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Contents

Case Report

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Case Report; Primary Aortoenteric Fistula Due to Plum Kernel

Hilal Hocagil[1], Abdullah Cüneyt Hocagil[1]

[1]Department of Emergency, School of Medicine, Bülent Ecevit University. Zonguldak, Turkey.

Abstract

Aortoenteric fistulas are defined as an abnormal connection between the aorta and any portion of the gastrointestinal tract. It can occur after aortic repair surgery or spontaneously (primary). The common clinical features of primary aortoenteric fistula are upper gastrointestinal bleeding, abdominal pain, and pulsatile abdominal mass. The reasons are inflammatory destruction of an aortic aneurysm, infection, tumors and ingestion of foreign bodies. We represent a case of a primary aortoenteric fistula due to plum kernel which is rarely seen. A 76-year-old man admitted to emergency department with abdominal pain. On computed tomography angiography, there was hypodense restricted lesion with widespread air image which was located around the abdominal aorta. It was determined that abdominal aorta is surrounded by organised hematoma. A plum kernel, was detected in the operating room, was the reason for the aortoenteric fistula. Graft was placed in the abdominal aorta. He died in the first day after surgery. Primary aortoenteric fistulas should be considered in differential diagnosis of abdominal pain, upper gastrointestinal bleeding and shock in patient with aortic aneurysm. Kernels of any fruit considered to be harmless just as plum kernel and should be noted that may cause aortoenteric fistula.

Keywords: Primary aortoenteric fistula, gastrointestinal bleeding, abdominal pain, plum kernel

Introduction

Aortoenteric fistulas (AEFs), are defined as an abnormal connection between the aorta and any portion of the gastrointestinal tract, are rare and life-threatening condition (1). The disease is classified into two types as primary and secondary. Primary AEFs occur spontaneous native aorta and secondary AEFs occur after vascular repair surgery. Clinical signs of AEFs consist of gastrointestinal bleeding, abdominal, back or flank pain, mild epigastric tenderness, pulsatile abdominal mass, hemorrhage shock and sepsis (2).

We represent a case of a primary aortoenteric fistula due to plum kernel which is rarely seen. The aim of this case report is to remind the AES, is a rare cause of gastrointestinal bleeding which is often seen in emergency department. Secondly, recalling that the fruit seed, is frequently passes through the intestine spontaneously, can cause a serious complication as aortoenteric fistula.

Case Report

A 76-year-old man admitted to the emergency department with nausea, vomiting, abdominal and back pain which had been continued for 15 days. Past medical history of the patient was included chronic obstructive pulmonary disease, hypertension, diabetes mellitus and bladder stones. Vital signs in the emergency department were notable for a temperature: 36 pulse: 110 beats/min and blood pressure: 80/50 mm Hg**.** The patient's general condition was poor**.** Physical examination revealed widespread tenderness in the abdomen. Other examinations of the patient were unremarkable and the patient was evaluated for the etiology of abdominal pain. His laboratory results were as white blood cell count 22.000 per microliter, pH: 7.30 and lactate:14.5. The computed tomography angiography of the patient was evaluated by the radiologists as follows: There was hypodense restricted lesion with diffuse air image(abscess?) which was located around the abdominal aorta. This area, was measured as 26 mm as thickest, was surrounding the aorta in approximately 9 cm segment which was extending from the infrarenal level to the iliac artery bifurcation. Aneurysmatic dilatation was observed in proximal of the main iliac artery bifurcation, and diameter of abdominal aorta was measured as 4cm in approximately 3cm segment. Aneurysm associated extravasation was not observed. (Figure1-2)

Corresponding Author: Hilal Hocagil **e-mail:** drhocagil@gmail.com
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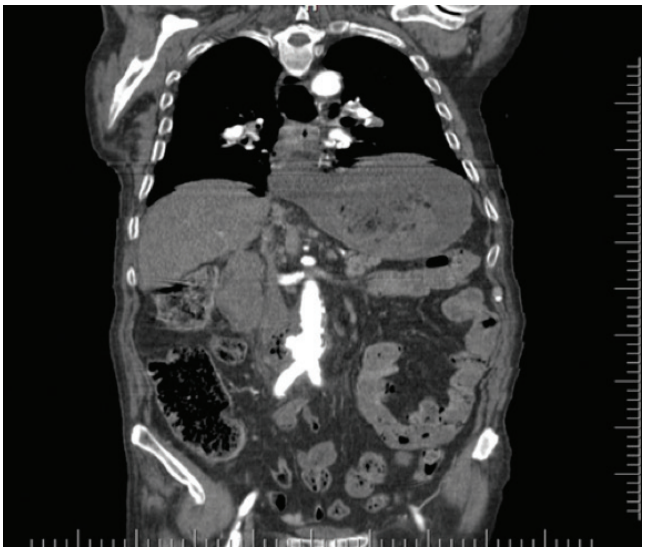

Figure 1. Coronal section from abdominal tomography image of patient.

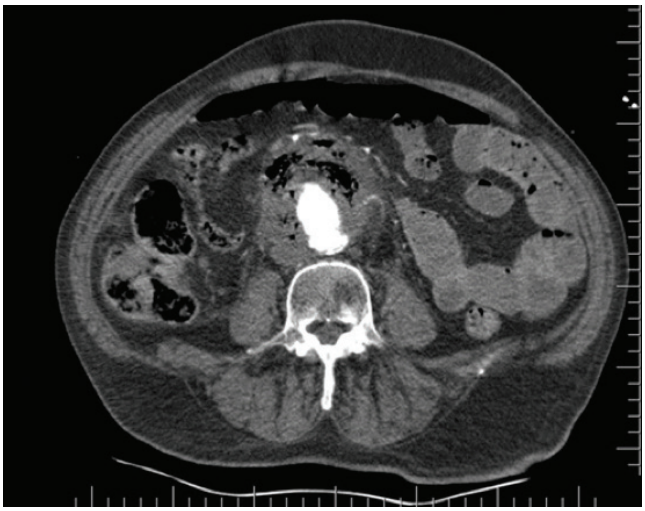

Figure 2. Axial section from abdominal tomography image of patient.

Hemoglobin level was 8.5g/L in the laboratory result. Hematemesis was seen in the nasogastric catheter and endoscopy was performed. Upper gastrointestinal endoscopy showed coagulated blood in the gastric lumen, but bleeding focus and fresh blood was not detected. In the pylorus, pyloric duct, bulbus and second part of the duodenum, a mass protruding lesion (external mass invasion? Vascular penetration?) was observed clot in the lumen, which is 3-4 cm distal above the bulb.

Operation was performed with the coordination of both cardiovascular surgery and surgery with suspicion of intraabdominal abscess, hematoma and aneurysm. It was determined that abdominal aorta is surrounded by organised hematoma and a plum kernel located posterior to the duodenum, was detected in the operating room. The plum kernel protruding from the duodenum to the aorta is thought to cause fistula. Abondane bleeding started after outbreak of plum kernel during surgery. Bleeding was stopped and infected and necrotic tissue was debrided. Aortic aneurysm was replaced with prosthetic graft and the duodenal fistula was closed. Our patient died in the first day after surgery.

Discussion: Aortoenteric fistulas are more commonly seen after aortic graft surgery and the incidence of those varies between in 0.19% to 2%. The aortic fistulas that develop after aortic surgery are called as secondary aortoenteric fistulas. Primary aortoenteric fistulas which are spontaneously occur, are rare than secondary aortoenteric fistulas. In an autopsy study which performed on patients who died of massive gastrointestinal bleeding, primary aortoenteric fistulas detected in 0.04% to 0.07% of patients (3). Men, are older than 60 years, are at greater risk for fistula. More than 75% of all AEFs are located in the duodenum, especially the third portion of the duodenum (4). In 83% of primary aortoenteric fistulas are accompanied with abdominal aortic aneurysm. Dynamic pressure between an expanding anaurysm and a relatively fixed portion of bowel such as the third portion of the duedonum and repetitive pulsation of the aneurysm are thought to cause fistula formation by creating mechanical stress (5). Some rare causes include radiation, infection, tumors, peptic ulcers, Crohn's disease, septicaortitis due to salmonella infection, diverticular abscess and ingestion of foreign bodies which are the reason in 1% of primary aortoenteric fistula (6). In adults, foreign body ingestion is frequently associated with food and in 95% of the cases are accidental. While in 80-90% of foreign bodies are excreted spontaneously from the intestines, only in 10-20% of them require endoscopic intervention and less than in 1% of them require surgical intervention (7). The etiology of PAEF was plum kernel which was seen in our patient.

The most common clinical feature of PAEF is upper gastrointestinal bleeding (64%). Bleeding episodes range from a herald minor hemorrhage to life-threatening massive bleeding. Other symptoms are abdominal pain (32%), and a pulsatile abdominal mass (25%). However, the classical triad of symptoms known as Cooper's triad, includes abdominal, back or flank pain; gastrointestinal hemorrhage; and a pulsating abdominal mass, is concomitantly presented only in 11% of the patients (2,3). Our patient was presented with abdominal-back pain and shock in the emergency department. AEF may not have specific findings in the early period of the disease just as seen in our patient and this may delay the diagnosis. Approximately one thirds of patients may admit to a hospital with hypovolemia, hypotension and septic shock. Surgical mortality is approximately in 21% of the patients who were presented with shock (8).

The diagnosis of primary aortoenteric fistula is difficult and often delayed because of its rarity. Computed tomography angiography, which is a noninvasive method, is often the first option for diagnosis in emergent cases with sensitivity 94% and specificity 85% (9). The specific

CT findings of AEF include ectopic gas within the aorta, focal bowel wall thickening, and extravasation of contrast material into the bowel lumen.

Upper endoscopy is a good diagnostic method to rule out other causes of upper gastrointestinal bleeding such as ulcers and varices. Endoscopy with a water-soluble contrast medium is helpful in documenting the presence of an AEF only when there is leakage of oral contrast material from the disrupted bowel wall into the periaortic space. But this method should be performed only on a hemodynamically stable patient and a negative endoscopy does not rule out the possibility of an AEF. On the side endoscopy has the potential risk of inducing massive hemorrhage by dislodging fresh thrombus in the AEF (2).

Two thirds of patients are diagnosed during surgery. Even laparotomy may be negative in one-half of cases. Unfortunately, as many as 50 percent of cases are diagnosed postmortem. Our patient was diagnosed during surgical intervention (4).

The foundation of treatment for primary aortoenteric fistula consist of accurate and timely diagnosis, initial resuscitation and hemodynamic support, antibiotic therapy, surgical resection and debridement, and arterial and enteric reconstruction. The surgeon should debride the infected and necrotic tissue, correct the bleeding source, and restore arterial and intestinal continuity (6).

Traditional open repair of aortoenteric fistula associated with high morbidity and mortality. Endovascular repair may be an alternative for patients with hemodynamic instability. It has been suggested as a less invasive method to temporarily or definitively manage the AEF (10).

Conclusion: Primary aortoenteric fistulas should be considered in differential diagnosis of abdominal pain, upper gastrointestinal bleeding and shock in patient with aortic aneurysm and swallowed a hard body until proven otherwise. Kernels of any fruit considered to be harmless just as plum kernel should be noted that may cause aortoenteric fistula.

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

Conflict of interest: *The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.*

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Case Report

Journal of Emergency Medicine Case Reports

Recurrent Temporomandibular Joint Dislocation Due to Antiemetic Induced Acute Dystonia: A Case Report

Nafis Vural[1], Emine Vural[2]

[1] Ereğli State Hospital, Department of Emergency Medicine, Konya, Turkey.

[2] Akdeniz University, School of Medicine, Department of Family Medicine, Antalya, Turkey.

Abstract

Drug-induced acute dystonic reaction is a common presentation to emergency department. Oromandibular dystonia is one of the focal dystonias, which can be presented as jaw clenching, jaw opening or jaw deviation and leads to impaired speech and swallowing. In this paper, we presented an adult patient with recurrent temporomandibular joint dislocation due to metoclopramide use. A 21-year-old female patient came to the emergency department with the complaints of inability chew and swallow, difficulty in speaking, pain at right temporomandibular region that started a few hours ago. On physical examination, she was having dystonia of the right mandibular region and left posterior servikal region and no dystonia at other parts of the body. The patient was diagnosed with metoclopramide-induced acute dystonia. She was treated with intravenous anticholinergic. Metoclopramide is an antiemetic drug that can cause serious adverse events such as acute dystonic reaction. Among these side effects are oromandibular dystonias, which may lead to TMJ dislocation. Physicians and other healthcare professionals working in the emergency department should be familiar with such side effects.

Keywords: Temporomandibular joint dislocation, dystonia, metoclopramide, emergency department

Introduction

Dystonia is a movement disorder that is characterised by intermittent or prolonged muscle contractions that result in abnormal, often repetitive movement patterns in all or parts of the body(1). It may be focal, multifocal, or generalized and may also be primary or secondary based on their etiology. It may manifest as oculogyric crisis, deviation of eyes in all directions, protrusion of tongue, trismus, lock jaw, torticolis, laryngeal spasm, difficulty in speaking, facial grimacing, opisthotonus, lordosis or scoliosis and tortipelvic crisis(2). Dystonia induced by drug treatment is an example of a secondary form of the disorder(3). Drug-induced acute dystonic reaction is a common presentation to emergency department. Drug-induced dystonia are secondary dystonias which occur commonly with drugs with antidopaminergic effects such as antipsychotics and metoclopramide(4) which we call tardive dystonia, is to be distinguished from acute dystonic reactions, which are transient, and from classic tardive dyskinesia, which is a choreic disorder that predominantly affects the oral region. We present 42 patients with tardive dystonia. The age of onset of dystonia was 13 to 60 years. Symptoms began after 3 days to 11 years of antipsychotic therapy. Younger patients tended to have more generalized dystonia. In a few patients, spontaneous remission occurred, but dystonia persisted for years in most. Therapy was rarely a complete success. The most frequently helpful medications were tetrabenazine (68% of patients improved.

