E-ISSN
e-ISSN 2149-9934
Volume: 11 **Issue:** 4 December 2020

Journal of Emergency Medicine Case Reports

EPAT
1999
Emergency Physicians Association of Turkey

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Editorial

Dear colleagues,

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

Prof. Dr. Başar Cander

Değerli Meslektaşlarımız

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

Prof. Dr. Başar Cander

Contents

Case Report

Journal of Emergency Medicine Case Reports

A Rare Case of Pneumobilia Caused By Ingestion of Methyl Ethyl Ketone Peroxide

Ömer TAŞKIN[1], Ufuk AKDAY[2], Gökhan SAĞLAMOL[3], Ayça AÇIKALIN[1]
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Abstract

Introduction: Methyl Ethyl Ketone Peroxide (MEKP) is a highly toxic substance which is used as a solvent. MEKP causes morbidity and mortality by leading to severe metabolic acidosis, kidney failure and liver failure due to necrosis of the hepatocytes. In this case report we aim to discuss the clinical and radiological findings of an accidental MEKP poisoning.

Case Report: A 64-year-old male was referred to our clinic after accidentalingestion of a corrosive substance. The vital signs were normal and fluid treatment was started. The patient was hospitalized after the computerized tomography scan (CT) showed severe esophagitis and pneumobilia. The CT scan revelaed regression on the 3rd day of hospitalization. The patient was discharged by his own will on the eighth day before providing total recovery.

Conclusion: Unlike other corrosive substances, MEKP may cause intra-abdominal free air such as pneumobilia. An early CT scan helps to evaluate the need for emergency surgical intervention and may prevent patients from unnecessary surgery.

Keywords: Methyl ethyl ketone peroxide, Pneumobilia, Computerized Tomography

Introduction

Methyl Ethyl Ketone Peroxide (MEKP) is a highly toxic substance which is a clear fluid without any colour. It is used as a solvent, a hardener in the production of resins, synthetic rubber and other petrochemical plastics. It can be drunk by accident because of its water like appearence[1]. MEKP is a strong oxidizer and corrosive agent. The exposure of MEKP leads to free radical formation and this results in lipid peroxidation. The lipid peroxidation may cause dysfunction in cellular level, specially liver and multiple organ failure[2]. MEKP causes liver failure due to necrosis of the hepatocytes, kidney failure, severe metabolic acidosis, edema of the pharynx and larynx, toxic inhalation pneumonitis, corrosive esophagitis, gastrointestinal tract bleeding and hollow organ perforation due to its corrosive effect[3,4]. In the chronic period, it may cause severe strictures on multiple sections of the gastrointestinal tract as well[5]. Gastrointestinal system endoscopy is an important traditional method in the diagnostic staging and treatment of corrosive esophagitis and gastritis.

In this case, we aim to report formation of pneumobilia after exposure of MEKP and to discuss utilization of computed tomography (CT) scan which will be more informative than traditional methods.

Case Report

A 64-year-old male who is a constructor was referred to our clinic with the complaints of burning in the mouth and back of his sternum, nausea and vomiting after intake of a solvent fluid by accident. The patient applied to our clinic on the third hour of intake. His vitals were stable and in normal limits, the Glaskow Coma Scale score was 15. The physical examination revealed hyperemia in oropharynx and he had tenderness in epigastrium. His electrocardiyogram (ECG) was normal sinus rhytm. The bottle containing the fluid he had drunk as labelled as Methyl Ethyl Ketone Peroxide (MEKP). His blood tests revealed normal biochemical and hematologic parameters, but metabolic acidosis (pH: 7,23; pCO2: 45mmHg; hCO3:17 mmol/L). On his chest and abdomen x-ray there was neither sign of perforation nor other pathologies. His oral intake was stopped and daily fluid treatment was started.

After the increase in retrosternal and epigastric pain, he was scanned with intravenous (IV) contrast enhanced abdominal computed tomography (CT). His CT revealed diffuse thick heterogeneous appearance at all levels of esophageal walls and increase in wall thickness. In addition to air densities and para-esophageal fluid densities at the hiatus level near the esophageal wall, air densities (pneumobilia) in intrahepatic biliary tract were also observed (Figure 1).

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Received: 25.08.2020 • **Accepted:** 26.10.2020
DOI: 10.33706/jemcr.785272

After the consultation with the general surgery department, he transferred to the intensive care unit for observation and treatment. The IV contrasted abdominal CT scan of the third day showed regression of all findings (Figure 2). After the regression of his complaints, he discharged from the hospital by his own will.

Discussion

Common presentations and diagnosis after MEKP intoxications are corrosive esophagitis, gastritis and hepatic necrosis[6]. The traditional diagnostic method of corrosive esophagitis is endoscopy. However, the CT scan is more informative for transmural damage than endoscopy in these patients. The CT imaging provides information about the entire gastrointestinal tract and is very useful in excluding perforation. There are similar CT findings reported in the literature, emphysematous widespread gas formation in the bile ducts and organ walls in the gastrointestinal tract after MEKP exposure[6]. The CT scan is very useful to determine the indication for surgery by the evaluation of gastrointestinal tract, detection of perforation, edema and emphysematous gas formation in the liver and biliary tract.

The gastric decontamination is contraindicated in the patients with MEKP exposure like other corrosive intakes. N-Acetylcysteine is recommended in the patients with acute liver failure and hemodialysis is recommended in the patients with kidney failure[6,7]. To the best of our knowledge of the 30 patients with MEKP exposure reported in the literature, almost all had gastrointestinal tract and liver damage and 10 resulted with mortality[5,8].

In the case of Jung Oh Chang et al, MEKP exposure had caused gastrointestinal tract damage and liver necrosis[6]. The authors claimed that early endoscopy and CT scan can be used to identify perforation and bleeding in gastrointestinal tract. Endoscopy is diagnostic but CT scan be used to assess the urgent need for surgery. More than 50-100 mililiters (ml) of intake is mortal[8,9].The drinken amount was nearly 10 ml in our case. Our patient had severe esophagitis in the acute period but acute organ failure did not develop. We believe that the small amount of intake resulted in better response to treatmentandgood outcome.

Conclusion

MEKP is an agent that may cause death due to multiple organ failure in addition to the corrosive esophagitis. CT imaging in these patients will be more informative than traditional methods and will also help to evaluate the need for emergency surgical intervention.

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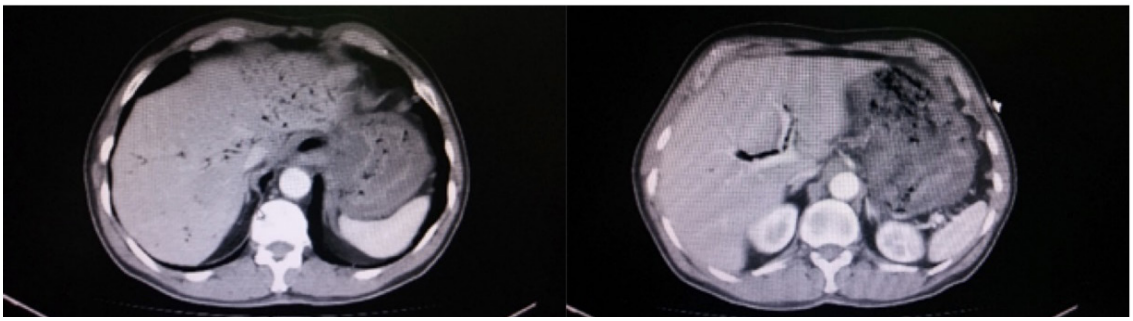

Figure 1: CT image indicating pneumobilia and esophageal wall thickening at day 1 after MEKP accidental oral intake

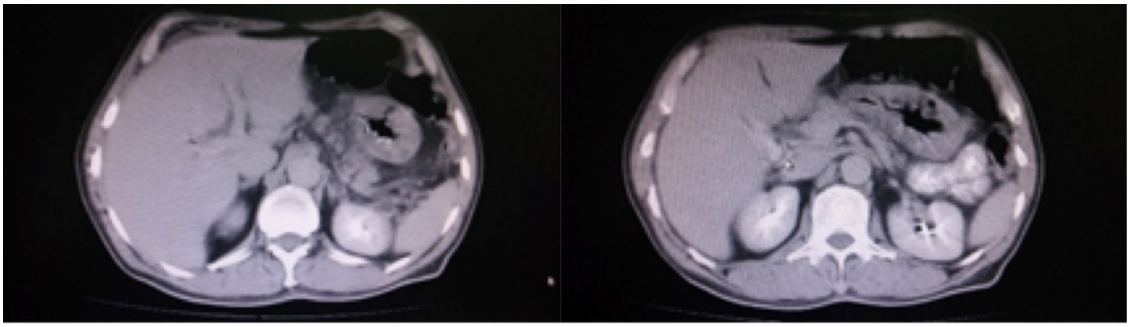

Figure 2: Control CT image at the 3rd day of hospitalization

DOI: 10.33706/jemcr.785272
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Case Report

Journal of Emergency Medicine Case Reports

Oral Snake Skin Resulting in Anaphylaxis: How and Why?

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Abstract

Introduction: The use of snake skin, both transdermal and orally comes from ancient Chinese medicine. People eat snake skin for skin disorders, convulsions, gallbladder disorders and hypertension. People also apply snake skin by transdermal way for skin disorders such as sores,boils, itching, psoriasis, scabies, hemorrhoids, eye infections, cloudy spots in the eye..etc. There isn't enough reliable information available about snake skin to know if it is safe or what the side effects might be.

Case: The patient described in the case report has given his informed consent for publication. We present a case of anaphylaxis developed after 10 days of snake skin eating and dicharged after 4 days treatment with full recovery.

Conclusion: It must be kept in mind that natural products are not always necessarily safe and also dosages can be important. Availability of exotic foods for different uses is steadily increasing. No matter how developed we are, those who try traditional medicine methods instead of chemical drugs will always be. So we must be careful and always be awake for different food consumptions.

Keywords: Allergens, anaphylaxis, angioedema, snake skin, traditional treatments

Introduction

Availability of exotic foods for different uses is steadily increasing. The use of snake skin, both transdermal and orally comes from ancient Chinese medicine. People eat snake skin for skin disorders, convulsions, gallbladder disorders and hypertension. No matter how developed we are, those who try traditional medicine methods instead of chemical drugs will always be So, it must be kept in mind that natural products are not always necessarily safe and also dosages can be important.

Case Presentation

A 36-year-old 75 kilos man with no medical history presented to the emergency department complaining of common urticaria, itching and mild shortness of breath. He had itch in the mouth and troat, facial and body urticaria, angioedema and breating difficulties (Picture 1). In physical examination wheezing was heard due to bronchoconstriction and uvula edema was seen. He had mild hypotension (90/60 mmHg) and heart rate was 110/min. Intramuscular adrenalin 0,5 mg, intravenous prednisolone 80 mg and ranitidine 50 mg were applied urgently. Salbutamol 0,15 mg/kg was given for one time and nasal oxygen 4 lt/min was started. After 20 minutes, the hemodynamics were more stable (blood pressure 130/80, heart rate 105/min) and he was free of symptoms accept common body and facial urticaria (Picture 2). We also applied feniramine 45,5 mg intravenously against urticaria. He had normal sinus rhythm and no ST-T wave changes. Troponin T and creatinekinase-MB (CKMB) leves were normal. Arterial blood gas results were completely normal. Routine biochemistry and coagulation tests were normal. The percentage of eosinophils was % 6 (normal 0,0- 2,0) , white blood cell count was 14,2 10^3/µL (normal 4,8-10,8) and the other parameters of hemogram were normal. There was no pathology in chest X-ray. He told he hasn't used any medication and has not consumed any different nutrients recently. At the end of 6-hour follow-up at emergency service, he still had urticarial lessions all over the body , so he was hospitalized prednisolone 160 mg/day and feniramine 45,5 mg/day was ordered. Despite this treatment, urticarial lesions were still same the next day. They decreased after drug administer but when the drug effect is over they were same again. Serum tryptase and allergen-spesific IgE levels are not measured in our hospital so we couldn't evaluate

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Received: 27.08.2020 • **Accepted:** 04.11.2020

DOI: 10.33706/jemcr.786358

DOI: 10.33706/jemcr.786358
Journal of Emergency Medicine Case Reports 2020; 11(4): 95-97

them. We added oral cetirizine 10 mg of single dose in the evenings. On the second day of his hospitalization,we asked if he ate any different nutrient again and then he telled us that he has been eating snake skin for the last 10 days. He has been treated for perianal warts for about 20 days with drugs and electrocoterization by general surgeon and his lesions were about to disappear. After being recommended by relatives to eat snake skin with meals everyday to treat the warts, he bought shed snake skin which was found on mountain skirts. Then he started to consume snake skin by smashing it over his dinner. Another factor in allergy etiology has not been found so we attributed the current clinical picture to snake skin consumption. After 4 days of treatment, he was free of symptoms and discharged with full recovery. He was also advised to use oral cetirizine 10 mg for about 10 days after discharge.

Discussion

To the best of our knowledge, this is the first report of allergic reaction to snake skin which was taken orally. Availability of exotic foods for different uses is steadily increasing as our anaphylaxis example due to snake skin eating. Allergies to food and environmental antigens have steeply grown to epidemic proportions. The most common cause of anaphylaxis are foods. Nutrients are responsible for 33.2% - 56% of anaphylaxis cases[1]. The frequency of foods that cause anaphylaxis varies regionally.

The use of snake skin, both transdermal and orally comes from ancient Chinese medicine. People eat snake skin for skin disorders, convulsions, gallbladder disorders and hypertension. People also apply snake skin by transdermal way for skin disorders such as sores,boils, itching, psoriasis, scabies, hemorrhoids, eye infections, cloudy spots in the eye.. etc. But there is no scientific evidence to rate snake skin for these uses. It is told that snake skin is used in ointments and creams in order to reduce pain and stiffness in some places. There isn't enough reliable information available about snake skin to know if it is safe or what the side effects might be.

In new terminology, anaphylaxis formed by immunological mechanisms such as IgE, IgG, immunocomplex and complement system is defined as immunological anaphylaxis[2]. IgE antibodies are key mediators of allergic disease, including life-threatening anaphylaxis[3].

The epidermis of snakes is a multi-layered system consisting of keratin and associated ß-proteins[4]. In our patient, symptoms appeared approximately 10 days after the first snake skin consumption. This late allergic reaction may be due to late digestion or non-digestion of keratin in the human gastrointestinal system.

Dietery metal exposure can be readily detected in shed snake skins, including at trace levels of exposure that may be consistent with, or below, environmental exposures. Lead, cadmium and mercury are frequently evaluated as part of wildlife bioaccumulation and health monitoring studies[5]. In developing countries, different herbal or animal resources are frequently consumed as healing and medicine for different diseases. So we have to be careful for heavy metals also.

