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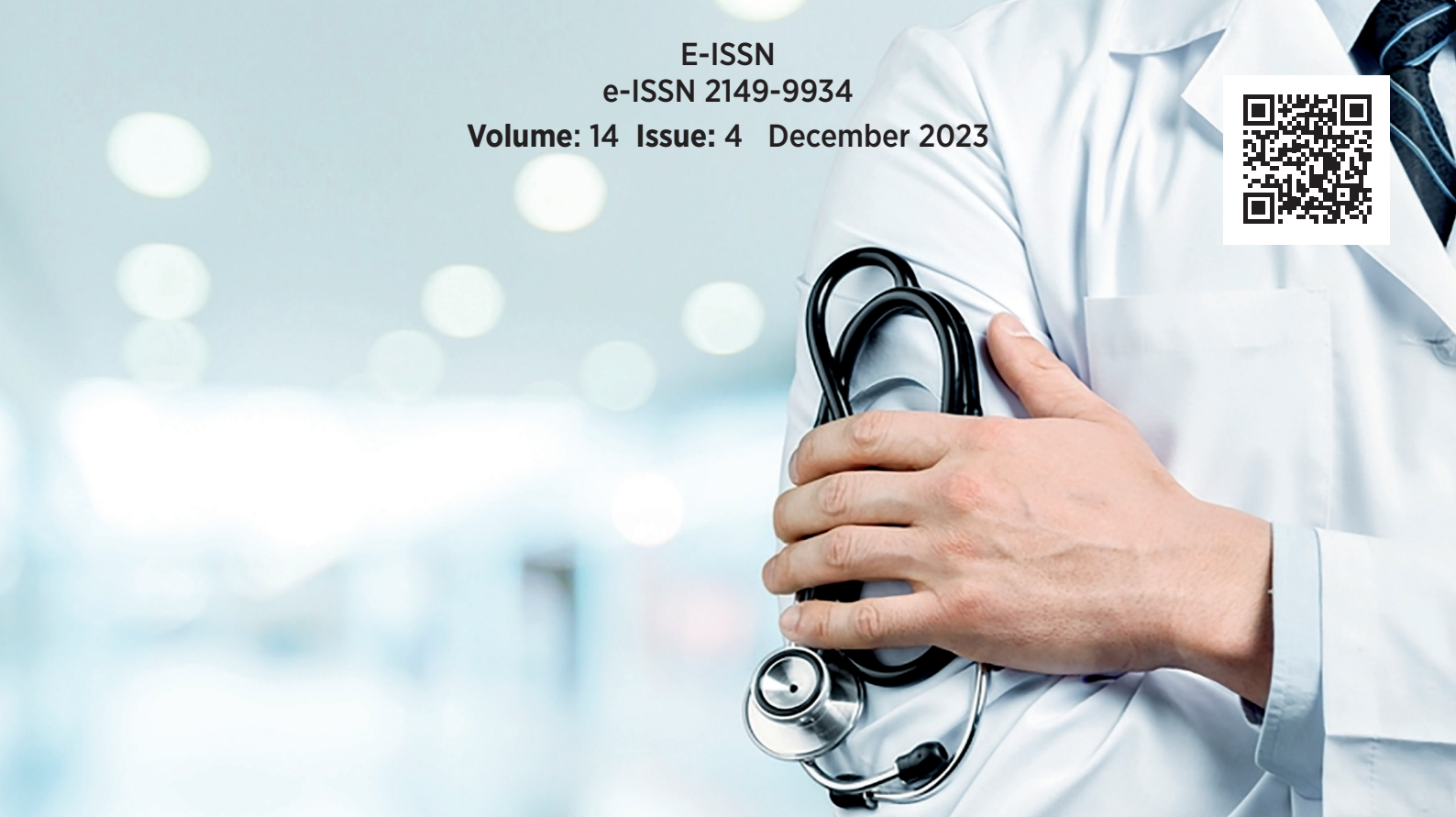

Journal of Emergency Medicine Case Reports

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Contents

Case Report

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Hypokalemic Paralysis Due to Distal Renal Tubular Acidosis, Case Report

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Abstract

Distal renal tubular acidosis (dRTA) is a metabolic disease characterized by hypokalemia, hyperchloremic metabolic acidosis and urine pH above 5.5. These findings may be accompanied by hypercalciuria, nephrocalcinosis, nephrolithiasis, jaundice, osteomalacia or rickets in children. Although hypokalemia is frequently seen as a laboratory finding in dRTA, weakness, which is the clinical finding of this deficiency, is rare. A 33-year-old female patient was brought to the emergency department (ED) with complaints of weakness, loss of strength in the extremities, and difficulty in breathing. Laboratory analyzes of the patient revealed metabolic acidosis and hypokalemia. Urea and creatinine values were normal. The patient was admitted to the internal medicine department with a preliminary diagnosis of dRTA and hypokalemic paralysis. Initially, parenteral infusion of KCl and NaHCO3 was administered in the treatment. In the follow-up of the patient, it was observed that hypokalemia and metabolic acidosis improved from the 3rd day and clinical findings improved within 36 hours following the replacement therapy. dRTA, which is rare in adults, is among the secondary causes of hypokalemic paralysis. dRTA should be considered among the differential diagnoses in the presence of hypokalemia and metabolic acidosis in patients presenting with bilateral weakness.

Keywords: Renal tubular acidosis, hypokalemia, paralysis

Introduction

Hypokalemic paralysis is an uncommon, life-threatening syndrome with widespread muscle weakness, including respiratory muscles, and hypokalemia. If it is diagnosed and treated appropriately, it resolves without sequelae (1). Periodic paralysis is distinguished by a normal potassium level, except for attacks. In the presence of normal anion gap metabolic acidosis, renal tubular acidosis (RTA) should be considered among the differential diagnoses. In this case, secondary diseases that may cause RTA should also be investigated. RTA Type 1 and 2 are seen with hypokalemia, while RTA Type 4 is associated with hyperkalemia. Clinical findings may present with nonspecific findings such as constipation, weakness, growth retardation in childhood, nausea-vomiting, polyuria-dehydration, as well as presentations with complications related to the kidney and musculoskeletal system (2, 3). Complications associated with the kidney; nephrocalcinosis, urolithiasis (which may be the first initial finding in adults), chronic interstitial nephritis and severe hypokalemic crisis. During the hypokalemic crisis, the patient may apply to the emergency department (ED) with the symptoms of dehydration, shock, arrhythmia, vomiting, flaccid weakness, respiratory distress, drowsiness, and coma (4). Here, we present a female patient who presented with acute muscle weakness and dyspnea and was diagnosed with hypokalemic periodic paralysis due to distal RTA (dRTA), with clinical and laboratory findings.

Case Report

A 33-year-old female patient was brought to the ED by the 112 with complaints of weakness, loss of strength in the arms and legs, and difficulty in breathing. The patient, who has a history of type 2 diabetes mellitus and hypothyroidism, was using metformin 2x850 mg and levothyroxine 50µgr/day. The patient had no smoking or alcohol use. There was no pregnancy history. In her anamnesis, it was learned that she had previously received a vitamin B12 ampoule injection due to numbness in her arms. It was learned that the patient felt weakness and numbness especially in his upper extremities for 2-3 days, and she admitted to the orthopedics outpatient clinic with the thought of nerve compression. Blood gas analysis revealed pH: 7.15, pCO2: 32 mmHg, HCO3: 10.9 mmol/L, base excess (BE): -16.8 mmol/L, potassium (K): 2.02 mmol/L, ionized calcium (Ca): 1.39 mmol/L, chloride (Cl):115 mmol/L. In laboratory findings, hemoglobin 13.88 gr/dL, hematocrit 41.9%, white blood cell 14.400/mm3, platelet 358000/mm3, glucose 119 mg/dL, urea 36 mg/dL, creatinine 0.69 mg/dL, sodium (Na) 138 mmol/L, K:

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2.06 mmol/ L, Cl: 116 mmol/L, Creatine Kinase 574 u/L were detected and liver function tests were normal. In urine analysis; pH 7, protein (-), Na: 61 mmol/L, K: 10 mmol/L Cl: 75 mmol/L Ca: 9.2 mmol/L and urine density was 1002. The patient's electrocardiography (ECG) was normal. The patient was admitted to the internal medicine department with the preliminary diagnosis of dRTA and hypokalemic paralysis. Thyroid function tests, plasma renin, aldosterone values were within normal limits, Parathormone: 8.3 ng/L (15-68.3) low, vitamin B12: >2000ng/L high, anti-DNA, RF, AMA, ASMA, anti-SSA, anti-SSB, anti-Jo-1, CCP, c-ANCA, p-ANCA, anti-ds-DNA were negative, ANA was (++) granular appearance. No pathological finding was detected in urinary ultrasonography. In the treatment, a parenteral 80 mEq/day KCl and 150 mg/kg NaHCO3 infusion was administered initially. In the follow-up of the patient, it was observed that his hypokalemia and metabolic acidosis improved from the 3rd day. During the follow-up period, the potassium values are respectively; 2.7, 2.2, 2.6, 3, 3.51, 3.58 mmol/L were detected. After the initial KCl and NaHCO3 parenteral infusion, oral effervescent potassium tablet 2x40 mmol was administered. It was observed that clinical findings improved within 36 hours following the replacement therapy. Sodium hydrogen carbonate and oral potassium treatment containing potassium citrate and potassium bicarbonate were prescribed and she was discharged with the recommendations of rheumatology outpatient clinic control. An informed consent form was obtained from the patient for this case report.

Discussion

Potassium, which is the main cation of the cell, is of great importance in the stimulation of muscle, nerve and myocardial cells in addition to its many functions in the body (5). Hypokalemia, defined as a plasma potassium level below 3.5 mEq/L, is a common clinical problem. Findings vary between individuals. It rarely causes symptoms unless it falls below 3 mEq/L (4). Weakness, muscle aches and loss of strength in the lower extremities are common. Cerebrovascular diseases are considered primarily in patients presenting with paresis and loss of strength in the ED (6). However, in severe hypokalemia, clinical pictures ranging from progressive loss of strength, hypoventilation due to the involvement of respiratory muscles, and complete paralysis can be observed (7). The coexistence of hypokalemia and metabolic acidosis can be seen in diabetic ketoacidosis, and Type 1 and Type 2 RTA (7).

Hypokalemic periodic paralysis is a disease characterized by decreased serum potassium level due to primary and secondary causes and accompanying transient acute flaccid paralysis. Secondary causes include dRTA, although rare.

