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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

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Cause of the Emergency Department Visit: Internal Jugular Vein Thrombosis

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Abstract

Introduction: Detection of the internal jugular vein thrombus (IJVT) in the Emergency Department with bedside ultrasonography is not common.

Case Report: A 43 years old male patient was admitted to our emergency department with the complaint of pain, swelling and redness on the left side of the neck, which was noticed after chemotherapy 1 day ago. The swelling and redness of the hand, starting from the left clavicle and extending to the corner of the left mandible, were detected in the physical examination of the patient. The patient underwent bedside ultrasonography imaging by the emergency medicine specialist. Ultrasonography examination of the patient was performed with 5-12 MHz linear probe. After visualization of the vascular structures carotid, vascular structures were observed in the sagittal and longitudinal plane by gradual compression. Hyper echoic thrombus was seen in the incompressible internal jugular vein.

Conclusion: IJVT can lead to complications such as pulmonary embolism, septic embolism, cerebral venous thrombosis. These complications may decrease with early diagnosis and treatment. It is a rare condition in IJVT Emergency Services. The increasing use of bedside ultrasonography by emergency physicians will prevent possible complications that will enable these patients to be easily diagnosed and successful treatments in recent years.

Key words: Doppler Ultrasound Imaging, Emergency Department, Jugular Vein, Venous Thrombosis.

Introduction

Swelling in the neck can be caused by a variety of reasons, although rarely, there may be a complaint to the Emergency Department. Internal jugular vein thrombosis is also one of the rare conditions that can cause swelling in the neck. Deep venous thrombosis localization is common at lower limbs. Swelling and tenderness can be the first symptom especially at younger (2). A retrospective study of 1948 DVT patients demonstrated that jugular venous thrombosis is rare (1.5%) [X]. Vascular ultrasonography, "two-point compression method" are used frequently in emergency services to detect thromboembolic events, especially thrombi in femoral and popliteal veins. Detection of the internal jugular vein thrombus (IJVT) in the Emergency Department with this method is a rare condition. We presented the diagnosis process of a patient with complaints of neck swelling and internal jugular vein thrombosis in bedside ultrasonography imaging with images.

Case Report

A 43 years old male patient was admitted to our emergency department with the complaint of pain, swelling and redness on the left side of the neck, which was noticed after chemotherapy 1 day ago. It was learned that the patient, who was diagnosed with lung cancer (non small cell) about 1 time ago and who received 3 cycles of chemotherapy, had pathological lymph nodes in the left and right supraclavicular region in the ultrasonography imaging performed 15 days ago. He smoked one pack per day 15 years and no alcohol use history. The patient's systolic blood pressure was 116 mmHg, diastolic blood pressure was 66 mmHg, heart rate was 116 / min, oxygen saturation was 98%. The swelling and redness of the hand, starting from the left clavicle and extending to the corner of the left mandible, were detected in the physical examination of the patient. To evaluate lymph adenitis, mass, cellulite or vascular abnormality, the patient underwent bedside ultrasonography imaging by the emergency medicine specialist. It was observed that there was no flow in the internal jugular vein with doppler. Innominate, subclavian axillary veins were open. In the laboratory findings, white blood cell count 6.3 k/µL, platelet count 502 k/µL, hemoglobin level 9.6 g/dL, coagulation studies international normalized ratio (INR) 1.22, prothrombin time (PTZ) 17.1. Basic biochemical blood values were normal. The patient underwent pulmonary computed tomography imaging with suspected pulmonary embolism. No signs of pulmonary thrombus or embolism were seen on tomography imaging.

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The patient was recommended to treat enoxaparin sodium 0.6mL (6000IU AntiXA), 2 times a day for 10 days subcutaneously. After 10 days, the patient's control ultrasonography showed that the IJVT sign regressed completely.

Ultrasonography technique and findings

Vascular ultrasonography evaluations can be performed in emergency services with the help of high frequency transducers and linear probes. In particular, the lack of full compression of the arterial vessels in the horizontal section provides the distinction from other vascular structures. Detection of the arterial vascular structure helps to identify its adjacent anatomical structures. However, excessive manipulation and pressure can also cause undesired disintegration of an existing thrombus. The use of color doppler also helps to define blood flow. Especially in the evaluation of the internal jugular vein, evaluation should be made from the clavicle to the skull base. The possibility of incompressible structures such as lymph node etc. should not be forgotten. Sonographically reactive lymph nodes can be detected with oval hypoechogenic hilus and low doppler appearance[1].

Ultrasonography examination of the patient was performed with Samsung HS70A machine and 5-12 MHz linear probe. After visualization of the vascular structures carotid, vascular structures were observed in the sagittal and longitudinal plane by gradual compression. Hyperechoic thrombus was seen in the incompressible internal jugular vein (Figure 1 (A-B)). After 10 days, the patient's control ultrasonography showed that the IJVT sign regressed completely (Figure 2 (A-B)).

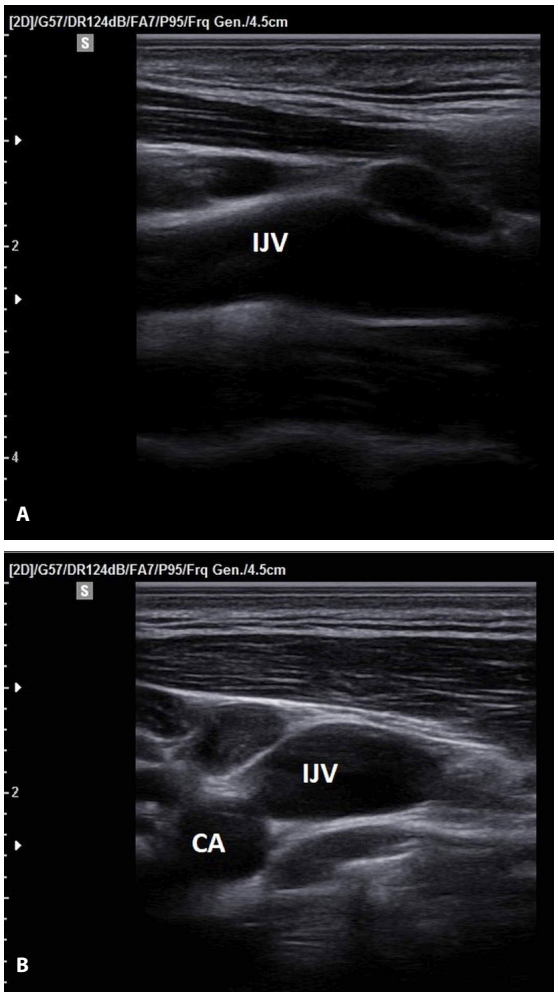

Figure 2: Longitudinal (**A**) and transverse (**B**) ultrasonography image of the internal jugular vein and carotid artery after treatments.

Discussion

The deep vein thromboses are frequently seen in the lower extremities. IJVT accounts for about 1.5% of all deep vein thrombosis in emergency services[2]. IJVT cancer can be monitored due to central venous catheter and ovarian hyper stimulation syndrome. Behçet's disease, middle ear, sinus or oropharyngeal infections, neck abscesses are other detectable etiologies[3,4,5]. It is stated that malignancy is the most common cause that triggers IJVT, especially in cases with cervical metastasis in the analyses[6]. Cancer patients are mostly patients with hypercoagulability. Venous thromboembolism is a clinical condition observed in approximately 15% of these patients, especially in patients receiving chemotherapy[6]. Approximately 20% of patients with symptomatic deep vein thrombosis have a known active malignancy[7]. Therefore, it should be considered that IJVT may be the first sign of malignancy. These cases, like our case, often refer to the hospital with painful and sensitive swelling in the neck[6]. Also in literature a heavy weight lifter nutritional supplementary (calcium fructopyranose borate) admitted emergency department complaining headache ,neck and arm diagnosed JVD, was described. This case report complet blood count(CBC) parameters was abnormal elevated; hemoglobine (18.1 g/dl), hematocrit (52.3 percent). Our case CBC parameters was decrased[11].

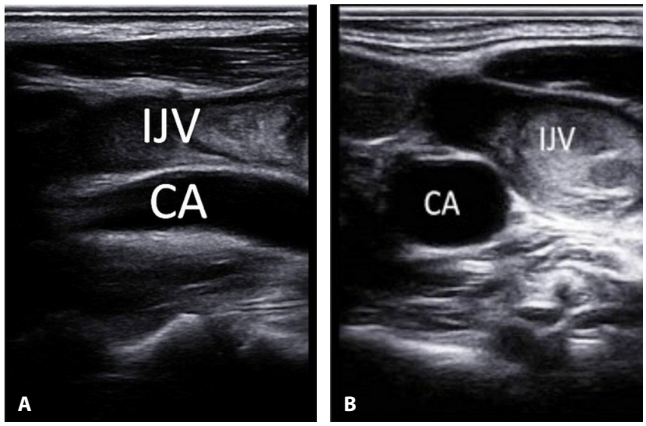

Figure 1: Longitudinal (**A**) and transverse (**B**) ultrasonography image of the internal jugular vein thrombus with the carotid artery.

The IJVT was detected in 61 of 210 patients with upper vein deep vein thrombosis in a retrospective study. Only 21

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patients with isolated IJVT were detected in the same study. This clinical condition was more common in women[7]. There was no other vein thrombosis accompanying IJVT In our case.

As IJVT complication, pulmonary embolism can be seen in approximately 10% of cases[8]. Apart from this, it can also cause post-thrombotic syndrome. Septic embolism may also develop following infected thrombophlebitis[9]. Failure to detect or properly treat deep vein thrombosis may result in cerebral venous thrombosis, increased intracranial pressure, and severe morbidity and mortality with cerebral edema[10].

There are no defined guidelines for IJVT treatment. It is treated like other deep vein thromboses. It is generally recommended to start treatment with low molecular weight heparin and to continue anticoagulant treatment with vitamin K antagonists. It is recommended that anticoagulant use should not exceed 3 months. Primarily 3-6 months anticoagulant use, thrombolysis, thrombectomy and vena cava superior filter are applied in selected case[2].

Conclusion

It is a rare condition in IJVT Emergency Services. Especially, patients with malignancy and neck swelling, pain, etc. should be evaluated in terms of internal jugular vein thrombosis by considering possible complications. However, in cases where IJVT is detected, other possible etiologies should be kept in mind. The increasing use of bedside ultrasonography by emergency physicians will prevent possible complications that will enable these patients to be easily diagnosed and successful treatments in recent years.

Funding

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Ethical approval

Ethical approval is not required at our institution to publish an anonymous case report.

Conflicts of interests

The authors declare that they have no competing interests.

The funders had no role in the design, conduct, analysis, or interpretation of data or in writing the manuscript.

Contributors

AG wrote the first draft of this paper. All authors approved the final version.

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Case Report

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A New Risk Factor for Hepatic Encephalopathy: Ingestion of Mad Honey

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Abstract

Introduction: Hepatic encephalopathy (HE) is a neuropsychiatric syndrome that occurs in the severe liver dysfunction and is characterized by a wide range of central nervous system symptoms. Hepatic encephalopathy precipitated with mostly infections, gastrointestinal bleeding, diuretic overdose, electrolyte imbalance, constipation and drugs. Traditional local foods for example honey can contain toxins for the liver and could cause acute decompensation in patients with chronic liver disease (CLD). This toxins can cause dose related severe symptoms. Spectrum of the symptoms are from nausea, vomiting to life threatening bradycardia and/or hypotension.

Case Report: A 66-years-old man with prior hepatitis B-related Child-Pugh B cirrhosis admitted to the hospital with nausea, vomiting, confusion, gross disorientation and bizarre behavior. By the aid of medical history, physical examination, laboratory tests and imaging techniques; hepatic encephalopathy diagnosed with distinct aetiology, is mad honey consumption.

Conclusion: In this paper; we reported first time in English literature a cirrhotic case with hepatic encephalopathy due to consumption of mad honey.

Key words: Grayanotoxins (GTS), hepatic encephalopathy (HE), mad honey (MH)

Introduction

Hepatic encephalopathy (HE) is a neuropsychiatric syndrome and is characterized by a wide range of central nervous system symptoms and findings including trivial lack of awareness, euphoria, anxiety, shortened attention span, lethargy, somnolence, stupor, coma as well as dead, flapping tremor in connection with higher serum ammonia levels[1].