A study showed that the thickness and lipid concent of shed snake skin and human stratum corneum were not significantly different ($p>0.05$), whereas the water content of shed snake skin was significantly lower than that of human stratum corneum ($p<0.05$) [6]. Low water content may make digestion more difficult when snake skin is consumed as

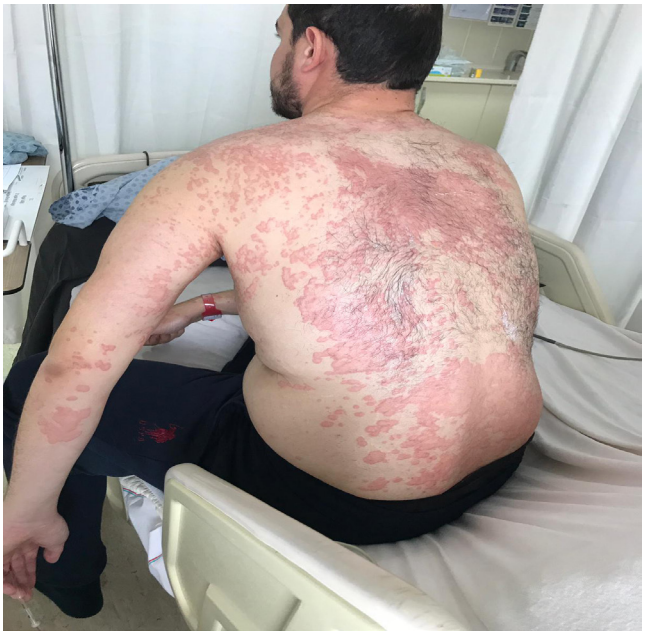

Figure 1: Common urticaria at arrival to emergency service

Figure 2: 20 minutes after the first treatment common urticaria still exists

food and maybe this late digestion could mean longer contact with allergen in our patient.

In the beginning of covid-19 pandemic, there were rumors that the Coronavirus outbreak in China has been caused by a freshwater snake that has been frequently eaten there. The discovery of bioactive remains found in freshwater is thought to be proof of the beginning of biological warfare. It is likely that the current virtual information will be confirmed but it needs more investigation and real scientific publications than possibilities. It should not be dismissed that any food consumed outside the routine can have many risks such as allergies, infections, epidemics..etc.

Conclusion

In developing countries, different herbal or animal resources are frequently consumed as healing and medicine for different diseases. It must be kept in mind that natural products are not always necessarily safe and also dosages can be important. We must be careful against traditional treatment materials (both animal ve herbal) especially those that are not widely consumed because of acute anaphylaxis risk and both known and unknown toxic side effects due to chronic exposure.

Conflict of interest: The authors have no conflict of interest.

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Case Report

Journal of Emergency Medicine Case Reports

An Unusual Occurrence of Acute Cerebellar Infarct After Self-Cervical Manipulation: A Case Report

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Abstract

Introduction: Spinal manipulation is a widely used method in the treatment of neck pain, but it has the potential for serious complications. Although controversial, stroke can occur especially after cervical manipulation. Stroke secondary to self-cervical manipulation is rare and only a few cases have been reported.

Case Report: We reported a 28-year-old male patient with complaints of dizziness, neck pain and right hemiparesthesia. Except the cerebellar gait and right hemiparesthesia, his neurological examination was normal. It was learnt that his complaints started an hour after the self-cervical manipulation. His brain computed tomography, brain and cervical computed tomography angiography were normal limits. The brain diffusion magnetic resonance imaging (MRI) revealed an acute infarction area. He was discharged without sequelae with antiaggregant treatment, after 8 days of follow-up in the hospital.

Conclusion: Although it is controversial whether the risk of stroke increases after cervical manipulation, we believe the risk of stroke may increase, especially when the manipulation is performed by non-professionals. Especially in younger patients with a history of cervical manipulation and neurological complaints, stroke should be suspected even if the neurological examination is normal.

Keywords: Stroke, cerebellar infarction, chiropractic manipulation, Self neck manipulation

Introduction

Stroke, which is one of the leading causes of morbidity and mortality in the world, is divided into two groups as ischemic and hemorrhagic. Ischemic stroke is 4 times more common. Various causes such as atherosclerosis, cardioembolism, genetics, trauma and arterial dissection play a role in the etiology of ischemic stroke[1].

Manual cervical spinal manipulation, the place of which is being discussed in the etiology of stroke, is a common treatment technique, especially for neck pain. However, although the results have been controversial, it may cause complications, such as cervical artery dissection (CAD), vertebral artery dissection (VAD), vertebrobasilar insufficiency (VBI), or a cerebellar or brain stem infarction[2-4]. In the literature, several cases of infarction have been reported after chiropractic therapy and, to a lesser extent, after self-cervical manipulation. And all of these cases are associated with vascular pathologies[5-8].

In this case report, we present a 28-year-old male patient with an unusual case of cerebellar infarction, without any vascular pathology who admitted to our emergency department with neck pain, dizziness and right hemiparesthesia.

Case Presentation

A 28-year-old male patient with no known medical history or drug use was admitted to our emergency department with complaints of dizziness, neck pain and right hemiparesthesia that began the previous day. It was learned from the patient's history that he was a plumber and sometimes experienced neck pain. He stated that he was not subjected to any neck trauma and was practicing self-rotational neck manipulations to relieve his neck pain. It was learnt that his complaints started an hour after the neck manipulation. The patient then applied to another emergency department the previous day but had not been diagnosed. After he was discharged from the hospital, he applied to our emergency department because his complaints did not recede. The patient's arterial blood pressure was 125/79 mmHg, heart rate was 85 beats/min, oxygen saturation was 95%, and electrocardiography (ECG) showed a normal sinus rhythm.

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Received: 02.09.2020 • **Accepted:** 12.12.2020
DOI: 10.33706/jemcr.789735

On neurological examination, there were cerebellar ataxia, hemiparesthesia in the right upper extremity, mild effacement in the left nasolabial sulcus and paresthesia in the left half of the face. Dysmetria or dysdiadochokinesia was not detected in the patient's cerebellar tests. He had no nystagmus. There were no motor findings giving sides. Routine laboratory tests, including complete blood tests, liver and kidney function tests, and electrolyte values, were within normal limits. Cervical and brain computed tomography (CT) of the patient was within normal limits. However, the brain diffusion magnetic resonance imaging (MRI) revealed an acute infarction area in the left cerebellar hemisphere inferior region (Figure 1). Brain and cervical CT angiography was performed and both vertebral arteries and the basilar artery were intact and patent. The patient was hospitalized with the diagnosis of cerebellar infarction secondary to cervical manipulation. The anticoagulant enoxaparin sodium 6000 anti-Xa IU/0.6 ml 1x1 was administered subcutaneously and antiaggregant treatment with acetylsalicylic acid (ASA) 100 mg 1x1 P.O. was given. During the follow-up examination, a transthoracic echocardiography (TTE), a transesophageal echocardiography (TEE), a bilateral carotid-vertebral color Doppler ultrasonography, and a control brain CT scan were performed and found to be normal. The patient's tests for coagulopathy, thrombophilia and connective tissue diseases (ANCA, ANA, anti-dsDNA, lupus anticoagulant, anti-Sm, anti Sm-RNP, anti-SSA, anti-SSB, antiphospholipid IgG , anticardiolipin IgG-IgM, anti beta-2 glycoprotein IgG-IgM, antithrombin III, protein c, protein s, homocysteine) were seen within normal limits. After 8 days of follow-up, the patient was discharged without any sequelae with antiaggregant treatment (ASA 100 mg 1x1 po). Informed consent was obtained from the patient for the publication of his information and images.

Discussion

Chiropractic manipulation, which is an alternative treatment method used for cervical pain, is very common worldwide and performed by licensed people in some countries[9]. However, it is difficult to determine the frequency and results of all these practices. The safety of a spinal manipulation or the incidence of complications, such as infarction and dissection, after manipulation remains unknown[10]. A case-controlled study found that spinal manipulation therapy is associated with VAD, regardless of pain, even if it controls neck pain[11]. In the literature, there have been several infarction cases reported following chiropractic treatment and self-cervical manipulation. The common feature of these cases is that they all have vascular pathologies such as vertebral or cervical or cerebral artery occlusion or dissection[5-8]. Contrary to these cases, no findings in favor of dissection or occlusion were found as a result of imaging performed in our case. Both the carotid arteries and vertebral arteries were intact. As in our case (28-year-old), the history of neck manipulation, especially in young patients without comorbid disease, suggests that the cause of the stroke may be manipulation. Rothwell et al. founded that patients under the age of 45 had an increased risk between vertebral artery dissections or occlusions after cervical manipulation[2]. However, it was stated in a systematic review and meta-analysis that there is a weak relationship between cervical neck manipulation and cervical artery dissection, but studies in the literature may be

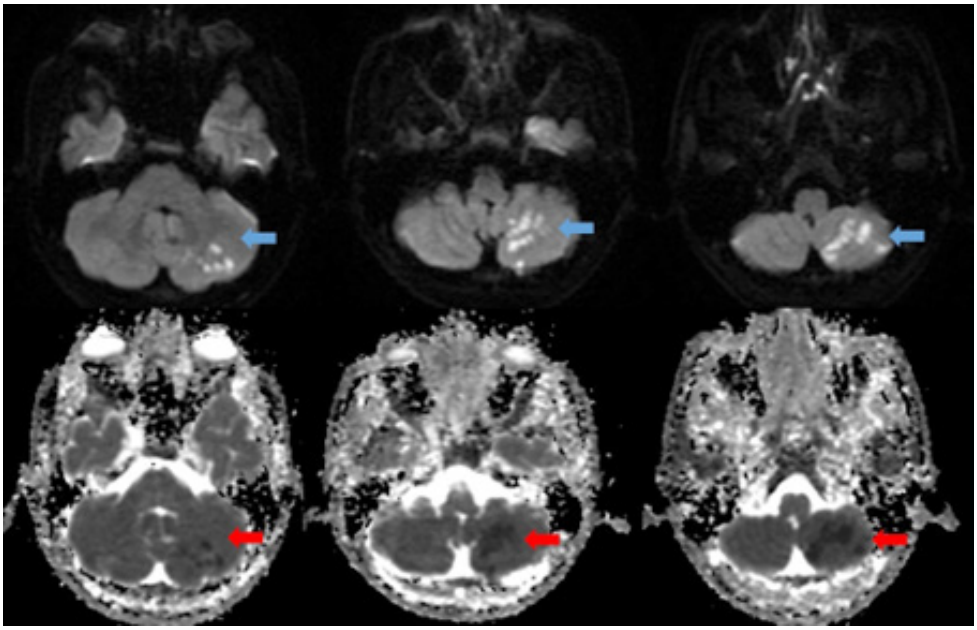

Figure 1: The acute infarct area in the left cerebellar hemisphere, is indicated by the arrow on the diffusion-weighted imaging (DWI) and Apparent diffusion coefficient (ADC) sequence in the diffusion MRI.

DOI: 10.33706/jemcr.789735
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biased and there is no causal evidence[12]. In a cross randomized controlled trial published in May 2019, MRI measurements were performed to investigate the effects of cervical manipulation on vertebral arteries and cerebral perfusion. The results suggest that cervical manipulation did not cause cerebral perfusion changes compared with a neutral neck position or a maximal neck rotation, and it may not increase the risk of cerebrovascular events with a hemodynamic mechanism[13]. However, as in our case, the possible risk of injury following manipulation, especially when performed by non-professionals, cannot be ruled out. As no etiology was found to explain the infarction in our case, such as any vascular pathology, infection, connective tissue disease, we concluded that cervical manipulation may be the etiological cause of this infarct. A study by the American Heart Association and American Stroke Society (AHA/ASA) in 2014 suggests that patients should be informed about the cause and effect relationship before manipulation since the risk of cervical dissection secondary to cervical manipulation treatment is not clear[14]. Since the effects and complications of cervical manipulation are very controversial, studies with a higher degree of scientific evidence are needed.

Conclusion

Stroke is a common cause of emergency admissions, and the patient's story may not always be clear. Clinicians should keep all kinds of etiology in mind. Although it is controversial whether the risk of stroke increases after cervical manipulation, we believe the risk of stroke may increase, especially when the manipulation is performed by non-professionals.

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Case Report

Journal of Emergency Medicine Case Reports

Is Computed Tomography the Gold Standard in Aortic Dissection?

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Abstract

Introduction: Aortic dissection is a disease with high mortality, which is characterized by a tear in the aortic wall. Thanks to early diagnosis and treatment, patients' survival rates are high. Chest pain is the most common symptom. Imaging methods help in diagnosis. Its treatment is surgery.

Case Report: A 47-year-old male patient was admitted to the emergency department with chest pain. The diagnosis of aortic dissection in computed tomography was evaluated as motion artifact and valve motion, and he was asked to be discharged from the emergency service after his diagnosis was missed. However, transesophageal echocardiography was performed because of the patient's clinical symptom and echocardiographic findings supported the aortic dissection. When a dissection flap was seen in transesophageal echocardiography, the patient was transferred to surgery. In surgery, a dacron graft was placed in the patient's aorta and a prosthetic valve was placed on the aortic valve, and left main coronary repair and right coronary is bypass were performed. The patient was discharged without any problem.

Conclusion: Aortic dissection is a clinical diagnosis, it is a disease with high mortality. Imaging methods are helpful in diagnosis, but the fact that imaging methods rule out dissection does not always rule out the disease. The important thing is to suspect the disease and to consider the patient's current clinical symptoms and signs.

Keywords: Aortic dissection, computed tomography, transesophageal echocardiography, surgery.

Introduction

Aortic dissection is the filling of blood into the aortic wall as a result of a tear in the aortic intima. Aortic dissection is one of the real emergencies due to high mortality. Therefore, diagnosis and treatment should not be delayed. It is frequently seen in men between the ages of 40-70. The most common cause of aortic dissection is uncontrolled hypertension[1]. Autoimmune diseases, bicuspid aorta, aortic aneurysm history, chronic constipation, pregnancy, congenital anomalies, blunt traumas, connective tissue diseases are among other etiological reasons[2-3]. The typical clinical picture of aortic dissection is very severe chest pain in the form of tearing or rupture starting from the anterior chest wall and other clinical symptoms that increase with the progress of the dissection. Clinical findings occur as a result of the effects of branches separated from the aorta. While neurological symptoms such as syncope and stroke are prominent in proximal aortic involvement, mesenteric ischemia, lower extremity sensory and motor losses, and renal failure may occur in distal aortic involvement[4].

Case report

A 47-year-old male patient, who had no known history of systemic or coronary disease and had a history of smoking one pack per day for 30 years, was examined in another center 1 week ago due to chest pain and syncope, and no cardiac pathology was found. Computed tomographic (CT) angiography was performed with a preliminary diagnosis of aortic dissection, but it was evaluated by radiology as motion artifact and valve motion, and aortic dissection was not considered (Figure 1a). During his admission to our center for control purposes, the patient had atypical chest pain, hemodynamically stable, and there was no pathological feature on electrocardiography. In echocardiography, the ejection fraction was 60%, there was no wall motion disorder, but the patient had a 0.5 cm pericardial effusion in the posterior and lateral wall. Advanced aortic insufficiency was detected without an organic pathology in the aortic valve. The ascending aorta was not wide and measured 40 mm from its widest point. After the rupture of a fibrous band at the sinotubular junction and a suspicious dissection image, the patient underwent CT angiography again (Figure 1b). It was evaluated by the radiology

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Received: 03.09.2020 • **Accepted:** 04.11.2020
DOI: 10.33706/jemcr.789856

DOI: 10.33706/jemcr.789856
Journal of Emergency Medicine Case Reports 2020; 11(4): 101-103

as motion artifact and valve motion again and dissection was not considered. The patient was consulted with cardiovascular surgery, and the patient was asked to be discharged from the emergency department, since dissection was not considered as a result of tomography. However, due to the dissection image and high clinical suspicion in the echocardiography performed by the cardiology, the patient was performed transesophageal echocardiography (TEE) and a dissection image starting from the sinotubular junction was observed (Figure 1c). At the same time, advanced aortic regurgitation was detected that completely filled the left ventricular outflow tract (LVOT) (Figure 1d). In the operation performed on the patient, it was observed that the aortic wall was teared up to the tunica adventitia, the tear extended to the left main coronary and right coronary ostium, and extended to the pulmonary artery and limited itself by developing hematoma (Figure 2). A dacron graft was placed in the aorta, and then a prosthetic aortic valve was placed. The left main coronary ostium was repaired and the right coronary artery was bypassed saphenous (Figure 3). The patient, whose postoperative follow-up did not develop any problems, was discharged.