In Type 1 RTA, which is the distal type, there is an inability to remove the daily acid load. If alkali treatment is not applied, hydrogen ion accumulation gradually increases and the plasma bicarbonate concentration may fall below 10 mEq/L. Despite acidosis, the urine pH is above 5.5. Serum potassium levels are variable. Due to chronic acidosis, both bone resorption and renal tubular reabsorption of calcium are affected and hypercalciuria is observed. Therefore, nephrolithiasis and nephrocalcinosis often develop (3). In our patient, the urinary calcium level was also high. In type 2 proximal RTA, hypokalemic metabolic acidosis develops due to the defect in bicarbonate reabsorption in the proximal tubules. Since the functions of the distal tubules are normal, partial absorption of bicarbonate occurs and blood bicarbonate levels are higher than in type 1. It is usually associated with other proximal tubular reabsorption disorders. With the administration of bicarbonate, the urine pH becomes alkaline in type 2 RTA, while the absence of a significant change in urine pH in type 1 RTA may help in the differential diagnosis. Kidney stone formation is not seen in type 2. Type 3 RTA is characterized by hyperkalemic hyperchloremic acidosis. It develops due to aldosterone insufficiency or resistance to the effect of aldosterone in the tubules (8, 9). Our patient, who was examined from the beginning for hypokalemia and hyperchloremic metabolic acidosis, also has a urine pH above 5.5 and a high urinary calcium, which is compatible with Type 1 RTA. In some patients who presented with hypokalemic paralysis and were found to have dRTA, underlying causes were found to be rheumatoid arthritis, collagen tissue diseases such as systemic lupus erythematosus (sometimes with hyperkalemia), and the use of amphotericin B, lithium, ibuprofen or some plants (7,8,10). Therefore, all patients diagnosed with RTA should be investigated for other diseases that may cause RTA. Our patient's clinic and examinations are not compatible with collagen tissue diseases in terms of diseases that may cause dRTA, and he did not use any drugs or herbs. Failure to define family history caused the case to be evaluated as sporadic. Treatment is with bicarbonate and potassium replacement. With bicarbonate, the symptoms disappear. In addition, with treatment, the development of kidney failure is prevented or kept at the same level. Administration of 80-200 mg/kg (1-3 mEq/kg/day) NaHCO3 in the dRTA eliminates acidosis. As a matter of fact, bicarbonate and potassium replacement were applied in our patient and clinical findings improved.

Conclusion

Electrolyte disturbances, especially hypokalemia, should be considered in patients presenting to the ED with bilateral weakness. The possibility of dRTA, which is rare in adults, should be considered in cases presenting to the ED with paresis in the extremities with or without respiratory distress.

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Case Report

Journal of Emergency Medicine Case Reports

Detection of Sternum Fracture with POCUS Despite Computed Tomography Findings Reported as Normal: Case Report

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Abstract

Sternal fractures are extremely rare in children. It often develops after high-energy chest trauma. Many methods other than ultrasonography are used in the diagnosis of sternal fractures. However, Point-of-care ultrasound (POCUS) can outperform other methods due to its ease of use, less radiation, and fast results.A thirteen-year-old male patient was brought to the Pediatric Emergency Department by ambulance due to chest pain that started following blunt chest trauma after falling from a height of 160 cm. The patient's respiratory and cardiovascular examinations were normal in his initial evaluation in the trauma room. On palpation, there was local tenderness in the middle 1/3 of the sternum. No pathology was detected in Extended-Focused Assessment with Sonography in Trauma (E-FAST). The portable postero-anterior chest radiograph was normal. POCUS was performed after the patient did not respond to analgesic treatment and had local sensitivity on the sternum. A sternal fracture was detected. Suspicious cortical irregularity was detected in repeated lateral chest X-ray and thorax Computed Tomography (CT) was reported as normal by the radiology department. When CT was re-evaluated, a greenstick fracture was observed in the sternum body. The patient was discharged without any complications.In this article, the importance of diagnosing a sternal fracture case whose CT was reported as normal with POCUS and integrating rapid, noninvasive and radiation-free ultrasonography into the physical examination will be discussed in managing these patients.

Keywords: Sternal fracture, trauma, POCUS

Introduction

The incidence of sternum fractures in children is reported to be between 0.5-3%. Although it often occurs with a high-energy trauma mechanism such as motor vehicle accidents, it can also be seen as a result of blunt traumas directly to the chest area without intra-thoracic injury or flexion-compression injuries without spinal injury (1).

The first question that often comes to mind when suspecting a sternal fracture is whether pulmonary and/or cardiac injury accompanies this fracture due to the anatomical location of the sternum. In this case, it is necessary to find answers to which radiological method should sternal fracture be demonstrated, and whether further imaging should be performed in the presence of fracture (1-2).

The purpose of this case is to emphasize the importance of integrating rapid, noninvasive, and radiation-free ultrasonography into physical examination in emergency departments. Point-of-care ultrasound (POCUS) may help diagnose sternum fractures in children.

Case Report

A thirteen-year-old male patient was brought to the Pediatric Emergency Department (PED) by ambulance due to chest pain that started following blunt chest trauma after falling from a height of 160 cm. In his initial triage evaluation, he was conscious and his vital signs were normal. In his initial evaluation in the trauma room, the patient's respiratory and cardiovascular examinations were found to be normal, with active chest pain. On palpation, there was local tenderness in the middle 1/3 of the sternum. No pathology was detected in the Extended-Focused Assessment with Sonography in Trauma (E-FAST). Portable postero-anterior (PA) chest radiograph was normal. Electrocardiography (ECG) revealed sinus rhythm and 1 mm ST elevation in leads V2, V3. Cardiac biomarkers were normal. Oral analgesic was given. The PED and emergency department physician performed POCUS to evaluate the sternal fracture, as no fracture was observed in the PA chest X-ray of the patient who did not respond to analgesic treatment and whose local

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sensitivity on the sternum continued. A sternal fracture was detected (Figure 1 determined with POCUS). No significant pathology was observed in bedside echocardiography (ECHO). Upon detection of a sternal fracture, chest lateral X-ray and thorax CT were taken to the patient. Suspected cortical irregularity was detected in the chest lateral X-ray (Figure 2). Since the detection of the fracture with POCUS is user dependent and the suspected irregularity in the lateral chest radiograph was taken later, a thorax CT was performed to confirm the diagnosis and exclude additional serious injuries. And the thoracic CT was reported normal. When CT was re-evaluated, a greenstick fracture was observed in the sternum body (Figure 3). Cardiac monitoring was performed throughout the observation period. No pathology was observed in ECG and cardiac markers during follow-up. The patient was discharged with a non-steroidal anti-inflammatory drug without any complications.

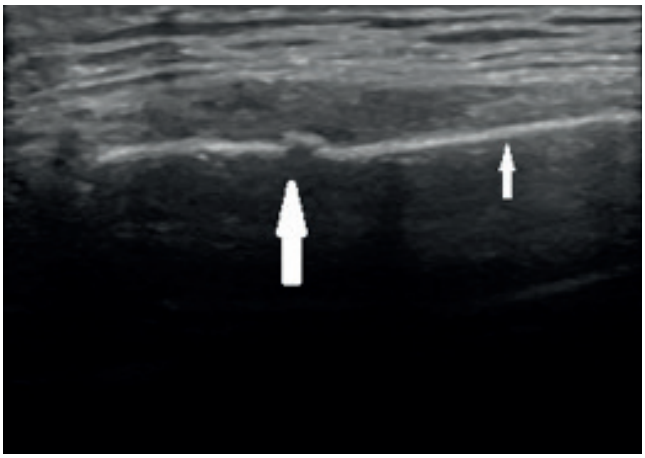

Figure 1. Sternal image with linear probe. Cortical irregularity (thick arrow) on the sternum (thin arrow)

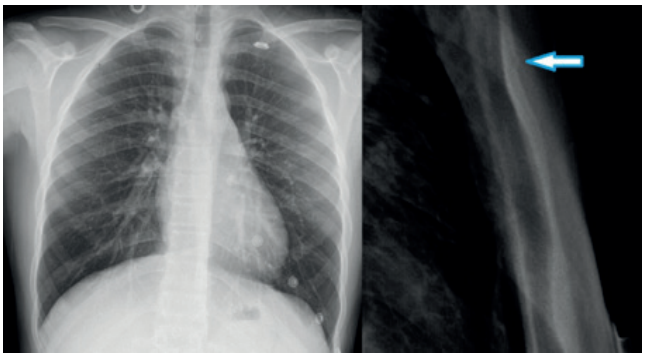

Figure 2. Normal appearance of the sternum in the anterior-posterior chest X-ray, irregularity in the cortical bone in the middle 1/3 of the sternum in the lateral chest X-ray (blue arrow)

Sonographic examination:

The sonographic examination was performed by a pediatric emergency and an emergency medicine specialist who were unaware of each other and had two years of POCUS experience. The sternal fracture was visualized with a high-frequency linear probe (3.6-12 MHz), and cardiac examination with a phased array probe (1-4 MHz). The sonographic examination was completed supine with the GE Logic E (GE Medical Systems, USA).

Discussion

The approach in patients with chest trauma depends on the severity of the trauma and the underlying suspected injury. Life-threatening conditions such as tension pneumothorax, hemothorax, cardiac tamponed, or great vessel injury in children with severe thoracic trauma and respiratory or circulatory disorders at the time of first presentation should be immediately identified and stabilized quickly. The exact extent of the injury can be determined later by a detailed history, careful physical examination, and diagnostic tests (3).

Imaging modalities that can be used to evaluate children with chest trauma include X-ray, POCUS by an experienced clinician, ECHO, and CT. In many studies, it has been reported that abnormal respiratory system auscultation findings and tenderness on palpation of the chest wall may indicate thoracic injury (4).

An X-ray should be performed in the primary evaluation of children with major thoracic trauma or multi-trauma (4). It is easy to access, inexpensive, and can also identify many clinically significant life-threatening injuries. Children with normal blood pressure, Glasgow coma scale scores of 15, and no localizing findings on chest examination are unlikely to have pathology in the radiograph. Therefore, imaging may not be required in children with isolated

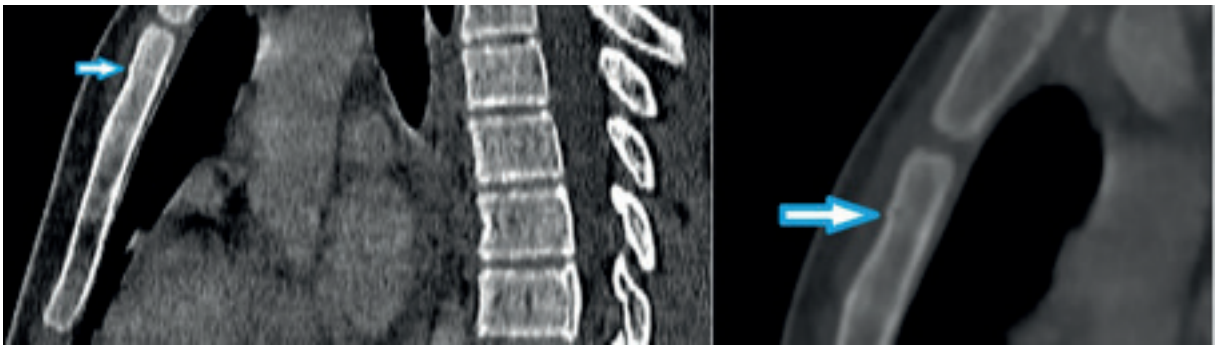

Figure 3. CT image of the torus fracture on the cortical bone in the middle 1/3 of the sternum (blue arrows)

minor thoracic trauma (4). However, in isolated blunt chest trauma, if the patient describes localized pain, especially in the sternal region and the pain increases with palpation, it should be considered that there may be a sternal fracture (1-2). We first evaluated our case as an isolated thorax injury. For this reason, we wanted to exclude life-threatening situations by taking only postero-anterior radiographs in the first evaluation. We performed POCUS because his pain continued and he had localized findings during follow-up. Since a fracture and ECG changes were detected, we wanted to confirm the diagnosis and perform a CT scan. Although the CT report was normal, when we re-examined the tomography, we saw the fracture on the tomography and confirmed our diagnosis. Although the guidelines do not recommend further examination, we performed a CT scan considering that it is a rare fracture in the childhood age group and that thoracic injuries in children may cause internal organ injuries without bone fractures, as stated in the literature. We wanted to discuss this case for this purpose.

