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Contents

Case Report

Journal of Emergency Medicine Case Reports

A Case of Perforated Retroperitoneal Appendicitis Mimicking Pyelonephritis

Levent Cankorkmaz[1], İsmail Şalk[2], Mehmet H. Atalar[2], Gökhan Köylüoğlu[3]
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Abstract

Introduction: Appendicitis is one of the most commonly diagnosed surgical diseases in childhood with emergency abdominal pain. Retrocecal retroperitoneal appendicitis is uncommon and potentially difficult to diagnose. This condition might present with atypical clinical, and radiological signs. Here, we present a patient with perforated retrocecal appendicitis in whom the clinical findings mimicked acute pyelonephritis.

Case Report: A 3-year-old boy presented with right flank pain, fever, and vomiting for two days. The abdomen was soft but the right costovertebral angle tenderness was positive. There was pyuria. The clinical impression was pyelonephritis. Ultrasonography showed perirenal fluid collection but did not show any collections pericecal area. In the Multi-detector CT (MDCT) MDCT a long inflamed retrocecal retroperitoneal appendix was seen with surrounding inflammation that extended to anterior pararenal space, and retroperitoneal air. At surgery, there was retrocecal retroperitoneal appendicitis with perforation and retroperitoneal collection. Appendectomy and drainage were performed.

Conclusions: Patients with retroperitoneal appendicitis is that these patients often present with atypical and less severe abdominal complaints. In this situation, MDCT could be a rapid and efficient tool for localizing the appendix and for the differential diagnosis.

Keywords: Retrocecal retroperitoneal appendicitis, perinephric collection, child

Introduction

Appendicitis is the most common surgical emergency. Accurate diagnosis is sometimes hindered due to various presentations that differ from typical signs and anatomic anomalies of appendicitis (1). A delay in diagnosis and treatment increases the likelihood of complications such as perforation which is associated with an increase in morbidity and mortality. The appendix is typically in the intraperitoneum,either anterior or retrocecal; however, in 30–65% of appendicitis cases, it may be retroperitoneal location (2).

A perinephric abscess is a collection of purulent material in the space between the kidney and Gerota's fascia. It is rare in adults and even less common in children.Sparsely, the spread of infection from inflammatory lesions of adjacent viscera, diverticulitis, and retrocecal appendicitis have been implicated in the pathogenesis of perinephric abscess (3).

We present a boy with perforated retrocecal and retroperitoneal appendicitis in whom the perirenal collection and atypical symptoms and signs of appendicitis were diagnosed by Multidetector CT (MDCT).

Case

A 3-year-old boy presented with right flank pain, fever, and vomiting since two days. His fever was 40° Celsius. The abdomen was soft but the right costovertebral angle tenderness was positive with right hypochondrial tenderness. A complete blood count revealed leukocytosis (19.8 × 109/L) with a left shift. There was pyuria on urinalysis. The clinical presentation was acute pyelonephritis. The patient's abdominal Ultrasonography (USG) findings were normal renal parenchymal echogenicity, and perirenal fluid collection but did not show any collections pericecal area. MDCT was performed for differential diagnosis. In the MDCT a long inflamed retrocecal appendix was seen with surrounding inflammation that extended to anterior perirenal space, and retroperitoneal air (Figure-1). There was also pleural effusion of the right lung base.

At surgery, there was retroperitoneal appendicitis with evidence of perforation into the retroperitoneum and retroperitoneal collection. Appendectomy and drainage of the perinephric abscess were performed. The patient received postoperative ceftriaxone (IV) and metronidazole

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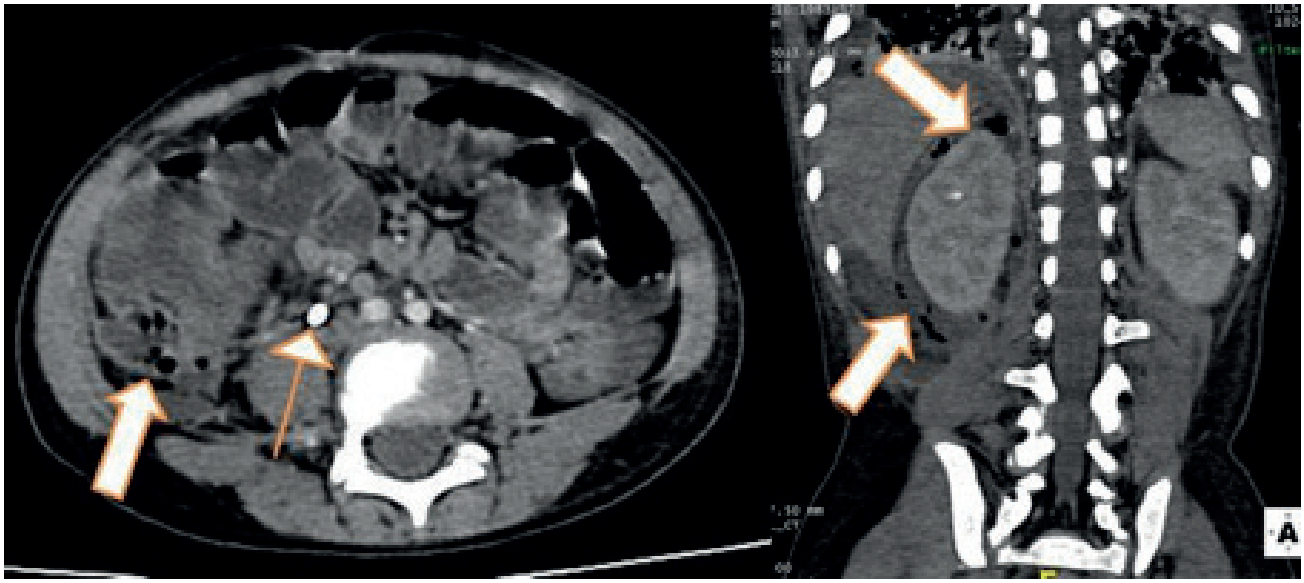

Figure 1. Axial and coronal contrast-enhanced CT images showed retroperitoneal air and inflammation (thick white arrows). In addition, the axial CT image showed appendicolith (thin white arrow).

(IV) for 12 days. The patient made a good recovery. He was discharged on the post-op 14th day in stable condition.