Discussion

Chest pain is the most common clinical presentation of aortic dissection. However, symptoms vary in some patients according to the involvement level of the aorta. In dissections involving the proximal part of the aorta, such as Type A dissection, the ostium of the coronary arteries may also be affected by this tear. Patients can sometimes present to the emergency department with the clinic of myocardial infarction with ST segment elevation, while in some patients, electrocardiographic changes have not yet occurred at an early stage, and the coro-

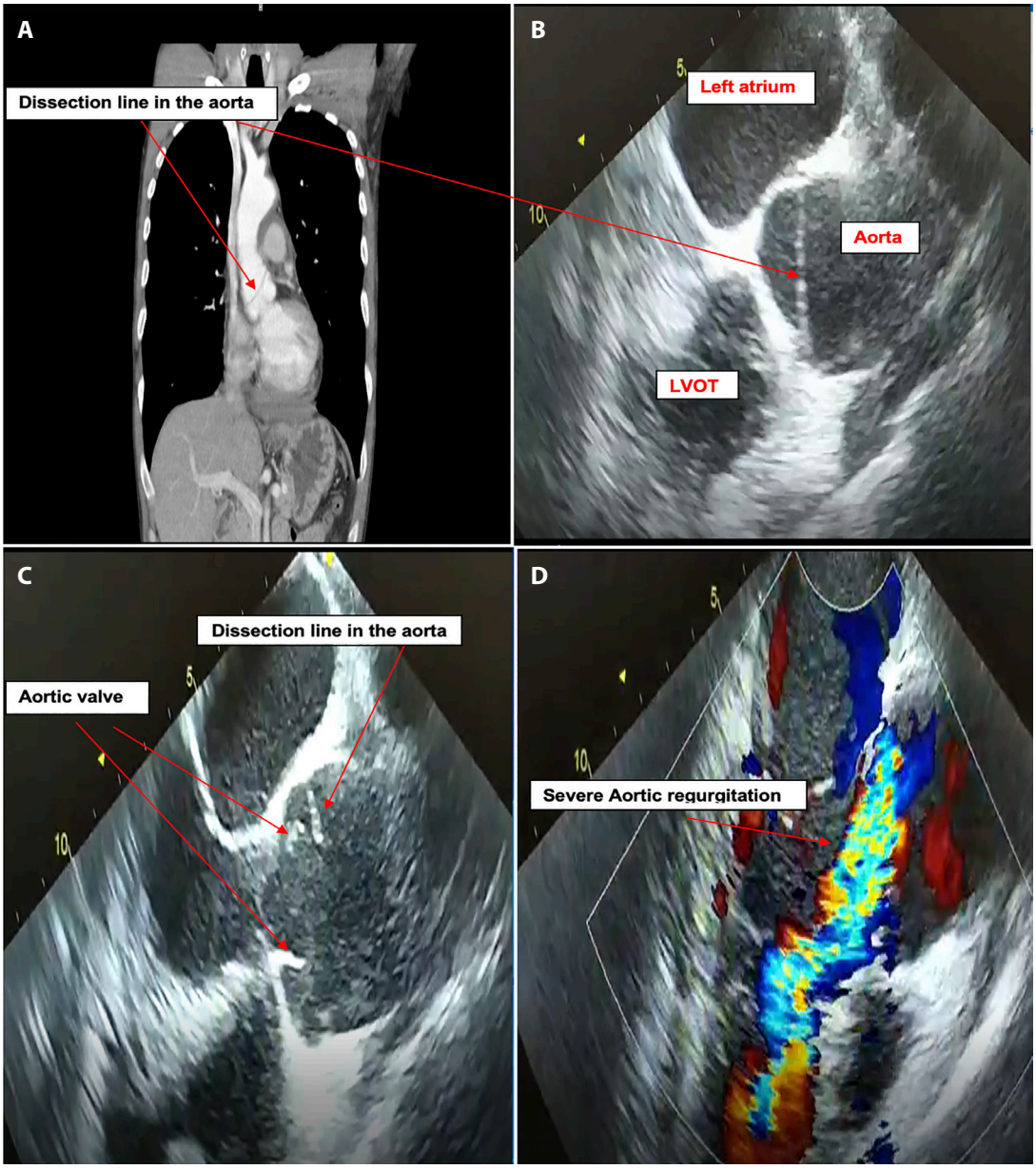

Figure 1: **A;** Dissection line is seen on tomography. **B- C;** Transesophageal echocardiography shows a dissection line. **D;** Transesophageal echocardiography shows severe aortic regurgitation.

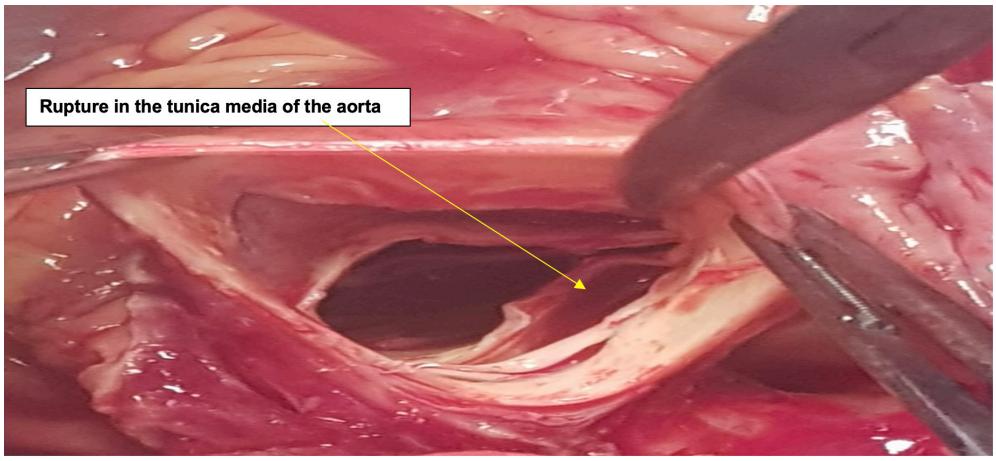

Figure 2: The tear in the aortic wall appears to extend to the tunica adventitia.

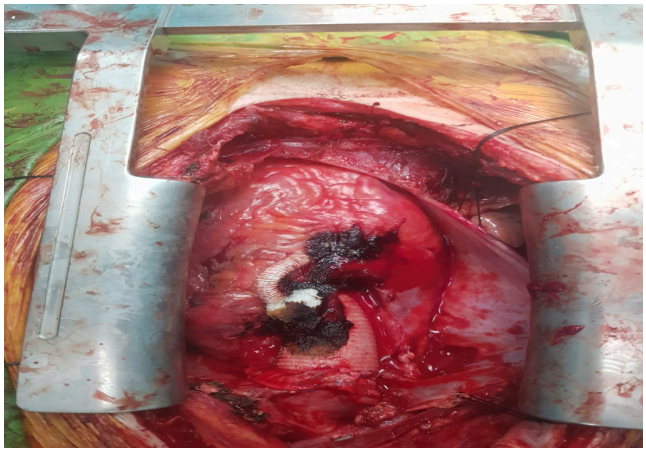

Figure 3: Repair of the coronary ostium

nary ostium may close with the advancement of the dissection line in the following hours. With the closure of the coronary ostia, mortality increases further. The right coronary artery is the most commonly involved coronary ostia, and patients may present with inferior myocardial infarction[5]. Routine coronary angiography in these patients is still controversial. In the studies performed, no significant difference was found between the patients who underwent preoperative coronary angiography and those who did not undergo mortality, length of stay and discharge[6]. Surgical techniques such as Bentall, David or Cabrol can be applied depending on the involvement of the aortic valve and the distance of the flap to the coronary ostium[7]. Dissections starting from the aortic root are sometimes accompanied by aortic insufficiency. Aortic dissection should be kept in mind especially in patients who develop severe valve insufficiency without primary valve pathology. Aortography, magnetic resonance imaging (MRI), echocardiography and computed tomography can be used in the diagnosis of aortic dissection. However, considering the difficulties in the imaging technique, difficulties in transportation, and the clinical condition of the patient, MRI is not a practical method. Computed tomography is the most preferred diagnostic method because it is available in most hospitals, easy accessibility and fast results. Although computed tomography is accepted as the gold standard method for the diagnosis of dissection, it should be kept in mind that dissection diagnosis can be missed with tomography as in our case. Excluding the diagnosis of dissection by CT angiography in young patients with clinical symptoms, severe aortic regurgitation without primary valve pathology, and pericardial effusion should not exclude clinicians from this pre-diagnosis.

Conclusion

Aortic dissection is a clinical diagnosis, it is a disease with high mortality. Imaging methods are helpful in diagnosis, but the fact that imaging methods rule out dissection does not always rule out the disease. The important thing is to suspect the disease and to consider the patient's current clinical symptoms and signs.

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Case Report

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Two-edged Knife: Massive Pulmonary Embolism and Thrombolytic Contraindication

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Abstract

Introduction: Pulmonary embolism is a common cause of death among emergency department admissions, and it has a high mortality and morbidity rate. Etiological reasons are generally associated with immobility. Radiological imaging methods are at the forefront in diagnosis. Anticoagulant and thrombolytic therapy may be preferred in treatment according to the hemodynamic condition of the patient.

Case Report: A 56-year-old female patient admitted to the emergency department with sudden onset of dyspnea and syncope with a condition of cardiogenic shock, and echocardiography revealed an enlargement of the right heart chambers and impaired functions, and a tomography was performed with the pre-diagnosis of pulmonary embolism. When systemic thrombolytic therapy was contraindicated in the patient who had embolism on tomography, catheter-based thrombectomy and selective low-dose thrombolytic therapy to the pulmonary artery were administered. The patient, who became hemodynamically stable and his shock condition improved, was discharged with anticoagulant therapy.

Conclusion: When left untreated, pulmonary embolism is a disease with a high mortality rate. Although systemic thrombolytic treatments are contraindicated in some patients, successful results can be obtained with locally effective interventional treatments in these patient groups.

Keywords: Pulmonary embolism, catheter-based thrombolysis, thrombolytic therapy.

Introduction

Pulmonary embolism (PE) is the clinical condition that occurs after thrombus occlusion of the pulmonary artery and/or its branches. Pulmonary embolism is the third most common cause of death in the USA after myocardial infarction and stroke, and is frequently encountered in emergency room admissions[1]. Mortality in hospitalized patients is as high as 12% as in outpatients[2]. Genetic and acquired risk factors play a role in the occurrence of PE. Genetic factors are less common and the most common are disorders of the coagulation system. Among the acquired risk factors, conditions such as recent hospitalization history, previous surgical intervention, immobilization, cancer, hormone and steroid therapy (risk 2-3 times higher), pregnancy should be questioned[3]. It should be kept in mind that 30% of patients with pulmonary embolism do not have any risk factors[4].

Case report

A 56-year-old female patient with a history of hemorrhagic cerebrovascular accident (CVA) and cerebral artery aneurysm was admitted to the emergency room with sudden shortness of breath and syncope. Microcoil embolization was performed to the wide necked aneurysm of 10 mm diameter in the left anterior cerebral artery (ACA) and then to the saccular aneurysm of 5 mm diameter in the right anterior-inferior cerebellar artery (AICA) by interventional radiology 2 months ago. The patient was evaluated in the emergency department with a blood pressure of 90/60 mm / Hg, an oxygen-free saturation of 85%, and a pulse rate of 120 / min during admission and pH: 7.49, PCO2: 23, PO2: 56, sO2: 89 in blood gas that these values were consistent with pulmonary embolism. On physical examination, the general condition was moderate to poor, tachypnea and anxious. Electrocardiography of the patient had tachycardia (120 / min) and incomplete right bundle branch block. The patient had a troponin height (troponin: 1.27 ug / L ref: 0-0.016). In bedside echocardiography; Right heart cavities were large (Right ventricle (RV) basal diameter: 4.5 cm, D-shape left ventricle) (Figure 1a) and severe impairment in right ventricular systolic functions (RV-Sm: 9 cm/s TAPSE: 12 mm). Subsequent pulmonary computed tomography (CT) angiography revealed a filling defect consistent with embolism in both pulmonary arteries lobar and segmental branches (Figure 2). In bilateral lower extremity venous doppler ultrasonography for deep vein thrombosis of

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Received: 03.09.2020 • **Accepted:** 13.10.2020
DOI: 10.33706/jemcr.790114

the patient, acute thrombus was observed in the popliteal vein and cranial segments of the deep crural veins. The patient was taken to the cardiology intensive care unit with the diagnosis of massive pulmonary embolism. Thrombolytic therapy was considered an absolute contraindication, after consultation with neurology and neurosurgery, due to the indication for thrombolytic therapy, but had a previous hemorrhagic CVA and cerebral artery aneurysm.It was decided to give pulmonary artery thrombectomy followed by selective thrombolytic therapy to the pulmonary artery in the patient whose general condition was poor, who was hypotensive despite dobutamine 10 mcg/kg/min inotropic support and fluid therapy, and who had contraindications for systemic thrombolytic therapy. We opted for catheter-directed low-dose thrombolytic therapy as it was hemodynamically unstable. Tissue-type 10 mg plasminogen activator (tPA) was infused rapidity into the pulmonary artery via a pulmonary arterial catheter (Figure 3). Clot removal performed using a manuel catheter-directed approach. Then, the same catheter was left in the thrombosed proximally pulmonary artery and an additional 16-hour infusion of 10 mg t-PA was administered for 16 hours. While there was a significant improvement in the clinical progress of the patient, the need for inotropes gradually decreased and was discontinued after a while. No neurological adverse events occurred during the 24-hour period. Control echocardiography was performed on the patient, who had no need for inotropes and achieved significant hemodynamic improvement, regression in right heart chambers (RV basal diameter: 4 cm) and improvement in right heart systolic functions (RV-Sm: 16 cm/s TAPSE: 14 mm) were observed (Figure 1b). The patient, who was followed up in the cardiology intensive care unit for about 3 days, was in good health, hemodynamically stable, and the patient who did not develop any neurological complications, was discharged with a new generation oral anticoagulant treatment (Rivaroxaban15 mg 2*1) and was discharged after 3 weeks and a switch to a 20 mg 1 * 1 dose of rivaroxaban was planned. The patient was evaluated 3 weeks later in the cardiology outpatient clinic, and a significant clinical and echocardiographic improvement was detected.

