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Contents

Case Report

Journal of Emergency Medicine Case Reports

Isolated Liver Injury Due to Ground Current Effect of Lightning Strike: The First Case of The Literature

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Abstract

A 26-year-old shepherd man was admitted to the emergency department in June 2020 after ground current effect of lightning strike. With the severity of the trauma, the patient fell to the ground and then fainted. Since the severity of the trauma was not known exactly, there was an open wound in the head, and the patient had dyspnea and elevated liver function tests. Cranial, thorax and abdominal computed tomography (CT) were performed. On CT scan, approximately 7-cm-in length grade 3 liver laceration, extending between segment V and segment VIII was seen without intra-abdominal pathology. The patient was admitted to the intensive care unit (ICU). During the follow-up, liver function tests returned to normal. Liver laceration was managed conservatively. The patient was discharged. On the 6th day of his hospitalization, the patient was discharged without any complications or signs of massive bleeding (the patient's hemoglobin level was 12.4 g/dL before discharge), his vital signs were stable, and his symptoms improved..

Key Words: ground current, isolated, laceration, liver injury, lightning strike

Introduction

Electrical injuries constitute up to 2%–5% of admissions to the burn centers. In such traumas, the excess of electrical voltage directly affects both the severity of trauma and mortality. Among all electrical injuries, the highest mortality group is the lightning strike cases, with 17.6%[1].

Lightning strike is defined as sudden, transient, high-voltage atmospheric electrical discharge. The incidence of lightning fatalities varies depending on region and season; nearly 24,000 lightning related fatalities are reported annually. Lightning can affect people by direct strike, contact injury, side flash, the ground current by pressure or shock wave paths. Also, victims may get injured or die indirectly by reasons such as falling, hit by an object, smoke inhalation, and fire[2]. Penetrating injuries related to a lightning strike caused by shrapnel from the explosion of a nearby structure have also been reported[3]. Lightning strike injuries also have increased morbidity rate because of critical alterations of the circulatory system, respiratory system, and central nervous system. Most lightning-related deaths occur soon after injury due to arrhythmia or respiratory failure[4].

In this study, a case of isolated liver laceration treated conservatively due to ground current effect of lightning strike was presented. Also, this is the first case in the literature in which isolated liver injury occurred after a lightning strike.

Case Report

A 26-year-old shepherd man was admitted to the emergency department of Erzurum Regional Education and Research Hospital, Erzurum, Turkey in June 2020 after lightning strike trauma. The lightning strike occurred about 1 meter away from the patient at the Agri mountain. At the time of the incident, there were no objects near the patient. With the severity of the trauma, the patient fell to the ground and then fainted. The patient did not remember the event after fainting.

Vital signs on admission were the following: blood pressure 115/65 mmHg, heart rate 130 beats per minute, respiratory rate 22 per minutes, and O_2 saturation on room air 88%, body temperature 37.8° Celsius. There was no wound on the body except partial opening near the right temple with headache. Breath sounds were diminished. There was only sinus tachycardia in electrocardiography (ECG). On echocardiography, cardiac functions were normal without tachycardia. There was tenderness and pain on deep palpation in the right upper quadrant.

Pathological laboratory parameters of the patient were as follows: white blood cell count (WBC) 14,100/mm^3, alanine transaminase (ALT) 163 IU/L, aspartate transaminase (AST) 135 IU/L, creatine kinase (CK) 509 U/L and C-reactive protein (CRP) 28 mg/L. In addition, hemoglobin (Hb) level of the patient was 13.6 g/dL. The remaining laboratory parameters were unremarkable.

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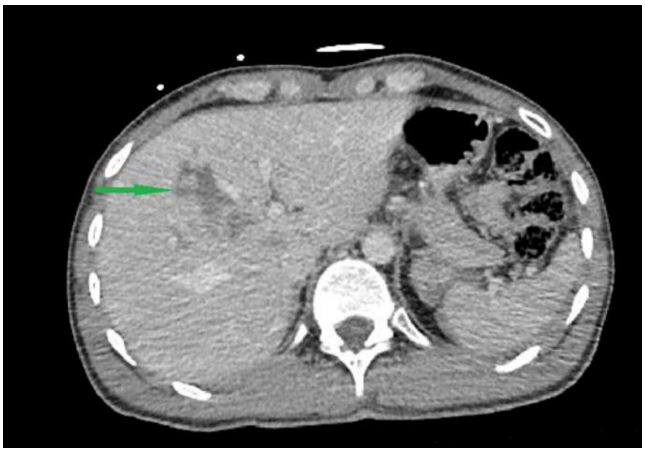

Figure 1. A 26-year-old man who admitted to hospital after lightning injury. Axial image of the CT scan shows approximately 7-cm-in length grade 3 liver laceration, extending between segment V and segment VIII.

Since the severity of the trauma was not known exactly, there was an open wound in the head, and the patient had dyspnea and elevated liver function tests. Cranial, thorax and abdominal computed tomography (CT) were performed. There was no obvious pathology found on brain and thorax CT. On abdominal CT scan, approximately 7-cm-in length grade 3 liver laceration, extending between segment V and segment VIII was seen without intra-abdominal pathology (Figure 1). The patient was admitted to the intensive care unit (ICU) for closely follow-up due to high pressure trauma, cardiac instability, dyspnea, and liver laceration. Supportive oxygen of 2 liters per minute and intravenous 0.9% NaCl hydration were started. The possibility of infection could not be ruled out due to leukocytosis and unreliable history on the initial evaluation, therefore antibiotic treatment of intravenous amoxicillin clavulanic acid (1.2 grams vial twice a day) was started.

During the follow-up, the WBC count, CRP level, creatine kinase level and liver function tests returned to normal (WBC count, 8,100/mm^3; CRP, 3.8 mg/L; AST, 22 U/L; ALT, 28 U/L; creatine kinase, 108 U/L). On the electrocardiography, cardiac rhythms had returned to normal (heartbeat 78 beats per minute). The blood pressure of the patient was 132/72 mmHg. O_2 saturation of the patient on room air increased to 98%. In this case, liver laceration after lightning strike was managed conservatively. The patient was discharged. On the 6th day of his hospitalization, the patient was discharged without any complications or signs of massive bleeding (the patient's hemoglobin level was 12.4 g/dL before discharge), his vital signs were stable, and his symptoms improved. Hemoglobin value of the patient was 12.6 g/dL on the 7th day after discharge. In the control ultrasonography performed on the same day, there was no peri-hepatic and intra-abdominal fluid. There was only intra-parenchymal hematoma of the liver of approximately 5 cm in size.

Discussion

Lightning strike is a frequent occurrence with an estimated 50 occurrences per second and 20% of those resulting in ground strikes. It is important that the clinician has a sound understanding of the injuries that can occur and the proper treatment of lightning-related injuries[5].

Lightning strike carries an enormous amount of energy, the intensity of which exceeds 10 million volts. Lightning strike injuries can be classified into four categories: a direct strike, contact injury, side splash or ground current. Direct strikes occur after directly struck of lightning. Contact injuries occur by touching an object that is struck. Side splash injury happens when the current jumps or "splashes" from a nearby object then following the path of least resistance to an individual. Ground current is the most common mechanism of injury, accounting for one-half, and occurs when lightning strikes an object/ground near an individual and then travels through the ground to an individual[6]. In this patient, trauma mechanism was ground current, and the patient with isolated liver injury was discharged after conservative treatment.

Lightning can be occurred in multiform pattern. Geographic and climate factors play a major role on etiology. Mountainous areas will often experience more lightning strikes than surrounding lower elevation areas. Lightning strikes are more common in rainy seasons such as June, July and August[7]. In addition, approximately 80% of victims are males. Most deaths occur in people between 20 and 45 years old age. Occupation is also an important factor in etiology. Shepherds, woodsmen and military personnel have a tendency to be exposed[8]. Consistent with the literature, the victim of this case report was a young man shepherd who was injured in the mountain in June.

Lightning strike is a rare and serious trauma. Lightning-related injuries can range widely, depending on the injury type and the affected organ[9]. Mortality is reported up to 30% of cases, most frequently causes of mortality are cardiovascular pathologies. Cardiovascular pathologies vary from benign changes to sudden cardiac death. The ST elevation, cardiomyopathy, atrial fibrillation, and QT prolongation may occur[10]. In patients who are unconscious, have head burn, or have abnormal neurologic examination, head CT should be performed because of the increased risk of intracranial hemorrhage. In this case, initial assessment revealed sinus tachycardia. However, the rhythm converted back to normal sinus rhythm without any additional cardiac medication or intervention. Also, the patient had a laceration of head. Therefore, a CT of the head was performed, and no pathology was found on CT.

Injuries may not be visible at first exploration. Possible indirect injuries due to lightning should always be considered. Renal, pulmonary, orthopedic, neurological, dermatological, otologic and ophthalmologic pathologies can also

be seen due to lightning injuries after detailed evaluation of the patient. These pathologies may occur at the time of the event or develop within days or weeks following the event[11]. Multi-disciplinary follow-up and treatment may be required in lightning injuries. However, there was only isolated laceration of liver in this case and multidisciplinary approach was not required.

There are no required laboratory tests or imaging for all patients struck by lightning strike, but certain tests and studies are recommended for selected patients. Patients with a direct strike, chest pain, dyspnea, status post-arrest, should have an ECG and echocardiogram and be monitored on telemetry for 24 hours with other imaging and labs guided by history and physical exam[7]. In this patient, due to cranial trauma, dyspnea, and right upper quadrant pain with increased liver function tests, computed tomography was taken.

Conclusion

In this case report, it is aimed to present the first case of the literature about isolated liver laceration due to ground current effect of lightning strike. In the case of lightning strike, which is a serious and rare trauma, first aid should be given depending on the resuscitation rules. All systems of patients should be evaluated as a whole at hospital. Necessary tests should be performed according to the patient's symptoms and findings at the time of admission. After evaluation with multidisciplinary approach, the necessary treatment should be started quickly. Although a case of isolated liver laceration treated conservatively is presented in our case, it should be kept in mind that surgical treatment will be required in patients who are hemodynamically unstable and have massive decrease in hemoglobin value.

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Case Report

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Unusual Transorbital-Penetrating Intracranial Injury by A Metal Bar: A Case Report

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Abstract

The orbit has a fine bony structure and represents the most vulnerable structure in the cranium. Therefore, penetrating orbital injuries are often associated with traumatic brain injury. Although transorbital-penetrating intracranial injury is not commonly encountered in emergency medicine practice, this occurrence has the potential to cause severe and fatal brain injury. In this study, we report the case of a 21-year-old male patient presented with an injury to his left orbit, caused by falling while holding a metal bar. A local examination revealed a perforating injury at the entry location of a metallic bar (S-shaped) that penetrated his left orbit. CT imaging showed the appearance of a metallic foreign body that entered anterior superior to the left orbit, passed into the left half of the frontal sinus, and extended into the intracranial area. The metallic bar was removed by craniotomy. The patient fully recovered and was discharged on postoperative 9th day. The penetration of foreign bodies through the orbitofrontal region is rare but potentially life-threatening. An emergent surgical initiative is necessary to save the life of the patient. When managed in a timely and efficient manner, the associated mortality and morbidity can be reduced.

Key Words: foreign body, penetration, transorbital, intracranial injury

Introduction

Transorbital-penetrating intracranial injury (TOPI) is rare and considered to represent a severe traumatic head injury. Although TOPI is rarely encountered in general emergency department (ED) practice, and cases that present are predominantly accidental injuries. The morbidity and mortality associated with TOPI remain high, and TOPI can result in severe structural and functional damage. TOPI accounts for 0.04% of all traumatic head injuries[1]. TOPI can be the result of either intentional or unintentional events, including gunshot wounds, stab wounds, and motor vehicle or occupational accidents involving nails, screwdrivers, metal poles, ice picks, keys, pencils, chopsticks, and power drills. TOPI is typically the result of a high-speed injury but can also be caused by comparatively insignificant trauma. The orbit is thinner in children than in adults and consists of bony walls that can be easily fractured even by low-velocity penetrating foreign bodies. Because the frontal bone section of the orbital roof is very thin, foreign bodies will most commonly be observed in this area with a certain penetration angle. This condition often results in frontal lobe damage[2].

Neurological disorders such as intracranial bleeding, central nervous system infections, cerebrospinal fluid leakage, cerebrospinal fluid fistulas, pneumocephalus, orbital cellulitis, carotid-cavernous sinus fistula and vascular complications may develop in patients as a result of penetrating head trauma[3]. The prevalence of vascular complications following TOPI has been reported as high as 50%, and this complication can be life-threatening[4]. Surgical treatment is the primary strategy for this type of injury. The mortality rate for TOPI is 33% when timely surgical treatments are applied but increases to 53% in cases where surgery is delayed[5].

In this article, we present a case report of a penetrating, metal, orbitocranial foreign body in a young male patient. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Case Report

A 21-year-old male patient presented with an injury to his left orbit, caused by falling while holding a metal bar. Local examination revealed a perforating injury at the entry location of a metallic bar (S-shaped) that penetrated his left orbit (Figure 1). Neurological examination revealed a Glasgow Coma Scale of 15/15, and the patient had normal bilateral vision. His left pupil was miotic, reactive to light, anterior segment of cornea intact, and eye movement was normal. Also, no lacerations were found in the sclera and conjunctiva. The examination of the cranial nerves was normal. No other motor or sensory deficits were observed, and the patient's vital signs were within normal ranges.

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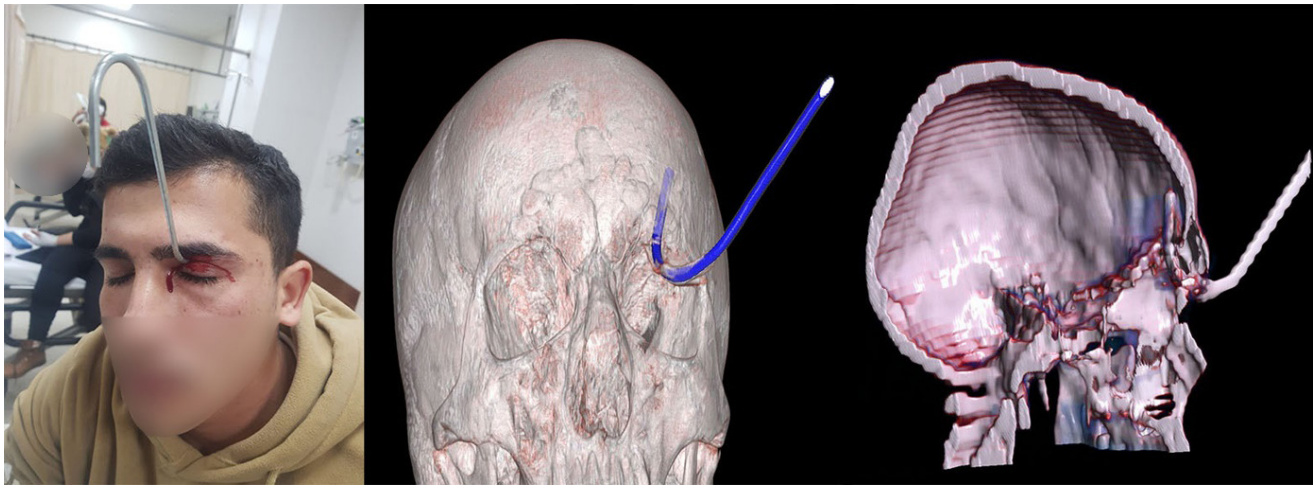

Figure-1: Photograph and a 3-dimensional CT images of the patient who has a perforating injury at the entry location of a metallic bar (S-shaped) that penetrated his left orbit.