Discussion

Complications and relatively high mortality rate associated with retroperitoneal perforation of appendicitis, it is important to be able to recognize the early signs. This can delay diagnosis, which may contribute to higher rates of complications and mortality. If the appendix is in a retrocecal position, it may be positioned intraperitoneal or retroperitoneal area (4).In the case presented here, the only sign apparent on the MDCT exam was retroperitoneal air and fluid. Patients may present only with minimal perinephric fluid as well. If there is no urogenital pathology on USG, as in our patient, other sources of infection should be searched for, including perforated retrocecal appendicitis.

A perinephric abscess is rare in adults and even less common in children (3,5). Rarely, the spread of infection from inflammatory lesions of adjacent viscera, diverticulitis, and retrocecal appendicitis has been implicated in the pathogenesis of perinephric abscess (3).The most common cause of perinephric abscess is the direct extension or hematogenous seeding from other sites of infection (3).

Patients with acute retrocecal and retroperitoneal appendicitis are that these patients often present with atypical and less severe abdominal complaints cecal in whom the clinical and USG outcomes mimicked acute pyelonephritis. USG is less sensitive in cases with perforated appendicitis, whereas contrast-enhanced computed tomography particularly MDCT with thick coronal multiplanar reformat (MPR) images has better sensitivity and specificity in this clinical setting (6). MDCT can be used effectively for the diagnosis of retrocecal or retroperitoneal appendicitis without additional preparation or focused examination. We postulate that, in this case, the MDCT exam was performed early in the course of perforation, and therefore only retroperitoneal fluid was seen, without intra abdominal fluid.

If there is no urogenital pathology with the USG, as in our patient, other sources of infection should be searched for, including perforated retrocecal and retroperitoneal appendix. In such cases, MDCT can be a more effective method than ultrasound (USG) in the differential diagnosis of retrocecal appendicitis.

Conclusions

In conclusion, retrocecal and retroperitoneal perforated appendicitis may present with various atypical clinical signs such as only with minimal perinephric fluid. In such cases, MDCT can be a more effective method than the USG in the differential diagnosis of retrocecal appendicitis.In such cases, when the appendix cannot be seen clearly or seems in an unusual localization, MDCT can be used effectively as a useful method for the correct diagnosis of retrocecal appendicitis without additional preparation or focused examination.In this situation, MDCT could be a rapid and efficient tool for localizing the appendix and for the differential diagnosis. Perirenal collection and atypical symptoms and signs of perforated appendicitis were diagnosed by MDCT.

We propose that, with increasing use of MDCT exams early in the course of clinical presentation, more cases of acute appendicitis with perforation into the retroperitoneum presenting with retroperitoneal fluid or air may occur, leading to earlier interventions and more successful clinical outcomes.

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Case Report

Journal of Emergency Medicine Case Reports

A Misleading Case: Lichtenberg Figures Caused by Illegal Use of Electricity from High-Voltage Transformer

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Abstract

Although injuries with high voltage are relatively rare in emergency departments, their morbidity and mortality are high. Findings in the body after the event can guide the diagnosis and treatment. A 21-year-old female patient was brought by her relatives with the complaint of electric shock. Lichtenberg figures, which are known to occur because of high voltage, were observed in the body examination of the patient, who stated that the incident occurred after contact with an open cable at home. When the anamnesis was detailed again, it was learned that she entered the transformer for illegal use. Voltage information is key in electrical injury management. With criminal concerns, patients may describe events differently. The clues in the examination, like Lichtenberg figures, can change the management of the patient.

Keywords: Emergency department, electrical injuries, Lichtenberg figure

Introduction

Electrical injuries, which were initially caused by lightning strikes, have diversified with the beginning of the use of electricity in cities. While the incidence of electrical accidents decreases with age in pediatric groups, it rises among adults, particularly affecting electricians, and construction workers (1). Patients may not have any symptoms and may present from superficial burns to cardiac arrhythmias and even death. In the United States, there are 10,000 emergency room visits due to electrical injuries each year, and approximately 350 deaths are reported (2). Voltage levels affect patient management. In high-voltage injuries, internal organ damage is not directly proportional to the severity of skin burns. Therefore, the percentage of burns should not be relied upon in fluid therapy (3). Even if there are no findings, the observation period should be longer in these injuries, especially in terms of the development of cardiac findings. In this case, a rare electrical accident accompanied by a Lichtenberg figure with anamnesis-examination mismatch is presented.

Case

A 21-year-old female patient presented to the emergency department with the complaint of electric shock. She accidentally stepped on an open cable at home. The patient was conscious and there was no loss of consciousness period, but she was unsure of the contact time.There was paresthesia and pain in both legs. She did not describe any significant medical history.She had no smoking or alcohol use. The patient's vital signs were blood pressure 125/77 mmHg, heart rate 120/min, temperature 36.6° C, respiratory rate 20/min, oxygen saturation 100% in room air respectively. Her Glasgow coma score was 15. She was agitated because of the event and described numbness and pain in the distal of both lower extremities. The patient's left leg Visual Analogue Scale (VAS) pain score was 4/10, and the right leg VAS score was 7/10. Her muscle strength was normal in all extremities, with no additional neurological examination findings. There were no obvious signs of trauma and no electrical input and output hole. The patient, who did not describe any chest pain, had palpitations and

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her ECG was sinus tachycardia. No additional ischemic features were observed. A bolus of 1000 cc %0,9 NaCl isotonic fluid was started for the patient's tachycardia and the patient was monitored. The patient had leukocytosis in blood tests (Table-1). The urine sample was clear with noclinically significant findings.

In the control examination of the patient, performed at the approximately second hour, the most prominent in the posterior of the right lower extremity (Figure-1) and the pink-colored, painless, non-branching, nonfluffy figures were observed on the abdominal skin (Figure-2). We determined that this finding, which was not noticed during the first physical examination but became evident later, was the Lichtenberg figure. After the detection of the finding that can be seen in high voltage injuries, the patient's anamnesis was suspected and repeated. The patient sustained injuries from a transformer while attempting to use illegal electricity. She had walked in barefoot and probably stepped on the exposed wire. Afterwards she was thrown upwards with a loud noise and then she was unconscious for a short time. She explained her anamnesis differently because she was worried about being arrested.

The patient's neurological, cardiological and dermatological findings showed high voltage exposure. Management of the patient was changed. She was followed up in the emergency room for 24 hours with intravenous/oral hydration and analgesia. In blood tests performed before discharge, leukocytosis regressed, and no significant increase was found in cardiac and muscle-destruction markers (Table 1). The vital signs were normal, the patient did not have any active complaints and was discharged in a healthy manner.