Metoclopramide, a dopamine-2 receptor antagonist used for various gastrointestinal disorders, may cause or exacerbate a variety of extrapyramidal movement disorders(5). The anti-emetic action of metoclopramide is the result of its antagonist activity at D2 receptors in the chemoreceptor trigger zone in the central nervous system; this action prevents the nausea and vomiting triggered by most stimuli(6). Extrapyramidal side effects are estimated to be 0.2 %. Acute dystonic side effects usually occur 1–2 days after first taking the drug(7).

Oromandibular dystonia is one of the focal dystonias, which can be presented as jaw clenching, jaw opening or jaw deviation and leads to impaired speech and swallowing(8). Sometimes oromandibular dystonia is so severe that it can cause temporomandibular joint dislocation. Thus far, few

Corresponding Author: Nafis Vural
e-mail: 42nafisvural@gmail.com
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case reports have been published about temporomandibular joint dislocation due to antiemetic medications(9). Published articles are also generally in the pediatric population. Hence the present article reports a case of oromandibular dystonia with temporomandibular joint dislocation with metoclopramide.

Case Report

A 21-year-old female patient, who had no known additional disease, came to the emergency department with the complaints of inability chew and swallow, difficulty in speaking, pain at right temporomandibular region that started a few hours ago. She was diagnosed with temporomandibular joint dislocation. Subsequently dislocation was reduced by traditional intraoral tecnique. The patient was discharged with oral symptomatic treatment and a recommendation for plastic surgery outpatient control. After a few hours, she came back to the emergency department with the same complaints and additional cervical contraction.

On physical examination, she was having dystonia of the right mandibular region and left posterior servikal region and no dystonia at other parts of the body. Her vitals and systemic examination were normal. When the patient's anamnesis was detailed, it was learned that the patient had used metoclopramide 2 days ago with a prescription due to nausea and vomiting. The patient was diagnosed with metoclopramide-induced acute dystonia. She was treated with intravenous (IV) biperiden 5 mg slow infusion and IV fluids to maintain hydration. The patient was observed in the emergency room for 4 hours after treatment. In the follow-up, dystonia decreased and the patient was able to close her mouth and started eating. The patient was discharged with full recovery after 8 hours observation. If the patient needed antiemetics again, it was recommended not to use metoclopramide.

Discussion

In the present case we reported an unconventional presentation of oromandibular dystonia with TMJ dislocation in a patient treated with oral metoclopramide 10 mg. If dystonia is severe, it can cause temporomandibular joint dislocation (2).

Metoclopramide may cause extrapyramidal symptoms, usually occurring as acute dystonic reactions within the first 24-48 hours (6). In studies conducted in the developed world, the incidence of metoclopramide-induced dystonic reactions is reported as 1:500 patients (10). Female patients, children, adults younger than 30, and patients taking high doses of metoclopramide have higher chances of developing dystonic reactions (6)(11). Our patient was a 21-year-old female, therefore, had a slightly higher risk of developing a dystonic reaction to metoclopramide compared with the general population.

Management of acute dystonia includes primarily discontinuation of metoclopramide followed by administration of injectable anticholinergic and antihistamine drugs, mostly benztropine and diphenhydramine. The response to treatment is often dramatic and typically occurs within minutes of intravenous drug administration. If initial treatment is successful, therapy is continued orally for two to three days to prevent recurrence. Alternative treatments for dystonia include benzodiazepines , amantadine, or biperiden (12). In this case, our patient had jaw dislocation and cervical contraction. It could not be applied to the patient because there were no drugs that should be used in the treatment management in the first place. Therefore, our patient was given an alternative drug, biperiden, intravenously. Since there was a situation requiring urgent intervention, the medicine in our institution was given to the patient. Medications to be used in the treatment of such extrapyramidal symptoms should be available in emergency departments using metoclopramide.

Acute dystonic reaction in the emergency department is a very serious problem due to the high probability of misdiagnosis. In the event of a dystonic reaction, the fatal differential diagnosis that physicians must make is tetanus, partial seizures, strychnine poisoning, hypocalcemia, or other electrolyte imbalances (13).

TMJ dislocation is a rare complication of oromandibular dystonia, but it can cause significant fear and discomfort to the patient and their relatives. In our case, dislocation of the temporomandibular joint was first misdiagnosed and oromandibular dystonia was not recognized. Metoclopramide-induced TMJ dislocation has been reported in the pediatric population in studies conducted so far, but no such case has been reported in adult patients.

In this case, it was shown that TMJ dislocation can also be seen in the adult patient population due to the side effect of metpamide. In these patients, it was emphasized that the anamnesis should be deepened and drug use should be questioned.

Conclusion

Metoclopramide is an antiemetic drug that can cause serious adverse events such as acute dystonic reaction. Among these side effects are oromandibular dystonias, which may lead to TMJ dislocation. Physicians and other healthcare professionals working in the emergency department should be familiar with such side effects. The most rapid treatment of acute dystonic reaction caused by metoclopramide

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is intravenous or intramuscular administration of anticholinergics and antihistamines.

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Case Report

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Posterior Reversible Encephalopathy Syndrome in Covid 19 Disease: A Rare Case

Öztürk Taşkın[1], Ayşe Yılmaz[2], Veysel Garani Soylu[3], Ufuk Demir[1], Gizem Kurada[2]

[1]Department of Anestehesiology And Reanimation,Kastamonu Universty Medicine Faculty , Kastamonu, Turkey.

[2]Department of Anestehesiology And Reanimation, Kastamonu Training and Research Hospital, Kastamonu, Turkey.

[3]Department of Intensive Care, Kastamonu Universty Medicine Faculty, Kastamonu, Turkey.

Abstract

Covid-19 disease can have many neurological complications, posterior reversible encephalopathy being one of them. A 42-year-old female patient was admitted to the emergency department on the 4th day of PCR test positivity with decreased consciousness. Patient with a Glasgow coma score of 6 (E1M3V2). The patient's vital signs were stable and there was no abnormality in his laboratory. No pathology was found in the brain tomography to explain the loss of consciousness. In MRI evaluation, the patient was diagnosed with Posterior reversible encephalopathy syndrome (PRES) according to radiological and clinical information. The standard Covid -19 treatment added to the antiedema treatment was applied to the patient. Brain MRI was repeated four days later and previous MRI findings showed regression. The patient was discharged to the ward with stable neurological status on the 22nd day of hospitalization. PRES is a rare but serious complication in Covid-19. Especially in the Covid-19 intensive care unit, intubation and sedation suppress the clinical picture, and the difficulty in getting patients on magnetic resonance imaging can complicate the diagnosis. Therefore, PRES should be kept in mind in the presence of neurological symptoms such as encephalitis, meningitis, encephalopathy and cerebrovascular disease.

Keywords: Covid-19, posterior reversible encephalopathy syndrome, intensive care unit, neurology

Introduction

Approximately 250 million cases and 5 million deaths due to the Covid -19 disease have been reported due to the SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) virus, which emerged in 2019 in Wuhan, China (1). Most of the Covid 19 patients are asymptomatic and the most common symptoms are fever, cough, shortness of breath, myalgia and headache. The most common serious manifestation of the disease is pneumonia. In severe cases, Severe Acute Respiratory Syndrome is the most important complication (2). In addition to these symptoms, neurological symptoms were also observed in more than 30% of the patients. These neurological symptoms are myalgia, dizziness, headache, hyposmia, hypogeusia, polyneuropathy, encephalitis and encephalopathy (4). Although the pathophysiology of the damage caused by the SARS-CoV-2 virus has not yet been resolved, theories exist. One of them is that the virus can enter the brain via the transcriptional route or by using ACE-2 receptors in endothelial and glial tissues, as in other central nervous system pathogens (3). Cerebral edema, which can be seen in Covid 19 patients, also supports this theory.

Posterior reversible encephalopathy syndrome (PRES), a condition that can develop due to eclampsia-preeclampsia, hypertension, drug intoxications and many metabolic diseases, was first described by Hinchey et al. in 1996. The syndrome may progress with clinical findings in the form of seizures, headache, nausea, vomiting, mental status changes, visual loss, and focal motor loss (4). The pathophysiology of PRES is unknown. Two possible mechanisms are thought to be hypertension and endothelial dysfunction. As a result, the blood-brain barrier deteriorates and vasogenic cerebral edema develops (5). In the diagnosis of PRES, nonspecific changes can be seen in computerized brain tomography (CT), while specific findings are seen in brain magnetic resonance imaging (MRI). Therefore, MRI rather than CT is recommended for diagnosis because characteristic findings of cerebral edema with bilateral and symmetrical involvement in the occipital and parietal regions are easily demonstrated by MRI (5). The fact that this syndrome is reversible reveals the importance of rapid diagnosis and initiation of treatment . In the treatment of PRES, it is essential to control blood pressure and seizures, as well as to adjust the dose if cytotoxicity has developed due to drug use and to treat the known metabolic disease.

In this case report, we aimed to review the posterior reversible encephalopathy syndrome (PRES), which is a consequence of the endothelial dysfunction associated with COVID-19.

Corresponding Author: Öztürk Taşkın
e-mail: drozturk275@hotmail.com
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Case Report

A 42-year-old female patient was admitted to the emergency department on the 4th day of PCR test positivity with regression in consciousness. It was learned from her family that the patient did not have any additional disease, was not vaccinated against Covid-19 and did not use the favipiravir given for treatment. The patient, whose glaskow coma score was calculated as 6 (E1M3V2), was intubated and received mechanical ventilator support. Her heart rate was 103 / min rhythmic, arterial pressure was 123/67 mmHg, Sp02 was 98 and fever was 37.8 ^{0} C . Muscle strength was 2-3/5 on the upper left, 1-2/5 on the lower left, and Babinski was positive on the left, and nuchal rigidity could not be evaluated as optimal due to agitation. While the patient's thorax tomography had a ground-glass image compatible with bilateral covid, there was no pathology to explain the loss of consciousness in the brain tomography. The patient who underwent bedside echo did not have any cardiac etiology. Patient admitted to covid intensive care unit . Laboratory findings are; ph:7.39, pC02: 21.1 mmHg, p02:158 mmHg, lactate:1.9 mmol/L, Na:145 mEq/L, glucose: 247mg/dL, urea: 56mg/dL, creatinine: 0.87 mg/dL, alt :18U/L, ast 23 U/L, hs troponin: 5 ng/L, ldh: 498 U/L, ferritin: 480 ng/ml, hs troponin: 5 ng/L, sedimentation: 56, CRP: 75.4 mg/L, wbc: 30.82 10^{3}µ/L, hgb:15.9 g/dL, hct:49.4 %, plt:480 10^{3} µ/L, lymph: 2.22 10^{3} µ/L, INR:1.32, and D-Dimer: 1.94mg/L.

Lumbar puncture was performed on the patient for the differential diagnosis of meningitis and encephalitis. Sample and simultaneous biochemistry were studied for cerebrospinal fluid culture and cell count. The patient was administered favipiravir, meropenem and other supportive treatments. Brain magnetic resonance imaging was performed at the 20th hour of hospitalization of the patient who had no growth in the cerebrospinal fluid culture, no leukocytes in the cell count, and no abnormality in biochemical parameters. In MRI evaluation, it was observed that there was an increase in subcortical and cortical intensity in the bilateral posterior region and occipital area in the left hippocampus posterior and temporal lobe level (Figure 1). The patient was diagnosed with Posterior reversible encephalopathy syndrome (PRES) based on radiological and clinical information. The patient was administered 3 mg/h midazolam infusion and antiedema treatment. Brain MRI was repeated four days later, and previous MRI findings showed regression (Fig. 2). Midazolam was discontinued. The patient was awakened and cooperation was established. The patient was extubated on the 15th day of hospitalization and respiratory physiotherapy was started. The patient's covid treatment was continued and completed simultaneously. She was discharged to the service in a neurologically stable condition on the 22nd day of her admission to the care.