Non-contrast computed tomography (CT) and a 3-dimensional (3D) CT scan were performed on the patient because the foreign body extended toward the cranial cavity. CT images revealed a metallic foreign body in the left parietal region (Figure 2). CT and 3D CT imaging showed the appearance of a metallic foreign body that entered anterior superior to the left orbit, passed into the left half of the frontal sinus, and extended into the intracranial area (Figure 1-2). Although the evaluation provided by this imaging sequence was suboptimal due to artifacts introduced by the foreign body, the integrity of the globe and optic nerve appeared to be preserved. The tip of the foreign body extended into the frontal lobe parenchyma, and pneumocephalus was observed near the tip (Figure 2).

In this case, the first approach involved the maintenance of airway, breathing, and circulation, with local hemostasis.

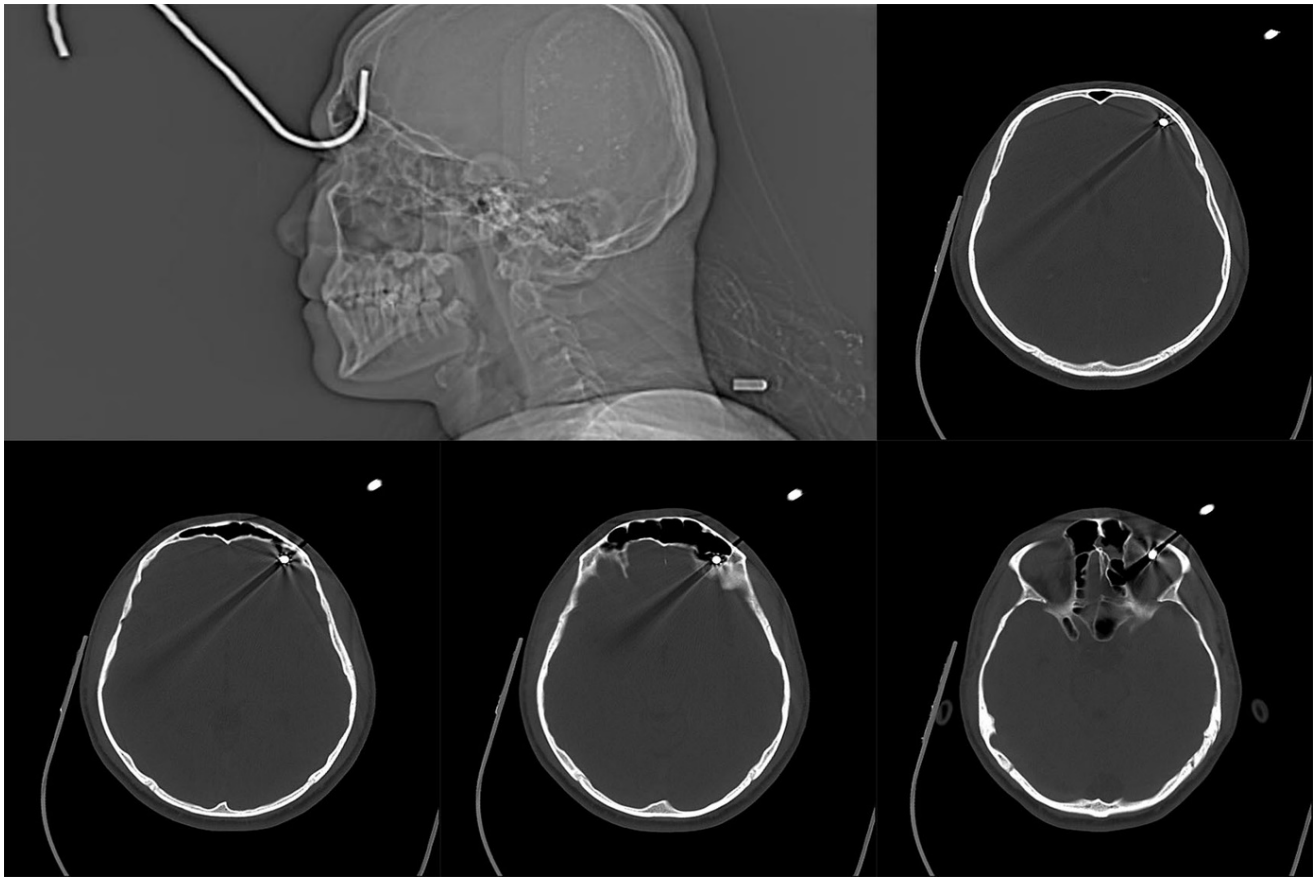

Figure-2: Non-contrast CT scan of head in axial plane showing pneumocephalus and the foreign body.

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Broad-spectrum antibiotic prophylaxis and tetanus vaccination were administered immediately. A consult with neurosurgeons and ophthalmologists was performed, as a result of the evaluations made, the metallic bar was removed by craniotomy by neurosurgeons. The patient fully recovered and was discharged on postoperative 9th day.

Discussion

Although TOPIs are not common, they can cause severe brain damage and significant ophthalmic and neurological disabilities. Therefore, the management of such cases needs a multidisciplinary approach involving both neurosurgeries as well as ophthalmology departments[5]. Previous reports have been published describing intracranial penetration via the orbit with various types of foreign bodies. Penetrating orbitofrontal foreign bodies is more common in males than in females and in younger people than in older people. The management and prognosis of TOPI depend on the composition and localization of the foreign body and the presence of secondary infections.

In TOPI cases, pathophysiology depends on tracing the trajectory of the foreign body through the skull. In these injuries, intracerebral hematoma, cerebral contusion, intraventricular hemorrhage, pneumocephalus, and brain stem damage can be considered immediate complications. Some of these injuries can extend through the cerebellum and brain stem. Penetrating injuries through the superior orbital fissure may affect the III, IV, V, and VI cranial nerves, the arteries of Willis circle, and the carotid artery[6]. The orbit, which is shaped like a quadrangular pyramid, is directed to certain anatomical regions of the intracranial cavity with penetrating foreign bodies. The degree of neurological damage that occurs is associated with the orbital bone anatomy, in addition to the size, shape, and trajectory of the foreign body. Foreign bodies can access the cranial cavity via various routes. Foreign bodies typically penetrate the orbit from the medial canthus, passing through the optic canal and superior orbital fissure before lodging in the ipsilateral or contralateral side of the cranium[7]. Intracranial penetration can occur through the orbital roof due to the fragile structure of the superior orbital fissure of the frontal bone, often resulting in frontal lobe damage[2].

In our case, the metal bar entered anterior superior of the left orbit and passed through the left half of the frontal sinus, extending intracranially. Therefore, our case is an example of injuries in which penetration occurs through the superior orbital fissure. Diagnostic methods that can be used to assess the detection and localization of penetrating orbitofrontal foreign bodies include plain radiographs, ultrasonography (USG), CT, and magnetic resonance imaging (MRI). CT is excellent for identifying high-density impurities such as metal or glass but is not suitable for similarly sized organic objects. MRI allows for accurate localization and provides a better separation of organic foreign bodies from soft tissue but is contraindicated in the presence of a ferromagnetic foreign body[8]. USG has shown promising results, especially in the detection of radiolucent foreign bodies, and its sensitivity of 95% for detecting foreign bodies. However, the biggest disadvantage of USG is the imaging of the air[9]. CT scan is useful and can readily be applied to determine the extent of intraparenchymal injury, identify bony defects in the skull, and locate the foreign body, allowing for the location, position, size, and shape of the foreign body to be accurately reproduced[10]. CT and 3D CT were performed on our patient to provide a detailed analysis of the bone pathology and determine the position and trajectory of the foreign body involved. In this case, CT revealed that the metal bar passed through the roof of the left orbital toward the ipsilateral frontal lobe. In our case, parenchymal damage extending into the frontal lobe and pneumocephalus was observed due to the transorbital penetration of the foreign body.

The most appropriate management of transorbital penetration cases that present to the ED is the retention of the transorbital object *in situ* and the careful transfer of the patient to the surgical department. Metal bars with extracranial components should be carefully surgically removed, and broad-spectrum antibiotic treatment should be initiated. If not treated promptly, these injuries can result in serious neurological damage or even death. Treatment aims to minimize brain damage and prevent mortality by controlling bleeding through early surgical intervention. In our case, because the neurological and radiological investigations showed no vascular injuries, the foreign body was surgically removed. An intensive antibiotic treatment regimen is recommended during both the perioperative and postoperative periods to prevent late infections, which were also applied in our case. Antiepileptic drugs are also recommended during the early stages after injury to prevent seizures and were applied in our case. In our case, during the acute period and in the follow-up evaluations performed one month later, no complications, neurological deficits, or mortality were observed.

In conclusion, the penetration of foreign bodies through the orbitofrontal region is rare but potentially life-threatening. If not treated immediately, TOPIs can result in critical neurological damage or even death. An emergent surgical initiative is necessary to save the life of the patient. We would like to emphasize that radiological imaging is important for both diagnosis and planning appropriate surgical intervention, and patient disability and mortality rates will decrease when a multidisciplinary approach is used.

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Case Report

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COVID-19 Cases with Acute Necrotizing Pancreatitis

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Abstract

COVID-19 is a multisystemic condition that presents with different signs and symptoms. Studies conducted have shown that it shows its effect on different tissues through Angiotensin Converting Enzyme 2 receptors. In particular, gastrointestinal system symptoms have started to attract more attention. We present two COVID-19 cases, aged 82 and 70, presenting with acute pancreatitis without any other risk factors. An 82-year-old female patient had a fatal outcome despite the treatment, while a 70-year-old female patient was discharged for outpatient follow-up. While COVID-19 patients are typically admitted with symptoms of respiratory system, some patients may have symptoms such as nausea-vomiting, diarrhoea and abdominal pain.

Key Words: covid-19, pancreatitis, necrotizing pancreatitis

Introduction

SARS-COV-2 is a virus that poses a threat to global public health by causing a pandemic as a result of rapid transmission. The agent often presents with the pathology it creates in the respiratory system[1]. However, it also presents with different symptoms by affecting many systems such as gastrointestinal, hepatobiliary and pancreas[2]. Two patients whose COVID-19 RT-PCR tests were positive, who were admitted with complaints of abdominal pain, nausea and vomiting and who were diagnosed with acute pancreatitis are presented in this study.

Case Report

Case-1: 82-year-old female patient was admitted to the emergency service as a result of vomiting in addition to fever and abdominal pain that had been going on for three days. The patient's anamnesis included hypertension and cholecystectomy five months ago. Physical examination showed that she was tachycardic (121/minutes), hypoxic (pulse oxymeter 85% in room temperature), she had 38.3 C fever and bilateral rales in lower zones of both hemithorax. In abdominal examination, it was found with deep palpation that she had tenderness and defense in all quadrants, especially in the epigastric region. Symptomatic treatment for fever and abdominal pain was started and laboratory and imaging tests were requested. Emergency laboratory tests showed 19580/uL leukocyte count, dominated by neutrophil (18320/uL), albumin was found as 29mg/dL and CRP was found as 223.5 mg/L (Table-1). Thoracic and abdominal computed tomography taken to explain the current clinical picture of the patient showed consolidated area containing air bronchograms at the level of the left lung lower lobe posterobasal segment, pleura effusion in both hemithoraxes (Figure-1a), dirty appearance and free fluid in peripancreatic localization in and around choledochus, hypodense air values around the pancreas and heterogeneous density areas (Figure-1b). Since it is the pandemic period, the thoracic findings were not excluded and interpreted as COVID-19 disease although the patient did not have contact history. Nasopharyngeal swab sample was taken and RT-PCR test was sent. Although the patient's laboratory findings were not supporting pancreatitis, clinical and image findings belonged to pancreatitis. Since the RT-PCR test in the emergency service gives result within 12 hours, the patient was isolated with a pre-diagnosis of acute pancreatitis and monitored in the internal diseases clinic. When the RT-PCR test result was found as positive, hydroxychloroquine, oseltamivir phosphate, azithromycin and oral intake for acute pancreatitis were discontinued and fluid infusion, imipenem/cilastatin sodium treatment was started. During follow-up, acute hypoxic respiratory failure requiring high flow oxygen therapy and sepsis due to emphysematous pancreatitis developed. As a result of the increase and advancement in gastrointestinal and pulmonary symptoms, the patient died on day 58 of the follow-up.

Case-2: 70-year-old female patient was admitted to the emergency service due to abdominal pain radiating to the

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Table-1: Laboratory results on admission

Laboratory Tests	Level	Normal range
WBC count (x10^3/uL)	19.58	4-10 x 10^3/uL
Neutrophil (x10^3/uL)	18.32	2-7 x 10^3/uL
Lymphocyte (x10^3/uL)	0.88	0.8-4 x10^3/uL
RBC count (x10^6/uL)	3.90	3.5-5.5 x 10^6/uL
Hemoglobin (g/dL)	12.2	11-16 g/dL
Platelet count (x10^3/uL)	173	100-400 x 10^3/uL
Hemotocrit (%)	34.6	37-54 %
AST (U/L)	17	0-35 U/L
ALT (U/L)	16	0-35 U/L
Albumin (g/L)	29	35-42 g/L
Amylase (U/L)	93	28-100 U/L
Blood sodium level (mmol/L)	133	136-146 mmol/L
Blood potassium level (mmol/L)	3.34	3.5-5.1 mmol/L
Blood calcium level (mg/dL)	7.5	8.8-10.6 mg/dL
Blood Urea Nitrogen ((mg/dL)	38	17-43 mg/dL
Creatinine (mg/dL)	0.68	0.51-0.95 mg/dL
CRP (mg/L)	223.5	0-5 mg/L

back, nausea, vomiting and shortness of breath that had been going on for two days. The patient's anamnesis showed that she had hypertension, coronary artery disease and congestive heart failure. Physical examination showed that her vital findings were stable and there were occasional rales in both hemithorax lower zones. Abdominal examination showed tenderness and defense with palpation in epigastric region. Regarding the symptoms, oral intake was stopped, fluid infusion and analgesic treatment were started and tests were requested. Laboratory tests in the emergency service showed 11430 /uL leukocyte count, amylase 1403 /u Land CRP 0.6 mg/L (Table-2). Thoracic and abdominal computed tomography showed mosaic attenuation in bilateral lungs, subsegmental atelectasis in the left lung lower lobe and several ground-glass density nodules in the lower lobe posterior segment (Figure-2a), while fluid and inflammatory density increase were seen in the peripancreatic region (Figure-2b). Although the patient did not have a contact history, her existing thoracic imaging findings supported COVID-19 disease due to being in the pandemic period. For this reason, RT-PCR test was taken from nasopharyngeal swab sample. Because the test result would be taken in 12 hours, she was followed in the internal medicine clinic with a diagnosis of acute pancreatitis. Since the result was positive, imaging test

Table-2: Laboratory results on admission.