Precipitating factors of acute HE are infections, gastrointestinal bleeding, diuretic overdose, electrolyte disorders, constipation, psychoactive drugs, dehydration and consumption of large amounts of animal protein containing foods[2].

Mad honey (MH) is a natural form of honey which obtained from forested areas of Asia and believed to beneficial to health in many parts of Asia especially in northeast part of Turkey. MH has also some toxic properties including grayanotoxins (GTS) which causes bradycardia, hypotension and agitation. Consumption of honey containing GTS obtained from Rhododendron plant species is termed as MH poisoning, and it is mostly reported in north east of Turkey, Korea, and Nepal[3,4].

Diet management of the patient with chronic liver disease (CLD) takes an essential role in preventing HE, there is lack of studies regarding dietary advice about honey consumption for patients with cirrhosis in medical literature. We present the first case HE due to consumption of MH.

Case Description

A 66-years-old man with prior hepatitis B-related Child-Pugh B cirrhosis admitted to the hospital due to nausea, vomiting, confusion, gross disorientation and bizarre behavior. His wife declared that, he was consumed a large amount of MH due to belief in anti-cirrhotic effect before the emergency admission. He was taking lactulose, propranolol and L-ornithine L-aspartate regularly for two years. He was non-smoker and taking no alcohol. Also his family history was not remarkable about CLD. Physical examination showed a blood pressure of 100/70 mmHg and a heart rate of 60 bpm, with normal respiratory and cardiac auscultation. He had a flapping tremor, with a palpable spleen 3 cm below the left costal margin. There were no sign of ascites and gastrointestinal bleeding. The electrocardiogram and chest X-ray were normal. His laboratory tests were as follows white blood cell count: 2.770/mm^3; hemoglobin: 11,4 g/dl; platelets: 74.000/mm^3; alanine aminotransferase: 195 U/L; aspartate aminotransferase: 335 U/L; alkaline phosphatase: 229 U/L, ɣ-glutamyl transferase: 210 U/L ; lactate dehydrogenase: 358 U/L; total bilirubin: 1,69 mg/dl; direct bilirubin: 0,6 mg/dl; prothrombin time: 16.2 seconds; serum albumin: 28,9 g/L; serum creatinine: 0.9 mg/dL. His plasma ammonium level was also 240 U/L (normal range; 0-80). While serology tests for Hepatitis A, C and E viruses were negative; he tested

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positive for hepatitis B surface antigen with low levels of hepatitis B virus DNA by PCR. Thus following investigations were carried out and revealed that liver size was in 120 mm, and had lobulated contours with increased caudate lobe. There was no solid or cystic lesion. Gallbladder had 37 mm in transverse diameter and its wall thickness was 3 mm which was in normal limits. His choledochus size was 9 mm which was in normal limits. Pancreas had normal contours and its parenchyma was homogeneous. Axial diameter of splenic size was 165 mm. Additionally, portal vein doppler ultrasonography was performed and showed us: portal vein and its branches were clear, no evidence of thrombosis, both of portal and splenic veins' sizes were increased which are compatible with portal hypertension. His cranial computed tomography and magnetic resonance imaging of brain revealed no striking findings. His final diagnosis was advanced stage liver cirrhosis due to chronic hepatitis B infection with HE. He was treated with colonic cleansing with lactulose twice per day and rifaximine 1200 mg per day orally. At the end of the second day of hospital stay, his ammonium level was reduced to 100 U/L with normalization of HE findings. He referred to another hospital for liver transplantation.

Discussion

We described the first case report has shown that HE may has been due to MH poisoning in a compensated patient with cirrhosis.

MH intoxication may occur after ingestion of the honey contaminated with lipid-soluble toxins called GTS[5]. GTS are mainly obtained from the nectar of Rhododendron ponticum which is a member of Ericaceae botanical family. Those plants are mostly growing on the mountains of the eastern Black Sea region of Turkey also in Japan, Nepal, Brazil and some parts of North America and Europe[6].

MH has been used in these regions as an indigenous medicine to relief abdominal pain, arthralgia, dyspepsia and to treat gastritis, hypertension, diabetes mellitus and also used for sexual stimulating effect[7].

Symptoms of MH intoxication are dose related. Minor symptoms are including dizziness, weakness, excessive perspiration, hypersalivation, nausea, vomiting and paresthesia and close follow-up is enough. However, when severe intoxication happened, it may lead to life threatening cardiac complications such as hypotension or atrioventricular blocks[8].

In our case there were no bradycardia or hypotension which is related to muscarinic effect of the mad honey. In addition there were no precipitating factors for developing HE including pulmonary and urinary tract infections, intracranial events, constipation, electrolyte disturbances, gastrointestinal bleeding, history of alcohol consumption, use of anti-depressions and narcotic drugs. Thus we postulated that described case's clinical picture was related to MH poisoning.

Currently there are a few case reports involving MH intoxicated non-cirrhotic patients with seizures probably due to neurotoxic effects of GTS[9,10]. A Turkish study showed that intracerebral administration of GTS successfully stimulates convulsive generalized seizures in adult Wistar rats[11].

Rat studies also revealed that M2-muscarinic receptors are involved in cardiotoxicity of GTS[6]. In addition, GTS facilitate the entry of calcium into cells by modifying action potential of sodium channels and increasing permeability of sodium ions in excitable membranes. This calcium-related effects resulted in prolonged depolarization period of the excitable cells including nerve and muscle fibers[12]. Thus inactivation of excitable cells are inhibited[13].

GTS are lipid-soluble neurotoxins which have cardio-toxic effects via M2- muscarinic receptors. On the other hand, almost 30% of GABAergic neurons in cortical neuronal system express M2 receptors[14]. It has also been a well known fact that, the mechanism of HE due to hyperamoniemia is related with activity of GABAergic neurons in brain[15].

To the best of our knowledge, this is the first case report that describing MH poisoning causing HE in a patient with cirrhosis. But there is no available data about GTS' capability of passing the blood brain barrier. There is also need further studies in the field of neurophysiology about GABAergic effects driven by GTS.

We finally concluded that dietary management of the patient with CLD takes an essential role in preventing HE. Mad honey consumption could be a risk factor for HE in these patients. Further large scale studies are necessary to determine whether and how MH consumption as a risk factor for HE in the literature.

Conclusion

Mad honey consumption is the first reported aetiology in the literature of a common condition called hepatic encephalopathy. Clinicians should be alert on honey - mad honey consumption, before regarding hepatic encephalopathy with unknown aetiology.

Ethics

The authors have no ethical conflicts to disclose. Written informed consent to publish the case report was obtained from the patient.

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Case Report

Journal of Emergency Medicine Case Reports

Pseudoulnar Palsy Due to Ischemic Stroke; Case Report

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Abstract

Isolated motor nerve paresis can occur due to central nervous system lesions. This condition is extremely rare, they are often misdiagnosed as a peripheral nerve lesion. 83-year-old male, applied to our hospital with 4th and 5th digit weakness. Neuroimaging revealed cortical-subcortical diffusion restriction in the medial gyrus of the precentral gyrus. The patient was diagnosed as ischemic stroke. In this article, we presented a case of ischemic stroke that mimics ulnar nerve paresis and be easily overlooked.

Key words: Ulnar Nerve, Paresis, Stroke

Introduction

Isolated motor paresis due to central nervous system lesion was first described by Lhermitte and was reported as pseudoperiferal palsy[1]. These lesions may be ischemic stroke or brain tumors, abscesses and hemorragie[2-4]. These types of paresis, which are associated with ischemic stroke, are rare and often diagnosed as perfieric nerve paresis[5]. Hand motor area is located in the precentral sulcus. Any ischemic infarcts in this area can lead to isolated motor paresis[6]. In this article, we presented a case admitted to our hospital 4th and 5th digit weakness, was diagnosed ischemic stroke.

Case Report

83-year-old male complained sudden onset loss of strength his 4th and 5th finger, was admitted emergency service. He stated that this complaint developed two hours before the application. He denied any dizziness, loss of consciousness, speech disorder, gait disturbance or loss of balance. The patient has hypertension, ischemic heart disease and chronic myeloid leukemia had no history of chemotherapy or radiotherapy. He was using imatinib, ramipril, clopidogrel and trimetazidine for existing diseases. His arterial blood pressure was 130/70 mm Hg, body temperature was 36.4 Celcius, heart rate was 86 bpm, and oxygen saturation was 99%. On neurological examination, he was conscious, cooperative. The patient speech was normal and had normal cranial nerve examination but right hand's 4th 5th digit flexion, abduction and adduction motions were 3/5 muscle strength (Picture 1). There was no abnormality was detected in laboratory examinations, no pathology was found on computed brain tomography, whereas cortico-subcortical diffusion restriction was detected in the medial precentral gyrus in diffusion-weighted magnetic resonance imaging (Picture 2). No pathology was found in the bedside echocardiography, cervical spinal magnetic resonance imaging and electroneuromyography. He was admitted to the Neurology Service with the diagnosis of ischemic stroke.

Discussion

Pure motor monoparesis due to ischemic stroke is a rare condition[6]. Typically, monoparesis tend to worsen over time as they develop as a result of compression. If it is develope suddenly, it should be consider ischemic stroke[4]. Monoparesis due to ischemic stroke are mostly seen in the hand region[6-8]. In our case, a suddenly developing ischemic stroke affect patient's right hand. Representation of the hand in the motor cortex is in the precentral gyrus. Due to the node-like feature of this structure, the region is called precentral node[4]. In the literature, there were many ischemic strokes in the precentral node region caused monoparesis. Among these motor monoparesies, ulnar, radial and median motor paralysis are found separately[2,9-11]. Ulnar or radial motor monoparesis was more likely to occur[12,13]. Similar to that, ulnar motor paresis occurred after ischemic stroke in our case. Strokes in the medial parts of this region are mostly associated with ulnar paresis, and in the lateral part with radial paresis[14]. The lesion in our patient was in the medial part of region and ulnar

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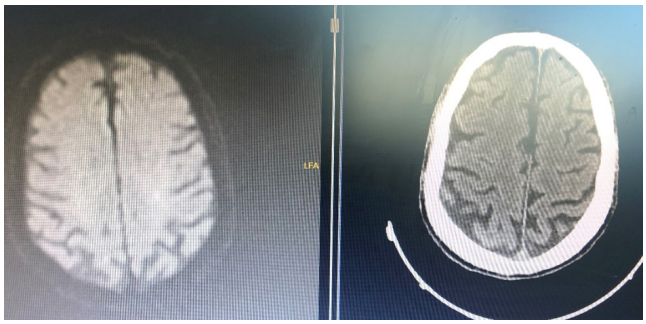
Picture 1



Picture 2

nerve involvement developed. In the etiology of ischemic stroke cases, patients with ulnar nerve paresis have been associated with hemodynamic reasons and those with radial nerve paresis have been associated with embolic causes[15]. In a case series Timsit et al. found that there were hemodynamic causes in the etiology of patients[16]. In our case, there was no etiological cause was found.

Conclusion

Diagnosis of ischemic stroke should be kept in mind in patients admitted to the hospital with sudden isolated nerve paresis and appropriate cranial imaging tests should be performed in the differential diagnosis.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Cause of Epidural Abscess: Esophageal Perforation After Radiotherapy

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Abstract

Introduction: Spinal epidural abscess is a rare neurosurgical emergency that is usually seen invasive interventions to the spinal region, trauma or infection. In our case, anteriorly located spinal epidural abscess, which is caused by spontaneous esophageal rupture as complication of radiotherapy, is described.

Case report: A 60-year-old female patient was admitted to the emergency department with complaints of confusion, high fever and weakness in both legs. The patient had been treated for lung metastasis of breast cancer with radiotherapy. Spinal anterior epidural abscess was observed in the spinal imaging, and it was considered that the abscess developed after esophageal rupture secondary to radiotherapy. After surgery the patient had antibiotic treatment during the postoperative period. Although there are cases of spinal epidural abscess that developed after esophageal rupture in the literature, they were generally observed after interventions to the esophagus or trauma to the chest. In our case, epidural abscess developed after radiotherapy, that caused esophageal rupture, was presented.

