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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

The Effect of Steroid Dosage in the Treatment of Adult-Onset Still's Disease: A Case Report

Ebru Yilmaz
Kocaeli Government Hospital

Abstract

Introduction: Adult-onset Still's disease (AOSD) is a rare systemic inflammatory disorder of unknown etiology accompanied with high spiking fever, arthralgia, arthritis, myalgia, salmon-colored evanescent rash and leukocytosis. However, the clinical presentation of AOSD is heterogeneous and the spectrum of differential diagnoses-including infection, neoplasia and other autoimmune disorders, which should be ruled out before the diagnosis of AOSD can be made- is wide.

Case Report: The case of a 25-year-old male with no significant medical history presented with fever, arthralgia and evanescent, salmon-pink, maculopapular eruption for two days. His laboratory tests demonstrated particularly the presence of leukocytosis with neutrophilia, elevated AST and ALT and marked hyperferritinemia. Throughout his hospitalization, the patient was evaluated for other potential differential diagnoses. After an extensive workup, the patient was diagnosed with AOSD based on Yamaguchi criteria. He was promptly started on i.v. pulse methylprednisolone (1 gr/day for 3 days) and followed by oral corticosteroid (40 mg/day for 4 weeks). Once the patient became asymptomatic, the oral steroid dose was decreased related to side effects (4 mg/p.o./daily), and he was started on methotrexate 20 mg/s.c./weekly. However, the dosage of steroid had to be increased again because of the onset of fever and rash in the patient.

Conclusion: Although methotrexate (MTX) is well known to control both chronic systemic symptoms and arthritis and as a steroid-sparing agent for tapering dose of steroid, it seems that prednisolone (especially high dose) own a significant place in the treatment of Adult-onset Still's disease.

Key Words: Adult-onset Still's disease, Steroid therapy, Methotrexate therapy

Introduction

Adult-onset Still's disease (AOSD) is an uncommon, systemic autoinflammatory disease of unknown etiology characterized by intermittent high spiking fevers, arthralgias/arthritis, myalgia, maculopapular rash, leukocytosis, sore throat, pharyngitis, anorexia, nausea, weight loss, generalized lymphadenopathy, liver dysfunction and splenomegaly. AOSD, one of the most important causes of fever of unknown origin, is diagnosed after ruling out infection, malignancy, and rheumatologic diseases[1]. AOSD is generally treated with non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and disease-modifying anti-rheumatic drugs (DMARDs) such as methotrexate, azathioprine, tacrolimus and cyclosporine. The biological agents including tumor necrosis factor (TNF) inhibitors, tocilizumab or anakinra have also been shown to be beneficial in the management of patients with AOSD refractory to corticosteroids or conventional DMARDs or in patients presenting life-threatening manifestations[2].

The optimal management of AOSD is corticosteroids ± methotrexate. The patients with AOSD are treated initially with high dose steroids for control of disease activity. The dose of steroids needs to be reduced to the minimum, when the disease is controlled and inflammatory parameters have returned to normal. Methotrexate generally provides a complete remission of the disease or at least a significant reduction in daily corticosteroid intake[3].

In this paper, it was aimed to report a case that occur quickly relapse in spite of adding methotrexate to reduce the dosage of steroid when the patient's symptoms and biological parameters returned to normal after initially high dose of steroid treatment.

Case Report

A 25-year-old male presented to the emergency department with spiking fever with maximum temperature of 40°C and chills for two days, accompanied with loose stools, decreased appetite, evanescent rash, myalgia and generalized joint pain. He also had a salmon pink, maculopapular rash observed with the fever spike localized on the trunk and both lower extremities (Figure 1). He complained of joint pain affecting knees,

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Table 1. Yamaguchi Criteria (1992)

Major Criteria	Minor Criteria
1) Fever of at least 39°C	1) Sore throat
2) Arthralgia > 2 weeks	2) Lymphadenopathy or splenomegaly
3) Still rash	3) Liver dysfunction
4) Neutrophilic leukocytosis > 10.000	4) RF and ANA negativity

wrist, and hands. Her past and family history was insignificant. He was admitted to the department of Infectious Diseases to investigate the etiology of fever. He had been given Ceftriaxone 1 g/i.v./twice a day and Metronidazole 500 mg/i.v./3 times a day as empirical treatment. His significant laboratory findings showed marked leukocytosis (11,700 elements/mm^3; 80,6% neutrophils), elevated C reactive protein (CRP) of 132,27 mg/L, elevated erythrocyte sedimentation rate (ESR) (60 mm) and slightly elevated AST (62 IU/L) and ALT (61 IU/L). Ferritin level was also high at 1650 ng/mL. TORCH (toxoplasma, rubella, cytomegalovirus, herpes simplex virus) and EBV (Epstein-Barr virus) panels were both negative. Salmonella and Brucella tube agglutination, Rose Bengal tests, hepatitis panel, tumor markers and HIV/AIDS and VDRL tests were all negative. No proliferation was observed in the throat, blood, stool and urine cultures. Chest X-ray, echocardiography and computerized tomography (CT) of the chest, abdomen and pelvis, also abdominal ultrasonography (USG) did not show any abnormal finding. Despite the use of antibiotic treatment, the patient remained cyclically febrile. At this point, the department of Physical Therapy and Rehabilitation was consulted and noted a non-pruritic generalized erythematous maculopapular rash occurring only during febrile episodes. Rheumatoid factor (RF), anti-CCP (anti-cyclic citrullinated peptide), anti-nuclear antibody (ANA), anti-neutrophilic cytoplasmic antibody (ANCA), and anti-double-stranded DNA (anti ds-DNA) were negative and complement dosage was normal. In light of the available examinations and tests, AOSD was diagnosed according to the classification criteria of Yamaguchi et al. (Table 1)[6]. The patient received intravenous pulse methylprednisolone therapy (1 g daily for 3 days), followed by oral corticosteroids (40 mg daily for 4 weeks). He had an excellent therapeutic response to methylprednisolone during his hospital stay with a decrease in febrile frequency. Once the patient became asymptomatic, the oral steroid dose was decreased related to side effects (4 mg/p.o./daily), and he was started on methotrexate 20 mg/s.c./weekly and then discharged in a stable condition with normal laboratory findings. One week later, while he was on tapering dose of steroid, he applied to the outpatient clinic with complaints of fever and rash again. The dosage of oral steroid was increased from 4 mg/day to 40 mg/day and methotrexate was continued. He continued to remain complete remission on methotrexate and methylprednisolone as maintenance therapy after six months.

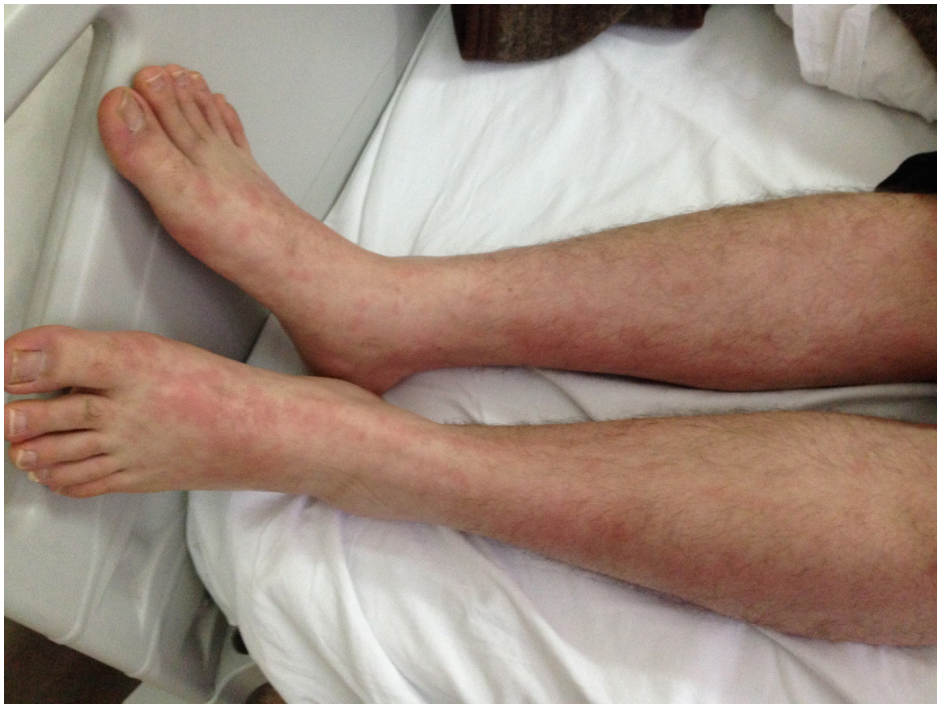

Figure 1. Diffuse maculo-papular erythema in legs

Discussion

AOSD is a rare disease. The incidence of AOSD is estimated to be about 0.16 per 100,000[1]. The age of AOSD onset had a bimodal range with two group: 15–25 and 36–46 years of age[4]. The pathogenesis of AOSD is not clear. Several factors have been suggested to contribute to the disease occurrence, including genetics, viral and bacterial infections, and immune dysfunction. The pro-inflammatory cytokines such as tumor necrosis factor (TNF)-α, interleukin (IL)-1, IL-6, IL-18and IFN-γ seem to play a major role in this disorder. The diagnosis of AOSD can be very difficult. The diagnosis of AOSD requires the exclusion of other possible disorders because it lacks specific clinical and histopathological findings. There is no specific laboratory or imaging test available for diagnosing AOSD and diagnosis is usually based on a symptom complex. AOSD is a multi-systemic inflammatory disorder accompanied by a triad of spiking fever, salmon-colored rash and arthralgia[5]. Yamaguchi's criteria are the most widely used and sensitive diagnostic tool to diagnose AOSD (Table 1)[6]. The patient had four major features of Yamaguchi criteria: fever, arthralgia, leukocytosis and rash. He also met two features of Yamaguchi minor criteria: hepatic dysfunction and negative ANA and RF.

Laboratory findings in AOSD are leukocytosis (mostly neutrophils), anemia, elevated ferritin, C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), abnormal liver function tests (AST and ALT) and ANA and RF negativity[6]. Our patient also had high levels of ferritin, ESR, CRP, AST, ALT and leukocytosis and ANA/RF negativity.

Early diagnosis and intervention may help prevent the development of complication and improve the patient outcomes[7]. AOSD treatment is based on anti-inflammatory medications, including steroids, NSAIDs, and anti-rheumatic agents to control symptoms such as fever, joint pain and systemic inflammation. Non-steroidal anti-inflammatory drugs have limited efficacy, and corticosteroid therapy and disease-modifying anti-rheumatic drugs are usually required. Corticosteroids are usually the first-line treatment. In general, the treatment of AOSD involves corticosteroids, generally medium-high to high doses (0.5–1 mg/kg/day) of prednisone equivalent[8]. Presence of high fever attacks, severe articular symptoms or internal organ involvement may justify corticosteroid (usually prednisolone) use at a dose of 1 mg/kg. The response to corticosteroids is often quick and it occurs within a couple of hours or a few days. The tapering begins usually after 4 to 6 weeks[9]. It was preferred intravenous infusion therapy of high-dose methylprednisolone as initial treatment to achieve quicker response. In this case, intravenous infusion of high-dose methylprednisolone therapy dramatically reduced the fever and rash within 3 days. Then, intravenous steroid was switched to peroral methylprednisolone 40 mg/day.

With inadequate response to corticosteroids, methotrexate appears the best choice to control disease activity and allow for tapering of steroid use. In addition, methotrexate (MTX) was introduced to avoid chronic use of high doses of corticosteroid[6].Unfortunately, steroid dependence occurs in 42% to 45% of the cases. This exposes the patients to serious mid- and long-term side effects. Thus, methotrexate should be added to prednisone when the latter fails to control the disease or in case of steroid-dependence (9).The reason why we added methotrexate to methylprednisolone therapy are: 1) to decrease the dosage of steroid, 2) to prevent the side effects of steroid, 3) to avoid the steroid-dependence. In addition, it was reduced the dosage of steroid from 40 mg/day to 4 mg/day for adding methotrexate. However, the dosage of steroid had to be increased again because of the onset of fever and rash in the patient.

Conclusion

Although methotrexate (MTX) is well known to control both chronic systemic symptoms and arthritis and as a steroid-sparing agent for tapering dose of steroid, it seems that prednisolone (especially high dose) own a significant place in the treatment of AOSD.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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Case Report

Journal of Emergency Medicine Case Reports

Iliac Artery Dissection: A Case Report

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Abstract

Introduction: Iliac artery aneurisms are generally observed associated with abdominal aortic aneurisms. Iliac artery dissection happens to be one of the rarely encountered cases and very few cases have been reported in literature. We aimed to present a patient with both iliac artery aneurisms associated with left iliac artery dissection in this study.

Case: A male, 34-years old patient applied to emergency service with complaints of abdominal pain and paresthesia in his left leg. There was no pain or loss of strength in the left leg of the patient, but there existed a numbness described by himself. The brain computed tomography (CT) of the patient was considered as normal but his abdominal ultrasonography was reported as "an aneurismal dilatation in both iliac artery at bifurcation level on a nearly 3 cm-segment, reaching up to 3 cm at the widest section and a mural thrombus reaching up to 70 % stenosis within the vein lumen were observed". Thereon, the patient went through a CT-angiogram and as a result dissection was observed in the left iliac artery together with aneurismal dilatation and mural thrombus in both iliac arteries The patient was hospitalized in the intensive care unit of cardiovascular surgery clinic.

Conclusion: In regards to the patients who apply to emergency services with complaints of abdominal pain, numbness in extremities, acute abdomen or neurologic symptoms atypically seen like loss of strength; we should also keep in mind aortic dissection and/or iliac artery dissection among our preliminary diagnoses.