Discussion

PRES is characterized by headache, confusion, seizures, blurred vision (with normal pupillary reflex and fundus examination and intermittent hallucinations) and often accompanied by hypertension (6). No seizure was observed in our patient, and bilateral light reflex was found to be positive and the fundus examination was normal in her

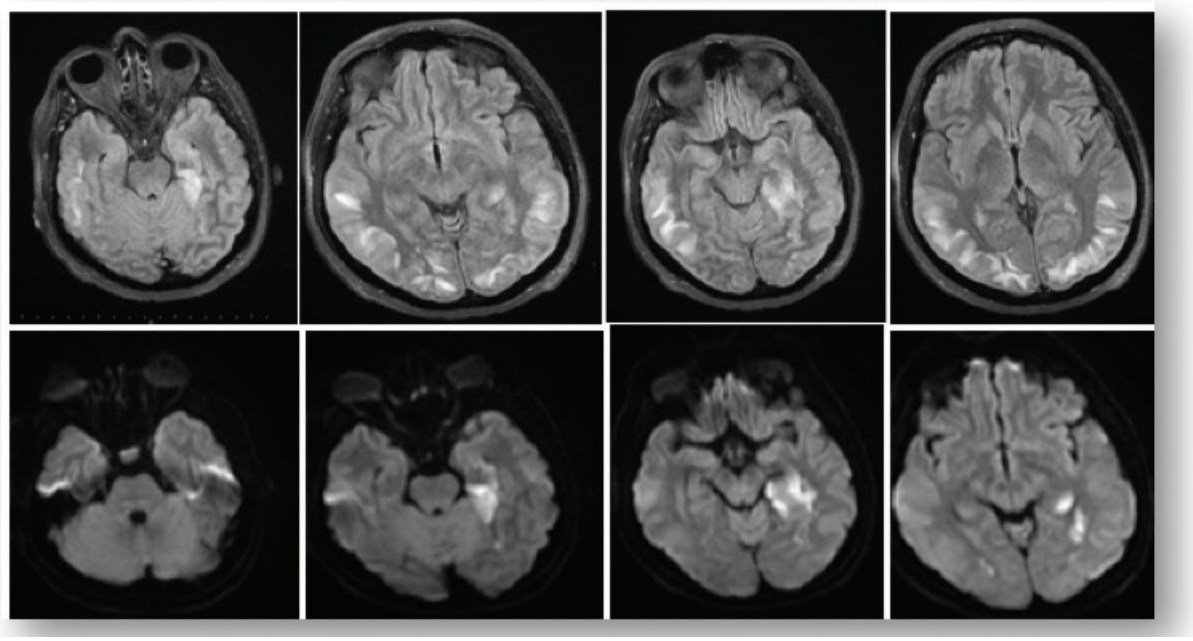

Figure 1. Patient's first Brain MRI Findings.

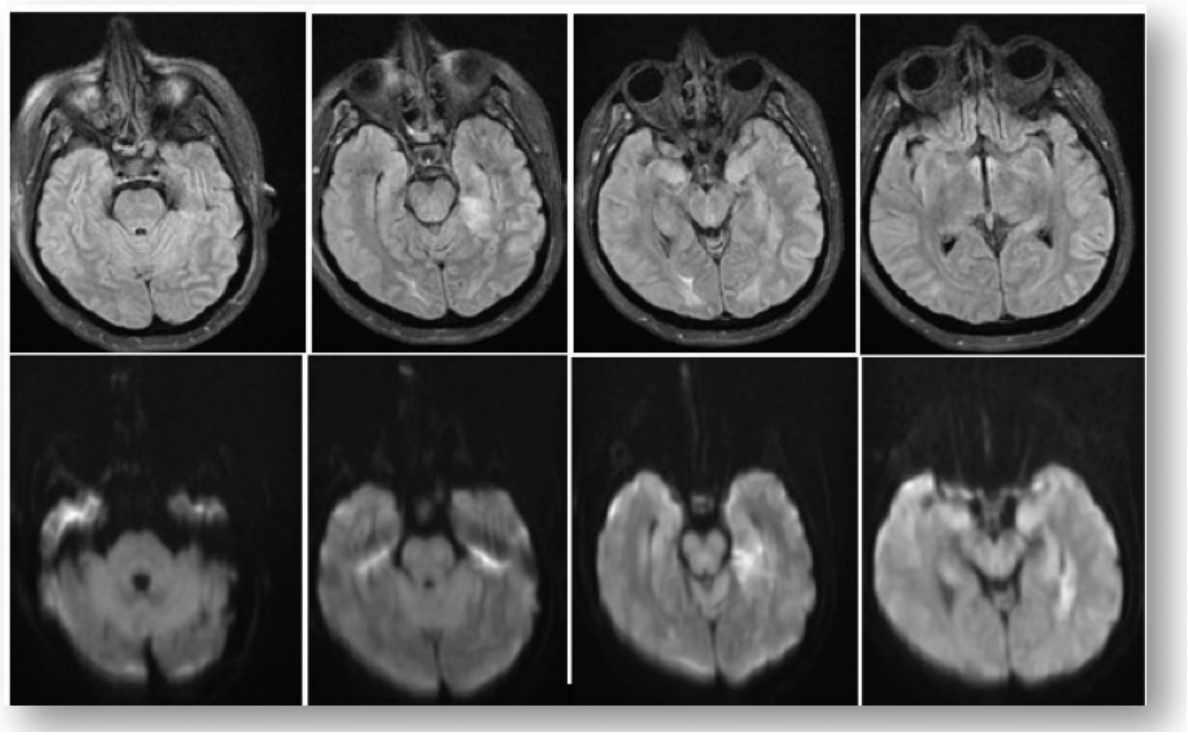

Figure 1. Patient's first Brain MRI Findings.

neurological examination. In severe covid 19 disease, PRES may develop due to comorbidities, inflammatory response and endothelial dysfunction. Although the pathophysiology of this syndrome has not been fully resolved, PRES is mostly characterized by impaired cerebral blood barrier autoregulation after severe hypertension or as a result of severe infection or vasotoxicity. Most of these patients have either an autoimmune condition or an immune disorder (7). While the most common cause was hypertension, our patient had normal blood pressure. Endothelial dysfunction in Covid 19 disease may be associated with PRES and other vascular complications. In recent studies, this hypothesis was supported by detecting microcirculation and vascular glycocalyx changes in covid 19 patients (8). The absence of hypertension and severe inflammation in our patient also supports this theory. Due to severe infection, the immune system is activated and cytokine production is stimulated. As a result, especially tumor necrosis factor a and vascular endothelial growth factor levels may increase. This increases the blood-brain barrier permeability, leading to the development of cerebral edema. This situation has also been described in viral infections such as sepsis, influenza A and parainfluenza (9). A similar complication may develop in PRES associated with Covid 19. Infection values were also high in our patient.

PRES cases have also been reported during Tocilizumab (IL-6 Antibody) treatment, which is used for cytokine storm in Covid 19 disease (10). Tocilizumab was not used in the treatment of our patient. Renal failure is also an important risk factor for PRES, and our patient's kidney function tests were normal (8).

PRES can be seen in patients with covid 19 due to many potential triggers. Especially uncontrolled hypertension, excessive cytokine production, endothelial dysfunction, renal failure and immunomodulatory treatments may cause the development of PRES.

Conclusion

In conclusion, PRES is a rare but serious complication in covid 19. Especially in the covid 19 intensive care unit, intubation and sedation suppresses the clinical picture and the difficulty of taking the patients to magnetic resonance imaging can make the diagnosis difficult. Therefore, PRES should be kept in mind in the presence of neurological symptoms, such as encephalitis, meningitis, encephalopathy and cerebrovascular disease. It should be known that early diagnosis and treatment are of vital importance and that recovery without sequelae is possible.

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Case Report

Journal of Emergency Medicine Case Reports

A Clinical Picture of Anticholinergic Toxidrome Developed Due to Scutellaria Orientalis: A Case Report

Yıldız Yıldırımer[1], Sema Yeşilkaya[1], Huzeyfa Yeşilkaya[1]
[1]Doğubayazıt Dr. Yaşar Eryılmaz Devlet Hastanesi, Ağrı, Turkey.

Abstract

Scutellaria orientalis; also known as yellow eucalyptus, is a plant species with yellow flowers. Generally, it is used by locals as an antidiarrheal and antihemorrhagic, and to relieve fatigue in the regions where it grows endemically in eastern Turkey. The current case comprised a 23-year-old patient, who ate the plant in question, and presented with an overall clinical picture dominated by anticholinergic toxidrome. Although anticholinergic toxidrome is a syndrome that can be diagnosed using various clinical findings, its treatment is symptomatic and physostigmine can be used in the treatment of severe cases. Depending on the clinical course of the patient, the possibility of follow-up and treatment in intensive care units may arise.

Keywords: Scutellaria orientalis, anticholinergic toxidrome, antidiarrheal

Introduction

Scutellaria orientalis, also known as *kulîlka kûçik* (small yellow flowers), or *dermana telî* in Ağrı Doğubayazıt, is a plant that is consumed by local people for the treatment of diarrhea, fatigue, etc. Lamiaceae family of Scutellaria species of the genus have been identified worldwide 360, turkey also comes with 16 types (1). While anticholinergic toxidrome may present with symptoms related to peripheral nervous system effects, such as flushing, urinary retention, ileus, dry mouth, and blurred vision, it may also present with findings related to effects on the central nervous system, such as agitation, delirium, stupor, and coma (2). Anticholinergic syndrome can be caused by drugs or substances that prevent the binding of acetylcholine to muscarinic receptors (3). In this article, it was aimed to discuss a patient who ate Scutellaria orientalis herb for 2 days for the treatment of diarrhea and was brought to the emergency room due to fatigue and fever.

Case Report

A 23-year-old male patient was brought to the emergency room by his relatives due to malaise. His vital signs were noninvasively measured fever of 38 °C, blood pressure of 110/70 mmHg, heart rate of 132 beats/min, and respiratory rate of 20/min, on admission electrocardiography of sinus tachycardia, and blood oxsigen level of 100%. On arrival at the emergency department, his Glasgow coma score was evaluated as 15, he was conscious, presenting with abdominal bloating, but no tenderness. In the anamnesis taken from the patient, it was learned that he had not been able to urinate for a while. On first examination, his general condition was poor and his breathing was irregular. The face and neck of the patient were flushed, the oral mucosa was dry, the pupils were mydriatic, the vesical globe was detected, and 1000 cc of stomach contents were drained using a nasogastric tube. After the urinary catheter was inserted, 1000 cc of urine was drained. The laboratory fparameters were pH of 7.23, lactate level of 8.8, urea: 102 mg/dL, creatinine: 3.32 mg/dL, aspartate Aminotransferase (AST): 118 U/L, alanine aminotransferase (ALT): 97 U/L, and creatine kinase (CK): 3227 U/L. There was no leukocytosis or hyperglycemia. The other blood parameters were within the normal ranges.

No infiltration was detected in the thoracic computed tomography of the patient. In the abdominal computed tomography of the patient, the stomach and intestines were dilated. This was evaluated together with general surgery and radiology clinics and it was determined that no pathology requiring urgent surgical intervention was detected. The light reflex of the patient, whose pupils were mydriatic in the neurological examination, was bilateral. Antipyretic, empirical antibiotics, and fluid replacement

Corresponding Author: Yıldız Yıldırımer
e-mail: yildiz_yildirimer@hotmail.com
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therapy were started. According to the information obtained from the relatives of the patient, it was learned that the patient, whose family history and personal history was unremarkable, had diarrhea for two days, and had consumed Scutellaria orientalis (yellow eucalyptus) for 2 days to treat it. Photographs were taken of the flower brought during the hospital follow-up and its type was confirmed (Figure 1); however, no case related to this plant was found in the literature. In the follow-up of the patient, while his breathing had become shallower, with a blood oxsigen level of 70 and Glasgow coma score of 7, the patient was intubated with drug induction. Spontaneous circulation was established after 20 min of cardiopulmonary resuscitation in the patient, who had arrested 10 min after intubation, and he was transferred to the intensive care unit. In light of this information, anticholinergic toxidrome was considered based on the patient's clinical findings and anamnesis.

Discussion

Worldwide, 360 species of the genus Scutellaria of the Lamiaceae family have been described, 16 species of which are in Turkey (1), with Scutellaria orientalis being one of them. It is used by locals as an antidiarrheal in the regions where it grows endemically in Turkey.

In the literature review on this species, although in the vitro anti-leukemic activity of the plant was studied (4), no studies on its anticholinergic content could be found.

Anticholinergic toxidrome is characterized by both central and peripheral physical findings. Central anticholinergic syndrome, which is a term used to describe symptoms resulting from decreased cholinergic activity in the central nervous system, is primarily characterized by signs and symptoms consistent with hyperactive delirium. Peripheral anticholinergic syndrome include mydriasis and blurred vision, chills, ataxia, fever/hyperthermia, red and dry skin, dry oral mucosa, decreased bowel sounds, constipation, and urinary retention. In advanced cases, central anticholinergic syndrome may be associated with seizures, coma, respiratory failure, and cardiovascular collapse (5).

Anticholinergic syndrome may be caused by drugs or substances that prevent acetylcholine from binding to muscarinic receptors (3). These drugs include antihistamines (diphenhydramine, hydroxyzine, promethazine), antiparkinsonian agents (benztropine, trihexyphenidyl), antipsychotics (phenothiazines, butyrophenones), belladonna alkaloids (aladonna alkaloids and similar, hyocyanin, ipratropium), and mydriatics (cyclopentolate, tropicamide) (2)

During the patient's application, the history of such drug use was carefully questioned, but no drug use history was found. However, after the patient's deterioration in the follow-up, the patient's relatives presented the plant that they brought with them, and toxidrome came to mind in the diagnosis.

In anticholinergic syndrome, the pupils are usually dilated and their response to light is poor. Clinical conditions such as fever, flushing, ileus, cardiac arrhythmias, tachycardia, urinary retention, hypoactive bowel sounds, choreoathetosis, myoclonus, visual and auditory hallucinations, convulsions, and coma may also be observed (2).

Benzodiazepines for sedation are frequently used to control the delirium and agitation resulting from anticholinergic findings (2). Physostigmine is a specific antidote, it crosses the blood-brain barrier and inhibits reversible anticholinesterase. Physostigmine should be given in cases of tachycardia, coma, and respiratory arrest (6). Delirium and agitation were not present in the current case. However, anticholinergic syndrome was considered for the patient, whose general condition deteriorated during the follow-up. The patient who was intubated due to respiratory and cardiac arrest, was referred to a center where physostigmine could be administered, since physostigmine was not exist in the hospital. It was learned that he died 1 day after his transfer to the intensive care unit.

Conclusion

Poising cases have an important place for morbidity and mortality in Emergency Medicine services. Among these, poisoning cases due to plants are quite rare (7). In patients presenting to the emergency department with agitation, delirium and anticholinergic symptoms, poisoning due to plants used in the treatment of many diseases common in our country should be considered. It should not be forgotten that plants that are widely used among the public often cause poisoning and toxic effects.