There is no standard protocol for diagnostic examination and treatment. Diagnosis of sternal fracture in adults can be made by X-ray. Still, the sensitivity is limited and some studies have reported that X-ray misses the diagnosis in 25-50% of cases. When evaluated together with lateral radiographs, the sensitivity increases slightly (5).

Ultrasonography may be more sensitive and specific than X-ray in the diagnosis of sternal fractures, but it depends on the experience of the person doing it (5). There is no definite information about which patients with sternum fractures should have a CT scan. Although it is said that CT is not required in patients with non-displaced, isolated sternal fractures and no associated multiple trauma, thoracic CT imaging continues to be the preferred method in adults when fractures are detected in X-ray or ultrasonography (6). The CT scan for children remains an essential life-saving tool for diagnosing serious thoracic injuries, as long as it is performed properly and justified clinically. However, ultrasonography remains an individualized diagnostic tool with a smaller scanning area than other imaging modalities such as MRI, X-ray or CT (7).

X-ray is the first method used to diagnose sternum fractures in children. However, the sternal fracture may be overlooked in the evaluation of anteroposterior chest radiographs, especially in severely traumatized cases (7). However, in addition to X-ray and CT, USG should also be considered to demonstrate a sternum fracture. Recently, the success of USG in the diagnosis of sternal fracture has also been reported (1-2, 7). USG is overlooked by X-ray in diagnosing fractures, as it reflects the image of the cortical surface very well and allows acceptable cortical changes to be seen clearly (8). In a study, the sensitivity and specificity of X-ray were 70.8% and 75%, while the sensitivity and specificity of USG were 100% and 95% (9). Another study with 31 patients showed that multi-detector CT was 100% sensitive, but axial slices were 65% and coronal slices 59% sensitive (5).

It should be evaluated by long-axis scanning technique using a high-frequency linear probe for superficial, subcutaneously located bones and a curved probe for deeper bones. It can best show long bone fractures. On POCUS, fractures appear as a marked deterioration in the echogenic line corresponding to the cortical surface of the bone (10). For sternal fractures, scanning should be performed along the entire sternum in both the longitudinal and transverse planes, with the patient lying supine. POCUS is helpful in children who can show the sensitivity area, and the sensitivity area should be evaluated by comparing it with other areas. It should be borne in mind that the ossification zones in children fuse at about 25. Therefore, ossification sites should not be confused with fractures. While the edges of the ossification centers are round; Sonographic distinction can be made with the appearance of irregular and notched fractures (8).

Conclusion

Since the probability of intrathoracic injury is low in patients with isolated sternal fractures, aggressive investigation is not required. Stable patients can be followed safely without hospitalization. POCUS can be superior to other methods due to its ease of use, lack of radiation risk, and immediate results. Complementary advanced diagnostic imaging modalities may be performed if clinically relevant intrathoracic injuries are suspected.

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Case Report

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A Rare Case of Rickettsia and Herpes Simplex Virus 1 Co-infection in a Male Patient with Meningoencephalitis

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Abstract

Herpes simplex viruses (HSVs) belong to the *Herpesviridae* family. Close contact is the primary mode of transmission for both HSV-1 and HSV-2, leading to a persistent lifelong infection. HSVs are widely recognized as causative agents of viral infections affecting the central nervous system, capable of presenting as both meningitis and encephalitis. Herpes simplex virus type 1 (HSV-1) is the predominant viral cause of encephalitis, accounting for the majority of cases. Here, a rare co-infection case of meningoencephalitis, associated with HSV-1 and rickettsia is described. A 42-year-old man presenting with non-remitting headache for 6 days, fever, sweating, and muscle aches was admitted to the Emergency Department. His Weil-Felix test was positive for Proteus OX2 indicating rickettsial infection. Therapy started promptly however patient's condition deteriorated. Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis, and elevated protein concentration. CSF molecular analysis was positive for HSV-1. His cranial MRI indicated cytotoxic edema and gyral enhancement at the right temporal lobe. He was administered acyclovir for 14 days during his hospital stay and was successfully discharged. This case report highlights that HSV-1 meningoencephalitis can co-occur with rickettsia infection in immunocompetent individuals, and co-infection with other agents should always be considered to avoid the progression of the disease.

Keywords: Rickettsia, HSV-1, co-infection, meningoencephalitis, polymerase chain reaction

Introduction

Herpes Simplex Virus (HSV) is a viral agent that causes infections in humans which can result in the formation of painful blisters or ulcers. The infection persists throughout a person's lifetime, and is characterized by periodic re-activation at the initial site of infection [1]. HSV is classified into two distinct types, Herpes simplex virus 1 (HSV-1) and Herpes simplex virus 2 (HSV-2). HSV-1 is mainly spread through oral-to-oral contact and commonly leads to orolabial herpes, also known as cold sores. In addition, HSV-1 can cause less common conditions such as keratitis, ocular complications, and encephalitis. While HSV-1 genital infection resulting from oral-to-genital contact is increasingly observed, re-activation is less frequent compared to HSV-2. HSV-2 is primarily transmitted through sexual contact and causes genital herpes [2]. HSV has the capacity to invade the central nervous system (CNS) during primary infection or through latent state re-activation. Following the primary infection, HSV can become latent in lymphoid tissue or in ganglia. Typically, HSV-1 travels through the trigeminal and/or olfactory ganglia to the CNS, where it duplicates and has the potential to induce encephalitis [3]. Among the herpes viruses currently identified, the neurotropic HSV-1 can invade both CNS and the peripheral nervous system (PNS). HSV encephalitis (HSVE) is a life-threatening medical emergency. The worldwide incidence of HSVE is estimated to be 2-4 cases/1,000,000 [4].

Mediterranean spotted fever (MSF) is a tick-borne rickettsiosis of the spotted fever group (SFG), endemic in the Mediterranean basin. *Rickettsia conorii* is a vector-borne, obligate intracellular bacterium which is the causative agent of MSF, mainly in the Mediterranean area and the surrounding countries [5]. The arthropod vector of this bacterium is known to be the brown dog tick *Rhipicephalus sanguineus*. A recent study in Cyprus has shown that the number of rickettsiae positive patients have gradually increased between 2016 and 2020 [6]. While typical symptoms of MSF are diverse and include fever, maculopapular rash, and a characteristic eschar ("tache noire"), atypical clinical features and severe multi-organ complications may also be present. Meningitis, encephalitis, and acute disseminated encephalomyelitis are the primary neurological conditions frequently observed in cases of rickettsial infections [7].

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Journal of emergency medicine case reports. 2023;14(4): 81-83

In this case report, we present a rare condition of meningoencephalitis due to HSV-1 and rickettsia co-infection in an immunocompetent individual.

Case Report

A previously healthy 42-year-old man, presenting with non-remitting headache for 6 days, fever, sweating, headache and muscle aches was admitted to the Emergency Department at Near East University Hospital, Cyprus. He had used azithromycin previously. He reported not feeling well the night before admission, and had a shoulder fracture after he fell at home. Body temperature was 39°C on admission. On the first physical examination, the patient had decreased level of consciousness, confusion, tendency to sleep, and difficulty in cooperation. In the physical examination of the patient, neck stiffness and Kernig and Brudzinski signs were evaluated as negative. The patient indicated having had tick bites earlier although none were observed upon admission. He also noted living in an area where Phlebotomus flies were abundant. No tissue related to rickettsiosis was found during the examination. The patient was consequently referred to the Infectious Disease Department.

Laboratory test results showed an increased C-reactive protein level (2,05 mg/dL), high peripheral leukocyte cell count (11,1 x 103/µl), high procalcitonin (0,22 ng/mL), and a high aspartate aminotransferase (serum glutamic-oxaloacetic transaminase, AST-SGOT) level of 49 U/L. The Weil-Felix agglutination test was performed for OX 19, OX 2, OXK strains of Proteus species. The serum Weil-felix (Proteus) test was positive for Proteus OX2 at 1/640. Patient's blood, urine and rectal cultures, brucella agglutination (Rose Bengal) and Grubel-Widal salmonella tube agglutination tests were negative. The patient was immediately treated with 2x1 500 mg ciprofloxacin, however his overall condition deteriorated several hours later.

Patient's serum creatinine levels were high (1,51 mg/dL) therefore brain magnetic resonance imaging (MRI) could not be performed. Patient's creatinine levels were lowered to 0.69 mg/dL with fluid intake and MRI was consequently performed. His cranial MRI indicated signal increase at right temporal lobe on T2W images (Figure 1) and gyral enhancement on T1W C+ images (Figure 2).

Lumbar puncture (LP) was immediately performed and cerebrospinal fluid (CSF) was collected. CSF biochemical analysis revealed high glucose (104 mg/dL) and elevated protein levels (6313 ml/dL) due to the traumatic LP. The cellular CSF analysis indicated pleocytosis, with a leukocyte cell count of 280,000 cells/µL, 90% lymphocytes, suggestive of a possible viral aetiology. RT-qPCR analysis of CSF was performed using QIAstat-Dx Meningitis/Encephalitis Panel and was positive for herpes simplex virus type-1 (HSV-1) (Figure 3).