Laboratory Tests	Level	Normal range
WBC count (x10^3/uL)	11.43 x 10^3/uL	4-10 x 10^3/uL
Neutrophil (x10^3/uL)	6.67 x 10^3/uL	2-7 x 10^3/uL
Lymphocyte (x10^3/uL)	3.94 x 10^3/uL	0.8-4 x10^3/uL
RBC count (x10^6/uL)	4.18 x 10^6/uL	3.5-5.5 x 10^6/uL
Hemoglobin (g/dL)	13.1 g/dL	11-16 g/dL
Platelet count (x10^3/uL)	276 x 10^3/uL	100-400 x 10^3/uL
Hemotocrit (%)	38.5 %	37-54 %
AST (U/L)	40 U/L	0-35 U/L
ALT (U/L)	21 U/L	0-35 U/L
Albumin (g/L)	43 g/L	35-42 g/L
Amylase (U/L)	1403 U/L	28-100 U/L
Blood sodium level (mmol/L)	136 mmol/L	136-146 mmol/L
Blood potassium level (mmol/L)	3.95 mmol/L	3.5-5.1 mmol/L
Blood calcium level (mg/dL)	9.6 mg/dL	8.8-10.6 mg/dL
Blood Urea Nitrogen (mg/dL)	59 mg/dL	17-43 mg/dL
Creatinine (mg/dL)	1.20 mg/dL	0.51-0.95 mg/dL
CRP (mg/L)	0.6 mg/L	0-5 mg/L

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Case-1

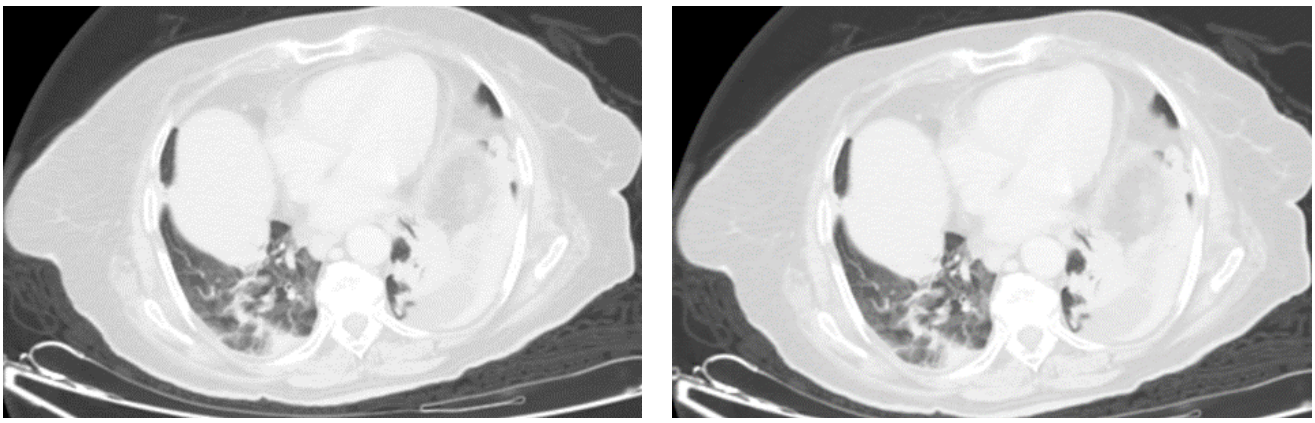

Figure 1. Thoracic CT showed concoliated area containing air bronchograms at the level of the left lung lower lobe posterobazal segment, band consolidated/atelectasis area at the level of the right lung lower lobe posterobazal segment and pleura effusion in both hemitoraxes.

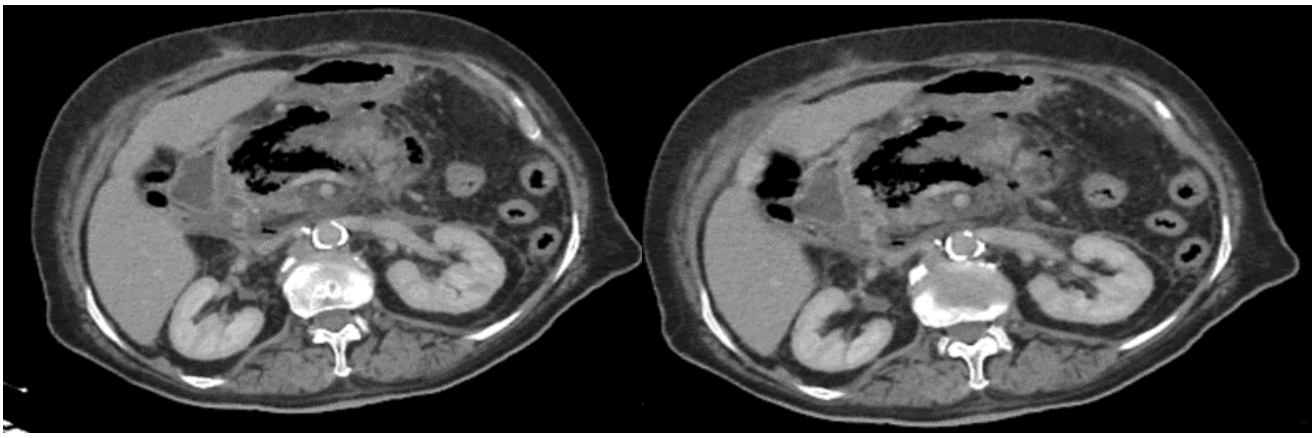

Figure-1b: Abdominal CT showed dirty appearance and freefluid in peripancreatic localization in and around choledochus, hypodense air values around the pancreas and heterogeneous density areas.

supported COVID-19 disease and there was an increase in shortness of breath, she was treated with oxygen, hydrocyclorocin, fluid resuscitation and empiric antibiotics. Supportive treatment was continued during the follow-ups and she was discharged on day 7 since the signs and symptoms decreased gradually.

Discussion

In this study, we saw that COVID-19 pneumonia occurred with pancreatitis in two patients who were admitted with symptoms of the gastrointestinal system. Coronaviruses are in the single stranded RNA virus family, which are highly contagious and which cause many different symptoms[3]. Viral pathogen has cellular entrance through Angiotensin Converting Enzyme 2 receptors (ACE2) which are expressed by gastrointestinal system and epithelial cells of the blood veins[4]. This is supported by studies which show that the agent exerts its effect especially in the liver and pancreases through this receptor[5, 6].

Systematic inflammatory and immune responses with the cytopathic effects caused by the virus lead to enzyme abnormalities with pancreatic damage[5]. In this study, absence of other etiological causes in addition to the temporal relationship between pancreatitis and COVID-19 in both cases suggests that they may result from the cytopathic effect caused by coronavirus. In addition, coexistence of pancreatic damage and pneumonia is an indicator of the systemic inflammatory effect caused by SARS-CoV-2 infection in the immune system and that several organ systems may be influenced[7].

As a conclusion, while COVID-19 patients are typically admitted with symptoms of respiratory system, some patients may have symptoms such as nausea-vomiting, diarrhoea and abdominal pain[2]. Knowing about this is important for clinicians and it can prevent neglecting other systems. However, further studies are needed for a better understanding of the effects of this virus.

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Case-2

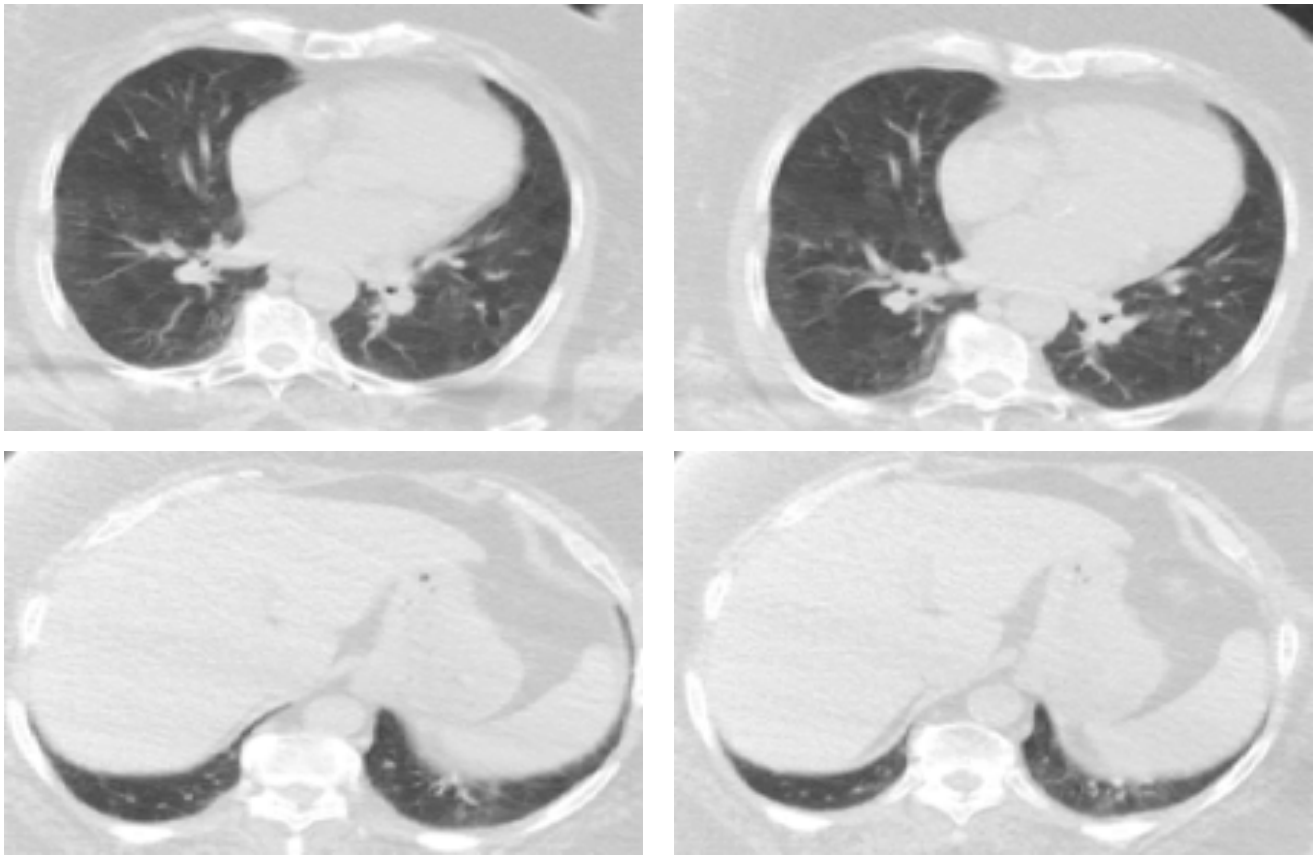

Figure-2a: Thoracic CT showed mosaic attenuation in bilateral lungs, subsegmental atelectasis in the left lung lower lobe and several ground-glass density nodules in the lower lobe posterior segment.

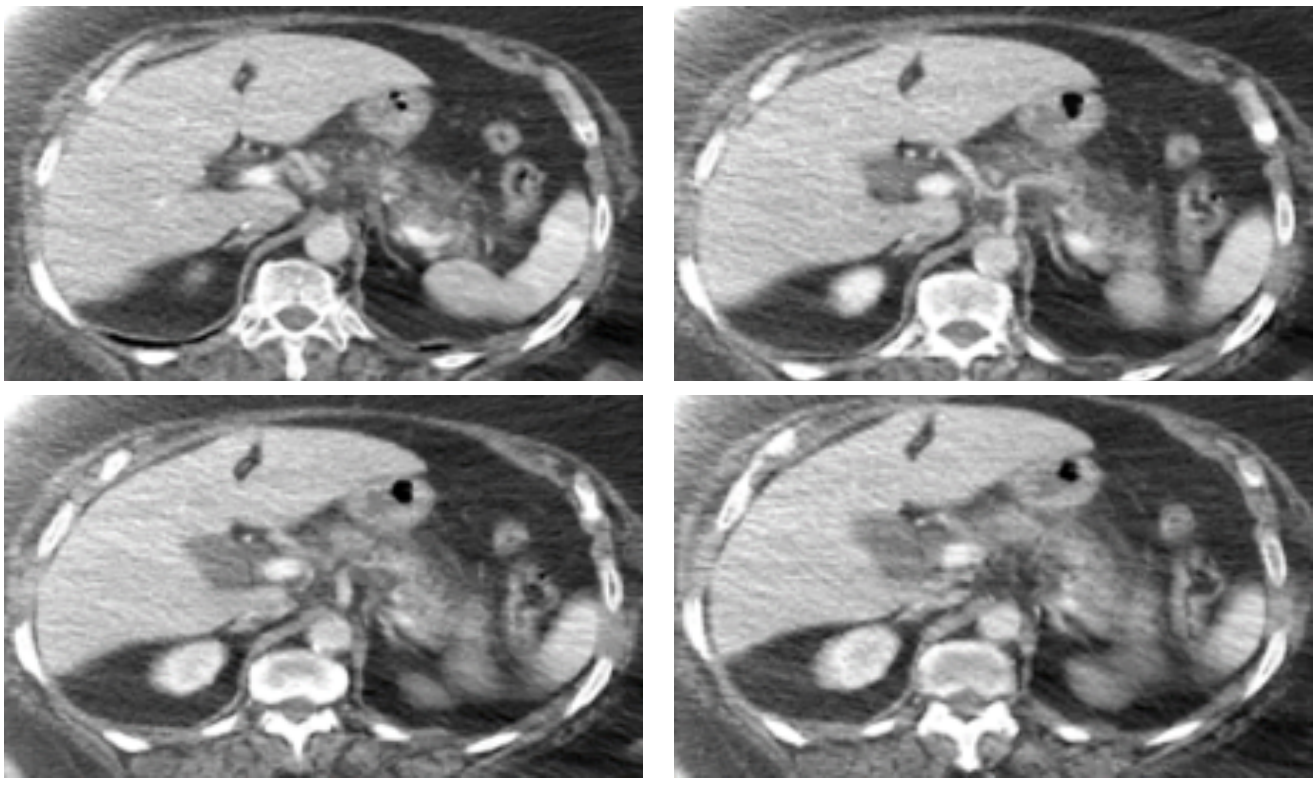

Figure-2b: Abdominal CT showed fluid and inflammatory density increase were seen in the peripancreatic region.

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Case Report

Journal of Emergency Medicine Case Reports

Spontaneous Coronary Artery Dissection in The Left Main Coronary Artery: A Case Report

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Abstract

Spontaneous coronary artery dissection (SCAD) can be defined as epicardial coronary artery dissection, which is not associated with trauma, atherosclerosis, or iatrogenesis. It is known that its incidence increases in the presence of some risk factors. In this case report; We wanted to present a case of a young-middle-aged female patient who had no risk factors other than a history of drug use, spontaneous left main coronary artery dissection was detected in coronary angiography and percutaneous coronary intervention was applied in the treatment. With typical chest pain, SCAD should be considered in young women with suspected myocardial infarction. Revascularization strategies may be preferred instead of conservative treatment in the high-risk patient group.

Key Words: spontaneous dissection, coronary artery dissection, left main coronary artery

Introduction

Spontaneous coronary artery dissection (SCAD) can be defined as epicardial coronary artery dissection, which is not associated with trauma, atherosclerosis, or iatrogenesis. It is a rare cause of acute coronary syndrome (ACS). SCAD is considered a rare disease of the coronary arteries and occurs largely as myocardial infarction (MI) in young women. Impaired coronary artery circulation develops due to myocardial injury, intramural hematoma formation, and/or intimal impairment during SCAD[1]. Risk factors that predispose patients to SCAD include fibromuscular dysplasia, systemic inflammatory conditions (systemic lupus erythematosus, rheumatoid arthritis), connective tissue diseases (Marfan, Ehler-Danlos), postpartum condition, and multiparity[2]. Epidemiologically, SCAD accounts for 0.1% to 4% of all ACS cases in the United States[3]. Further analysis indicates that approximately 25% of ACS cases in women under 50 years of age are caused by SCAD[4]. It has also been shown to be increasingly common in older and postmenopausal women even though it is typically thought to affect young women between the ages of 43-52 (± 10)[4, 5]. Overall, it is difficult to measure the actual incidence of SCAD because this disease is often inadequate and/or misdiagnosed.

Patients with SCAD typically present with ACS symptoms and chest pain is observed in 96% of cases. Other symptoms include arm, neck, or jaw pain, nausea/vomiting, and sweating[5]. Most commonly, the left anterior descending (LAD) coronary artery is affected and accounts for 40-70% of cases[6].

We identified a young to middle-aged female patient with no risk factors other than a history of drug use, presenting with spontaneous left main coronary artery dissection, and undergoing percutaneous coronary intervention treatment due to high-risk characteristics in this case report.