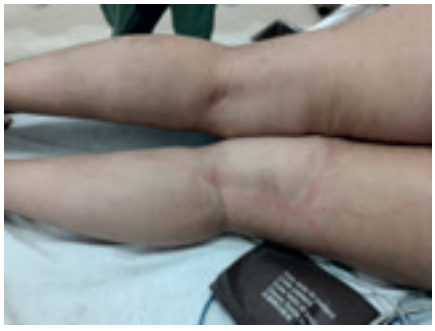

Figure 1. Lichtenberg figures (posterior of the right lower extremity)

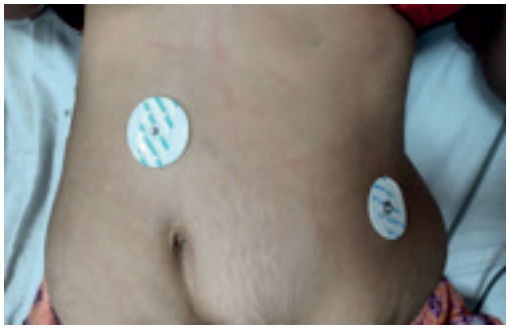

Figure 2. Lichtenberg figures (Left upper quadrant of the abdomen)

Table 1: Comparison of patient's admission and discharge laboratory analysis

Labs (range and unit)	Admission	Before Discharge
AST (<31 U/L)	22	19
ALT (<34 U/L)	22	18
ALP (35-104 U/L)	114	89
Total protein (64-83 g/L)	82.3	66.9
Albumin (35-52 g/L)	48.9	41.6
Total bilirubin (0.1-1 mg/dL)	0.33	0.22
LDH (135-225 U/L)	209	200
CK (34-145 U/L)	76	119
Myoglobin (25-58 µg/L)	74.43 H	35
Troponin T (<14 ng/L)	<13	<13
Urea (10-50 mg/dL)	20	17
Creatinine (0.6-1.1 mg/dL)	0.66	0.7
Sodium (136-145 mEq/L)	140	142
Potassium (3.5-5 mEq/L)	3.7	4
Chloride (96-110 mEq/L)	105	108
Calcium (8.6-10.2 mEq/L)	10.2	9.2
Magnesium (1.5-2.6 mEq/L)	1.96	1.89
CRP (0-5 mg/L)	4.7	4.8
Beta-hCG (<5.3 U/L)	<0.1	-
Complete Blood Count		
WBC (4.5-11x10^3/µL)	15.41 H	11.71 H
Hemoglobin (11.7-15.5 g/dL)	14.3	12.2
RBC (3.8-5.1x10^6/µL)	5.76	4.88
Hct (35-45 %)	44.7	38.8
Platelet (150-450x10^3/µL)	379	245

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase ALP: Alkaline phosphatase, LDH: Lactate dehydrogenase, CK: Creatinin kinase, CRP: C reactive protein,
Beta-hCG: Beta-human chorionic gonadotropin, WBC: white blood cells, RBC: Red blood cells, Hct: Hematocrit H: High

Discussion

Although the pathophysiology is not clearly known, they often appear with typical branching and pink-red shapes after lightning injuries (4-6). One hypothesis suggests that these patterns result from the activation of keratocytes, and T-cells triggered by high-voltage discharge. Extravasated blood cells also may contribute to its appearance (6). Typically, these figures begin to appear approximately 1 hour after the event and vanish within 48 hours without leaving a trace (4,7). Notably, the figures were not clearly visible before the patient's discharge. As far as we know, there are two more cases reported in the literature for Lichtenberg figures resulting from an industrial electrical accident. In the first reported case, a 49-year-old male patient who was caught in a 3 of 4 25000 V alternating current was presented and was followed up in the burn intensive care unit with the diagnosis of 15% total second-degree body burns and rhabdomyolysis (5). In the other case, a 47-year-old male

patient with a second-degree burn covering 7% of his body due to 20000 V alternating current was reported (6). Interestingly, although the electrical voltage in our case was not known (the electrical current in transformers in Turkey ranged from 10000 to 36000 volts (8), no complications were observed unlike the reported cases. Transient leukocytosis seen in the patient's blood results may be associated with acute inflammation findings because of the severity of the event (5). No additional evidence of infection was detected in the patient's physical examination and anamnesis. The patient did not remember which foot she had contacted. The patient's pain was mostly in his right leg, and these patterns were more prominent in the dorsal side of the same leg. Therefore, in our case, as mentioned in George et al., the electric arc may have terminated in the leg (9). The patient had a short-term loss of consciousness, and her cardiac rhythm was sinus tachycardia. Benign arrhythmias such as sinus tachycardia are frequently seen in these injuries at presentation and are temporary. Although the accompanying sinus tachycardia and high respiratory rate can be attributed to anxiety, the patient was continued to monitor. According to the literature, patients with symptoms such as altered consciousness, arrhythmia and chest pain should be followed up for at least 24 hours for deterioration (2,3). It should be noted that rhabdomyolysis may accompany another treatable fatal condition.Fluid therapy in appropriate amounts should be started without waiting for blood test results.

Conclusion

Lichtenberg figures emerge as a result of high-voltage electrical injuries, primarily associated with lightning incidents but also observable in industrial accidents. In such cases, patients may be unconscious. Even when conscious, obtaining relevant medical history may be challenging due to forensic concerns. Knowledge of these pathognomonic findings by emergency service healthcare providers can contribute to preventing patient mortality through the implementation of appropriate care.

Informed Consent:

The written informed consent of the patient was taken.

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Case Report

Journal of Emergency Medicine Case Reports

Transient Complete Right Bundle Branch Block Due to Lung Contusion: Case Report

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Abstract

Right bundle branch block (RBBB) may occur in lung diseases and cardiovascular diseases. A 25-year-old male patient was admitted to emergency department after a motorcycle accident. Contusion was detected in bilateral lungs, and pneumothorax with laceration were detected in the right lung. ECG revealed103 bpm, right axis deviation, and complete RBBB. ECGrevealed 83 bpm, complete RBBB continued at the 6th hour after tube thoracostomy, andhigh sensitive TnI was normal. Chest X-ray wasnormal on the 4th day of hospitalization, and ECG revealed74 bpm, normal sinus rhythm. In thoracic trauma, ECG changes may develop as a result of lung contusion. ECG findings improve as the lung contusion heals.

