erythematous maculopapular eruption and diffuse exfoliation, tending to merge in approximately 80% of the body

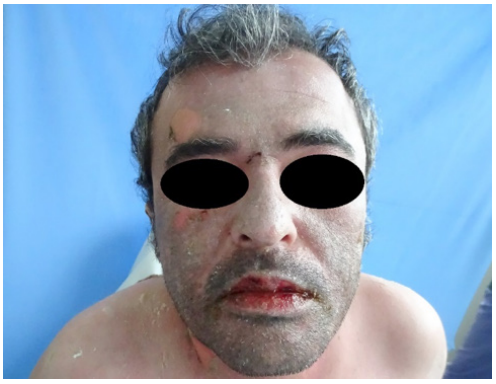

Figure 2: Oral mucosa and eye involvement

of Toxic Epidermal Necrosis (SCORTEN score) is a severity-of-illness scale. The SCORTEN score was evaluated as three due to pulse, serum bicarbonate and body surface areas. The patient, who had a definitive diagnosis of TEN, was hospitalized in the reanimation intensive care unit. Cutaneous biopsy was taken for differential diagnosis.

IV ceftriaxone 70 g / day (1 g / kg / day) was administered for 14 days with the recommendation of the infectious diseases department. 350 g (70 g / day, total 5 g / kg) intravenous immunoglobulin (IVIG) treatment was administered for five days. 1 mg / kg / day (70 mg) methylprednisolone treatment was started and was gradually tapered after (2 weeks) after the response was obtained. Levocetirizine 5 mg was given three times a day until the patient was discharged from the hospital. Local streoid, eau de borique %2, antibiotic cream and sterile dressings, which made by plastic surgeons, treatments were applied. Nutritional and electrolyte follow-up of the patient was done by the reanimation unit. No secondary infection or metabolic disorder developed in the case. In terms of eye and genital mucosa involvement, recommended local treatments were applied to our case, who was consulted with urology and ophthalmology.

The patient whose bullous lesions regressed with the treatments at the end of two weeks was taken to the dermatology service from the intensive care unit. The patient was discharged with almost complete regression of the lesions followed in the service for two weeks (Figure 3).

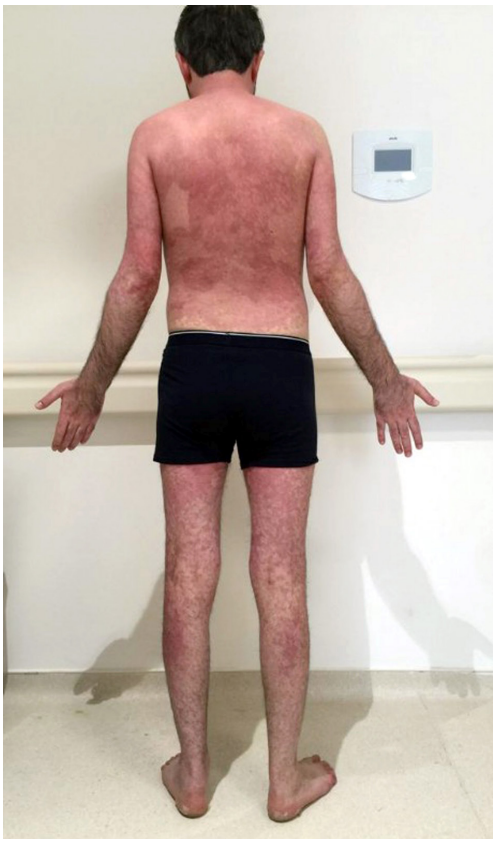

Figure 3: Almost complete regression in lesions after approximately four weeks

Discussion

The treatment of toxic epidermal necrolysis first begin with the detection and discontinuation of the etiological agent[1]. The patient should then be transferred to a hospital with a burn unit (or intensive care unit). It is necessary to do wound care, maintain the ambient temperature of 30–32 °C, and maintain fluid-electrolyte balance[2]. The SCORTEN score should be calculated on day 1 and 3 in terms of mortality and risk follow-up[3]. In addition to steroid therapy IVIG, tumor necrosis factor (TNF) inhibitors or cyclosporin should be considered as adjuvant therapy within first 24-48 hours[5,6]. We planned IVIG treatment for our case whose the Scorten score we calculated three and was in the high risk group because of his high CRP, infection parameters and high safety profile of IVIG.

IVIG, which is used in the treatment of toxic epidermal necrolysis, has been shown to contain antibodies that block

Fas in vitro and inhibits the formation of the Fas-FasL (Fas ligand) composition[7]. However, there are opinions that argue that the duration and extent of necrolysis and mortality rates in patients using IVIG are not different than expected. In a study (using IVIG treatment) in which 64 patients were evaluated retrospectively, mortality did not decrease. However, the low number of patients in this study and its retrospective nature were important limitations of the study[8].

It has been shown that there is no significant reduction in mortality in a large meta-analysis study in which 221 patients were evaluated, but it decreases mortality at doses above 2 g / kg IVIG treatment[9,10]. In our case, it was planned to give 210 g IVIG from 3 g / kg in 3 days, but since the lesions continued, the treatment was completed in 5 days and 350 g IVIG was given from a total of 5 g/kg. As mentioned in the literature, with the effect of using high IVIG doses, the patient's condition was improved within weeks and he was discharged in a healthy.

Allopurinol can cause reactions such as hypersensitivity reaction, Drug Reaction with Eosinophilia and Systemic Symptom (DRESS), Stevens-Johnson syndrome (SJS) and TEN. In a meta-analysis study, it was stated that allopurinol is the leading cause of SJS / TEN in many countries (11). In the same study, it was reported that HLA-B * 5801 tissue typing can be used to detect allopurinol-induced SJS / TEN worldwide, regardless of geographical ethnic differences. Thus, it is thought that the development of SJS / TEN can be prevented by controlling allopurinol prescription in people who carry this allele more frequently. However, this practice has not yet entered the clinical practice routine.

Colchicine, another drug used by our case, is a commonly used agent in dermatology and rheumatology, with a wide safety profile in terms of cutaneous reactions. In a meta-analysis related to SJS and TEN in 2019, there were only four cases in the literature due to colchicine[1]. It is known that agents that inactivate microtubules induce apoptosis. Colchicine has been shown to induce apoptosis in fibroblasts, osteosarcoma cells, myeloid leukemia cells, cerebellar granular cells and adenocarcinoma cells[12].

It is known that the use of alcohol together with drugs increases the development of TEN[1]. Our case was using colchicine and alcohol along with the use of allopurinol, which is known to cause TEN. We think that the combination of colchicine and alcohol besides allopurinol probably facilitates reaction development. Allopurinol, which is held very responsible for drug reactions, should be used very carefully. Other drug history and alcohol use should also be questioned in cases that are planned to be used.

Conclusion

TEN cases, one of the most serious of dermatological emergencies, should definitely receive a multidisciplinary care under the leadership of the dermatologist under intensive care conditions[6]. In this article, we wanted to draw attention to our toxic epidermal necrolysis case with allopurinol, colchicine and alcohol use and the possible side effects of allopurinol may increase with the combination of drugs and alcohol. Again we wanted to point out that high-dose IVIG, previously recommended in the literature at a dose of 2-4 g / kg (5 g / kg in our case), is a safe and effective option.

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Case Report

Journal of Emergency Medicine Case Reports

Ruptured Pulmonary Hydatid Cyst: A Case Report

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Abstract

Introduction: Hydatid cyst disease (hydatidosis or echinococcosis) is an important parasitic disease in all societies engaged in agriculture and animal husbandry, where environmental health and preventive medicine measures are insufficient. Lung hydatid cyst is a zoonotic infection caused by larval forms (metasestod) of E. granulosus (EG).

Case report: In our case, a 77-year-old woman was admitted to the Emergency Department with cough sputum and high fever. The patient had complaints of cough and sputum after exercise for 5-6 days in her anamnesis, fever and fatigue in the last 2 days. Physical examination revealed that the oropharynx was normal, both hemithoraxes were participating in the respiration equally, and the right lung basal breathing sounds were reduced by listening. Upon detecting an appearance compatible with abscess on chest radiography, the patient underwent thoracic tomography for further examination and treatment. After our evaluations, the patient was operated by thoracic surgery with the diagnosis of ruptured pulmonary hydatid cyst.

Conclusion: This case is presented to emphasize the importance of physical examination and imaging methods in patients presenting to the emergency department with classic upper respiratory tract infection or community-acquired pneumonia clinic.

Keywords: Pulmonary Hydatid Cyst, Pulmonary Abscess, Case Report

Introduction

Hydatid cyst is a parasitic infectious disease caused by Echinococcus granulosus, which is common in nature and is frequently encountered in our country. It is prevalence is approximately 50-400 / 100,000 and its incidence is 3.4 / 100,000. Echinococcus granulosus is very common in the world and is a common parasitic disease in countries where agriculture and animal husbandry are common, mostly transmitted to human by dog feces. Embryo, which is separated from the egg in the duodenum by the water and food after the eggs are eaten by the dog faeces and spread to the external environment, settles the liver most frequently via the vena porta or lymphatic system and secondly into the lungs. They take the form of larvae in the settled areas, causing hydatidosis (hydatid cyst) formation[1].

Complications such as hemoptysis, respiratory failure, anaphylactic shock, pneumothorax, empyema, lung abscess may occur due to ruptured cyst in the lung[2]. This case was presented to the emergency department in order to draw attention to the importance of physical examination and imaging methods in patients presenting with classic upper respiratory tract infection or community-acquired pneumonia clinic.

Case Report

A 77-year-old female patient was admitted to the emergency department with complaints of cough sputum and high fever for 2 days. Vital signs of the patient; fever: 38,0 ^{O}C Respiratory Rate: 19/min Pulse: 90/min Blood Pressure: 132/78 mmHg Blood Sugar: 150 mg/dL SO_2: % 95. The patient had complaints of cough and sputum after exercise for 5-6 days in her anamnesis, fever and fatigue in the last 2 days, she was admitted to the green area of emergency department. Physical examination revealed that the oropharynx was normal, both hemithoraxes were participating in the res-

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Received: 11.05.20 • **Accepted:** 26.10.20
DOI: 10.33706/jemcr.735428

piration equally, and the right lung basal breathing sounds were reduced by listening. Abdominal and neurological examination was normal. It was recorded that she had essential tremor in her medical history and that she used Dideral (Propranolol) regularly.