Case Report

A 46-year-old female patient had chest pain that started 3 hours before admission to the emergency department. The pain was in the retrosternal region, spreading to the left arm. She described it as eight out of ten. Fever was 36.5 degrees, respiratory rate was 20/min, blood pressure was 90/60 mm/Hg, SpO2 was 98%. Lung respiration sounds were normal and heart sounds were rhythmic and normal on physical examination. ECG taken revealed T wave negativity in sinus rhythm, heart rate 85/min, V1-V6 derivations. There was mild hypokinesis in the anterior and apical wall on the echocardiography image. There was mild insufficiency in the mitral valve. There was a history of oestrogen and progesterone-containing tablet use due to dysmenorrhea. She had been smoking for approximately 20 years. There was no family history, no pregnancy, and no chronic disease history. No significant pathology was detected in biochemical and hemogram parameters. An approximately 25-fold increase in troponin level was observed in the emergency department (1195 ng/L [determined reference range 0-47 ng/L]).

The patient was admitted to the coronary angiography catheter laboratory with the diagnosis of non-ST elevation

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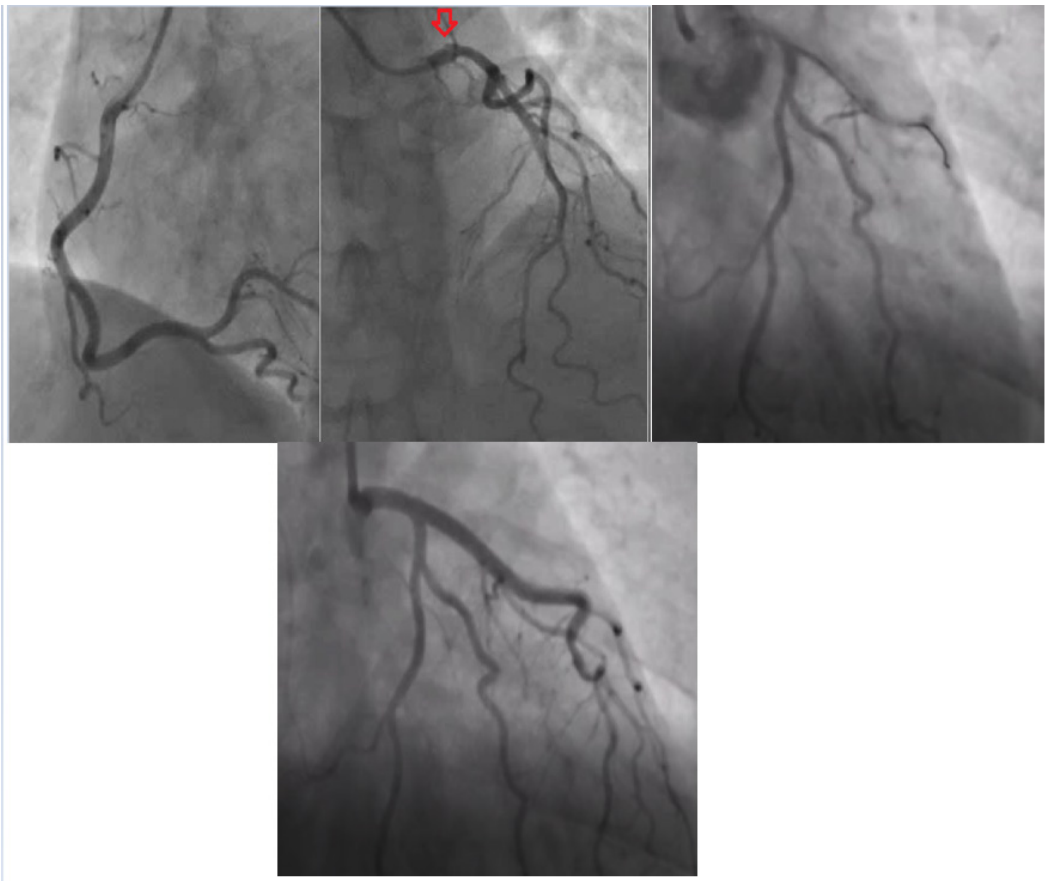

Figure-1: Coronary angiography images of the patient

myocardial infarction. Coronary angiography revealed no pathology in the right coronary artery (RCA). Dissection appearance consistent with type 1 SCAD was observed in the left main coronary artery (LMCA). No pathology was observed in the left circumflex artery. The patient was defibrillated upon the development of VT and VF, and 200 mg amiodarone intravenous push was administered during the imaging process. Sinus rhythm was achieved. Afterward, it was observed that the dissection line advanced to the mid region of the LAD artery. It was lowered to the actual lumen with the help of a guidewire upon seeing TIMI 1 flow in LAD. A wire was sent to the Cx artery for protection. The LAD artery was predilated with 2.0*10 mm and 1.20*15 mm diameter balloons starting from the midsection. LMCA was implanted under 3.0*36 mm DES 16 atm P starting from the ostial to close the dissection line in the LAD artery. 2.75*18 mm and 2.5*24 mm DES were implanted to overlap each other, respectively, upon the continuation of the dissection line in the midsection of the LAD. The hemodynamics of the patient improved after the procedure. Intracoronary abciximab was administered due to thrombus in the Cx ostial region. Afterward, abciximab infusion, acetylsalicylic acid, ticagrelor, heparin was administered and taken to the coronary intensive care unit. The patient, who was stable in follow-up, was discharged from the hospital on the 4th day of hospitalization (Figure-1, Video).

Discussion

SCAD is an important cause of acute coronary syndrome in women[7]. SCAD is usually seen in ≤50 young women, some studies have found the mean age between 43 and 52 years[8-10]. The patient was a middle-aged female in our case. Risk factors other than female gender may include pregnancy and postpartum period. In addition, hormone treatments are also a risk factor, as in the case we reported. The underlying arteriopathies contribute to the risk of SCAD in systemic inflammatory diseases[11]. There was no such risk factor in the case we presented. 3 types of SCAD were identified angiographically. It is called type-1 if there is a visible dissection flap and linear filling defect, type-2a in the presence of a flat stenosis that can be limited to normal segments, type-2b if it extends to the end of the coronary artery, and type-3 if it mimics an atherosclerotic lesion. We evaluated it as type-1 SCAD in the first image we obtained in our case. Tortuous vascular structure is also a potential risk factor for SCAD[12]. Intracoronary additional imaging techniques such as IVUS and OCT can be used in cases where the diagnosis of SCAD is uncertain. Especially OCT is important in terms of providing us with clear images. However, it can lead to the dissection line progression since a serious contrast load is also required during OCT. IVUS is commonly used since it does not require contrast in this respect[7].

A relationship was found between SCAD and tortuous coronary artery. Tortuous coronary arteries are characterized by a curvature of 90-180 degrees in 3 consecutive regions seen at the end of the diastolic in major coronary arteries ≥2 mm in diameter[13]. In our case, there was no tortuous vascular structure. Revascularization strategies should be preferred instead of conservative treatment in patients with high-risk features such as ongoing ischemia, recurrent chest pain, left main coronary artery dissection, ventricular arrhythmias, or hemodynamic instability[14,15]. Coronary artery bypass graft surgery is one of the treatment options and transplantation can be another treatment option in some cases. It was observed that our patient had many of the above-mentioned characteristics that occurred in chest pain that continued approximately three hours before hospitalization and then in coronary angiography. Coronary angiography revealed dissection consistent with TIMI-1 flow starting from the left main coronary artery and extending to LAD. The left main coronary artery is less affected by SCAD, with some reports showing that it accounts for only 2% of affected cases, while it accounts for 13% of cases in a series of STEMI SCAD patients only. LAD is often shown as the most commonly affected vessel and accounts for approximately 40-70% of SCAD cases[16].

We believe that this case is worth reporting due to the fact that this patient is young and middle-aged, that there is no majority of risk factors that increase the susceptibility to SCAD, and that there are catastrophic and serious consequences. SCAD is often misdiagnosed and underdiagnosed. We tried to understand this situation and improve the prognosis with an atypical case with less common associated risk factors, as with this patient.

Conclusion

It should be kept in mind that SCAD may be present in especially young-middle-aged female patients presenting to the emergency department, if they have typical chest pain, myocardial infarction findings, and no risk factors for atherosclerosis. Coronary angiography should be recommended. In addition, keeping the comorbid conditions that cause this condition under control and reducing the risk factors are very important for primary prevention.

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Case Report

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A Rare Case of Paraneoplastic Limbic Encephalitis leading to Epileptic Seizure in a Patient with Ovarian Carcinoma

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Abstract

Paraneoplastic limbic encephalitis (PLE), a rare and diagnostically-challenging encephalopathy, is frequently associated with an underlying malign neoplastic tumor. Epileptic symptoms are uncommon but can be the first sign of the disease. We present a patient admitted to our intensive care unit (ICU) unit with epileptic seizure and a Glasgow Coma Scale (GCS) of six. All tests and investigations that had been utilized for this patient's diagnosis, including blood tests, serological analyses, magnetic resonance imaging (MRI), electroencephalogram (EEG) and cerebrospinal fluid (CSF) test results were evaluated. The patient had been diagnosed with ovarian carcinoma within the last year. The patient's cancer history, her most recent complaints and MRI results were strongly suspicious for paraneoplastic limbic encephalitis. Her neurological condition improved rapidly in a few days with steroid therapy. This case showed that any neurological deterioration based on an ovarian oncologic disease can bring PLE to mind. The possibility of PLE must be taken into consideration in patients presenting with epileptic seizures after neoplastic diagnoses.

Key Words: paraneoplastic limbic encephalitis, ovarian carcinoma, seizure, epileptic

Introduction

Paraneoplastic limbic encephalitis is a rare condition which is often difficult to diagnose. It is an immune-mediated encephalopathy that is often found in relation with an underlying malign neoplastic tumor. Corsellis and colleagues first described PLE as a clinicopathological entity in 1968[1]. The limbic system is the area that is primarily involved, but other areas of the nervous system, particularly the brain stem may be affected. The diagnosis of PLE is very difficult. Symptoms of this entity are similar to that of various other diseases, such as, brain metastases of primary cancers, toxic and metabolic encephalopathies, hypertensive encephalopathy, side effects of cancer therapy, and viral or bacterial infections of the central nervous system (especially herpes simplex)[2]. Most of the cases are diagnosed by clinical presentation, EEG findings, MRI studies, CSF sampling and neuropathological examination. Specific antineural antibodies have not been investigated for PLE[2,3,4,5,6].

Paraneoplastic limbic encephalitis has increasingly been shown to cause epileptic seizures in patients. These seizures are suggested to occur by autoimmune-mediated mechanisms of antibody-associated limbic encephalitis[7,8]. Epileptic symptoms are uncommon, but may as well be the first symptom of PLE. We present a 73-year-old woman with ovarian cancer who was diagnosed with PLE after presenting with epileptic seizures.

Case Report

A 73-year-old woman was admitted to ICU from another hospital with a preliminary diagnosis of status epilepticus. On the first evaluation, she was unconscious with a Glasgow Coma Scale of six. In her history, she was found to have a mild headache, anxiety and had demonstrated personality changes within the last three weeks. She had been evaluated by a psychiatrist and given risperidone treatment. In spite of the treatment, her complaints and symptoms had progressed. She had short term memory loss, agitation, depressive mood and personality changes. She was admitted to the previous healthcare center with stupor one day before; however, she had an epileptic seizure on the same day even though the patient had no history of epilepsy. Her epileptic seizure was still ongoing. Her MRI report was showing temporal cortical and subcortical, insular and right thalamic regional hyperintensities on T2 and flair sequences (Figure 1 and 2). Afterwards, she was referred to our hospital for further diagnosis and management. While evaluating the medical history of the patient we found that she had ovarian carcinoma which was diagnosed 1 year ago. She had been operated on and had received six cycles of chemotherapy after the operation. She was still attending follow-up studies in oncology.

When she was admitted to the ICU, she was unconscious and intubated. She was under sedation with midazolam. Her physical examination revealed pupillary isocoria, positive

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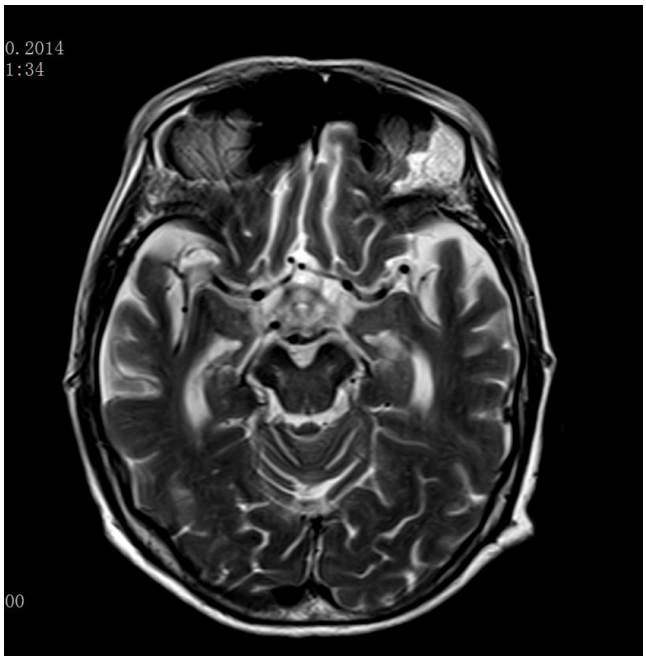

Figure 1. T2 image of first MRI scan presenting intensity changes at temporomesial and limbic areas

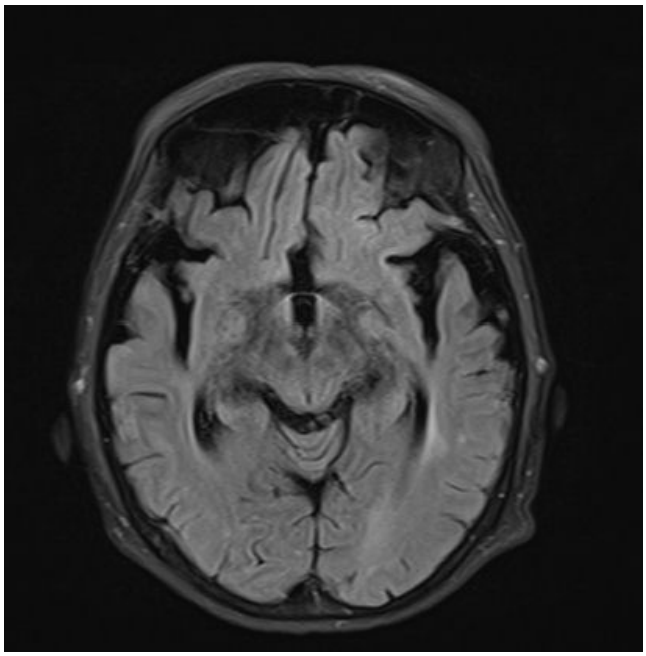

Figure 2. Flair image of first MRI scan presenting intensity changes at temporomesial and limbic areas

light reflex (bilaterally), incomprehensive verbal response and strained arms and legs. In addition to midazolam, we have continued to parenteral levetiracetam for treatment of the status epilepticus. There was no fever. Blood test results, including CBC, sedimentation, CRP and procalcitonin levels, were not signifying an infection. An infectious diseases specialist was consulted and lumbar puncture was performed. The CSF was clear and had unpressurized flow. A sample was sent to the laboratory for further tests. Blood and CSF PCR test results (especially for antineural antibodies) were all uneventful.