Keywords: Abdominal pain, numbness in extremities, iliac artery dissection

Introduction

Iliac artery aneurisms are generally observed associated with abdominal aortic aneurisms. While isolated iliac artery aneurisms are lesser seen, they constitute 0.4 % and 2 % of all aneurismal diseases[1]. Iliac artery dissection happens to be one of the rarely encountered cases and very few cases have been reported in literature[2-4]. We aimed to present a patient with both iliac artery aneurisms associated with left iliac artery dissection in this study.

Case

A male, 34-years old patient applied to emergency service with complaints of abdominal pain and paresthesia in his left leg. The vital parameters of the patient were body temperature 36.6°C, pulse 76/min, blood pressure 135/76 mmHg and respiratory rate was 17/min. Although the abdomen was painful and sensitized in the physical examination of the patient, there was no defense-rebound. There was no pain or loss of strength in the left leg of the patient, but there existed a numbness described by himself. There was no difference in the right-left blood pressure and his peripheric pulses were palpable on upper extremities, but regarding the lower extremities, extremity pulses were weakly taken, the left lower extremity being pulsed more weakly. Other system examinations of the patient were natural. With respect to blood tests of the patient, his hemogram was at normal level and in his biochemical values, creatinine 1.28 (normal range 0.72-1.25 mg/dl) and other parameters were also normal. Electrocardiography (ECG) of the patient was normal, and sinus rhythm and troponin value was also normal. Following the physical examination, the patient was demanded a cranial tomography for intracranial pathologies and an ultrasonography for abdominal pain. The cranial computed tomography (CT) of the patient was considered as normal but his abdominal ultrasonography was reported as "an aneurismal dilatation in both iliac artery at bifurcation level on a nearly 3 cm-segment, reaching up to 3 cm at the widest section and a mural thrombus reaching up to 70 % stenosis within the vein lumen were observed". Thereon, the patient went through a CT-angiogram and as a result dissection was

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observed in the left iliac artery together with aneurismal dilatation and mural thrombus in both iliac arteries (Figure 1). The patient was asked for a neurological consultation for intracranial pathologies and a cardiovascular surgery consultation for his iliac artery aneurism + dissection. The neurology department stated that his present stated was not related to any intracranial case and the patient was hospitalized in the intensive care unit of cardiovascular surgery clinic.

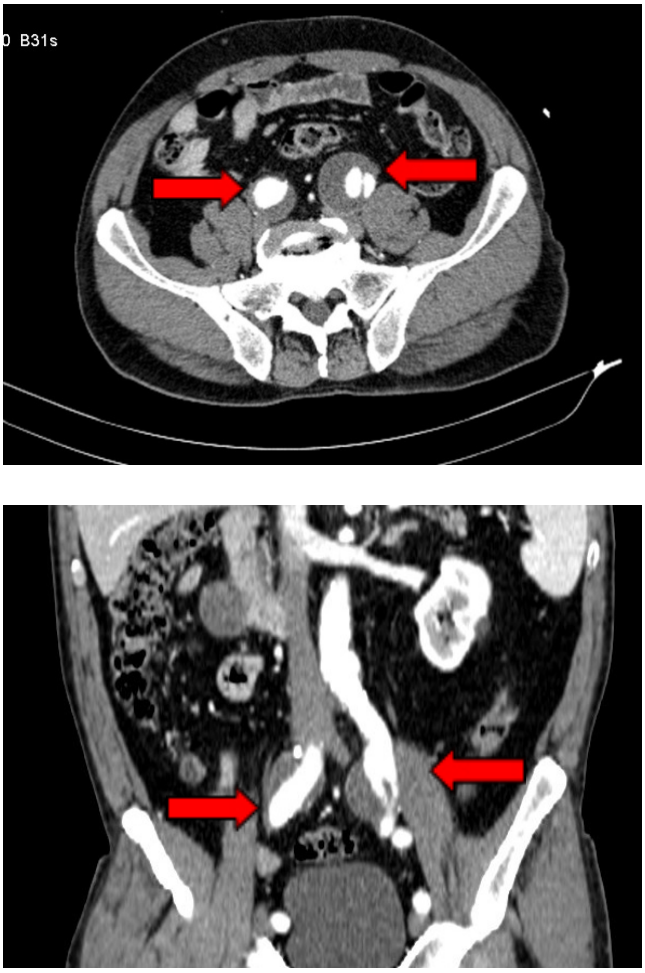

Figure-1: Image of BT-angiogram compatible with aneurismal dilatation in both iliac arteries, mural thrombus and dissection in the left iliac artery.

Discussions

Iliac artery aneurisms are often coincidentally diagnosed during the imaging studies conducted for other reasons. Most of the findings occur depending on the erosion and rupture within the tissues. In nearly more than half of the patients, the symptoms are existent. The patients rather have lower abdominal and flank pain. The findings generally take place depending on the compression on the surrounding anatomic structures. The most frequent findings are glomerulonephritis, pain during defecation due to rectal compression, paresthesia in lower extremities due to pelvic nerve compression and ischemia in the lower extremities. As these symptoms are not directly related to the arterial system, the diagnosis might be delayed. Besides, findings like thrombosis, emboli and fistulae might be seen less often associated with iliac aneurisms. In case the aneurisms are ruptured; abdominal pain, hypotension and bradycardia could be observed[5]. In our case, the patient had abdominal pain along with a complaint of paresthesia in the left leg. Atherosclerosis is known as the most common reason in the etiology of this type of aneurisms. Rarely encountered reasons in etiology can be counted as paraanostomatic pseudoaneurysm, penetrating pelvic trauma, iatrogenic lesions, bacterial infections, Kawasaki syndrome, Behçet's disease, fibromuscular dysplasia, Takayasu arteritis, connective tissue diseases (cystic medial necrosis, Marfan syndrome and etc.)[6,7]. Many of the conducted researches showed that symptom or rupture risk of isolated iliac artery aneurisms are closely related to the diameter of the aneurism. The risk of rupture increases considerably, particularly if the diameter of iliac artery aneurism is over 3 cm[8]. On the other hand, iliac artery dissections are very rarely encountered cases. Again atherosclerosis is put forward as the most frequent reason in cases reported in literature. Almost in all of the cases related to atherosclerosis, the age of the patients are over 50. In our case, the patient was young and 34 years old. Unless there is a story of dissection or trauma developed on aneurism basis, it becomes hard to make diagnose in iliac artery dissection cases. It is not usually possible to make correct diagnosis during first application even in cases who have a trauma story[9]. The initial application of the patients might be induced from aneurism rupture and this situation is generally encountered in cases who have developed iliac artery dissection developed on aneurism basis. The type of treatment in these cases changes depending upon the symptoms of the patients during their application which covers a wide spectrum ranging from medical therapy to urgent surgical interventions.

Conclusion

In regards to the patients who apply to emergency services with complaints of abdominal pain, numbness in extremities, acute abdomen or neurologic symptoms atypically seen like loss of strength; we should also keep in mind aortic dissection and/or iliac artery dissection among our preliminary diagnoses.

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Case Report

Journal of Emergency Medicine Case Reports

Clinical Course Of A Death From Ranolazine Overdose: A Case Report

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Abstract

Introduction: Ranolazine is a new medication for angina pectoris. It is usually used for refractory angina pectoris. Ranolazine overdose may cause some serious effects on heart like other cardiac medications.

Case Report: We report the case of a 21-year-old girl was brought to the emergency department because of an attempt to commit suicide with a large number of Ranolazine tablets. She went into the coma and was unresponsive to verbal and painful stimuli after two hours of admission. There was no pathological findings on brain computed tomography (CT) and Magnetic Resonance Imaging (MRI). The QT interval was 420 msec and more extended than previous measurements. The patient had three episodes of polymorphic nonsustained ventricular tachycardia (NSVT). Despite the amiodarone treatment, the NSVT evolved to polymorphic sustained ventricular tachycardia (Torsade de Pointes; TdP) and Ventricular fibrillation (VF). The patient underwent CPR and accepted as death end of the resuscitation due to asystole on the ECG.

Conclusion: Ranolazine overdose is known that may cause some serious ECG changes and seizures. But suddenly entering a coma and death has never been reported with ranolazine intoxication in literature.

Keywords: Ranolazine overdose, Death, Torsades De Pointes

Introduction

Ranolazine is an anti-anginal and anti-ischemic medication. It was approved for the treatment of chronic, stable angina by the Food and Drug Administration (FDA) in 2006. İt improves exercise performance and prevents the symptoms of myocardial ischemia during exercise in patients with chronic angina[1]. Ranolazine is a safe drug, but it can cause side effects at higher dosages. Common side effects include nausea, constipation, dizziness and prolonged QT (QTc) intervals in a dose dependent manner. It has also been reported that ranolazine can cause seizures[2].

In this report, we presented a case with high dose ranolazine usage that was related to new onset convulsions and QT interval changes, and ultimately, to death due to ventricular arrhythmia.

Case Report

A 21-year-old woman was brought to the emergency department by her family. She had attempted to commit suicide by ingesting a large number of ranolazine tablets one hour prior to her admission. She had no history of medical problems and was not taking any medications. On admission, the patient had dysarthria and dysmetria; otherwise, her neurological examination was normal except that she was disoriented to time, place and person and had difficulties in following commands. The patient's vital signs were within the normal range. Her electrocardiogram (ECG) showed that her heart was in a sinus rhythm, and that her QT interval was nearly normal (Figure 1) [heart rate (HR) = 92 beats per minute (bpm); QT interval = 360 milliseconds (msec); cQT = 446 msec with Bazzet's formula]. A venous blood sample was collected, and ranitidine [50 milligrams (mg)] and omeprazole (40 mg) were administered as a slow intravenous injection to protect the stomach. A nasogastric tube was inserted, and gastric lavage was performed by passing water through the tube. After completion of the gastric lavage, 100 grams of activated charcoal was administered through the nasogastric tube. The results of blood chemistry studies, including liver function tests, blood glucose level test, and urea, creatinine and serum electrolytes levels [Sodium (Na), Potassium (K), Magnesium, Calcium (Ca) and Chloride (Cl) were within reference ranges. A complete blood count, arterial blood gas test and troponin I test were also within reference ranges. Two hours after admission,

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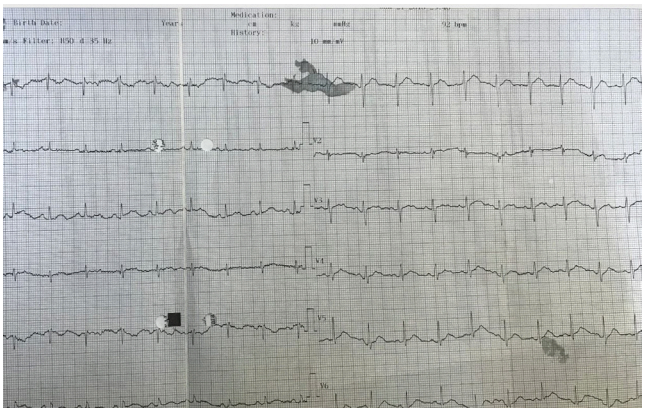

Figure-1

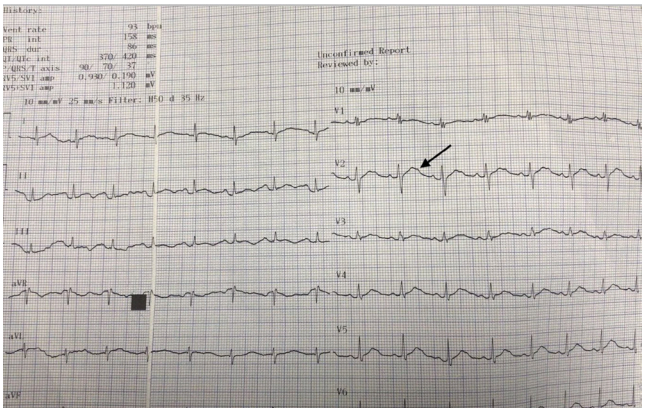

Figure-2

she was unresponsive to verbal and painful stimuli. The patient was intubated due to respiratory failure. The ECG was repeated two hours after the first ECG; the QT interval was 420 msec and was more extended than in the first ECG (Figure 2) (HR = 93 bpm, QT interval = 420 msec, cQT = 523 msec with Bazzet's formula). A computed tomography scan (CT) and magnetic resonance imaging (MRI) were performed on the brain, and there were no pathological findings.

The patient had two episodes of generalized tonic-clonic convulsions within a short time period—two hours after admission. Diazepam was given as an intravenous 45 mg bolus for convulsions, and the patient was admitted to the intensive care unit (ICU) for closer monitoring. The patient developed three episodes of polymorphic nonsustained ventricular tachycardia (NSVT) six hours after admission to the ICU; therefore, continuous intravenous amiodarone was administered. Despite the amiodarone, the NSVT evolved to polymorphic sustained ventricular tachycardia (Torsades de Pointes, TdP) that required a 300 Joules (J) cardiac defibrillation. TdP evolved to resistance ventricular fibrillation (VF) after the cardiac defibrillation. Resistance VF continued against the 360 J defibrillation, and cardiopulmonary resuscitation (CPR) was started. Atropine, adrenaline and magnesium were administered during CPR. Asystole was observed during CPR. The patient underwent CPR for 95 minutes and was declared dead at the end of the resuscitation due to asystole on the ECG.

During the autopsy, medicine tablet particles were found in the stomach contents. Also, 38 nanograms per milliliter (ng/ml) diazepam, 27 ng/ml atropine, 121 ng/ml metoclopramide and 477 ng/ml ranitidine were detected in the blood due to medications which were administered during treatment and resuscitation. The forensic medicine department reported that the cause of death was medicine intoxication (no medication name was specified). We interviewed the laboratory manager for forensic medicine and confirmed that ranolazine was not among routine medications they look for in samples in the forensic laboratory. This told us that the tablets in the stomach contents were ranolazine, and that they caused cardiac arrest due to overdose.