Pneumonia was considered as a preliminary diagnosis and a chest radiograph was taken and routine blood tests were performed. Laboratory tests results were Hemoglobin: 12,77/gr, white blood cell (WBC); 14.890/mm^3, neutrophil; %78 , C reactive protein (CRP);108.8 mg/dL, glucose: 165 , creatinine: 0,71, D-Dimer: <140 mg/dL. A chest x-ray showed a circular lesion in the lower lobe of the right lung that yielded air-fluid levels (Picture 1). Tomography of the lung showed a large ruptured hydatid cyst in the lower lobe of the right lung (Picture 2). The patient was administered intravenous paracetamol and 500 mg metronidazole in the emergency department in accordance with the clinical symptoms and diagnosis. At the same time, the patient with shortness of breath inhaled Ipratropium bromide-salbutamol and Budesonide 2 times 20 minutes apart.

In the patient's history, it was learned that cough with serous water came about 5 to 6 days ago after exertion but when it comes to water swallowed. The patient was consulted with Thoracic Surgery for the diagnosis of ruptured lung hydatid cyst and was operated with the diagnosis of ruptured lung hydatid cyst. Cystotomy was performed through the right lower lobe posterolateral through the parenchyma. Intracystic fluid aspirated, membrane was removed and was cleaned with povidone iodine. Two thoracic drains were followed up in the intensive care unit and then in the thoracic surgery clinic. The patient was discharged with full recovery after being followed up in the thoracic surgery department for 1 week postoperatively.

Discussion

The diagnosis of hydatid cyst is made by taking anamnesis, physical examination, laboratory findings and imaging methods. Chest X-ray, Thorax Ultrasonography and Tomography imaging methods are diagnostic methods. Indirect Hemagglutination, Indirect Fluorescent Antibody and Enzyme Immunoassay (EIA) and Complement Fixation (Weinberg) tests are used in diagnosis. Techniques such as Indirect Hemagglutination Test and ELISA are used for the detection of antibodies (Ig G1, G2 and G4) produced by Echinococcus multilocularis and Echinococcus granulosus. Western Blot test is used as verification test. Does not exclude diagnosis due to negative test results of up to 50% in hydatid cyst. The presence of scolexes in pathological examination of sputum is pathognomonic finding in diagnosis[1].

The diagnosis of hydatid cyst is made by radiological examinations. Noninvasive imaging techniques such as chest radiographs and thorax tomography are used for the diagnosis of pulmonary cysts. Intact cysts appear as smooth rounded opacity on plain chest X-ray, and well-circumscribed, smooth and thin wall surrounded by homogeneous fluid density on computed tomography. Air penetration between the pericyst and cystic membrane creates the appearance of crescent. This view is called ‘meniscus sign’. It is a very specific radiological finding for hydatid cyst, indicating that the cyst is about to explode. If the parasite membrane is fully ruptured and some amount of cyst fluid is expectorated, the membrane collapses and falls on to the fluid. In this situation, the collapse membrane images floating on the liquid together with the air-liquid level form the “lotus flower”

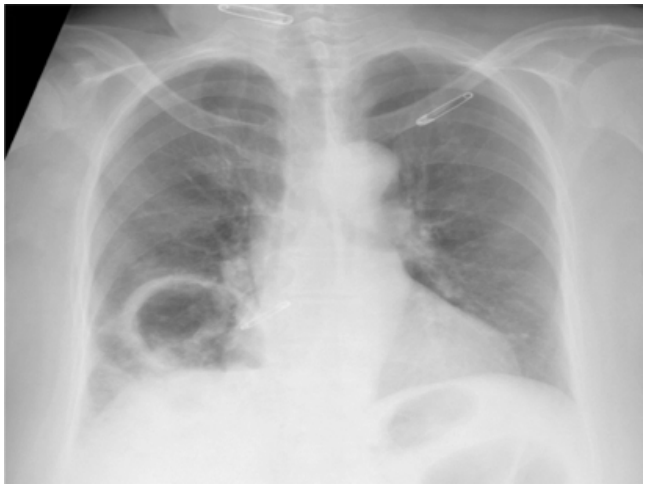

Figure 1: Chest radiograph consistent with abscess giving air fluid level in the lower lobe of the right lung

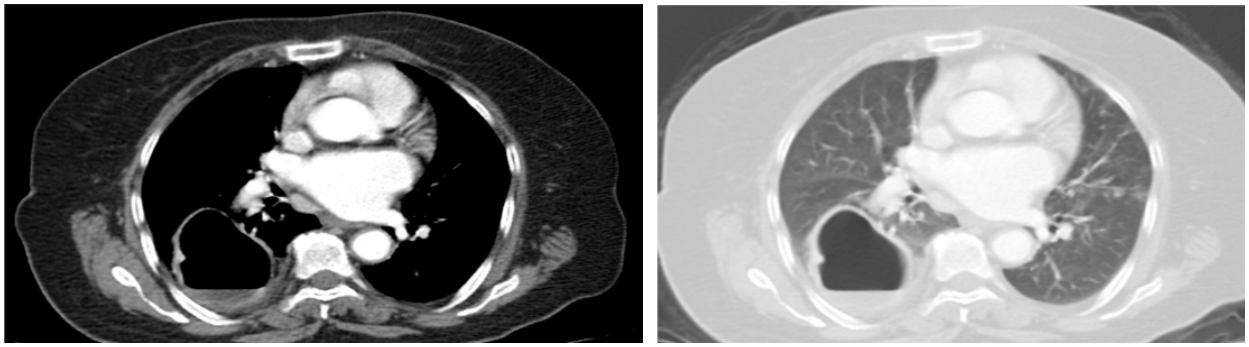

Figure 2: : Thorax tomography revealed a large ruptured hydatid cyst in the lower lobe of the right lung

DOI: 10.33706/jemcr.735428
Journal of Emergency Medicine Case Reports 2020; 11(3): 68-70

sign[3,5,6,7]. In our patient, imaging findings were consistent with abscess giving air-fluid level in the right lung lower lobe on chest X-ray, and a lesion compatible with large ruptured hydatid cyst cavity in the right lower lobe on thorax tomography.

Primary treatment of pulmonary hydatid cyst is surgery. Intact cysts should be operated immediately due to the risk of infection and rupture. Even if the parasite inside the cyst has died, the remaining germinative membrane should be removed by operation as it may be the source of infection. Today, parenchymal preservative surgical methods (enucleation, cystotomy-capitonage, pericystotomy and wedge resection) in hydatid cyst surgery used[8,9,10]. Resective surgery can be performed if more than 50% of the lobe is devastated[4]. Surgical method applied in our patient, the most common surgical method was cystotomy-capitonage.

Considering the clinical and diagnostic methods, this case is presented to draw attention to the importance of physical examination and imaging methods in patients presenting to the emergency department with classical upper respiratory tract infection or community-acquired pneumonia clinic.

Conclusion

We believe that physical examination findings and imaging methods are also important in patients presenting to the emergency department with cough, nonspecific fever and sputum complaints. Since our country is geographically located in the endemic region, we think that it should be considered in the differential diagnosis because of the prevalence of hydatid cyst cases.

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Case Report

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Giant Jejunal Diverticula Causing Pseudoobstruction

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Abstract

Introduction: Small bowel diverticula is a rare condition and is rarely seen in the jejunum compared to the ileum. It is usually incidentally detected. It is mostly asymptomatic. Herein, we aimed to discuss a case of multiple jejunal diverticula that have caused pseudoobstruction in the light of the literature.

Case report: A 55-year-old male patient had complaints of abdominal pain, abdominal swelling, nausea and vomiting. Mechanic intestinal obstruction was considered. Multiple giant diverticula were found in entire jejunum.

Conclusion: Jejunal diverticula may produce a different clinical picture. It should be kept in mind that jejunal diverticula that is a rare condition may cause pseudoobstruction.

Keywords: Jejunum, Diverticula, Pseudoobstruction.

Introduction

Diverticle is defined as outpocketing of the intestinal wall. Intestinal diverticula is a commonly seen condition. However, small bowel diverticula is a rare condition and is more frequently seen in the ileum. The prevalence of small bowel diverticula differs between 0.3% and 4.5% in autopsy studies[1-3]. Jejunal diverticula are mostly asymptomatic[1]. The diagnosis is usually made incidentally. The most commonly expected symptoms are abdominal pain, bleeding, and rarely intestinal obstruction[2]. It is more common in male gender and advanced ages[4].

Herein, we aimed to discuss a rare case of giant multiple jejunal diverticula progressing with abdominal pain and vomiting, in light with the literature.

Case Report

A 55-year-old male patient was admitted to the emergency department with complaints of abdominal pain, nausea, vomiting, abdominal swelling, weight loss, and excessive fatigue. In the physical examination, the patient was cachectic. The inspection revealed abdominal distention and abdominal fullness on palpation. He had a history of hypertension.

Blood pressure was 90/50 mmHg, and pulse was 100 bpm. Among the laboratory values, hemoglobin was 10 g/dL, and albumin was 3 g/dL. İnitially, intestinal malignancy was considered in the patient. He was admitted to the general surgery clinic. Parenteral feeding was initiated.

In the endoscopic examination, no pathology was found in the upper and lower gastrointestinal systems. On the all abdominal computed tomography, dilatation and multiple diverticula like structures were observed at the small intestinal proximal level (Figure 1).

The patient was taken to the operation after 5-day resuscitation. Parenteral nutrition was started for the patient. Electrolyte imbalance of the patient was corrected. Multiple giant diverticula with the largest one reaching 10 cm were found in entire jejunum (Figure 2).

Diverticula began after the 4th part of the duodenum and ended at the level of ileojejunal junction. Despite dilation in

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Received: 28.05.2020 • **Accepted:** 6.10.2020
DOI: 10.33706/jemcr.744147

DOI: 10.33706/jemcr.744147
Journal of Emergency Medicine Case Reports 2020; 11(3): 71-73