The patient's cancer history, her most recent psychiatric complaints and MRI results were strongly suspicious for paraneoplastic limbic encephalitis. The patient was treated at second day of hospitalization with high dose (1000mg / day) intravenous methyl prednisolone additionally to intravenous levetiracetam 1000mg / day for five days. The patient's sedation was stopped on the 3rd day and the seizures were under control. The general condition of the patient improved rapidly. She was extubated on the same day. Despite having mild confusion, she could communicate verbally. All four extremities were moving actively and spontaneously,

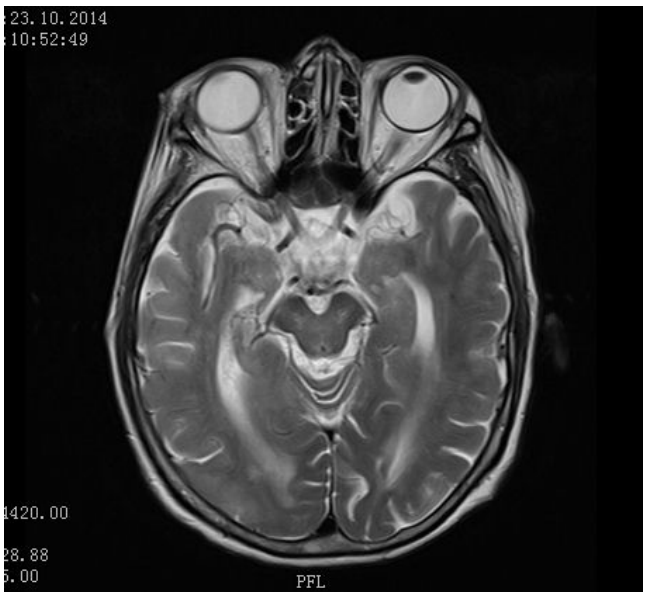

Figure 3. T2 image of control MRI scan presenting regression of intensity changes at first week of the treatment

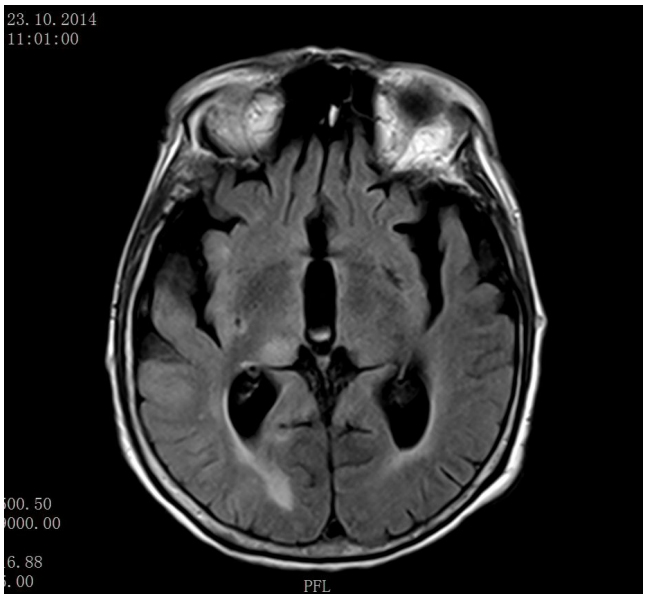

Figure 4. Flair image of control MRI scan presenting regression of intensity changes at first week of the treatment

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and she was responding to commands. Laboratory CSF examination results were negative for CMV IgM, HSV Type 1 IgM, HSV Type 2 IgM, indicating that there was no central nervous system infection. When cranial MRI examination was repeated, lesions in the limbic field were found to be in regression (Figure 3 and 4). After EEG examination, paroxysmal epileptiform activity in both temporal fields (more dominant in the right field) was observed. Following five days with high dose methyl prednisolone and antiepileptic treatment and supportive care, it was observed that the patient's neurological status was recovering swiftly. After she was discharged from the intensive care unit, she was followed in a normal ward and showed dramatic regression of psychiatric symptoms. Unresponsiveness to initial antiepileptic therapy and rapid and good recovery with additional high dose steroid therapy was confirmed our initial diagnosis of PLE. After a few days follow up in the department, her neurological examination was unproblematic and she was discharged with low dose oral prednisolone (2x40mg per a day) and oral levetiracetam (2x500mg) treatment.

Discussion

In the literature, patients with PLE often have an acute or subacute onset of recent memory disorder associated with psychiatric manifestations, including personality changes, depression, hallucination, irritation and agitation[1,3,5]. In our case, headache, personality changes and following agitation, dementia and progressive rapid course that finally led to an epileptic seizure were typical for the diagnosis of limbic encephalitis. Her symptoms and ovarian adenocarcinoma history were the key features. In patients with PLE, tumor detection rate is around 60%. Sometimes, PLE is the first diagnostic sign of a neoplasm. While the most frequent malignancy observed together with PLE is small cell lung cancer, ovarian tumors are not rare. As seen in our case, patients may apply to the hospital with epileptic seizures[7,8]. Treatment of PLE includes steroid therapy, immunosuppression and immunomodulation. Patients with PLE respond often demonstrate dramatic response to therapy. In our case, the patient's epileptic seizure and psychiatric manifestations improved after a 5-days course of glucocorticoid therapy, similar to the results of another study[6].

Conclusion

Our case showed that any neurologic deterioration based on an oncologic disease must bring PLE to mind. It should not be forgotten that, when PLE is diagnosed, a dramatic recovery can be possible with steroids and immune therapy.

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Case Report

Journal of Emergency Medicine Case Reports

De Winter Wave with ST Segment Elevation Equivalent with Speech Disorder; A Case Report

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Abstract

Acute coronary syndrome is a clinical condition that may require urgent coronary intervention. The most important is myocardial infarction with ST elevation. Emerging clinical studies now show equivalents of ST elevation myocardial infarction without ST elevation. The de Winter wave has been recognized as the equivalent of the ST-elevation myocardial infarction described since 2008. A 71 year old male patient was admitted to our emergency deparment with a symptom of speech disorder and later angina. We also detected myocardial infarction with de Winter wave in the patient. In the coronary angiography of the patient, we demonstrated the presence of acute thrombus in the circumflex artery, which is much less common in the literature. Early diagnosis is very important in this acute coronary syndrome that develops without ST elevation. We think that the speech disorder and feeling of worsening in our patient progressed as a result of a vital dystrhythmia that occured before coming to our emergency deparment. Widespread ischemic changes in magnetic resonance imaging in the flair phase may be caused by brain hypoperfusion due to this dysrhythmia. Coronary angiography should be planned for these patients.

Key Words: de winter wave, myocardial infarction, speech disorder

Introduction

As an equivalent of ST segment elevation myocardial infarction (STEMI), de-Winter et al. described an electrocardiogram (ECG) pattern in 2008 (1). Symmetrical and high T waves with ST segment depression (STD) in the precordial leads were found to have a positive predictive value of over 95% for acute occlusion of the left anterior descending (LAD) artery (2,3). This sign was found in 2% of acute myocardial infarction (AMI) cases (1,4). This pattern in the ECG may not always indicate LAD occlusion. However, de Winter wave is an important sign for acute coronary syndrome that needs to be diagnosed early and requires immediate percutaneous coronary intervention (PCI) when detected (5).

Our aim in this case report is to emphasize the importance of the de Winter wave, which we view after chest pain in a patient presenting with a neurological symptom.

Case Report

A 71-year-old male patient was admitted to our emergency department with speech disorder and deterioration. Physical examination of the patient was normal. Neurological examination revealed no lateralizing finding and dysarthria. Systolic blood pressure was 110/70 mm/Hg, pulse 73/min, respiratory rate 19/min, and oxygen saturation 95%. Central imaging was performed in our patient who presented with a neurological symptom. While there was no acute finding in brain computed tomography (BCT), diffusion brain magnetic imaging (Dif MRI) showed acute diffusion restriction of 3-4 mm in the left parietal and diffuse ischemic lesions in the flair phase. (Figure 1) The patient with a known diabetes mellitus (DM) diagnosis said that he felt pressure-like pain in the chest afterwards. In the first ECG (Figure 2) of the patient, there was a prominent T wave in the precordial leads. In the control ECG (Figure 2) of the patient, there were upsloping ST depression and high symmetrical T waves in V2-V6 leads. Segmental wall motion abnormality was viewed on echocardiography (ECHO). Coronary angiography (CAG) was scheduled due to the appearance in accordance with the de-Winter pattern in the ECG. The patient's troponin T resulted as 3100 ng / L. In the CAG, it was observed that the proximal part of the circumflex artery (CX) was occluded with acute thrombus at a rate of 90%. (Figure 3) A 3.0x15 mm stent at 16 atm pressure was placed in the lesion in CX. A 3x0.9 NC balloon was dilated under a pressure of 20 atm inside the stent to provide patency. The patient was discharged home after 4 days of follow-up without any sequelae.

Discussion

In this report, our aim is to emphasize the importance of the de Winter wave, which we view after progressing angi-

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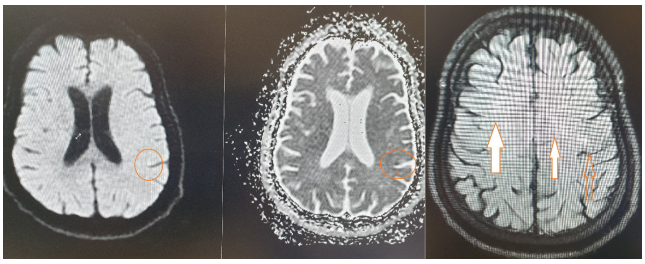

Figure 1. Brain diffusion magnetic imaging: diffusion phase, adc mapping, flair phase

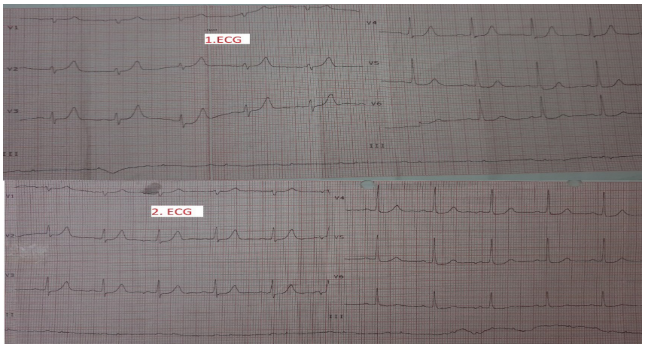

Figure 2. Electrocardiography (ECG) images: 1. The patient's arrival ECG, 2. The ECG taken when chest pain develops.

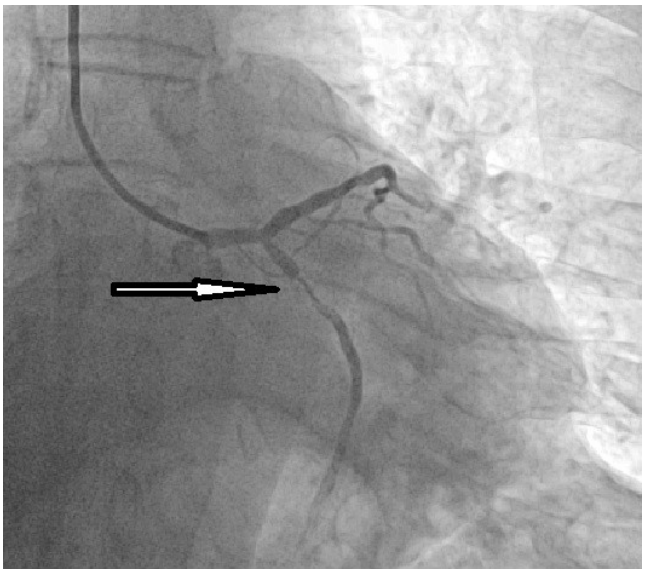

Figure 3. Coronary angiography images: Proximal part of the circumflex artery (CX) was occluded at a rate of 90%.

na in a patient with neurological symptoms, for emergency coronary intervention. In the study conducted by Xu et al. in patients with anterior STEMI, they showed Winter wave pattern in 15 of 441 patients. In these patients, they showed that the vessel responsible for infarction was LAD and its branches. As a result of this study, they stated that the Winter pattern is a symptom of ischemia and can turn into AMI (6). In other case reports, it was observed that a patient with the Winter pattern transformed into STEMI, and a patient with STEMI turned into Winter wave. This situation is thought to be caused by the thrombus in the artery that does not cause full-thickness occlusion or the thrombus undergoes spontaneous lysis (7,8). In our case, our patient previously had a syncope described as a speech disorder and deterioration. There were markedly high T waves in the first ECG. Precordial upsloping ST depression and high and prominent T waves developed in the control ECG with the description of angina in the following period of our patient. This situation made us think that the patient had a fatal dysrhythmia and syncope before coming to the emergency department. The multiple ischemic focus in the dif MRI flair phase image is an indication of this. In the 2013 American Heart Association (AHA) guideline, Winter recommends coronary interventional treatment within 2 hours in patients with an ECG pattern (9). In our case, PCI was made as a result of this dynamic de Winter view we saw on the ECG. Acute occlusion in CX, elevation in troponin T, indicated that this condition was AMI. Although Winter wave is an important finding for LAD, it can also be seen in CX lesions (10). This is proof that the Winter wave cannot be seen on the first visit of the patients. Alahmad et al. Applied a chemical cardiology in a patient with atrial flutter. Winter's wave was detected due to thrombus in the left atrium that developed on the 2nd day. Acute thrombus was observed in LAD D2 in the patient after PCI (11). In our case, no cardiac thrombus was observed, but it is the first case of Winter in the literature to have central thrombus and coronary thrombus together, albeit minimally.

Conclusion

Although the constution mechanism of the de Winter wave is not known exactly, it should be treated like STEMI due to its consequences. Early interventional coronary artery treatments provide good outcomes.

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Case Report

Journal of Emergency Medicine Case Reports

Thyrotoxic Hypokalemic Periodic Paralysis: Case Report

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Abstract

Thyrotoxic Hypokalemic Periodic Paralysis (THPP) is a rare hereditary disorder which is characterized by thyroid hormone elevation, low blood potassium level and recurrent acute muscle weakness. Basic pathology is thought to be the increase in activity in the sodium-potassium pump (Na+/K+ATPase). Here we report the case of a 31-year-old male that presented with weakness in his legs, and inability to walk. The patient had elevated thyroid hormone levels (FT3 and FT4) and lower TSH levels, lower serum potassium levels, and recurrent acute muscle weakness. The diagnosis was made to be Thyrotoxic Hypokalemic Periodic Paralysis precipitated after intense physical activity. THPP is a reversible medical emergency. Early diagnosis, and rapid treatment is lifesaving. Although rare, THPP must be considered as a differential diagnosis in patients presenting with hypokalemia and paralysis.

Key Words: endocrine, thyrotoxic hypokalemic periodic paralysis, sodium potassium pump, hypokalemia

Introduction

Thyrotoxic Hypokalemic Periodic Paralysis (THPP) is a rare endocrinological entity, characterized by thyrotoxicosis, hypokalemia, and acute proximal muscle weakness. Its incidence has been reported to be 2% in Japanese and Chinese populations with thyrotoxicosis, and even lower (0.1-0.2%) in Caucasian populations[1]. Although hyperthyroidism is more frequently seen in women, THPP is more frequent among men. THPP can be triggered by physical activity, cold exposure, stress, heavy carbohydrate or alcohol intake. Pathophysiologic mechanism is when the catecholamine discharge increases secondary to hyperthyroidism, intracellular passage of potassium increases. This leads to an increase in the number and activity of Na+/K+ATPase, and it manifests as paralysis.[2,3]. Additionally, it may present with fatal hypokalemia, reversible muscle weakness, and attacks of paralysis[4]. Fortunately, THPP is a reversible medical emergency. To get serum potassium levels back to normal, beta-blockers can be initiated and euthyroidism can be ensured with hyperthyroidism treatment. Death can be easily prevented with accurate diagnosis and management. Therefore, here we report the case of a 35-year-old male patient with THPP.

Case Report

A 35-year-old male patient presented to our Emergency Department with weakness in his legs, and inability to walk. He worked as a dockworker, and he reported that he previously experienced episodes of muscle weakness a few times at night. Because these weaknesses were not severe, he didn't seek medical help. Two months ago, he consulted another medical center with complaints of dizziness and shivering. Propranolol and methimazole were initiated as treatment, however he didn't use these medications regularly. The patient's additional medical and family history was unremarkable. He had no history of smoking, alcohol substance or additional drug use.