His HSV-1 IgG was also found to be positive with 27,94 RU/mL (normal range: 0-16 RU/mL), while HSV-1 IgM

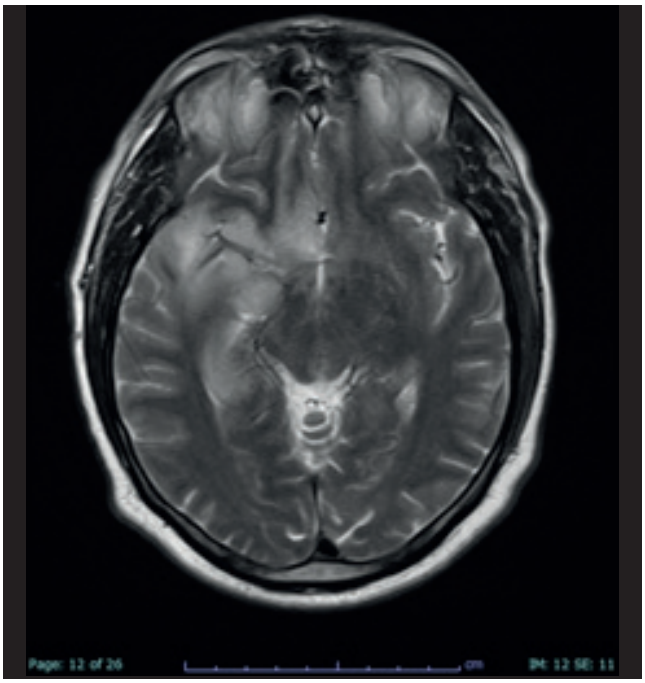

Figure 1. Patient cranial MRI axial T2W image showing hyperintensity at right temporal lobe, due to cytotoxic edema

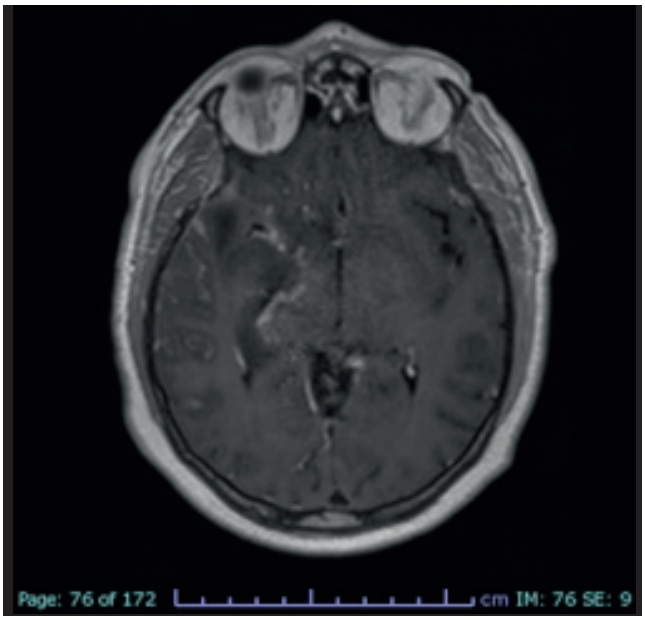

Figure 2. Patient cranial MRIaxial T1W image with IV contrast material indicating gyral enhancement

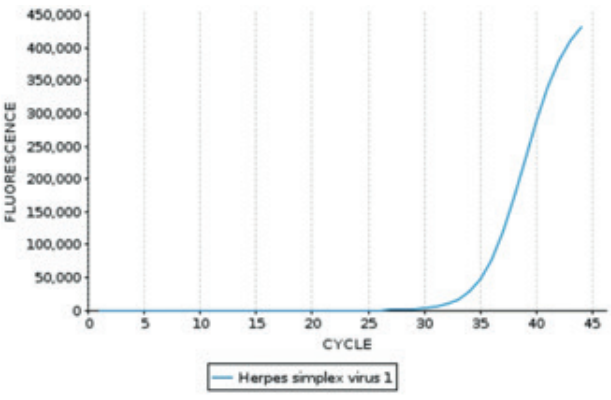

Figure 3. RT-qPCR amplification of Herpes Simplex Virus-1 DNA in the patient CSF sample

was negative. He was immediately administered with 750 mg of acyclovir intravenously three times daily (2250 mg total) and 2x1 1 g ceftriaxone intravenously for 14 days. The patient recovered successfully within two weeks and was followed up to ensure no neurological sequelae.

Discussion

The current study represents a rare case of co-infection of HSV-1 and rickettsia in a patient presenting non-remitting headache, fever, sweating, headache, and muscle aches. Analysis of CSF revealed a positive HSV-1 PCR test, lymphocytic pleocytosis, and elevated protein concentration. The patient had positive HSV-1 IgG levels which indicated HSV-1 re-activation, resulting in HSV-1 encephalitis. Furthermore, his serology tests also showed a rickettsia co-infection.

Despite the Enteroviruses being the most common agents, HSVs are also among the main agents responsible for viral meningitis and may cause neurological morbidity [8]. HSV can lead to diverse neurological symptoms such as meningitis, myelitis, polyradiculopathy, and encephalitis. While these syndromes may indicate primary HSV infection, they typically arise from the re-activation of the latent virus in the sensory ganglia. Apart from viral encephalitis, HSV-1 is infrequently responsible for meningoencephalitis and may lead to permanent neurologic sequelae. In the diagnosis of viral meningitis, detection of HSV DNA in CSF via polymerase chain reaction (PCR) is considered as the gold standard with high sensitivity [9]. In the case of HSV encephalitis, as symptoms are not specific to the HSV-1, prompt diagnosis and therapy are essential for reducing mortality and morbidity.

Rickettsia is an obligate bacterium that causes flea, tick, or mite-borne diseases. In addition, they can cause neurological illnesses including encephalitis, meningitis, and acute disseminated encephalomyelitis. In cases of neurological involvement, convulsion, altered sensorium, fever, behavioural changes, confusion, headache, rash, and semi-comatose conditions have been reported in the literature. Serological evaluation of different rickettsial species is usually performed by the Weil-Felix test which is an easy and rapidly performed test that can be implemented to confirm the diagnosis of rickettsial infections. Although the test has low sensitivity and specificity, it is simple and cost effective, hence can guide the clinicians for the appropriate treatment.

This report presents a rare case of rickettsia and HSV-1 co-infection without blisters or ulcers. The patient presented with diverse symptoms not only specific to HSV-1 encephalitis. He did not have any travel history or rash to suggest rickettsial infection but he indicated living in a rural area where *Phlebotom*us flies were frequently observed. Following the LP procedure, laboratory investigations revealed CSF pleocytosis with mononuclear predominance, suggesting a viral infection. A positive RT-PCR test for HSV-1 confirmed the viral aetiology assumption and the resulting diagnosis was HSV-1-associated encephalitis. The elevated protein levels observed in the CSF analysis have been reported to be commonly detected in HSV-1-associated encephalitis cases [8].

As the patient was immunocompetent and had no history of recent symptoms, the serological analysis was suggestive of HSV re-activation. It can be hypothesized that HSV-1 re-activation occurred due to the rickettsial co-infection. However, the opposite is also conceivable, in which infections due to both agents occurred simultaneously. In the literature, a similar viral co-infection with *Rickettsia helvetica* and HSV-2 has been documented in a meningoencephalitis case [10].

In conclusion, the early diagnosis of encephalitis is essential for the rapid implementation of appropriate therapy. This study highlights the significance of investigating the presence of rickettsial infections in endemic areas for meningitis cases of uncertain aetiology. During diagnosis, co-infections should also be always considered by the clinicians.

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Case Report

Journal of Emergency Medicine Case Reports

Traumatic Left Tension Pneumothorax with Concomitant Congenital Diaphragmatic Hernia

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Abstract

Blunt thoracic trauma with clinical identification of a tension pneumothorax necessitates immediate decompression. Concomitant diaphragmatic hernia is challenging and rather uncommon; there is potential bowel perforation due to the thoracocentesis. We report a case of traumatic left tension pneumothorax, prompting an immediate left thoracostomy and an incidental finding of an uncomplicated congenital diaphragmatic hernia from a chest x-ray and computed tomography (CT) thorax. Early identification with clinical assessment and bedside imaging is essential, and surgical repair is the definitive management.

Keywords: Thoracic injuries, pneumothorax, diaphragmatic hernia

Introduction

Blunt thoracic trauma represents approximately 15% of all emergency department visits worldwide and is the second leading cause of death in a motor vehicle accident after traumatic brain injury (1). Tension pneumothorax is a rapidly lethal condition that should be diagnosed on clinical grounds during the initial primary survey. A finger thoracostomy followed by rapid chest tube insertion is life-saving.

Congenital diaphragmatic hernia (CDH) usually presents in childhood but may not be diagnosed in healthy adult patients with good reserves. Presentation at a later age is rarely reported in the literature. Traumatic diaphragmatic rupture only occurs in 0.8–1.6% of blunt trauma cases, and it frequently manifests asymptomatically at the time of injury (2). However, congenital or traumatic injuries are clinically undifferentiable if no previous record or imaging is available.

There have only been a few cases described of simultaneous traumatic pneumothorax and diaphragmatic hernias. The physician's goal in treating a blunt thoracic injury is to identify any actual or impending life-threatening illnesses, treat them, and avoid complications. Early identification of pathologies with bedside ultrasound and imaging helps to prevent complications.

Case Report

A 24-year-old Malay gentleman was brought to the emergency department after being involved in a motor vehicle accident. The car skidded and hit a tree on the roadside. The patient wore a seatbelt on the passenger seat, but the airbag was not deployed. The fireman evacuated him from the wrecked car. Upon retrieval, he was alert but complained of left-sided chest pain and difficulty breathing. He appeared tachypnic and in shock, with a blood pressure of 79/59 mmHg and an oxygen saturation of 86% on room air. He was put on a high-flow oxygen mask and given an intravenous fluid bolus.

Upon arrival at the emergency department, the primary survey revealed a left-sided tension pneumothorax. The trachea shifted to the right side; there was a decreased chest rise, hyperresonance on percussion, a positive chest spring on palpation, and a reduced air entry on auscultation over the left chest. Bedside lung ultrasound noted the absence of a sliding sign over the left lung and a partial absence of a hyperechoic line representing a normal diaphragmatic profile. Following an immediate left-sided finger thoracostomy over the safety triangle, there was a gushing of air, and chest tube insertion revealed fluctuations and bubbling in the underwater seal.

The chest x-ray showed a left chest tube in situ and residual left pneumothorax but also noted a rounded hyperdense ring lesion in the lower zone (Figure 1). The patient complained of persistent left-sided chest pain and colicky abdominal pain and was urged to pass motion despite regular analgesia and well-functioning chest drainage. Contrast-enhanced computed tomography (CECT) thorax and referral to the surgery team followed the suspicion of a concurrent traumatic diaphragmatic hernia (TDH).