Conclusion: Clinicians should consider spinal epidural abscess, which is a rare complication in patients with walking difficulties after radiotherapy, in the differential diagnosis.

Keywords: abscess,rupture,spine,radiotherapy,paresis

Introduction

Spinal epidural abscess (SEA) is a rare neurosurgical emergency that has to be treated early[1]. Incidence of SEA is 5.1 per 10,000 hospital admissions[2]. SEA mostly occurs in patients aged 30 to 60[1]. Invasive interventions to the spinal region, trauma, systemic or surrounding tissue infection play a role in its etiology[3]. In spite of being cases of spinal epidural abscess that seconder to esophageal rupture in the literature, these generally developed after interventions to the esophagus or traumatic blows to the thoracic wall or vertebrae[4]. In our case, anteriorly located spinal epidural abscess, which is caused by spontaneous esophageal rupture as complication of radiotherapy, is described.

Case report

A 60-year-old female patient was admitted to the emergency department with complaints of confusion. In adittion there was weakness in the lower extremities and difficulty in walking that started two days ago. As far as it was learned, she had fever and swallowing difficulties despite her antipyretic and antibiotic therapy for about ten days. Lung metastasis was detected 2 years ago in the patient who was diagnosed with breast cancer and the final radiotherapy was applied to the thorax 1.5 months ago.

At the time of admission, the patient's body temperature was 38.2°C, arterial blood pressure: 110/70 mmHg, peak heart rate: 121 beats / min. The patient was confused. The motor muscle strength of both lower extremities was evaluated as 2/5. The deep tendon reflexes of the patient, who described hyperalgesia below the T10-T11 level, could not be obtained bilaterally. Babinski reflex was positive in bilateral lower extremities. In the routine blood samples taken from the patient, WBC: 14.67 / mm3, NEU: 13.66 / mm3, CRP: 187 mg / L were reported. No acute pathology was found in the cranial imaging of the patient. It was observed that her state of consciousness improved after her body temperature decreased with antipyretic therapy. In spinal CT examination, C7-T1 level prevertebrally located hypodense area, diffuse free air images in the prevertebral area of T1-T5 vertebrae and adjacent prevertebral area, and suspicious participation between air images and middle esophagus segment was detected. Also, there were free air images in the posterior part of the spinal cord (Picture 1). In the contrast-enhanced thoracic vertebra MRI examination showed osteomyelitis in the upper thoracic vertebrae, abscess in the prevertebral area, a hyperintense lesion in T2AG with a width of approximately 1 cm in the epidural space at the T2-3 level in the right ante-

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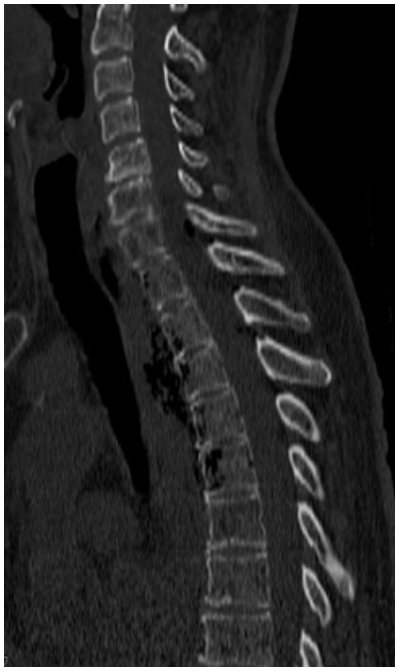
rior section. The lesion was evaluated as an epidural abscess in contrast-enhanced series (Picture 2). Gastroscopy was planned in terms of esophageal fistula and perforation that could cause abscess in the prevertebral region. In the gastroscopic examination, the area covered with secretion and exudate covering ¼ of the lumen in an area of approximately 23 cm in the upper esophagus and the perforation opening from the lower part were observed.

The patient underwent decompression surgery with T1-3 level total laminectomy due to cord compression secondary to the spinal epidural abscess. Samples of the abcess obtained were sent to the microbiology and pathology laboratory. It was observed peroperatively that there were extensive hard granulation tissues around the dura mater. In the same session, a stent was placed in the esophagus by the general surgery clinic for esophageal perforation.

The microorganisms could not be isolated from the material taken during the operation. She was transferred to a physical therapy unit for rehabilitation after antibiotherapy.

After 6 weeks from the surgery, there was no progression of neurological status in proximal of the lower extremities, while there was partial recovery in the leg muscles, full strength was achieved in foot dorsiflexion and plantar flexion.



Picture 1: Diffuse free air images in the prevertebral area of T1-T5 vertebrae

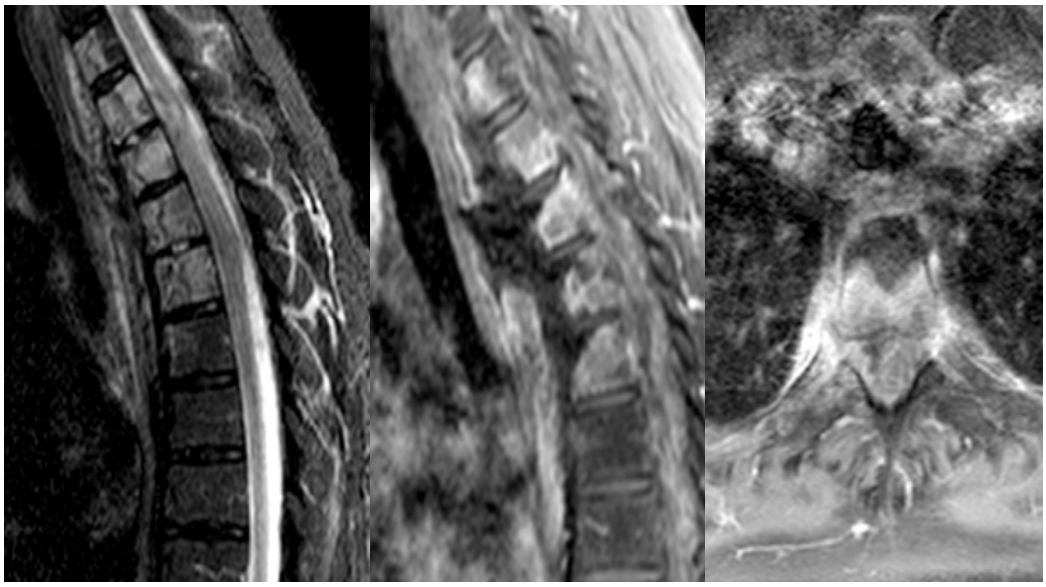
Discussion

SEA is a rare condition in which devastating consequences can be avoided with early diagnosis and treatment. Paraplegia, quadriplegia and even death can be seen in late intervention. Although death can be seen in cases with sepsis or comorbidities, its rates have decreased with the use of antibiotics. The reversible neurological deficit is associated with early diagnosis and decompression of the spinal canal, especially in the first 24 hours[2-4].

In a study by Ghobrial et al., cases of SEA that were treated and not treated in the first 24 hours were compared, and no statistically significant difference was found between the two groups in terms of neurological progression[5].

In our case, the complaints of difficulty in walking started two days ago and she was operated 8 hours after her admission. In the postoperative follow-up of the patient, partial improvement was observed in her neurological deficit.

SEA, which are frequently located in the lumbar and thoracic regions, are located posteriorly in the canal; however, Lu et al. found that anteriorly located spinal epidural ab-



Picture 2: SEA at the T2-3 level in the right anterior section

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scesses were more common[6]. In our case, an anterior abscess was observed in the thoracic region.

Spinal epidural abscess clinically presents with low back pain, local tenderness, fever, loss of muscle strength, incontinence and radiculopathy. The classical triad of low back pain, fever and neurological dysfunction are seen in very few cases of SEA[2,3].

Our patient, who was admitted to our hospital with fever and paraparesis, had no pain or sensitivity with palpation on the spine, but had hyperalgesia in the bilateral lower extremity.

Risk factors published in the literature are diabetes mellitus (DM), intravenous drug use, alcohol addiction, HIV (Human Immunodeficiency Virus) infection, degenerative joint disease, recent trauma or surgery. Transition of microorganisms to the epidural space can be caused by hematogenous pathways, direct spread of infection from neighboring structures or by catheters in the epidural space[3,7].

In our case, two of the risk factors were seen; these are DM and infections that develop after esophageal rupture. We consider that the development of SEA is due to infectious agent passage to the prevertebral area and epidural area after esophageal rupture secondary to radiotherapy. Similarly, in a case report published by Pulle et al., spontaneous esophageal rupture was observed after radiotherapy[8].

The sensitivity and specificity of contrast-enhanced MRI in the diagnosis of spinal epidural abscess is 90%. Contrast-enhanced spinal CT examinations have low sensitivity, especially in early spinal epidural abscesses, and should not be preferred except in cases MRI is not available[3].

Recently, it has been shown that air can pass into the spinal canal from the mediastinum; this can be considered as an indication that there may also be transmission of infection through neighborhood[7].

In our case, spinal CT scan was performed firstly since it is easier to access under emergency conditions and in order to rule out possible bone pathologies that may cause paraparesis. Contrast-enhanced MRI examination of the patient who had no bone pathology, prevertebral area abscess, SEA and osteomyelitis in the upper thoracic vertebrae was detected.

Usually monobacterial pathogen is seen in SEA, and Staphylococcus aureus is the most common agent. Depending on the comorbidities of the patient, gram-negative pathogens or fungal factors may also cause it[2,3,9,10]. In our case, unlike the literature, there was no microorganism growth from the samples taken. On the other hand, samples sent to the pathology laboratory were reported as active chronic nonspecific inflammation. In cases of spinal epidural abscess, it is recommended that antibiotherapy be started empirically immediately after sampling, and then it is recommended to continue treatment for causative microorganism for 4-16 weeks[1,3].

Conclusion

Epidural abscess has many risk factors but secondary to esophageal rupture is rare in the literature. In our case we present a spinal epidural abscess due to esophageal rupture secondary to radiotherapy. Clinicians should consider spinal epidural abscess, which is a rare complication in patients with walking difficulties after radiotherapy, in the differential diagnosis.

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Case Report

Journal of Emergency Medicine Case Reports

Binocular Diplopia After A Snakebite

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Abstract

Introduction: Ocular manifestations in snake bite injuries are quite rare. Although ophthalmoplegia following snake bites is not a severe neurotoxic complication, symptoms such as diplopia, dizziness, and ocular discomfort can be emotionally devastating for patients. We presented a case with bilateral diplopia and ptosis after snakebite and was recovered with the anticholinesterase therapy and antivenom.

Case report: Blurred vision developed at the 10th hour of the follow-up of a 48-year-old man who had complaints of nausea, vomiting, fatigue, diarrhea, as well as swelling, redness, and pain on the lateral malleolus of the right foot ankle after a snakebite. His physical examination revealed binocular diplopia and bilateral ptosis.

Conclusion: Ophthalmoplegia (diplopia and ptosis) is a rare symptom of snakebites. It may induce anxiety and ocular discomfort for an extended period. Treatment of anticholinesterase combined with antivenom may facilitate the recovery from ophthalmoplegia.

Keywords: neuromuscular paralysis, snakebite, ophthalmoplegia, anticholinesterase

Introduction

A snakebite often results in puncture wounds inflicted by the snake's fangs and sometimes resulting in envenomation. Cases related to snakebite are common in emergency departments, especially in the summer months. Venom may cause a complex condition: local damage, neuromuscular dysfunction, or systemic vascular damage leading to hemolysis.

Although the World Health Organization classified snakebite as a tropical disease, envenoming can also become a severe public health problem outside of tropical regions. In particular, neurotoxicity develops from poisoning with pit vipers such as rattlesnake. Neurotoxicity related to snakebites is the leading cause of mortality and morbidity, and it develops mainly due to the neuromuscular paralysis[1]. Neuromuscular paralysis secondary to the snakebite can be explained by the inhibition of synthesis, packaging, transport, and release of the neurotransmitter acetylcholine in the presynaptic field, blockade of postsynaptic nicotinic acetylcholine receptors, acetylcholine esterase inhibition, muscarinic effects of some snake toxins, and inhibition of voltage-gated calcium gates, changes in the neuromuscular junction. Apart from these, neurological manifestations can also result from non-neurotoxic effects of envenoming, such as cerebral hemorrhage and infarction due to coagulopathy and myotoxicity[1].