Discussion

Ranolazine is a new anti-ischemic medication recommended in patients with stable angina. Ranolazine significantly improves exercise duration, time to angina and time to ST-segment depression either as monotherapy or in combination with anti-anginal medications (e.g., amlodipine, atenolol and diltiazem). Ranolazine interrupts the pathophysiology of myocardial ischemia and prevents diastolic failure by inhibiting the myocardial late inward sodium current related to ischemia. Thus, ranolazine could increase the diastolic myocardial oxygen supply during ischemia[1, 3].

A combination assessment of ranolazine in stable angina (CARISA) trial studied the role of ranolazine on angina frequency and revealed a significant reduction in mean angina attacks each week. The same trial also reported significant increases in treadmill exercise performance and significant decreases in angina frequency and nitroglycerin consumption[4]. Ranolazine is widely metabolized primarily through the cytochrome P450 3A4 (CYP3A4) pathway with a small amount (5 percent) excreted unchanged in the urine, and it is contraindicated in patients with hepatic and severe renal impairment, those with QTc prolongation or those who are using drugs known to prolong the QT interval[5].

The most common adverse effects reported in clinical studies include dizziness, constipation, nausea, hypotension and headache along with post-marketing reports of angioedema, ataxia, and Torsade de Pointes. Dizziness and nausea are the most common reasons for discontinuation of ranolazine. Also, postural hypotension and syncope developed in healthy volunteers with higher doses and without cardiac arrhythmias or QT prolongation[4, 6]. Kalra et al. reported that ranolazine caused myoclonus and hallucinations[7]. Our patient had a generalized tonic-clonic convul-

sion, which might be related to ranolazine blockage of the sodium (Na) channel in the brain.

Ranolazine increases the action potential duration and therefore prolongs QT intervals in a dose dependent manner[1]. Another study reported that ranolazine is associated with TdP, which is related to the QT interval prolongation usually due to the inhibition of the rapid outward potassium currents (IKr)[8]. We assumed that cardiac arrhythmia developed due to potassium channel blockage as a result of a ranolazine overdose in the patient. Also, T-wave notching has been observed at high ranolazine concentrations in the plasma, and our patient's last ECG showed T-wave notching (Figure 2, black arrow)[9].

Conclusion

In this case report, we presented an overdose of ranolazine that was related to convulsions and TdP. The QT interval was associated with malignant arrhythmias such as TdP and VF. TdP evolved to resistive ventricular fibrillation and then to asystole in the patient.

To our knowledge, suddenly total loss of consciousness(-coma) and death from ranolazine intoxication has never been reported. Because fatal ECG and consciousness changes were observed in our patient, we recommend that an ECG should be acquired at baseline with close follow-up for at least 24 hours in the ICU to assess the QT and vital sign changes in patients who are suspected of ranolazine overdoses.

CONFLİCT OF İNTEREST

We declare that there is no conflict of interest.

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Case Report

Journal of Emergency Medicine Case Reports

Successful Computed Tomography-guided Pericardiocentesis After Performing a Failed Conventional Transthoracic Echocardiography-guided Approach: A Case Report

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Abstract

İntroduction: Computed tomography-guided pericardiocentesis has been shown to be technically feasible and safe for treating pericardial effusion. However, no recommendations for computed tomography-guided pericardiocentesis have been made and standardized among the established guidelines by the European Society of Cardiology or the Japanese Circulation Society. We report on a case of successful computed tomography-guided pericardiocentesis after performing a failed conventional transthoracic echocardiography-guided approach in a patient with cardiac tamponade.

Case Report: A 71-year-old man was admitted to our hospital with progressive chest discomfort and cardiac tamponade. Transthoracic echocardiography-guided pericardiocentesis was primarily performed on the patient; however, the quality of the transthoracic echocardiography images was inadequate to guide the procedure, resulting in procedure-related pericardial injury. When cardiac tamponade recurred, a computed tomography-guided approach successfully treated the patient, with the success owed to precise intraoperative visualization of the needle and organs.

Conclusion: Our case suggests that computed tomography guidance is safe and useful in pericardiocentesis, especially for patients who are ineligible to undergo an echo-guided approach.

Keywords: pericardiocentesis, computed tomography,

Introduction

Computed tomography (CT)-guided pericardiocentesis has been shown to be technically feasible and safe for treating pericardial effusion (PE). However, unlike that with echocardiographic guidance, there are no recommendations regarding treatment by the CT approach for pericardiocentesis in the current standard guidelines, including those from the 2015 European Society of Cardiology and 2017 Japanese Circulation Society[1,2]. These guidelines introduce no alternative to surgical management as a second-best approach to treat cardiac tamponade in patients ineligible for conventional echocardiography-guided pericardiocentesis. Conventional echocardiography-guided pericardiocentesis is contraindicated in patients due to the absence of an optimal acoustic window, which is typically associated with chronic obstructive pulmonary disease, obesity, or post-thoracic surgery. One potential reason for the lack of guidance concerning CT-guided pericardiocentesis is that evidence supporting the competitive advantage of CT guidance over transthoracic echocardiography (TTE) is still lacking[3-5]. Thus, the optimal application of CT guidance for pericardiocentesis is unclear. We report on a case of successful computed tomography-guided pericardiocentesis after performing a failed conventional transthoracic echocardiography-guided approach in a patient with pericardial effusion.

Case Presentation

A 71-year-old man visited our hospital with progressive chest discomfort. There was no past and family history of disease and no alcohol consumption. He had smoked 20 cigarettes daily for

50 years. His vital measurements on arrival were as follows: blood pressure, 105/75 mmHg; heart rate, 84 beats/min; respiratory rate, 24 breaths/min. He was admitted to the hospital under suspicion of pneumonia with pleural effusion.

Approximately 36 h after admission, his circulatory and respiratory condition worsened. His blood pressure decreased to 86/54 mmHg. and heart rate and respiratory rate increased to 112 beats/min and 40 breaths/min, respectively. Repetitive TTE and his physical signs and symptoms confirmed emergent cardiac tamponade (Figure 1A).

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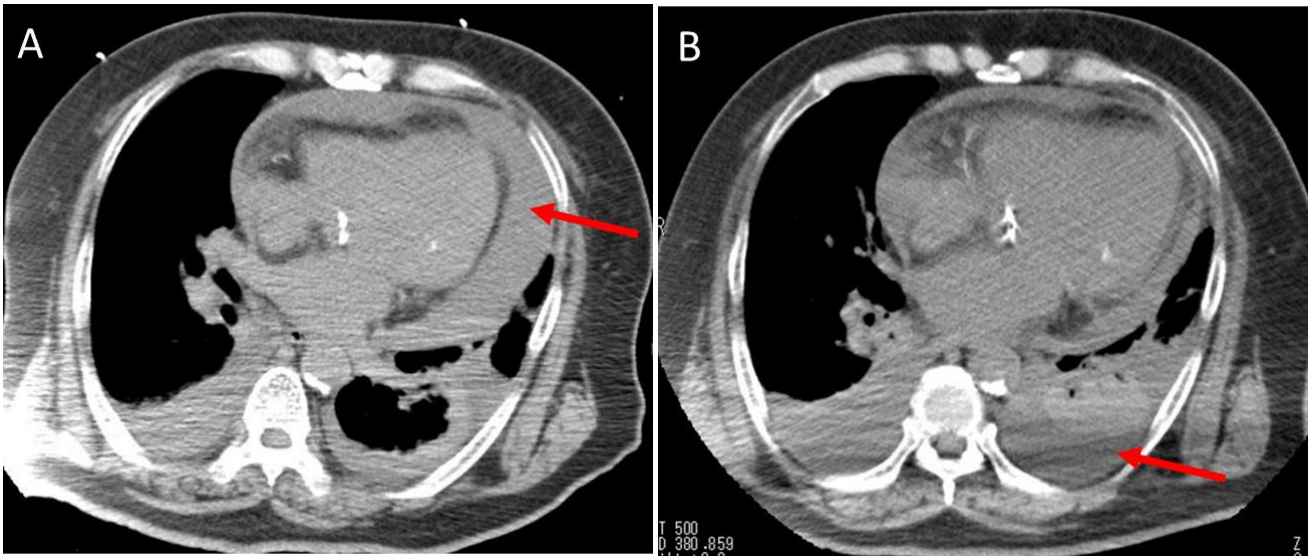

Figure-1: Computed tomography (CT) imaging before and after transthoracic echocardiography- guided pericardiocentesis. (A) Pre-procedural CT scan revealing the presence of extensive fluid collection (arrow), which was predominantly in the pericardial cavity. (B) Procedure-related epicardial injury causing a shift of fluid from the pericardial cavity to the thoracic cavity (arrow).

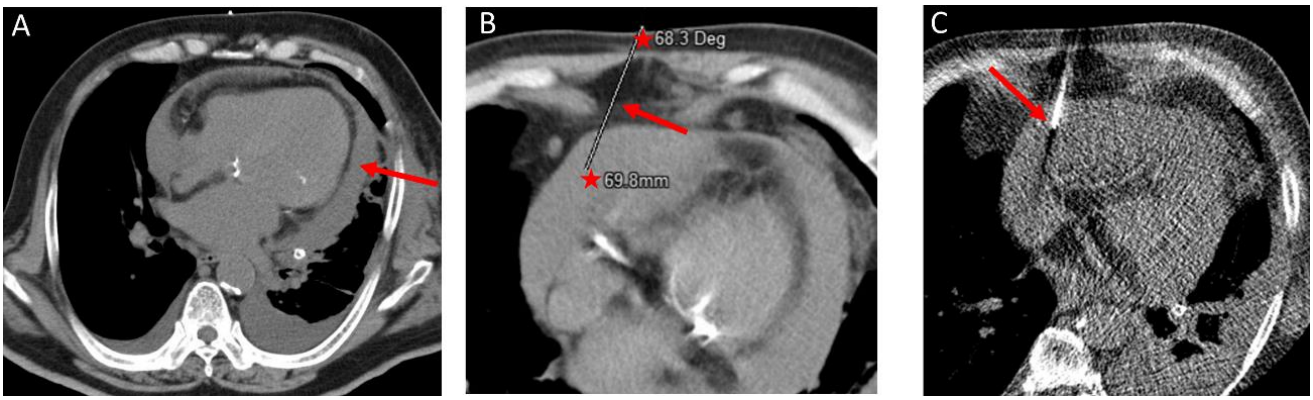

Figure-2: Procedural images of the computed tomography-guided approach. (A) Re-accumulation of pericardial effusion (arrow) was observed. (B) Both the distance to the pericardial cavity and angle of approach (stars) to the pericardial cavity were calculated based on the pre-designed route of needle insertion (arrow). (C) Computed tomography fluoroscopy provided accurate information about the position of the needle tip.

TTE-guided pericardiocentesis was attempted using a commercially available pericardiocentesis kit (Merit Medical Systems, South Jordan, Utah, USA). However, the quality of the TTE images was insufficient for complete visualization of the puncture needle due to ultrasound attenuation by intervening tissues and organs. Needle insertion was forced several times under suboptimal TTE image guidance, which resulted in injury-induced pericardial fenestration and subsequent relief from tamponade through efflux of pericardial fluid into the thoracic cavity (Figure 1B). Nine days later, cardiac tamponade recurred with a smaller amount of PE than what had accumulated during the first pericardiocentesis (Figure 2A). We decided to use a CT-guided approach to overcome the technical difficulties experienced with the previous TTE-guided procedure. Axial images acquired using a multidetector-row CT scanner (Aquilion Prime, Toshiba Medical Systems, Japan) clearly visualized both the puncture needle and surrounding structures. The images enabled us to determine the optimal puncture site and direction satisfying the following criteria to avoid injury to the surrounding organs: confirming that the parietal pericardium was directly attached to the thoracic wall and that sufficient pericardial space for needle penetration was present (Figure 2B). An 18-gauge puncture needle was advanced into the pericardial cavity under intermittent-mode CT guidance (Figure 2C). After controlled penetration of the needle into the pericardial cavity, a 0.035-inch guidewire was advanced through the needle sheath. A 6 Fr pigtail drainage catheter was inserted in the pericardial cavity over the guidewire using the Seldinger technique. A post-procedural CT scan was performed to confirm that the drainage catheter was placed within the pericardial space and that there were no procedure- associated complications (Figure 3A, B). Hemodynamic compromise was successfully relieved. Cytological analysis of the fluid samples provided evidence of malignant adenocarcinoma. His total duration of hospital stay was 45 days, and she was discharged home without neurological defects.

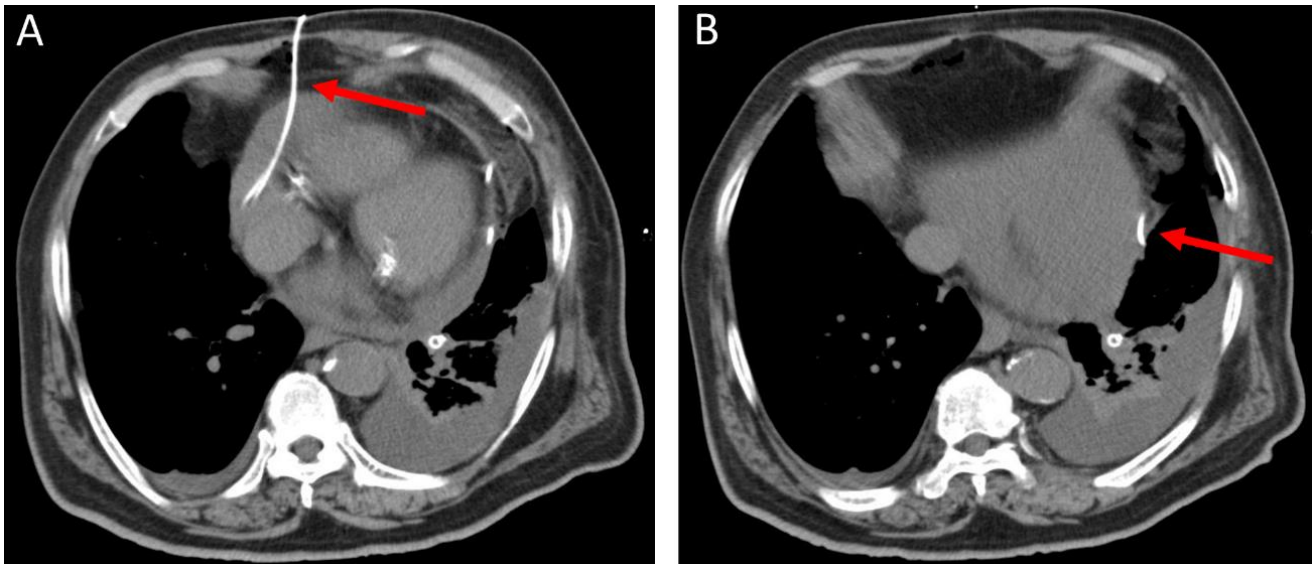

Figure-3: Post-procedural computed tomography imaging. (A, B) A drainage catheter (arrows) was successfully positioned within the pericardial cavity and the pericardial effusion was removed.