The CECT thorax showed focal thickening and discontinuity of the central left hemidiaphragm. There

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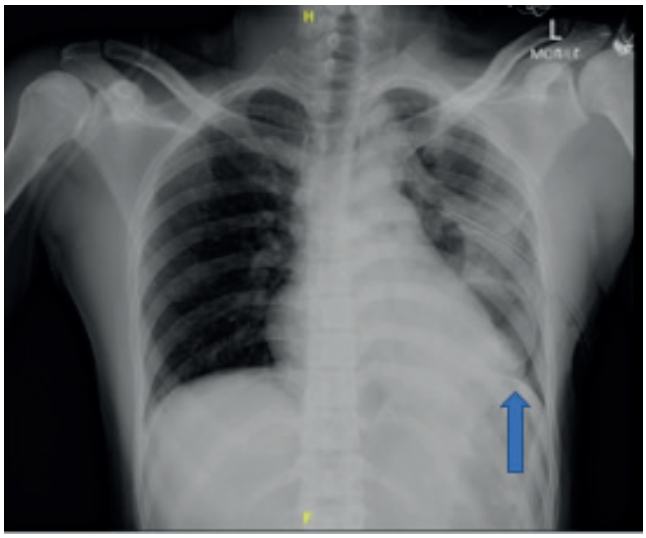

Figure 1. Chest x-ray post left chest tube insertion shows left chest tube in site, residual left pneumothorax and a rounded hyperdense ring seen in the left lower zone (blue arrow)

was intrathoracic herniation of the stomach and peritoneal fat and focal constriction of the stomach at the herniation site. The herniated stomach was distended with the air-fluid level seen within. No diaphragmatic or peri-diaphragmatic contrast extravasation was identified. There was a left pneumothorax with evidence of a left chest tube traversing the left fifth intercostal space. Consolidation involving the left upper and lower lobes was suggestive of a pulmonary contusion. There was no haemothorax or pleural effusion bilaterally, and there was no significant mediastinal shift. Left second- to seventh-rib fractures were seen (Figure 2).

The features suggested a traumatic left diaphragmatic rupture or injury, with associated left pneumothorax, left pulmonary contusion, and multiple left rib fractures. The patient was sent for a surgical laparotomy and diaphragmatic plication. During the surgery, a smooth, well-defined, non-traumatic defect measuring 6 cm by 4 cm and involving part of the stomach and omentum was found at the left posteromedial diaphragm. The hernia contents were reduced, and the diaphragm was repaired. The patient recovered well post-operation, had the chest tube removed on day three, and was eventually discharged well after five days. Written informed consent was obtained from the patient.

Discussion

Motor vehicle accidents, workplace accidents, and falls are the main causes of blunt injuries, and 70% of all are chest trauma (3). The mechanism of blunt trauma involves compression, acceleration, and deceleration injuries, as well as direct strikes on the thorax, resulting in a serious blunt impact injury. High-impact blunt thoracic trauma commonly results in rib fractures and may cause damage to underlying tissues, such as pulmonary contusions and pneumothorax. Tension pneumothorax is a deadly condition that mandates decompression manoeuvres as life-saving measures. It happens when the air becomes trapped in the pleural space under positive pressure, dislodging mediastinal structures and impairing cardiac function. The diagnosis is made based on clinical evidence, while extended-focused assessment sonography for trauma (E-FAST) is sensitive in ruling out pneumothorax (4).

In this case, an emergency finger thoracostomy relieved the tension in the pneumothorax. The gushing of air and the lack of abdominal content that can be felt when the finger is swept across the pleural space can differentiate other rare pathologies like fecopneumothorax or gastrothorax. An improper thoracostomy may result in an intestinal injury or stomach perforation. The CECT thorax shows that the chest tube is just one intercostal space away from the upper border of the herniated stomach. Fortunately, complications like an iatrogenic bowel injury are avoided in this case.

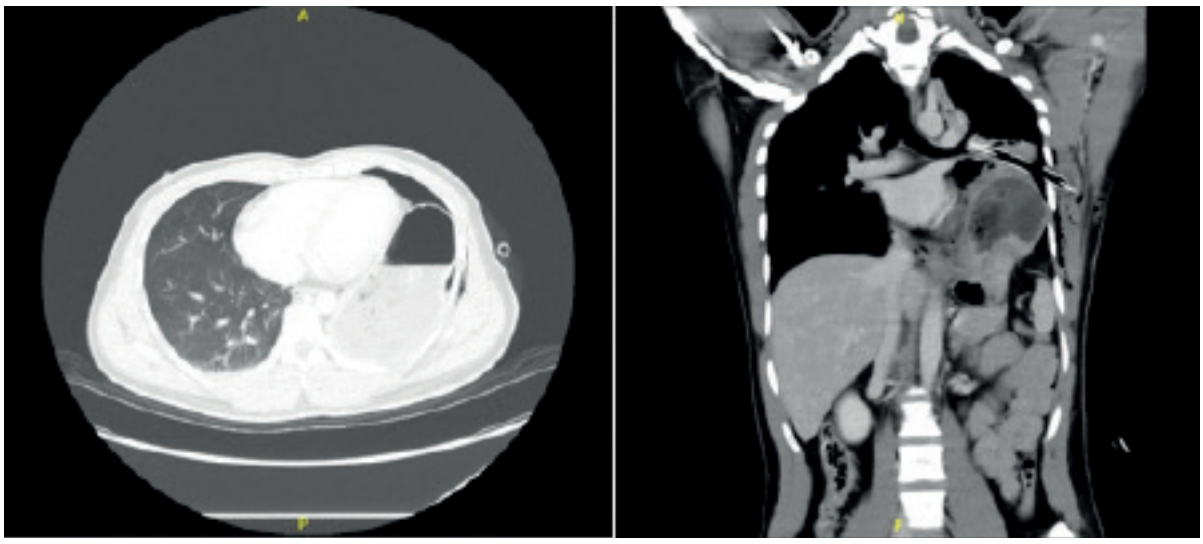

Figure 2. CECT thorax Axial and sagittal view shows focal thickening and discontinuity of the central left hemidiaphragm, intrathoracic herniation of the stomach and peritoneal fat, and focal constriction of the stomach at the herniation site. The herniated stomach is distended with the air-fluid level seen within. No diaphragmatic or peri-diaphragmatic contrast extravasation is identified. There was a left pneumothorax with evidence of a left chest tube traversing the left fifth intercostal space. No haemothorax or pleural effusion bilaterally. No significant mediastinal shift.

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Diaphragmatic hernia is an uncommon, potentially fatal ailment that can arise congenitally or from trauma. It occurs when abdominal contents protrude into the thoracic cavity through a diaphragmatic defect. Congenital diaphragmatic hernia (CDH) is most commonly detected in the neonatal period and is more common on the left side of the chest, most frequently a Bochdalek hernia (5). The pathology is infrequent in adults, who can be diagnosed incidentally or when symptomatic. The patient may present with respiratory or gastrointestinal symptoms or be acutely ill due to strangulation, volvulus, or perforation of hernia contents.

Traumatic diaphragmatic hernia (TDH) can occur due to penetrating and blunt injuries, yet it is hard to find due to the concealment of accompanying organ injuries. It rarely causes death on its own, but delay in diagnosis may lead to higher mortality with respiratory problems and strangulation of eviscerated organs. The concomitant injuries and complications determine the outcome (6). TDH's clinical symptoms include respiratory discomfort, diminished breath sounds on the affected side, chest auscultation of bowel sounds, palpable abdominal contents upon chest tube insertion, and a less full abdomen on palpation. The diagnosis of a diaphragmatic hernia could go unnoticed, but luckily it was detected in the initial chest x-ray without complication. An emergency operation to reduce the herniated organs and repair the diaphragm was performed. However, the CDH is identified intraoperatively.

Some clinical evaluations and bedside ultrasonography indicators support the presence of a diaphragmatic hernia. First off, having both respiratory and gastrointestinal symptoms at the same time raises the suspicion of a coexisting diaphragmatic hernia. In this case, even after the installation of a chest tube, the patient complained of ongoing chest and stomach pain and an intermittent urge to defecate. Bedside ultrasonography of the left hemithorax suggests a diaphragmatic hernia if there is no A profile in the left lower zone and only part of a hyperechoic line is seen, which is an abnormal diaphragmatic profile. There are a variety of ultrasound characteristics of a diaphragmatic hernia, including (i) a partial absence of the hyperechoic line representing the normal diaphragmatic profile; (ii) a partial absence of the pleural line in the affected hemithorax; (iii) the absence of A-lines in the affected area; (iv) the presence of a multi-layered area with hyperechoic contents in motion (normal gut); and (v) the possible presence of parenchymatous organs inside the thorax (i.e., liver or spleen) (7).

The chest x-ray revealed a round, hyperdense ring lesion in the lower left zone, which represented the abdominal content of the thorax and had focal constriction (collar sign). Other signs of a diaphragmatic hernia include a raised hemidiaphragm (more than 4 cm higher on the left), a distorted diaphragmatic border, and a visible nasogastric tube (8). 73% of TDH detected on the initial chest radiography has an additional 25% found on subsequent films (8). A CT scan is also the preferred modality whenever there is suspicion of diaphragmatic damage. Various significant signs are described in the literature with variable significance, which can be divided into three categories: direct signs of rupture, indirect signs that are consequences of rupture, and signs of uncertain origin (9). Blunt diaphragmatic rupture should be considered if any signs are present. The reference standard is multidetector computed tomography (MDCT), with a sensitivity and specificity of 61–87% and 72–100%, respectively (10).

This case serves as a reminder that tension pneumothorax and diaphragmatic hernia can coexist in blunt trauma patients. Evaluating the patient's history, performing a thorough clinical evaluation, and using imaging can help determine the specific types of thoracic injuries and their related issues. This information can also serve as a guide for life-saving and definitive surgical intervention. Diaphragmatic hernia's delayed consequences can be avoided by early discovery and surgical treatment, which also has a favourable prognosis and low recurrence.

Conclusion

CDH might be asymptomatic in childhood and manifest later in life due to a traumatic chest injury. It's possible to confuse this uncommon disorder with tension pneumothorax or have both life-threatening conditions together. Vigilant clinical examination and imaging aid in early detection and life-saving intervention.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Cause of Abdominal Pain: Herlyn-Werner-Wunderlich Syndrome

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Abstract

Herlyn-Werner-Wunderlich (HWW) syndrome is a congenital urogenital syndrome. Typical features are unilateral blind-ending hemivagina with uterine didelphy and ipsilateral renal agenesis. Patients usually progress asymptomatically until menarche, and they are diagnosed with radiological imaging by applying to the hospital with these conditions such as abdominal pain, abdominal bloating, primary amenorrhea along with menarche. In this case report, it is aimed to present a patient who applied to our emergency department with abdominal pain and is diagnosed with HWW syndrome. In this case, it is emphasized that emergency physicians should regard to HWW syndrome in the differential diagnosis of abdominal pain.