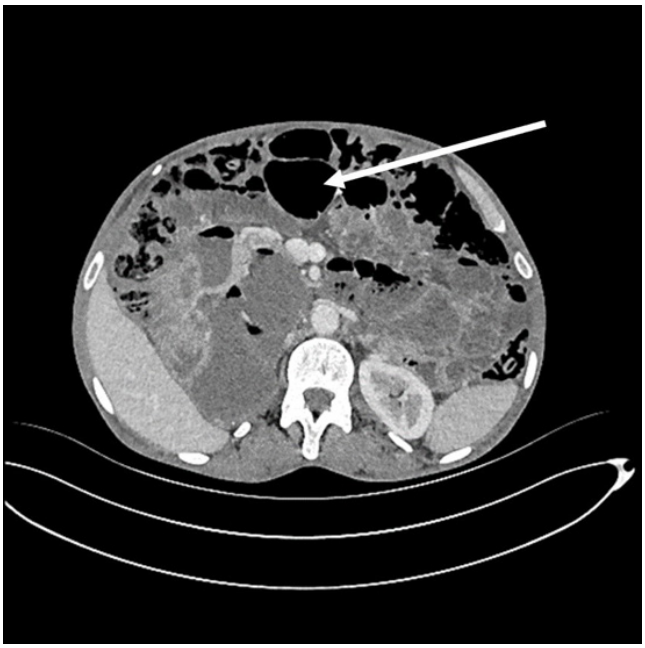

Figure 1: A section of abdominal computed tomography

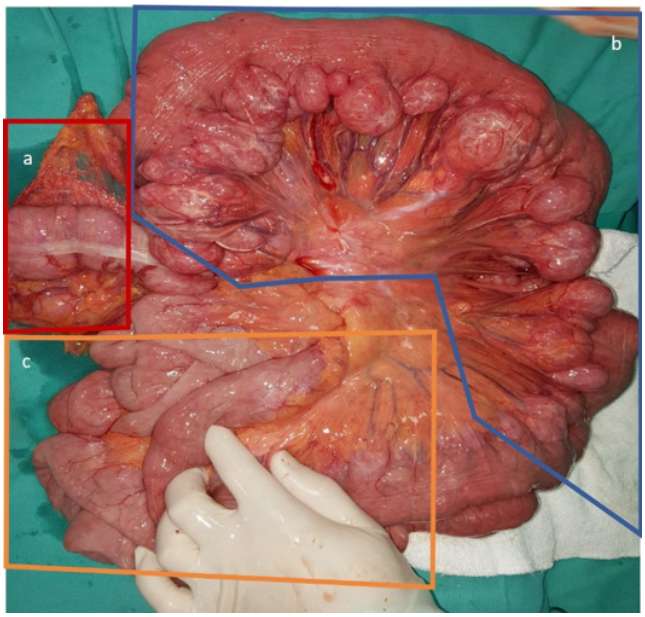

Figure 2: Operation image (a: normal colon, b: jejunal diverticula, c: normal ileum).

the jejunal loops, there was no any cause leading to obstruction. Resection was not performed due to general status of the patient. Postoperative period was complication free and oral intake was started on the third day. The patient who tolerated oral intake was discharged with a medication containing otilonium bromide and simethicone.

Discussion

Mechanisms of the formation of jejunal diverticula with is a rarely seen disease has not been fully clarified. In general, intestinal wall smooth muscle dysfunction or myenteric plexus defects have been accused[5]. Irregular jejunal contractions cause elevation in intraluminal pressure. Following the increase in intraluminal pressure, pulsion of the mucosa and submucosa develops in the weakened intestinal wall (at the vasa recta entrance region). Diverticula usually occur in the mesenteric surface. In our patient, all diverticula developed in the mesenteric region. Intestinal wall was thick and dilated. The number of diverticula was beyond measure.

Majority of cases have an asymptomatic course. Rarely they may be symptomatic. Symptomatic cases show perforation, obstruction, malnutrition and bleeding[5, 6]. Perforation is usually progress with abdominal pain and fever. In the imaging examinations, inflammatory mass, abscess, free air or mesenteric air may be observed. The diagnosis is usually confused, increasing the mortality rate up to 50%[6]. In our case abdominal pain was prominent. However, there was no acute abdominal finding in the physical examination. Oral intake of the patient was disrupted because of nausea and vomiting. Malabsorption, which is seen in such cases, accelerated weight loss in our patient. He lost 15 kg within the last 3 months.

Bleeding is one of the complications seen in these patients. Although rarely, fatal bleeding may also occur[2]. Bleeding manifests as melena or hematemesis. Hematemesis is due to the traume occurring in diverticula and subsequent ulcer[7]. Endoscopic diagnosis investigations usually fail[7, 8]. Mesenteric angiography or scintigraphy are successful in showing the localization. No active bleeding was seen in our patient. However, hemoglobin value was low. We thought that the existing chronic anemia was related to occult bleeding or malnutrition.

Diverticula related obstruction is rarely developing clinical picture. Patients develop adhesion and stricture due to experienced diverticulitis attacks. Diverticula can establish a focus for intussusception[8, 9]. These manifestation cause vomiting and distension in the patient. Large diverticula can develop pseudoobstructrion table due to dysmotility. This table shows clinical signs in the patient including mechanical intestinal obstruction[9]. Our patient had the symptoms of intestinal obstruction. No pathology that will explain intestinal obstruction could be found during the operation. Although there was no obstruction in distal of the jejunum, dilatation was observed in all areas where diverticula were localized. This supported pseudoobstruction. Resection was not considered in the patient with poor general status. No problem was found at postoperative follow up.

Conclusion

In conclusion; jejunal diverticula may be encountered with a different clinical picture. It should be kept in mind that there may be pseudoobstruction table without mechanical intesti-

nal obstruction in patients with intestinal obstruction table due to jejunal diverticula. More nonoperative approaches should be attempted in patients preoperatively diagnosed with jejunal diverticula.

Conflict of Interest Statement: The authors declare no conflict of interest.

Financial Disclosure: The authors declared that this study has received no financial support.

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Case Report

Journal of Emergency Medicine Case Reports

Use of REBOA As an Adjunct to The Conventional Femoral Cut-Down For Bleeding Control During Veno-Arterial ECMO Decannulation

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Abstract

Introduction: One of the potential complications of Extracorporeal Life Support is the arterial bleeding during decannulation. We present a case that describes the use of REBOA for hemorrhage control during femoral decannulation.

Case report: 42-old-male who was suffering from ARDS and cardiogenic shock was admitted to the Intensive Care Unit and Veno-arterial (VA) ECMO was commenced. On day 4, his cardiac performance improved and we decided that there was no longer a need for cardiac support with ECMO. Thus, a conversion from VA to Veno-venous (VV) ECMO was planned. An additional venous cannula was placed and the arterial cannula in the femoral artery was withdrawn and the femoral artery wall was repaired just after a Zone III total REBOA was achieved from the contralateral femoral artery. There were not any significant changes in blood pressure, heart rate, and lactate levels before and after the procedure.

Conclusion: REBOA might be a useful tool in the hands of non-vascular surgeons to avoid unintended bleeding during the decannulation of a large-bore arterial cannulae.

Introduction

Extracorporeal Life Support is a complex modality requiring cannulation of major arteries of the body such as the femoral artery with large bore cannulae, usually 15-25 Fr. Also withdrawing these arterial cannulae when ECLS is no longer needed might be a challenging process because of the potential arterial bleeding following the decannulation. In addition, a "bloodless area" at least for a short time is mandatory for repairing the femoral arterial wall. We present a case in which REBOA was used for temporary bleeding control during the decannulation of a 21 Fr femoral arterial cannula and femoral arterial wall repair in addition to a conventional femoral cut-down.

Case Report

A 42-year-old male patient was admitted to the intensive care unit with acute respiratory distress syndrome (ARDS) associated with cardiogenic shock in whom the return of spontaneous circulation (ROSC) was achieved after conventional cardiopulmonary resuscitation for 8 minutes. A veno-arterial ECMO with a 21 Fr arterial in the common femoral artery and a 7 Fr backflow cannula in the superficial femoral artery was then initiated regarding the acute heart failure (Left ventricle ejection fraction: 20%) unresponsive to fluid and positive inotropic agent infusions. On the 4th day of the follow up the native cardiac functions of the patient improved with LVEF of 60% in bedside transthoracic echocardiographic re-evaluation, and the positive inotropic agents were no longer needed. Due to persistent respiratory failure and increased native cardiac performance, we have planned to convert VA-ECMO to VV-ECMO. An additional infusion cannula was placed through the right internal jugular vein, and a veno-arterio-venous (VAV) ECMO was established. The femoral infusion cannula which was already in place for VA ECMO was then clamped. Following the confirmation of the patient did not hemodynamically deteriorate for one hour, the right femoral arterial cannula was planned to be withdrawn.

A femoral cut-down was performed and the common femoral artery and its branches superficial and deep femoral arteries were secured proximally and distally (Figure-1). The cannula was slightly withdrawn for about 10 cm in a

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Received: 02.06.2020 • **Accepted:** 03.10.2020
DOI: 10.33706/jemcr.747023

stepwise fashion to facilitate a safe passage of a 7 Fr Fogarty catheter (Edwards Lifesciences, Irvine, CA, USA) which was placed in the contralateral common femoral artery through a 7 Fr introducer sheath (Avanti + Sheath introducer; Cordis, California, USA) to perform a complete aortic balloon occlusion in Zone 3 (Figure- 2). Following the inflation of the balloon and the detection of the loss of the pulse in ipsilateral dorsalis pedis artery, the ECMO cannula was withdrawn without significant bleeding associated and the femoral artery was repaired with a 3-0 prolene suture. The total time of the balloon occlusion was 4 minutes. Minimally oozing following the deflation of the balloon from the suture site was successfully managed with compression with gauze (Ankaferd Blood Stopper; And Ilaç, Istanbul, Turkey). The popliteal, anterior tibial and dorsalis pedis arterial pulses were bilaterally palpable and there was no sign of ischemia in both legs. The cut down layers were closed without placing a drain. The backflow and the contralateral 7 Fr sheaths were also withdrawn, and the bleeding was managed with simple compression with gauzes. There were not any significant changes in vital signs of the patient during and after the procedure and the patient did not need any blood product and / or positive inotropic agents. Also, lactate levels remained stable even 1 hour after the completion of the procedure (2,5 mmol/L prior to and, 2,7 mmol/L 1 hour after the procedure).

Figure 1: Femoral cut down and securing the branches of the common femoral artery

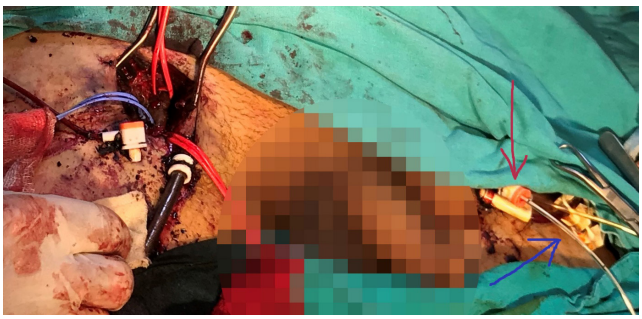

Figure 2: REBOA in place after the decannulation of the femoral arterial cannula. (Red arrow: 7 Fr arterial sheath; Blue arrow: Fogarty balloon embolectomy catheter)

Discussion

REBOA has become an alternative to emergency thoracotomy and cross-clamping of the aorta in patients with massive hemorrhage, in addition to its use in bleedings originated from non-compressible areas such as bleeding due to pelvic fractures[1]. Considering these developments, the non-traumatic use of REBOA is also increasing. In the literature, there are several case reports showed that massive vaginal hemorrhage[2] or postpartum hemorrhage can be controlled with REBOA[3]. In addition, it is used successfully in repairing a ruptured splenic artery aneurysm[4]. In this case report, we wanted to describe a different use of REBOA as well.