Discussion

In the present case, CT-guided pericardiocentesis was successfully performed, even with a comparatively small volume of PE, in a patient for whom initial TTE-guided pericardiocentesis failed due to the poor quality of the sonographic images. While ultrasound imaging is insubstantial due to air-filled organs or bone, CT overcomes the potential limitations of TTE by enabling precise visualization of the intervention needle independent of procedural skill or experience, even when lesions are deep-seated.

Historically, ultrasonography and X-ray fluoroscopy were considered the standard imaging modalities for pericardiocentesis. Current guidelines for the treatment of PE recommend TTE as a routine imaging modality, according to previous reports describing the high success rate of conventional ultrasound-guided pericardiocentesis[1,2,6,7]. However, physicians should be aware that most available evidence supporting the usefulness of TTE guidance have little information regarding the resultant imaging quality of TTE, and have assessed the procedure based on selection of patients deemed technically eligible for TTE-guidance[7-10].

The CT-guided procedure is an established strategy for assisting with percutaneous interventions in a variety of diseases. CT-guided pericardiocentesis is a safe and effective treatment option for patients with PE. Neves et al. reported that CT-guided pericardiocentesis was successful in 94% of patients, and that unsuccessful cases had localized or organized PE, which was shown to be unsuitable for percutaneous drainage[4,8]. Furthermore, Bruning et al. reported that CT-guided pericardiocentesis was successfully performed in 10 of 11 cases, including eight patients where TTE guidance was determined to be inappropriate[3]. These studies indicate a clinical utility for CT-guided pericardiocentesis. Our case further highlights, in a straightforward manner, the importance of CT guidance for pericardiocentesis in patients who could not be appropriately treated using conventional echocardiographic guidance.

An additional advantage of CT guidance revealed by our case is its ability to detect procedure- related injury to non-cardiac organs, especially the lungs, which are difficult to visualize using ultrasonography. Intraoperative CT scans facilitate immediate intervention for serious complications without the need to leave the procedure room[10], It is important for physicians to recognize fluctuations in the size of the pericardial space during the cardiac cycle, especially in patients with small volumes of PE. Continuous-mode CT imaging, which can provide real-time tracking of the needle position, is an additional option. It can minimize technical complications at the expense of a higher radiation dose.

Conlusion

Our case suggests that CT guidance is technically safe and useful, especially to treat for pericardiocentesis in patients ineligible for an echo-guided approach. CT enables optimal imaging to guide needle insertion, thus making the procedure effective and safe to perform. Physicians should be aware that CT-guided pericardiocentesis is readily adapted to a wide variety of clinical presentations, including pericardiocentesis.

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Case Report

Journal of Emergency Medicine Case Reports

Acute Kidney Injury After Near-Drowning In A Pool Case Report

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Abstract

Introduction: Acute kidney injury is a neglected complication of submersion injury.

Case Report: A young man presented to a hospital after a near drowning event. He was clinically stable and discharged well from the hospital. He sought medical attention few days later for non-specific symptoms. His was found to have to severe acute kidney injury despite being fairly well and clinically stable. He was treated and recovered without need for hemodialysis.

Conclusion: Acute kidney injury presents in a delayed fashion after submersion injury. Acute kidney injury may not be apparent immediately after a drowning event and victims should be re-evaluated for acute kidney injury a few days after the initial event.

Keywords: Submersion, drowning, acute kidney injury, acute renal failure, rhabdomyolysis

Introduction

Drowning is a traumatic scenario that is uncommonly encountered. Pulmonary and neurologic complications are the most worrying. Healthcare providers tend to neglect other less apparent complications when the victim is well.

Case Report

A 35 year old male with no past medical history of note, first presented to the Emergency Department (ED) of another hospital after a near-drowning (ND) incident. He was swimming in a pool (freshwater) together with his young son who was seated around his shoulder. At one point his son panicked and struggled and grabbed patient around his neck, resulting in him submerging on and off underwater. The duration of struggle was less than 2 minutes and there was no loss of consciousness. He was able to pull himself out of the water after a passerby threw him a float. He felt generalized weakness, slightly breathless and nauseous after that.

His initial parameters in the ED were temperature 35.8°C, pulse rate 108/min, blood pressure 129/58 mmHg, respiratory rate 18/min and oxygen saturation of 99% on room air. Physical examination was largely unremarkable except cold peripheries. His chest x-ray was clear and he was discharged well on the same day with temperature at 37.2°C and oxygen saturation at 100% on room air.

He attended our ED 3 days later with non-specific symptoms. His chief complaint was feeling lethargic since then, associated with nausea and having a metallic taste in his mouth. Other complaints include vomiting 1-2 times a day, mild upper abdominal discomfort, feeling feverish, mildly breathless and low backache. His oral intake decreased past few days but he was still passing same amount of urine with no discoloration. His parameters then were temperature 36.9°C, pulse rate 71/min, blood pressure 129/65 mmHg, respiratory rate 19/min and oxygen saturation of 98% on room air. Physical examination was again unremarkable. His chest x-ray was clear. However, laboratory investigations revealed elevated creatinine (Cr) (1065 µmol/L) and urea (20.5 mmol/L). His venous blood gas showed a pH of 7.38, pCO2 of 30 mmHg, bicarbonate 18 mmol/L, base excess of -7 and lactate of 0.54mmol/L. His creatine kinase (CK) was also found to be elevated at 25404 U/L.

He was started on intravenous hydration and admitted. Aggressive intravenous hydration was commenced with good urine output. He was reviewed by Nephrology and started on oral sodium bicarbonate. An ultrasound of the

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kidneys and bladder showed both kidneys of normal size but increased renal echogenicity. His Cr levels increased to a peak of 1267 µmol/L 3 days after admission (6 days after ND event) before improving while his CK levels steadily decreased after hydration. He was not started on dialysis. Patient reported improvement in symptoms only after 2-3 days of treatment. He was discharged well 9 days after admission with a creatinine of 152 µmol/L, urea of 8.9 mmol/L and creatine kinase of 103 U/L. His creatinine levels 7 weeks after the ND incident was back to normal levels.

Discussion

Acute kidney injury (AKI) in drowning cases had been described previously but due to its scarcity and poorly understood mechanism, it is often under-recognized.

Commonly described mechanisms for AKI in drowning include:

- ❑ Renal hypoxic ischemia (from hypoxemia from pulmonary insufficiency or systemic hypotension) with subsequent reperfusion injury
- ❑ Rhabdomyolysis

Other mechanisms:

- ❑ Hypothermia (may or may not be associated with drowning) induced renal impairment has been described. Reduced renal blood flow is considered as the mechanism although the specific pathophysiology is unknown
- ❑ Profound renal vasoconstriction due to intense sympathetic activity and stress-related release of angiotensin II with resulting renal hypoperfusion and hypoxia[1]
- ❑ Increased oxygen demand for tubular transport (due to enhance solute delivery and reactive oxygen species mediated mechanisms at the cellular level)[11]
- ❑ Unknown

This is an unusual case of acute kidney injury (AKI) associated with drowning. Most cases of acute kidney injury in the context of drowning are in the context of severe hypoxemia with multi-organ involvement. A retrospective analysis of 95 cases by Gorelik et al.[1] this year also showed that AKI in drowning is associated with need for resuscitation, mechanical ventilation and with the degree of acidemia, lactemia, and ventilatory failure. In this case, the patient was relatively well with stable vital signs, not requiring any form of supplemental oxygen or hemodynamic support.

Table 1. Summary of cases of delayed diagnosis of AKI in well victims

Case Report	Type of presentation	Year of publication	Type of water body	Time from drowning to presentation (days)	Duration of observation during first visit (hours)	Time between initial and 2nd visit (days)	Presenting complaint	Peak creatinine levels (µmol/L)	Hemodialysis required
Amir[3]	Delayed	2013	Fresh	4	-	-	Flank pain, nausea, loss of appetite, polyuria	1213 (5 days after incident)	Unknown
Logan[4]	Delayed	2017	Sea	1	-	-	Nausea, fatigue	451 (3 days after incident)	No
Hegde[5]	Delayed	2003	Sea	2	-	-	Myalgia, reddish urine and subsequent anuria	707 (2 days after incident)	Yes
Alp[6]	Reattendance	2016	Sea	Immediate	24	2	Nausea, flank pain, vomiting	707 (3 days after incident)	No
Seong[7]	Delayed Reattendance	2012	Lake	3	12	3	Tiredness, anorexia, anuria	1017 (6 days after incident)	Yes
Hottelart[8]	Reattendance	2004	Lake	Immediate	24	5	Tiredness, anuria	1600 (6 days after incident)	Yes
Current case	Reattendance	Pending	Fresh	Immediate	<6	3	Lethargic, nausea, metallic taste in mouth	1267 (6 days after incident)	No

A case series of 30 patients by Spicer et al.[2] in 1999 showed serum bicarbonate, pH and base excess to be predictors of AKI in drowning on univariate logistic regression analysis. Base excess was the best predictor by multivariate logistic regression analysis.

Our literature search revealed that AKI in seemingly well victims of near drowning is often diagnosed after a delay of a few days from the initial event. This is due to victims presenting late[3-5] or due to AKI not being evident or picked up during the immediate medical visit post drowning[6-8] and only detected the 2nd time the victims re-attended. Summary of the cases is shown in Table 1. Similarly, AKI in our case is only picked up 3 days after the initial near-drowning event even though the patient had been evaluated at a medical facility immediately after the event.

AKI often continues to progress beyond the initial presentation as seen in case reports.[4,9,10] Serum creatinine of 67% of patients with AKI continued to peak beyond the first day in the case series by Spicer. [2] The case series by Gorelik[1] noted that mean creatinine levels reached a maximum at 48 hours from presentation for cases with higher degree of renal impairment.

Conclusion

We suggest that for all near-drowning or immersion cases, a 24 hour period of observation in the emergency department observation unit may not be enough to pick up AKI and that provision should be made for a follow-up re-evaluation after a few days if the victim is deemed well enough to be discharged from the ED following an immediate presentation after near-drowning.

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Case Report

Journal of Emergency Medicine Case Reports

Spontaneous Spleen Rupture and Apixaban

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Abstract

Novel oral anticoagulants (NOACs) are now widely used. Spontaneous splenic rupture (SLR) is a rare condition. It is life-threatening, especially if not immediately recognized. Previous studies have shown an association between NOAC use and SLR. We report a case of 53-year-old female patient who responded to cardiopulmonary resuscitation (CPR) after SLR. The patient, who had an external cardiac device for heart failure 13 months ago was admitted to our emergency department. She was unconscious, pale, hypotensive and had tachycardia. Free fluid was detected in the peritoneum by bedside ultrasonography. The patient was resuscitated for 20 minutes after arrest during the follow-up period. She was stabilized and computed tomography showed a large subcapsular splenic hematoma with active extravasation around the spleen, but persistent low urine output developed with hypotension despite resuscitation. She was taken to the operating room for splenectomy and abdominal washing. She was then recovered and discharged 17 days later. We wanted to draw attention to a patient with SLR due to NOACs. Rapid diagnosis and aggressive early resuscitation are critical in the management of NOAC associated SLR.

Keywords: Resuscitation, NOAC, spontaneous splenic rupture

Introduction

Novel oral anticoagulants (NOACs) are more preferred today. Non-traumatic, spontaneous splenic rupture is very rare and especially life-threatening if not immediately recognized[1, 2]. Although organ-specific bleeding is much rare with NOACs compared to warfarin use, there is still a risk of intracranial bleeding and gastrointestinal (GI) bleeding[3]. Splenic rupture is rare compared to these bleeding complications. In many cases, splenic rupture is associated with an underlying pathological process (atraumatic-pathological rupture) rather than being atraumatic and idiopathic. Previous studies have shown an association with the use of NOACs and spontaneous splenic rupture in patients with risk factors[4, 5] and underlying serious chronic diseases[6, 7]. Although splenic rupture is well known in infectious mononucleosis, more atypical relationships with infections include follow-up of intravenous immunglobulin therapy for pure red cell aplasia associated with babesiosis[4] and parvovirus B19[8]. In the case of ectopic pregnancy in the spleen[9], the underlying pathological mechanism is more clear. Gonzva and colleagues described the first non traumatic spontaneous splenic rupture case associated with NOAC use in a 67-year-old patient from France with an acute shock presentation that was good after resuscitation, splenectomy and intensive care[10]. Another case was described with spontaneous splenic rupture, who was at age 57 and anticoagulated for pulmonary embolism resulted with shock[11] .