In patients with newly developed diplopia, 3rd and 6th cranial nerve pathologies, orbital trauma, especially neuromuscular junction toxins, should be considered. For differential diagnosis, botulism, tick paralysis, snake venom, organophosphate and carbamate toxicity, hypermagnesemia/hypocalcemia, and some medications such as lithium, magnesium sulfate, phenytoin, and other anticonvulsants should be evaluated[2-3]. Ocular manifestations in snake bite injuries are quite rare. Although ophthalmoplegia following snake bites is not a severe neurotoxic complication, symptoms such as diplopia, dizziness, and ocular discomfort can be emotionally devastating for patients[2].

We presented a case with bilateral diplopia and ptosis after snakebite and was recovered with the anticholinesterase therapy and antivenom. In the Black Sea region of Turkey, poisonous snake envenomation is very rare. However, Turkey's cases, resulting in neuromuscular paralysis after snakebite, are rarely seen in the literature.

Case Report

A 48-year-old man was admitted to the emergency department with complaints of nausea, vomiting, fatigue, diarrhea, swelling, redness, and pain on the right foot ankle after a snakebite. He was a farmer, and he had no comorbid disease and medication. He was not a cigarette smoker and alcohol drinker. He declared that he did not use any illicit drugs.

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The patient was awake and alert and was non-toxic-appearing. His temperature was 37.1◦C, his blood pressure was 105/82 mmHg, his heart rate was 92 bpm, and his respiratory rate was 12 /min. The initial physical examination was regular without swelling and redness on the right foot ankle (Figure 1). All systemic examinations were regular. His serum electrolytes and cardiac enzymes were normal. Prothrombin time and activated partial thromboplastin time were at normal range, but white blood cell count (WBC) was elevated (14,61 thousand/uL), creatine phosphokinase (CPK; 765 U/L) and the international normalized ratio (INR; 1.33) were minimally elevated. ECG showed a normal sinusoidal rhythm. Sultamicillin 2 gr per day and %0.9 NaCl solution were administrated intravenously. He was hospitalized to the emergency observation unit for follow-up.

At the tenth hour of the follow-up, blurred vision developed. In the second physical examination, binocular diplopia and bilateral ptosis were revealed (Figure 2). The examination of eye movements showed a presence of partial bilateral paresis of third cranial nerves. Cranial CT and diffusion MR imaging had no pathology. He was consulted with a neurologist and an ophthalmologist.

The second dose (10 ml polyvalent snake serum) of antivenom was administered intravenously. The visual field was found in the normal range by an ophthalmologist. After the neurologist's consultation, pyridostigmine 60 mg tablet (3 tabs per day) was added to his treatment. The symptoms were improved, and the eye movements were regular at the 12th hour of the treatment (Figure 3). WBC, INR, and CPK were at a normal range. The patient was discharged from our emergency department with full recovery on the third day after the admission. Informed consent was obtained from the patient for the publication of his information and images.

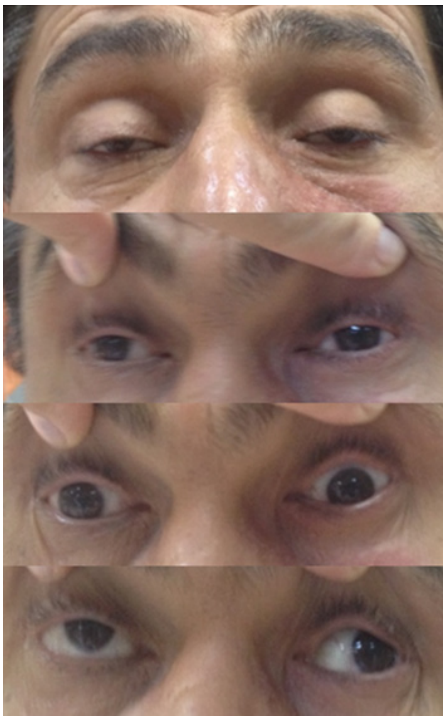

Figure 2: Diplopia and ptosis

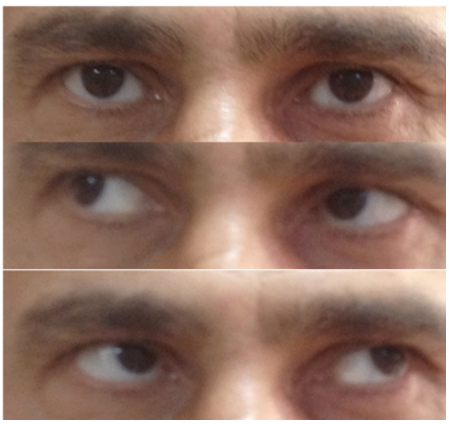

Figure 3: Improved eye findings after the treatment

Discussion

Localized and systemic symptoms may be seen after snakebites such as focal edema, pain, focal necrosis, muscle degeneration, disseminated intravascular coagulation or hemolysis, and acute renal failure[2]. Localized symptoms on his foot ankle due to snakebite were seen in our patient, but no necrosis. Neuromuscular paralysis or oculomotor nerve paralysis associated with snakebites was considered in our patient because of aberrant history and physical examination findings that exclude other causes affecting the neuromuscular junction. The normal results of our patient's cranial imaging supported the snake envenomation instead of structural brain pathologies.

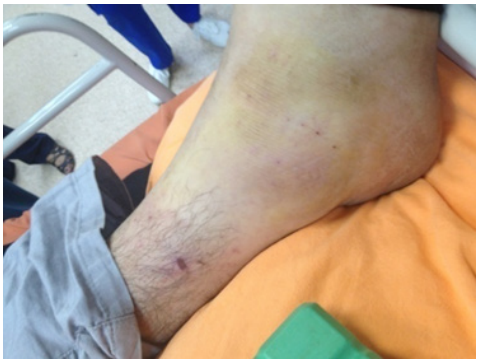

Figure 1: Swelling and redness on lateral malleolus of the right foot after a snakebite

According to the literature, a few snake species such as Europa viper (Vipera apsis), North America Viper (Agkistrodon blomhoffi), and some other vipers may lead primarily to extraocular muscle paralysis. In some reports, the most common cause of eye symptoms following a snake bite is Agkistrodon blomhoffi. The most common eye symptom is ophthalmoplegia, and the medial rectus muscle is commonly involved[4]. In our case, the patient had bilateral ptosis and paresis of the bilateral medial rectus muscle, but more

prominent in the left medial rectus muscle. The species of the snake which bit our patient was not known.

Snake neurotoxins bind to the neuromuscular junction both pre-and post-synaptically causing muscle weakness. Alpha-bungarotoxin of krait binds to acetylcholine receptors, and inhibits acetylcholine receptor sites in the postsynaptic membrane. Alpha-cobra toxin, which has a similar action, produces features of myasthenia gravis in the experimental animals. Phospholipase A2 enzyme and Beta-bungarotoxin act pre-synaptically to cause neurotoxicity[3]. Neurotoxic paralysis may also begin within the first hour of snake bites and is seen first as ptosis, then blurred vision and diplopia, followed by facial weakness and dysarthria. In severe cases, the limbs' weakness, paralysis of respiration, and fixed and dilated pupils may be observed[4]. Our patient experienced blurred vision and diplopia in the late period.

The primary treatment of neurotoxic paralysis following snakebites is an injection of antivenom. Indications for antivenom are hemostatic disturbance, cardiovascular abnormalities, neurotoxicity, elevated CPK and aminotransferases with a definite local envenomation sign, and others. Because the binding to the presynaptic portion is irreversible, clinical recovery occurs slowly and only with the formation of a new neuromuscular junction. However, the toxin binding to the postsynaptic part may act post-synaptically to produce a competitive or noncompetitive acetylcholine receptor blockade[4]. Although an antivenom may induce a certain degree of reversal of the paralysis by postsynaptic neurotoxin, the clinical recovery may be prolonged. He was brought to our emergency department from another hospital due to hypotension, unresponsive to iv fluid treatment (80/60 mmHg) after the intravenous administration of snake antivenom. In the previous hospital, the ED physician has given a polyvalent antivenom because it was challenging to identify the snake species that had bitten the patient at the treatment time[4-6].

Anticholinesterase is potentially valuable for patients with suspected myasthenia gravis[6]. Sung and Hah[7] reported a case of extraocular muscle paresis following a snakebite. It was reported that a patient, supported with a mechanical ventilator due to the neuromuscular paralysis, which was developed about 5 hours after snakebite, was resuscitated successfully using intravenous neostigmine and oral pyridostigmine[8]. In our patient, symptoms developed at the tenth hours, and after treatment, the symptoms improved fastly similar to the other cases in the literature. The patient was recovered fully on the third day of the admission. Although the absence of signs of cholinergic crisis such as muscle fasciculation and presence of improvement at the physical examination, the use of anticholinesterase may be effective for patients with neurotoxicity following snakebites.

Conclusion

Ophthalmoplegia that may induce anxiety and ocular discomfort for an extended period is a rare symptom of snakebites. Combined treatment of anticholinesterase with antivenom may facilitate the recovery from ophthalmoplegia.

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Case Report

Journal of Emergency Medicine Case Reports

A rare cause for sciatalgia: Piriformis syndrome

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Abstract

The clinical symptoms comprising piriformis syndrome emerge as a result of the compression of the sciatic nerve due to anatomical variations or pathological conditions related to the piriformis muscle. The underlying cause in 6% of cases of lumbar pain, piriformis syndrome might be also defined as entrapment neuropathy of the sciatic nerve. While piriformis syndrome is not rare, it is not well known. As the clinical symptoms may be misleading, it can easily be overlooked, or lead the clinician to an incorrect diagnosis (such as discopathy). In this report, our objective was to present the case of a 18-year-old female patient who presented to the emergency department with sciatalgia and received a diagnosis of piriformis syndrome caused by pyomyositis of the piriformis muscle. The patient arrived at the emergency department reporting severe pain and paresthesia in the right pelvis, thigh, and leg with the inability to walk due to pain. Vital signs were normal except for a high fever. Her Lasègue test was positive, and the patient had severe pain with both internal and external rotation. After radiological examination, the patient was diagnosed with an abscess in the piriformis muscle due to pyomyositis. It was determined that the cause of sciatalgia was piriformis syndrome, with the abscess in the piriformis muscle compressing the sciatic nerve. The abscess was drained after the patient was admitted to the hospital; the patient was then discharged without further complication. The need for high-cost therapeutic methods or even death may result in instances where piriformis syndrome is overlooked by emergency department clinicians, or when it is given the misleading diagnosis of sciatalgia.

Keywords: sciatalgia,pyomyositis,piriformis syndrome,piriformis abscess,differential diagnosis

Introduction

Sciatalgia, a clinical symptom occurring as a result of the sciatic nerve being compressed at any point along its path, is characterized by pain spreading through the lumbar and sacral dermatomes[1]. Piriformis syndrome is seen as a result of the piriformis muscle compressing the sciatic nerve due to anatomic variation or its pathologies. In other words, piriformis syndrome can also be defined as an entrapment neuropathy of the sciatic nerve[2,3]. Here, we aimed to present a case admitted to the emergency department with sciatalgia for which the diagnosis was piriformis syndrome due to piriformis muscle pyomyositis discovered via clinical examination and radiological imaging.

Case Report

An 18-year-old woman was admitted to the emergency department reporting severe pain in her right hip and right thigh and leg and inability to walk due to numbness and pain. She stated that the pain started in her hip and spread to her right leg, gradually increasing in intensity through the past couple of days. Three days previously, the patient had been started on an analgesic and myorelaxant oral tablet at another health center, with lumbar discopathy being the general diagnosis as the cause of the pain. There was no history of systemic disease, recent trauma, or intramuscular injection in her medical record. The vital signs of the patient upon admission were normal except for high fever (38.8°C). In her physical examination, there was minimal tenderness to palpation in the right gluteal region, but no other signs of inflammation. Her Lasègue test was positive (that is, the patient could not raise her leg to a 45-degree angle without pain). While the patient brought her thigh to flexion and extension with ease, she felt severe pain during external rotation and especially internal rotation. Lower extremity peripheral pulses were equally palpable using the hands. The patient had no complaints related to her left gluteal region or left leg. Rectal and other systemic examinations showed no abnormalities. Her laboratory tests were as follows: white blood cell count (WBC): 17,000/MM^3, creatine kinase (CK): 500 U/L. Other parameters were normal.