At the time of his admission to our hospital, he was first evaluated by the Neurology Department. Cerebral and spinal imaging studies were clean, but his blood tests revealed the presence of hypokalemia. Hence, he was transferred to the Internal Medicine Department for further evaluation. Here the physical examination findings were as follows: TA: 120/70 mmHg; pulse rate: 90/min, rhythmic; body temperature: 36.9 C; respiratory rate: 16/min. His skin had a natural color but was sweaty. Thyroid gland manifested Stage 1b goiter. He had open consciousness with complete cooperation, full time and place orientation. He was agitated due to complaints of weakness. Muscle strengths were 4/5 and 5/5 for proximal and distal parts of upper; 3/5 and 5/5 for proximal and distal parts of lower extremities, respectively. Sensory loss was not detected. Babinski reflexes were negative, and bilateral DTRs were decreased. Rest of the examination was normal.

For diagnostic purposes, biochemical and hematologic tests were requested, and they revealed lower serum potassium levels of 2.1mmol/L (reference range: 3.5-5 mmol/L), creatinine levels of 0.38mg/ dL (reference range: 0.74-1.35

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mg/dL), hemoglobin levels of 13.5 gm/dL (reference range: 14.0-17.5 gm/dL). On the other hand, the patient's blood urea was higher with the level of 31 mg/dL (reference range: 6-24 mg/dL). His other biochemical findings were within the normal range. Arterial blood gas analysis, complete urinalysis and peripheral smear were unremarkable. ESR was higher than the reference range with a level of 30 mm/hr (reference range: 0-22 mm/hr), but iron /lipid panel, CRP and phosphorus levels were within normal limits. Posteroanterior chest X-ray, EMG, and ECHO did not demonstrate any abnormality. On EKG, a mildly elongated PR interval, and slight T- wave flattening were detected. Thyroid US, and scanning results were consistent with Graves' disease. Thyroid Function tests reported a lower TSH level of 0.01 miU/L (reference range: 0.2-5.5 miU/L), and higher levels of FT3: 45 pmol/L (reference range: 3.1-6.8 pmol/L) and FT4:57 pmol/L (reference range: 10-24.5 pmol/L). Anti-TPO and TSH receptor blocking antibody levels were higher with levels of 194 IU/mL (reference range: < 9 IU/mL) and 27 IU/L (reference range 0.79-3.47 IU/L) respectively.

For treatment, potassium replacement was started. Initially, infusion of 40 mEq/L KCL in 1000 cc isotonic saline was delivered within 4 hours. In the third hour of treatment, the patient's complaints were reduced; and in the fifth hour, the patient was able to walk. After he received a total of 80 mEq/L KCl, his potassium levels normalized and was 4.5 mmol/L (reference range: 3.5-5 mmol/L). The patient was also started on daily doses of 3*10 mg p.o. Methimazole, and 2*40 mg p.o. Propranolol treatment. He was advised about the importance of quitting smoking, using non ionized salt, and complying to drug therapy. His general health state improved without any additional complaints and he was discharged with recommendation of ambulatory control in the Endocrinology Outpatient Clinic. During his 2-month long follow-up, he didn't have any recurrent episodes of the condition. His control tests revealed significant improvement.

Discussion

Thyrotoxic Hypokalemic Periodic Paralysis (THPP) is a rare hereditary disorder which is characterized by thyroid hormone elevation, low blood potassium level and recurrent acute muscle weakness. Ion channel defects in the familial paralysis tables, and relevant family histories are not seen in this disease. THPP can be differentiated from other entities with concomitant presence of hyperthyroidism, and hypokalemia. The basic pathology in THPP is thought to be due to the disequilibrium between intra and extracellular potassium values. This condition is related to increased number, and activity of Na/K/ATPase pump secondary to thyrotoxicosis with resultant increase in intracellular potassium inflow[5]. Case presented here is consistent with the definition of THPP since the patient had elevated thyroid hormone levels FT3: 45 pmol/L (reference range: 3.1-6.8 pmol/L) and FT4:57 pmol/L (reference range: 10-24.5 pmol/L), lower serum potassium levels of 2.1mmol/L (reference range: 3.5-5 mmol/L) and recurrent acute muscle weakness.

Among factors precipitating the attacks, higher amounts of carbohydrate intake, trauma, upper respiratory tract infection, emotional stress, alcohol intake, and heavy exercise have been reported[6,7]. Heavy intake of a diet rich in carbohydrates develops hyperinsulinemia and induces intense beta-adrenergic activity which speeds up intracellular inflow of potassium. Similarly, higher rates of THPP developed in men are associated with the androgenic effect induced increase in Na+/K+ATPase. In our case, the patient was male and an androgenic effect might be present. He presented to the Emergency Department after a work day where he does intense physical activity. It might also be possible that the THPP attack was precipitated after a heavy exercise.

Among etiologic factors of hypokalemia, diuretic use, renal and gastrointestinal causes should be priorly considered, and inquired. One should be attentive about cardiac arrhythmias and respiratory problems in the cases of hypokalemia. Our patient, although had hypokalemia, presented with only mild changes in the ECG and no abnormalities in respiration. In the case of reversible paralysis, it's important to remember possible cardiac and respiratory complications of THPP.

In most of the cases with THPP, Grave's disease was detected; while other reported etiologies include toxic nodular goiter, thyroiditis, TSH-secreting adenoma, and exogenous thyroid hormone intake[8,9]. Here our case was in parallel with the previous literature as the Anti-TPO and TSH receptor blocking antibody levels: 194 IU/mL (reference range: < 9 IU/mL) and 27 IU/L (reference range 0.79-3.47 IU/L) respectively, thyroid US, and scanning results were consistent with Grave's disease.

THPP is a reversible medical emergency that can be treated in two parts: treatment of hypokalemia and treatment of hyperthyroidism. Treatment of hypokalemia can be instituted based on the clinical manifestations of the patient through oral route or intravenous route in compliance with relevant protocols. In the literature, development of rebound hyperkalemia has been reported in 40 % of the patients who received IV KCl at a rate of 10 mmol/hour. Therefore, slow replacement with IV KCL is recommended for these cases[10]. That's why we did not see rebound hyperkalemia in our case. For hypokalemia treatment, non-selective beta-blockers such as Propranolol should be initiated, which will lead to correction of Na+/K+ATPase, hence a marked improvement in hypokalemic state. Our case was consistent with this information as well. For long-term effective treatment in cases with THPP, euthyroidism should be ensured with hyperthyroidism treatment that normalizes the function of the pump. Based on the underlying thyroid pathology, medical treatment, radioactive iodine, or surgery can be preferred.

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Conclusion

Early and accurate diagnosis, and rapid treatment in THPP is lifesaving. Although rare, THPP must be considered as a differential diagnosis in patients presenting with hypokalemia and paralysis.

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Case Report

Journal of Emergency Medicine Case Reports

Case Series About Atropa Belladonna (Deadly Nightshade) Intoxication and Experience of Physostigmine

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Abstract

Atropa belladonna (deadly nightshade) is a poisonous herb that can be especially seen in Northern regions of our country and cause anticholinergic toxic syndrome (ATS) when consumed. ATS can be seen when consuming the plant which contains atropine or in overdose of psychoactive drugs containing atropine (such as antipsychotics, tricyclic antidepressants). A 65-year-old female patient was admitted to emergency service with the clinic of agitation, speech disorder and consciousness change. It was learned that she consumed atropa belladonna fruits about an hour ago. She was followed up with the diagnosis of ATS and treated with conservative approach. A 58-year-old male patient was admitted to the emergency servise due to impaired consciousness, agitation, difficulty in speaking, vomiting and fever. He also ate the same fruits with his relative, case 1. He also followed up with a diagnosis of ATS and treated with both conservative approach and physostigmine. Consuming atropa belladonna fruits and leaves can lead to ATS and life-threatening consequences can be seen in severe toxicity. Early diagnosis and treatment are important in prognosis. Physostigmine must be kept in mind in cases who does not benefit from conservative approach.

Key Words: atropa belladonna intoxication, anticholinergic syndrome, physostigmine

Introduction

Atropa belladonna is a herb which is a member of Solanaceae family and also called 'deadly nightshade'. The leaves of this herb are green, the fruits are small, shiny, round, sweet and black in color (Fig.1). Intoxication can occur when consuming the fruits, leaves or root of the Atropa belladonna plant. The fruit and leaves of the plant contain high levels of atropine, scopalamine and hyoscyamine alkaloids[1-3]. People generally consume the fruit of the plant because they do not know that it is poisonous but sometimes they think consuming will be good for their underlying chronic diseases, especially diabetes mellitus. In mild intoxication cases; nausea, vomiting and dizziness can be seen but; impaired consciousness, fever, flashing, mydriasis, dry skin and mucous membranes, tachycardia, ileus, urinary retention, muscle fasciculations, dysarthria and visual hallucinations can be seen in severe cases.

Case Report

Case-1: A 65-year-old female patient was referred to us from a district state hospital with the clinic of agitation, speech disorder and consciousness change. It was learned that; the patient ate 5 or 6 fruits of Atropa belladonna plant 8 hours ago for treatment of her existing chronic disease, diabetes mellitus.The patient had complaints of dry mouth and dyspnea about half an hour after eating the fruit. Afterwards, when she had meaningless speech and difficulty inspeaking, she applied to the state hospital. Her blood pressure was 120/90 mmHg, body temperature was 36 °C and peripheral oxygen saturation was %93 at the hospital admission. Her level of consciousness was confused, her Glasgow Coma Scale (GCS) score was 15, her pupils were dilated and bilateral pupillary light reflex were positive (Fig.2). The remaining system examinations were evaluated as normal. The patient was referred to our hospital with a initial diagnosis of cerebrovascular disease. At the time of admission to our hospital; her GCS score was 15, blood pressure was 155/97 mm/Hg, heart rate was 107 beats per min, and peripheral oxygen saturation was 97. The patient had a history of diabetes mellitus, hypertension, asthma and panic attacks. She had difficulty in speaking, amnesia and visual hallucinations. Her pupils were mydriatic and oral mucosa was dehydrated and hyperemic. Other system examinations were normal. Normal sinus rhythm was detected on electrocardiogram (ECG) and heart rate was 99 per min. There was no abnormality in the complete blood count and biochemical blood tests. Cranial tomography and diffusion magnetic resonance imaging (MRI) were performed to rule out cranial pathologies and no immediate pathological image was detected. The patient was admitted to the emergency department intensive care unit with the diagnosis of Atropa belladonna

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intoxication. Conservative treatment was initiated. On the second day of follow-up, difficulty in speaking and dryness of oral mucosa disappeared, but visual hallucinations continued. On the third day her hallucinations also disappeared. No pathological finding was found in her physical examination and laboratory tests and the patient was discharged with full recovery on the third day. Since the patient's complaints decreased with a conservative approach after her arrival; physostigmine was not considered in the treatment.

Figure 1: Atropa belladonna plant and fruit photo was taken by the patient's relatives. (Permission was obtained from the patients for the photograph).

Case-2: A 58-year-old male patient was admitted to the district state hospital due to impaired consciousness, agitation, difficulty in speaking, vomiting and fever. It is learned that he ate 6 or 7 fruits of atropa belladonna about 8 hours ago, because he had thought it would be good for his current disease; diabetes mellitus. He had dryness in his mouth one hour after eating. In the next few hours, hot flashes, gibberish, and agitation developed. He was referred to our hospital for further research since his complaints did not resolve. His blood pressure was 115/60 mmHg, heart rate was 105 beats per min and body temperature was 37.5 °C at the time of admission to our hospital. He was disoriented and cooperation was difficult because of his rapid and meaningless speech. He had dry and sticky oral mucous membranes. His GCS score was 14 and he had severe agitation and visual hallucinations. Pupillary light reflexes were bilateral positive and pupils were mydriatic (Fig.2). His motor system examination and reflexes were normal. Abdominal examination was normal and there was no sign of urinary retention. He had sinus tachycardia during first minutes of follow- up (115-120 beats per min). Leucocyte count was 15,820 cells per microliter. Biochemical blood tests were normal. Cranial tomography and diffusion MRI were performed to rule out cranial pathologies and no immediate pathological image was detected. The patient was admitted to the emergency department intensive care unit with the diagnosis of Atropa belladonna intoxication. 5 mg diazepam infusion was administered and 5 mg haloperidol tablet was given to control the patient's agitation. Intravenous (IV) midazolam is also administered intermittently for this purpose. Despite all these conservative medication; patient's hallucinations, difficulty in speaking and agitation continued, 2 mg physostigmine was administered intravenously. A significant improvement in neurological findings was observed half an hour after the treatment. During his follow-up period, neurotoxic findings of the patient completely regressed within 24 hours. The patient, who was asymptomatic on the third day of follow-up period, was discharged with full recovery.

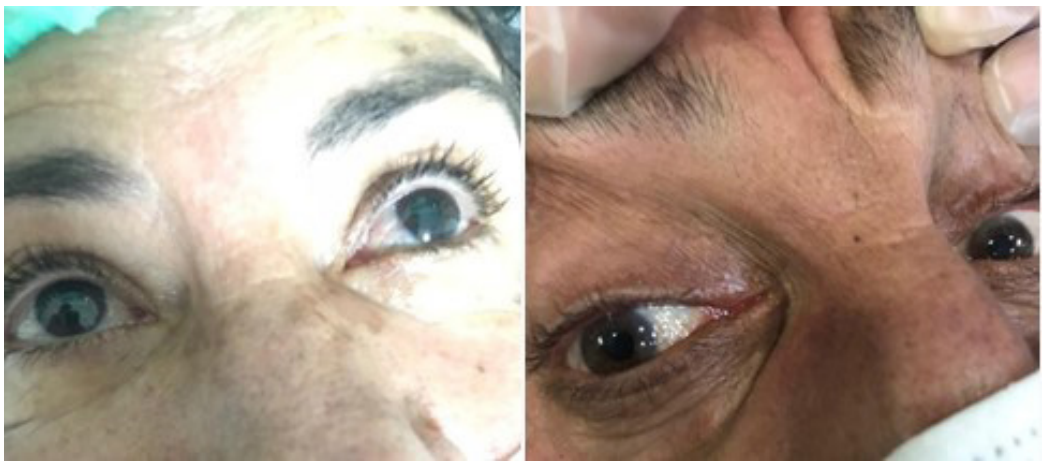

Figure 2: Mydriasis is observed in patients during acute poisoning. (Permission was obtained from the patients for the photographs)

Discussion

ATS has a broad clinical spectrum range from nausea-vomiting to life threatening situations like coma. In mild cases, we can see dryness of skin or mucosa, fever,abdominal pain and nausea vomiting but in severe cases we can see serious symptoms like acute psychotic attack, convulsion and coma[4,5]. At high doses, atropine alkaloids cause ATS clinical features by competitively blocking parasympathetic postganglionic muscarinic receptors and acetylcholine binding sites in the central nervous system. While symptoms such as hallucinations, amnesia, delirium, agitation, cooperation and orientation disorders, acute psychosis, cardiovascular and respiratory failure and coma are observed due to central nervous system involvement; findings such as mydriasis, dryness and redness of the skin and mucous membranes, urinary retention and ileus may be observed due to peripheral nervous system involvement[6]. Ahıskalıoğlu et al reported a 62-year-old male patient who was admitted to the emergency room with GCS score of 5, 2 hours after consumption of atropa belladonna fruit. He had a low-grade fever (37.5 °C) and tachycardia (128 beats per min). His pupils were mydriatic, oral mucosa was dry and acute globe vesicalis was observed. 2 mg iv physostigmine was administered after gastric lavage and activated charcoal administration and the patient is extubated within 24 hours[7]. Ethemoğlu et al. reported a 52-year-old male patient whose complaints started 2 hours after consuming the cooked leaves of the plant. He had restlessness, inability to urinate and he was in a state of severe delirium and agitation.His pupils were mydriatic and mouth was dry, bowel sounds were decreased. He had no fever but tachycardia (109 beats per min). The patient was taken under control with a conservative treatment approach because physostigmine could not be reached[8]. In a case series included 4 adult intoxication patients reported by Schneider et al.; visual hallucinations and delirium were observed in 3 of them and the other was intubated[9]. A 49-year-old female patient presented by Demirhan et al. was accepted to the emergency service with a poor conscious state and a GCS score of 7. Her body temperature was normal but pulse was 132 beats per min. She had a dry oral mucosa, dilated pupils and acute globe vesicalis. In laboratory tests; white blood cell count was 15.700 cells per milliliter and blood glucose level was 224 mg/dl. Gastric lavage was performed and activated charcoal were given after the patient was intubated. Conservative treatment approach was administered because physostigmine could not be reached[10]. In the study of Çaksen et al. in which they evaluated 49 pediatric patients with Atropa belladonna intoxication, meaningless speech, mydriatic pupils, tachycardia and rash were reported as the most common signs and symptoms[2]. We detected both central and peripheral findings of ATS in our cases. In our first case, which had a milder clinical course, there were altered consciousness, amnesia, visual hallucinations and agitation as central findings, while mydriatic pupils and dry mouth were present as peripheral findings. In our second case, there were severe agitation, aggression, visual hallucinations, amnesia and speech disorder as central findings; while dry mouth and mydriatic pupils were observed as peripheral findings. There was no urinary retention in our patients. Among the vital signs, both patients had tachycardia, fever was normal in our first case but subfebrile fever was detected in our second case. While there was no significant finding in our first patient in laboratory tests, leukocytes were detected as 15,820 cells per microliter in our second patient. Since the patients were brought to hospital 6 hours after their complaints started, gastric lavage and activated charcoal were not applied, while IV diazepam was started for the agitation of both patients. Haloperidol was administered to second case because of persistent agitation with the recommendation of psychiatry. While the complaints of first case regressed with conservative treatment approaches, in our second case, there was no significant regression, so physostigmine was provided and 2 mg IV was administered in two minutes and a significant improvement was observed in his complaints 30 minutes after the treatment. Both patients were observed as asymptomatic for 24 hours and then they were discharged from our emergency service intensive care clinic with recovery.