Case Description

We present a case of a 53-year-old female patient responding to CPR after arrest due to spontaneous splenic hemorrhage. The patient underwent an external cardiac device 13 months ago due to heart failure was admitted to our emergency department. She was unconscious, pale, hypotensive and had tachycardia. Bedside ultrasonography detected free fluid in the peritoneum. The patient was arrested during the follow-up period and was revived with 20 minutes of CPR following endotracheal intubation and underwent massive transfusion protocol. The patient was given 4 units of packed red blood cells, 1 unit of fresh frozen plasma, prothrombin complex concentrate and calcium. The first hemoglobin and hematocrit were 9.2 g / dL per microliter and 28.4% respectively. The second hemoglobin was 6.0 g / dL and hemotocrit was 18.4% after three hours of admission. The patient's prothrombin time and international normalized rate were 19.5 seconds and 3.89. Patient's blood pressure and heart rate improved. Computed tomography of the relatively stabilized patient (Figure 1) showed intra-peritoneal hematoma with

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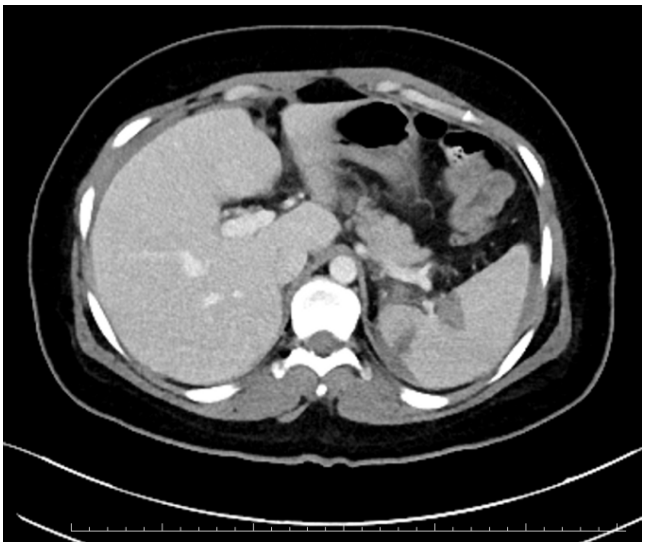

Figure 1.

active extravasation around the spleen, and despite resuscitation, persistant low urine output with hypotension developed. She was taken to the operating room for a splenectomy and abdominal wash. She recovered after the post-op intensive care unit follow-up and was discharged 17 days later. We wanted to draw attention to a case of arrest as a result of non-traumatic spontaneous splenic rupture due to NOACs. Rapid diagnosis and aggressive early resuscitation are critical in the management of spontaneous splenic rupture.

Discussion

Atraumatic-pathological splenic rupture includes neoplastic (29.3%), infectious (28.3%), inflammatory (20%), iatrogenic (10.2%) and mechanical (7.2%.) etiology and rarely drug assocaited cases were described[1]. Mortality was associated with advanced age, the underlying neoplastic process, known history of splenomegaly, and especially with delay in diagnosis[2]. With the popularity of NOACs, as well as the evolving pharmacological strategies for bleeding control (fresh frozen plasma, activated prothrombin concentrates, recombinant activated Factor VII, idaricizumab), it is important to recognize this rare but increasingly well documented problem of atraumatic splenic rupture. Here we present a case that previously appeared relatively healthy, 53 years old, using an external heart device, with shock. We wanted to draw attention to intra abdominal bleeding suspicion with bedisde ultrasonography, rapid resuscitation with blood product transfusion and rapid surgery with treatment.

Conclusion

Spontaneous splenic hemorrhage should be bear in mind in a patient on apixaban treatment admitted with shock. Rapid diagnosis and aggressive early resuscitation resulted with full healing of our patient. Accurate diagnosis in a minimum of time was the major key in the management of this patient.

Declaration of competing interest

None

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Case Report

Journal of Emergency Medicine Case Reports

Concurrent Pulmonary Embolism and Acute Myocard Infarction: A Case Report

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Abstract

İntroduction: Pulmonary embolism and ST-elevation MI are both life threatening diseases. Although rare, concomitant pulmonary embolism and myocardial infarction pose even greater risk. Here we aimed to report a case of ST-elevation acute coronary syndrome developing one hour after diagnosis of pulmonary embolism. Case-Report: A 48-year-old male patient with known coronary artery disease had admitted for chest pain and dyspnea. He had got deep vein thrombosis in 10 days prior to this presentation. There wasn't ST elevation on his electrocardiogram. Pulmonary embolism was detected in the pulmonary CT angiography taken to the patient. The ECG was repeated after the patient, whose treatment was started, had new chest pain and sweating. The patient, whose ST elevation was detected in the inferior leads in the new ECG, was taking in to PCI. The patient, who had a stent in RCA, left the hospital voluntarily on the 3rd day of his admission to the coronary intensive care unit from where he was hospitalized with the diagnosis of ACS and PTE. In conclusion, it should be kept in mind that these two conditions may be present in patients presenting with complaints suggesting both AKS and PTE such as chest pain and shortness of breath.

Keywords: chest pain, electrocardiography, ST elevation, acute coranary syndrome, pulmonery thromboembolism

Introduction

Pulmonary Thromboembolism (PTE) is one of the most important clinical conditions of Venous Thromboembolism (VTE) and often develops on the background of underlying deep vein thrombosis (DVT)[1]. Since patients have complaints of chest pain and dyspnea during their arrival, Electrocardiography (ECG) is performed as soon as possible at the time of admission. ECG is evaluated in terms of whether there are findings for cardiovascular differential diagnoses. Although there is no specific ECG change for PTE, sinus tachycardia, right bundle branch block and S1Q3T3 pattern can be seen frequently[2]. The co occurrence of PTE and ST elevation ACS together is a rare situation in the literature[3].

In this case report, we aimed to draw attention to the subject by handling a case that presented to us with chest pain and dyspnea and was diagnosed with inferior myocardial infarction after PTE.

Case Report

A 48-year-old male patient applied to our emergency department with the complaints of sudden dyspnea and chest pain that started in the morning. At the time of admission, his general condition was moderate, his body surface was sweaty and had tachypnea. Vital findings: blood pressure arterial 110/70 mmHg, pulse 70 / min, fever 36.6° C, finger tip oxygen saturation in room air was 88%.There was no pathological finding in the lung sounds and heart sounds of the patient who has no jugular venous filling. History revealed that patient was hospitalized in another institution 10 days ago due to deep vein thrombosis (DVT) and he taken on low molecular weigh the heparin (LMWH). He was also diagnosed with coronary artery disease (CAD) 1 year ago. On this medical history, the patient's ECG was taken in order to exclude ACS and cardiac events, and no pathology was detected in the ECG except for T negativity in leads D1, AVL, V3,4,5,6 (figure 1). In his blood tests, complete blood count and emergency biochemistry tests were normal. Troponin test was evaluated as 14 pg / mL (normal). Since the patient had DVT, PTE was primarily considered in the etiology of the current presentation table and Pulmonary Computed Tomography angiography (Pulmonary CT Angio) was performed. The patient was diagnosed with PTE after pulmonary CT angiography, '' acute embolic filling defects were observed in all lung lobes on the right and lower lobes and pulmonary artery branches leading to the lower lobe segments on the left ''(Figure 2). The patient began to describe

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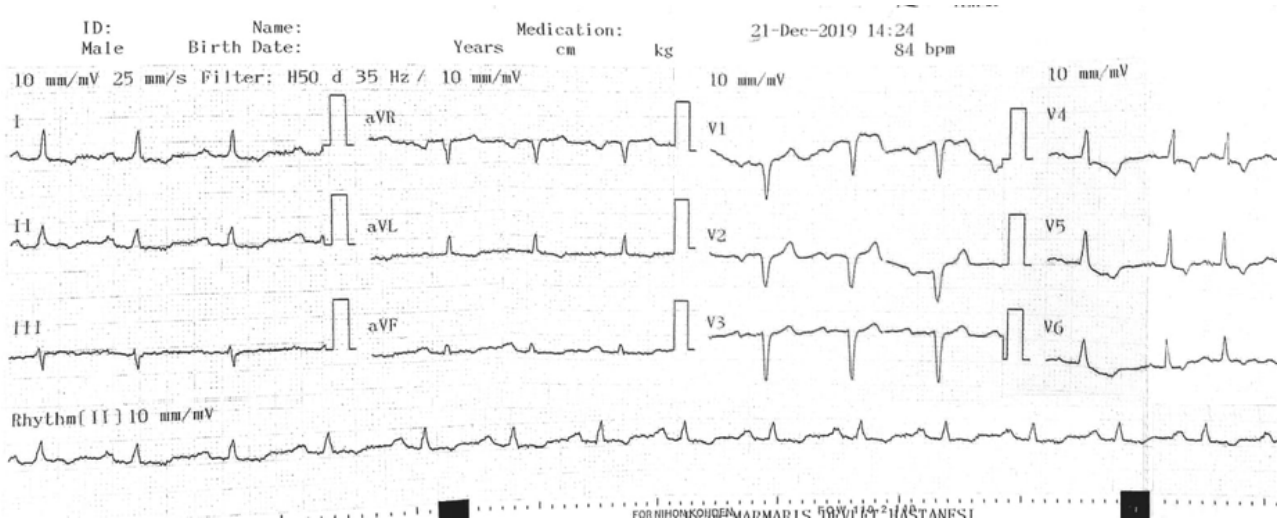

Figure 1. The first ECG of the patient

chest pain in the pressure style, which was started newly and spread to the left arm while the consultation procedures were going on in the emergency room. ECG was repeated after emerging chest pain occured (Figure 3).In the newly performed ECG, ST segment elevation in DII-III, AVF leads, ST segment depression and T negativity in DI, AVL, V3-6 leads were detected. Emergency cardiology consultation was performed with the diagnosis of ST elevation ACS. The control troponin test was found as 1559 pg / mL.The patient, whose emergency trans thoracic echocardiography (Echo) was planned by cardiology and chest pain and ACS with Echo findings, was taken into percutaneous coronary intervention (PCI) by cardiology. In addition, although a patent foramen ovale evaluation was performed in Echo, it could neither be clearly diagnosed nor said anything.Stent was placed in the patient with stenosis in the right coronary artery on angiography (Figure 4). After wards, the patient, who had 100% full openness, was hospitalized in the coronary intensive care unit.The patient, who was also examined by chest diseases in intensive care unit, was followed up with good condition on the 3rd day of hospitalization and left the hospital with his own consent.

Discussion

In our case, it was presented as an remarkable case since both PTE and ST elevated MI were seen together in the same time frame. It is very rare to beseen these two diseases together in the literature. Both of these diseases are serious diseases even in terms of mortality and morbidity, more over, the concurrency of both makes follow-up and treatment more difficult[4].

One of the most important parameters used in the differential diagnosis of patients presenting with chest pain is ECG. Infact, it is used in ACS diagnosis. However, there can

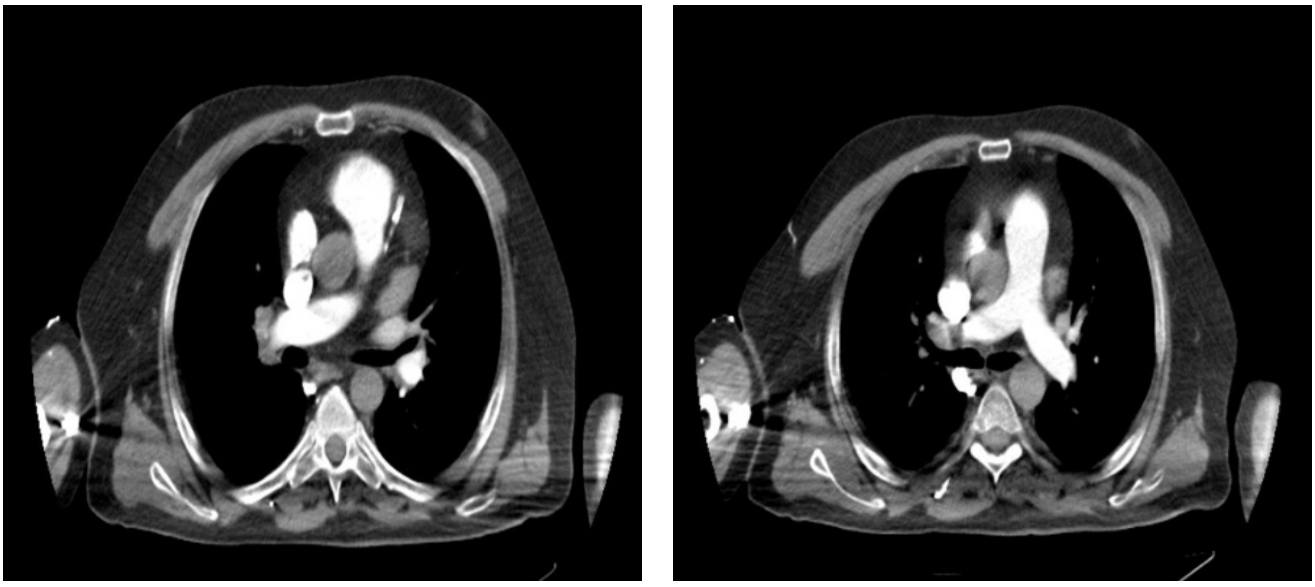

Figure 2. PTE compatible filling defects in the patient's pulmonary CT angiography.