A detailed anamnesis should be taken in patients presenting with anticholinergic findings, and the consumption of such plants should be questioned in addition to the intake of anticholinergic drugs in the anamnesis. Although no such case could be found in the literature, it is our belief that anticholinergic syndrome may develop as a result of the ingestion of Scutellaria orientalis (yellow eucalyptus).

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Case Report

Journal of Emergency Medicine Case Reports

Acute Kidney Injury in a Patient with Darifenacine Overdose

Dilber Üçöz Kocaşaban[1], Sertaç Güler[1], Serdal Ateş[1]
[1] University of Health Sciences, Ankara Training and Research Hospital, Department of Emergency Medicine, Ankara, Turkey.

Abstract

Darifenacin is a medication that has anticholinergic and antimuscarinic effects and is used in the treatment of overactive bladder. Adverse and overdose effects of the medication such as dry mouth and eyes, headache, nausea, constipation, urinary retention, and dyspepsia have been reported. An overdose effect on acute kidney injury (AKI) has not been reported in the literature, and no dose adjustment is recommended in patients with renal impairment. A 24-year-old male patient applied to the emergency department with a declaration of taking 25 drugs containing darifenacin active ingredient for suicidal purposes. The patient had complaints of nausea and difficulty urinating. The patient's physical examination was normal. In laboratory, creatinine was 2.09mg/dL, and hemoglobin (+++) and protein (++) were found in urine test. Renal ultrasonography revealed a grade 1-2 increase in echogenicity of both kidneys. The patient was followed up with intravenous fluid replacement therapy, and the patient whose creatinine levels regressed was discharged with the recommendation of nephrology outpatient control. Any molecule taken in overdose can cause AKI via acute tubular necrosis should not be ignored by emergency physicians. The fact that such a side effect or undesirable situation has not been reported with Darifenacin so far doesn't mean that the molecule is safe for this situation. An overdose of Darifenacin may cause AKI in patients.

Keywords: Acute kidney injury, darifenacine, drug overdose, emergency medicine, kidney tubular necrosis, suicide

Introduction

Acute kidney injury (AKI), which was previously called acute renal failure (ARF), was redefined by the KDIGO clinical guidelines in 2012, and an increase in serum creatinine level by more than 0.3 mg/dL within a 48-hour period and/or increase in serum creatinine level to ≥1.5 times baseline, which was obtained 1 week ago constituted the first 2 items of the new definition (1). Darifenacin is a medication that has anticholinergic and antimuscarinic effects and is used in the treatment of overactive bladder (2). Adverse and overdose effects of the medication (Emselex® 15 mg tablet) such as dry mouth and eyes, headache, nausea, constipation, urinary retention, and dyspepsia have been reported (2). An overdose effect on AKI has not been reported in the literature, and no dose adjustment is recommended in patients with renal impairment (2).

We present a 24-year-old male patient, who we followed up with the diagnosis of AKI, who drank 25 pieces of his father's medication with active ingredient Darifenacin (Emselex® 15 mg tablet) for suicide and presented to the emergency department with the complaint of difficulty urinating 3 days later.

Case Report

A 24-year-old male patient was admitted to the emergency department with complaints of nausea and difficulty in urinating. The patient drank 25 pieces of his father's medication with the active ingredient Darifenacin (Emselex® 15 mg tablet) 3 days ago for the purpose of committing suicide. The patient, who did not apply to any health facility during that period, did not have any complaints such as vomiting, abdominal pain, oral inability, syncope, palpitations. The patient, who had difficulty in urinating and complaining of constipation at first, stated that his urine output improved and he returned to normal. On examination, the patient's vital signs were stable, general condition was good, consciousness was clear, cooperation was complete, the abdomen was relaxed, mucous membranes and skin turgor were normal. There was no pretibial edema. There was no globe vesicale. There were no additional diseases in the medical history. The patient had no history of smoking, alcohol, or any other medication use. In the biochemical examinations, creatinine was detected as 2.09mg/dl (N: <1.2mg/dl). There was no metabolic acidosis. Bicarbonate and lactate levels were normal. Electrolyte

Corresponding Author: Dilber Üçöz Kocaşaban
e-mail: dilberucoz@hotmail.com
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values were found to be normal. His hemoglobin level was normal. A urinary catheter was inserted into the patient and treatment was started with the preliminary diagnosis of post-renal ARF and/or acute tubular necrosis (ATN) due to the antimuscarinic/anticholinergic side effects of the medication. Hemoglobin (+++) and protein (++) were detected in the complete urinalysis. The patient had urine output and it was normal, and intravenous (IV) salin hydration at a dose of 150 cc/hour and volume replacement was started. The patient was consulted with Internal Medicine and Urology with the preliminary diagnoses of ATN and post-renal ARF, and Psychiatry due to the suicide attempt, and was taken to the emergency critical care unit and followed up.

Hydronephrosis was not detected in both kidneys in the abdominal ultrasonography (USG) of the patient, and it was reported that the echogenicity of both kidneys increased by grade 1-2. No acute pathology was detected in the unenhanced abdominal computed tomography (CT) examination. The urology department did not consider the post-renal ARF in the patient who had urine output and did not have hydronephrosis. The patient's creatinine values, which were taken once every 6 hours and then twice a day, were found to be 2.19mg/dl, 2.85 mg/dl, 2.70 mg/dl, 2.78 mg/dl, 2.6 mg/dl, and 2.2 mg/dl, respectively. Nephrology did not plan emergency hemodialysis for the patient and did not (could not) perform scintigraphy, elective USG or renal biopsy to diagnose or rule out ATN because they could not hospitalize the patient. Our opinion that the patient, whose follow-up was performed by us, might have ATN was not shared by the Nephrology department together with poison counseling on the grounds that Darifenacin-induced ATN is not a highly anticipated clinical picture. Due to regression in creatinine level, good general condition, and good urine output, the patient was discharged to come for outpatient follow-ups by the Nephrology department after 3 days of emergency critical care hospitalization. We observed that the patient's creatinine level requested by the Nephrology department was 1.5mg/dl 3 days after discharge and was at normal level thereafter.

Discussion

Acute kidney injury is characterized by a sudden decrease in renal function. Causes can be prerenal, renal, or post-renal. AKI is observed in approximately 10% of hospitalized patients (3). We did not consider a pre-renal cause in our patient, since there was no condition that would cause real volume loss such as nausea, vomiting, diarrhea, bleeding, and burns, and hypotension, or third space loss did not develop. About 70% of AKI is caused by prerenal conditions and ATN. In a very small group of patients, the cause is urinary tract obstruction (approximately 10%) (4).

The medication has antimuscarinic and anticholinergic side effects (2), but unlike the nephrology department, we did not consider a post-renal cause in our patient due to the normal amount of urine output at the patient's admission, the absence of a condition such as a globe vesicale, the continuation of urine output after insertion of the bladder catheter, and the lack of condition such as hydronephrosis in renal imaging (USG and CT).

Darifenacine is rapidliy an almost completely absored (%97) from the gastrointestinal tract with maximum plasma levels being reached after 7 hours. The elimination half-life of darifenacine is approximately 3h. Darifenacine is lypophilic, exhibits high protein binding (%98). About %58 of the dose is excreted in the urine and %42 in the feaces. Only a small percentage (%3) is excreted in the form of unchanged drug. Metabolism is mediated by hepatic cytochrome P450 2D6 and 3A4, the main metabolic routes being monohydroxylation in the dihydrobenzfuran ring, dihydrobenzfuran ring opening, and *N*-dealkylation of the pyrrolidine nitrogen (5).

In our opinion, the basic condition in the patient is medication-induced ATN. Many endogenous and exogenous toxins can cause ATN, and the main ones have been reported as vancomycin, aminoglycosides, iron pigments, cisplatin, radiocontrast material, pentamidine, foscarnet, mannitol, IV immunoglobulins, and synthetic cannabinoids (6,7). We think that DFC metabolites may also cause this toxic effect.

Since our patient could not be hospitalized by the Nephrology department, the inability to perform further investigations (fractional excretion of sodium and urinary sodium concentration, advanced imaging, renal biopsy, etc.) in terms of differentiation and definitive diagnosis is an important limitation for this case, however, medication overdose (8), exposure to the agent within 24 hours, return of creatinine level to normal after elimination of the agent and the increase in renal echogenicity on USG are strong evidence suggesting the cause as ATN. In our era, side effects of the medications can be detected to a large extent after these molecules start to be sold. One of the conditions that increase the risk of medication-related nephrotoxicity is the high dose (7). Our patient also drank 25 pieces of this medication belonging to his father 3 days before his application. However, medication-induced AKI can be dose-dependent or idiosyncratic (6).

Conclusion

Since we know that technology has not produced perfect medications without side effects until now, the fact that any molecule taken in overdose can cause AKI via ATN should not be ignored by emergency physicians. The fact that such a side effect or undesirable situation has not been reported

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with Darifenacin so far does not mean that the molecule is safe for this situation. An overdose of Darifenacin may cause AKI in patients. We believe that our case will be clarfy for emergency medicine physicians in revealing the possibility of AKI in DFC overdose.

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Case Report

Journal of Emergency Medicine Case Reports

Spontaneous Spleen Rupture Due to Infectious Mononucleosis

Bilal Aslan[1], Semih Hot[1], İlknur Mansuroglu[1]

[1]Department of Surgery, Division of Surgical Oncology, Mersin University School of Medicine, Mersin, Turkey.
[2]Department of Surgery, Istanbul Cemil Tascıoğlu City Hospital, University of Health Sciences, Istanbul,Turkey.
[3]Department of Pathology,Istanbul Cemil Tascıoğlu City Hospital, University of Health Sciences, Istanbul,Turkey.

Abstract

Spontaneous spleen rupture (SSR) is a fatal and rare pathology causing acute abdomen. SSR secondary to infectious mononucleosis (IM) is quite rare (0.06-0.5%), and it is the most common cause of IM associated death. A 43 years old male patient, who had no previous severe disease history, chronic drug use, or previous operation applied to our emergency outpatient clinic of general surgery. The patient had complaints of severe abdominal pain, nausea, vomiting, and diarrhea. In abdominal computerized tomography, diffuse free fluid with respectively increased in density (hemorrhage?) was observed. Emergency operation was decided, because patient developed acute peritonitis signs. During exploration in the operation, it was observed that spleen was ruptured at multiple sites, and bleeding was ongoing, and splenectomy was performed. Serology was consistent with the previous EBV infection. It was mentioned in the pathology report that there was no neoplastic infiltration, and infectious causes should be investigated. No problem has been encountered during approximately two years' follow-up. SSR secondary to EMN is a rare, fatal, and very severe pathology. Diagnosis is delayed, or it is not even diagnosed because there is no trauma. Correct diagnosis on time, and decision of emergency surgical intervention can be life-saving.

Keywords: Spontaneous spleen rupture; infectious mononucleosis; surgery.

Introduction

Spontaneous spleen rupture (SSR) is a fatal and rare pathology causing acute abdomen. It constitutes only 1% of all spleen ruptures [1]. Four criteria are important in the diagnosis; a a careful anamnesis; no sign of any organ disease, which may cause rupture, other than the spleen; absence of previous trauma or perisplenic adhesions due to rupture or scar tissue; and normal macroscopic and histologic examination of the spleen [2]. Causes of SSR are examined in seven categories; neoplastic, infectious, hematologic, inflammatory, iatrogenic, primary splenic diseases, and idiopathic. SSR secondary to infectious mononucleosis (IM) is quite rare (0.06-0.5%), and it is the most common cause of IM associated death [3]. Patients diagnosed with IM are generally at the age of tens or young adults. Mortality is especially damaging in this population, and it confirms the importance of awareness of IM. Its incidence is reported 345-671 in 100.000 individuals [3]. We present this very rare case to increase awareness especially to emergency physicians and surgeons.