Keywords: Lung contusion, Right bundle branch block, RBBB, Thorax trauma

Introduction

Right bundle branch block (RBBB) is a common finding in older people and its prevalence increases with age. Complete right bundle branch block is associated with an increased risk of all-cause mortality in the population and in patients with heart disease. RBBB may occur in lung diseases in which right ventricular pressure increases, such as pulmonary hypertension/cor pulmonale, pulmonary thromboembolism (PE), and in cardiovascular diseases such as myocarditis, ischemic heart disease, congenital heart disease and hypertension. Transient or permanent RBBB may result from iatrogenic etiology during procedures such as right heart catheterization and septal ablation. Cases of temporary complete RBBB in spontaneous pneumothorax and traumatic pneumothorax have been reported. However, complete RBBB due to blunt trauma was mostly detected in cases of cardiac contusion (1-4).

In this case presentation, the relationship between lung injury and ECG changes in a patient who developed pneumothorax, lung contusion, and temporary complete RBBB as a result of thoracic trauma was discussed.

Case

A 25-year-old male patient was brought to the emergency department (ED) by ambulance after a motorcycle accident that occurred 1 hour ago. It was learned that the patient fell on his right side after being hit by a car while riding a motorcycle. He had pain in his chest and right shoulder. The patient was conscious, Glasgow Coma Scale was 15, blood pressure was 110/70 mmHg, pulse was 101 beats per minute (bpm) and rhythmic, respiratory rate was 32 breaths/min, and arterial oxygen saturation was 70%. The patient had no disease in his medical history. On physical examination, there was tenderness in the right chest and decreased breath sounds. There was a deformity in the right shoulder and proximal humerus, and there was a 3 cm incision on the right knee. Blood sugar was 180mg/dL. In arterial blood gas, pH: 7.48, pCO2: 39mmHg, pO2: 59mmHg, lactate: 2.1 mmol/L were measured. The patient's 12-lead standard electrocardiogram (ECG) and right-sided ECG were taken. ECG revealed rate: 103 bpm, PR interval: 160ms, QTc: 447ms, QRS interval: 124ms, QRS axis: 114°, right axis deviation, complete right bundle branch block (RBBB) (Figure-1). There were T-wave inversions from V3R to V6R in the right-sided ECG. (Figure-2). RBC: 5.28 106/µL, Hb: 15.8g/dL, hematocrit: 46.7%, high sensitive TnI: 21.4ng/L (reference range: 0-47ng/L) were measured. The patient was started on oxygen at 8L/min via mask. Computed tomography (CT) images of the patient were taken.

Thorax CT images were interpreted by the radiologist and the diameters of the heart chambers were measured.

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Thorax CT showed ground glass densities and septal thickening consistent with contusion in both lungs, more prominent on the right side. Pneumothorax in the right pleural space and laceration in the right lung were detected. Displaced fractures were observed lateral to the right 5th and 7th ribs. Left ventricle diameter: 41.1 mm, right ventricle diameter: 36 mm, inferior vena cava: 21.6 mm, ascending aorta diameter: 32.7 mm, main pulmonary artery diameter: 22.9 mm, heart and vascular structures were normal, there was no pericardial fluid (Figure-3). The patient had a tube thoracostomy placed on the right hemithorax in the emergency department, and there was a significant improvement in respiratory parameters. The patient was transferred to the intensive care unit. Oxygen therapy was continued at 8L/min via mask. Lung expansion was observed on chest X-ray (Figure-4). At the 6th hour of hospitalization, the ECG showed rate: 83 bpm, PR interval: 152ms, QTc: 441ms, QRS interval: 128ms, QRS axis: 66°, complete RBBB (Figure-5). RBC: 4.18 106/µL, Hb: 12.7 g/dL, hematocrit: 36.8%, high sensitive TnI: 195ng/L were measured. The chest tube was removed on the 3rd day of hospitalization. High sensitive TnI: 17ng/L was measured on the 4th day of hospitalization. Chest X-raywas normal on the 4th day of hospitalization (Figure-6). In the ECG, rate: 74 bpm, PR interval: 160ms, QTc: 390ms, QRS interval: 94ms, QRS axis: 62°, normal sinus rhythm was detected (Figure-7). The right-sided ECG was normal (Figure-8). The patient was discharged with full recovery.

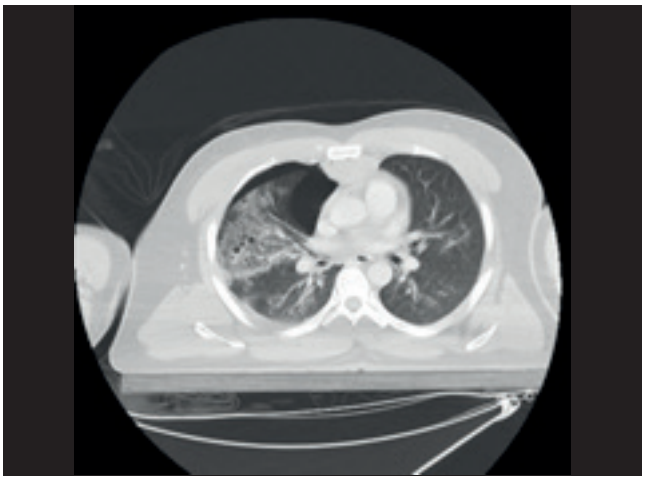

Figure 3. Thorax CT showed ground glass densities and septal thickening consistent with contusion in both lungs, more prominent on the right side. Pneumothorax in the right pleural space and laceration in the right lung were detected.

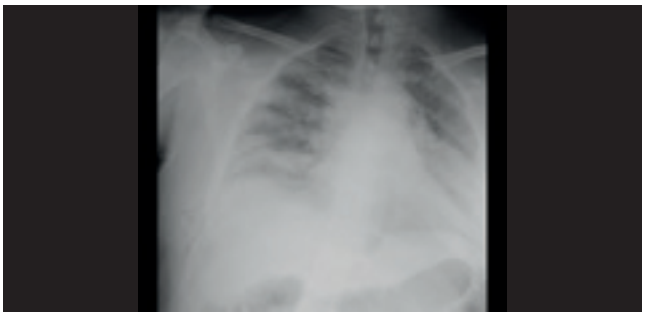

Figure 4. After tube thorocostomy, the lung expanded and lung contusion continues.