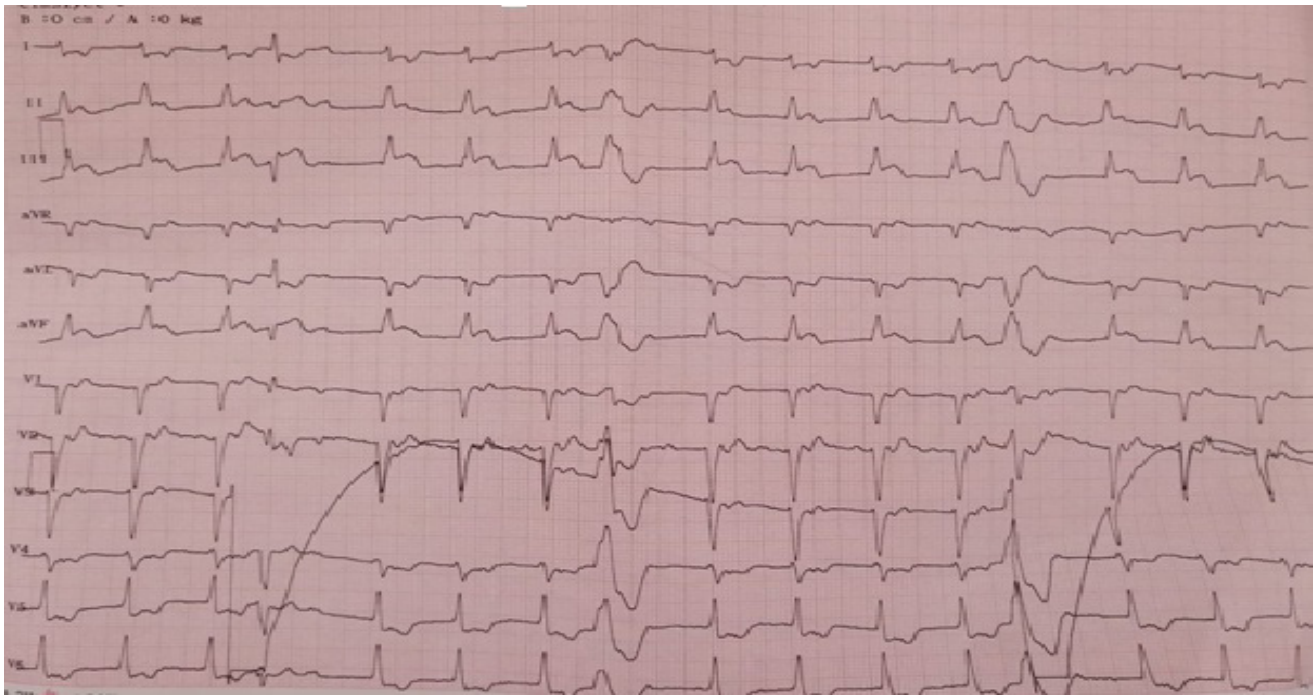

Figure 3. The 2nd ECG taken in our emergency department

alsobe various ECG changes in PTE.The most common of these are sinus tachycardia, right bundle branch block and S1Q3T3 pattern[2]. Depending on right ventricular tension and dilation, ST segment and T segment changes are reported in anterior leads in ECG[5,6]. With this change, we often encounter ST segment depression, and rarely, PTE cases accompanied by ST elevation have also been reported[7-11]. Therefore, in addition to ECG, history and physical examination are also important. Because similar ECG findings can be seen in ACS as well. ACS guidelines state that rapid differential diagnosis should be made in order to transfer cases with ECG with ST segment elevation to PCI without wasting time. However, if there is no ST elevation on the ECG, the patient's history, physical examination findings, ECG, troponin values and risk scores are evaluated together. Our patient also applied to the emergency department with the complaint of chest pain. Medical history had CAD. For this reason, in his ECG, T negativity was detected in the anterior leads. Although these ECG changes were seen in ACS, the patient's DVT history and troponin negativity led us to PTE rather than ACS and directed us to take a pulmonary CT angio. In pulmonary CT angio, images confirming the diagnosis of PTE were detected.

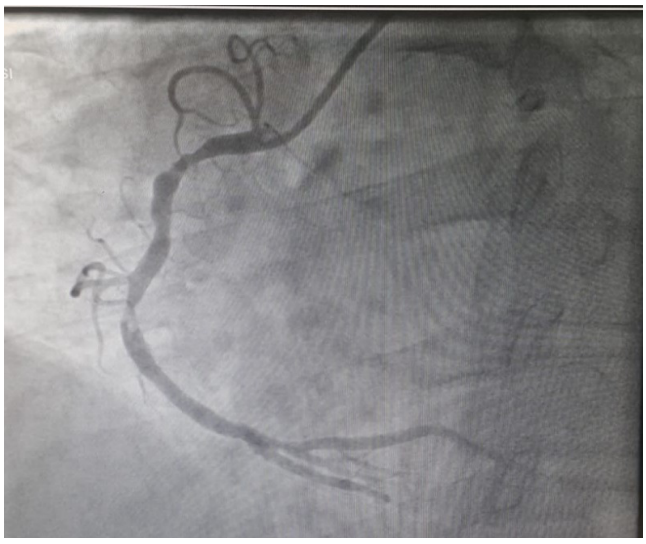

Figure 4. PCI image of the patient

What may be causing the concurrency of PTE and ST segment elevation ACS? When the literature is examined, it is seen that the underlying cause is right-to-left shunt for a reason such as patent foramen ovale, although not in all cases where this concurrency is reported[4,12,13]. In the review of the literature, in an article presenting a 35-year-old male patient, the researchers stated that the factor 5 leidan mutation caused PTE-ACS association[14]. Seguban et al.[3] reported that they detected ST elevation in the anterior leads of ECG in a 67-year-old patient with chest pain and dyspnea after an operation due to cancer. However, due to the patient's history and Echo findings, they stated that both pulmonary and coronary angiography were performed considering PTE and both conditions were detected. But the reason for this paradoxical situation in the case reports is not discussed. In a case where a 69-year-old male patient was presented, Wasek et al. stated that thei rpatients had been followed-up for hip fracture, but that he did not use DMAH regularly, and applied to their hospitals with complaints of chest pain and dyspnea.They explained that when they detected ST segment elevation in leads DII-III-AVF and V3-6 in their ECG, they received an emergency PCI and detected a large thrombus in RCA.They also expressed that they detected RV en-

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largement in the Echo performed for the patient whose dyspnea did not regress even two days after PCI and that they detected the embolism in pulmonary CT Angio for PTE. In this case, they pointed out that they detected patent foramen ovale as the cause of paradoxical thrombus[12].Similarly, in two different case reports, there are similar ST elevation MI and PTE concurrency.In these two case reports, it was stated that patent foramen ovale was detected in cases[4,13].In our case, the patent foramen ovale could not be excluded in the transthoracic Echo performed in the emergency department, but the patient had to be postponed to a later date since the patient was taken to the emergency angiogram. No data were found from the patient file regarding the examination of this condition while in hospital. However, in case presentations detected in foramen ovale, although embolism is observed in patients' coronary angiograms, the situation is different in our case because stenosis is in the foreground. For this reason, in our case, the absence of patent foramen ovale seems more reasonable. However, it seems difficult to make a definite judgment as the diagnosis cannot be clarified or the patent foramen oval is not ruled out.

In conclusion, it should be kept in mind that these two conditions may coexist in patients presenting with complaints such as chest pain and dyspnea, suggesting both ACS and PTE. Although the underlying cause is often a patent foramen ovale, this is uncertain. However, it should be remembered that PTE should also be considered in patients with DVT and PCI with coronary embolism.

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Case Report

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Unexpected Acute Aorta Dissection with Ischemic Stroke: A Case Report

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Abstract

Introduction: Both acute aorta dissection and ruptured aorta aneurism are prominent causes of death in cardiovascular diseases. The frequency of developing neurological complications in aortic dissections is reported to be between 2 to 8%. Stroke is more common in dissections involving the proximal aorta, but paraparesis is more common in distal aortic dissections due to circulatory impairment in the spinal arteries.

Case: The 70-year-old male patient was referred to our emergency service from an external center with diagnosis of acute ischemic stroke. The patient had experienced loss of strength on his left side and had syncope one hour before. After his physical examination, the patient received computerized brain tomography and diffusion MR imaging with the pre-diagnosis of acute stroke. For the patient who had an appearance of acute diffusion restriction in the right parietal region in the diffusion MRI and had a chance of thrombolytic treatment, thrombolytic treatment was planned. However, the general status of the patient was worsened in this checkup examination, and his GCS score regressed down to 7. The poor current condition of the patient could not be explained by the acute ischemic stroke in the right parietal region. Aorta dissection, which may progress with clinical signs of stroke, was considered for the patient, and as an advanced test, dynamic thorax CT angiography was taken. In the dynamic thorax CT angiography of the patient, aneurism in the ascending aorta, dissection and fluid around the pericardium and left lung (hemorrhage?) were observed. The echocardiography of the patient revealed that the fluid around the pericardium caused tamponade.

Discussion: Cardiovascular system diseases are the most common cause of natural sudden deaths and are mostly seen in middle and older ages. Acute myocardial infarction and coronary artery disease are the most common cardiovascular diseases, however, sudden deaths due to aortic dissection and rupture have been reported less frequently. The most typical symptom is the sudden start of severe chest or back pain. Patients typically visit with complaints of tearing chest and back pain, while they may visit with atypical clinical pictures wementioned in our cases such as abdominal pain, syncope, stroke. Sensory loss may also be seen in patients, and this is a neurological symptom which may extend from falling as leep to deep coma.

Conclusion: Patients who visit emergency services with symptoms that are not expected for aorta dissection such as syncope, altered consciousness, hypotension, atypical abdominal pain and loss of strength in the extremities.

Key Words: Syncope, ischemic stroke, aorta dissection

Introduction

Both acute aorta dissection and ruptured aorta aneurism are prominent causes of death in cardiovascular diseases. This situation, which threatens life, was recently classified as acute aortic syndrome. Acute aortic syndromes are defined as an emergency in the clinic and they may be listed as aorta dissection, intramural hematoma without intimal rupture, penetrant atherosclerotic ulcer and ruptured or almost ruptured aorta aneurism[1]. The frequency of developing neurological complications in aortic dissections is reported to be between 2 to 8%[2]. Stroke is more common in dissections involving the proximal aorta, but paraparesis is more common in distal aortic dissections due to circulatory impairment in the spinal arteries[2]. In our study, we found it worth presenting a case of acute aorta dissection in the ascending aorta that arrived without chest and/or back pain and atypically with left hemiplegia and syncope and caused ischemic stroke.

Case

The 70-year-old male patient was referred to our emergency service from an external center with diagnosis of acute ischemic stroke. The patient had experienced loss of strength on his left side and had syncope one hour before. He had a history of hypertension. In the examination of the patient at the external center, his general status was moderate, the Glasgow Coma Scale score was 10, and the left side of the patient was hemiplegic. After his physical examination, the patient

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received computerized brain tomography and diffusion MR imaging with the pre-diagnosis of acute stroke. For the patient who had an appearance of acute diffusion restriction in the right parietal region in the diffusion MRI (Figure-1) and had a chance of thrombolytic treatment, thrombolytic treatment was planned. However, the general status of the patient was worsened in this checkup examination, and his GCS score regressed down to 7. This blood pressure decreased down to 80/60mmHg. The patient was electively intubated and started with positive inotropic treatment. For the patient with a high score of NIH Stroke Scale, thrombolytic treatment was found not appropriate, and thus, not provided. The patient was brought to our emergency service in an intubated position by 112 (emergency services in Turkey). The vital parameters of the patient were as temperature: 36 °C, pulse: 120/min, BP: 80/50 mmHg and respiratory rate: 16/min. In the hemogram of the patient that was obtained at the time, his hemoglobin value was found as 10.4 g/dL, while his biochemical parameters were considered normal. In the ECG of the patient, sinus tachycardia and troponin values were negative. The poor current condition of the patient could not be explained by the acute ischemic stroke in the right parietal region. Aorta dissection, which may progress with clinical signs of stroke, was considered for the patient, and as an advanced test, dynamic thorax CT angiography was taken. In the dynamic thorax CT angiography of the patient, aneurism in the ascending aorta, dissection and fluid around the pericardium and left lung (hemorrhage?) were observed (Figure-2). The echocardiography of the patient revealed that the fluid around the pericardium caused tamponade. While cardiology and cardiovascular surgery consultation was being planned for the patient, the patient had cardiac arrest. Active cardiopulmonary resuscitation was applied for the patient for 45 min. The patient, whose heart movements did not come back for 45, was accepted as exitus.

Discussion

Cardiovascular system diseases are the most common cause of natural sudden deaths and are mostly seen in middle and older ages. Acute myocardial infarction and coronary artery disease are the most common cardiovascular diseases, however, sudden deaths due to aortic dissection and rupture have been reported less frequently[3].

Aorta dissection is accumulation of blood in the aorta wall with a rupture that occurs in the aorta intima as a result of high blood pressure and the structural anomalies of the aorta wall[4]. In diagnosis, aortography, magnetic resonance imaging, transthoracic or transesophageal echocardiography and dynamic computerized tomography are utilized. The diagnosis of aorta dissection is suspected if measurement of the aorta diameter in ECHO reveals valve anomalies, detection of intimal flap and real and pseudo lumens with the dissection membrane. There are two classifications that are used the most frequently for dissection. The DeBakey classification was divided in to three types (Type-1, 2 and 3) based on the starting point of dissection. The Stanford classification has two types (Types A and B) based on the involvement of the ascending aorta[5]. The most frequent risk factor for aortic dissection is uncontrolled hypertension (65-75% with history of hypertension). Other risk factors include age, male sex, smoking, previous aortic diseases or aortic valve disease, direct blunt trauma, family history, history of cardiac surgery and usage of intravenous drugs (cocaine or amphetamines)[1].