Tomography was performed in the pelvic area because of the increased sensitivity in the patient's gluteal region;

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heterogeneity consistent with an abscess was present in the piriformis, gluteus maximus and gluteus minimus muscles on the right. Subsequent to tomography, MR imaging was performed to determine the source of abscess formation and to explain the sciatic pain. A lesion that was hypointense and fat-suppressed in the T1-weighted images and showing a significant signal increase in the T2-weighted images was seen inside the right gluteus maximus, minimus and piriformis muscles in the MR. The appearance was interpreted as an abscess inside the piriformis muscle and as sciatic nerve compression not related to the abdomen. The patient was believed to have piriformis syndrome caused by an abscess inside the piriformis muscle. The patient underwent abscess drainage accompanied by tomography and was started on prophylactic antibiotherapy. *Staphylococcus aureus* grew in the abscess culture that was done. The patient's condition did not regress during follow-up appointments; she underwent surgery and the abscess was cleaned and closed after surgical drainage. Upon resolution of the patient's condition during follow-up appointments, she was discharged with follow-up recommendations after 20 days of hospitalization.

Discussion

Piriformis syndrome is the underlying cause in about 6% of those suffering from lower back pain; it is not uncommon, but remains a little-known disease[4]. Piriformis syndrome is seen more commonly in women; the fourth through sixth decades of life constitute the highest risk period for the disease[5]. Piriformis syndrome is easily overlooked due to its clinical symptoms being misleading and directing the physician to incorrect diagnoses (such as discopathy)[5,6]. Just as piriformis syndrome can occur due to soft tissue inflammation or muscle spasm due to trauma, overuse of the muscle (such as when engaging in exercise involving long walks or running) can also lead to the syndrome[5]. For this reason, piriformis syndrome is often overlooked in athletes and those practicing sports.

After starting inside the pelvis, in the front side of the 2nd to 4th sacral vertebrae, the piriformis muscle goes through the greater ischiatic foramen and ends at the femur trochanter major[7]. In the pelvic region, neurovascular structures pass under or over the piriformis muscle in the foramen ischiadicum majus. The sciatic nerve usually extends underneath the muscle[6,8].

Pyomyositis caused by the piriformis muscle leads to irritation and compression in the sciatic nerve; this condition is one of the rare causes of piriformis syndrome[9]. Pyomyositis is a primary pyogenic infection of the skeletal muscle that usually progresses to abscess formation[10] and is specific only to muscle tissue. It is believed to develop secondary to injuries in the muscle during an asymptomatic and undiagnosed, transient bacteremia and is not related to bone or soft tissue infections around the muscle[8]. Many patients with pyomyositis are admitted with a clinical picture that changes depending on the location and severity of the infection, but is insidious nonetheless[10].

The three stages of pyomyositis progress from diffuse inflammation to focal abscess formation and the potential for sepsis[10]. While there is minimal inflammation during the first stage, there is no pus. The second stage is the suppurative stage, which appears 10 to 21 days after symptoms begin. In the last stage, systemic complaints surface along with high fever and septicemia[11]. Delays in the diagnosis of the disease lead to extended hospital stays and a mortality rate around 10%[12]. In our case, sciatic pain was accompanied by high fever and leukocytosis, and thus we evaluated it as stage third.

Despite the pain that occurs in piriformis syndrome being very similar at first to other types of pain that also cause sciatalgia, a number of differences exist. Similar to discogenic pains, the pain in piriformis syndrome spreads through the hip and the sciatic nerve path; unlike discogenic pains, weakness and numbness are rarely observed. Recreating the pain with maneuvers that place the sciatic nerve under stress during the physical examination can help lead the clinician to a diagnosis[3,6,13]. In piriformis syndrome, patients also experience pain during walking or prolonged sitting. Despite symptoms related to the straight-leg lifting test being diverse and contradictory in PS, they are usually normal, with the internal rotation of the hip exacerbating the symptoms[4,13].

Imaging methods are used to rule out other conditions that might be confused with the syndrome[6]. MR imaging is particularly valuable in terms of showing nerve compression due to piriformis hypertrophy on the affected side[6]. While electromyography can be normal, it can be useful for diagnosis in long-term compression[14]. In the case of our patient, the presence of high fever; increased tenderness in the gluteal region with palpation; increased pain in the hip with flexion, abduction, and rotation; and elevated WBC count in addition to the typical clinical symptoms of sciatalgia led us to search for a preliminary diagnosis other than discopathy. At that point, the patient underwent tomography and then (due to the results of tomography) MR imaging.

Treatment of piriformis syndrome is related to its etiology. Medical treatment, physical therapy, acupuncture, therapeutic perisciatic block, botulinum toxin injection, or surgical intervention are among the potential treatment options. Abscesses due to pyomyositis are treated with percutaneous drainage or open surgery accompanied by ultrasound or computed tomography (CT) imaging[10]. In our case, surgical drainage and antibiotherapy was applied to the abscess inside the muscle because secondary piriformis syndrome had developed. In the culture performed, *S. aureus* grew, in accordance with the literature.

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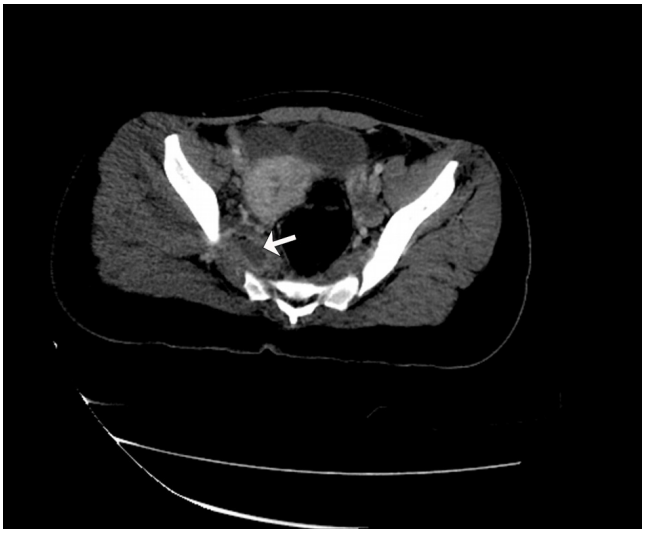

Figure 1: CT scan of pelvis demonstrate heterogeneity consistent with an abscess in the piriformis muscle on the right (arrow).

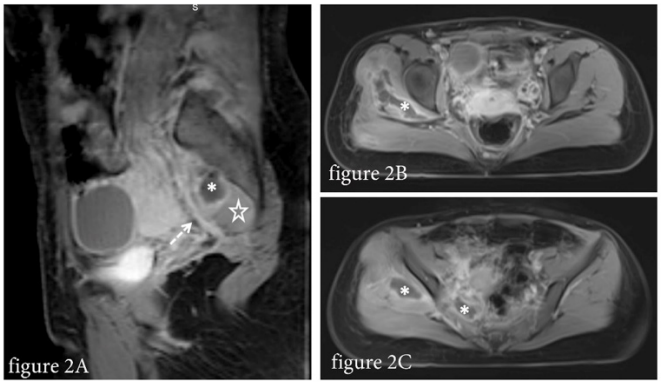

Figure 2: Sagittal (**A**), and axial (**B, C**) contrast enhanced (fat saturated) T1 weighted MR images show abscess formations in musculus piriformis and gluteal muscles (asterisks) and sagittal MR image (A) also shows compressed nervus ischiadicus (dashed arrow) below the abscess formation in the piriform muscle (star) (musculus piriformis syndrome).

Conclusion

In patients who report to the emergency department with symptoms of sciatalgia, the focus of the differential diagnosis should be on maneuvers in the physical examination that place the piriformis muscle under stress. If piriformis syndrome due to pyomyositis is overlooked by emergency clinicians with sciatalgia in mind, it can lead to death or the need for high-cost treatment methods.

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The authors have no conflict of interest to declare

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The written consent form is taken from the patient.

"The case report has written in an anonymous characteristic, thus secret and detailed data about the patient has removed. Editor and reviewers can know and see these detailed data. These data are backed up by editor and by reviewers."

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Case Report

Journal of Emergency Medicine Case Reports

Common Symptom, Rare Etiology: A Case Metastatic Cancers of Unknown Primary Origin Presenting with Epistaxis and Gingival Bleeding

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Abstract

Introduction: Epistaxis and gingival bleeding are among the most common presentation to the emergency department for patients with thrombocytopenia. Here, we present a case who was admitted to the emergency department with thrombocytopenia and was diagnosed with metastatic cancer of unknown primary origin.

Case Report: A 26-year-old male patient was admitted to the emergency department with gingival bleeding and epistaxis. The body temperature was 38.3 °C. Petechial rash, ecchymosis or organomegaly was not detected on physical examination. Laboratory results revealed thrombocytopenia as 31x 10^3 (159-388 x 10^3/µL). Although hemoglobin and leukocyte counts were normal, no band or precursor cell was observed in the patient's peripheral blood smear. There was no history of weight loss, night sweats, arthritis, malar rash, photosensitivity, contact with ticks, animals, or a COVID-19 patient. Serological tests performed for infections such as HIV, EBV, HCV, Crimean-Congo hemorrhagic fever were negative. Bone marrow biopsy was performed due to the unexplained cytopenia, reported as "signet ring cell metastatic adenocarcinoma". Gastrointestinal system endoscopy was performed to detect primary cancer. A biopsy was taken from the antrum and corpus revealed gastritis. An FDG PET-CT was revealed heterogeneously pathologically increased FDG attitude in all axial and appendicular bones. Despite all the modalities of diagnosis, the origin was not found and the patient was transferred to the oncology department for treatment with a diagnosis of cancer of unknown origin with bone marrow infiltration.

Conclusion: Bone marrow metastases should be kept in mind in patients presenting with thrombocytopenia.

Key words: epistaxis,gingival bleeding,thrombocytopenia,Bone Marrow metastases of Unknown Primary Origin

Introduction

Metastatic cancer of unknown primary origin (MCUP) that is 1-2% of all cancer types, is defined by metastatic tumors that primary origin not able to be detected despite all patient's history, laboratory findings and radiological imagine methods[1,2]. %60 of these tumors are adenocarcinoma and more common in male patients around 60 years old[3,4]. Survival of 25 % of the patients is less than one year[5]. Clinical presentation of the heterogeneous tumor group is depends on the site of metastases. While liver and thoracic metastases are mostly detected at older age, brain and bone metastases are seen more in younger population[3].

Bone marrow metastases are seen less than 10% of solid tumors that mainly originated from lung, breast and prostate cancers[6]. According to the article published by Kılıçkap et al, the most common cancers that metastasize to bone marrow are the breast (28%) and lung cancer (23%) while tumors of unknown primary origin are 8 %[7].

Signs and symptoms of the patients with bone marrow metastases are mostly related to cytopenia. Most frequent findings are anemia and thrombocytopenia, respectively[8,9]. Patients can be complicated with bleeding and infections.

In this report, we present a case who was admitted to emergency department (ER) with fever, epistaxis and gingival bleeding, which are frequent complaints of admissions to ER. The patient had thrombocytopenia in the first-line of laboratory tests and diagnosed with MCUP with further examinations. In this perspective, the literature regarding the clinical approach and the etiology is discussed.