Discussion

In pulmonary embolism, patients may have no symptoms or may present with cardiac arrest or shock. The most common symptoms are resting or exercise dyspnea, chest pain, palpitations, orthopnea, cough, and haemoptysis[5]. Chest pain, which is often of the pleuritic type. Dyspnea is often acute and rapid onset (seconds and minutes) and is more common especially in PE in the main or lobar vessels. In those with

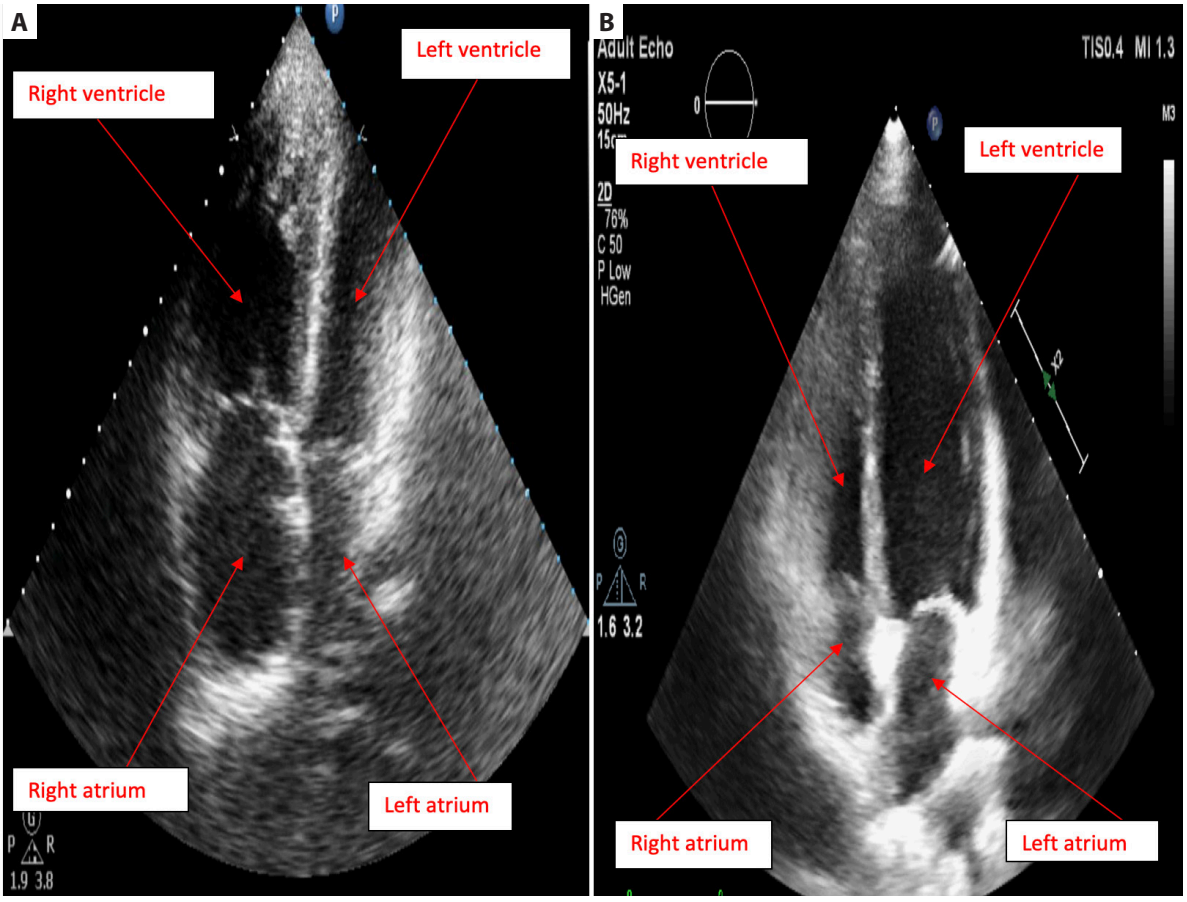

Figure 1: A; Before lytic therapy, the right atrium and right ventricle are markedly dilated. **B;** After lytic therapy, the right atrium and right ventricle are seen to return to normal sizes.

DOI: 10.33706/jemcr.790114
Journal of Emergency Medicine Case Reports 2020; 11(4): 104-107

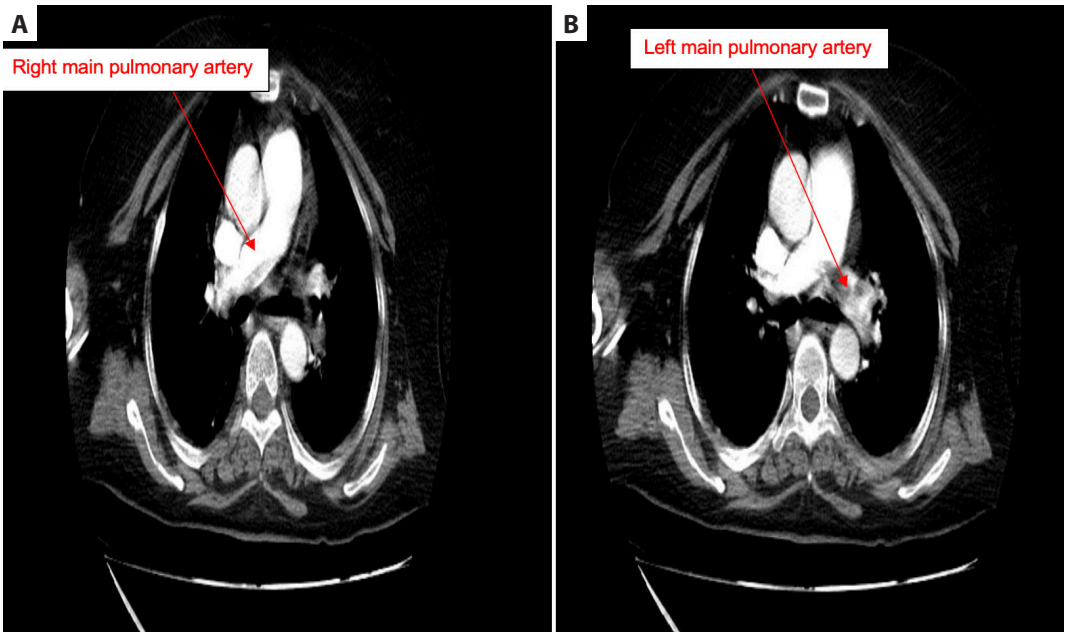

Figure 2: A; Filling defect consistent with embolism is observed in the mid-distal part of the right main pulmonary artery. **B;** The left main pulmonary artery is completely occluded with thrombus and distal filling is not observed.

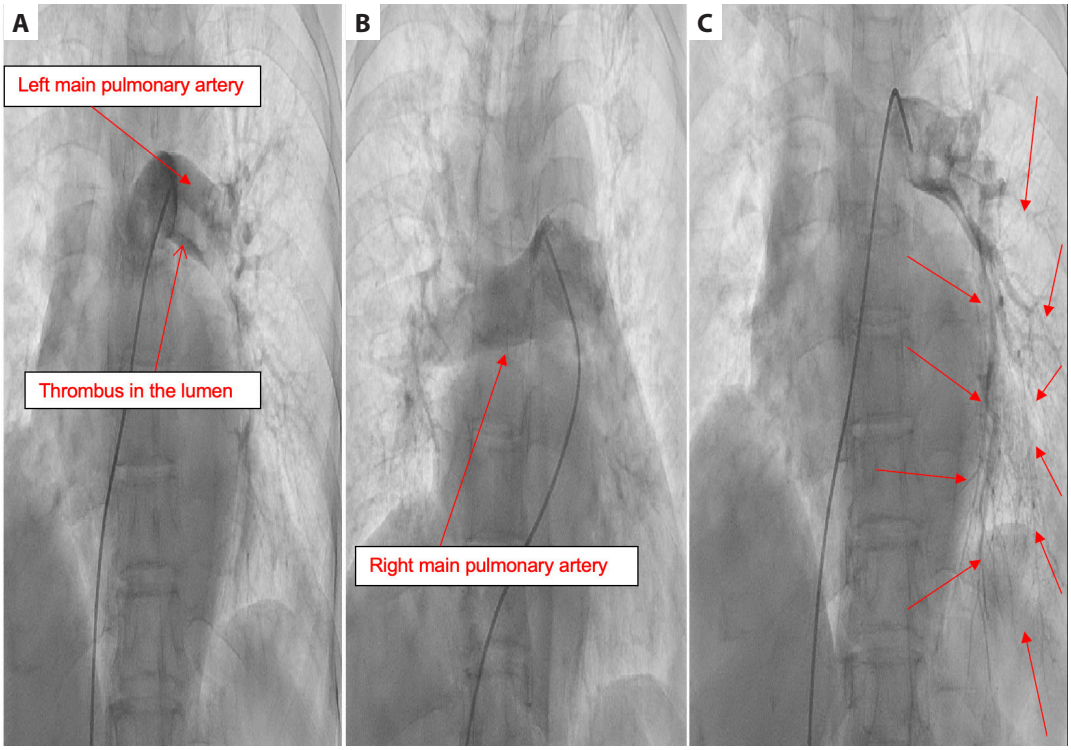

Figure 3: A; Selective pulmonary angiography. Left main pulmonary artery thrombus is completely occluded and distal filling is not observed. **B;** Filling defect consistent with embolism is observed in the mid-distal part of the right main pulmonary artery and distal weak filling was observed. **C;** Distal vascular filling was observed after lytic therapy.

heart or lung disease, an increase or worsening of shortness of breath may be the only symptom[6]. Although syncope is seen in less than 10% of patients, it may be the cause of first presentation[7]. Anamnesis, physical examination, laboratory tests and imaging methods are used in the diagnosis of pulmonary embolism. Pulmonary angiography, which was previously accepted as the gold standard method in detecting pulmonary embolism, has been replaced by pulmonary CT angiography. In pulmonary embolism, treatment strategy changes according to the clinical condition of the patient and the severity of the embolism. Hemodynamic support and respiratory support constitute the first step in treatment. Anticoagulant therapy and / or thrombolytic therapy can be applied according to risk classification and hemodynamic status. Reperfusion therapy is applied not only with systemic thrombolytic drugs, but also with percutaneous catheter-me-

diated embolectomy and surgical embolectomy. Systemic or selective thrombolytic therapy can be applied as the first choice in hemodynamically unstable patients or as a rescue therapy in patients who do not show clinical improvement despite anticoagulant therapy.Thrombolytic therapy is more effective than anticoagulant therapy in improving right ventricular function, decreasing pulmonary artery pressure and pulmonary vascular resistance, and improving clinical status. The failure rate of thrombolytic therapy has been reported as 8%, severe bleeding due to its adverse effects, 9%, and intracranial bleeding 7%[8]. In the presence of contraindications for systemic thrombolytic therapy such as recent surgery, intracranial mass, hemorrhagic cerebrovascular disease, catheter-mediated selective thrombolytic therapy has become a current issue. Mechanical reperfusion is performed by inserting a catheter into the pulmonary arteries through the femoral route, giving low-dose thrombolytics (10-24 mg). Current guidelines emphasize that reperfusion therapy can be given based on echocardiographic findings in a case of pulmonary embolism presenting with shock and hypotension in case of clinical compliance[9] The success rate considered as hemodynamic stabilization, improvement of hypoxia and discharge is higher and the risk of bleeding is lower than heparin. Systemic thrombolytic therapy has long been used as the first choice in high-risk and haemodynamically unstable patients. Bleeding complications for systemic thrombolytic therapy and the presence of contraindications for its administration have suggested catheter-based therapy (CDT) methods. According to current guidelines, CDT can be applied to high-risk patients for bleeding, unsuccessful systemic thrombolytic therapy and high-risk patients for death without systemic therapy being effective[10]. The guidelines recommend using CDT only in high-risk PE patients but most clinical studies have been conducted in intermediate-risk PE patients. In pulmonary embolism, CDT can be applied mechanically, pharmacologically and in combination of both. In our case we successfully performed mechanical thrombectomy and selective thrombolytic therapy.

Conclusion

We agree with guidelines which suggest that catheter-directed approaches are options for patients with persistent hemodynamic instability due to PE who are at moderate to high risk of bleeding. Importantly, catheter-directed techniques should be reserved for use in centers with appropriate expertise since they are not without risk (eg, further hemodynamic instability, bleeding). As in our patient, relatively lower tPA doses are now typically administered during CDT, and bleeding rates are now likely in the region of less than 4 percent. It can also be considered that the technique in this trial is different compared to more modern techniques in that the catheter is inserted into the pulmonary artery (proximal to the embolus) rather than "buried" in the embolus.

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Case Report

Journal of Emergency Medicine Case Reports

The İmportance of Hemodialysis in İntoxications with 3 Case Reports

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Abstract

Introduction: While many intoxication cases are cured with medical treatment, antidote therapy and symptomatic treatment; some cases may need hemodialysis treatment or even invasive mechanical ventilation support. We aimed to examine the place of hemodialysis in three rare intoxication cases seen in our hospital.

Case Reports: 43-year-old male patient was admitted to the emergency service with metformin intoxication. The patient with type B lactic acidosis was treated with urgent hemodialysis After a session of hemodialysis, the clinical and laboratory data returned to normal. 19-year-old female patient admitted to the emergency service with theophylline intoxication. The blood theophylline level was toxic. The patient with symptoms of intoxication (tachycardia, hypokalemia, severe nausea and vomiting, etc.) regressed after a session of hemodialysis. 22-year-old mental retarded male patient was admitted to the emergency service with loss of consciousness. The patient with metabolic acidosis was taken to the intensive care unit with coma of unknown cause. 4 sessions of hemodialysis was applied to the patient who developed acute renal failure. It was learned that the patient drank ethylene glycol (antifreeze).

Result: All our patients could be discharged with full recovery.Hemodialysis treatment has an important place in the treatment of intoxication cases.

Keywords: Intoxication, Hemodialysis, Intensive Care Unıt

Introduction

Intoxication could be very dangerous for life. Sometimes patients may be unconscious so that anamnesis cannot be taken from the patients, or they may be conscious enough to convey these complaints themselves.In our country, intoxication cases are quite common. Some of these cases may be in the form of drug-substance use for suicidal purposes, as well as drug-drug reactions or involuntary overdose of drugs. 0.91% of the patients admitted to the emergency service while intensive care unit (ICU) patients % 5.11 were intoxication cases in Turkey[1]. In addition to emergency medical treatment, reduce the absorption or increase the excretion of toxic substances from the gastrointestinal system, using specific antidotes, and to apply urgent hemodialysis treatment under certain conditions is required.

In intoxication cases; If the substance taken develops acute kidney damage, causes severe clinical symptoms and natural renal clearance is insufficient, hemodialysis is required. Continuous renal replacement therapy can be performed in the intensive care unit, as well as short-term renal replacement therapy when hemodynamic parameters are appropriate.

We aimed to present a case of ethylene glycol intoxication, which caused acute kidney damage as a result of involuntary use and was successfully treated as a result of sequential hemodialysis treatments, with two cases who received single-session hemodialysis due to severe symptoms resulting from suicidal metformin and theophylline intoxication.