The most typical symptom is the sudden start of severe chest or back pain. The painmay be sharp, and in the form of tearing or stabbing feelings, and typically different from other reasons for chest pain. Patients typically visit with complaints of tearing chest and back pain, while they may visit with atypical clinical pictures wementioned in our cas-

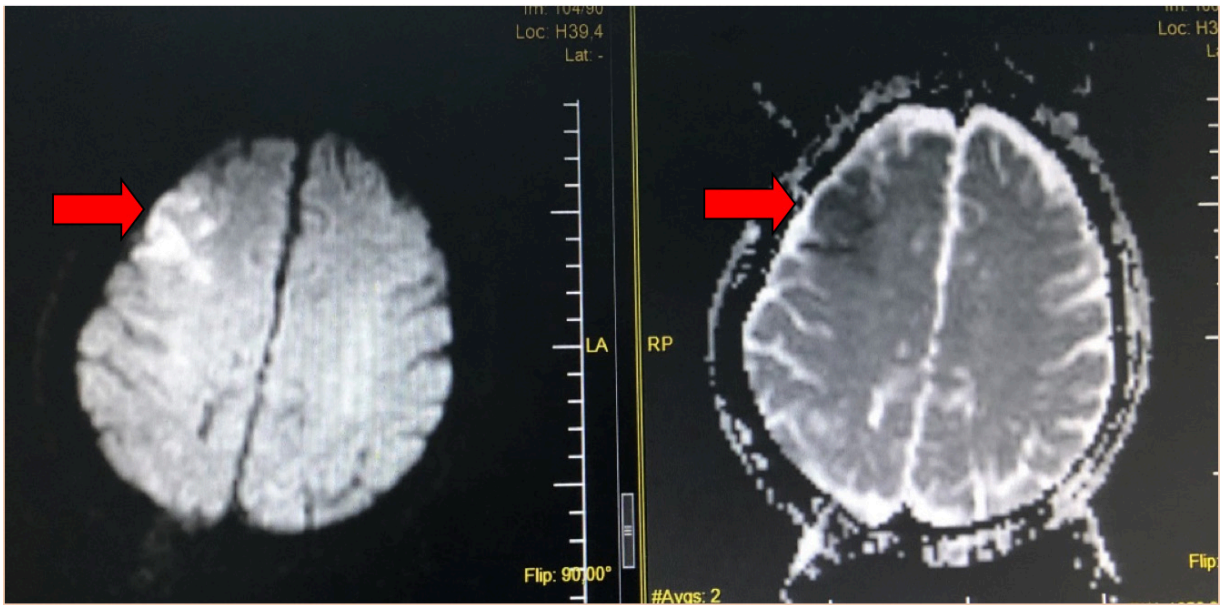

Figure 1: Acute diffusion restriction in the right parietal region in diffusion MR

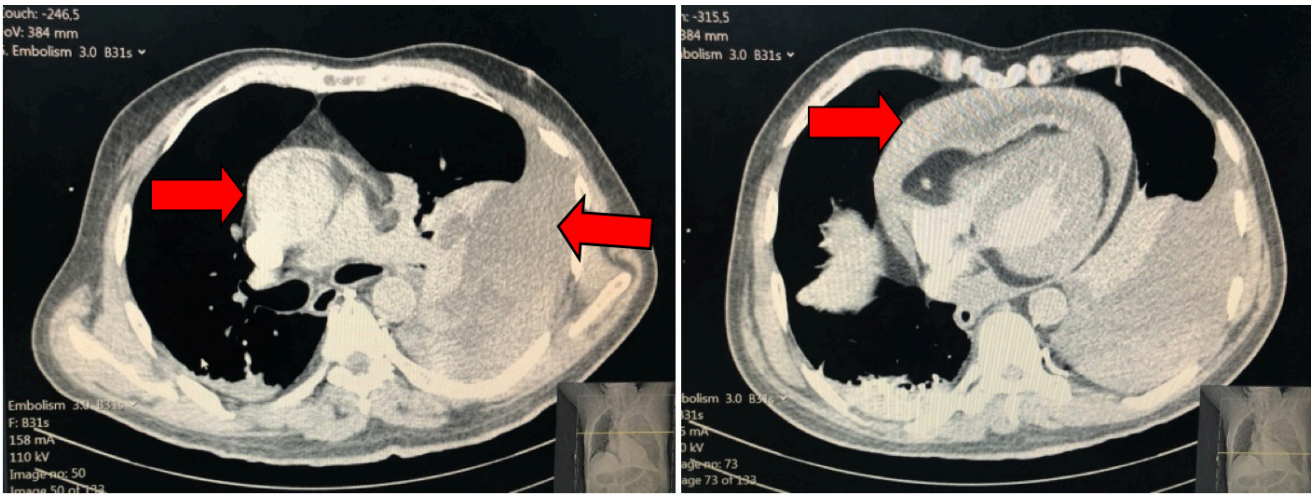

Figure-2: Aneurism in the ascending aorta, dissection, fluid around the pericardium and the left lung (hemorrhage?) in dynamic thorax CT angiography

es such as abdominal pain, syncope, stroke. Sensory loss may also be seen in patients, and this is a neurological symptom which may extend from falling as leep to deep coma. In studies, neurological complications have been reported as 17% in Stanford type A and 5% in Stanford type B[5].

In a review where Suzuki et al. examined type B aorta dissection cases, among the 384 cases, there was spinal cord ischemia in 10 (3%) and ischemic peripheral neuropathy in 8 (2%). It was reported that one patient died in each group[6]. In a review where Stewart Collins et al. examined type A aorta dissection cases, among the 617 cases, there was a neurological complication in 171 (29%). While there was a history of cardiac surgery in 23 of these cases, there was none in 148[7]. Stroke especially arises in proximal aorta lesions, while paraplegia emerges with involvement of spinal arteries in cases with distal lesions (by 2-8%). Cerebral infarction may be seen in cases of aorta dissection by 5-10%[8]. The neurological state is based on the extent of reduction in blood flow due to brain cerebral circulatory disruption, hypotension or distal thromboembolism. Other than these, a different picture like mesenteric ischemia is seen in 5% of both Type A and Type B aortic dissection patients[9]. As it is seen here, Aortic dissection may appear with highly variable clinical pictures. In agreement with the literature, there was also dissection in the proximal aorta (DeBakey type-2, Stanford type-A) in our patient, which led to stroke.

In our case, the patient did not have chest and/or back pain. The arrival of the patient with left hemiplegia and syncope led us to acute ischemic stroke as a pre-diagnosis. However, when the poor clinical status of the patient could not be explained by acute ischemic stroke, we considered aorta dissection by a clinical picture of stroke as the pre-diagnosis. As seen here, we may encounter aorta dissection with several different clinical issues that are impossible to consider at first.

Conclusion

Keeping dissection in mind is the most important factor in the diagnosis of acute aorta dissection. The clinical signs of our cases at the time of admission did not include the typical clinical signs of aorta dissection. The patient did not have chest and/or back pain. This is why appropriate examinations should be carried out by keeping the diagnosis of aorta dissection in mind in patients who visit emergency services with symptoms that are not expected for aorta dissection such as syncope, altered consciousness, hypotension, atypical abdominal pain and loss of strength in the extremities.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Cause of Acute Abdomen in Emergency Room: Isolated Superior Mesenteric Artery Dissection

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Abstract

Introduction: Isolated superior mesenteric artery dissection (ISMAD) has severe and sudden-onset abdominal pain with little physical examination findings. Misdiagnosis of ISMAD is seen commonly in clinical practice, which may lead to severe intestinal ischemic necrosis or even death. Here, we present a patient with abdominal pain and diagnosed as ISMAD.

Case Report: A 46-year-old male was admitted to the emergency department with sudden onset abdominal pain and constipation. He had hypertension and asthma in his history. There was epigastric tenderness in his physical examination. Laboratory tests revealed no pathological findings except high white blood cell count. His complaints did not alleviate despite medical treatment and contrast enhanced abdominal computed tomography was performed. The patient was diagnosed as ISMAD and underwent emergency surgery. The patient developed widespread bowel ischemic necrosis and exitus occurred.

Conclusion: Emergency physicians should be aware of ISMAD and include this in differential diagnoses, especially in patients with acute onset of abdominal pain.

Key Words: Acute abdomen, computerized tomography, isolated superior mesenteric artery dissection

Introduction

Arterial dissection is a life-threatening condition that can be seen in any artery. Isolated superior mesenteric artery dissection (ISMAD) is a dissecting lesion of the superior mesenteric artery (SMA) and its branches. Compared to other arteries, such as the splenic, hepatic, celiac and gastric arteries, superior mesenteric artery dissection is the most common visceral artery dissection[1]. SMA dissection is usually associated with aortic dissection. With the more common use of multidetector computed tomography, isolated superior mesenteric artery dissections which do not coexist with aortic dissection are reported more frequently today[2,3].

Isolated superior mesenteric artery dissection has very few clinical findings except severe and sudden onset abdominal pain. The ISMAD clinic may vary from unclear symptoms to fatal SMA rupture[2]. Delays or misdiagnosis of ISMAD can cause severe bowel necrosis and sometimes death[4].

In this case report , we present a patient with abdominal pain and diagnosed as isolated SMA dissection.

Case Report

A 46-year-old male was admitted to the emergency department with sudden onset abdominal pain. In his anamnesis he stated that the pain suddenly started in the evening, he had constipation and the pain decreased after enema was administered in another hospital. When the pain increased again later, he applied to our emergency department. He had hypertension and asthma in his history. His physical examination was normal except epigastric tenderness. Vital findings were: blood pressure 180/100 mmHg; heart rate 78/min; fever 36.5 °C; SPO_2 95%; respiration rate 16/min. No pathological findings were observed in the ECG. Laboratory tests revealed no pathological findings other than high white blood cell counts (16.140 U/L). His complaints did not alleviate despite medical treatment and contrast enhanced abdominal computed tomography (CT) was performed. CT showed a double lumen appearance of the dissection starting 3.5 cm after the superior mesenteric artery origin and continuing through the 4 cm segment and no luminal filling after dissection (Figure 1, 2). The patient was diagnosed as

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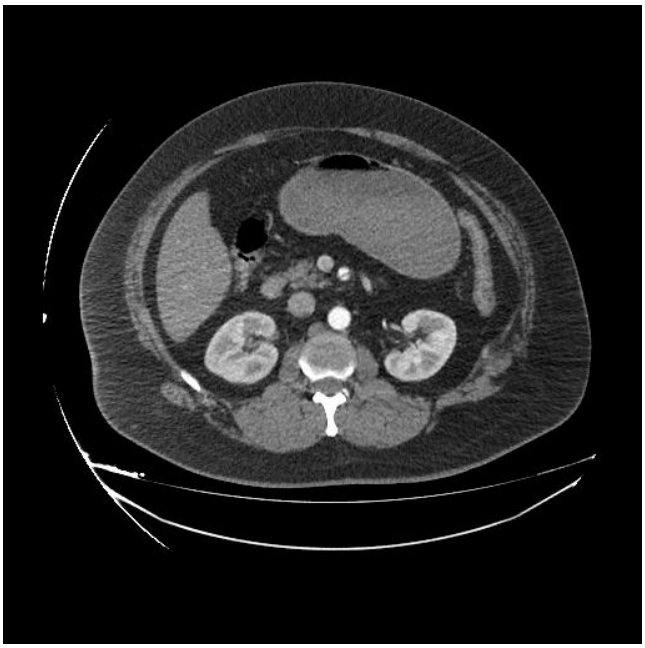

Figure 1: The dissection flap of superior mesenteric artery is seen.

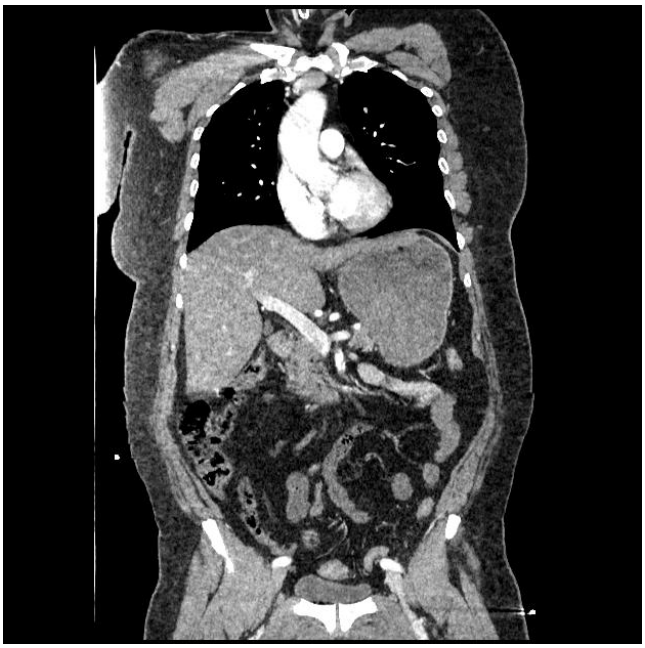

Figure 2: Dissection flap of superior mesenteric artery and disturbed blood flow in the distal part.

ISMAD and admitted to the general surgery clinic. The patient underwent emergency surgery due to acute abdomen findings during follow-up. Aneurysmatic dilatation was observed at the root of SMA in exploration. The blood supply of right colon and small intestine were disrupted and necrotic areas were observed. A bypass was performed between the SMA and iliac artery. It was observed that the blood supply was returned to 140 cm area from the ligament of Treitz in intestinal loop. Right hemicolectomy and end-lateral jejunotransvers anastomosis was performed with intestinal resection. Gastrointestinal contents from the abdominal drain were observed during the clinical follow-up and the patient was reoperated with suspicion of anastomotic leakage and the remaining bowel loops were found to be necrotic. The patient developed widespread bowel ischemic necrosis and exitus occurred on the postoperative 2nd day.

Discussion

Sudden onset abdominal pain is one of the major causes of emergency admissions even though vascular abdominal pain constitutes only a small part of these cases[5]. Acute mesenteric ischemia (AMI) accounts for 1–2% of acute abdominal emergencies. Similar to clinical findings in other types of acute mesenteric ischemia, SMA dissection causes severe and sudden onset abdominal pain with very few physical examination findings[6]. Spontaneous ISMAD represents 8% of all visceral artery dissections and has an overall prevalence of 0.09% in cadaveric studies[7]. On the other hand increased use of CT revealed that ISMAD was more frequent than previously thought.