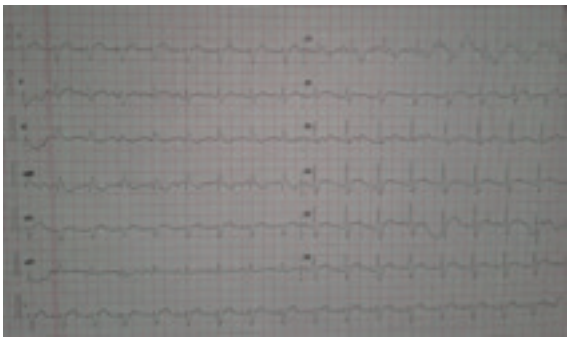

Figure 1. The patient's first ECG revealed tachycardia, right axis deviation, and complete right bundle branch block

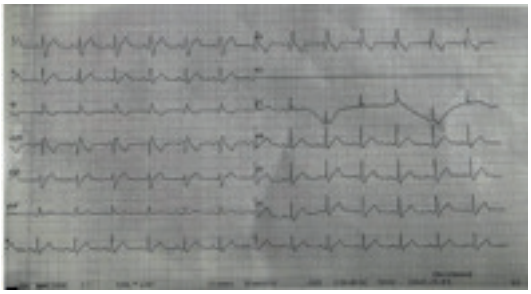

Figure 5. The ECG after lung expansion showed complete right bundle branch block.

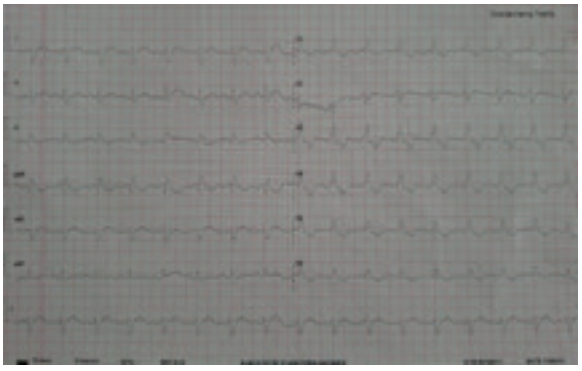

Figure 2. T-wave inversions from V3R to V6R in the right-sided ECG

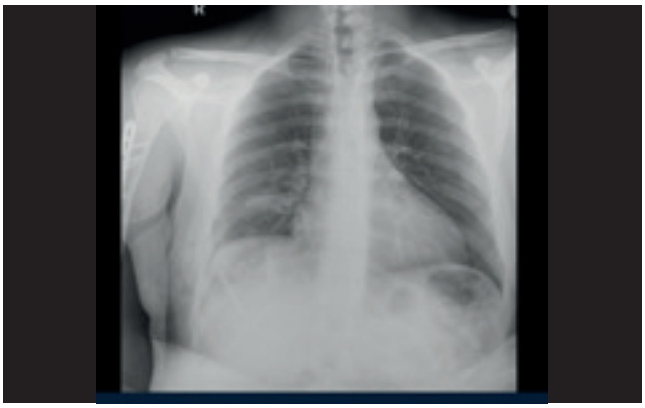

Figure 6. The patient's chest X-ray was normal on the 4th day of hospitalization

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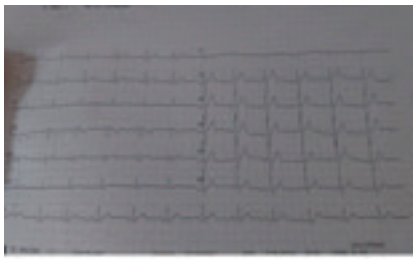

Figure 7. The patient's ECG was normal on the 4th day of hospitalization.

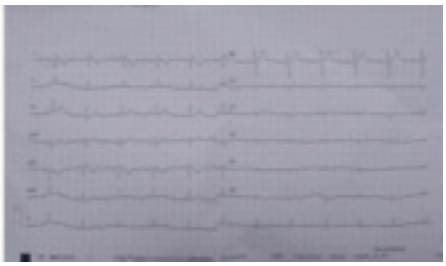

Figure 8. The patient's right-sided ECG was normal on the 4th day of hospitalization.

Discussion

Thoracic trauma accounts for 10-50% of trauma-related deaths and includes a wide variety of injuries that can cause significant morbidity and mortality. In the primary evaluation, injuries that are directly life-threatening and require rapid intervention are identified. Fatal injuries due to thoracic trauma include massive hemothorax, cardiac tamponade, tension pneumothorax, open pneumothorax, flail chest and lung contusion. Lung contusion is the most common parenchymal lung injury in blunt thoracic trauma and occurs in 25-80% of cases. The mortality rate in lung contusion is 10-25% (5–7).

In our case, contusion in both lungs, pneumothorax and laceration on the right side were detected as a result of thorax trauma. The ECG showed sinus tachycardia, right axis deviation, complete RBBB and long QTc. In the right-sided ECG, there were T-wave inversions from V3R to V6R. In reports investigating electrocardiographic changes in pneumothorax cases, phasic voltage changes and P, QRS and T axis deviation, right axis deviation, RBBB, left axis deviation, ST elevation, T inversion, and long QTc were detected in the ECG. Additionally, in these reports, a statistically significant correlation was found between the prevalence of ECG findings and the size of pneumothorax.3,4 The findings in our case were compatible with the literature. However, no report was found regarding right-sided ECG in thoracic trauma. In addition, in our case, although the lung expanded after tube throcostomy, complete RBBB continued in the ECG.

Clinicians use troponin I (TnI) levels to detect cardiac contusion in patients with thoracic trauma. However, in blunt chest trauma autopsy series, inequality was found between the incidence of elevated troponin and the incidence of cardiac contusion. Hypotension, hypoxia, and head trauma have been identified as other causes of trauma-related elevated troponin (8,9). In a study investigating the relationship between lung contusion and TnI, a correlation was found between the severity of pulmonary contusion and TnI (10). In a study conducted in patients with thoracic trauma, TnI increases in pneumothorax, hemothorax and lung contusion, apart from cardiac contusion. In addition, the most important diagnostic point in this study is that while troponin elevation continues in patients with cardiac contusion, it returns to normal in a short time in patients without cardiac contusion (11). The TnI level of our patient was normal. The TnI level measured 6 hours later was increased. But the troponin measured 3 days later was normal. The limited increase in troponin level and the rapid recovery of this increase led us to reasons other than cardiac contusion. In our case, complete RBBB that continued after lung expansion was attributed to lung contusion.