Case Report

A 43 years old male patient, who had no previous severe disease history, chronic drug use, or previous operation applied to our emergency outpatient clinic of general surgery. There was no specific finding in his family history. He was not taking alcohol or smoking. The patient had complaints of severe abdominal pain, nausea, vomiting, and diarrhea. The patient had influenza infection 10 days ago, and he had no trauma history. Besides, the patient was a compressor worker who was breaking concrete, so it was possible that he had chronic trauma in his abdomen. Laboratory tests revealed CRP: 76.48 mg/L, WBC: 13.6, Hb: 10.8 g/dL, Hct: % 33.6. The vital signs were blood pressure: 100/70 mmHg, pulse rate: 110/min and rhythmic, respiratory rate: 18/min. There was tenderness and defense in the left abdominal quadrant in the physical examination. In abdominal ultrasonography, there was a heterogenous image filling up the left subdiaphragmatic space, encircling the spleen, and causing irregular borders (hematoma?). In abdominal computerized tomography, diffuse free fluid with respectively increased

Corresponding Author: Bilal Aslan
e-mail: bilalarslan84@hotmail.com
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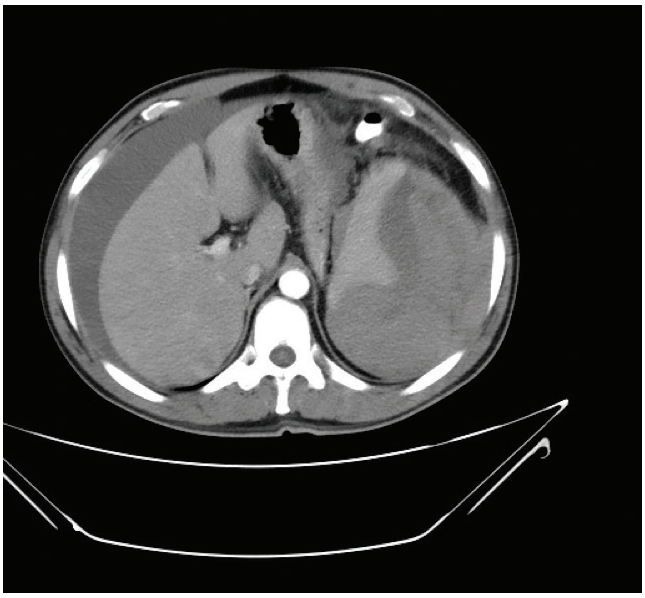

Figure 1. CT image of spontaneous splenic rupture.

in density (hemorrhage?) was observed, and spleen size was increased. There were heterogenous areas which were not enhanced by contrast substance, and signs indicating contusion. Also, it was reported that lateral borders were erased (Figure 1). Emergency operation was decided, because patient developed acute peritonitis signs, and he responded inadequately to medical treatment. During exploration in the operation, it was observed that spleen was ruptured at multiple sites, and bleeding was ongoing. Splenectomy was performed, and abdomen was washed with abundant amount of serum physiologic fluid. Postoperative period was non-problematic; intestinal movements returned normal in the first day, so nutrition was started with oral liquid nutrients. Causes which might lead to SSR were investigated. EBV IgG profile was anti-VCA gp125 IgG 1+, anti -VCA p19 IgG 3+, anti EBNA-1 IgG 3+, anti p22 IgG 2+, and anti -EA-D IgG was weak positive. Thus, serology was consistent with the previous EBV infection. When evaluated with clinical signs, it was decided that SSR was caused by EMN. Vaccines for capsuled bacteria were administered to the patient in postoperative second day. As there was no complication in postoperative period, the patient was discharged with recovery in the seventh day. Pathological examination reported mesothelial cell proliferation in spleen capsule; inflammatory reaction containing giant cell, neutrophil, and histiocyte; dilation in sinusoids; old and new bleeding foci; and germinal center development in the white pulp (Figure 2, Figure 3). It was mentioned in the pathology report that there was no neoplastic infiltration, and infectious causes should be investigated. Therefore, the report supported EMN as etiological cause. No problem has been encountered during approximately two years' follow-up.

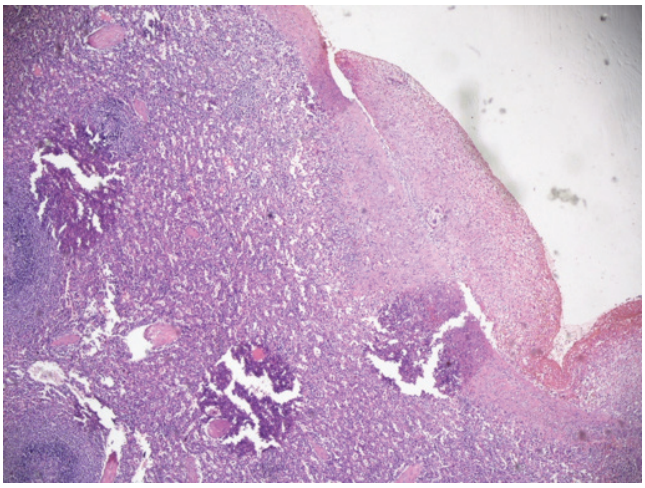

Figure 2. Mesothele cell proliferation in the splenic capsule,giant cell,-neutrophil,histiocytes (HE x 40 magnification)

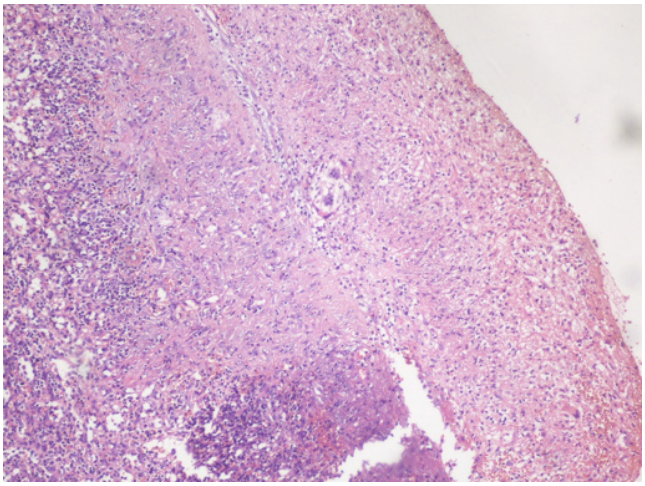

Figure 3. Inflammatory reaction,dilatation in sinosoids,new and old bleeding areas in white pulp (HEx 100 magnification)

Discussion

SSR was first reported in 1861 by Rokintnsy [4]. In 1991, Crate and Payne reported that viral infection, and viral antibody titers should be investigated as the fifth criteria for SSR diagnosis [5]. Generally, spleen reached 3-4 folds of its normal size in EMN patients [2]. Although spleen was non-palpable, splenomegaly was determined 100% by ultrasonography [5,6]. In SSR, the most common complaint is abdominal pain with 88%, and it is generally localized in upper left quadrant of abdomen. Other symptoms are nausea, vomiting, and Kehr's sign.

Since it is rarely encountered, there is no optimal treatment protocol for SSR. Conservative treatment is a well-known approach in traumatic spleen rupture, but 30-days' mortality rate of conservative approach has been reported as 22% in SSR [7]. Emergency surgery is performed in majority of patients, because there are signs of

acute abdomen; diffuse liquid diagnosed by using imaging methods; and there is especially hemodynamic instability. Also, if etiology of rupture is not clarified, then spleen should be removed histopathological examination, and to prevent a delayed secondary intervention.

During EMN infection, spleen rupture may present itself between the first day and 8th week. However, it is observed within the first 4 weeks in 84% of cases. According to a study, in which spleen size was analyzed in EMN patients by using ultrasonography, it was more common within the first 14 days of initiation of SSR symptoms [6,8]. While splenomegaly is severe in the fourth week in 16% of EMN patients, this sign is improved in the 8th week [8]. Spleen rupture was reported in the 3rd week of disease in a histological study which examined changes in spleen structure due to EMN [2]. However, in another study, it was reported that spleen reached its maximum size nearly on 12th day, so rupture was more common in the early phase [6]. Actually, as spleen rupture may occur spontaneously or due to undetermined reasons, it may be wise to avoid from severe and possible traumatic activities. Patient should be asymptomatic before restarting severe activities and exercises. This time period is approximately 8 weeks from initiation of the disease, and improvement of splenomegaly should be verified by using ultrasonography. During the first 8 weeks' time, all sportive activities, heavy lifting, and excessive active life style should be prohibited. However, these prohibitions are not based on controlled studies, national or international guidelines. Therefore, clinical judgement, and local experiences may variable. In this presented case, the patient was treated at outpatient clinic for influenza infection. He did not rest, and continued to work at his heavy job, which was the most facilitating factor for rupture of the spleen, which became fragile during the disease period. It should be kept in mind that the spleen, which becomes sensitive and fragile after infectious mononucleosis, may rupture as a result of trauma to which the patient may be exposed due to work or other conditions, albeit minor, as in our case.

Conclusion

SSR secondary to EMN is a rare, fatal, and very severe pathology. Diagnosis is delayed, or it is not even diagnosed because there is no trauma. Correct diagnosis on time, and decision of emergency surgical intervention can be life-saving.

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Case Report

Journal of Emergency Medicine Case Reports

Spontaneous Spinal Epidural Haematoma Mimicking a Heart Attack: A Case Report

Ismail Istemen[1], Akkan Avcı[2]
[1] Health Science University, Adana City Research and Training Hospital, Department of Neurosurgery, Adana, Turkey.
[2] Health Science University, Adana City Research and Training Hospital, Department of Emergency Medicine, Adana, Turkey.

Abstract

Spontaneous spinal epidural hematoma (SSEH) is a rare disease. Typical symptoms develop chest and back pain, numbness, radicular paresthesis, sensory deprivation and progressive paralysis. SSEH sometimes mimics other diseases like stroke, lung pathologies, myocard infarctus (MI). This situation makes it difficult to treat this pathology. We presented a case with epidural hematoma which compressing the spinal cord at the T2-4 level. He had applied to our emergency service with acute severe chest pain and numbness. He was given clopydogrel, acetylsalicylic acid for the medical treatment of MI by the cardiologist. Therefore, progressive paralysis of lower limbs, incontinence and sensory deprivation were developed. Laminectomy and hematoma evacuation were performed. After phisical therapy, he had a complete neurological recovery on the postoperative day 14. SSEH should be kept in mind and early surgical treatment should be performed if neurological deterioration develops.

Keywords: Chest pain, myocardial ischemia, spinal epidural hematoma, emergency

Introduction

Spinal hematoma is a rare disease and epidural hematomas are the most common type of this disease. The incidence of SSEH is 0.1 per 100000 patients per year[1]. The etiology is unclear in 40% of cases[2]. Neoplasm, trauma, vascular malformation, in prolonged valsalva maneuver, anticoagulation therapy, in patients with internal jugular vein thombosis, in pregnancy, and thrombolytic therapy are the most known causes[3]. Typical symptoms develop chest and back pain, numbness, radicular paresthesis, sensory deprivation and progressive paralysis. Delayed diagnosis and treatment cause poor prognosis. As many article reported, the primary treatment option is decompressive surgery. But some articles report that non surgical treatment or delayed surgery can be performed[4,5].

SSEH sometimes mimics other diseases like stroke, lung pathologies, myocard infarctus (MI). This situation makes it difficult to treat this pathology. We presented a case which mimics a heart attack.

Case Report

A 49-year-old man presented to our emergency service with acute severe chest pain and numbness. Because of the chest pain, he was referred to cardiology by emergency service. Electrocardiography showed T-wave inversion, but the troponin value was normal. He was admitted to the intensive care unit with a preliminary diagnosis of acute coronary syndrome and was given clopidogrel and acetylsalicylic acid by the cardiologist. Five hours later, the patient developed progressive paralysis of the lower limbs, incontinence and sensory deprivation. Physical examination showed paraplegia and sensory deficit below the T4 level. Urgent spinal magnetic resonance imaging (MRI) revealed a posterior epidural haematoma which compressed the spinal cord at the T2–T4 levels. In our patient, the T2-weighted images were hyperintense (Figures 1A and 1B). As soon as spinal spontaneous epidural haematoma was detected, the patient was operated on immediately. Clopidogrel and acetylsalicylic acid were discontinued, as they may increase

Corresponding Author: Akkan Avcı
e-mail: drakkanavci@gmail.com
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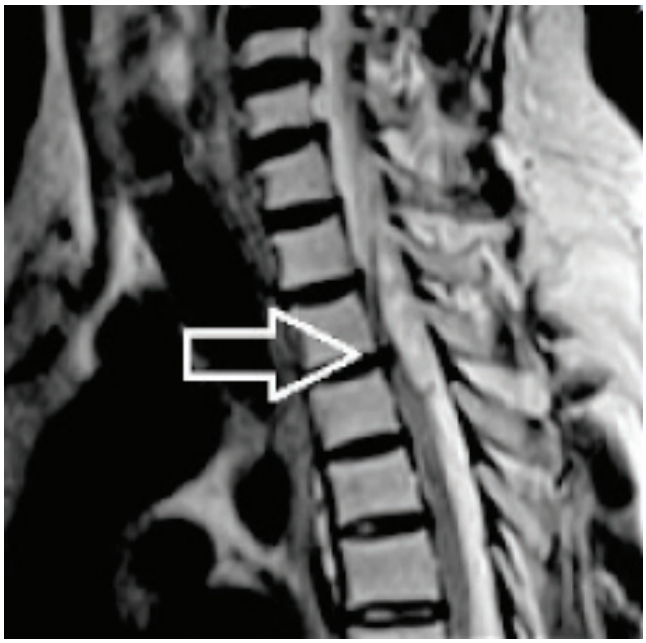

Figure 1A. T2-W sagital MR images (Pre-operative)

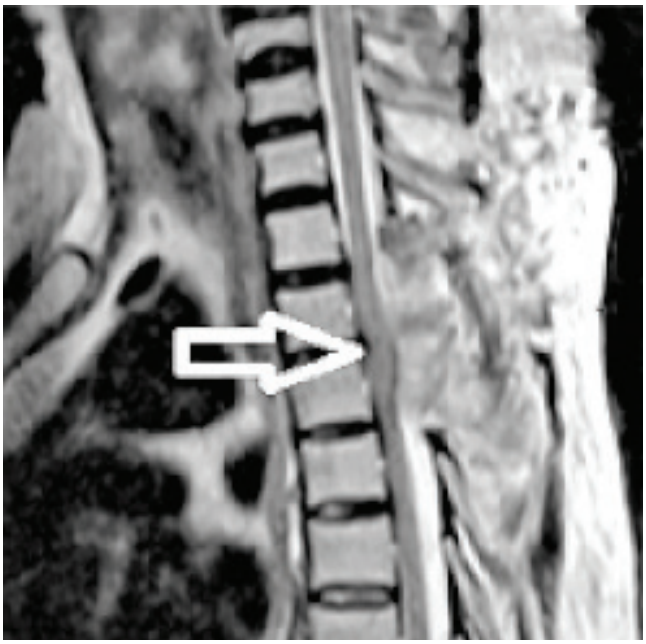

Figure 2A. T2-W sagital MR images (Post-operative)

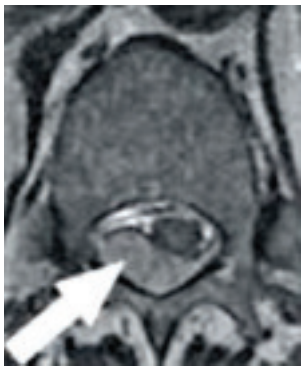

Figure 1B. T2-W sagital MR images (Pre-operative)

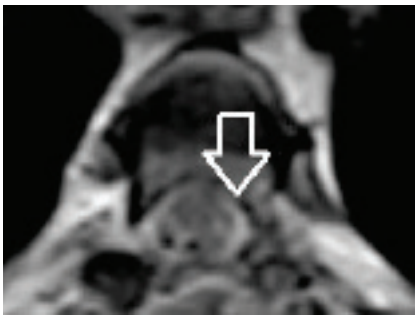

Figure 2B. T2-W axial MR images (Post-operative)

bleeding. Three levels of laminectomy from the lower T2 to the upper T4 and haematoma evacuation were performed. No evidence of vascular malformation or neoplasia was found during the operation. After the haematoma evacuation, the spinal cord returned to its normal shape. Histopathological examination confirmed haematoma. Chest pain healed in the early postoperative period. After physical therapy, the patient had complete neurological recovery on postoperative day 14. No urinary dysfunction occurred. Postoperative MRI showed decompression of the spinal cord (Figures 2A and 2B).