The extracorporeal life support (ECLS) system is a life-saving treatment option for acute cardiopulmonary insufficiency[5]. Since the goal of ECLS is to perform extracorporeal circulation as quickly as possible, the cannulation of the femoral artery (FA) and the femoral vein (FV) is the most common cannulation strategy. Therefore, the groin is the most common cannulation site in adults[5,6]. In VA-ECMO, blood is drawn from the vena cava inferior via venous cannulation and following oxygenation in the membrane oxygenator, re-infused into the aorta, thus providing complete cardiac and pulmonary support. Large bore cannulae are required to ensure high blood flow during ECMO. Three techniques are commonly used for cannulation: percutaneous (Seldinger), semi-open and open surgical cut-down[7]. In our patient, the cannulation was performed by surgical cut-down. Available options for vascular access site management after successful weaning of ECMO include manual compression followed by using a mechanical femoral compression system[8], and surgical cut-down technique, which is considered standard practice in cannulation for VA-ECMO[8,9] There are also studies in the literature that suture-based vascular closure devices might be useful[10]. Surgical approach and repair are often necessary for cannulas placed via surgical cut-down[7]. Therefore, the decannulation in our patient was performed by the femoral cut-down. Bleeding is one of the most common complications at this stage. The decannulation techniques described so far are neither standardized nor sufficiently proven to be used in large VA-ECMO patient groups[10]. We believe that uncomplicated vascular repair by surgical cut-down with the aid of REBOA is important in terms of both the use of REBOA in a non-traumatic indication and a new suggestion for VA-ECMO decannulation techniques. However, prospective randomized controlled trials are needed to decide which technique is the best for which patient.

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Case Report

Journal of Emergency Medicine Case Reports

Corona Virus Disease 2019 (COVID-19) presenting as carbon monoxide poisoning

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Abstract

Introduction: Corona Virus Disease 2019 (COVID-19) is a pandemic disease in the World and some patients are asymptomatic. In this report, we present an asymptomatic COVID -19 patient detected as a result of carbon monoxide poisoning.

Case report: A 76-year-old male patient was admitted to the emergency department with the complaint of syncope cause of carbon monoxide poisoning. In further investigations, COVID-19 was detected in the patient.

Conclusion: We describe the first COVID-19 patient presenting as carbon monoxide poisoning in literature.

Keywords: COVID-19, Carbon monoxide poisoning

Introduction

The pandemic of the 2019 novel coronavirus disease (COVID-19) infections that developed in Wuhan, Hubei Province has spread to other country in the world[1]. It is now evident that most cases of COVID-19 disease develop mild respiratory and constitutional symptoms, while some cases are asymptomatic[2]. In this case report, we aimed to present the asymptomatic COVID -19 patient detected as a result of cabonmonoxide poisoning.

Case Report

A 76-year-old male patient was admitted to the emergency department with the complaint of syncope. In detailed anamnesis taken from the relatives of the patient, the patient who lived alone in the house, fell asleep after burning the stove in the evening and was found unconscious by relatives in the morning. He had hypertension in his medical history. The patient's blood pressure, pulse, fever and respiratory rate were 160/110 mmHg, 100/min, 36.4 ^{0}C and 26/min. On his physical examination patient's consciousness was confusion. Glaskow Coma Score (GCS) of patient was 13. Oxygen saturation was evaluated as 80%. Patient was taken into security circle. Nasal oxygen therapy was started at 4L/min per hour. Then the causes of syncope of the patient was began to be investigated. There was no pathology except sinus tachycardia at his electrocardiography. Rectal interference was normal stool. Complete lood count (CBC), glucose, aspartate aminotransferase (AST), alanine aminotransferase (ALT), Troponin, blood gas, d-dimer, electrolytes, urea, creatinine levels were investigated. From the patient's blood tests, we found that glucoase value was 102 mg/dl, pH was 7,42 and carboxyhemoglobilin (COHb) level was 36%. The other blood tests were normal. The patient was diagnosed with carbon monoxide poisoning. Brain tomography was evaluated as normal. Because of the low saturation of the patient, chest tomography was also performed. In the chest tomography scan, we detected ground-glass opacities (GGOs) and incomplete consolidations in pleural neighborhoods, showing multifocal peripheral location in both lungs (Figure1-2). On top of that, the patient was isolated in our covid service. Oxygen therapy was continued with a mask. Nasopharyngeal swab was taken from the patient for the diagnosis of COVID-19. COVID-19 was positive as a result of reverse-transcriptase polymerase chain reaction (RT-PCR). On the second day of CO poisoning treatment, the patient's COHb values decreased to 0.1%. Hyperbaric therapy was not applied to the patient because of suspected COVID-19. The patient was taken Favipiravir and Plaquanil

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Received: 28.06.20 • **Accepted:** 03.10.20
DOI: 10.33706/jemcr.759423

DOI: 10.33706/jemcr.759423
Journal of Emergency Medicine Case Reports 2020; 11(3): 77-78

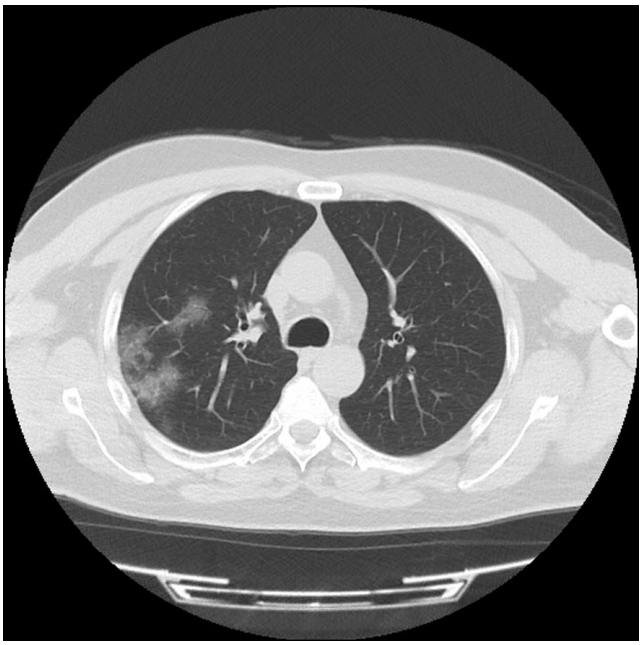

Figure 1: ??

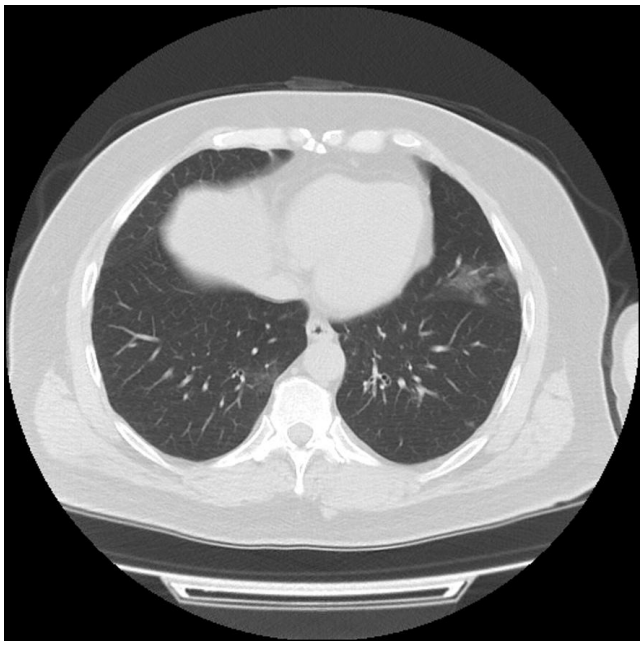

Figure 2: ??

treatment for COVID-19 and after 5 days, the patient's saturation was 98%. GCS became 15. The patient, who did not have any complaints, was discharged with suggestions of isolation at home.

Discussion

Diseases accompanied by asymptomatic cases of COVID-19 are occasionally reported in the literature[3]. In this report, we present a case that does not have covid-19 symptoms and admitted to the emergency department due to carbon monoxide poisoning was diagnosed with COVID-19.

In the literature, the tomography and chest radiography typical findings of COVID-19 pneumonia are discussed radiologically. CT findings are typically characterized by GGOs or bilateral pulmonary consolidations in multiple lobular and subsegmental areas[4]. In our case, there were no symptoms of COVID-19, but radiologically on the patienet's chest tomography we detected GGOs and incomplete consolidations in pleural neighborhoods, showing multifocal peripheral location in both lungs. These findings helped us to diagnose COVID-19.

COVID-19 cases usually apply to the emergency department with symptoms such as fever, cough, dyspnea. However, most cases are asymptomatic carriers and undetectable these cases[5]. In our case, he was asymptomatic carrier until he was not exposed to carbonmonix poisonig and COVID-19 was not detected.

Conclusion

We describe the first COVID-19 patient presenting as carbonmonoxide poisoning in literature. It should be kept in mind that patients presenting to the emergency department with any symptoms may have asymptomatic COVID-19 patients.

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Case Report

Journal of Emergency Medicine Case Reports

Incidental Thoracic Spinal Angiolipoma in a Young Woman Presenting with Trauma

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Abstract

Introduction: Spinal angiolipoma is a rare clinical entity and usually presents with clinical signs and symptoms of spinal cord compression. It has two types; non-infiltrating and infiltrating. It is more common in females at the ages of 40-50 and at the mid-thoracic levels.

Case report: We discussed an incidentally diagnosed non-infiltrating angiolipoma case in the lower thoracic spine level in a 33-year-old female patient who presented to the emergency department with severe low back pain after trauma.

Conclusion: Spinal angiolipoma should not be forgotten in the differential diagnoses of the patients presenting with trauma.

Keywords: Spine, Trauma, Back pain, Angiolipoma

Introduction

The first diagnosis of spinal angiolipoma (SAL) was described in 1690 during the autopsy of a 16-year-old boy[1]. Less than 200 cases were reported until 2020[2]. Although there are some SAL reports in the lower cervical, thoracic and lumbar spine, they tend to settle, especially in the mid-thoracic region[3,4]. They present with gradually increasing symptoms in accordance with the degree of cord compression, unless intratumoral bleeding or venous thrombosis occurs[4]. They consist of varying degrees of mature fat cells and abnormal capillary sinusoidal, venous, or arterial vascular elements[5]. The majority of SAL have favorable prognoses after surgical resection[4].

In this report, we are presenting a spinal angiolipoma located at the low-thoracic region that was diagnosed after admitted to the emergency department due to trauma.

Case report

A 33-year-old female patient was brought to the emergency department due to severe back and right hip pain after falling stairs. There were severe tenderness and pain on the lower thoracic and upper lumbar regions in the physical examination. The neurological examination had no pathology. Computerized tomography (CT) of thoracolumbar region revealed multiple fractures of right lumbar vertebra transverse processes (L1, 2 and 3). The spinal canal and vertebral corpuses were evaluated as normal (Figure 1). The patient stated that her pain did not relieve despite the respectively administered intravenous paracetamol, dexketoprofen, and tramadol during her follow-up in the emergency department. The attending physician planned thoracolumbar magnetic resonance imaging (MRI) and neurosurgery consultation for possible acute disc pathologies. Although MRI showed no disc-related acute pathology, the patient was hospitalized to the ward by the neurosurgery department for pain monitorization and further investigation.