Case report

Case Report 1: A 43-year-old male patient, who had no known illness, applied to the emergency service of Kastamonu Training and Research Hospital, after taking 50 tablets of the preparation containing 1 gram of metformin for suicide. The patient's consciousness was confused, Glaskow Coma Scale (GCS): 10, Pulse: 100 / min, respiratory rate: 26 / min, arterial blood pressure: 126/66 mmhg. Laboratory data: glucose: 126 mg / dl, ph: 7.25, pCO2: 29.4 mmhg, p02: 90 mmhg, HCO3: 20.3 mmol / L, lactate: 13.8 mmol / l, creatine: 1.93 mg / dl, aspartate transaminase (AST): 32 u / l, alanine transaminase (ALT): 45 u / l, potassium: 6.83 mEg / l. The patient was consulted poison counseling; He was administered activated charcoal and intravenous fluid therapy. The patient was admitted to the intensive care unit due to the severe type of lactic acidosis and GCS: 10. A hemodialysis catheter was placed in the intensive care unit, and she was taken for 4 hours without ultrafiltration After 2 hours following hemodialysis treatment,

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Received: 14.09.2020 • **Accepted:** 20.11.2020
DOI: 10.33706/jemcr.794850

her consciousness was cooperative and her blood lactate level was between 1.1 and 2.1 mmol / l in the follow-up of the patient with GCS 14. After two days of follow-up, the patient was discharged with full recovery without organ damage.

Case Report 2: A 19-year-old female patient, who had no known illness, applied to the emergency service of Kastamonu Training and Research Hospital with oral intake of 30 tablets of 100 mg theophylline for suicide. The patient was planned to apply activated charcoal in the emergency room, but could not be administered effectively due to severe nausea and vomiting, and intravenous fluid therapy was started. When the patient had severe symptoms and laboratory findings due to theophylline intoxication, he was admitted to the intensive care unit. Conscious, cooperative, oriented GKS: 15. Vital signs were: pulse: 148 /min, arterial blood pressure: 136/68 mmHg Spo2: 96, respiratory rate: 28/ min. Patient's white blood cell (WBC): 22000 / ul, glucose: 310 mg / dl, lactate: 6 mmol / l, potassium: It was 2.9 mmol / l, creatine: 0.9 mg / dl, b-hcg: 0.2 mIU / ml. The patient had severe nausea, vomiting and abdominal pain. Theophylline level was sent from the patient to the laboratory. In addition to reducing tachycardia with diltiazam, a calcium channel blocker, intravenous fluid therapy was initiated. In the follow-up of the patient, the complaints of nausea and vomiting increased, clinical symptoms did not improve despite medical treatment and theophylline level was 106 ug / ml, so the patient was taken to 4-hour ultrafiltration-free hemodialysis. After hemodialysis treatment, the patient's complaints of nausea and abdominal pain resolved, his tachycardia, blood lactate and glucose blood levels returned to normal. Hypokalemia was treated with potassium replacement. After hemodialysis, blood sample was taken from the patient again for theophylline blood level and sent to the laboratory and the theophylline blood level was 4 ug / ml. The patient was transferred to the internal disease service after two days of intensive care. The patient was discharged with full recovery, without organ damage, after two days of follow-up in the internal disease service.

Case Report 3: 22 years old male patient with mental retardation, he applied to the emergency service of Kastamonu Training and Research Hospital by his relatives with complaints of nausea, vomiting, sleepiness. There was substance use. The patient with GCS: 6 was intubated orotracheally and taken to the support of invasive mechanical ventilation. Arterial blood pressure: 96/45 mmhg, pulse: 124 / min, respiratory rate: 38. In blood tests performed in the emergency department, ph: 7.27, lac: 27 mmol / l, pco2: 23mmhg, sodium: 141 mEg / l, potassium: 5.55 mEg / l, AST: 11 u / l, ALT: 14u / l creatine: 2.12 mg / dl. A toxic blood panel was sent from the patient and the results were negative. Intracranial pathology was not detected in the imaging. The patient was admitted to the general intensive care unit with the diagnosis of coma and metabolic acidosis of unknown cause. We had a pre-diagnosis of intoxication in the patient. Since etiology could not be determined exactly due to the fact that ethylene glycol blood level could not be studied in the laboratory of our hospital, no antidote treatment was initiated and mechanical ventilator support and anti-poatassium treatment was applied.
When the patient became anuric during the first 12 hours of his admission to the intensive care unit and his creatinine levels increased to 5.08 mg / dl, he was placed on hemodialysis catheter with 2000 cc UF. In the following four days, when the patient was anuric and his creatinine levels were high, 3 cycles of hemodialysis were performed. The patient started to communicate on the 5th day of the ICU follow-up and on the 6th day his GCS was 14 and was extubated. After communicating with the patient, it was learned that he drank 1.5 liters of antifreeze (ethylene glycol). Since ethylene glycol blood level could not be studied in our institution, the level could not be monitored. The patient was transferred to the nephrology service on the 9th day of the intensive care follow-up. The patient, who had 1500-2500cc diuresis daily and whose kidney function tests returned to normal levels, was discharged with recovery without organ damage.

Discussion

Traditional hemodialysis is a frequently preferred treatment due to its widespread availability and proven efficacy for certain drugs and toxins. The primary determinants of the ability of a substance to be removed from blood by hemodialysis are the weight of the molecule, the volume of distribution (VD), the hydrophilicity or lipophilicity state of the substance, the protein or tissue binding capacity, and endogenous clearance. Substances with low molecular weight can be easily dialyzed. High efficiency high flow dialysers with diffusive modalities can remove medium molecular weight (<15,000 Da) materials. Convective modalities such as Hemofiltration and Hemodiofiltration allow removal of solutes approaching 25,000 Da[2].

Metformin is a biguanide antidiabetic agent commonly used in the treatment of type-2 diabetes mellitus. The plasma half-life of metformin is about six hours. Lactic acidosis may be seen in acute and chronic use of biguanides. There are few case reports associated with high doses of metformin, and it is generally fatal. Metformin is thought to reduce lactate metabolism by suppressing pyruvate carboxylase and increase lactate production by reducing pH in the liver. It also reduces glucose utilization and increases lactic acid release from hepatocytes. Lactic acidosis is defined as blood pH below 7.35 and serum lactic acid level above 2 mmol / l. It is divided into two subtypes, of which type A is the form associated with tissue hypoxia and mostly occurs in sepsis is the form associated with an external agent (metformin poisoning) as a result of a decrease in lactate clearance without hypoxia[3,4]. Metformin poisoning may present with nonspecific symptoms such as nausea, vomiting, abdominal pain, hypoglycemia, hypothermia, tachypnea, tachycardia / bradycardia, hypotension / hypertension, agitation, drowsiness, and coma. Therefore, anamnesis is important in differential diagnosis[5]. The classic

DOI: 10.33706/jemcr.794850
Journal of Emergency Medicine Case Reports 2020; 11(4): 108-110

triad of metformin toxicity can be listed as acute renal failure, high plasma metformin concentration, and severe lactic acidosis. Hemodialysis or continuous renal replacement therapies; It is preferred to eliminate overdose of drug, normalize serum potassium level and eliminate lactate, which is the cause of metabolic acidosis. Our patient had hyperlactataemia, hyperkalaemia and metabolic acidosis and was successfully treated with hemodialysis.

Theophylline is an effective bronchodilator used in the treatment of diseases like asthma, chronic obstructive pulmonary disease (COPD), neonatal apnea and bradycardia syndrome. A level of theophylline in the blood above 15 µg / mL carries the risk of intoxication. Therefore, when steady state is reached, 5-15 µg / mL blood level is considered as an effective and safe dose range[6]. When the toxic dose is reached, metabolic abnormalities such as nausea, vomiting, agitation, palpitations, hyperglycemia, hypokalaemia, acid-base imbalance and leukocytosis. traceable. In addition, life-threatening conditions such as convulsions and ventricular arrhythmias can be seen depending on the dose[7]. Our patient had nausea, vomiting, palpitations, hyperglycemia, hypokalaemia, acid-base disorder and leukocytosis, which are signs of severe theophylline intoxication. Our patient did not only have convulsions and agitation findings

Theophylline is well adsorbed by charcoal and therefore activated charcoal should be used even in poisoning with IV theophylline overdose. Hemoperfusion or high-throughput hemodialysis is indicated if vomiting prevents the use of activated charcoal, or it can be used as an adjunct therapy in patients with seizures, hypotension or arrhythmia. Hemodialysis / hemoperfusion should be considered in acute intoxications above 100 mg / L and chronic toxicity above 60 mg / L^2. In our patient, active charcoal treatment could not be successfully applied due to nausea and vomiting, and hemodialysis treatment was performed due to the toxic doses of blood theophylline and severe intoxication findings. After hemodialysis treatment, the patient's blood theophylline level came as 4 µg / ml and intoxication symptoms disappeared.

Intoxications should be considered in the foreground in cases of coma of unknown cause and acute renal failure. Ethylene glycol poisoning is one of the rare types of poisoning in our country with high mortality. In the first 12 hours after ingestion of ethylene glycol, it can cause central nervous system depression, confusion, ataxia, hallucination, ambiguous speech and coma. A cardiorespiratory phase (12 to 24 hours) then occurs with the onset of tachypnea, hypotension, and congestive heart failure. Finally, 24 hours after ingestion, oliguria and acute kidney injury often follow this process[8]. In our patient, we encountered a picture of acute renal failure following central nervous system depression, but we did not see a cardiorespiratory phase in our patient.

Intravenous ethanol, fomepizole and hemodialysis are the most important treatments in patients with ethylene glycol poisoning. The American Academy of Clinical Toxicology primarily recommends fomepizole as the treatment of choice in such situations. Fomepizole is a relatively new agent with a specific indication for ethylene glycol poisoning[9]. In a case report by Buchanan et al., A patient consuming high doses of ethylene glycol was treated with fomepizole alone without hemodialysis. They suggested that treatment with fomepizole may be feasible even with high-dose ethylene glycol consumption, provided that renal function is maintained[10]. Although fomepizole treatment could not be applied to our patient due to diagnostic difficulty, hemodialysis treatment was applied due to being anuric and impaired renal functions. In the ongoing process, three more hemodialysis sessions were applied, depending on the indication, and the patient was successfully treated.

Conclusion

Intoxication cases are medical emergencies, and severe cases may require urgent extracorporeal treatments to prevent or reverse major toxicity. Hemodialysis is the leading extracorporeal treatment. It can also be applied as short-term high-flow hemodialysis in suitable patients.

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Case Report

Journal of Emergency Medicine Case Reports

Ludwig's Angina: Case Series with Description of the Ultrasonographic Features of the Emergency Conditions

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Abstract

Introduction: Ludwig's Angina (LA) is a rare but rapidly evolving form of toxic cellulitis with heavy clinical symptoms and the potential of a rapid spread to adjacent cervical triangles.possible expansion.

Case Report: This case series describes the ultrasonographic (USG) features, imaging characteristics and aims to highlight the importance of USG imaging of spreading infections caused by dentomaxillofacial pathologies. Although the diagnosis of Ludwig's angina is more clinical, ultrasound is a necessary complementary examination to delineate the extent of the inflammation and to highlight its most common complications, such as obstruction of the airway, abscess formation, osteomyelitis and vascular clots which are indications for more immediate treatment.

Conclusion: The use of ultrasound for evaluating the extent of the condition is not often reported (and it is reported as isolated case reports) in the literature which allows for a high end and detailed estimation of the infection and its surrounding tissues.

Keywords: Ludwig's Angina, Ultrasonography, infection

Introduction

Ludwig angina (LA) is a rapidly evolving cellulitis of the upper neck area. It is diffused inflammation of bacterial origin and its expansion is by tissue continuity.In most cases it extends to include the sublingual submandibular and submental spaces[1-2]. Ludwig angina spreads into superficial and surrounding facial tissues. Generally, the original source of the infection is dental, mostly an untreated or an undiagnosed dental abscess, It can also arise from otitis media, tongue piercing, sialadenitis, or sialolithiasis of the submandibular glands[6-8]. In advanced cases airway obstruction is the major problem that preaents, while other complications of the condition include pneumothorax[5], thoracic empyema[6], septicemia etc[3,4,7-8] Overall, its rapid expansion and mortality rate about 8% makes it a potentially mortal infection[9]. Mortality caused by LA was greater than 50% in the preantibiotic era.[10]. The use of ultrasound for evaluating the extent of the condition is not often reported (and it is reported as isolated case reports) in the literature though modern machines allow for a high end and detailed estimation of the infection extend and its surrounding tissues possible expansion. The aim of this paper is to present the key ultrasonographic features of Ludwig angina through a case series presentation where ultrasonography was selected as the initial examination of imaging.

In many cases ultrasonography is the initial radiographic exam of choice. This is because it is readily available in hospital emergencies, it depicts the soft tissues with good enough detail to set in most cases the initial diagnosis and it has no radiation or the patient.

Case Series

During the past year in the radiology dept. of the AHEPA general Hospital of Thessaloniki and Dentomaxillofacial Radiology Department of Ankara University there was a total of four cases, three of them in the emergencies and one case were in the dental outpatient clinic that were studied with ultrasound and the diagnosis of Ludwig's Angina was set by the clinical and ultrasonographic findings. The informed consent were taken from all patients and legal guardian. The consent was included that the patient's clinical photo, radiographs can be used for scientific purposes and publishing.

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Received: 25.09.2020 • **Accepted:** 20.11.2020

DOI: 10.33706/jemcr.800027

DOI: 10.33706/jemcr.800027
Journal of Emergency Medicine Case Reports 2020; 11(4): 111-115

Case 1

53-year-old male recruited to emergency department with painful pulsating swelling of the submandibular area. The patient presented with a painful, pulsating swelling of the submandibular area, accompanying edema, and rubor of the skin. The clinical estimation was abscess of the submandibular area, the ultrasonographic image demonstrated thick walls of the lesions since it was a formed abscess and high vascularity diaphragms within the lesion. The pathological lesion was accompanied by multiple lymph nodes with a rounded image, while the entire neck area had high vascularity and superficial cellulitis, the abscess was punctured (Figure 1).

Case 2

36 years old female who had difficulty in swallowing and intense dental pain admitted to emergency. Collection of inflammatory material forming an abscess superficial to the mandibular ramuswas observedwith the use of ultrasound. There was accompanying edema under the skin, and increased vascularity with the use of color Doppler. The inflammation was expanding to adjacent tissues with intense thickening of the skin, and the presence of pathological lymph nodes with swelling and pain upon palpation in the entire neck area (Figure 2).