Discussion

Patients with SSEH present with severe and acute back-neck or chest pain. These complaints can be attributed to spinal musculoskeletal strains, heart diseases, degenerative spine diseases or lung diseases. With the onset of neurological deficits, such as paraplegia, paraparesia, urinary or anal sphincter dysfunction, spinal abscess, spinal mass and disc herniation should be considered. Some patients have nerve root symptoms with or without spinal cord and nerve root compression. Bilateral or unilateral sensorimotor deficits and the Brown-Sequard syndrome are common[6]. This situation causes confusion in diagnosis. Furthermore, a delayed diagnosis leads to poor prognosis. A meta-analysis study reported that the best neurological recovery occurred in patients who were operated on in the first 12 hours after the onset of symptoms[7]. MRI is the gold standard for early diagnosis[8]. T2-weighted MR images show heterogeneously hyperintense lesions as SSEH. In T1-weighted images, SSEH appears isointense or hypointense. There is no contrast enhancement except hyperacute stage haemorrhage.

The appropriate modality of treatment in SSEH is neural decompression and haematoma evacuation. If necessary, posterior spinal instrumentation can be added for fusion. In a review of 330 SSEH patients, the role of early surgery was

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emphasised[9]. Surgical treatment was recommended within 36 hours for complete spinal cord dysfunction or within 48 hours for incomplete spinal cord dysfunction.

Some authors advocate the spreading theory, which states that haematomas are spread along the epidural space and neural foramens. In this way, neural compression does not occur [5,8]. In a review of the literature, the mean length of the haematoma was found to be significantly higher in patients who undergo conservative treatment. Although this finding supports the spreading theory, it is not conclusive evidence. Haematoma length is not a guide for treatment[8]. Kim et al. reported that they administered anticoagulant and antiplatelet therapy in two patients, but this did not result in improved clinical outcomes[4]. In our case, the patient's neurological condition deteriorated after anticoagulant and antiplatelet treatment for acute coronary syndrome. As the spreading theory was not an appropriate reason for treatment delay with acute paraplegia, we operated on the patient immediately when paraplegia and a sensory deficit below the T4 level occurred. In a case report with SSEH, Sakaguchi et al.[10] emphasised that the antiplatelet drugs given to the patient should be discontinued because of the effect of increased bleeding. In our case, clopidogrel and acetyl salicylic acid were given primarily because acute coronary syndrome was considered. However, when spinal spontaneous epidural bleeding was detected in the patient, treatment was discontinued, as the drugs may contribute to increased bleeding. Chest pain healed in the early postoperative period. After physical therapy, the patient had complete neurological recovery on postoperative day 14.

Conclusion

Patients with SSEH may present with back or chest pain without neurological deficits and trauma, like heart attack. Anticoagulant and antiplatelet therapy for acute coronary syndrome can be administered. Although some authors advocate the spreading theory this therapy can lead to an increase in hematoma, and neural compression as in our case. Although it is rare, SSEH should be kept in mind and early surgical treatment should be performed if neurological deterioration develops.

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Conflict of Interest
The authors declared no potancial conflict of interest.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Diagnosis After a Traffic Accident Aortic Transection

Murat Tepe[1], Fatma Dilan Arslan[1], Ali İhsan Kilci[2], Hakan Hakkoymaz[2], Muhammed Semih Gedik[2]
[1]Acil Tıp, Kilis Devlet Hastanesi, Kilis, Turkey.
[2]Acil Tıp, KSÜ Tıp Fakültesi, Kahramanmaraş, Turkey.

Abstract

Traumatic aortic transection is mostly seen in blunt injury and associated with motor vehicle accidents. 80%-85% of the patients with the aortic transection die at the scene of the incident and the rest of the patients who is delivered to the hospital, have 20% mortality rate. Even with the endovascular repair, these patients have considerably high mortality. On the other hand the diagnosis and the prognosis of aortic transection has been continuously improving. Furthermore the treatment requires a combination of pharmacological and surgical techniques and the endovascular treatment playing a very important role in definitive repair. In this report we present a patient who is admitted to the ER after an in-vehicle car accident. The chest computerised tomography (CT) showed a pseudo aneurysm, measuring approximately 30 mm, in the proximal part of the descending aorta and an appearance consistent with hematoma, measuring approximately 30 mm, and aortic transection was considered. The possibility of aortic injury should always be considered in cases admitted to hospital due to trauma, and if there is any suspicion, necessary examinations should be performed also cardiovascular surgeon should be consulted immediately.

Keywords: Blunt trauma, aortic transection, traffic accident.

Introduction

Aortic transection after blunt traumatic injury is a very rare condition observed in the clinical practice and has a high risk of mortality and morbidity[1]. The underlying mechanism is a tear or damage to the aortic wall due to rapid deceleration of the body with the effect of hitting a solid object[2]. These cases are mostly spotted in car accidents and approximately 80-85% of cases lose their lives at the scene[3]. The location of injury is the aortic isthmus in approximately 85% of patients[1].

In this article, a case of aortic transection diagnosed in a patient with multiple trauma due to a traffic collision was presented.

Case Report

An 18-year-old male patient was admitted to the emergency department by the 112-emergency medical service ambulance due to a traffic collision. The patient had facial bleeding, pain in the midline of the chest, dyspnea, diffuse abdominal pain, right pelvic pain and left knee pain. There were no specific characteristic in the patient's history.

In the physical examination, the patient was conscious, oriented and cooperative, and the Glaskow Coma Score (GCS) was 15. Arterial Blood Pressure was 100/60 mmHg, the heart rate was 73 beats/min, the respiratory rate was 16/min and oxygen saturation was 99%. In the head and neck examination, a linear incision, measuring 6-7 cm was noted on the left side of the forehead. In the thoracic examination, the respiratory sounds were normal and no rhonchus or rale was heard. The first heart sound (S1) and second heart sound (S2) were rhythmic, and no additional sound or murmur was detected. No pathology was detected during the examination of the patient, who stated that he had sternum pain. In the abdominal examination, there was tenderness in the right upper quadrant; however, no rebound or defence was noted. In the extremities, the peripheral pulses were palpable, tenderness was detected on the right hip and on the distal end of left femur. Since the patient was exposed to high-energy trauma, cranial, cervical, thoracic and abdominal CT imaging were requested. Moreover, while the patient was being prepared for tomography, bedside chest radiograph, anterior-posterior pelvis radiography and bilateral radiographs of both femur with portable direct radiography were requested. The chest radiograph showed mediastinal widening (Figure-1), and antero-posterior pelvic radiograph demonstrated fracture on right femoral neck, and left femur

Corresponding Author: Muhammed Semih Gedik
e-mail: semihgedik86@hotmail.com
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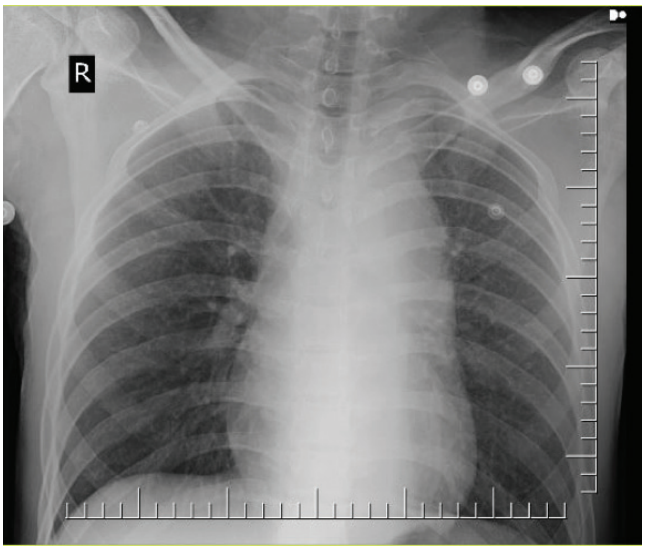

Figure 1. Antero-posterior Chest Radiograph; the axial sections of thoracic computerised tomography showed hematoma around the aorta and transection line.

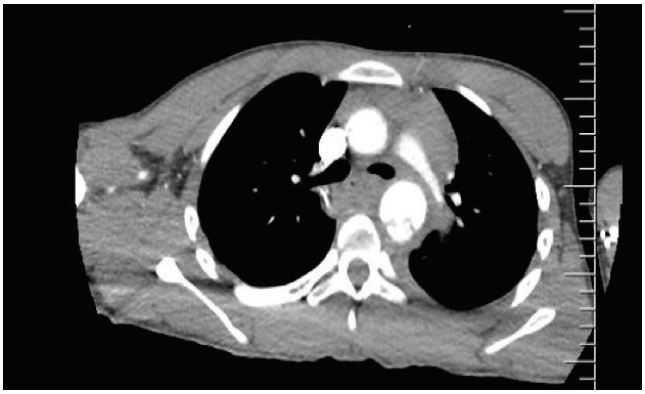

Figure 2. Thoracic CT showed hematoma around the aorta and transection line; the axial sections of thoracic computerised tomography showed hematoma around the aorta and transection line.

radiograph showed fracture in the distal femur. The chest CT showed a pseudoaneurysm, measuring approximately 30 mm, in the proximal part of the descending aorta and an appearance consistent with hematoma, measuring approximately 30 mm, and aortic transection was considered (Figure-2). The abdominal CT demonstrated an image compatible with hematoma, measuring 30 mm in its thickest section, in the paraaortic region, extending to the level of renal artery (Figure-3). In liver segment IV, a hypodense area, measuring 30 mm, consistent with laceration, and in left lower pole, a hypodense lesion and measuring 30x24mm, consistent with contusion were observed. The patient was transferred to the intensive care unit after being consulted with the relevant surgical branches (Figure-4). The patient underwent surgery in the orthopedic clinic for fractures of femur and underwent endovascular surgery for aortic injury and was discharged approximately in the postoperative 1st month.

Discussion

There are many diseases in the differential diagnosis of patients admitted to the emergency department with blunt thoracic trauma. These diseases include cardiac tamponade, pneumothorax, hemothorax, major vessel injury, lung contusion, cardiac contusion and rib fractures[4].

Traumatic aortic transection has a very high mortality rate. The autopsies have been performed, shows that traumatic aortic transaction has been responsible for the %15 of the death sin blunt trauma. The localization of injury is usually in the aortic isthmus; however, it may be rarely observed on the abdominal aorta and distal thoracic aorta[5]. Approximately 80% of aortic transection cases due to trauma die at the scene. They rarely can reach a hospital[6]. This case was also admitted to the emergency ward due to blunt trauma, and the transection was located on the aortic isthmus.

Blunt trauma can affect many organ systems, especially the central nervous system. The patient can be unconscious

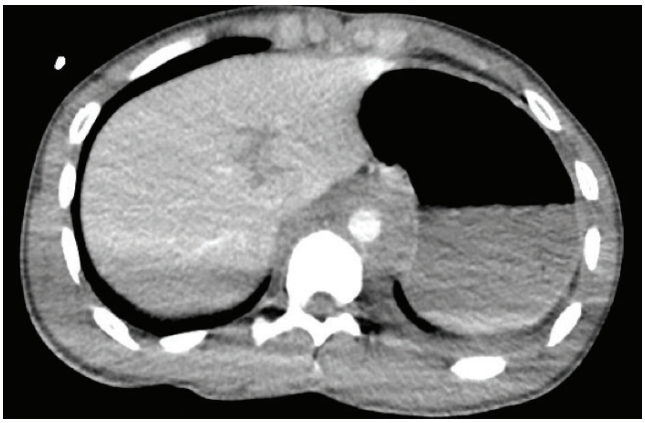

Figure 3. Hematoma around the abdominal aorta; the axial sections of thoracic computerised tomography showed hematoma around the aorta and transection line.