Case Presentation

26 year-old male with no significant medical history was admitted to ER with epistaxis and gingival bleeding. Concomitantly he had backache and loss of appetite. There was no history of fever, weight loss, night sweats, arthritis, ma-

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lar rash, photosensitivity, contact with ticks / animals and with a COVID-19 patient. On admission, he was febrile with a temperature of 38°C, with otherwise unremarkable vital signs. In physical examinations, minimal leaky epistaxis and gingival bleeding were detected. There were no petechia or ecchymosis all over the skin. The liver and the spleen were not palpable. In the first step laboratory examination; hemoglobin was 13.1 mg/dl (11.7-15.5), white blood cell count was 8700/ µL (4100-11200), platelet count was 31x10^3/µL (159-388x10^3), aPTT was 29.1 sec (22.5-32) and Prothrombin Time (INR) was 1.47 (0.8-1.2). Dipstick protein (+) and 64 erythrocytes were seen in the complete urinalysis. In the peripheral smear examination, 1-3 % fragmented erythrocytes and 2-3 thrombocytes per high power field are seen thus pseudothrombocytopenia was ruled out. In leukocyte subtype analysis; neutrophil, lymphocyte and monocyte percentages were 70, 20 and 8 respectively. The other remarkable laboratory test results are seen in Table-1.

Since thrombotic thrombocytopenic purpura (TTP) was in the differential diagnosis, ADAMTS-13 enzyme activity test specimen was sent to a private laboratory. Because the patient were from endemic area of the Crimean Congo Hemorrhagic Fever (CCHF), the blood sample was sent to The Public Health Laboratory for PCR. The patient was transferred to the internal medicine ward with high suspicion of CCHF and ribavirin treatment started. On the third day of the treatment, CCHF PCR was reported as negative and ribavirin stopped. Bone marrow biopsy was performed due to unexplained cytopenia. The pathology was metastatic signet ring cell adenocarcinoma. Gastrointestinal system endoscopy performed that revealed hyperemic and edematous antrum and corpus in line with gastritis. Tumor FDG PET-BT was taken to the patient to investigate the primary origin of the tumor. Heterogeneously pathologically increased FDG attitude was detected in all axial and appendicular bones.

Despite all the modalities of diagnosis, the primary origin of the tumor was not able to be detected and the patient was transferred to the oncology department for treatment of CUP with bone marrow infiltration.

Table-1.

	Patient's Value on admission	Reference
ALT (U/L)	86	<50
AST (U/L)	53	<50
ALP (U/L)	663	30-120
GGT (U/L)	28	<55
Bilirubin,total (mg/dl)	4,35	0,3-1,2
Bilirubin,indirect (mg/dl)	3,74	0-1,2
INR	1,47	0,8-1,47
aPTT (sec)	29,1	22,5-32
Fibrinogen (mg/dl)	139,2	180-350
D-dimer (mg/L)	25, 4	0-0,55
LDH (U/L)	519	<248
B2 microglobulin (ng/ml)	1108	609-2366
Na (mEq/l)	139	136-146
K (mEq/l)	3,9	3,5-5,1
Total Ca (mg/dl)	9,7	8,8-10,6
P (mg/dl)	3	2,5-4,5
Albumin (g/dl)	5	3,5-5,2
Globulin (g/dl)	3	1,5-4,6
BUN (mg/dl)	22	6-20
Creatinine (mg/dl)	0,65	0,67-1,17

ALT: alanine aminotransferase, AST: aspartate aminotransferase, ALP: alkaline phosphatase, GGT: gama glutamil transferase, INR: international normalized ratio, aPTT: activated partial thromboplastin time, LDH: lactate dehydrogenase, Na: sodium, K: potassium, P: phosphorus, BUN: blood urea nitrogen

Discussion

Admissions to ER with epistaxis or gingival bleeding are not uncommon[10,11]. The complete blood count is the first laboratory examination for the patients with bleeding diathesis, thus thrombocytopenia can be detected. Thrombocytopenia is defined by thrombocyte count is less than 150x10^3/µL. Reasons of thrombocytopenia are divided into 3 main groups: decreased production, increased destruction and splenic sequestration. Two main situations can explain decreased production in the bone marrow; bone marrow failures, like aplastic anemia and myelodysplastic syndrome, and bone marrow infiltrations such as leukemia, lymphoma, multiple myeloma, and metastatic tumors. Disseminated intravascular coagulation (DIC), thrombotic thrombocytopenic purpura (TTP) and portal hypertension are examples for increased destruction and splenic sequestration, respectively[12].

Thrombotic microangiopathies should be considered if thrombocytopenia and hemolytic markers (increased lactate dehydrogenase, decreased haptoglobulin, schistocytes in peripheral blood smear etc.) are present. Since TTP causes major organ damage rapidly and threatens life, it should be noticed in the early period and treatment should be initiated as soon as possible. It is valuable for the ER physicians to suspect TTP and take blood sample for ADAMTS-13 enzyme activity as soon as possible. DIC can be excluded by normal prothrombin time and activated partial thromboplastin time. In our case, thrombotic microangiopathies were excluded with absence of hemolytic markers and normal level of ADAMTS-13 enzyme activity.

Although CCHF was thought in the differential diagnosis as the patient came from an endemic region with fever and bleeding, it was excluded due to the PCR negativity.

Bone marrow biopsy is not recommended routinely for isolated thrombocytopenia[12]. If thrombocytopenia cannot be

explained despite all detailed patient history, physical examination, peripheral blood smear, hemolytic markers, liver and kidney function tests and viral serology; bone marrow biopsy should be performed.

Though bone marrow involvement is more frequent in hematologic malignancies, bone marrow metastasis is seen in 0.17- 1.19 % of solid tumors[13]. The most common solid tumors infiltrating bone marrow are lung, prostate, and breast cancers[14]. Although it is known that bone marrow metastases of solid tumors are seen in advanced stages, it were seen as the initial presentation in the case series of 25 patients reported in 1993 by Wong et al.[8]

Median life expectancy of the patients is five months after cancer diagnosis. Survival of 28% of the patients is only one year and thrombocytopenia is a predictor for poor prognosis[7]. Low mean thrombocyte volume may be a sign of bone marrow metastases of solid tumors[15]. When anemia, thrombocytopenia, increased alkaline phosphatase, hypercalcemia or leukoerythroblastic syndrome at peripheral blood smear are detected in chemo-radiotherapy naive solid tumor patients, bone marrow should be analyzed in terms of metastasis[16].

Conclusion

Epistaxis and gingival bleeding are common complaints for admission to ER. Complete blood count and peripheral blood smear are the first step laboratory examinations. In patients with thrombocytopenia, a wide differential diagnosis should be made by a clinical workup. Although MCUP with bone marrow metastases is rare (1-2% of all cancers) it should be kept in mind in patients with thrombocytopenia.

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Conflict of Interest

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Case Report

Journal of Emergency Medicine Case Reports

Iatrogenic Right Heart Failure Due To Central Vein Catheterization: A Rare Complication

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Abstract

Introduction: Central venous catheterization (CVC) is an invasive procedure used for many reasons in clinics such as hemodynamic monitoring, fluid and drug administration and hemodialysis. It is an unlikely problem to forget the central catheter wire (CW) in the intravascular area and this has dangerous consequences. Causes such as inattention, inexperience, time constraints due to workload, fatigue and the absence of accompaniment by a more experienced clinician are the main reasons for this complication.

Case Report: In this article, we present a case of forgotten CW starting from the subclavian vein and making a ring around the tricuspid valve in the right atrium, extending to the vena iliaca communis dextra. This prevented tricuspid valve closure and led to decompensated right heart failure. This complication was not reported previously.

Conclusion: CVC is a commonly used method. It provides great benefit in follow-up and treatment. It should be kept in mind that forgotten wires can cause right heart failure secondary to mechanical tension on the tricuspid valve as one of its complications.

Key words: Central venous catheterization, right heart failure, forgotten catheter wire

Introduction

Central venous catherization (CVC) is an invasive procedure that is frequently used in intensive care with many indications such as hemodynamic monitoring, total parenteral nutrition support, long inotropic and irritant drug administration and hemodialysis[1]. Besides the advantages of the CVC procedure, it can also cause different complications such as arterial puncture, catheter malposition, vascular erosion, air and thrombus embolism, pneumothorax, cardiac perforation, and cardiac tamponade[2]. In this article, we present a case of forgotten catheter wire (CW) starting from subclavian vein and making a ring around the tricuspid valve in the right atrium, extending to the vena iliaca communis dextra, preventing the tricuspid valve closure and leading to decompensated right heart failure. Also in this context, the reasons for forgetting the guide wire and the precautions to be taken to prevent it are discussed.

Case

A 69-year-old male patient was admitted to the emergency department with complaints of increasing breathlessness, cold sweats and swelling in the feet for the last three months. On physical examination, general condition was moderate, conscious and agitated. Vital findings were blood pressure; 140/90 mmHg, pulse; 115 beats/min, respiratory rate 14 breaths/minute, body temperature; 36.5 C. Pulmonary sounds were natural. A 3/6 systolic murmur was heard at the tricuspid focus. Jugular venous fullness and bilateral 2 (+) pretibial edema was found. In laboratory tests; glucose: 82 mg/dl, BUN: 37 mg/dl, Cre: 1,1 mg/dl, AST: 51 U/L, ALT: 58 U/L, GGT: 38 U/L, ALP: 131 U/L, Na: 142 mmol/l, K: 2.6 mmol/l, troponin 0.01 n /ml, WBC: 9000 / UL, hemoglobin: 12.7 g/dl, Hct: 36.9% were found. Electrocardiography (ECG) had no features other than sinus tachycardia. Echocardiography was planned to evaluate the cardiac function of the patient. Echocardiography revealed an ejection fraction of 35%, fibrotic tricuspid valve, third degree tricuspid insufficiency with color Doppler, and foreign body obstruction which prevent the closing of the flaps. Contrast-enhanced thoracic tomography for advanced examination of the patient revealed a CW continuing to the superior vena cava from the subclavian vena cava, making a ring around the tricuspid valve and preventing the tricuspid valves closing with mechanical pressure, and advancing toward to inferior vena cava and vena iliaca communis dextra (Figures 1,2,3,). After this, the patient's anamnesis

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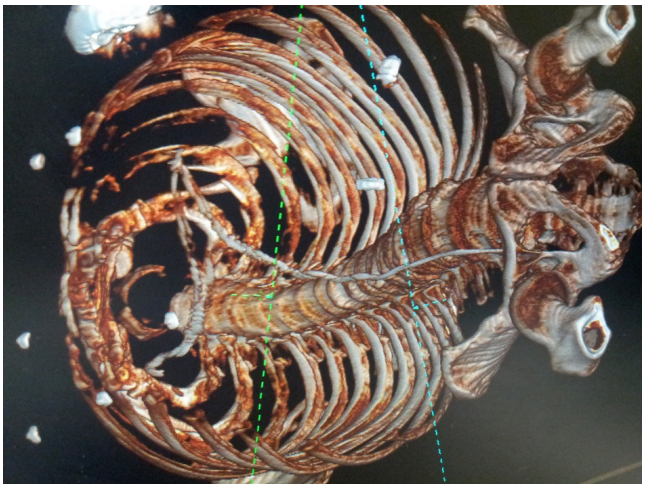
was deepened and it was learned that the patient was hospitalized for one week in the intensive care unit due to acute renal failure and a hemodialysis catheter was inserted using subclavian vein for hemodialysis three months previous. The patient complained that his feet started to swell, shortness of breath evolved, and gradually increased after he was discharged. Decompensated right heart failure which developed secondary to hemodialysis catheter, which was forgotten in the patient. was diagnosed. The patient consulted with a cardiovascular surgeon to remove the catheter wire. With the recommendation of the cardiovascular surgery specialist, the patient was referred to the center for removal of the CW. The central catheter of the patient was organized and a frequent follow-up with antiaggregant and anticoagulant treatment was planned because of the risk of removal. The patient was called for check-up 2 times a year.