Keywords: Herlyn-werner-wunderlich, müllerian anomaly, abdominal pain

Introduction

Abdominal pain constitutes an important part of emergency admissions. It has been observed that 40% of these not complying with the known symptoms and concluded any clear and understandable outcome and come up with a spontaneous sight (1). It is very difficult to determine the prevalence and incidence of uterine anomalies due to the use of different classifications, diagnosis with different methods and the absence of symptoms of many anomalies. On the other hand, in all studies conducted between 1950 and 2007, it was determined as 6.7% in the general population and 7.3% in the infertile population and women with recurrent pregnancy loss, this rate varies between 3% and 25% (2). No matter how the exact factors that caused these anomalies are not clear, it has been determined in studies that ionizing radiation, infective processes and some medicine (Diethylstilbestrol (DES), Thalidomide, etc.) exposed during the genital development process causing Müllerian anomalies (3).

Herlyn-Werner-Wunderlich (HWW) syndrome is among the uterine anomalies and important for early diagnosis of complications and permanency of fertility. Most of the patients consult with these complaints such as abdominal pain, abdominal mass, bloating, inability to menstruate and acute abdominal pain(4). Dysmenorrhea is the main symptom of HWW syndrome and it is usually established after puberty. The increase in pain is related to a rise in the volume of hematocolpus caused by an obstructed hemivagina. Endometriosis, inflammation, twisted cysts, and appendicitis are other differential causes of pelvic pain and these must be excluded. Although it is possible to interfere diagnoses with similar symptoms and there is not a typical examination finding, differential diagnosis can be easily made with scanning methods. Observing unilateral blind-ending hemivagina and uterus didelphy and ipsilateral renal agenesis on computed tomography (CT) or ultrasonography (USG) imaging performed on the patient is important for diagnosis(5). Making diagnosis in time makes it possible to prevent possible complications with further examination, treatment and surgical procedures depending on the condition of the anomaly (6).

In this case, it is aimed to present the HWW Syndrome that emergency physicians detect in a 19-year-old female patient who applied to the emergency department with the abdominal pain.

Case Report

A 19-year-old female patient has consulted to the emergency department with the abdominal pain. On admission to the

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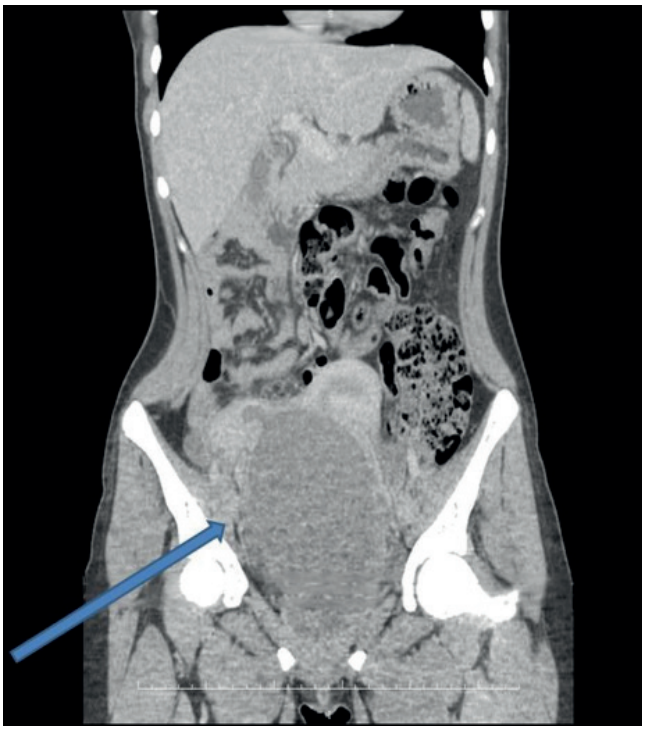

Figure 1.

emergency service, it has been observed that the general health of the patient is good and conscious. First vital signs; blood pressure: 115/75 mmHg, saturation: 96%, heart rate: 88 beats/min, fever: 36.6oC.In physical examination; there is tenderness and defense in the right inguinal and hypogastric area of the abdomen. Advanced vaginal examination could not made to the patient in the emergency room, but it is learned that she has the virginity in this statement. No significant finding is detected in the patient's other systemic examination. In her background, it is learned that the patient has occasional abdominal pain, but not having consistent dysuria or dysmenorrhea. The patient has not applied to any health institution with this complaint beforehand.

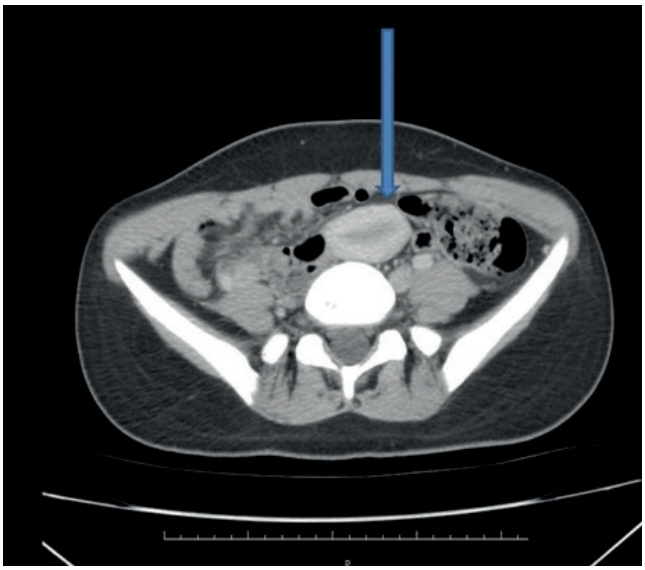

Figure 2.

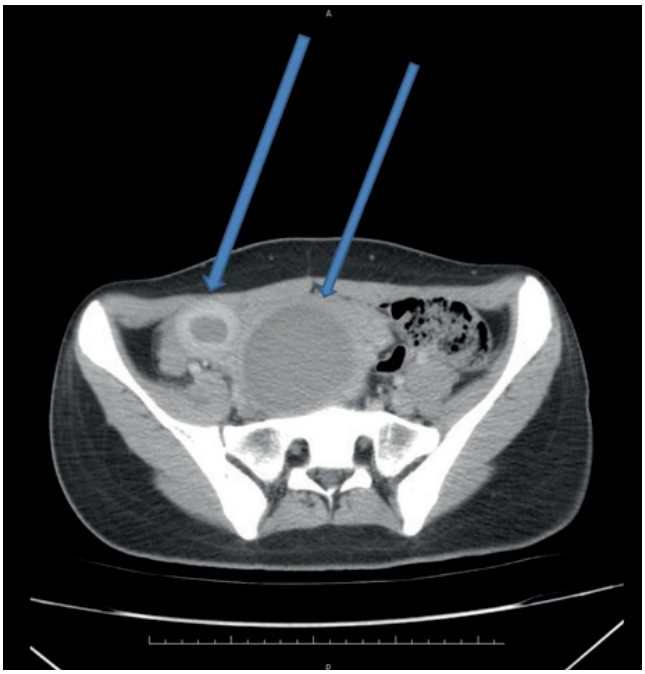

Figure 3.

By closing the oral intake of the patient in the emergency room, IV hydration is started, and necessary laboratory tests are requested. In the test results; b-Hcg<2 mIU/mL. White blood cell (WBC): 11360/mL C-reactive protein (CRP): <0.5 mg/dL and urinalysis: No leukocytes are observed. Little bit of erythrocyte is observed. The patient who has constant complaints, abdominal USG is taken for advanced diagnostic purposes. On USG, edematous didelphis uterus and concominant hematocolpos are observed. As the level of the cecum and ileocecal valve pushed to the left lower quadrant of the abdomen and renal pathologies could not be evaluated clearly, abdominal CT is performed for definitive diagnosis. On abdominal CT, a compatible sight is observed with hematocolpos, which fills the pelvis associated with right renal agenesis, uterine didelpys and right uterine cavity.

The patient who consulted with abdominal pain is diagnosed with HWW syndrome in the emergency room. Afterwards, she is discharged from the service with recommendation of outpatient control. The patient is transferred to obstetrics and gynecology service for further examination-treatment and follow- up process.

Horizontal and vertical one-centimeter incision is made in the hymen of the patient, who evaluated by the department of obstetrics and gynecology. Approximately 200 cc of old bleeding material is aspirated. Afterwards, the patient who followed up for the service is discharged next day and the outpatient clinic follow-up has started.

Discussion

Herlyn-Werner-Wunderlich (HWW) syndrome is a congenital malformation among Müllerian anomalies. In this

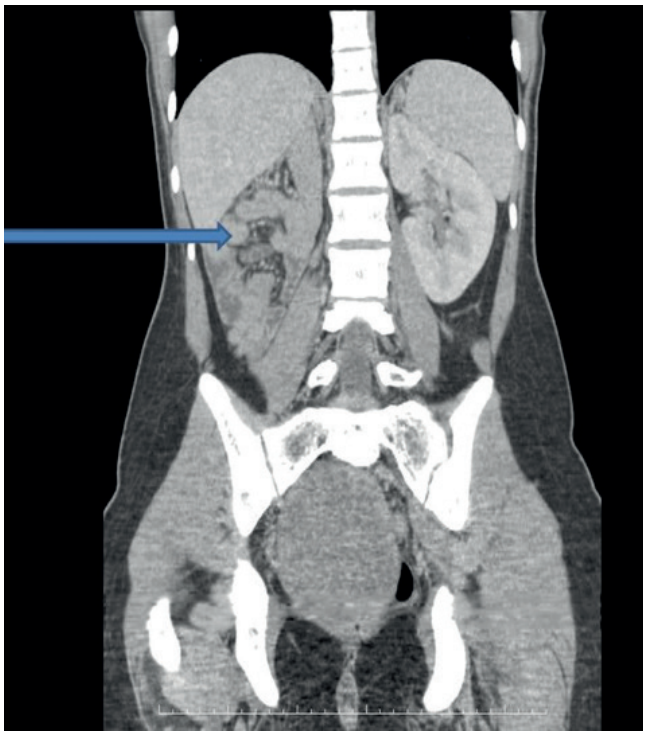

Figure 4.

syndrome, which generally progresses asymptomatically until puberty, it can be diagnosed rarely before puberty, and in some patients in the neonatal period with the appearance of a distended and protruding hemivaginal mass due to the effect of maternal estrogen (7). Symptoms of hematocolpos distension starting with puberty and menarche, bloating and abdominal pain varying from patient to patient depending on the degree of distension can be seen. There are many clinical manifestations that may cause infertility, such as fever, pyohematocolpos, peritonitis, which may occur with accompanying infective processes, and, albeit rare, urinary system stenosis and endometriosis (8-9).

In the developmental pathophysiology of the anomaly, the Müllerian canal shows migration to the midline at the 8th week of pregnancy and then there is a defect in its union with the uterus, cervix and superior part of the vagina (10). The incompleteness of this union causes the formation of two uteruses. The kidneys, fallopian tubes, cervix, and proximal vagina all develop from the same ureteral bud. Therefore, renal agenesis occurs following Müllerian duct dysgenesis (10).