The primary approach in the treatment of atropa belladonna intoxications is conservative treatments[10]. The patient should be monitored, airway patency should be ensured, conservative medical treatments should be started and attention should be paid to mechanical ventilation. Atropine is rapidly absorbed from the gastrointestinal tract, reaching peak levels in the blood 2 hours after ingestion. Gastrointestinal decontamination and activated charcoal administration should be given if the patient arrives at the hospital within this time frame[7]. In our cases, this prosedure was not performed because thepatients were brought 6 hours after oral intake. Benzodiazepines can be used if the patient has agitation[11]. We administered 5 mg IV diazepam to both of our patients. Especially in severe cases with central nervous system findings, physostigmine should be tried in the treatment[12]. We did not administer physostigmine to first case because it responded to conservative treatment, but we administered IV physostigmine to second case, due to non-regressive central nervous system findings with conservative approaches, and we observed a dramatic improvement after physostigmine. We discharged both of our patients asymptomatically.

Conclusion

Atropa belladonna intoxication is a rare condition that can lead to anticholinergic syndrome and life-threatening consequences in cases of severe toxicity. Early diagnosis and

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treatment are important in prognosis. Physostigmine is a difficult drug to find in most health care provider. However, it can be obtained from certain centers if needed. In this report, we wanted to report our experience on the treatment processes of two patients with different clinical features, who consumed the fruits of Atropa belladonna.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Case of Hyperlactatemia in The Emergency Department

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Abstract

Glycogen storage disease type 1a is a rare autosomal recessive syndrome characterized by hypoglycemia, hyperuricemia, hyperlipidemia, hepatomegaly, among other features. Case report: A 31-year-old woman genetically diagnosed with this disease in childhood was admitted to the Emergency Department with tachypnea. Her arterial lactate was 179 mg/dL, bicarbonate of 2 mmol/L, pH of 7.0 and pCO2 2.2 mmHg. She received IV glucose, isotonic bicarbonate, and antibiotics. Her urine culture was positive for *Escherichia coli*. She had a complete recovery from acidosis in 12 hours and was discharged three days later. Conclusion: This case highlights a rare differential of lactic acidosis that can, sometimes, be present in the Emergency Department.

Key Words: glycogen storage disease, von Gierke disease, hypoglycemia, lactic acidosis, emergency medicine.

Introduction

Hyperlactatemia occurs when lactate production exceeds lactate consumption. Lactic acidosis is an anion gap metabolic acidosis that can be classified into two types: those with tissue hypoxemia (type A) and those without tissue hypoxemia (type B)[1,2].

Type B etiologies include intoxication (i.e. metformin), inborn errors of metabolism (i.e. glycogen storage disease), diabetic ketoacidosis, malignancy, and alcoholism[1].

Glycogen storage disease (or glycogenosis) refers to a group of hereditary metabolic disorders with improper metabolism of glycogen, resulting in hypoglycemia and lactic acidosis. Depending on the type of disease, autosomal inheritance can be dominant or recessive[3].

We report a case of glycogen storage disease type 1a presenting with type B hyperlactatemia in the Emergency Department (ED) and performed a literature review about this rare condition.

Case Report

A 31-year-old woman presented to the ED with a two-day history of tachypnea. She denied fever, cough, and other systemic symptoms.

In her first year of life, she was genetically diagnosed with glycogenosis type 1a (also known as von Gierke disease). She was using atorvastatin, ciprofibrate, and a special corn starch diet for her condition.

At admission, she was afebrile, heart rate was 120, blood pressure of 120/76 mmHg, oxygen saturation was 99%, and respiratory rate of 36 ipm with good peripheral perfusion.

Workup exams demonstrated an arterial pH of 7, bicarbonate of 2 mmol/L, base excess -28 mmol/L, lactate 179 mg/dL, anion gap of 30, pO2 118 mmHg, pCO2 2.2 mmHg, mild elevation of inflammatory markers, and leukocytosis of 15.820/mm3. She promptly received IV glucose (250 mg/kg followed by 3-4 mg/kg/min), isotonic 8.4% bicarbonate (1 mmol/kg/h), and ceftriaxone (2g daily) to prevent hypoglycemia, manage acidosis and treat a potential infection, respectively.

Exams to determine the cause of decompensation demonstrated a urine culture positive for *Escherichia coli*. Blood cultures and a thorax computed tomography were negatives. As a result, we continued to use ceftriaxone for cystitis treatment.

We decided to start an oral diet after twelve hours (Table 1) when the acidosis was resolved. She was discharged three days later.

The patient provided informed written consent for publication.

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Table 1: Evolution in exams parameters during hospital stay

	Bicarbonate (mmol/L)	Lactate(mg/dL)	Glucose (mg/dL)
Admission*	2	179	87
2 hour	5.2	-	140
4 hours	6.9	-	-
8 hours*	8.6	259	220
10 hours	14.3	257	-
12 hours	21	257	180
20 hours	25.5	-	-
40 hours	20	23	-

*From arterial blood, the others samples are from venous blood

Discussion

As previously described, glycogenosis is a rare genetic disorder characterized by an inborn glycogen metabolism error. Type 1a is defined by a deficit in glucose-6-phosphatase (G6Pase) enzyme, which catalyzes the final phase of glycogenolysis and gluconeogenesis with glucose-6-phosphate transporter (G6PT). Type 1b, however, is caused by a G6PT mutation. The disease is autosomal recessive, with an overall incidence of 1:100 000 cases, with about 80% of these having type 1a[3].

The symptoms differ depending on the patient's age. In most cases, the diagnosis is made during childhood. In the neonatal period, doll-like facies, hypoglycemia, lactic acidosis, and hepatomegaly may be present. Hypoglycemia, lactic acidosis, hyperlipidemia, hypertriglyceridemia, and hyperuricemia are among the conditions that can occur during evolution. Type 1b has neutropenia and reduced neutrophil functions, but type 1a has a functional immune system. Inflammatory bowel disease is also more common in type 1b[3,4].

The diagnosis is based on clinical features and confirmation of mutations in the G6Pase and SLC37A4 (G6PT) genes[5]. Liver biopsy is not mandatory; however, a diagnosis may be made by measuring the G6Pase enzyme activity in a piece of snap-frozen liver biopsy tissue[3].

Management is based on appropriate food intake to prevent hypoglycemia – such as corn starch. Fructose and galactose metabolism is impaired when the G6Pase enzyme is deficient, hence these sugars should be avoided or minimized. Multivitamins and calcium may be required because of the restricted diet[3,6].

Allopurinol may be used for hyperuricemia as well as statins and fibrates for hyperlipidemia. For type 1b neutropenia, a granulocyte colony-stimulating factor is considered first-line therapy. For patients who develop microalbuminuria, ACE inhibitors should be initiated along with oral bicarbonate or citrate to treat persistent acidosis[3,6].

There is limited evidence in the literature for acute management. We followed the recommendation in the last European Guideline: a bolus of 250 mg glucose/kg followed by 3-4 mg/kg/min and IV bicarbonate for acidosis correction[6]. There is no report about the optimal concentration for bicarbonate repositioning or when the oral diet should be reintroduced. We opted for isotonic bicarbonate concerning the sodium level and restarted the oral diet once the acidosis was under control and the patient's tachypnea had improved. In cases of acute decompensation, it is important to search for precipitating factors including dietary non-compliance and bacterial or virus infections[2].

The issue with acute decompensation is respiratory fatigue rather than acidosis. To our best knowledge, this is the second case report of glycogenosis presented in the ED. The first case was described by Oster et al. in 2016. In their report, the patient had a catastrophic evolution caused by respiratory failure and required orotracheal intubation. Even dialysis was considered for acidosis management since sodium level was too elevated as a result of bicarbonate administration[2].

Conclusion

Glycogenosis is a rare cause of lactic acidosis, mainly in the pediatric population. The management should include IV glucose for hypoglycemia prevention and IV bicarbonate for acidosis control.

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Case Report

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A Rare Small Intestine Injury Without Free Gas Image on Radiological Imaging After Blunt Abdominal Trauma in A Child: Ileal Perforation

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Abstract

Introduction: Trauma continues to be one of the leading causes of death in children. Even though among traumas blunt abdominal trauma is frequently seen in children, small intestine injuries due to blunt abdominal trauma are very rare. In case the diagnosis of intestinal perforations is delayed and not treated rapidly, severe morbidity and mortality may develop.

Case Report: In this case, a 4-year-old male patient with ileal perforation, who applied to the emergency department with blunt abdominal trauma and did not show any signs of free gas in radiological imaging, was presented.

Conclusion: To the best of our knowledge, ileal perforation associated with blunt abdominal trauma is very rare in the literature. The aim of this case report is to emphasize the necessity of considering small intestine injury in the differential diagnosis of acute abdominal syndrome in children applying with blunt abdominal trauma.

Key Words: child, blunt abdominal trauma, small intestine injury, ileal perforation

Introduction

In the world, trauma is still one of the most common causes of death in childhood. In all periods of life, small intestine injuries associated with blunt abdominal trauma are rare, especially ileal perforation is a very rare. In the pediatric age group, spleen and liver injuries are observed after blunt abdominal trauma in general[1].

Small intestine perforations are generally determined by the findings of peritoneal irritation. In hollow-organ perforations in the abdomen, free gas image is generally seen in the abdomen in direct graphy[2].

The aim of this case report is to emphasize the necessity of considering small intestine injury in the differential diagnosis of acute abdumen, even if there is no free gas image on radiological imaging in children applying with blunt abdominal trauma.

Case Report

A four-year-old male patient applied to the emergency department due to the complaint of falling down from the height onto a hard floor. When the patient was examined at the emergency department, it was found that body temperature was 37.4 ^{O}C, blood pressure was 110/75 mmHg, pulse was 136/min, respiratory rate was 36/min.

In his physical examination, his consciousness was open and cooperated. In his abdominal examination, there was a four-cm wide ecchymotic area in the form of a strip under the umbilicus which went over the abdomen in the transverse plane, and palpation and percussion were normal. Other system examinations were normal. In his laboratory examination, hematological parameters, biochemical, coagulation and urinary tests were normal.

On the standing direct abdominal radiograph (SDAR), no free gas image was found (Figure 1). Thoracic and abdominal computerized tomography (CT) with non-contrast was assessed as normal. The patient was hospitalized at the department of pediatric surgery for follow-up.

He was started nutrition with oral liquid foods. He started to have significant tenderness under the umbilicus at deep palpation in the abdomen. Oral intake was terminated due to the onset of vomiting. In the second day of hospitalization, abdominal ultrasonograph (USG) and contrast-enhanced abdominal CT were conducted for the patient due to the development of an acute abdomen picture. In the USG, it was reported that ‘‘intestinal segments and mesenteric planes in the hypochondrium and pelvis were edematous”. In the CT, it was reported that “calibration increase in the ileal and jejunal loops, contamination was seen in the mesenteric fatty tissue at the right lower quadrant level, and no free gas image was observed”.

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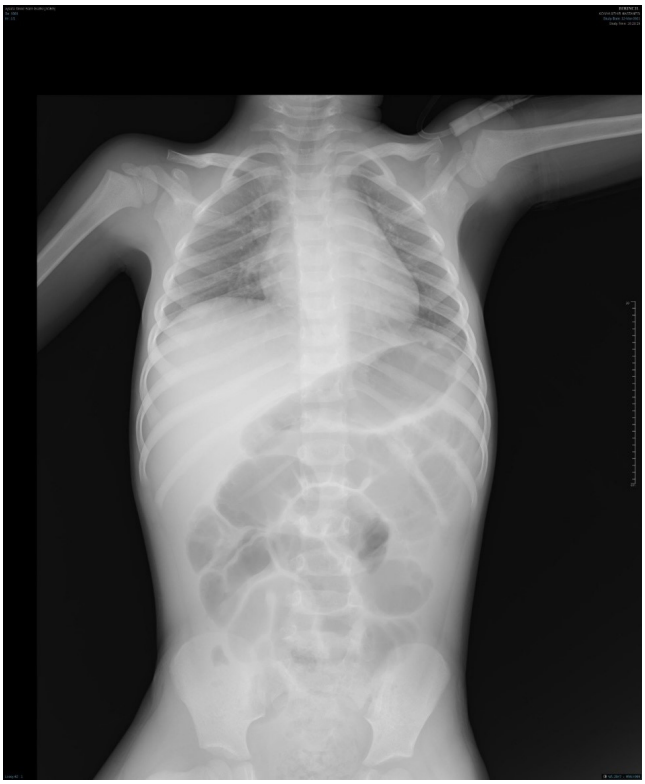

Figure 1: Absence of free gas image on standing direct abdominal radiograph.

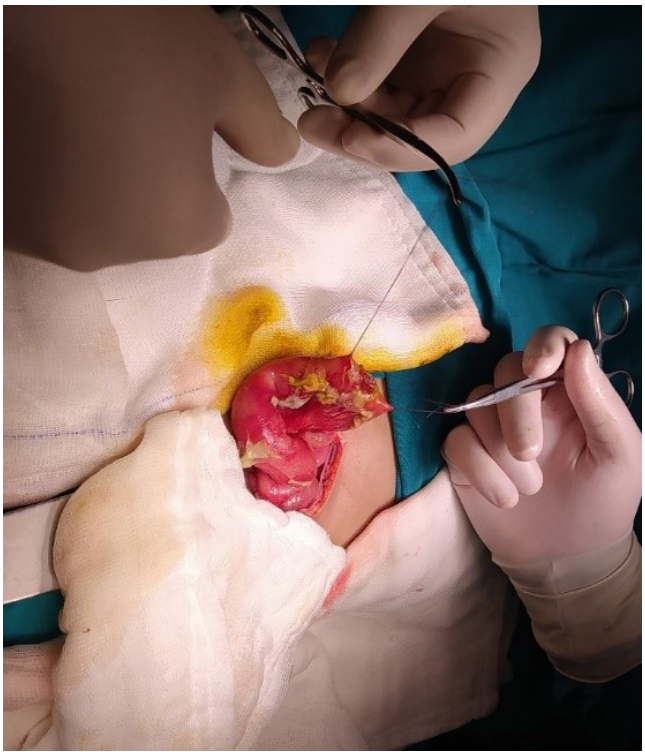

Figure 2: Image of ileal perforation during surgery.