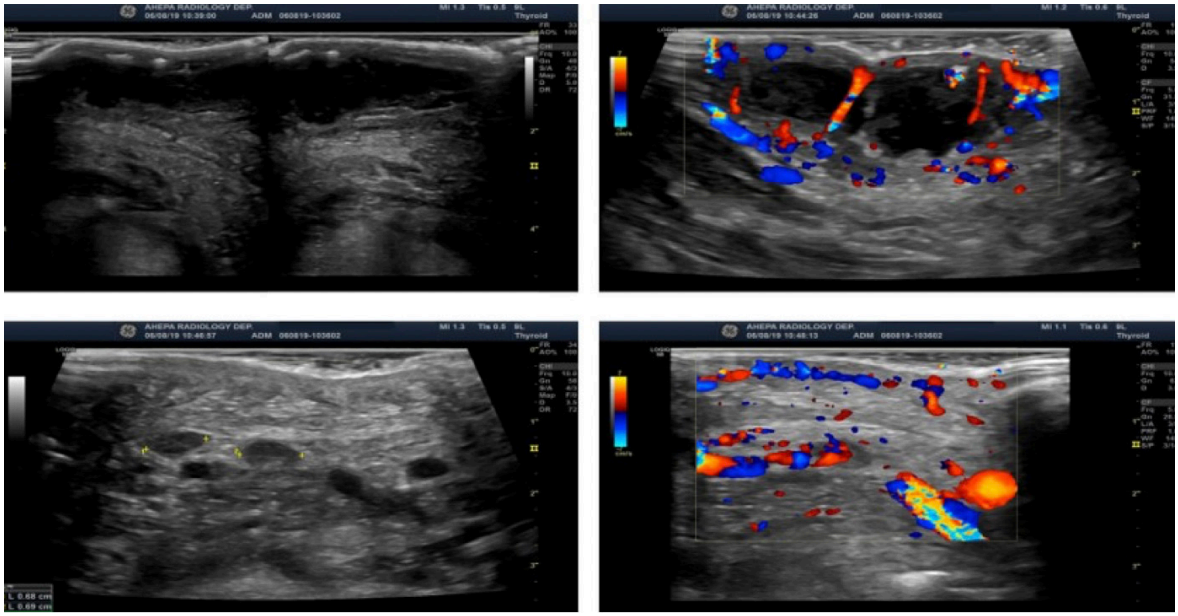

Figure 1: USG images show an abscess of the submandibular area with a thickened wall and increased vascularity with the use of color Doppler. Multiple, slightly rounded superficial lymph nodes with a reactive image, intense skin cellulitis and increased hemorrhage.

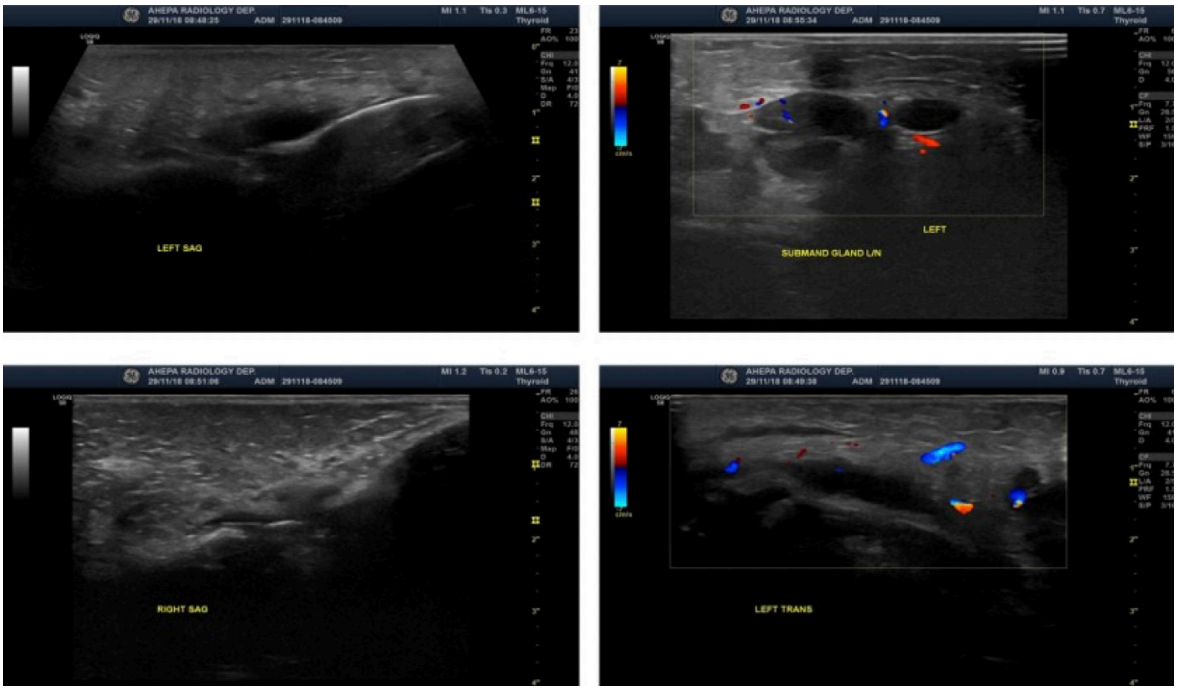

Figure 2: USG images revealed abscess formation superficial to the ramus of the lower jaw, subcutaneous edema and increased vascularity in Color Doppler. Expansion of the lesion to the adjacent tissues apparent thickening of the skin, and presence of abnormally enlarged lymph nodes in the underlying region.

Case 3

42 years old female admitted to oral diagnosis clinic of dental faculty. She had an abscess due to periapical infection of mandibular molar that was initially diagnosed with panoramic radiography. The patient later referred to USG imaging for further examination. USG showed a pus filled localized swelling in the soft tissue with deep tissue extension. The lesion had a hypoechoic echo pattern without pronounced borders and without increased vascularity in color doppler examination (Figure 3).

Case 4

4-month-oldinfant which arrived with difficulty in breathing, inability to feed and very high fever. The unusual in this case is the origin of the inflammation since LA is considered to be mostly of odontogenic origin. In this case of infantile inflammation, the condition was attributed to tonsillitis and was not of odontogenic origin. Though the initial image of the infant was of acute tonsillitis, the rapid expansion of the inflammation and its extent to adjacent tissues, the rapid deterioration of the condition of the infant, the high number of inflamed lymph nodes led to the diagnosis. Both submandibular spaces were affected. What was very characteristic of the inflammation expansion was the presence of continuous lymph nodes with abscesses and high vascularity forming a block of nodes, the content of the nodes was a thick and nonclear cystic fluid. There was edema under the skin and extended inflammation. The infant was intubated (Figure 4).

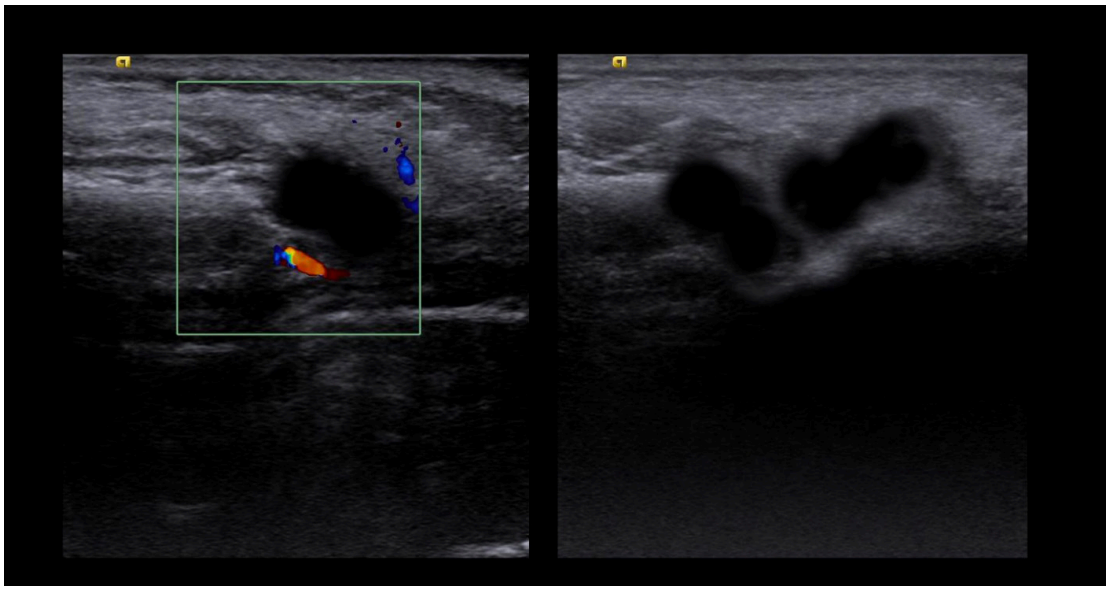

Figure 3: USG showed a pus filled localized swelling in the soft tissue with deep tissue extension. The lesion had a hypoechoic echo pattern without pronounced borders and without increased vascularity in Color Doppler examination.

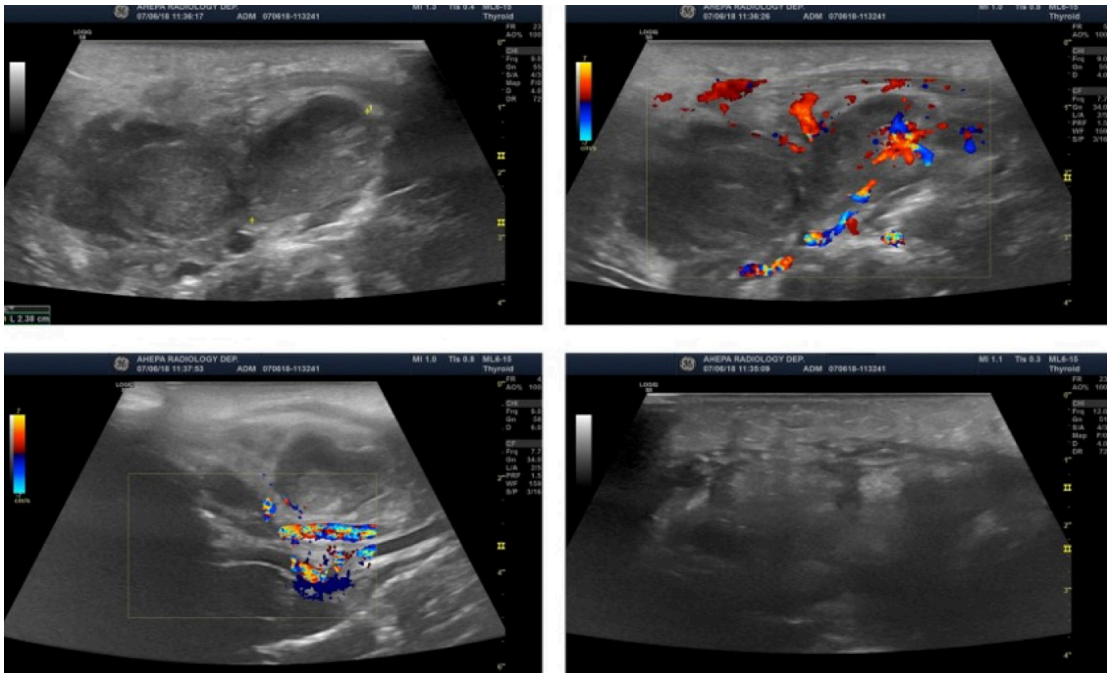

Figure 4: USG images of an infant which revealed expansion of the inflammation to the submandibular spaces with high vascularity and thick viscous fluid content.

DOI: 10.33706/jemcr.800027
Journal of Emergency Medicine Case Reports 2020; 11(4): 111-115

Discussion

Ludwig's Angina is a condition well established in the literature and first described by Wilhelm Frederick von Ludwig. Clinically LA is characterized by diffused abscesses and coexisting swelling of the upper cervical triangles[3-5]. When the submandibular and submental spaces are involved to the expansion of the inflammation, intense rigid and painful, hard upon palpation swellings are observed in the involved spaces in most of cases. Though it is not common to be able to palpate organized swelling and abscesses, there are small in size focal inflammatory areas that spread in all tissues involved and their enlargement and growth makes them palpable clinically. When there is sublingual space involvement, there is swelling of the floor of the mouth of the posterior section of the tongue. This swelling, when accompanied by ovula swelling may lead to airway obstruction and cause a threat for the patients' life, making the need of tracheotomy imperative[8-10]. Patients often complain of difficulty in swallowing and breathing, and in few cases even talking. The importance of proper diagnosis is crucial for the patient and the evolution of the inflammatory process. The infection progresses rapidly and can disseminate to the parapharyngeal space, retropharyngeal space, and also mediastinum[8]. Early diagnosis and treatment of the LA are important to avoid the complications that may occur due to LA. Airway obstruction, necrotizing fasciitis, osteomyelitis, mediastinitis, empyema, carotid arterial rupture or sheath abscess, pericardial effusion, thrombophlebitis of the internal jugular vein,aspiration pneumonia subphrenicabscess, and pleural effusionare the complications of LA. The radiographic findings of the condition are well described however the diagnosis is rarely set by ultrasonography. This is partly due to the fact that LA is not a frequently presenting condition particularly with the ofteninstructed early use of antibiotics to fight it and prevent its expansion[10]. The panoramic radiograph is usually an important radiographic feature which depicts the most frequent to the condition dental origin of the inflammation. Anteroposterior and lateral scull projections are often taken to observe initial inflammations of the adjacent spaces, while in advanced and potentially life-threatening cases, CT and/or MRI are selected. Computed tomography of the neck orfocused ultrasound is essential for surveying the details ofinvolved structures or newly formed well definedinflammatory areas caused by the expansion of the underlying pathology.As compared with computed tomography, ultrasound is aportable and without radiation tool with the ability of real-timeevaluation for patients. Ultrasound however provides usually adequate information to set the diagnosis and establish the etiology without giving any radiation to the patient. With the newest machines of linear high frequency transducers, the diagnosis is very easy to set and in high detail particularly when high vascularization is involved. In the cases studied the patients had ultrasonographic features that set the diagnosis of LA. Although the diagnosis of Ludwig's angina is more clinical, ultrasound is a necessary complementary examination to delineate the extent of the inflammation and to highlight its most common complications, such as obstruction of the airway, abscess formation, osteomyelitis. and vascular clots which are indications for more immediate treatment. At any case the following ultrasonographic features indicative of inflammation must present and help establish the diagnosis: Soft tissue edema,-Cellulitis, Skin thickening and infiltration, Swollen lymph nodes, Possible abscesses, High vascularity.

LA is a rare but rapidly evolving form of toxic cellulitis with heavy clinical symptoms and the potential of a rapid spreadto adjacent cervical triangles. Before presenting to the hospital the most common symptoms the patients complain of include tooth pain (79%), neck swelling (71%), dysphagia (52%), and neck pain (33%)[11-12].

Current and modern therapeutic approaches to LA include in most cases early diagnosis, immediate antibiotic treatment early surgical treatment when needed, and removal of the cause,usually of dental origin, that lead to the inflammation. A 50% mortality is mentioned in the literature in these patients due to upper airway edema. Recent therapeutic advancesinclude antibiotics and aggressive supportive therapy and their early use have lowered this mortality rate to 8%. The condition is due to odontogenic infections in most cases though additional inflammations of the neck may set its presence. Most of thecases involve the roots of the lower molars which are located just below the mylohyoid ridge and therefore, when they have periapical infection and/or abscess, the inflammation can spread directly to the soft tissue spaces of the submandibular, sublingual, and submental regions[11-12]. One of the major concerns in patients with LA is the maintenance of a clear airway. In the literature though the condition is not so frequent nowadays, there is mention that the patient may die from airway obstruction and not from septicemia as one would expect. The treatment may include a number of operations most frequent which is the surgical excision and drainage of possible abscesses, before they form organized inflammations in the neck area, the treatment with antibiotics and removal of the cause. Ultrasonography can be utilized to predict airway difficulty during the use of general anesthesia. There is a rapidly growing body of evidence showing its benefits. Lakhal et al,[13] compared the diameter of subglottic upper airway between MRI and ultrasonography on healthy volunteers. They found strong correlation on transverse diameter which is the smallest diameter of the cricoid lumen.In healthy young adults ultrasonography is a secure device to state it. Shibasaki et al found that, subglottic upper airway diameter measured by ultrasonography is a good predictor of endotracheal tube sizes for pediatric patients[14]. Particularly with infants and children the diagnosis of the condition is not easy since it is very rare, and it usually is mistaken

for other simpler conditions. However, care should be taken with infants and children on the pattern of inflammation expansion, the clinical condition and possible rapid occurring complications as well as the radiographic difficulties that may present. Ultrasonography is the method of choice in the follow up of children with the condition since it is radiation free and it even helps with the pediatric tracheotomy. More advanced radiographic techniques may be required prior to surgery and puncture of the abscesses, though nowadays most cases are treated conservatively with the use of antibiotics and other anti-inflammatory agents, making the use of ultrasonography adequate for the treatment.