In lung contusion, blood leaks into the alveolar space, resulting in decreased lung compliance, ventilation-perfusion mismatch, and intrapulmonary shunt. Reflex vasoconstriction and pulmonary hypertension that develop in the affected tissue may occur as a protective response to pulmonary contusion. Thus, blood is directed from areas with parenchymal damage to areas with better oxygenation (6,7). The development of acute pulmonary hypertension may lead to ECG changes, as in pulmonary embolism. Sinus tachycardia, complete RBBB, right axis deviation, and QTc prolongation, which was also seen in our patient, have been frequently reported in cases of pulmonary hypertension (12). Additionally, T wave inversions on the right-sided ECG have been reported in cases with acute pulmonary embolism (13). In addition, along with the improvement of our patient's clinical findings, the simultaneous return of the troponin level and chest X-ray to normal, and the normalization of the right and left sided ECG supported our diagnosis.

Conclusion

In thoracic trauma, both left and right-sided ECG changes may occur as a result of lung contusion. ECG findings improve as the lung contusion heals.

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Case Report

Journal of Emergency Medicine Case Reports

A Prodigious Diagnosis of Acute Epiploic Appendagitis: A Rare Case Report

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Abstract

Appendices Epiploicae, also referred as Epiploic appendages, are 50-100 fat filled finger like projection from the serosal surface of large intestine. Epiploic Appendagitis (EA) is a self-limiting, benign disease process which results from the inflammation of these Appendices Epiplocae or thrombosis of the draining vein of Appendices Epiplocae. We report a case of 49 years old female, generally well, presented to the Emergency Department with complaints of a painful lump in her left iliac fossa growing in size for last four weeks. She presented to us because of acute increase in size and pain, resulting in significant discomfort. Owing to her history of cervical intra epithelial neoplasia we arranged a CT abdomen and pelvis with contrast fearing some sinister underlying ongoing pathology causing her symptoms. But to our surprise CT reported an underrated cause of her abdominal pain, EA. We were surprised of her presentation as a hard abdominal lump which was quite unusual for EA to present as. We assume it was secondary to an extensive underlying local inflammatory reaction. Patient was reassured and treated with NSAID, antibiotics and follow up with surgery ambulatory care. We authors are reporting this case of primary EA because we think every emergency and primary care physicians should be aware of this very rare condition which might present to emergency as an acute abdominal presentation mimicking other common presentations like acute diverticulitis and acute appendicitis. Being aware of this condition is utmost important to diagnose it early and avoid more invasive surgical managements and unnecessary antibiotic usage.

Keywords: Epiploic Appendagitis, Acute Abdomen, Emergency Department.

Introduction

Epiploic appendages, also referred to as Appendices epiploicae, are fat filled finger like projections originating from the serosal surface of large intestine, out pouching into the abdominal cavity. They are originating parallel to the outer layer of three longitudinal muscle bands of large intestine called taenia coli and in two rows, anterior and posterior(1). There are 50-100 epiploic appendages in an adult human (2).

Epiploic Appendagitis (EA), first described by Lynn et al. in 1956, a rare cause of acute abdominal pain/ presentation in Emergency Departments (ED). Primary appendagitisepiploica is a self-limiting disease process, resulting from inflammation of one or more epiploic appendage/s or rarely thrombosis of draining vein of a epiploic appendages. Secondary appendagitisepiploica is a resulting secondary inflammation of epiploic appendages which springs from a primary inflammation of an adjacent peritoneal structure, examples: appendicitis or diverticulitis or cancerous process (3).

We aimed to discuss this case of a minacious presentation of acute abdominal pain, which to our pleasant surprise, lead us to a very rare but amicable diagnosis of Epiploic Appendigitis.

Case

49 years old female presented to the ED with complaints of a painful lump in her left illiac fossa growing in size for last four weeks. She presented to us in ED as she noticed it increased in size significantly in last few days and pain became much worse. She denied any fever, weight loss, nausea, vomiting, loss of appetite, dizziness. No dysuria/ diarrhoea. She further denied any vaginal discharge/ bleeding/ spotting/ dyspareunia. Only significant past medical history was, she was diagnosed as cervical intra epithelial neoplasia 1 (CIN1) and recent cervical smear 2 weeks back confirmed no progression of the disease. When the patient's vital signs and physical examination evaluated in ED: Blood Pressure: 136/76mmhg, heart rate-89/min, SpO2-98% on Room Air, respiratory rate-19/min, Body Temperature-36.5°C. Chest: Bilateral (B/L) normal vesicular breath sounds (VBS). CVS= normal S1, S2 audible and no murmur. Abdomen: a hard, tender lump palpated in left lilac fossa, non-mobile and undefined lower edges, approx. 5x5cm size. Rest of the abdomen was soft and bowel sound was normal. We were surprised of her presentation as a hard abdominal lump which was quite unusual for EA to present as. We assume it was secondary to an extensive underlying

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local inflammatory reaction. Urine dipstick and urine beta HCG both came back negative. Her blood report showed raised WBC and CRP and normal renal and liver function tests. At this point our main concern was a malignancy in background of the history of CIN-1. Other differentials we wanted to rule out were Diverticulitis/ diverticular abscess, ruptured ovarian cyst or ovarian torsion. Hence we decided to go ahead for a CT abdomen and pelvis with contrast, weighing risk of radiation exposure vs. missing a more sinister pathology undergoing. To all of our pleasant surprise, CT reported that there was an acute left iliac fossa (LIF) EA of the sigmoid colon with target sign on the coronal MPR causing significant surrounding fat standing reaching the left inguinal canal with adjacent soft tissue swelling. No convincing hernia as shown in Figure-1. We referred the patient for emergency review by general surgeons. Patient was initially hospitalized with Non-Steroidal Anti-inflammatory Drug (NSAIDS) and Co-Amoxiclav with follow up in surgery care unit.