When re-evaluating CT and MRI images, a suspicious lesion on the posterior level of T11-12 was seen in the sagittal sections, and we planned contrast-enhanced MRI. An extradural lesion with diffuse contrast-enhancing at the same level was observed (Figure 2). Surgical intervention for diagnostic and therapeutic purposes was planned. The lesion was reached by laminectomy. A bright, dark red colored lesion was observed, which seemed to emerge from the left neural foramen and erode the neural foramen partially. The lesion was easily peeled off from the dura (Figure 3). The histopathologic examination confirmed the diagnosis of SAL.

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Received: 29.06.20 • **Accepted:** 13.10.20
DOI: 10.33706/jemcr.759924

DOI: 10.33706/jemcr.759924
Journal of Emergency Medicine Case Reports 2020; 11(3): 79-81

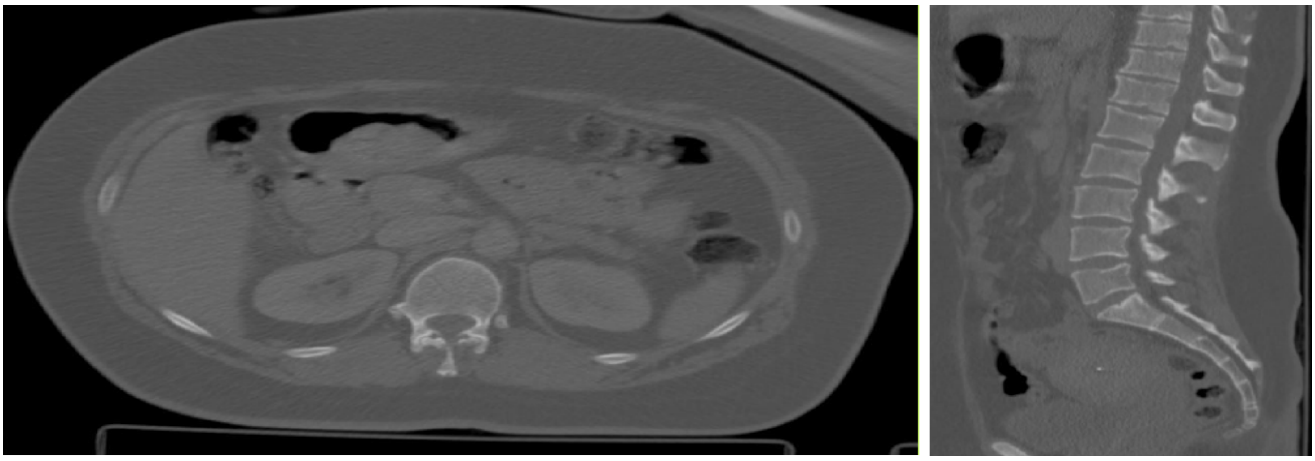

Figure 1: Computerized tomography of thoracolumbar region of the patient with transverse and sagittal views on the day of emergency department admission showed no pathology associated with the spinal canal and/or vertebral corpuses.

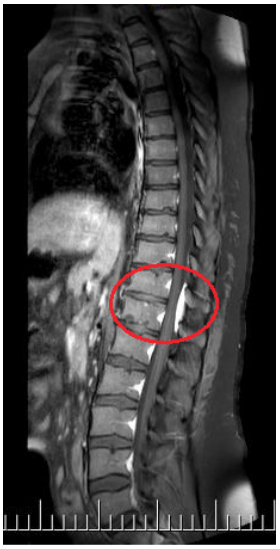

Figure 2: Magnetic resonance imaging with contrast of thoracolumbar region of the patient on the next day of emergency department admission showing a suspicious extradural lesion with diffuse contrast-enhancing on the posterior level of T11-12.

Discussion

SAL is a rare but well-defined clinical pathology that accounts for approximately 2-3% of epidural spinal tumors[6]. It is more common in females and usually seen in the fourth and fifth decades. However, several cases have been reported in the pediatric age group as well. SAL is categorized into two: non-infiltrating and infiltrating. The non-infiltrating type, which is encapsulated has good prognosis and located in the posterior or posterolateral of the spinal canal. The capsule-free infiltrating type which is found in intramedullary or intervertebral locations has poor prognosis[6]. Our case was a female patient in her third decade, and the tumor location was at the epidural area, posterior to the spinal canal.

The clinical picture of SAL presents with spinal cord compression-related symptoms. The common findings are progressive paraparesis, low back pain without radiculopathy, sensory changes in the lower extremity, and hyperreflexia[7]. Spinal cord width is determinative in the initial presenta-

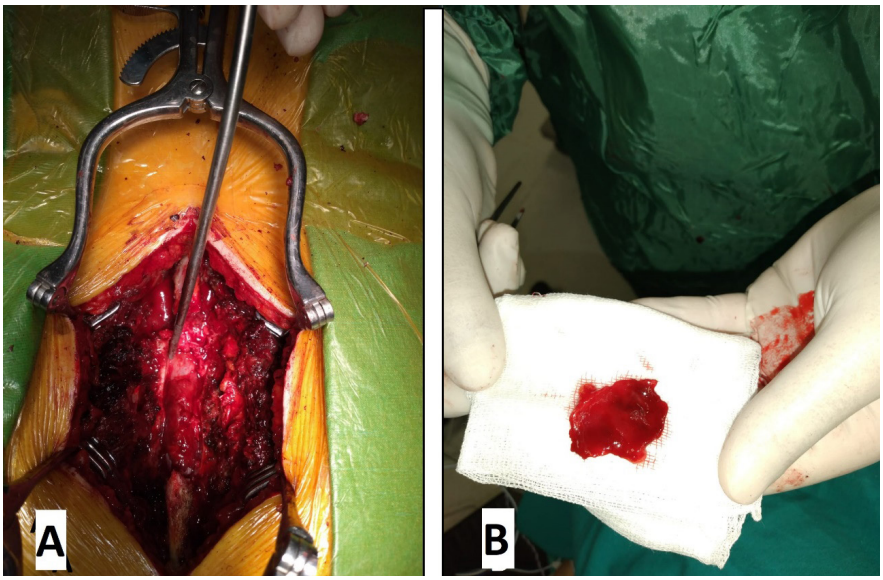

Figure 3: The appearance of the lesion after the laminectomy (A) and after peeling off from the dura.

tion of clinical findings, especially in extradural SAL cases[5]. They generally show slow progression, and the definitive diagnosis is usually made in around one year. However, the expected clinical course can be suddenly disrupted by vascular occlusion, growth, enlargement and degeneration of degenerated jab vessels, vascular stealing phenomenon, thrombosis, and bleeding into the lesion[5]. In some cases, the clinical course may be in the form of relapses as mimicking multiple sclerosis cases[5]. The medical history of our patient was insignificant except for occasional low back pain. She admitted to the emergency department with severe low back pain after trauma. There was no sensory defect or loss of strength in the neurological examination, but only severe pain on her lower back. There was no compatible traumatic finding with her persistent and refractory back pain in the tomography sections of the patient. Lumbar MRI was necessitated for the differential diagnosis of severe pain. The extradural lesion was detected during the contrast-enhanced MRI performed on the next day. The intraoperative view of the lesion was hemorrhagic. According to all, we conclude that hemorrhage was occurred due to trauma, and the pain was exacerbated by it. The appearance of the lesion on the control imaging studies, the decrease of pain in the post-operative period, and even disappearance of pain entirely after the next few days support our hypothesis.

The exact mechanism of SAL development is unknown, but the presence of various predisposing factors has been proposed like obesity, weight gain during pregnancy, and corticosteroid use[5]. Consistently, our patient was overweighted with a body mass index (BMI) of 32.5. The current data in the literature cannot point out clearly on this issue, so further investigations are needed to explain the relationship between BMI and SAL.

MRI is accepted as the gold standard in the diagnosis of SAL. It appears isointense on non-contrast T1-weighted images and hyperintense on T2-weighted images compared to other spinal tumors or epidural lipomatosis[6]. Although homogeneous enhancement on contrast-enhanced T1-weighted sections was reported in the literature, our case did not[5]. The differential diagnosis should contain lipoma, hemangioma, malignant lymphoma, and nerve sheath tumors[6]. Non-infiltrating type of SALs can be easily removed by laminectomy, and post-operative results after surgery are promising, as in our case.

Conclusion

Although SAL is rare, it is useful to keep in mind in the differential diagnoses of the patients presenting with severe back pain after trauma. Advanced radiological techniques such as MRI and contrast-enhanced techniques should be performed even if no compatible pathology on tomography, which could not explain the clinical presentation of the patient.

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Case Report

Journal of Emergency Medicine Case Reports

Pulmonary Langerhans Cell Histiocytosis X Incidentally Diagnosed in a Non-Smoker Because Of Simultaneous Bilateral Spontaneous Pneumothorax

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Abstract

Introduction: Pulmonary Langerhans cell histiocytosis X (PLCHx) is a rare interstitial lung disease of unknown aetiology and occurs mostly in young smokers. Lung involvement alone accounts for >85% of the cases; other organs may be involved too (5%–15%).

Case report: A 19-year-old boy was presented to the emergency service with sudden-onset, sharp, stabbing chest pain and dyspnoea. The patient had no history of smoking. His medical and family histories were negative for any disease. Thoracic radiography showed increased reticulonodular density with a near-total bilateral pneumothorax at the left and partially at the right. Hence, bilateral tube thoracostomy was performed. High-resolution computed tomography (HRCT) showed pronounced thin-walled cystic structures smaller than 10 mm and small millimetre-sized nodules in the upper and middle zones. Extrapulmonary Langerhans cell histiocytosis was excluded based on the radiological and laboratory findings. Based on the clinical and tomography findings, diagnosis of PLCHx was made.

Conclusion: Our case highlights that PLCHx should be suspected in non-smokers. Further studies elucidating the aetiology of PLCHx are needed.

Keywords: Histiocytosis X, spontaneous pneumothorax, interstitial lung disease.

Introduction

Pulmonary Langerhans cell histiocytosis X (PLCHx) is a rare interstitial lung disease of unknown aetiology and occurs mostly in young smokers. It is characterised by abnormal accumulation of Langerhans-type histiocytes in the lung parenchyma[1,2]. An epidemiological hallmark of PLCHx is that it almost exclusively occurs in patients with a smoking history of >20 cigarettes per day. No other epidemiologic feature has been identified so far[3]. Herein, we report a case of PLCHx in a 19-year-old non-smoker. PLCHx was diagnosed incidentally during the treatment for simultaneous bilateral pneumothorax.