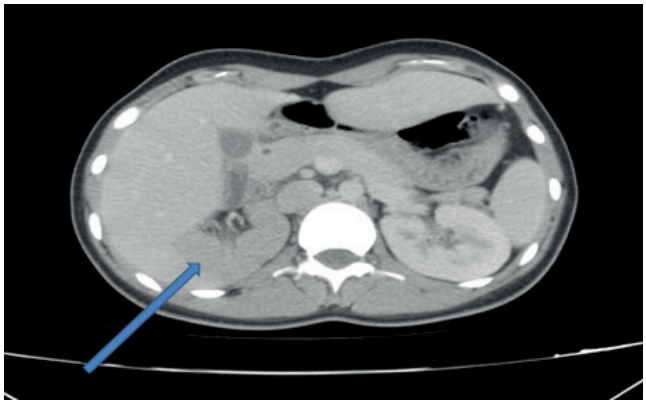

Figure 5.

Scanning methods play an important role during the diagnosis process. It can be diagnosed by visualizing genitourinary anomalies, uterine didelphis, bicornuate uterus, vaginal septum, renal agenesis, hematocolpos, free fluid in the pelvis in USG, CT, hysterosalphingography methods. However, magnetic resonance imaging (MRI) is accepted as the high standard in the diagnosis of Müllerian anomalies (6). Laparoscopy can be performed as an auxiliary intervention in the diagnosis and treatment when other imaging methods are insufficient (11). In treatment, vaginal septum resection can be applied to prevent obstruction, and unilateral hysterectomy can be applied in recurrent stenosis (12-13).

Conclusion

The patient, who presented to the emergency department with abdominal pain, is diagnosed with HWW syndrome as a result of the examinations. HWW is a rare disease caused by Mullerian canal anomaly. Emergency physicians should also keep in mind the causes of vaginal stenosis and HWW syndrome in female patients who apply with abdominal pain apart from the menstruation time.

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Case Report

Journal of Emergency Medicine Case Reports

Lung Parenchymal Damage Due to High Voltage Electric Shock

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Abstract

In electric shocks, lung tissue is a poor conductor and has low resistance. Therefore, they are not very sensitive to the development of tissue damage due to electric current. In this study, we wanted to present a rare case of lung parenchymal burn as a result of electric shock. A 21-year-old male patient was brought to the emergency department by ambulance after being electrocuted with a current of 15,000 volts after contact with a high voltage line while working with a construction machine. The general condition of the patient was good and vital signs were stable. However, his pulse oxygen value was 74 and his breathing was slightly tachypneic. There were no lesions in the chest and abdomen due to electrical burns. Radiologic imaging showed bilateral diffuse tissue damage in the lung tissue. The patient was referred to a tertiary care institution with an intensive care burn unit. In traumas caused by high-voltage electric shock, burns on the body surface may be insufficient to determine the severity of the trauma. A multidisciplinary approach must be provided to these patients

Keywords: Emergency medicine, high voltage, electrical injury, lung damage

Introduction

The most common type of injury in the body as a result of electric shock is burns (1). In high-voltage electrical injuries, myoglobinuria, renal failure and compartment syndromes may be observed due to muscle destruction in the body. The mortality rate of high-voltage injuries is quite high and surgical treatment is required in some cases in addition to medical treatment (2). In electric shocks, electric current causes damage to internal organs by creating a closed circuit current effect especially on wet surfaces. Although there are no entry and exit burn scars in the chest area, pneumonia, pleural effusion, hemothorax and liver capsule burn may occur (3). In electric shocks, lung tissue is a poor conductor and has low resistance. Therefore, they are not very sensitive in the development of tissue damage due to electric current (4).

In this study, we aimed to present the case of burns in the lung parenchyma tissue of a heavy equipment operator who was injured in an electric shock caused by the contact of the vehicle with a high voltage line with a current of 15,000 volts during the use of heavy equipment.

Case Report

A 21-year-old male patient was brought to the emergency department by ambulance after being electrocuted with a current of 15,000 volts after contact with a high-voltage line while working with a construction machine. The patient was conscious with a Glasgow Coma Scale (GCS) of 15. Vital signs; blood pressure: 118/72 mmHg, pulse rate: 98/minute, temperature: 36.7oC, pulse oxygen saturation: 74. The patient's respiration was mildly tachypneic and he stated that he had difficulty breathing. Physical examination revealed burns around the left shoulder and ear and electrical output burns on the right thigh, right foot and left foot (Figure 1). Laboratory and imaging tests were performed. According to the results, white blood cell count was 22.85 10^3 u/L, hemoglobin: 16.1 g/dL, platelet: 475 10^3 u/L, urea: 34 mg/dL, creatinine: 0.99 mg/dL, sodium: 137 mmol/L, potassium: 4 mmol/L, AST: 26 U/L, ALT: 26 U/L, CRP: 8.1 mg/dL, Troponin T: 5.1 ng/dL, CK: 3.3 µ/L, CK-MB: 2.7 µ/L, pH: 7.344, PCO2: 40 mm/Hg, PO2: 68 mml/Hg, HCO3: 21.3 mmol/L, lactate: 4.09 mmol/L. Electrocardiography (ECG) evaluation revealed normal sinus rhythm. Radiologic imaging revealed diffuse ground glass images in bilateral lung tissue on thoracic tomography (Figure 2). The patient had no recent known signs of lung infection (cough, sputum, fever, etc.). It was concluded that the images in the lung parenchymal tissue occurred after electric shock. The patient received tetanus prophylaxis, antibiotics, analgesics and fluid replacement therapy.

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Vital signs were stable and urine output was normal. After consultation with the relevant departments, the patient was referred to a center with a burn unit. The patient was referred to a tertiary health care institution with a burn unit and intensive care unit. After remote follow-up, the patient received treatment in the intensive care unit for eight days without intubation and was discharged with healing with the improvement of the lung tissue. It was learned that the patient did not develop any complications.

Discussion

As a result of electric shock; burns in the body at various degrees, musculoskeletal injuries, orthopedic injuries and cardiac arrhythmias may occur. The most important cause of death in electric shocks is cardiac arrhythmias (5). In electrical injuries, the voltage of the electric current and the duration of exposure to current may cause injuries in internal organs disproportionately to skin burns. Therefore, the voltage and duration of exposure to electric current must be questioned in persons exposed to electric current even in the absence of skin burns, and the patient must be evaluated extensively (6). In our case, the burn findings observed in the patient suggested that the patient had been exposed to high-voltage electric current for a long time. We attribute the fact that the patient responded to treatment in a short time without complications and did not end with death to the fact that the working environment was dry and the contact with the current was relatively short. Indeed, in a similar case reported by Yaşar et al. in 2006, an 18-year-old male patient was electrocuted during a car wash and was exposed to current for about 30 minutes on a wet floor. The burns observed in the lung and liver parenchyma tissue were severe and the patient died (6). In a case reported by Aydın et al. in 2019, a 52-year-old male patient was exposed to 36,000 volts of current with a transformer explosion and compartment syndrome and rhabdomyolysis occurred in the patient's arm. In this case, cardiac pathology, lung and liver tissue damage did not develop and electric current caused major tissue damage on musculoskeletal tissue (7). Araç et al. retrospectively analyzed electrical injuries admitted to the emergency department and observed pneumomediastinum in one patient (0.6%), pneumothorax in one patient (0.6%) and lung contusion in one patient (0.6%) in 178 patients (8).

Patients exposed to high-voltage electric current are generally younger and the morbidity and mortality rates are high in these cases. Follow-up of these patients would be more appropriate in centers with more comprehensive burn surgery and burn intensive care compared to low-voltage injury patients (8).Our patient was referred to a more comprehensive health center to reduce mortality because of his young age and exposure to high-voltage electric current.

Conclusion

In traumas caused by high-voltage electric shock, burns on the body surface may be insufficient to determine the severity of the trauma. A multidisciplinary approach should be provided to these patients and it should be kept in mind that diffuse parenchymal damage may develop in the lung tissue.

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Case Report

Journal of Emergency Medicine Case Reports

Acute Systemic Toxicity Associated with Ingestion of Juniper Tar

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Abstract

Juniper Tar has been utilized in traditional folk medicine to treat various ailments. Despite its historical use, documented cases are scarce regarding the potential complications associated with Juniper Tar, some of which have been fatal. A 62-year-old male patient with no previous medical history presented to the emergency department complaining of confusion and respiratory distress after drinking a glass of Juniper Tar approximately four hours ago. The patient, who experienced multiple seizures and cardiac arrest at the emergency department, was discharged after an 11-day intensive care unit monitoring period. We present this case to raise awareness among emergency physicians about Juniper Tar, which is widely used for various purposes in folk medicine and cosmetics but can cause poisoning due to its content of essential oils, triterpenes, and phenols. This is a case of cardiac arrest and status epilepticus associated with juniper tar poisoning.

Keywords: Juniper tar; toxicity; emergency department

Introduction

Juniper Tar, a dark-colored and highly aromatic liquid and also called "cade oil," is derived from the dry distillation of the wood and branches of the *Juniperus oxycedrus* (1, 2). It has been utilized in traditional folk medicine to treat a range of ailments, including hyperglycemia, obesity, tuberculosis, bronchitis, pneumonia, kidney stones, and the management of various dermatological conditions (3). Despite its historical use, there is a scarcity of documented cases regarding the potential complications associated with Juniper Tar, some of which have been fatal (4-8). Considering the widespread availability of Juniper Tar in Mediterranean countries, it is essential to emphasize the need for increased awareness among emergency physicians regarding its toxic effects. We report a case of acute systemic toxicity and cardiac arrest associated with Juniper Tar ingestion.

Case Report

A 62-year-old man with no significant medical history presented to the emergency department (ED) approximately four hours after ingesting a cup of Juniper Tar procured from a local herbal vendor. The patient exhibited confusion and respiratory distress.

On arrival, his blood pressure was 130/70 mmHg, heart rate was 101 beats per minute, temperature was 36°C, respirations were 28 breaths per minute and a pulse oximetry of 99% on room air. Initial assessment revealed an unconscious patient with equally reactive pupils. The remainder of the physical examination was unremarkable.

The parameters of the biochemistry, hemogram, arterial blood gas, infection screening tests, and culture results studied at the time of the patient's arrival are presented in Table 1. Other laboratory testing including urine drug screen (consisted of amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine, methadone, opiates, tramadol and phencyclidine) and ethanol levels were undetectable. His electrocardiogram revealed sinus tachycardia and troponin, and chest X-ray also were unremarkable. Symptomatic and supportive treatment was initiated.