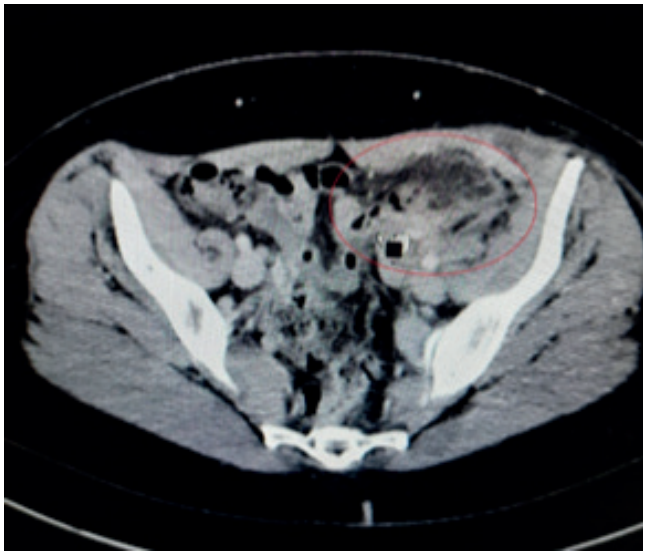

Figure 1. Left Iliac Fossa Epiploic Appendagitis of Sigmoid Colon with Target Sign with significant surrounding soft tissue swelling suggestive of inflammation, extending to left inguinal canal.

Discussion

Primary appendagitisepiploica is a rare, self-limiting inflammatory or ischaemic process involving the epiploic appendages, which are fat filled finger like projections originating from the serosal surface of large intestine into the peritoneal cavity. An adult human body they are usually 50-100 in number, between 0.5 and 5 cm long, containing a vascular stalk (two arterioles and one venule) attaching it to the surface of the colon/ large intestine. Primary appendagitisepiploica is a disease presenting mostly between 2nd to 5th decades of life (2). It is more common in obese women and one who recently lost weight, as these structures are larger in them, make them more prone to torsion (4).

Primary appendigitisepiploica presents with non-specific, localized abdominal pain, non-radiating, sharp in nature, mostly in the lower abdomen, left lower abdomen greater than right lower abdomen, usually pain does not get aggravated by movements, without any guarding or rigidity or palpable mass (5). Nausea, vomiting, abdominal bloating etc. are non-specific symptoms associated (6). In our case, patient although presented with a hard lump in the left lower abdomen, which not a common presentation of EA. We were surprised of her presentation as a hard abdominal lump which was quite unusual for EA to present as.

EA is primarily a CT/ Radiology diagnosis as this condition is rare and lacks any specific clinical feature to diagnose this condition. In CT, inflammation is mostly around the epiploic appendages and sparing the colonic wall and colonic diverticula, excluding acute diverticulitis or colitis (7). “Hyper attenuation ring sign” in CT scan is highly pathognomic of EA. In non-enhanced CT an solitary fat density which originates from the large bowel wall signifies the inflamed or ischaemic epiploic appendages. A hyper-attenuation rim surrounding this solitary fat density is the inflamed visceral peritoneum, giving rise to “Hyper attenuation ring sign” (8). “Fat stranding sign” is a more severe CT sign when inflammation spreads to the adjacent mesentry (5). In our case, the patient was diagnosed with EA by contrast-enhanced CT. CT showed Left Iliac Fossa EA of Sigmoid Colon with Target Sign with significant surrounding soft tissue swelling suggestive of inflammation (Figure 1).

Conclusion

We authors are reporting this case of primary EA because we think every emergency and primary care physicians should be aware of this very rare condition which might present to emergency as an acute abdominal presentation mimicking other common presentations like acute diverticulitis and acute appendicitis. Being aware of this condition is utmost important to diagnose it early and avoid more invasive surgical managements and unnecessary antibiotic usage.

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Case Report

Journal of Emergency Medicine Case Reports

A Case of Sigmoid Volvulus Associated with Constipation

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Abstract

Sigmoid volvulus (SV) is a rare cause of intestinal obstruction in adolescents. Delay in diagnosis may lead to complications such as necrosis and perforation of the volvulated colon. Clinical presentations are non-specific and the diagnosis is based on high clinical suspicion. Herein, we report a 14-year-old male patient who presented to the emergency department with abdominal pain and no stool output. It was learned that the patient had these complaints for the last four years and had been receiving constipation treatment. In abdominal X ray, the classic coffee bean finding of the sigmoid volvulus has seen. A diagnostic laparotomy was performed, which revealed a 180-degree rotation of the mesentery and a significant diameter difference in the colon due to significant dilatation proximal to the volvulus. This case with SV without any complications, reminds us that SV should be considered as a differential diagnosis in patients presenting with acute or recurrent abdominal pain and intestinal obstruction.

Keywords: Sigmoid volvulus, constipation, emergency

Introduction

Sigmoid volvulus (SV) is a rare but potentially life-threatening condition in pediatric patients. It may resolves pontaneously; therefore the diagnosis is usually missed or delayed (1). Delay in diagnosis may lead to complications including necrosis and perforation of the volvulated colon (2). Clinical presentations are non-specific and the diagnosis is based on high clinical suspicion. SV manifests acutely with abdominal pain, bloating and vomiting. However, the chronic form has an insidious course with vague symptoms at the time of diagnosis (3). In this article, we present a 14-year-old patient who had multiple emergency room visits and was evaluated as constipation in almost all of his visits, which eventually diagnosed as SV through detailed examination and evaluation. Key points;

1. Sigmoid volvulus should be considered in patients presenting with recurrent abdominal pain and constipation.
2. The clinical course of sigmoid volvulus in pediatric patients may be occult.

Case

A 14-year-old male patient was admitted to the pediatric emergency department with complaints of abdominal pain and no stool output for the last three days. When his history was questioned, it was learned that he had spontaneously regressing abdominal pain and constipation for the last four years, he did not have nausea and vomiting during these periods, he frequently applied to the emergency department with complaints of abdominal pain and was discharged after constipation treatments were arranged. The patient was evaluated in the emergency department; his vitalsigns were as follows: pulse rate 90/min, respiratory rate 19/min, oxygen saturation 98%, blood pressure 110/70 mmHg. There was no fever and vomiting. Physical examination revealed abdominal distension and diffuse tenderness in all quadrants. On rectal palpation, the ampulla was empty and there was no fecal contamination or gas discharge. There was no free air under the diaphragm in the standing abdominal X ray, but dilated loop of large bowel and the classic coffee bean finding of sigmoid volvulus were observed