Conclusion

Ludwig angina is a rare, life-threading condition. Early diagnosis and treatment are important to reduce the rate of mortality. Ultrasonography is an effective way to detect the condition and also useful for critical care procedures.

Ethical approval and informed consent

An informed consent was obtained from all patients whose pictures are shown in this case series. A copy of these informed consents can be delivered on demand. Since the data was retrospectively evaluated, no ethical approval was necessary.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Side Effect Secondary to Warfarin Use: Retropharyngeal Hematoma

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Abstract

Introduction: In this case report, we aimed to explain the importance of urgent management in the diagnosis and treatment of a patient who developed retropharyngeal hematoma as a result of the toxicity of warfarin he used due to atrial fibrillation.

Case Report: A 76-year-old male patient came to the emergency room with complaints of sore throat, difficulty swallowing, and shortness of breath. At the time of admission of the patient using warfarin due to atrial fibrillation, oropharynx examination revealed normal uvula, hematoma near the right lip mucosa, and stridor was detected in the lung auscultation of the patient whose trachea was deviated to the left. In the laboratory tests at the time of the patient's admission, INR was: 13.4. The patient was given 10 mg phytomenadione(K1) iv infusion and 80 mg prednisolone iv. 4 units(1000 ml) of FFP(fresh frozen plasma) was requested. The patient who developed respiratory distress was intubated and transferred to intensive care unit.

Conclusion: Warfarin toxicity may rarely cause bleeding in the upper respiratory tract, and if it creates pressure on the respiratory tract, it may pose a serious life-threatening risk.

Keywords: Warfarin, hematoma, retrofarengeal hematoma

Introduction

Bleeding into the upper respiratory tract secondary to anticoagulant use is a rare but life-threatening condition. It is vital that the pathology be recognized and treatment started as soon as possible.

In this presentation, we aimed to explain the toxicity of warfarin, which he used for atrial fibrillation, and the importance of emergency management in retropharyngeal hematoma examination, diagnosis and treatment.

Case Report

A 76-year-old male patient came to the emergency room with complaints of sore throat, difficulty swallowing, and shortness of breath for 4 days. At the time of admission of the patient with known chronic renal failure who used warfarin due to atrial fibrillation, the pulse rate was 100/min, blood pressure:124/79 mmHg, Spo2 was 96%. Physical examination revealed a glaskow coma scale 15, palpable swelling and hematoma on the right neck (Figure.1). Oropharynx examination revealed normal uvula, hematoma near the right lip mucosa, and stridor was detected in the lung auscultation of the patient whose trachea was deviated to the left (Figure 2).

In the laboratory examinations at the time of the patient's admission, creatine: 2.16 mm/dl, BUN (blood urea nitrogen):117 mg/dL, hemoglobin:9.9 g/dL, platelet: 295,000/uL, INR: 13.4. There was no pathology in other laboratory findings

The patient was given10 mg phytomenadione (K1) iv infusion and 80 mg prednisolone iv. 4 units (1000 ml) of FFP (compensated plasma) was requested, intubation decision was made for the patient whose respiratory distress worsened. Cardiopulmonary resuscitation was performed according to AHA criteria for 5 minutes in the patient who developed cardiac arrest secondary to respiratory arrest before being intubated. The patient who met difficult intubation criteria due to retropharyngeal hematoma was intubated with a bougie.

1000 mg TDP was applied. The patient was transferred to intensive care unit. Inintensivecare, tranexamicacid 250 mg iv and 10 mg phytomenadione iv infusion were administered for 3 days. The patient, whose neck hematoma completely healed, was extubated 7 days later, and was discharged 15 days later in good health.

Discussion

Oral anticoagulants are frequently used in patients with a history of atrial fibrillation, prosthetic valve disease, myo-

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Received: 25.09.2020 • **Accepted:** 04.11.2020
DOI: 10.33706/jemcr.800205

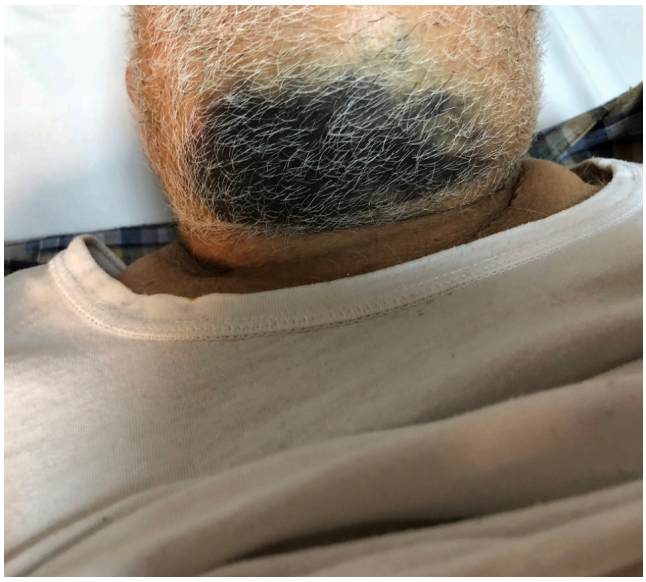

Figure 1: Patient with hematoma on the right neck

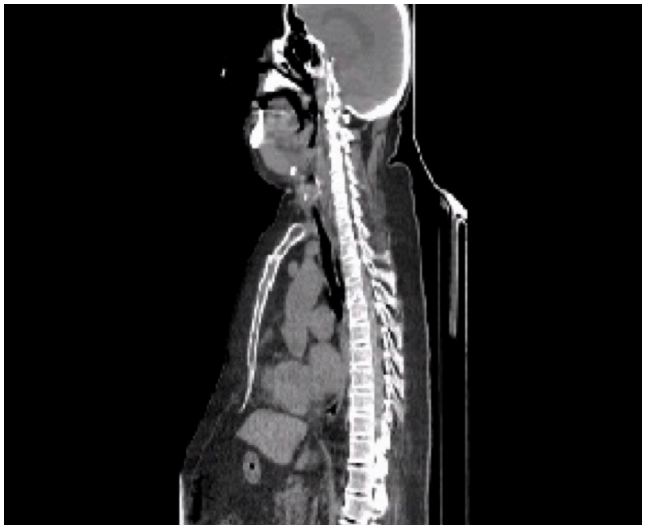

Figure 2: CT of the patient

cardial infarction, arterial or venous thromboembolism. The efficacy of warfarin is measured by INR, and the use of warfarin is difficult due to genetic differences in dose response, narrow therapeutic range of warfarin, and drug-diet interaction. The risk of haemorrhage secondary to anticoagulant use is most often in the gastrointestinal system, genitourinary system, nose, skin, and retroperitoneum[1].

Warfarin inhibits vitamin K reductase and cycloepoxitreductase, which are involved in the carboxylation of factor 2,7,9,10 and other vitamin K-related proteins, preventing the activation of coagulation factors and inhibiting coagulation[2].

In patients who need to use warfarin, discontinuation of the drug may cause clinical symptoms such as arterial occlusion[3]. In a study comparing new generation anticoagulants, two groups using Dabigatran and Rivoraxaban were compared, and these groups were not superior to each other in terms of complication rate[4].

Hematomas causing obstruction in the upper respiratory tract are rare, and patients may present with dyspnea, styrodor, dysphagia, odynophagia, neck swelling, and pain in the throat[1,2,5]. Bleeding may be due to trauma, vigorous tooth brushing, local infection, severe coughing, sneezing, yelling, or it may be spontaneous[1]. 'Capps triad', which includes evidence of ecchymosis on the anterior face of the neck and chest, tracheal displacement, and evidence of tracheal and / or esophageal compression[5]. The etiology was also unremarkable, he had dyspnea, stridor, dysphagia, odynophagia and sore throat, and he was in line with the Capps tradition.

The mainstay of retropharyngeal hematoma treatment should be to control coagulability and protect the respiratory tract. Intubation, tracheostomy and cricotrioromia may be considered depending on the clinical situation. Corticosteroids may be given to resolve edema[1]. Tomography or magnetic resonance may be ordered to better visualize the hematoma. The conservative method may take 3 weeks for the symptom and underlying hematoma to resolve[5]. Our patient was urgently decided to intubate, and he could only be intubated with a bougie. Meanwhile, cardiopulmonary resuscitation was applied to the patient, who developed cardiac arrest, for 5 minutes, and his return to sinus rhythm was ensured by preventing him to remain hypoxic.

Patients with INR> 9 and no significant bleeding can be given vitamin K1 2.5-5 mg, significantly decreasing INR within 24-48 hours 1. K1 administration is not recommended unless there is INR> 10 or bleeding occurs. If toxic levels of intake are considered for suicide, activated charcoal can be given within 1 hour. If there is a life-threatening condition due to warfarin toxicity coagulation, the patient; Factor 4 or 3 prothrombin complex concentrate or fresh frozen plasma and intravenous Vitamin K1 10 mg should be administered, K1 can be administered every 12 hours as needed. Tranexamic acid can be considered[6].

We applied vitamin K1 and fresh frozen plasma to our patient with INR: 13.4. In previous studies, retropharyngeal hematoma cases were reported due to trauma, surgical complications, clopidogrel use, or warfarin intoxication as in our study[6-8]. Although higher levels are reported in the literature, we think that our high INR level will contribute to the literature.

Conclusion

Warfarin toxicity may rarely cause bleeding in the upper respiratory tract, and if it creates pressure on the respiratory tract, it may pose a serious life-threatening risk. Detailed anamnesis and physical examination of patients with classical upper respiratory tract infection history such as sore throat and difficulty swallowing can be life-saving.

DOI: 10.33706/jemcr.800205
Journal of Emergency Medicine Case Reports 2020; 11(4): 116-118

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Case Report

Journal of Emergency Medicine Case Reports

When your breath dyes away Need for Surgical Airway in a Case of Hair dye Poisoning

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Abstract

Introduction: The use of hair dye for deliberate self-harm is seen in many parts of the world. Paraphenylenediamine (PPD) is the main constituent of hair dye formulations and is found to be highly toxic. The other constituents are resorcinol, propylene glycol, sodium ethylene diamine tetra acetic acid, preservatives and perfume. Clinical features of PPD poisoning include severe cervicofacial odema, chocolate colored urine, oliguria and shock. Management is mainly supportive and there are no specific antidotes.

Case Report: A young lady was brought to ED with complains of sudden onset swelling of chin, neck and tongue. She was sitting upright with hoarseness of voice. She had edema over her chin and lower half of face extending to the neck. The tongue was hard and edematous. As her symptoms worsened she asked for a piece of paper to write and wrote "VASOMOL", which is a brand name for the hair dye containing paraphenelyenediamine.

Conclusion: The difficult airway management is an essential skill needed for the emergency physician. Hair dye poisoning has been known to cause severe angioneurotic odema. Ours is a case of PPD poisoning which presented with life threatening airway odema. We followed the ABCD assessment and on determining the need for airway control, we planned a surgical airway immediately.

Keywords: Paraphenylenediamine, Angioneurotic odema, Rhabdomyolysis, Acute tubular necrosis

Introduction

The use of hair dye for deliberate self-harm has been seen in parts of Africa, the Middle- east and the Indian subcontinent[1]. Paraphenylenediamine (PPD) is the main constituent of hair dye formulations and is found to be highly toxic [2]. In India, Super Vasmol 33™ is a brand of hair dye containing PPD. The other constituents are resorcinol, propylene glycol, sodium ethylene diamine tetra acetic acid, preservatives and perfume. PPD is a synthetic aromatic amine and its main oxidation product is Bondrowskis base which is allergenic, mutagenic and highly toxic[3]. Clinical features of PPD poisoning include severe cervicofacial odema, chocolate colored urine, oliguria and shock[4]. The triad of angioneurotic odema, rhabdomyolysis and acute tubular necrosis is seen in cases of PPD poisoning[5]. Management is supportive and there are no specific antidotes. The angioneurotic odema of the airways in PPD poisoning is life threatening and if not managed promptly can be fatal[6].

Case Report

A 21 year old lady was brought to our emergency department with complains of sudden onset swelling of chin, neck and tongue which was noticed by her relatives 2 hours back. She was sitting upright on a stretcher with hoarseness of voice. Her chest was clear on auscultation. She was normotensive with a room air saturation of 89% and respiratory rate of 25. She had edema over her chin and lower half of face extending to the neck. The tongue was hard and edematous (Figure 1). There was no history of fever, throat pain, dental infections or allergies. Once shifted to the priority area, she was under continuous monitoring. We noticed her respiratory rate increasing and she also started drooling and had nausea. She was given intramuscular adrenaline, intravenous antihistamines, corticosteroids and fluids but her symptoms did not show any resolution. As her symptoms worsened she asked for a piece of paper to write and wrote "VASOMOL", which is a brand name for the hair dye containing para-

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Received: 01.10.2020 • **Accepted:** 12.12.2020
DOI: 10.33706/jemcr.804013

DOI: 10.33706/jemcr.804013
Journal of Emergency Medicine Case Reports 2020; 11(4): 119-121

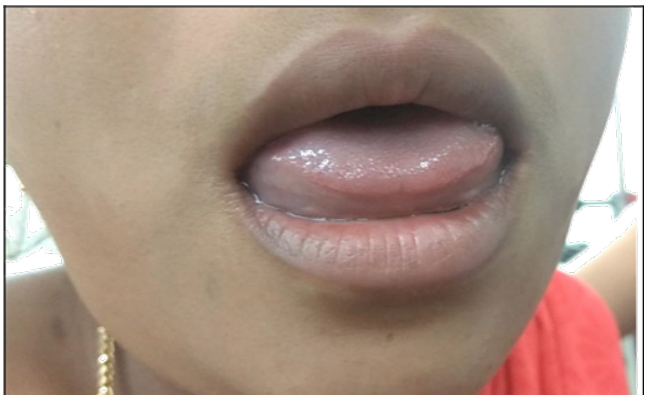

Figure 1: Clinical picture showing swollen tongue.