Case Report

A 19-year-old boy was presented to the emergency service with sudden-onset, sharp, stabbing chest pain and dyspnoea. Physical examination revealed decreased respiratory sounds bilaterally, with louder sound on the left. The arterial blood pressure was 130/85 mm Hg, pulse was 115 bpm and fingertip oxygen saturation was 90%. The patient had no history of smoking. His medical and family histories were negative for any disease. Laboratory examination was negative for any pathology. Thoracic radiography showed increased reticulonodular density with a near-total bilateral pneumothorax at the left and partially at the right (Figure 1). Hence, bilateral tube thoracostomy was performed. Postoperative chest radiography showed improved lung expansion except for minimal pneumothorax detected on the left (Figure 2). High-resolution computed tomography (HRCT) showed pronounced thin-walled cystic structures smaller than 10 mm and small millimetre-sized nodules in the upper and middle zones (Figure 3). The right chest tube was removed on the 5th day and the left tube on the 12th day. Extrapulmonary Langerhans cell histiocytosis was excluded based on the radiological and laboratory findings. Because thoracotomy or thoracoscopy was not conducted, lung biopsy could not be performed. Based on the clinical and tomography findings, diagnosis of PLCHx was made. The patient was advised to avoid inhaling cigarette smoke. Steroid treatment was not started because he was clinically and medically stable following the pneumothorax treatment. He is on regular follow-up and is stable.

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Received: 05.07.2020 • **Accepted:** 26.10.20
DOI: 10.33706/jemcr.763236

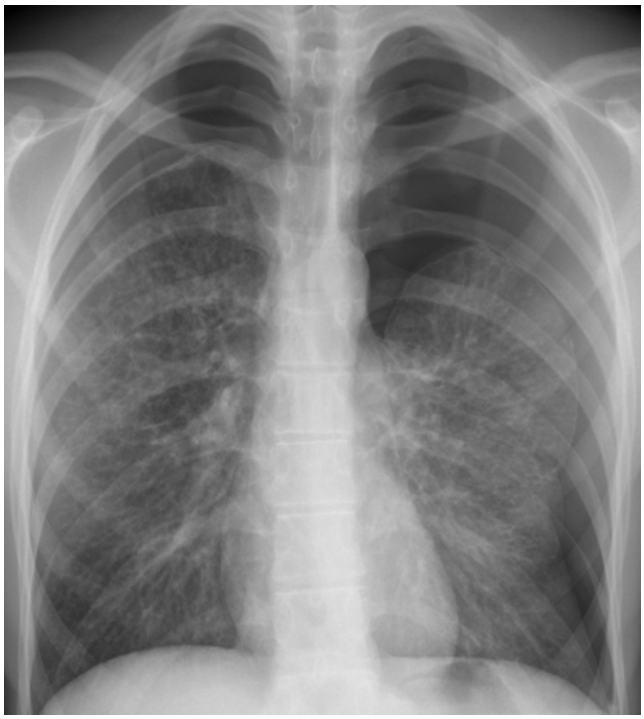

Figure 1: Chest radiograph with bilateral pneumothorax.

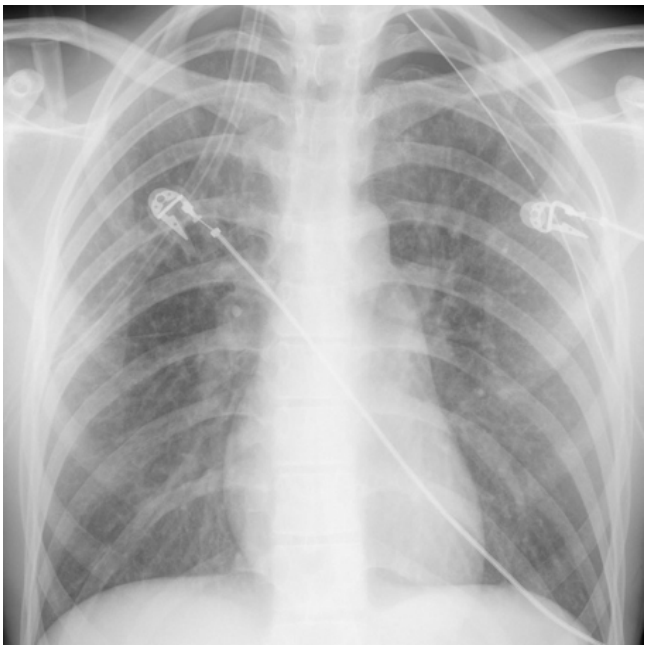

Figure 2: Bilateral lung re-expansion after bilateral drainage tube insertion

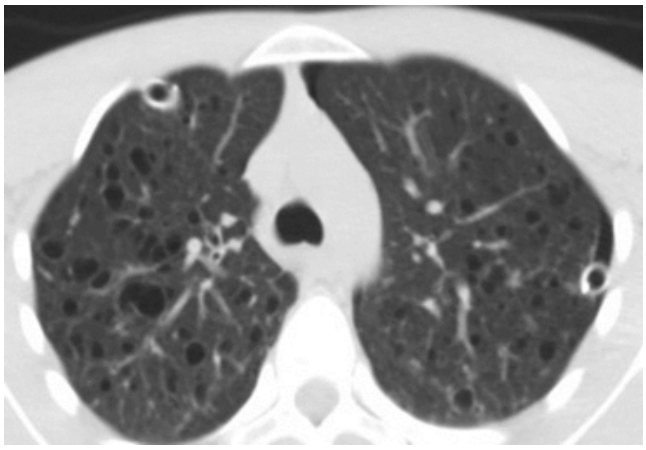

Figure 3: Chest computed tomography scan showing pronounced thin-walled cystic structures smaller than 10 mm in the upper and middle zones

Discussion

Langerhans cell histiocytosis is a group of disorders characterised by infiltration of a large number of Langerhans cells into tissues to form granulomas and is associated with a wide range of clinical features and outcomes. PLCHx is the pulmonary sub-form of this disorder; it is also known as pulmonary eosinophilic granuloma, pulmonary Langerhans cell granulomatosis and pulmonary histiocytosis[3,4]. Lung involvement alone accounts for >85% of the cases; other organs may be involved too (5%–15%)[2]. PLCHx occurs mostly in young adults; however, its actual incidence and prevalence are unknown. Almost all patients with PLCHx have a history of smoking; this indicates the critical role of smoking in its pathogenesis. However, no reliable epidemiological studies exist[2-4]. Our patient only presented with lung involvement, had never smoked and was not exposed to cigarette smoke. These findings imply that genetic and environmental factors could also be involved in the aetiopathogenesis of PLCHx. Therefore, further studies on this subject are required.

PLCHx is asymptomatic in approximately 25% of the cases and is mostly diagnosed incidentally during thorax radiography. Most patients present with cough, dyspnoea and laboured breathing. Recurrent unilateral or bilateral pneumothorax is seen in 10%–20% of the cases. Pneumothorax is resistant to air leak, and offering treatment options ranging from video thoracoscopy to pleurodesis might be required[5-7]. While our patient had no previous respiratory symptoms such as cough and dyspnoea, he presented with simultaneous bilateral pneumothorax. Pneumothorax was treated with tube thoracostomy, and no further surgical intervention was required.

PLCHx has characteristic findings on thorax radiography that help diagnosis, including irregularly limited or satellite nodules of 2–10 mm diameter, reticulonodular opacities, cysts and honeycomb appearance in the upper and middle zones and costophrenic angles. On HRCT imaging, thin-walled cysts sized <10 mm and nodules sized <5 mm are seen mostly in the upper zones. Cavitation can be observed in these nodules showing centrilobular and peribronchiolar distribution[1,2,8]. Thoracic radiography, HRCT and clinical findings are deemed sufficient for diagnosis when patients choose not to undergo surgical procedures and tissue samples cannot be obtained. The differential diagnoses basis tomography findings include lymphangioleiomyomatosis,

DOI: 10.33706/jemcr.763236
Journal of Emergency Medicine Case Reports 2020; 11(3): 82-84

tuberous sclerosis, hypersensitivity pneumonia, sarcoidosis and end-stage idiopathic pulmonary fibrosis.

The main treatment objective is cessation of smoking. Smoking cessation has been shown to stabilise the disease symptomatically, radiologically and physiologically[2-4]. However, reports of no progression up to 12 years in patients who continue smoking exist[9]. Similar to our case, PLCHx has been diagnosed in patients who never smoked and had no exposure to cigarette smoke. Reports have suggested that patients with predominant nodular lesions may respond to corticosteroid therapy. Cytotoxic drugs such as vinblastine, methotrexate, cyclophosphamide and etoposide can only be used in patients with multisystem involvement or steroid-resistant progressive disease[2-4].

Conclusion

Our case highlights that PLCHx should be suspected in non-smokers. Further studies elucidating the aetiology of PLCHx are needed.

Acknowledgments

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: This was not an industry supported study. The authors declare that this study has received no financial support.

All authors contributed to the design and implementation of the research, to the analysis of the results and to the writing of the manuscript. All authors discussed the results and contributed to the final manuscript.

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Case Report

Journal of Emergency Medicine Case Reports

Importance of Clinical Suspicion in Rapid Diagnostic Test Negativity in Malaria: Two Case Reports

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Abstract

Introduction: Malaria is a life-threatening disease caused by infection with Plasmodium parasites. Rapid diagnostic tests (RDTs) have been used for the diagnosis of malaria without special equipment by unskilled personnel over the last 15 years. The treatment should only be given after the clinical diagnosis confirmed by RDT or microscopy. RDTs' specificity and sensitivity have been reported as >95% by the World Health Organization - Foundation for Initiative New Diagnostics (WHO-FIND).

Case report: A 30-years-old male and a 23-years-old female presented to our emergency department with fever and history of a visit to a malaria-endemic country. Plasmodium trophozoites were seen in the blood smear samples via light microscopy. However, RDTs were negative. The patients were treated according to their pathogens.

Conclusion: Rarely, RDT might result in a false negative in the diagnosis of malaria. People travelling to endemic areas should be closely monitored. Emergency department physicians should not neglect microscopy which is the gold standard for diagnosis of malaria.

Keywords: Clinical Microbiology; Emergency Departments; Infectious Diseases; Parasitology; Plasmodium

Introduction

Malaria is one of the most common reasons to seek medical care and associated with considerable morbidity and mortality in many sub-Saharan countries. Africa has the greatest malaria burden, with 91% of malaria-related deaths worldwide [1]. *Plasmodium* parasites cause acute febrile diseases, ranging from 6 to 30 days after transmission. Among them, *Plasmodium. falciparum* is the most severe form of malaria and is responsible for more than 80% of the deaths worldwide[2]. Currently, the routinely used parasitological tests are microscopy and rapid diagnostic test (RDT). Light microscopy with the application of Giemsa stain is the "gold standard" for laboratory diagnosis of malaria, however, during the last World Health Organization - Foundation for Initiative New Diagnostics (WHO-FIND) reported high sensitivity (>95%) regarding *P. falciparum* detection of RDTs[3, 4].