The risk factors that cause ISMAD are not fully known. Dou et al. reported that atherosclerosis, smoking and hypertension are the most common risk factors. Connective tissue diseases and genetic susceptibility are also contributing factors to SMA dissection. Strong mechanical stress in the SMA wall is the main factor for SMA dissection[6]. However, no specific underlying cause of SMA dissection has been identified in the majority of reports[8]. Kimura et al reported that isolated SMA dissection was more common in men and hypertension was the most important comorbidity[9]. Similarly our patient was male and had hypertension.

Isolated SMA dissection should be suspected in patients with severe abdominal pain and atherosclerosis. The size of the aneurysm and remaining lumen, the number of collaterals determine the clinical presentation[7]. The main complaint of the patients is sudden onset severe pain. Acute epigastric pain, nausea, vomiting, melena, and abdominal distention are other common clinical findings[5]. Occasionally, patients may be asymptomatic. Rupture of the artery may rarely cause hemorrhagic shock in patients[8]. Our patient presented with acute onset epigastric pain, abdominal distention and constipation.

Direct radiographs or blood tests have no role in the diagnosis of SMA dissection[1]. In this case laboratory findings of the patient were normal except white blood cell count. Symptoms didn't relieve with medical treatment so a contrast enhanced CT angiography was performed in suspect of gastric perforation. CT showed isolated dissection of the SMA. CT angiography can show false lumens and intimal flaps and is helpful in diagnosis. The size of the lesion and bowel infarction can also be detected by CT angiography[6]. Today catheter angiography remains the gold standard diag-

nostic test for ISMAD. On the other hand, this test should be performed only in patients who require surgical or endovascular treatment and whose condition worsens[9].

The treatment options for ISMAD include expectant management, anticoagulation, open surgery, and endovascular intervention; however, the optimal initial treatment remains controversial[3]. It was reported that conservative treatment in ISMAD without arterial rupture or bowel necrosis should be first line therapy. Conservative management includes anti-platelet therapy, optimizing blood pressure control, anticoagulation, and bowel rest[10]. Since ISMAD causes low morbidity and mortality medical treatment is advocated. However, results of medical treatment have been reported in series with small number of patients and small dissection sizes[7].

Emergency revascularization should be applied to patients who are hemodynamically unstable and have radiological progression findings such as formation of thrombus, narrowing or saccular aneurysm formation. Our patient had signs of bowel ischemia so he underwent urgent revascularization and died from total ischemic necrosis. Since SMA dissection is a rare clinical entity, its management, diagnostic methods and outcomes have not been determined in detail yet[1]. Clinical experiences and outcomes related to ISMAD management mainly consist of case series in the literature.

Conclusion

Isolated SMA dissection is rare and important differential diagnosis of acute abdomen which may result in a life threatening condition such as mesenteric ischemia or aneurysmal SMA rupture. Emergency physicians should be aware of ISMAD and include this in differential diagnoses, especially in patients with acute onset of abdominal pain. Timely and accurate diagnosis is lifesaving.

Conflict of interest: The author have no conficts of interest to declare.

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Case Report

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Energy Drinks Induced Atrial Fibrillation

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Abstract

Introduction: Atrial fibrillation (AF) is most common in adulthood supraventricular arrhythmia. Some of our ways of life the occurrence of this arrhythmia possible. Recently, it is common to consume energy drinks (ED), especially among young people. Caffeine is found in energy drinks. Their adverse effects, not yet completely known, are usually considered secondary to a high concentration of caffeine. Here we focus on a particular acute cardiac complication of EDs overconsumption: atrial fibrillation.

Case report: A 23-year-old man, was referred to our emergency department for dyspnea, palpitations, and anxiety. The patient had a negative family, personal, and pharmacological history. About two hours before, he has consumed 750ml of ED. At admission, blood pressure was 104/62. An ECG revealed a high rate of atrial fibrillation at 162bpm. Laboratory investigation revealed that complete blood count and routine biochemical blood levels were within normal limits..Transthoracic echocardiogram excluded the presence of cardiac disease, the left atrium was slightly dilated, as expected. According to guidelines patient was treated with propafenone and recovered a normal sinus rhythm.

Conclusion: As in our case, PAF can occur due to ED consumption, and awareness about this adverse effect of ED consumption is important.

Keywords: Arrythmia, atrial fibrillation, energy drinks

Introduction

Atrial fibrillation (AF) is most common in adulthood supraventricular arrhythmia. Some of ourways of life theoccurrence of thi sarrhythmia possible. Completely prevent AF formation from lifestyle changes not expected. However, it is possible to reduce the likelihood of AF or the frequency of attacks with some precautions. Recently, it is common to consume energy drinks (ED) especially among young people. Caffeine is found in energy drinks.Their adverse effects, not yet completely known, are usually considered secondary to an high concentration of caffeine. Here we focus on a particular acute cardiac complication of EDs overconsumption:atrial fibrillation.

Case Report

A 23-year-old man, was referred to ou remergency department for dispnea, palpitations and anxiety. The patient had a negative family, personal, and pharmacological history. About two hours before, he has consumed 750ml of ED. At admission, blood pressure was 104/62. An ECG revealed a high rate atrialfibrillation at 162bpm (Figure-1). No other physical signs were present. Laboratory investigation revealed that complete blood count and routine biochemical blood levels were with in normal limits. (Thyroid, renal, hepatic functions were normal; the hypoxia, metabolic acidosis, electrolytes imbalance,troponin increase were excluded; the toxicological results were negative.) Transthoracic echocardiogram excluded presence of cardiac disease, the left atrium was slightly dilated, as expected. According to guidelines[1] patient was treated with propafenone and recovered a normal sinus rhythm (Figure-2). The patient was monitored for 6 hours, followed by sinus rhythm.

Discussion

Energy drinks contain stimulants and other substances, including large amounts of caffeine, taurine, ginseng, guarana, theophylline, sugars, vitamins, and herbs. Acute high doses have little effect on the occurrence of supraventricular arrhythmias[2]. Recent ingestion of a large amount of EDs, paroxys-

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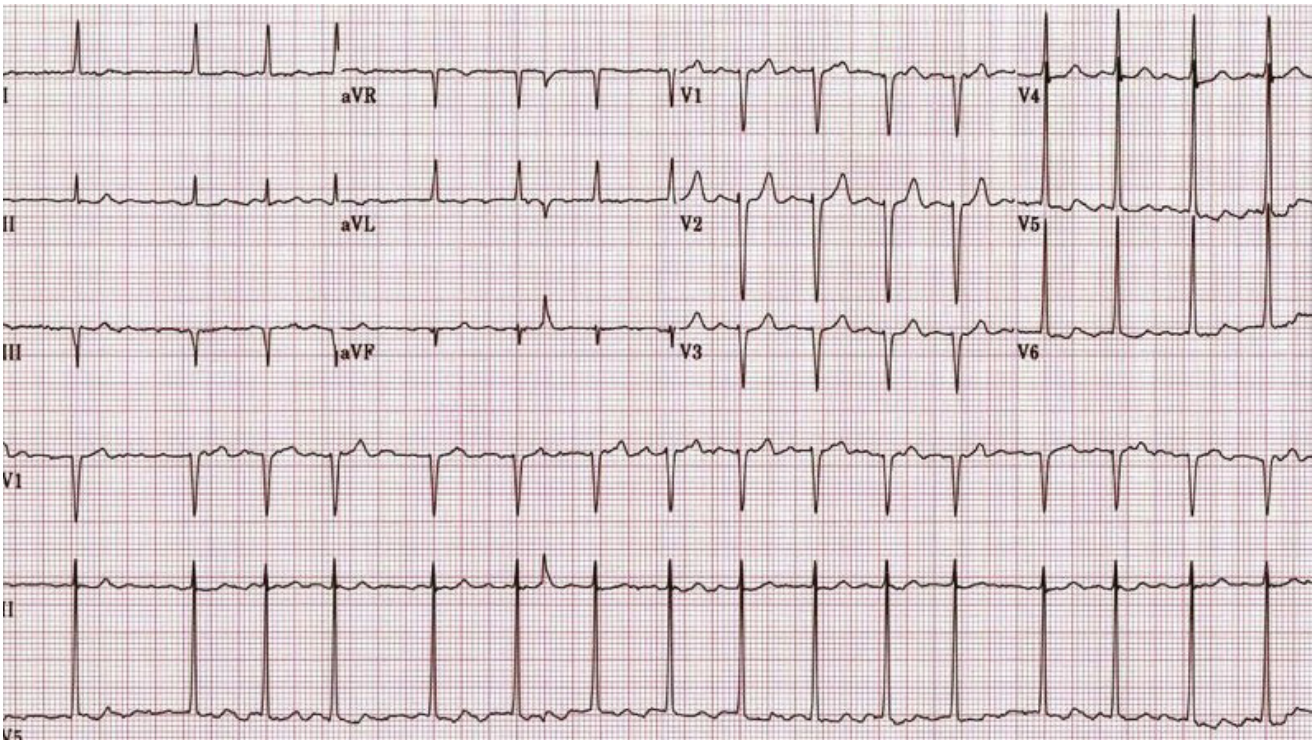

Figure 1: Atrial fibrillation after consumed energ drinks

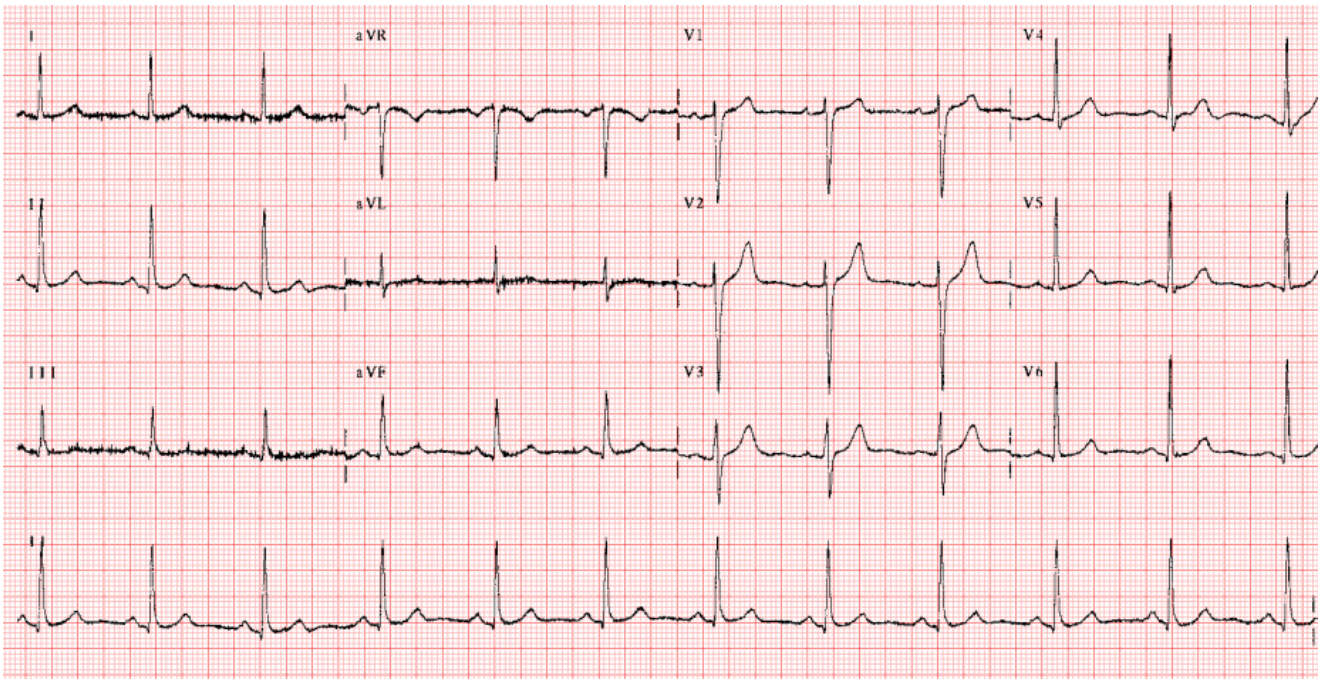

Figure 2: Treated with propafenone and recovered a normal sinus rhythm

mal AF at a high rate without underlying cardiac disease, and absence of laboratory abnormalities, indicating concomitant dysfunctions of other organs. Caffeine effects on cardiovascular system are well known and we want to exclude this trigger for atriall fibrillation[3]. A direct pathogenetic link between abuse of EDs and acute onset of high-rate AF is supported by the absence of recurrence of this complication after a complete abstinence from this beverage[4].The mechanism of this phenomena is unclear, as previous studies involving high doses of caffeine have shown no increased risk of AF[5]. Caffeine is a methylxanthine compound that primarily causes neurohormonal stimulation and activation of the sympathetic nervous system by inhibitin gphosphodiesterase and adenosine A_1, A_{2A}, A_3, and A_{2B} receptors and increases calcium concentration in thecytoplasm by blocking the reuptake of calcium in the sarcoplasmic reticulum[6]. Acute caffeine consumption was reported to increase plasma renin and norepinephrine and epinephrine levels, increasing blood pressure and heart rate[7].

It is also possible that the other ingredients and herbs in these energy drinks could potentially trigger an arrhythmia.There is no demonstration that caffeine alone increases the risk of AF[8], on thecontrary, few information is available on the risk of AF related to other substances (i.e. taurine, maltodextrin, inositol, carnitine, creatine,guarana, ginseng, and ginkgobiloba) included in EDs. On the other hand it is unknown whether these individuals had a genetic susceptibility that predisposed to the arrhythmia.

Conclusion

As in our case, PAF can occur due to ED consumption, and awareness about this adverse effect of ED consumption is important.

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