Discussion

CVC is a frequently-used invasive procedure, especially in patients monitored in intensive care. Internal jugular vein, subclavian vein, and femoral vein are the anatomic structures used for CVC. Many factors such as local skin lesions, coagulopathy, infection, anatomical integrity, and edema play a role in the choice of the anatomy to use by the clinician[3]. However, if the correct technique cannot be applied, it can cause serious complications. Complications such as arterial puncture, pneumothorax, hemothorax, hydrothorax, chylothorax, catheter malposition, air and thrombus embolism, arrhythmia, hematoma, cardiac perforation, and trauma to neighboring nerves and vessels may develop during central venous catheterization. In addition, there may be catheter-related thrombosis, coagulopathies, and catheter-related infections after administration[4]. Risk factors that facilitate the development of complications include, poor technical use, poor quality of the material used, a body mass index less than 20 kg/m2 or more than 30kg/m2, presence of coagulopathy, hemodialysis catheter with large lumen,



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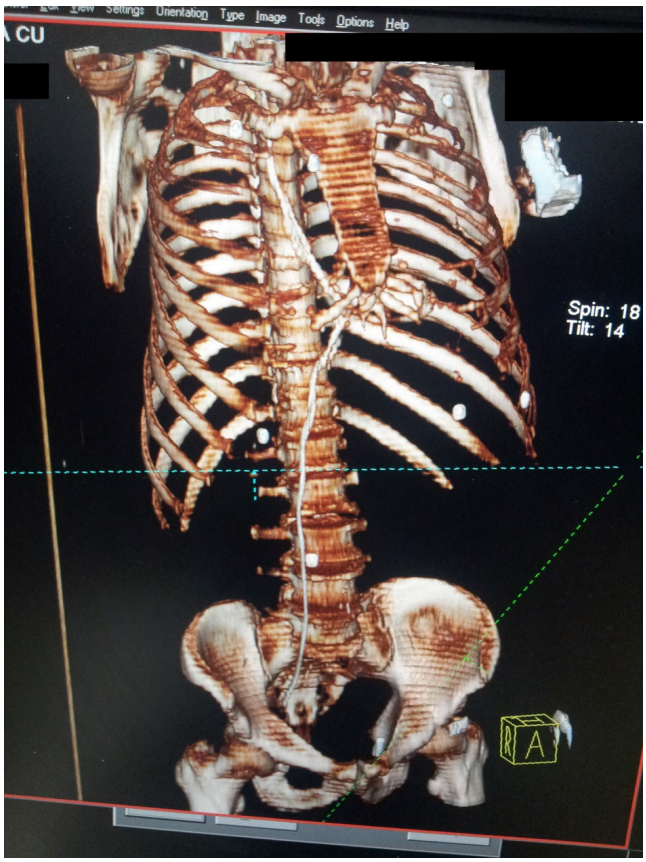

Figure 1:

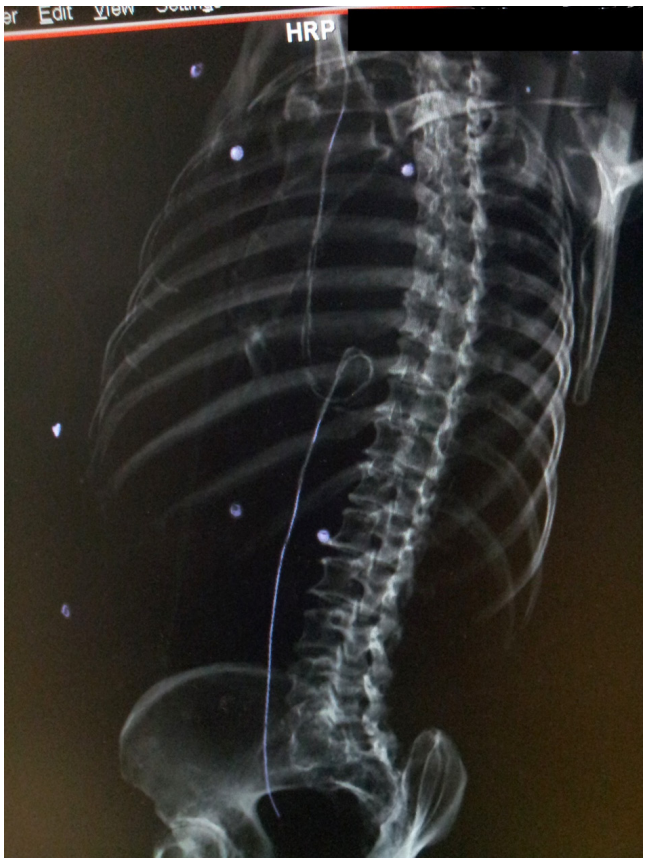

Figure 3:

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and false anatomical site selection. Rarely, central CW can be seen to be twisted, knotted, broken, forgotten or not retracted[5]. But this complication was not reported previously. The complication of the wire continuing to the superior vena cava from the subclavian vena cava, making a ring around the tricuspid valve and preventing the tricuspid valves closing with mechanical pressure, and advancing toward the inferior vena cava and vena iliaca communis dextra may occur, as reported in our case. The reason for forgetting the CW in the vascular area is carelessness, inexperience, haste, fatigue, multiple failed attempts and inadequate supervision by experienced clinicians[5]. In a study conducted by Taylor et al., it was reported that the presence of an experienced person reduced the complication rate by half. In the same study, the complication rate increased sixfold in cases of continuing the procedure after three and more unsuccessful attempts[6]. Complicated cases of forgotten CW in the vascular area which can be removed with noninvasive methods using Dormier basket and endovascular forceps under local anesthesia or with invasive methods such as venous exploration, laparotomy, thoracotomy under general anesthesia were reported[5]. Resistance should not be encountered during insertion of the CVC, and it should be withdrawn and retried if encountered. In addition, the catheter should never be advanced without seeing the distal lumen of the guide wire. After the process, the guidewire should be checked so it is completely removed and has no broken parts, and the CW should never be left until the end of the process. Inability to find the CW after the procedure, resistance to injection in the distal catheter lumen and poor venous return show that the CW was forgotten in the intravascular area[7]. Ultrasonically guided catheterization and checking the radiological accuracy of the catheter site after the procedure are among the recommended methods to reduce complications[8].

In conclusion, CVC is a commonly-used method. It provides great benefit in follow-up and treatment. It is an invasive application that increases the morbidity and mortality of the patient if it is not implemented correctly. It should be kept in mind that the forgotten wire can cause right heart failure secondary to mechanical tension on the tricuspid valve as one of its complications. For this reason, the CVC procedure should be done carefully and any probable previous procedure should be questioned in the presence of suspicious symptoms. Anamnesis should be taken in this way and with great care. Further investigation should be planned in case of doubt.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Effect Of Covid-19: Cerebral Venous Sinus Thrombosis Case Presentation

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Abstract

Introduction: Many studies have shown that coagulation disorders and increased risk of thrombosis may occur during coronavirus disease 2019 (COVID-19) infection. Although cardiac or pulmonary vascular pathologies has been detected in most cases, cerebral sinus thrombosis are rare. During COVID-19 infection patients rarely present with neurological symptoms.

Case Report: A 19-year-old man was admitted to our emergency department with neurological symptoms. Cerebral venous sinus thrombosis (CVST) was detected in Brain Computerized Tomography and Magnetic Resonance Imaging examinations. Our patient was hospitalized in the neurology department of our hospital for medical treatment and was discharged after clinical recovery.

Discussion: In this case report we wanted to draw attention to cerebral venous sinus thrombosis which is a rare but treatable complication of COVID-19 infection in a young patient. We examined our patient in the light of literature.

Conclusion: Clinicians should keep in mind the diagnosis of CVST that may occur due to infection associated thrombosis in COVID-19 patients presenting with neurological symptoms and consider adding anticoagulants to the treatment if necessary.

Key words: cerebral venous sinus thrombosis, coronovirus, antikoagulant, MR Venography

Introduction

The SARS-CoV-2 (Severe Acute Respiratory Syndrome causing Coronavirus) infection which was first detected in Wuhan city of China in 2019 caused a pandemic all over the World[1].

Hypercoagulability state in COVID-19 (Coronavirus Disease Infection-2019) is predicted to be hyperviscosity secondary to hypoxia. Cellular infection initiates localized inflammation, endothelial activation, tissue damage and irregular cytokine release. The septic situation which develops with an increase in leukocytes and platelets causes coagulopathy and eventually diffuse intravascular coagulation. Therefore many organ failures may occur. Most commonly cases of pulmonary embolism and cardiac thrombosis have been identified. Cases of COVID-19 associated cerebral venous sinus thrombosis (CVST) are rare in the literature[2,3,4]. In this article we aimed to investigate, the rare case of COVID-19 associated CVST in a young patient.

Case

A 19-year-old male patient was admitted to our emergency department (ED) with severe headache and vomiting. On his medical history; ten days ago, he was admitted to the internal medicine unit of our hospital with mild headache, multiple joints pain and fever. After normal blood test and brain computed tomography (CT) (Fig. 1) results, he was tested for COVID-19 with a PCR test. He received five days of antiviral treatment (Hydroxychloroquine+Favipiravir) with a positive test result. The patient was non-smoker and had no comorbidity. He had undergone an operation of right parietal lobe of brain for traumatic injury 15 years ago. On examination his vital signs were stable. There were no signs of meningeal irritation or any neurological deficit. Bilateral pupillary light reflex was +/+ and ophthalmoscope examination revealed mild papillary edema on the left. Blood tests showed leukocytosis (15.57 10^9/L), lymphopenia (0.63 10^9/L), elevated D-dimer (1.52 mg/L) and normal CRP (0.18 mg/dl) level. Observation of hyperdense appearance in confluence sinuum and transverse sinuses (especially on the left) on brain CT (Fig. 1), led to performing contrast-enhanced brain MRI (magnetic resonance imaging) and contrast-enhanced MR venography (MRV) with suspicion of thrombosis. MRI and MR-venography showed filling defects in the superior sagittal sinus, transverse sinuses and left sigmoid sinus. (Fig. 2). Therewith the patient was hospitalized with the final diagnosis of dural sinus thrombosis.

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Low weight heparin treatment was started. On the 5th day of medical treatment his headache completely disappeared and control CT revealed significant decrease of hyperattenuation in the left transverse sinus. Our patient was discharged with an anticoagulant prescription, since no new neurological symptoms were observed. A follow up repeat MRI was performed three weeks after the initial MRI which demonstrated almost complete resolution of the venous thrombosis (Fig. 3).

Discussion

CVST is a rare cause of stroke due to occlusion of dural venous sinuses[5]. The risk of CVST rises up during COVID-19 infection with an increased tendency to thrombosis. A limited number of CVST cases associated with COVID-19 infection have been reported in the literature. Most of these patients are middle-aged male patients with comorbidities[6,7]. Our patient differs from the others since he is young and has no comorbidities.

The localization of the thrombosed sinus is important in the occurrence of clinical findings. The superior sagittal sinüs is the most commonly affected dural venous sinüs. However, more extensive multi-site thrombosis is the typical finding as in our case. Headache and papillary edema associated with increased intracranial pressure are prominent in CVST[8,9]. Our patient also applied with similar complaints.

Prognosis of CVST is good if diagnosed and treated early. The use of anticoagulants in patients with CVST decreases morbidity[10]. The prognosis of our patient was very good.

For patients who have severe headache at admission to the ED, the most commonly used examination is brain CT. CT scans are useful to rule out the presence of haemorrhage. CVST is often overlooked on initial CT scans. Attention should be paid to signs of clot such as the dense clot sign and the empty delta sign. Brain MRI is superior to CT in detecting CVST. The presence of increased signal in the dural sinuses on multiplanar T2 Weighted (W), T1W and FLAIR sequences associated with the disappearance of normal flow suggests the diagnosis. Contrast-enhanced CT or MRI examination provides additional contribution to pre-contrast studies by showing filling defects. However, brain MRV is the most valuable non-invasive method to reveal dural sinus pathologies, especially if it is performed with contrast agent, as in our case.

Conclusion

During the COVID-19 pandemic, clinicians focus mostly on respiratory symptoms, but as the cases are examined in detail, it is noteworthy that organ damages resulting from intravascular coagulation are frequently seen. Clinicians should keep in mind the diagnosis of CVST that may occur

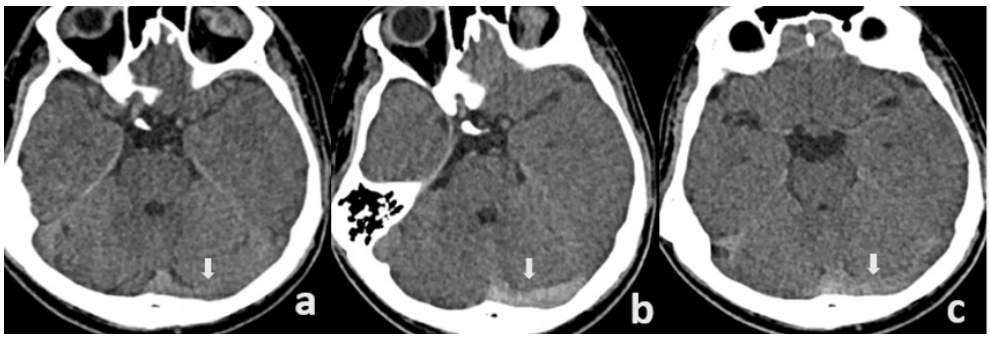

Figure 1: a. Prior non-enhanced CT which was performed 10 days ago shows no attenuation abnormality in the left transverse sinus. **b.** Non-enhanced CT performed at hospital admission demonstrates hyperdense left transverse sinus (dense clot sign). **c.** CT imaging -on the fifth day of medical treatment- indicates significant decrease of hyperattenuation in the left transverse sinus.