In this paper, we describe two cases of patients with *P. falciparum* and *P. vivax* infections found on light microscopy, but for whom the RDTs were negative.

Case 1

A 30-years-old male presented to the Emergency Department (ED) of our university hospital in Turkey with a history of fever, productive cough and sore throat persisting for two days. The patient stated that his body temperature suddenly escalates during sleep, his fever had been more apparent at night. There was no remarkable illness or medication in the patient's medical history. His physical examination showed as follows: a body temperature of 38.7 ºC, blood pressure of 110/63 mmHg, heart rate of 92 beats per minute, respiratory rate of 17 per minute, and pulse oximetry of 99% in room air, severe oropharyngeal hyperaemia, and postnasal drip. Laboratory findings were as follows: haemoglobin, 15,9 g/dL; white blood cell count, 4,6 × 10^3 /µL; platelet count, 17,1 × 10^3 /µL; blood urea nitrogen, 18 mg/dL; creatinine, 1,01 mg/dL; and the chest radiograph was normal. He lives in Turkey where is not one of the malaria-endemic countries. It was learned that he had been in a malaria-endemic country twice in the last month for 2 days but had no insect bite history. First, an RDT was planned for malaria screen-

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Received: 05.08.20 • **Accepted:** 29.09.20

DOI: 10.33706/jemcr.777328

DOI: 10.33706/jemcr.777328
Journal of Emergency Medicine Case Reports 2020; 11(3): 85-87

ing. It was negative (Figure 1). Considering the findings above, he was evaluated as an upper airway infection and discharged with a prescription. The next day he presented to the ED with fever (39.1 ºC) and diffuse muscular pain. Physical examination and laboratory tests were similar with first results except for platelet count (Platelet count: 5,3 × 10^4 /µL). Because of the development of thrombocytopenia, an infectious diseases physician was consulted. RDT was repeated and Giemsa painted thick and thin smears were ordered additionally. RDT resulted before microscopy and it was negative again. In contrast with RDT, *P. falciparum* was detected under microscopy (Figure 2) and the patient was started with the treatment immediately. The patient was started on treatment with artemether-lumefantrine combination therapy for three days, according to WHO recommendations (Table 1). The patient fully recovered after this treatment. Clinical and laboratory examination of the patient was found as normal after 15 days of discharge.

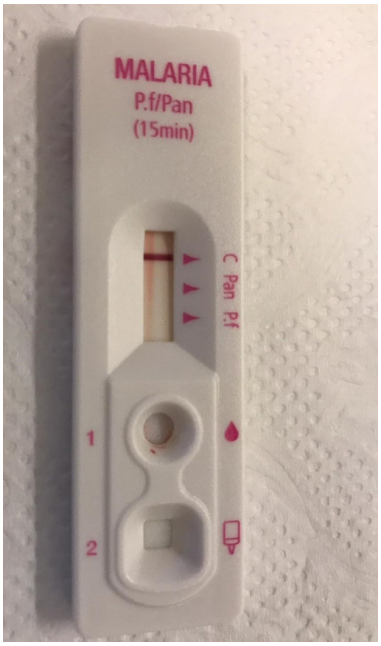

Figure 1: Negative rapid diagnostic test (only the control line is seen).

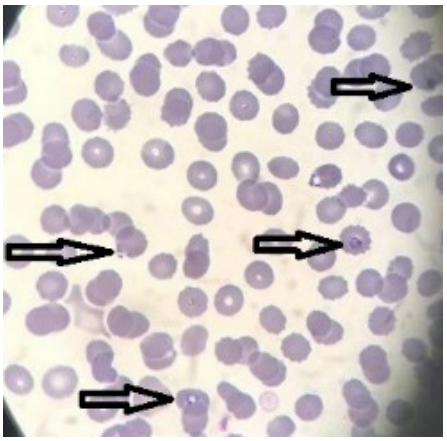

Figure 2: Blood smear microscopy shows Plasmodium falciparum parasites (arrows) infecting the patient's red blood cells (Giemsa stain, x100 magnification).

Table 1. Artemether-lumefantrine based combination therapy doses (tb: tablet)

Patient Weight (kg)	First Dose	8th Hour	24th Hour	36th Hour	48th Hour
<15	1 tb	1 tb	1 tb	1 tb	1 tb
15 – 24	2 tb	2 tb	2 tb	2 tb	2 tb
25 – 34	3 tb	3 tb	3 tb	3 tb	3 tb
>34	4 tb	4 tb	4 tb	4 tb	4 tb

Case 2

A 23-years-old female presented to the ED of our university hospital in Turkey with an insect bite lesion and a history of fever for the past three days. She stated that she was in Africa, an endemic country for malaria, for 48 hours, 18 days before her application. It was learnt that she has been experiencing periodic fevers. Her physical examination showed as follows: a body temperature of 38,6 ºC, blood pressure of 100/60 mmHg, heart rate of 90 beats per minute, respiratory rate of 20 per minute, and pulse oximetry of 100% in room air. Laboratory findings were as follows: haemoglobin, 13,2 g/dL; white blood cell count, 3,85 × 10^3 /µL; platelet count, 99 × 10^3 /µL; blood urea nitrogen, 3 mg/dL; creatinine, 0,55 mg/dL; and the chest radiograph was normal. Abdominal ultrasonography did not detect hepatosplenomegaly. RDT and microscopy from the blood sample obtained through antecubital cannulation planned for malaria screening and resulted as negative. The patient left the hospital to travel and was strongly advised to apply to an infectious diseases specialist in another city. She presented to another university hospital on her 4th day of fever. When infectious diseases department of the university was contacted, it was reported that the microscopy test from the blood sample obtained from patient's fingertip revealed *P. vivax,* but RDT was still negative. The patient was treated with artemether-lumefantrine based combination therapy were used for 3 days and primaquine for 14 days. Patient full recovered after this treatment and was discharged from the hospital. Clinical and laboratory examination of the patient was found as normal after 15 days of discharge.

Discussion

These cases focus on two malaria diagnostics (microscopy and RDT) that are likely to have the largest impact on malaria control. In many countries sub-Saharan Africa, an electricity supply is not available in villages, thus hampering the use of microscopy for malaria diagnosis which needs a reliable electricity supply. RDTs are used as an alternative in those locations. RDTs have a specificity problem and may result in a significant number of false-positive test results

when compared with the golden standard, expert microscopy. RDTs were false-negative in our cases, which were performed on the second and third days of the fever. The false negativity of RDT may be explained by the following reasons: The first, the occurrence of deletion or mutation on the histidine-rich protein 2 gene[5]. Second, the presence of an inhibitor in patient's blood preventing the development of the control line[6]. The last, lower parasite densities may lead to the false negativity of RDT[7]. According to the literature, most false-negative results of RDTs occur at lower parasite densities. RDTs have demonstrated sensitivities approximate to 100% for the detection of *P. falciparum* at densities above 100 asexual parasites/μL or >0.002% of parasitized red blood cells[8].

Experienced microscopists, increased examination time and the number of microscopic fields examined could facilitate the identification of *Plasmodium* species correctly. However, in health facilities with a high workload, the use of microscopy alone is not recommended since it requires more time to perform than an RDT. Health workers with poor supervision or inadequate training should preferably use RDTs which are comparatively easier to use than a microscope. Each of these two parasitological tests has its strengths and weaknesses for an accurate diagnosis of malaria[4]. Therefore, WHO recommends that both tests should be performed for the diagnosis of malaria in the clinical applications.

Prompt and accurate diagnosis will not only improve malaria treatment but also possibly reduce mortality and morbidity due to other febrile illnesses, however, malaria diagnosis is the most neglected area of malaria research in 2004, accounting for less than 0.25% ($700.000) of the United States of America's $323 million investment in research and development[9].

In developed countries, RDTs can be useful in screening febrile returnees from endemic areas. People travelling for touristic or work-related purposes to the endemic areas must be monitored closely[10].

Conclusion

Physicians should not forget that RDTs might provide false negative results. Both microscopy and RDTs should be performed for diagnosis of malaria. Presence of fever and a history of travel to an endemic region in the last 30 days, regardless of the duration of the stay nor the existence of a bite lesion, must be considered highly suspicious for infection. Combining RDTs and microscopy holds great importance in the accurate diagnosis and treatment.

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Case Report

Journal of Emergency Medicine Case Reports

Case Report: Postoperative Parotitis and Review Of The Literature

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Abstract

Introduction: Anesthesia mumps (mumps), also called postoperative parotitis and characterized by swelling of the salivary glands after general anesthesia. Usually occurs within the first two weeks postoperatively. It is a benign and usually noninfectious complication and usually unilateral. Patients complain of painful swelling. The etiology is unknown. Postoperative parotitis is usually mild is a preventable clinical picture. In order to prevent this complication, it is recommended to prevent compression of the parotid gland or duct by providing an appropriate position. In addition to providing normal neck venous drainage, it is necessary to select anticholinergics with low antisialagogue activity in premedication and to avoid maneuvers that increase intraoral pressure.

Case report: In this case repot we presented a 52-year-old woman that developed postoperative parotitis after surgery with general anesthesia for senile cataract.

Conclusion: Postoperative parotitis is usually mild and preventable. Some pharmacological agents can be used in addition to an appropriate position to prevent this complication. Although it is generally mild, the patient should be closely monitored in terms of airway and the patient and his relatives should be informed about this clinical picture.

Keywords: Postoperative period, parotitis, anesthesia

Introduction

Anesthesia mumps (mumps), also called postoperative parotitis and characterized by swelling of the salivary glands after general anesthesia. Usually occurs within the first two weeks postoperatively[1,2]. It is a benign and usually noninfectious complication and usually unilateral. Patients complain of painful swelling. The etiology is unknown. Increased pressure in the mouth, inappropriate head and neck position of the patient, loss of muscle tone of stensen duct orifice (due to the use of muscle relaxants), accumulation of salivary gland ducts, and dirty abdominal surgeries are among the accused factors. In this study, we aimed to present the bilateral parotitis after senile cataract operation in the light of the literature

Case Report

A 52-year-old woman weighing 72 kg and 165 cm was scheduled for surgery with general anesthesia for senile cataract. In the preoperative anesthesia examination, physical examination findings and routine laboratory tests were found to be normal. The patient was evaluated as ASA I and operated under general anesthesia. The patient was inducted with 0.02 mg/kg midazolam, 2 mg/kg propofol, 1 µg/kg fentanyl and 0.6 mg/kg rocuronium. Endotracheal tube 7 was spiral orotrecheal intubated. Anesthesia was maintained with 1.5 l/min O_2 (50%), 1.5 lt/min air (50%) and 2% sevoflorane. Hemodynamic parameters of the patient were stable peroperatively. Neuromuscular block was antagonized with sugammadex 2 mg/kg at the end of the operation. Approximately four hours after the operation, redness and painful swelling developed in the bilateral parotid gland, more prominent on the right side (Figure 1) Ear and nose throat consultation was requested and the patient was diagnosed as postoperative parotitis by parotid ultrasonography. Etodolac 400 mg 2 * 1 and ceftriaxone 500 mg 2 * 1 were administered for 10 days. The patient's complaints began to regress from day 2 postoperatively and healed completely on day 10.