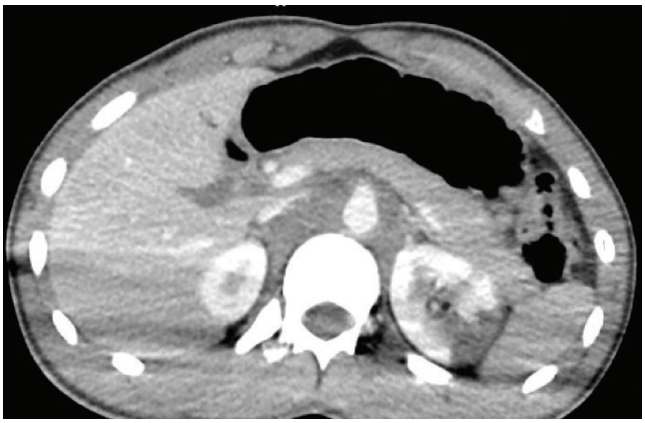

Figure 4. Hypodense lesion compatible with laceration in liver segment 4, and contusion observed in the left kidney; the axial sections of thoracic computerised tomography showed hematoma around the aorta and transection line.

or conscious at the admission. Patients with aortic transection may have chest pain spreading to the neck and back or may have no complaints(2). Other symptoms that may accompany include dyspnea, dysphagia, and extremity pain due to spinal ischemia. Chest radiography, one of the radiological images, may show findings, such as mediastinal widening, effacement in the aorta, and displacement of the trachea, even if it does not provide definite information for the diagnosis. Intravenous contrast-enhanced CT and echocardiography play an important role in diagnosis[7].

A significant proportion of deaths due to aortic transection occur before reaching a hospital. Approximately 18% of the aortic transection cases die before they reach a hospital[8]. Mortality during open surgery can reach up to 23% in aortic transections due to trauma, depending on the condition of other concomitant traumatic disease[9]. In a study, the mortality rate in endovascular repair was found to be 8.6%[8]. There are clinical studies showing that significant reductions in mortality and morbidity rates were achieved in endovascular repair[10]. As this case had serious concomitant disease and was hemodynamically stable, endovascular repair was performed in department of cardiovascular surgery after intensive care follow-up and necessary preparations.

Conclusion

Aortic transection due to blunt chest trauma has serious mortality and morbidity, and many patients pass away before reaching hospital. Therefore, it should be kept in mind as a differential diagnosis for patients who are exposed to multiple trauma. In case of a suspicion of aortic injury, clinical tests, such as computed tomography imaging and echocardiography, should be planned and a cardiovascular surgeon should be consulted.

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Case Report

Journal of Emergency Medicine Case Reports

Do Not Try at Home Alone; Spontaneous Pneumomediastinum Due to Handstand

Emrah Yürek[1], Bedran Aslan[1], İbrahim Altundağ[1], Sinem Doğruyol[1]
[1] Department of Emergency Medicine, Haydarpasa Numune Training and Research Hospital, Istanbul, Turkey.

Abstract

Spontaneous pneumomediastinum (SP) is the accumulation of air in the mediastinum without a surgical or traumatic etiology. It is thought that the basic mechanism triggering SP is the pressure difference that develops after a strong Valsalva maneuver. We presented a 50-year-old male patient was admitted to the emergency department with sudden onset chest and epigastric pain that started after standing up for a handstand. He had atrial fibrillation with rapid ventricular response with a hearth rate of 285 beat/min. Heart rate control was achieved after medical treatment. His computerised tomography was consistent with air in mediastinum. First line treatment was started and the patient was transferred to the thoracic surgery ward. Spontaneous pneumomediastinum is one of the rare diagnoses that should be considered in patients presenting with chest pain and shortness of breath. It is important to correctly evaluate what may be associated with the valsalva maneuver among the sportive movements performed at home, and to keep in mind that SP can also occur in different age groups in order to make the correct diagnosis.

Keywords: Spontaneous pneumomediastinum, pneumomediastinum, valsalva maneuver

Introduction

Spontaneous pneumomediastinum (SP) is the accumulation of air in the mediastinum without a surgical or traumatic etiology (1). Spontaneous pneumomediastinum is a clinical condition that was first described by Louis Hamman in 1939. (2). The main factor in the formation mechanism of SP is the sudden increase of pressure in the intrathoracic cavity (3). As a result, increased intraalveolar pressure causes alveolar rupture and then air leak into the lung interstitium and tracheobronchial tree. It is thought that the basic mechanism triggering SP is the pressure difference that develops after a strong Valsalva maneuver.

Spontaneous pneumomediastinum usually presents with symptoms such as chest pain, neck pain, shortness of breath, and difficulty in swallowing. It is a benign and self-limiting clinical condition. SP is usually seen in the young patient population around 25 years of age (4). Diagnosis process begins with symptoms and physical signs and ends with radiological confirmation of air in the mediastinum (5). The prognosis of patients is generally good and bed rest, oxygen and analgesics are often sufficient for the treatment of patients with SP.

In this case report, we aimed to present a relatively older CP patient with an atypical presentation.

Case Report

A 50-year-old male patient was admitted to the emergency department with sudden onset chest and epigastric pain that started after standing up for a handstand. The patient was diagnosed as atrial fibrillation 20 years ago, but was treated successfully. There is no regular medication usage in his history. In physical examination general condition was fair, he did not have respiratory distress but with a blood pressure of 80/60 mmHg, a heart rate could not be detected and a fever of 36,5°C. Electrocardiogram (ECG) was consistent with atrial fibrillation with rapid ventricular response (A-fib with RVR) with a hearth rate of 285 beat/min (Figure 1). Heart rate control was achieved after rapid intravenous bolus injection of 5 mg of metoprolol (Figure 2). In laboratory examination hemoglobin was 11,4 g/dl, blood ure nitrogen

Corresponding Author: Sinem Dogruyol
e-mail: dogruyolsinem@gmail.com
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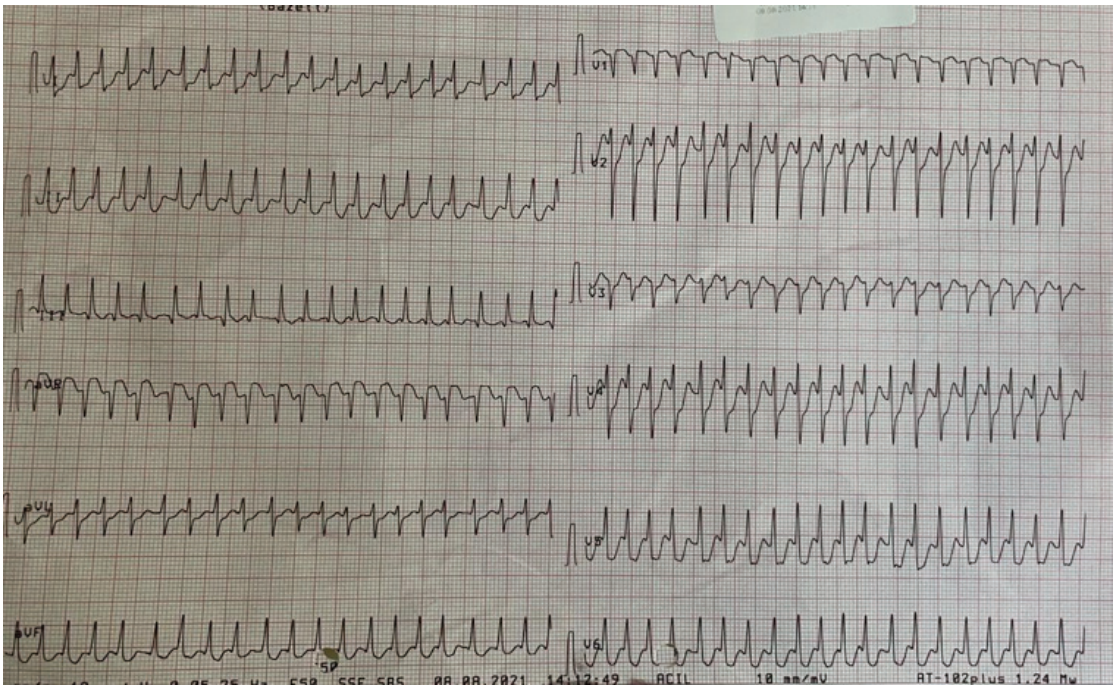

Figure 1. Electrocardiogram of the patient was consistent with atrial fibrillation with rapid ventricular response.

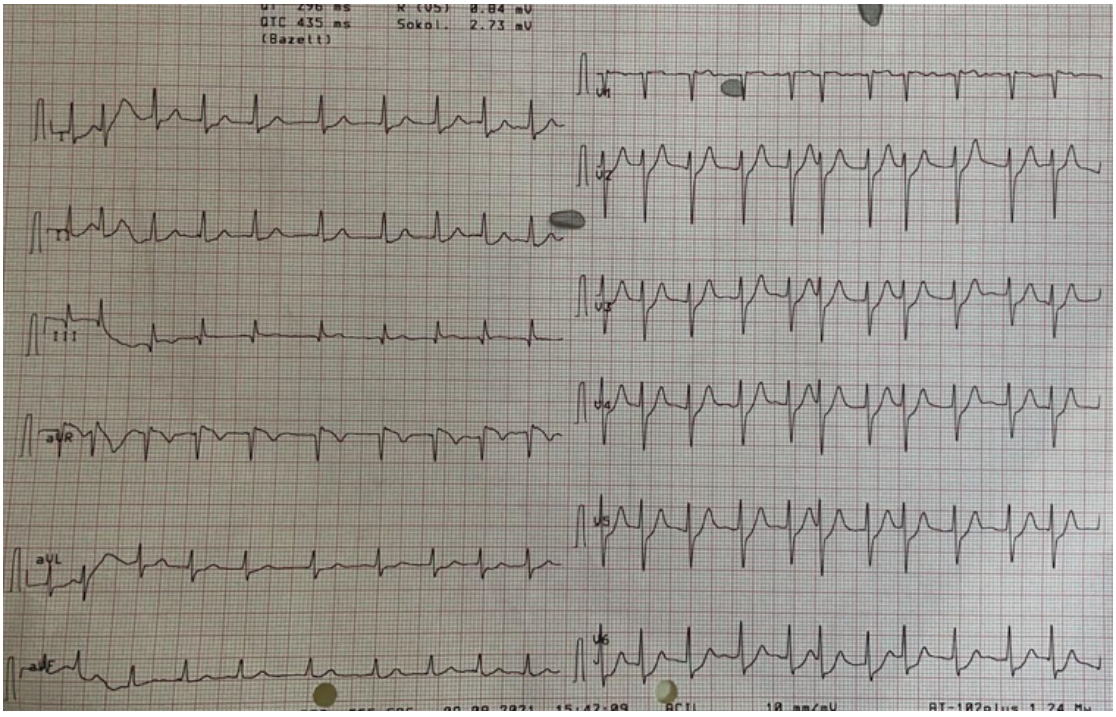

Figure 2. Electrocardiogram of the patient after heart rate control was achieved.

38 mg/dL, creatinine 1.2 mg/dL, sodium 128 mmol/L, potassium 4,3 mmol/L and Troponin I 0.06 ng/m. Contrast-enhanced thorax computerised tomography angiography was planned in the patient's emergency department follow-up to investigate the causes of ongoing chest pain and sudden on-set shortness of breath. Computerised tomography with intravenous contrast was consistent with air in mediastinum (Figure 3). Analgesic medication and oxygen treatment was started in the emergency department and the patient was transferred to the thoracic surgery ward. Acute coronary syndrome was excluded by bedside echocardiography before the patient was transferred to the thoracic surgery ward. The patient did not undergo any surgical intervention and was given oxygen treatment and analgesic medication

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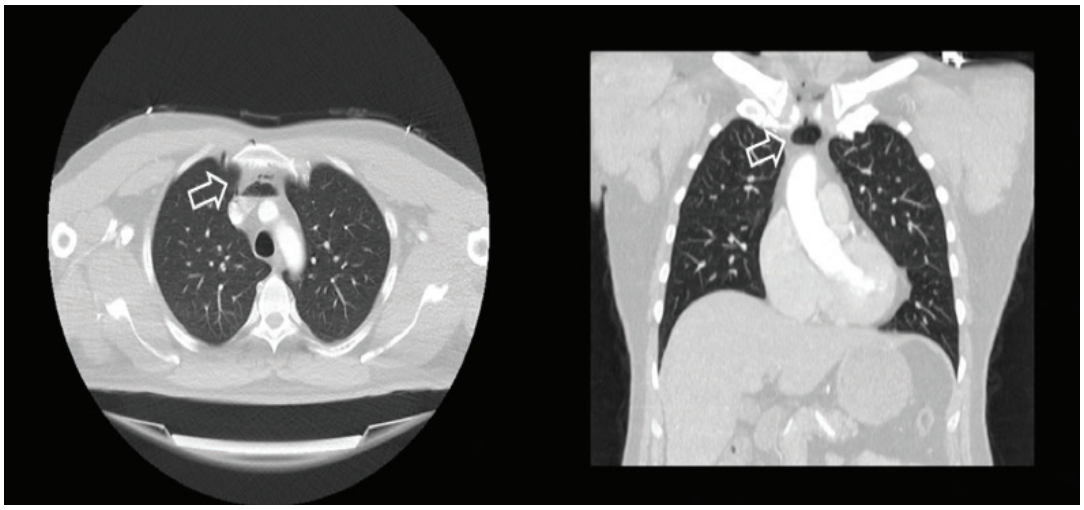

Figure 3. Axial and coronal computerised tomography sections of patient (white arrows show the free air in mediastinum).

in the thoracic surgical ward where he was admitted. The patient was discharged after 7 days.

Discussion

Spontaneous pneumomediastinum is a very rare clinical condition with a prevalence of 0.01%. Conditions that increase intrathoracic pressure come first in SP etiology. Our case described here stated that his complaints started after doing a handstand when he was at home. A case who presented with a similar mechanism has not been described in the literature. Only Lee reported a case of SP developing after running in army trainee (6).