Explorative laparotomy was performed 56 hours after the trauma. During laparotomy, 0.5-1 cm sharply-circumscribed perforation was detected approximately 15-20 cm proximal of the ileocaecal valve on the antimesenteric side in the terminal ileum.. Leakage here was circumscribed by small intestines. Other abdominal structures were found as normal (Figure 2). Perforation was primarily repaired. A drain was placed in the abdomen. On the 3rd postop day, the patient started oral intake and the drain was removed on the 5th postoperative day. The patient was discharged on the 10th postoperative day with full recovery.

Discussion

It is reported that small intestine injuries are about 1% of all the abdominal traumas[2,3]. Small intestine injuries were reported in 27 out of 2550 children with blunt trauma. Small intestine perforation was reported in only two of these 27 patients. The authors reported that the diagnosis of small intestine perforation in children is usually delayed, thus mortality and morbidity rates are higher[2].

The radiological finding of perforation in the traumas of hollow organs is generally the free gas image in the abdomen This image is usually observed in the subdiaphragmatic region in SDAR[4,5]. Since abdominal CT contains radiation, primarily abdominal USG is preferred. Most frequently encountered findings of perforation in abdominal CT are free gas image and the free contrast agent in the abdomen given via oral route. The absence of pneumoperitoneum does not exclude the hollow-organ perforation. Even a small amount of peritoneal fluid may be the only sign that indicates intestinal perforation[5,6]. In this case, pneumoperitoneum was not detected even though we made radiological imagings.

In blunt abdominal traumas, small intestine perforations have been generally reported on the antimesenteric side of the small intestine[5]. As in the current case, diagnosis of small intestine perforations in blunt abdominal traumas is usually delayed[2,3,7,8]. In their study, Holland et al.[7] stated that the mean surgical intervention period of small intestine perforations in children was 29 hours. In the current case, this period was 56 hours. In patients with blunt abdominal trauma, close monitorization of the patient and performing the physical examination again guide the diagnosis and treatment and also prevent delayed treatment[5].

Conclusion

Children applying with blunt abdominal trauma should be followed closely. Although the radiological images are normal, it should be considered that small intestine injury may be present in the differential diagnosis in such patients. If the diagnosis of intestinal perforations is delayed and not treated rapidly, severe morbidity and mortality may develop.

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Case Report

Journal of Emergency Medicine Case Reports

Spontaneous Pneumomediastinum in Covid-19 Pneumonia: Two Cases and Review of the Literature

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Abstract

Spontaneous pneumomediastinum (PM) is a rare presentation associated with Coronavirus disease 2019 (COVID-19). Here, we report two cases presented with spontaneous PM associated with COVID-19 pneumonia.A 58-year-old female patient was admitted to the Emergency Department (ED) with dyspnea, hoarseness, myalgia, and cough. A chest CT scan revealed subcutaneous emphysema, diffuse PM, bilateral diffuse areas of ground-glass opacity, and consolidations. The patient was transferred to the critical care unit. In critical intensive care, the patient developed multi-organ failure and expired on the 10th day. A 34-year-old male patient admitted to the ED with dyspnea and cough. A chest CT scan revealed diffuse PM, bilateral diffuse areas of ground-glass opacity, and consolidations. The patient was transferred to the critical care unit. His progress was uneventful and he was discharged after 12 days. Patients suspected of PM should be examined closely during the differential diagnosis of chest pain, dyspnea, subcutaneous emphysema, and various lung- and heart-induced states. Patients diagnosed with spontaneous PM should be hospitalized for observation because the condition can be associated with complications, including death.

Key Words: COVID-19, Dyspnea, Pneumomediastinum, Complication

Introduction

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which was first identified in Wuhan, China, in 2019. Pneumomediastinum (PM), also known as mediastinal emphysema, is defined as the presence of air or other gas in the mediastinum. PM can be classified as traumatic, spontaneous, or iatrogenic. Spontaneous PM occurs due to increased alveolar pressure, associated with coughing, vomiting, straining, and the Valsalva maneuver, resulting in the spontaneous rupture of alveoli. Spontaneous PM has been associated with asthma, chronic obstructive pulmonary disease, mediastinal infections, mechanical ventilation, excessive exercise, cannabis or cocaine intake, and diffuse interstitial fibrosis[1]. Spontaneous and traumatic PM symptoms are nearly identical, with the most common symptom being chest pain that begins acutely and is localized behind the sternum. The clinical presentation of PM can range from non-specific symptoms to life-threatening respiratory failure. Subcutaneous emphysema in the neck and chest and Hamman's sign during heart auscultation on physical examination can also be caused by serious cardiovascular collapse[2,3]. Here, we present the clinical cases of two patients who tested positive for COVID-19 and presented with spontaneous PM and discuss the possible mechanisms underlying this association.

Case Reports

Case 1

A 58-year-old female patient with a past medical history of asthma presented to the Emergency Department (ED) with dyspnea, hoarseness, myalgia, and cough, lasting for five days. The patient's vital signs were as follows: blood pressure was 120/80 mmHg; tachycardic heart rate of 105 bpm; mild tachypnea, with a respiratory rate of 20 breaths per minute; temperature was 36.2 °C; and transcutaneous oxygen saturation was 84% on room air. Physical examination revealed decreased breath sounds, bilaterally, and soft tissue crepitus in the bilateral clavicle, sternum, and anterior cervical area. Auscultation of the heart revealed a loud crunch-like sound during systole, consistent with "Hamman's crunch." The patient's abdominal examination was normal, as were skin, neurological, and psychiatric evaluations. Routine laboratory tests, cardiac enzymes, and electrocardiogram (ECG) were normal. The RT-PCR analysis for COVID-19 was positive 10 days prior to presentation, and the patient was treated with favipiravir for 5 days. The initial laboratory tests revealed an elevated C-reactive protein concentration of 198 mg/L (reference: < 8 mg/L). Complete blood count showed a leukocyte count of 8.0 × 109/L (reference: 3.5–1.5 × 109/L), with a relative left shift (9% band neutrophils [reference: <37%]) and lymphopenia

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(5% [reference: 10%–50%]). Serum levels of D-dimer and ferritin were both elevated, at 1,082 µg/L (reference: <500 µg/L) and 2,636 µg/L (reference: 23.9–336.2 µg/L), respectively. The ECG showed sinus tachycardia at a rate of 105 bpm without signs of ischemia. The posteroanterior chest radiograph showed bilateral pulmonary infiltrates, PM, and subcutaneous emphysema into the neck. A chest CT scan revealed subcutaneous emphysema, diffuse PM, bilateral diffuse areas of ground-glass opacity, and consolidations, which are all features compatible with COVID-19 pneumonia (Figure 1). No evidence of pneumothorax, pneumopericardium, rib fractures, or tracheal or bronchial injuries was observed. Management was conservative and consisted primarily of supportive care. The patient was transferred to the critical care unit for monitoring and the regulation of medical treatment. The patient was started on anti-infective therapy, with 4,5 g of piperacillin/tazobactam three times a day and 400 mg moxifloxacin once a day, low-dose intravenous methylprednisolone (1 mg/kg) following 1 g of Methylprednisolone for three days, enoxaparin sodium for therapeutic anticoagulation, convalescent plasma therapy 250 cc for two times, and symptomatic supportive therapy, which was adjusted according to clinical conditions. The patient was hemodynamically stable, and high-flow nasal cannula oxygen therapy was attempted. The fraction of inspired oxygen (FiO2) concentration and oxygen flow rate were 100% and 60 L/min, respectively. In critical intensive care, the patient developed multi-organ failure, requiring mechanical ventilation and vasopressor support. On the 10th day, the patient expired due to cardiopulmonary arrest.

Case 2

A 34-year-old male patient with a past medical history of cerebral palsy presented to the ED with dyspnea and cough, lasting for nine days. The blood pressure was 130/75 mm Hg, pulse was 98 beats per minute, respiratory rate was 16 breaths per minute, temperature was 36.5°C, and transcutaneous oxygen saturation was 91% on room air. The breath sounds were normal and equal in both lungs. Routine laboratory tests, cardiac enzymes, and ECG were normal. The RT-PCR analysis for COVID-19 was positive 9 days prior to presentation, and the patient was treated with favipiravir for 5 days. The initial laboratory tests revealed an elevated C-reactive protein concentration of 71,3 mg/L. Complete blood count was completely normal. Serum levels of D-dimer and ferritin were both elevated, at 1,656 µg/L and 5,483 µg/L, respectively. A chest CT scan revealed diffuse PM, bilateral diffuse areas of ground-glass opacity, and consolidations, which are all features compatible with COVID-19 pneumonia (Figure 2). The patient was transferred to the critical care unit for monitoring and the regulation of medical treatment. The patient was started on anti-infective therapy, with 400 mg moxifloxacin once a day, low-dose intravenous methylprednisolone (40 mg/day), enoxaparin sodium for therapeutic anticoagulation, and symptomatic supportive therapy, which was adjusted according to clinical conditions. The patient was hemodynamically stable, and high-flow nasal cannula oxygen therapy was attempted. His progress was uneventful and he was discharged after 12 days.

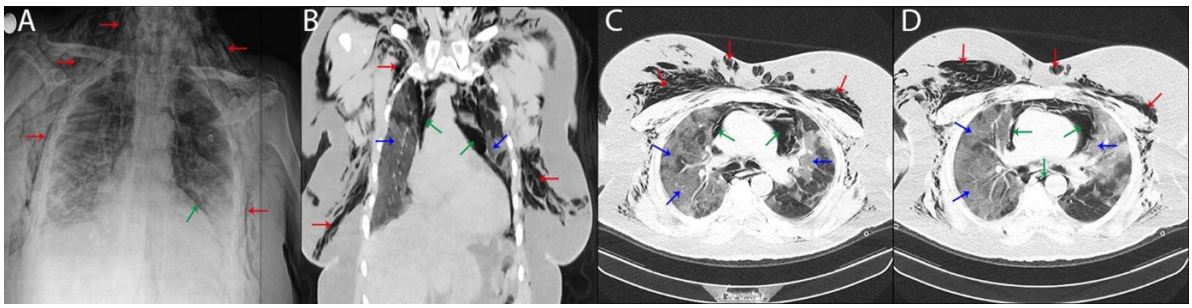

Figure 1: Radiological findings of Case 1. The posteroanterior chest radiograph (A) showed PM (green arrow), and subcutaneous emphysema (red arrow) into the neck. A coronal (B) and axial (C and D) chest CT scans revealed subcutaneous emphysema (red arrow), diffuse PM (green arrow), bilateral diffuse areas of ground-glass opacity, and consolidations (blue arrow).

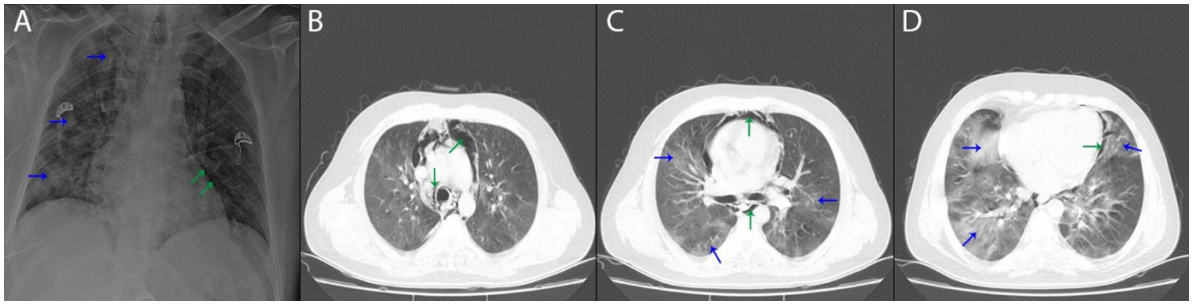

Figure 2: Radiological findings of Case 2. The posteroanterior chest radiograph (A) showed consolidations (blue arrow), and PM (green arrow). An axial (B, C and D) chest CT scans revealed diffuse PM (green arrow), bilateral diffuse areas of ground-glass opacity, and consolidations (blue arrow).

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Discussion

PM, which is defined as the presence of air in the mediastinum and is also referred to as mediastinal emphysema, is a rare condition that typically occurs secondary to trauma, pulmonary barotrauma, or mediastinal infections. Spontaneous PM is most often caused by increased airway pressure, secondary to mechanical ventilation or airway obstruction. The pathophysiology of spontaneous PM in COVID-19 patients is associated with increased distal airway pressure due to repeated coughing spells or the rupture of the alveolar membrane, secondary to diffuse alveolar membrane damage induced by the virus[4]. The symptoms of COVID-19 infection have been widely described, with the most frequent signs including high fever, chills, cough, dyspnea, diarrhea, myalgia, fatigue, expectoration, and hemoptysis[5]. Spontaneous PM associated with COVID-19 has occasionally been reported[4,6-8]. The management of spontaneous PM in the ED consists of the maintenance of respiratory integrity, hemodynamic stabilization, and the initiation of interventions designed to prevent further complications, such as tension PM, mediastinitis, airway obstruction, and pneumothorax. Both of our patients, whose RT-PCR COVID-19 tests were positive, presented with CT findings consistent with the advanced stages of COVID-19 infection. First patient's chest CT showed bilateral confluent areas of ground-glass opacities and alveolar consolidation, PM, and subcutaneous emphysema (Figure 1). Similarly second patient's chest CT scan revealed diffuse PM, bilateral diffuse areas of ground-glass opacity, and consolidations, which are all features compatible with COVID-19 pneumonia (Figure 2). The probable cause of spontaneous PM and subcutaneous emphysema associated with COVID-19 in both of our cases was alveolar damage. Alveolar rupture can occur when a pressure gradient exists between the alveoli and the perivascular sheath. Therefore, in these cases, severe diffuse alveolar damage and episodes of coughing or Valsalva maneuver can lead to spontaneous PM. Subcutaneous emphysema detected on physical examination may occur due to the spread of extra-alveolar air to the neck, face, and anterior chest wall. In addition to subcutaneous emphysema, a physical examination may reveal a crackling sound synchronous with the heartbeat (Hamman's crunch), which is pathognomonic for PM. On chest X-ray of first case, we observed air in the mediastinum and free air in the soft tissues of the cervical area. These findings were consistent with PM and subcutaneous emphysema, respectively. Thoracic CT is the current gold standard for the diagnosis of PM. Bronchoscopy and esophagoscopy examinations can be utilized to distinguish possible ruptures in the bronchial tree and esophagus (Boerhaave's syndrome), respectively, to complement chest CT. Subcutaneous emphysema detected on physical examination in patients occurs due to the spread of extra-alveolar air to the neck, face, and anterior chest wall[9,10]. Spontaneous PM and subcutaneous emphysema are not common for the clinical presentation of COVID-19 infection; however, these presentations can represent potentially aggravating factors during the management of COVID-19 pneumonia. Despite early diagnosis and optimal management, spontaneous PM associated with COVID-19 resulted in the death of our first patient. However, our second patient was discharged home.

Conclusion

Spontaneous PM is a rare complication of viral pneumonia that can progress from a mild clinical condition to a life-threatening clinical situation. Spontaneous PM and subcutaneous emphysema must be recognized as possible complications of COVID-19 infection and deserve early recognition. Even with a normal chest X-ray, a patient suspected of traumatic or spontaneous PM should undergo a CT scan. During the ongoing COVID-19 pandemic, physicians should approach the diagnosis and treatment of this complication with caution.

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