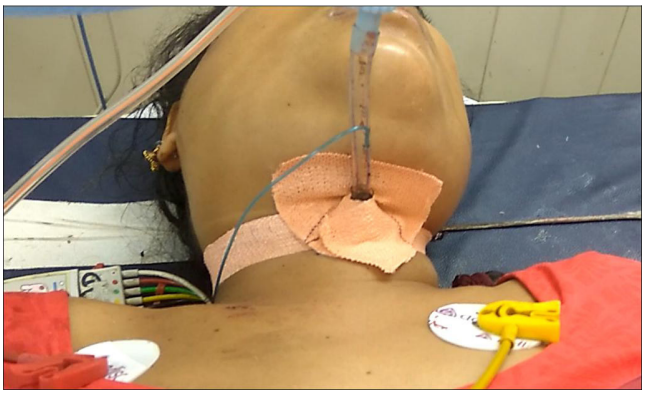

Figure 2: Emergency tracheostomy after failed intubation.

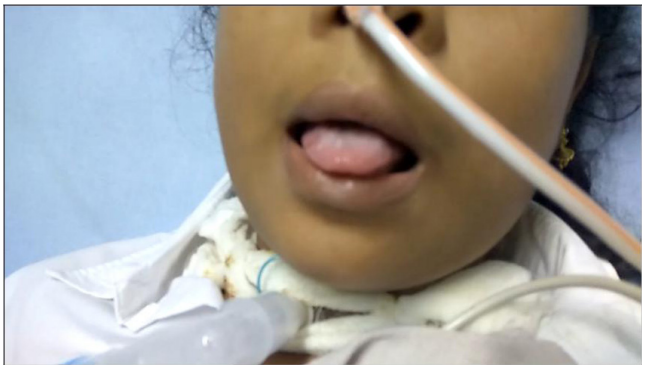

Figure 3: Clinical picture showing subsided neck and tongue edema.

phenelyenediamine. We immediately assessed her airway as she had begun to develop respiratory distress. She had narrow mouth opening due to the edema and the Mallampati grade was 4. Anesthetic backup was called in view of difficult airway prediction and decision to intubate was taken in view of impending life threatening airway edema. Prior to intubation, supraglottic devices and the equipment for surgical cricothyrotomy were arranged at the patient's bedside. Delayed sequence intubation was attempted using etomidate after 3 minutes of bag mask ventilation using two hand technique. Three attempts of laryngoscopy were made but could not visualize anything beyond the base of tongue. She was ventilated back to above 95% saturations after each attempt. She was also having increasing secretions. In view of pharyngeal odema, use of supraglottic device was deferred and we proceeded to attempt surgical cricothyrotomy (Figure 2).

The patient's neck was approached from the right hand side and vertical incision made over the cricothyroid membrane after raising a wheal of local anesthetic while stabilizing the trachea with the other hand. The skin was dissected and horizontal stab incision given after locating the cricothyroid membrane using a scalpel. The incision was then widened with the artery forceps and a bougie was passed downward which did not face any resistance. A 6 size endotracheal tube was then railroaded over the bougie and as soon as it entered the skin the cuff was inflated and bougie removed. Bilateral air entry was confirmed and patient's saturations were around 98%. She was then connected to a ventilator and shifted inside the ICU. She was given aggressive fluid hydration with isotonic crystalloids along with other symptomatic measures. The surgical team was notified and she was then taken up for a definitive tracheostomy. Her neck and tongue edema gradually subsided (Figure 3) and her renal and liver functions were within the normal limits. She was weaned off the ventilator and discharged home after 7 days of hospitalization.

Discussion

Hair dye poisoning has been known to cause severe angioneurotic odema. Ours was a case of PPD poisoning which presented with life threatening airway odema. We followed the ABCD assessment and on determining the need for airway control and difficult airway prediction we planned accordingly for the contingencies. Anesthetic backup was called and after 3 attempts at laryngoscopy with changes in operator and positioning, immediate realization of the 'cannot intubate' scenario was made. Due to unsuitability of the use of the laryngeal mask airway and unavailability of the higher airway devices like the video laryngoscope and fiberoptic devices, we went ahead with the surgical cricothyrotomy and were able to secure the airway. Our patient was also given fluid resuscitation and also bicarbonate therapy to prevent renal injury. PPD is one of the rare toxins where the toxin itself is responsible for airway distortion in the patient, hence requiring expert airway management with often front of neck access (FONA). The NEAR III study put the incidence of surgical airways in airway encounters at 0.45% [7]. In our case we used a scalpel –bougie-tube technique. Bleeding occurred into the field while making the incision and distorted the view, but we used palpation to find the membrane and once the endotracheal tube was placed used pressure to control the bleeding which was successful. The performance of surgical airways requires situational awareness with effective communication and resource management[8]. The advent of newer airway control devices like the fiber optic bronchoscope and the video laryngoscope has significantly decreased the incidence of surgical airways in the emergency department [9]. But in scenarios where such

equipment are not available the emergency medicine physician should be well aware of the alternatives.

Conclusion

The difficult airway management is an essential skill needed for the emergency physician. This case highlights two aspects, the need for airway management training in emergency medicine department and also the airway compromise in cases of PPD poisoning which if neglected, can be life threatening.

Conflict of Interest
None

Acknowledgements
None

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Case Report

Journal of Emergency Medicine Case Reports

Focal Myocarditis Mimicking Subendocardial Ischaemia: A Case Report

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Abstract

Introduction: Myocarditis is an infrequent, possibly life-threatening, and inflammatory myocardial disease with various number of clinical complaints and symptoms, often caused by infectious agents.

Case Presentation: A 24-year-old male came to the emergency department (ED) with the complaints of angina-like retrosternal chest pain and tightness lasting for three days. The ECG suggesting inferior submural ischaemia and echocardiographic assessment was normal. Laboratory tests showed troponin T levels increased. Coronary angiography was normal. CMRI showed patchy contrast uptake. It looked more like viral myocarditis.

Conclusion: Acute myocarditis diagnosis is most difficulty and predictively in connection with the variety of clinical presentations. The differential diagnosis between myocarditis and AMI can be troublesome in ED.

Keywords: Angina pectoris, endomyocardial biopsy, ischaemia, myocarditis

Introduction

Myocarditis is an infrequent, possibly life-threatening, and inflammatory myocardial disease with various number of clinical complaints and symptoms, often caused by infectious agents[1]. Myocarditis occurs with acute decompensation of heart failure and come with disproportionate dyspnea on exertion, chest pain, arrhythmias and sudden death. Acute myocarditis diagnosis is most difficulty and predictively in connection with the variety of clinical presentations. To confirm the diagnosis, the clinical presentation, physical examination, laboratory testing, electrocardiographic (ECG) changes and a normal coronary angiography should be considered[2]. Endomyocardial biopsy (EMB) is the best diagnostic option, but is not used as commonly. This report presents a case of young male suffering from angina-like chest pain who had ECG changes and cardiac enzyme levels suggesting acute myocardial ischemia of which the diagnosis of acute myocarditis was confirmed using cardiac magnetic resonance imaging (CMRI).

Case

A 24-year-old male came to the emergency department (ED) with the complaints of angina-like retrosternal chest pain and tightness lasting for three days. He did not have any chronic disease, cardiovascular risk factors and medication. One week before, he was diagnosed with an acute gastroenteritis. At presentation, his pulse rate was 110 bpm, blood pressure was 110/80 mmHg and temperature was 36.9 ˚C. The initial physical examination showed heart sounds were normal. The first ECG in the ED demonstarted sinus tachycardia with negative T-waves in the inferior leads (Figure 1), suggesting inferior submural ischaemia. The echocardiographic (ECHO) assessment revealed a left ventricular ejection fraction around 56% and no hypokinesis. Laboratory tests showed white blood cells counts of 9,9×109/mL, C-reactive protein (CRP) of 112 mg/dL (reference range[rr]:0-6), creatine kinase (CK) of 696 IU/L (rr:0-190), muscle brain fraction of CK (CK-MB) 54 IU/L (rr:3-25), troponin T of 574,3 ng/L (rr:=0-14). Based on these findings, initial diagnosis of acute non st elevation myocardial infarction was considered. Coronary angiography was performed immediately. It looked more like viral myocarditis as epicardial coronary arteries were normal. The diagnosis of myocarditis was confirmed by CMRI, which showed patchy contrast uptake at inferolateral wall and left ventricle apex (Figure 2). β-blocker therapy was advised as complementary medication. Four weeks later, he had no complaint, and so β-blockers were discontinued.

Discussion

Myocarditis is a confusing diagnosis due to the variety of clinical presentations ranging from fatigue, mild chest pain,

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Received: 05.10.2020 • **Accepted:** 15.11.2020

DOI: 10.33706/jemcr.805411

sings of congestive heart failure, ECG changes to life-threatening cardiogenic shock, ventricular arrhythmia and sudden death. Common symptoms included dyspnea (71.7%), unspecific chest pain (31.9%) and arrhythmic events (17.9%). Patients with severe myocarditis characteristically experience serious heart failure or cardiogenic shock symptoms[1]. Chest pain in acute myocarditis may imitate typical angina and may be linked to ECG abnormalities, including ST segment changes[2,3]. The European Society of Cardiology Working Group on Myocardial and Pericardial Diseases recommend all clinically suspected myocarditis cases should be considered for selective coronary angiography. Damage to cardiac myocytes lead to fluctuations in electrical activity which brings about ECG ST - T wave changes, ST segment elevation, atrial and ventricular arrhythmias, atrioventricular and intraventricular conduction defects and prematüre repolarization. However, ECG is sensitive for myocarditis by 47% only[4]. Cardiac Tn increases in the early stage of myocarditis and shows a gradual drop as the patient recovers. Sensitive tests specific for myocyte injury, such as cTnI, is likely to assist in the diagnosis of myocarditis[5]. Erythrocyte sedimentation rate and CRP levels are often raised in myocarditis, but are not used to diagnose myocarditis[1]. The European Study of Epidemiology and Treatment of Cardiac Inflammatory Disease (ESETCID) reported that 78.3% of the patients with an ejection fraction higher than 45% and in patients with normal or only mild reduction of ejection fraction, chest pain (42.1%) was the main symptom[6]. Speckle-tracking ECHO characterized by the precise evaluation of local contractility could be supportively used to diagnose acute myocarditis and inflammatory cardiomyopathy[7,8]. At the same time ECHO helps to rule out non-inflammatory cardiac diseases such as heart valve diseases. Today CMRI is used as one of the most powerful devices for detecting and diagnosing myocarditis. CMRI can detect myocardial edema and myocyte damage noninvasively. Myocarditis is characterized by a characteristic contrast enhancement pattern that originates primarily from the epicardium and protects the subendocardial layer. Conversely, myocardial infarction characteristically shows a subendocardial improvement on MRI. The European Society of Cardiology Working Group on Myocardial and Pericardial Diseases recommend CMRI for diagnosis. On the other hand, the single process for a definite diagnosis of myocarditis is endomyocardial biopsy, with a sensitivity from 43% to 64%, an overall complication rate of 6%, and a 0.4% incidence of death due to perforation. In 2013, The European Society of Cardiology Working Group on Myocardial and Pericardial Diseases recommended heart biopsy as a routine test for all cases of suspected myocarditis. Conversely, the routine application of EMB was not recommended by the 2013 American College of Cardiology Foundation/American Heart Association, which shows a lack of consensus on EMB for diagnosing myocarditis9. Almost 50% of the patients with acute myocarditis tend to naturally get better within a month's period, approximately 25% will develop persistent impaired cardiac function and up to 25-30% may either progress to dilated cardiomyopathy that requires heart transplantation. In patients with chest

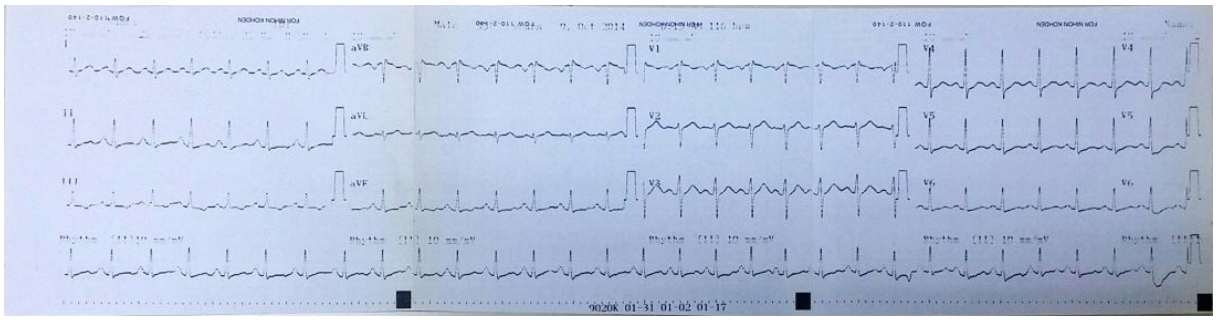

Figure 1: ECG shows negative T-waves in the inferior leads

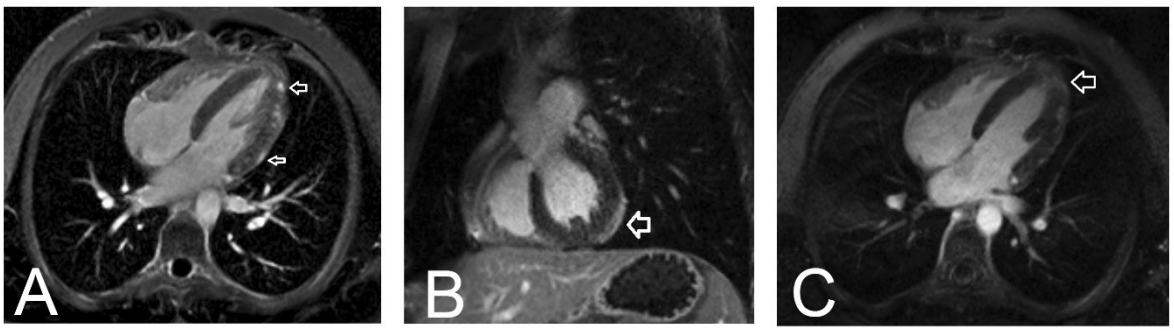

Figure 2: Contrast-enhanced cardiac magnetic resonance identified areas of myocardial inflammation (arrows) and minimal pericardial effusion due to focal myocarditis in the inferolateral wall. A. CMRI shows minimal pericardial effusion, B. CMRI shows minimal pericardial effusion, C. CMRI shows patchy contrast uptake at inferolateral wall and left ventricle apex.

DOI: 10.33706/jemcr.805411
Journal of Emergency Medicine Case Reports 2020; 11(4): 122-124

pain, elevated myocardial enzymes and ECG changes, AMI is the primary diagnosis. Coronary heart disease chiefly occurs in patients over 40, and younger male and female patients have different risk factors, clinical presentations, and prognosis than older patients. Myocarditis may affect people of all ages, but it is often encountered in the patients at young ages[3]. In this case, AMI was primarily considered due to angina-like chest pain, inferior negative T-waves on the ECG, elevated cardiac markers, for this reason coronary angiography had been performed. Young age, previous acute gastroenteritis with fever, lack of segmental wall motion on ECHO and a normal coronary angiography supported to diagnosis of myocarditis. The diagnosis of acute myocarditis was confirmed using CMRI.

Conclusion

In summary, the patients with focal myocarditis may complain of angina-like chest pain, ECG changes and elevated cardiac enzymes mimicking AMI. The differential diagnosis between myocarditis and AMI can be troublesome in ED. CMRI can be a useful diagnostic tool to distinguish between myocardial ischemia, infarction, or spasm from acute myocarditis.

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