Discussion

The parotid gland is more prone to inflammation than other large salivary glands due to reasons such as the anatomical

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Received: 19.08.2020 • **Accepted:** 15.10.20

DOI: 10.33706/jemcr.782379

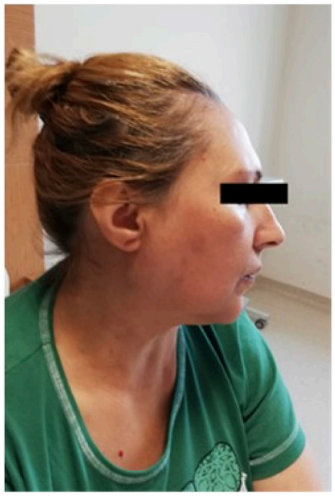

Figure 1: Redness and painful swelling developed inn the bilateral parotid gland, more prominent on the right side

features of the buccal orifice and the absence of mucin secretion in the secretion of the parotid gland.

Different mechanisms are considered in postoperative parotitis formation such as various types of metastatic, ascending and traumatic pathways[3].

Surgical procedures lasting more than 5 hours, surgical procedures performed with prone position, sitting position or extension of the head have been reported as predisposing factors[1,4,5]. Pneumoparotitis is another accused factor and is characterized by painless swelling. Pneumoparotitis is also observed in crepitation. In obese and short, thick necked patients, perfusion may be affected as a result of compression of position-related vessels. Developing ischemic sialadenitis usually presents with unilateral painful swelling[1]. Straining has also been reported to play a role in the development of postoperative parotitis[6]. In our case, the patient was evaluated as mallampati[2]. There was no intubation difficulty. There were no problems during operation and during wake-up.

Postoperative parotitis can be detected after each operation. However, some studies have reported an association with infected cases such as appendicitis, peritonitis, perforated gastric ulcer perforation[7].

Mouth dryness after anesthesia may be another predisposing factor for postoperative parotitis. Preoperative fasting, dehydration, mechanical obstruction of the head position and retention of secretion may result in postoperative parotitis[4,6,8]. Morphine and atropine however, it is one of the other factors accused especially in bilateral parotitis. Other accused mechanisms include depolarizing neuromuscular agents increasing intraoral pressure by fasciculation and reducing salivary secretion of fentanyl and sevoflurane[1,4]. In our case, fentanyl was applied during induction. Maintenance with sevoflurane was achieved. Postoperative parotitis may present as a painful condition that may require surgical excision of a mild painful swelling[1]. Generally, airway swelling is not affected, but in the literature, cases of airway obstruction have been reported[4,9]. In our case, bilateral swelling was observed on the right side. There was no shortage of airway. Response to symptomatic treatment was achieved and surgical intervention was not required. In order to prevent the development of postoperative parotitis, it is recommended to prevent compression of the parotid gland or duct by providing an appropriate position. In addition to providing normal neck venous drainage, it is necessary to choose anticholinergics with low antisialagog activity in premedication and avoid maneuvers that increase intraoral pressure (such as patient pushing, pressurized mask ventilation).

Conclusion

Postoperative parotitis is usually mild and preventable. Some pharmacological agents can be used inaddition to an appropriate position to prevent this complication. Although it is generally mild, the patient should be closely monitored in terms of airway and the patient and his relatives should be informed about this clinical picture.

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Case Report

Journal of Emergency Medicine Case Reports

Unexpected Fatal Empyema in a Previously Healthy Woman

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Abstract

Streptoccoccus pyogenes is gram + aerob coccus and it is described as group A Streptococcus (GAS) according to the Lancefield classification. GAS may cause pneumonia and pleural effusion rarely.Herein we reported a 43 years old previously healthy woman presented to the Emergency Department (ED) with the complaint of chest-back pain and shortness of breath. During her follow up she developed septic shock due to the empyema. 13rd hour of her admission, cardiopulmonary arrest developed and she died in spite of the appropriate and immediate treatment. 2 days later after her admission, streptoccoccus pyogenes was detected in her pleural effusion and blood culture. In conclusion clinicians should be aware of this potential fatal condition also in healthy patients and start the treatment immediately.

Case Presentation

A 43 years old woman presented to the Emergency Department (ED) with the complaint of chest-back pain and shortness of breath. Her sypmtoms had begun 3-4 days ago. Her vital signs were: Blood Pressure: 81/52 mmHg, Heart Rate: 140/min, Respiratory rate: 30/min, 0_2 sat: 97% (without O_2 support). Her Glasgow Coma Scale score was 15 but she was agitated. In her initial examination: The lung sounds decreased in right hemithorax, the distal peripheral pulses couldn't be palpable.. The upper extremities were cold and pale.. Her ECG showed only sinus tachycardia. Her arterial blood gases analyses (ABG) were as following: pH: 7.32, pO_2:82, p CO_2: 25, O_2 sat: 98, HCO_3: 15, lactate : 8,9 Aortic dissection and pulmonary embolismus were firstly predicted in the differential diagnosis.The triple rule out Computerized Tomography (CT) was applied to the patient. After the CT procedure, bradichardia was developed in her cardiac monitorization at 2nd hour of her admiision. She was administered 0.5 mg Atropine iv. Then she was arrested and the arrest rythym was pulseless electrical activity. After 1 cyclus of the CPR, ROSC was gained. The first ABG after the ROSC revealed ph: 6.82, pO_2:51, pCO_2:77, O_2 sat: 56%, HCO_3:12, lactate:13. The acute phase reactants results were as following: Leucocyte count: 2600 , C-reactive protein:45,9 mg/dL, procalsitonin: 11,98 ng/mL. The CT was reported as massive pleural effusion on the right side of the lung, no pathologic finding on the coronary and pulmonary arteries and also on aorta by the radiologist (Figure 1). A thoracostomy tube was inserted to the right side of the lung and approximately 2000 cc purulent effusion was drained (Figure 2). Meropenem was administered to the patient for the empyema. She was administered vazopressor agent by titrating the dosage but hypotension was resistant.pH value couldn't be reached over 6,8 during the follow up in spite of $NaHCO_3$ infusion.

Her relatives were asked whether she took any medications in overdose, recently having an influenza infection including fever, cough; any acutely ill patient in the home living with them. They told she had a boy having cerebral palsy and he didn't hospitalized for a long time. The possible cause of the empyema couldn't be found. Approximately

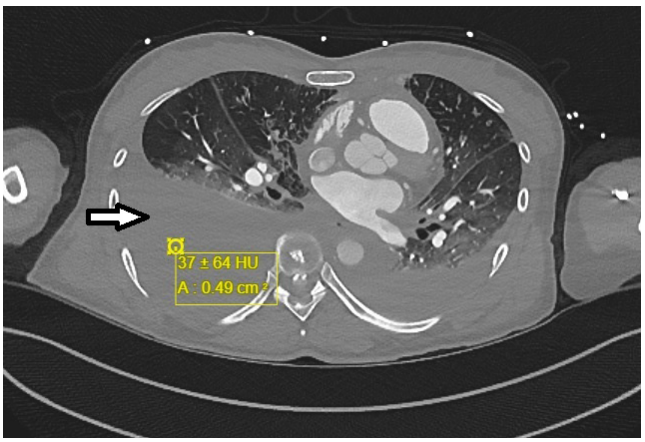

Figure 1: The massive pleural effusion on the right side of the lung with high HU*.
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Received: 19.08.2020 • **Accepted:** 09.09.2020
DOI: 10.33706/jemcr.822598

Figure 2: The drainage chest tube of the patient including purulent effusion.

13rd hour of her follow up, cardiopulmonary arrest developed and ROSC could not be achieved and she died. 2 days later after her admission, streptoccoccus pyogenes was detected in her pleural effusion and blood culture.

Discussion:

Streptoccoccus pyogenes is gram + aerob coccus and it is described as group A Streptococcus (GAS) according to the Lancefield classification.The exotoxin of this microorganism may cause toxic schock syndrome (TSS). GAS may cause pneumonia rarely and mostly seconder to the viral infection[1]. After the development of the pneumonia pleural effusion may occur rapidly and occasionally in the left side of the lung and frequently it is empyema. The prevelance of GAS associated pleural effusion is 0,7% in all pleural effusions[2]. Bacteriemia may develop 80% in pnemonia due to GAS. The 25% of these patients will develop TSS with high mortality rates[3]. Shock and multiorgan failure will develop in hours because of its exotoxins which cause tissue necrosis rapidly.

The mostly of these patients having a risk factor (minor trauma, rutine non steroidal antiinflamatory drug usage, recent surgeries, viral infections [influenza, varicella, HIV], iv drug abuse, malignancy, burns, diabetes mellitus and immunsupression.). The risk of the GAS assosiated TSS development in a healthy patient is so low.

Hypotension is resistant in spite of the vasopressor treatment. Our patient had also resistant hypotension and metabolic acidosis. Mortality rate is 30-70%[3].The absolute diagnosis can be made by the GAS grown in sterile area cultures like blood, pleural effusion, pericardial effusion, cerebrospinal fluid or surgical wound.

The management includes the treatment of septic schock, the debridement of the tissue necrosis if there is and antibiotic therapy.

Tamayo et al could showed streptococcus pyogenes in only one patient among 40 GAS associated pneumonia[3]. During the 2009 influenza A (H1N1) pandemia, there were increase in hospitalization of the children with the pleural effusions[4].

Asai et al reported a patient which presented with a severe Streptococcal pyogenes empyema following influenza A infection. This patient had no medical history apart from influenza. He was recoveried by drainage with intrapleural urokinase and antibiotic therapy[5].

Sakaia T et al reported a case report in which previouly healthy patient presented with empyema and TSS due to streptoccocus pyogenes and recoveried after the intensive combined therapies including drainage, anti-DIC agent and antibiotics[6]. Our patient was also a healthy woman and despite the detailed history and examinations we couldn't explain the possible cause of this fatal infection.. In conclusion clinicians should be aware of this potential fatal condition also in healthy patients and start the treatment immediately.

Acknowledgements: None

Conflict of interest: All authors declare no conflict of interest

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