Approximately 15 minutes after his arrival, the patient experienced a 5-minute generalized tonic-clonic seizure, and was administered 5 mg of intravenous diazepam. Head computed tomography without contrast was performed, which did not show any acute hemorrhage or mass. Following the seizure, the patient did not regain

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Table 1: Laboratory, imaging and culture results of the patient

Tests	Results
Fingerstick Glucose	166 mg/dL
Sodium	137 mEq/L
Potassium	3.5 mEq/L
Chloride	104 mEq/L
BUN	28.4 mg/dL
Creatinine	1.21 mg/dL
White blood cell count	10.6 x 10^3/µL
Hemoglobin	14.6 g/dL
Total creatinine kinase	Within normal range
pH	7.24
PaCO2	64 mmHg
HCO3	21.5 mEq/L
fMetHb	0.5
Lactate	3.6 mMol/L
C-reactive protein	Negative
Procalcitonin	Negative
Urine Culture	Negative
Blood Culture	Negative
Cerebrospinal fluid Culture	Negative
Urine Drug Screen	Undetectable
Ethanol Level	Undetectable
Troponin	Within normal limits
Urinalysis	Unremarkable
Chest X-ray	Unremarkable

consciousness and experienced another generalized tonic-clonic seizure, leading to a cardiac arrest. Cardiopulmonary resuscitation was performed, and the patient was intubated and sedated with midazolam infusion. After intubation, the arterial blood gas results showed a pH of 6.98, lactate level of 12.7, pCO2 of 85.6, and HCO3 of 14.1. The patient developed hypotension, tachycardia, and shock.

The patient was discussed with the National Poison Information Center, and systemic poisoning related to Juniper Tar-induced phenol toxicity was suspected. In addition to symptomatic treatments, the patient received N-acetylcysteine therapy, mechanical ventilation, hemodynamic support, correction of acid-base disturbances, and anticonvulsant treatment with levetiracetam.

The patient was admitted to the intensive care unit and empirical antibiotics were initiated. Severe gingival erosions were observed during follow-up examinations. Generalized seizures and respiratory distress persisted in the intensive care unit. Neurology consultation was obtained, and a brain MRI was performed, which did not reveal any acute processes. The patient was successfully extubated after seven days and ultimately discharged on the 11th day, with no sequelae observed during a 6-month clinical visit follow-up period.

Discussion

Juniper Tar, commonly used in alternative medicine, especially in Mediterranean countries, contains etheric oils, tripertene (raisin cadinene), and phenols (guaiacol and cresol derivatives) (9). Phenol, the most toxic component, causes systemic poisoning symptoms when it is converted into free radicals exceeding hepatic conjugation capacity (7, 8). Juniper Tar, which is mostly produced and sold without prescription by local herbalists, is used orally and percutaneously, and it leads to a wide range of reported clinical manifestations such as urticaria, nausea, vomiting, dizziness, and itching to more severe conditions including acute respiratory distress syndrome (4), acute pancreatitis (6), coma (5), and multiorgan failure (7). There have been reported cases in the literature, mostly involving infants aged 0-1, where serious poisonings caused by topical applications were observed.

The management of Juniper Tar toxicity is not well-defined. Treatment may include skin decontamination, supportive care, correction of hemodynamics, treatment of acid-base imbalances, and administration of anticonvulsants. Hemodialysis is essential in cases of anuric renal failure. N-acetylcysteine may be recommended to prevent the accumulation of free radicals due to hepatic biotransformation and to bind toxic metabolites. If methemoglobinemia develops, methylene blue can be administered (6).

The use of herbal products remains popular among the public, increasing the likelihood of emergency physicians encountering problems related to this use. Physicians, especially in regions where traditional medicine is widely practiced, should be aware of this reality. During history-taking, routine questioning should include herbal product usage alongside prescribed medications. The main challenge is the lack of standardized documentation regarding appropriate indications, dosages, side effects, and specific age groups for herbal use. Therefore, it is essential for physicians to contribute to the pool of data and report phytopharmacovigilance to obtain information about possible adverse effects.

Conclusion

Further research and investigation are needed to comprehensively explore the potential toxic effects and complications associated with Juniper Tar. By expanding the existing literature and disseminating this information among medical practitioners, we can facilitate a safer and more informed approach to the use of Juniper Tar in both traditional and contemporary healthcare settings.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Entity in Emergency Department: Trapdoor Fracture

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Abstract

Trapdoor fracture also known as, 'White-eyed blowout fracture" is generally seen among pediatric patients with orbital floor blowout fracture. It is presented as "open door fracture" in adults because of the mineralized, fragile orbital bones. An 11-year-old boy was admitted to the emergency department (ED) because of falling and hitting his on the occipital region. His main complaint was diplopia. He did not have nausea or pain. No visible lesion detected on his head. When the orbital CT scan was examined carefully, a fracture of the right orbital floor and herniation of orbital soft tissue was noticed. The patient was transferred to the plastic surgery department for surgical intervention. Even though there is no direct trauma, or visible pathology around orbital region, emergency physiscians should keep in mind trapdoor fracture, especially in pediatric patients with head trauma.

Keywords: Diplopia, emergency medicine, trapdoor fracture, trauma

Introduction

Trapdoor fracture also known as, 'White-eyed blowout fracture'' is generally seen among pediatric patients with orbital floor blowout fracture. It is presented as ''open door fracture'' in adults because of the mineralized, fragile orbital bones. The part of the fractured bone dose not replace, so no muscle or soft tissue entraps. Because children have elastic, cancellous bone, an increased intraorbital pressure displaces a part of bone that immediately replace to its main position. At the same time usually the inferior rectus muscle and the inferior oblique muscle entrap and it causes diplopia and limited ocular motility without periorbital oedema, ecchymosis or hemorrhage.(1,2) Because of lack of external symptoms, emergency physicians may often misdiagnose this pathology .

Case Report

An 11-year-old boy was admitted to the emergency department (ED) because of falling and hitting his on the occipital region. His main complaint was diplopia. He did not have nausea or pain. No visible lesion detected on his head. On the neurological examination; it was noticed that, the patient had limited upward movement in his right eye (Figure 1). Because of diplopia, a non-contrast cranial computerized tomography (CT) was requested. There wasn't detected any fracture on the occipital bone and any intracranial hemorrhage or herniation. The patient was consulted to the neurological surgeon.But the consultant physician did not determine any neurosurgical pathology. There was not any oedema, ecchymosis or hemorrhage around or on his orbital region. When the orbital CT scan was examined carefully, a fracture of the right orbital floor and herniation of orbital soft tissue was noticed (Figure 2,3). When we detailed the anamnesis, the patient said that; one of his friends fell onto his right eye with his ankle during playing football. The patient was diagnosed with 'trapdoor fracture'' and immediately consulted to ophtalmology and plastic surgery departments. The patient was transferred to the plastic surgery department for surgical intervention.

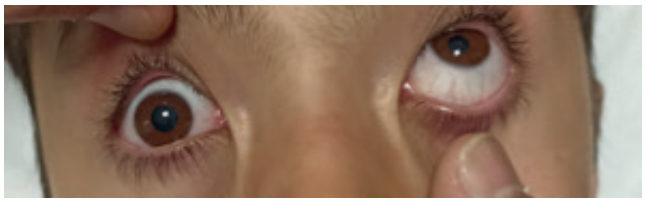

Figure 1. Restricted upward movement of right eye

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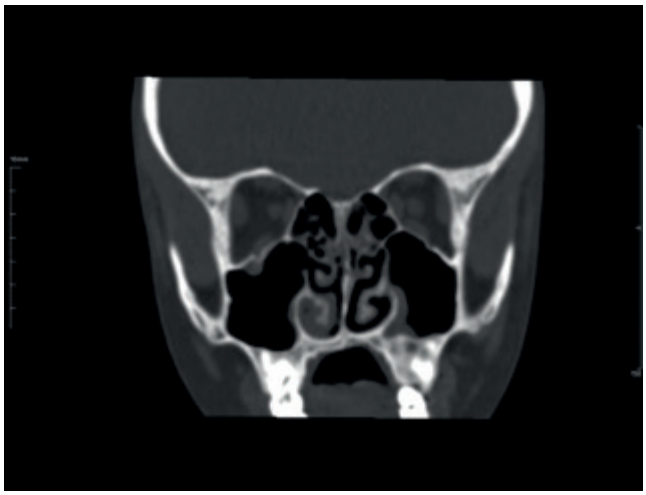

Figure 2. Coronal view of computed tomograpy. Right orbital floor fracture with entrapment (arrow)

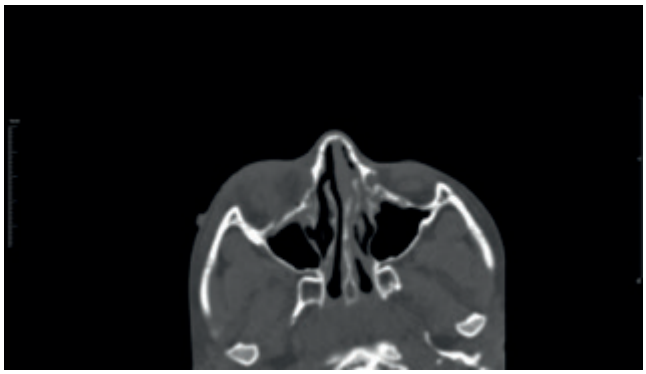

Figure 3. Axial view of computed tomograpy. Right orbital floor fracture (arrow)

Discussion

The term ‘’white-eyed blowout fracture’’ has been firstly used by Jordan and collegues to identify this typical presentation of blowout fracture: diplopia and limited ocular motility without evidence of trauma (3). Due to the oculocardiac reflex that is triggered by orbital floor fracture, syncope, nausea, vomiting and bradycardia can be seen. Limited ocular motility and diplopia are the frequent clinical findings in lots of the studies (4). Motility is usually limited in both the direction toward and opposite the fracture. The limitation opposite entrapped muscle is in most cases significantly more limited (5,6). In another study the most common clinical findings were ocular motility restriction (81%) followed by diplopia (62%), enophthalmos (48%), eyelid ecchymosis (42%), and ptosis (25%). Of the 42 children with ocular motility restriction, 32 (76%) had diplopia (7). And early surgical intervention within 48 hours of diagnosis is suggested in many studies for a successful recovery (8). In a study conducted by Gerbino and et all. In all 24 patients, surgery was undertaken as soon as possible after patient presentation (ie, within 12 hours of presentation); thus, the time between the occurrence of the trauma and surgery was related to the timing of the presentation (9). In the cases with trapdoor fracture and restricted ocular movement, early intervention was associated with better postoperative function. It is thus recommended that the symptomatic trapdoor orbit fracture be considered an urgent indication for surgical intervention. Practitioners therefore must have a high index of suspicion for these injuries (10). Therefore, to pay attention to the trapdoor fracture and do a comprehensive ocular examination is suggested in all pediatric cases of head trauma in emergency room. Early diagnosis and surgery is taught to lead a good prognosis and less complications.

Conclusion

Even though there is no direct trauma, or visible pathology around orbital region, emergency physiscians should keep in mind trapdoor fracture, especially in pediatric patients with head trauma.

Declaration of patient consent

We certify that we have obtained all appropriate patient consent forms. Patient's father has given the consent for the child's images and other clinical information to be reported in the journal.

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