In the literature, it was stated that SP is generally seen in young male patients (7). Considering the age of our patient, it was considerably higher than the classical average age of SP cases.

CP most commonly presents with chest pain and shortness of breath (8). Most cases of SP are related to conditions that result in vigorous Valsalva maneuvers such as strenuous physical activities, coughing, vomiting. Our patient's complaints were consistent with the literature. However, the handstand activity that triggered the strong valsalva causing SP was a rare situation that differed from the literature.

Vital signs of patients with SP are generally stable at the time of admission. Only in malignant pneumomediastinum, which is a rare condition, instability findings due to compression and obstruction on the trachea, esophagus and other mediastinal organs can be detected with excessive air accumulation in the mediastinum (9). Although there was no massive air accumulation in the mediastinum in our patient, his vital status at the time of admission was unstable. However, we think that this situation is related to A-fib with RVR, which responded quickly to medical treatment.

Conclusion

Spontaneous pneumomediastinum is one of the rare diagnoses that should be considered in patients presenting with chest pain and shortness of breath. It is important to correctly evaluate what may be associated with the valsalva maneuver among the sportive movements performed at home, and to keep in mind that SP can also occur in different age groups in order to make the correct diagnosis.

Conflict of interest statement
None of the authors have any conflict to disclose.

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

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Case Report

Journal of Emergency Medicine Case Reports

A Report of Three Cases Presenting to the Emergency Department due to Near-Drowning

Erdal Yavuz[1], Kasım Turgut[1], Umut Gülaçtı[1], İrfan Aydın[1], Ebru Arslan[1], Oğuzhan Özcan[1]
[1] Department of Emergency Medicine, Adiyaman University Medical Faculty, Adiyaman, Turkey.

Abstract

The frequency of drowning increases with the warming of the weather, especially in the summer months. In this paper, the clinical features, radiological imaging and laboratory values of three patients who presented to the emergency department due to near-drowning are discussed comparatively. In Case 1 and 3, advanced life support was applied at the scene and spontaneous circulation was restored. Case 2 presented with the complaint of dyspnea. Cases 1 and 3 died after being admitted to the intensive care unit, while Case 2 was discharged with full recovery. Cardiopulmonary resuscitation performed at the scene is a sign of poor outcome. Diffuse ground-glass opacities were detected in the early thorax computed tomography imaging of all three cases. In addition, there were early increases in laboratory values in all three cases, with the highest increases being observed in aspartate aminotransferase, alanine aminotransferase and creatinine kinase values. This suggests that tissue perfusion disorder develops in the early period.

Keywords: Drowning, emergency department, clinical features, laboratory values

Introduction

The frequency of drowning increases with the warming of the weather, especially in summer months. Near-drowning is defined as immediate survival after being removed from water. In cases of near-drowning, mortality increases as the time in water increases. It is more common in children younger than five years, individuals who drink alcohol and the elderly[1,2]. Hypoxia and acidosis are conditions seen in the early period after near-drowning and therefore treatment should be aimed at ensuring recovery from these conditions3. In this paper, the clinical features, radiological imaging and laboratory values of three patients that presented to the emergency department (ED) due to near-drowning are discussed comparatively.

Case Report

Case 1: A 39-year-old male patient was started on advanced life support at the scene due to the absence of a heartbeat and respiratory failure caused by near-drowning in freshwater, which he had entered to cool off. As a result of the successful advanced life support performed at the scene, in the ambulance and the ED, the heartbeat returned after 30 minutes. The patient was referred to our ED to the requirement of intensive care. It was seen on Electrocardiography (ECG) that the rhythm of the patient, whose first cardiac rhythm was detected as asystole, turned into sinus tachycardia in the emergency room after resuscitation. In the examination of the patient, his Glasgow Coma Scale (GCS) score was 3, pupillary fixation was dilated, there was no spontaneous breathing and endotracheal intubation was present. The patient was examined in the ED and the most increased values according to the reference intervals were determined as aspartate aminotransferase (AST) (630 U/L), alanine aminotransferase (ALT) (409 U/L), lactate (5.6 mmol/L) and creatinine kinase (CK) (1.015 U/L) (Table 1). Diffuse ground-glass opacities were detected in thorax computed tomography (CT) imaging (Figure 1). It was seen that the patient had cerebral edema in the brain CT was taken in the emergency room (Figure 2). The patient was admitted to the intensive care unit and died on the 11th day of hospitalization. It was learned from the patient's history that he did not have any disease and that he did not smoke.

Corresponding Author: Erdal Yavuz
e-mail: erdal_yavuz15@hotmail.com
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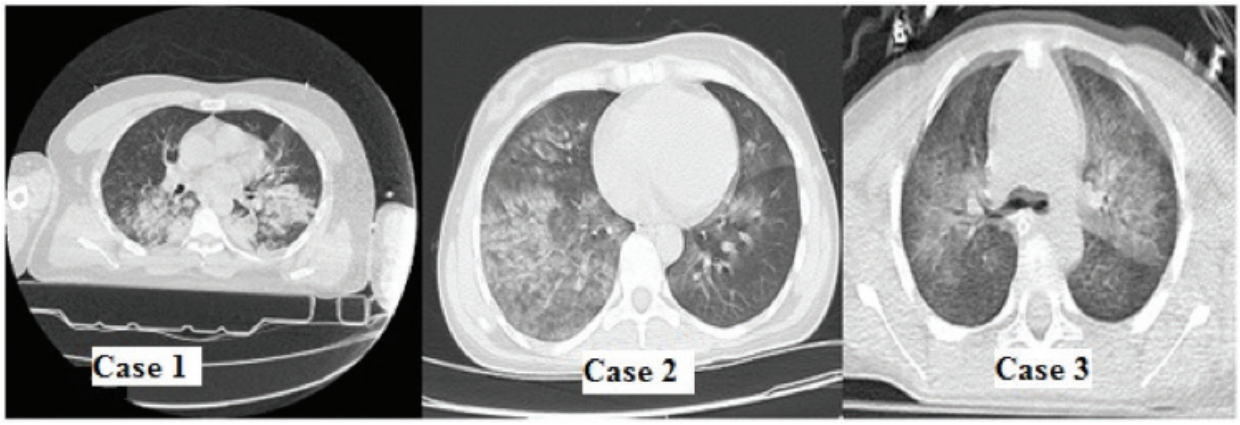

Figure 1. Electrocardiogram of the patient was consistent with atrial fibrillation with rapid ventricular response.

Case 2: A 34-year-old female patient was admitted to the ED due to near-drowning. The patient had complaints of chills, shivering and shortness of breath. Her vital signs were normal. The first ECG of the patient was evaluated as compatible with sinus tachycardia. The patient was started on oxygen support. In her laboratory tests, the highest increases were observed in AST (127 U/L), ALT (120 U/L), lactate (3 mmol/L) and CK (501 U/L) (Table 1). Since the patient was conscious, only thorax CT was got. Diffuse ground-glass opacities were detected in thorax CT imaging (Figure 1). The patient was admitted to the intensive care unit and discharged with full recovery following antibiotic and supportive treatments. The patient was not pregnant and has not no chronic health problem.

Case 3: A five-year-old male patient was given 10-minute advanced life support due to no heartbeat and no pulse at the initial evaluation after near-drowning. The patient's heart rate was recovered and he was transferred to our ED by ambulance. It was seen on ECG that the rhythm of the patient, whose first cardiac rhythm was detected as pulseless electrical activity, turned into sinus tachycardia in the emergency room after resuscitation. At the time of presentation, his GCS score was 3 and he was intubated. His laboratory tests revealed the highest increases in glucose (433 mg/dl), AST (484 U/L), ALT (325 U/L), lactate (16 mmol/L), CK (469 U/L) and amylase (501 U/L) (Table1). Diffuse ground-glass opacities were detected in thorax CT imaging (Figure 1). It was seen that the patient had cerebral edema in the brain CT was taken in the emergency room (Figure 2). The patient was admitted to the intensive care unit and died 30 hours after hospitalization. It was also learned that the patient did not have any health problems before.

Discussion

The main basis of damage caused by drowning in the body is hypoxia4. When hypoxia occurs, oxygen in the blood is directed to vital organs (brain, heart) through physiopathological mechanisms in the body. When hypoxia persists, irreversible damage occurs in cells of the heart and brain. With early intervention, damage to vital organs is minimized[5]. All three of our cases were in danger of drowning in freshwater and early intervention was undertaken because there were witnesses. Spontaneous circulation was restored in two of our cases with effective advanced life support.

Table 1: Laboratory values of the cases at the time of admission to the emergency department

Variables	Case 1	Case 2	Case 3
WBC(×10⁹/L)	6.7	16.6	15.1
Hemoglobin (g/dL)	16.7	10.9	12.4
pH	7.21	7.39	6.83
Lactate (mmol/L)	5,6	3	16
PCO2 (mmHg)	22	36	29
HCO3 (mmol/L)	11.3	21.9	4.6
AST (U/L)	630	127	484
ALT (U/L)	409	120	325
Na (mmol/L)	139	134	137
K (mmol/L)	5.2	3.5	3.2
Cl (mmol/L)	106	104	105
CK (U/L)	1015	501	469
Urea %	46	27	33
Creatinine (mg/dl)	1.55	0.69	0.80
CRP	20.8	8	30
Glucose (mg/dl)	240	97	433
Amylase (U/L)	631	120	501

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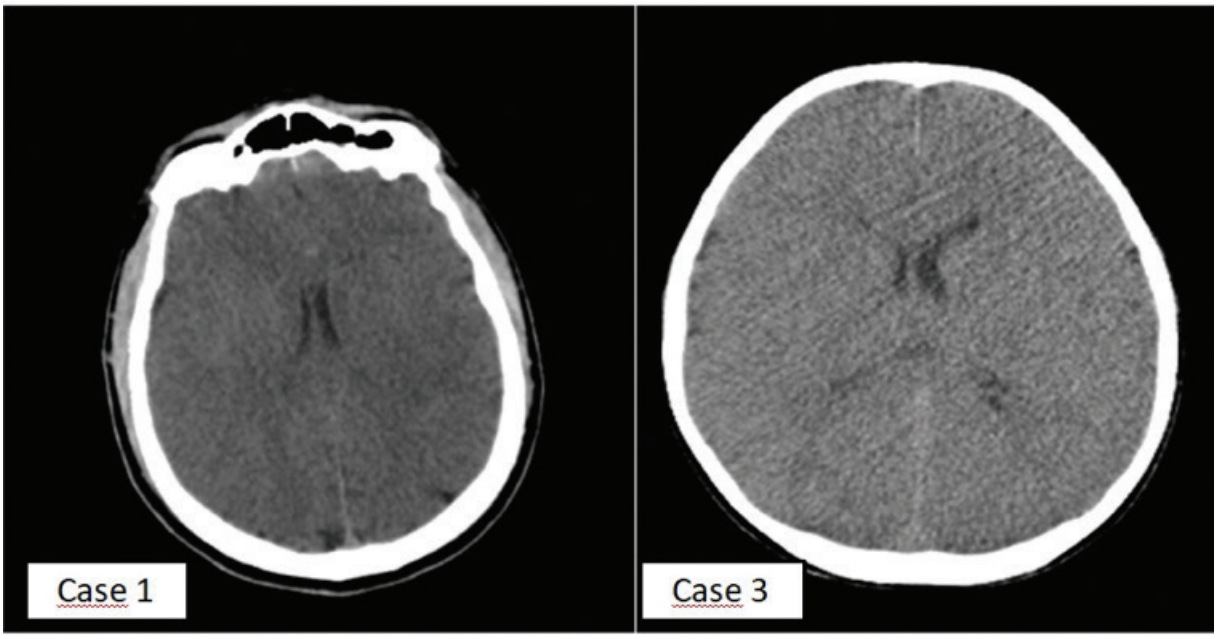

Figure 2. Brain computed tomography images of the two cases presented to the emergency department due to near-drowning.

In the study of Mosaybi et al.[6] on child drowning cases, it was observed that the mortality rate was higher in cases with a pH <7.2 and who underwent cardiopulmonary resuscitation at the scene. Similarly, in our study, cases 1 and 3 who underwent CPR at the scene died, while case 1 without CPR survived. Differently, although the pH was higher than 7.2, Case 1 died. Performing CPR at the scene was considered a more important mortality parameter

Oehmichen et al.[7] stated that electrolyte disturbances were not observed in the majority of patients presenting with near-drowning. Early changes were due to hypoxia and multi-organ damage was detected in four patients. Similarly, in our study, elevated liver enzymes, amylase and creatinine may indicate multi-organ damage.

Blood lactate level is a parameter indicating tissue hypoxia[8]. In the study of Şık et al.[9] the blood lactate level was found to be higher in the group with a high mortality rate. Although it is not correct to make inferences with our 3-case study, it was observed that the blood lactate levels of two patients (cases 1 and 3) who died in our study were higher than those of the surviving patient (case 2).

In the study performed by Nucci-da-Silva et al.[10] it was found that 78.2% of the patients admitted after drowning had cerebral edema in brain MRI scans. In our study, brain edema was observed in cases 1 and 3 on tomography.

Conclusion

The patients who underwent CPR at the scene are more likely to die. It was determined that the values that increased the most in the early period after near-drowning were AST, ALT, lactate and CK in all three cases. This shows that tissue perfusion disorder develops in the early period. High values of these variables may be associated with mortality. In all three of our cases, diffuse ground-glass opacities were present in the early period. There are usually no professional paramedics during the first minutes of intervention in near-drowning cases. Therefore, it would be beneficial for everyone to receive basic life support and first aid training.

Patient consent form - Ethics:

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

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