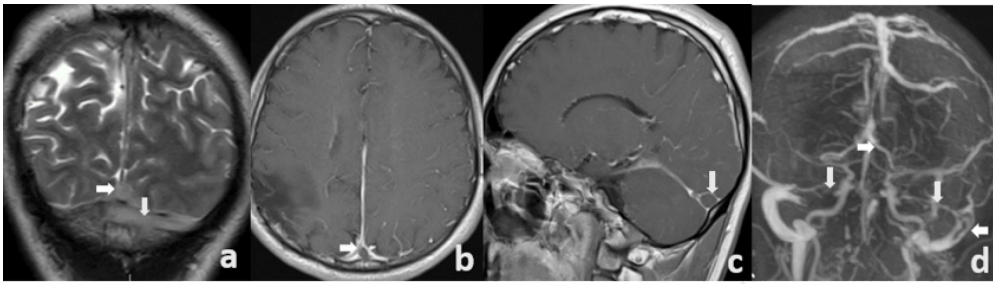

Figure 2: a. Absence of normal flow void in the left transverse sinus and distal superior sagittal sinus on pre-contrast T2 weighted image (coronal plane). **b-c.** Post-contrast axial and sagittal plane T1 weighted images show filling defects of superior sagittal sinus and left transverse sinüs due to thrombosis (empty delta sign). Note the expanded transverse sinus. **d.** Coronal MIP projection of contrast-enhanced MR Venography demonstrates non-visualization of the left transverse sinüs, medial part of the right transverse sinüs and superior sagittal sinüs. Additionally filling defect was observed in the left sigmoid sinus.

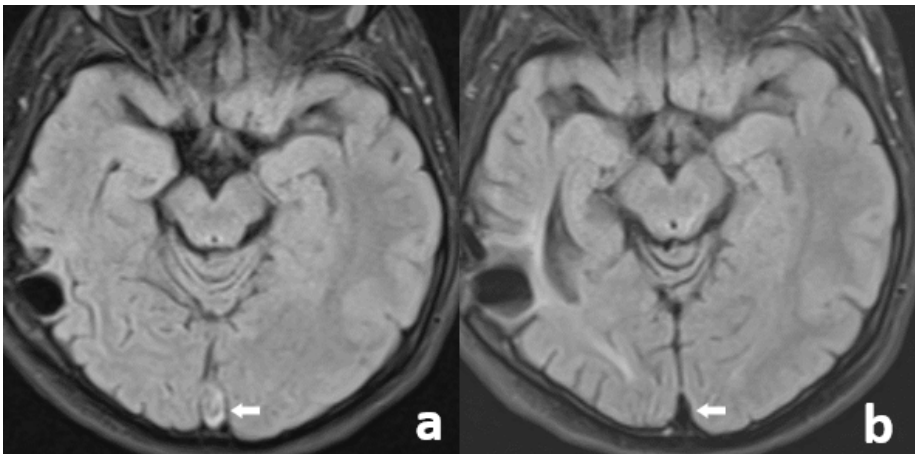

Figure 3: a. Absence of normal flow void in the superior sagittal sinüs on FLAIR image. Note the expanded sinüs. **b.** Normal flow void in the same localisation on the third week of medical treatment.

due to infection associated thrombosis in COVID-19 patients presenting with neurological symptoms and consider adding anticoagulants to the treatment if necessary.

The case report has written in an anonymous characteristic, thus secret and detailed data about the patient has removed. Editor and reviewers can know and see these detailed data. These data are backed up by editor and by reviewers.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Case of Patient Forgetting His Native Language with the Diagnosis of "Posterior Reversible Encephalopathy Syndrome"

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Abstract

"Posterior Reversible Encephalopathy Syndrome" is a reversible rare disease manifesting with a variety of neurological findings often accompanied by high blood pressures. Here we present a case of forty-nine years old male patient who was admitted to our emergency department with the complaint of having difficulty speaking his native language (Turkish) and later diagnosed with PRES Syndrome. Based on the interesting nature and rare occurrence of the case, the etiology, physical exam findings, and differential diagnosis of the disease are presented and discussed in light of the literature.

Keywords: Posterior reversible encephalopathy syndrome, emergency department, posterior leukoencephalopathy syndrome, hypertensive encephalopathy.

Background

"Posterior Reversible Encephalopathy Syndrome" (PRES) is a syndrome referring to a disorder of reversible vasogenic brain edema. Clinical features include a variety of acute neurological symptoms from headaches to seizures. Renal failure, fluctuations in blood pressure, some cytotoxic drugs, autoimmune disorders, and preeclampsia or eclampsia are associated with PRES.

Pathophysiology of PRES is considered to be caused by endothelial injury related to the disorder of cerebrovascular autoregulation resulting in brain edema. Another hypothesis is blood-brain barrier dysfunction caused by a rapid increase of arterial blood pressure leading to hyperperfusion of the posterior brain and producing vasogenic edema. Its radiographic and clinical features are usually reversible and usually, the disease has a favorable outcome[1-4]. Neurological impairments such as encephalopathy, cognitive deficits, or coma may be presented[3]. At magnetic resonance imagining (MRI) of the brain, radiological findings referring to PRES are mainly located at the parietooccipital lobe, posterior frontal lobe, temporal lobe, and at some less common areas as well[4].

Diagnostic criteria of PRES have not been established but suggested criteria by Fugate et al. are neurological symptoms of acute onset, (focal) vasogenic edema on neuroimaging, and reversibility of clinical and/or radiological findings[5].

Our main goal to present this case is to attract the attention of emergency department staff and readers on the diagnosis since it is rare, interesting, and could be manifested with other various neurological and clinical deficits.

Presentation of the Case

We report a forty-nine years old male patient who gets peritoneal dialysis treatment due to hypertension and chronic kidney failure was brought to our emergency department by his relatives because of his confusion started the day before, confusing his clothes, thinking his wife's shoes are his own, and losing the ability to speak his native language, Turkish, yet still able speaking English with his wife who is a native English speaker.

On admission, the patient's general condition was fine, conscious, oriented, cooperated, normal speech and motor output, comprehension preserved to a large extent, however, the patient was unable to follow complex commands, naming and repetition was impaired for both Turkish and English. The patient's other physical examination findings were normal. Blood pressure was 240/170 mmHg (mean arterial pressure: 193 mmHg) and other vital signs as pulse, fever, partial oxygen saturation and respiratory rate were normal. Metabolic causes of encephalopathy such as uremic encephalopathy were excluded in the differential diagnosis regarding the patient's laboratory values were at his baseline values. (e-gfr: 4,31 mg/lt/24h, BUN: 58, Glucose: 102, Na:134 mmol/L).

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Computerized brain tomography and diffusion-weighted magnetic resonance scan performed at the time of patient's admission, intracranial hemorrhage, and stroke were excluded, no acute pathological finding was observed.

Intravenous Esmolol therapy was initiated because of continuous hypertension since normal blood pressure goals could not be achieved by antihypertensive therapy given. Nephrology department consultation was requested for further treatment options. The patient was transferred to peritoneal dialysis treatment in the emergency department and peroral Captopril was added to intravenous Esmolol treatment. The patient's blood pressure was gradually reduced to 155/100 mmHg at the end of the 24th hour.

Bilateral hyperintensities observed mainly in the occipitoparietal, cerebellar, and brain stem in conventional brain magnetic resonance imaging performed the next day following admission founded to be compatible with PRES.

Outcome and follow-up

After blood pressure was regulated, the patient's confusion regressed, his ability to speak in his native language (Turkish) and his neurological condition was restored.

The patient's clinical findings were resolved after the 24th hour of his admission. The patient was admitted to the Nephrology department for further examination and treatment. He was clinically asymptomatic on the 3rd day of his admission and discharged without any neurological deficit.

Discussion

We report this PRES case since its unusual clinical features make it both rare and interesting.

Our patient was admitted to our clinic with the complaint of confusion, disarranging his clothes, thinking his wife's were his own shoes, and losing the ability to speak his native language Turkish, yet speaking English with his native English speaking wife. He was suffering from hypertension and in his brain MRI occipitoparietal, cerebral, and bilateral hyperintensities in the brainstem were detected. He was discharged to home with full recovery following hospitalization and advanced treatment in the nephrology department following ours.

The differential diagnosis of PRES is broad, and history might be limited. Venous sinus thrombosis or subdural, intracerebral or subarachnoid hemorrhage, autoimmune encephalitis, and metabolic encephalopathies such as deranged serum glucose, sodium (including central pontine myelinolysis), uremia, or drug toxicity (for example cyclosporine) can also have similar progressive symptoms[3].

Other than MRI, diagnostic tools such as EEG and lumbar puncture are found to be useful[5]. Seizures are a common feature of PRES, however, our patient did not have any seizure episodes and clinically recovering so, no further testing was ordered.

Various neurological findings related to the etiology of PRES have been reported in the literature, yet our case presented with an unusual symptom of not being able to speak his native language and being able to speak the foreign language (English) he spoke with his wife at home. After excluding the clinical findings such as hemorrhagic and ischemic stroke and uremic encephalopathy, PRES syndrome should be considered in the differential diagnosis in hypertensive patients with the presence of similar symptoms.

In the beginning, although his main complaint was the inability to speak his native language seems shocking, it is not known whether the dominant language has changed in the bilingual process, although the patient's native language is Turkish. Therefore it was observed that the patient, who had limited cognitive status in general, actually had difficulties at naming and repetition in speaking both languages, yet probably because of him speaking English every day with his wife recently, he received a low score in naming Turkish words[2-3].

Conclusion

After ruling out uremic encephalopathy, hemorrhagic and/or ischemic cerebrovascular events among patients who admitted to an emergency department with hypertensive emergencies, inability to speak their native language, and impaired speaking ability, the diagnosis of PRES, should be kept in mind though it is a rare case.

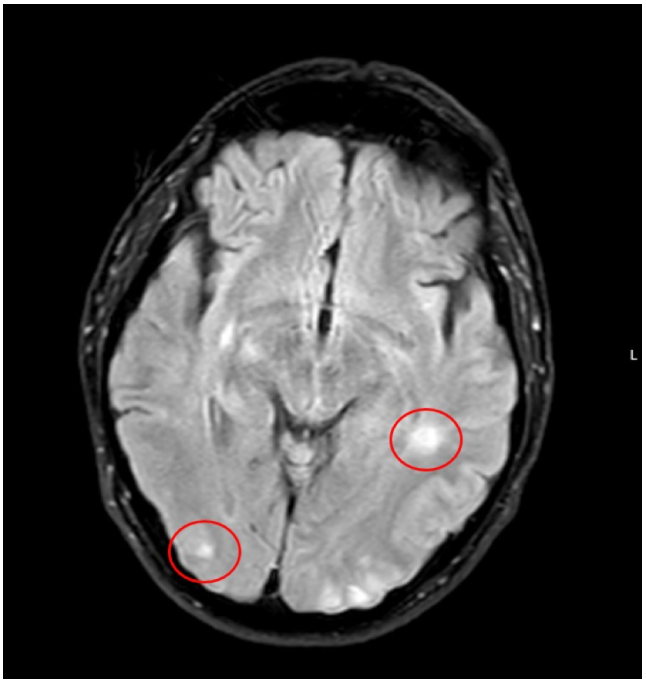

Figure 1: Fluid-attenuated inversion recovery image of brain MRI showing bilateral occipito-parietal white matter hyperintensities.

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