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(Figure-1). Laboratory tests were as follows: Hemoglobin (Hb) 14.5 g/dl, White Blood Cell (WBC) 4.24 x10.e3/uL, Neutrophil count (NEU) 1.90 x.e3/uL, C-ReactiveProtein (CRP) 2.9 mg/L and liver and renal function tests were within normal range. Abdominal computed tomography (CT) was performed as a further investigation because the patient had no stool output. The transverse colon was 9 cm in diameter and appeared dilated, and 180-degree rotation of the mesentery was observed at the level of the descending colon where the dilated are a terminated. Sigmoid colon and rectum were thin and collapsed. Redundant sigmoid colon volvulus was observed. The patient was evaluated by pediatric surgery department. After oral intake was stopped and a nasogastric catheter was inserted, a small amount of fecal contamination was observed with bedside rectal irrigation. Diagnostic laparotomy was performed by the pediatric surgeon and a 180-degree rotation of the mesentery and a significant diameter difference in the colon due to significant dilatation proximal to the volvulus area were observed. There was no evidence of necrosis and the rest of the colon was healthy and normal (Figure-2). Considering that resection anastomos is might not be safe due to the diameter difference in the colon, it was decided to open a colostomy first. The pathologic examination of the biopsy material from the distal part of the dilated loop of large bowel was evaluated for Hirschprung's disease and no aganglionic area was observed. He was discharged on the 5th postoperative day. After 2 months, the colostomy was closed by resection anastomosis from the redundancy to the sigmoid colon and no complications were reported.

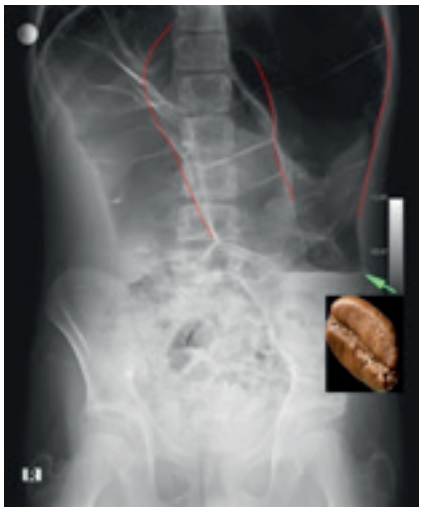

Figure 1. Coffee bean image seen in sigmoid volvulus

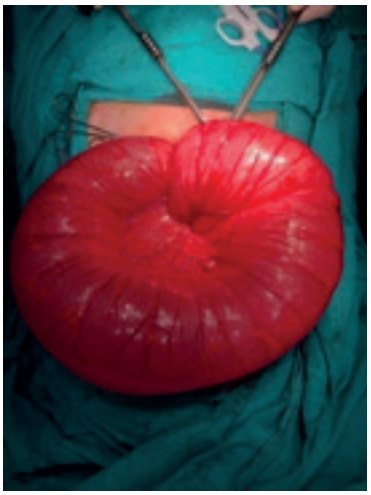

Figure 2. The intestinal anus volvulated 180 degrees and appears to be greatly dilated

Discussion

SV is a rare cause of intestinal obstruction inadolescents (2). Most patients with SV present with nausea, abdominal pain, bloating and vomiting which start insidiously, progresss lowly and settle within days (4). In our patient, other findings except vomiting were present. Only three studies were made with pediatric volvulus cases. One of them was made by Salas et al., in which a total of 63 cases were reported from 1941 to 2000 having the largest sample among all studies (4). Also, Colinet and colleagues reported 13 cases from 2001 to 2012 (5) and Atamanalp and co-authors presented 19 cases from a Turkish hospital (6).

The median age in SV is seven years and the incidence is higher in males than females (3.5:1) (4). These patients are usually relieved with stool or flatulence and are therefore frequently followed up with a diagnosis of constipation. Because of its insidious course, most patients present to the hospital 3 to 4 days after the onset of symptoms (1). Our case was also evaluated as constipation on multiple hospital admissions.

Various predisposing factors have been described among children. Hirschsprung's disease is one of the conditions associated with both transverse colonic and sigmoid volvulus. This disease was excluded in our patient by the abundant ganglion cells demonstrated in the biopsy specimen.

Physical symptoms are non-specific and the diagnosis can be made with complementary imaging studies such as

radiographs and CT scanand high clinical suspicion (1). Abdominal X-rays in children are usually non specific and lessuseful in differentiating volvulus from other disorders. A plain abdominal X-ray is suggestive of sigmoid volvulus in approximately 29% of patients (7). While abdominal X-ray is not very sensitive in detecting sigmoid volvulus findings in children, our patients howed a classical finding.

The diagnosis of sigmoid volvulus requires obtaining a detailed history, conducting a thorough clinical examination, and accurately interpreting plain abdominal radiographs. Diagnostic findings include the "whirlpool" sign, formed by the expansion of the sigmoid colon around the mesocolon and vessels, and the "Birds beak" appearance of afferent and efferent colon segments on barium enema. However, as in this case, the classic "coffee bean" sign may not be prominent in most pediatric cases and is not specific for distinguishing from other intra-abdominal pathologies (8).

Sigmoid resection is the definitive treatment for both children and adults, but nonoperative decompression should be tried for elective resection in patients without peritonitis findings (7). Since there were no sign of peritonitis in our patient, decompression was tried but was not successful. Our case was treated with sigmoidectomy, and endcolostomy (Hartman) and primary anastomosis of there maining healthy small intestine. It has been reported that the prognosis of sigmoid volvulus is quite good when diagnosed and treated rapidly (4).

If left untreated, SV can progress to ischemic colon, hemorrhagic infarction and even death; since these outcomes are potentially life-threatening, physicians should consider SV as a differential diagnosis in patients presenting with acute or recurrent abdominal pain and intestinal obstruction (1).

Conclusion

Clinicians and surgeons should be cautious and skeptical not to over look the diagnosis of sigmoid volvulus in a case presenting with symptoms of constipation, regardless of the patient's age.

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Case Report

Journal of Emergency Medicine Case Reports

Rare Diagnosis in The Emergency Department: Traumatic Testicular Dislocation

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Abstract

Traumatic Testicular Dislocation (TTD) is a rare form of scrotal trauma, involving the displacement of the scrotum directly towards the surrounding tissues due to direct compression, often occurring towards the inguinal area. In this report, we present a case of TTD in an adult patient involved in a motor vehicle accident and provide a brief review of this rare condition. An 18-year-old male patient was brought to our emergency department following a motorcycle accident. The patient reported a new swelling in his left groin. On physical examination, the left side of the scrotum was found to be empty, without the presence of hematoma. A swelling in the described area was palpated in the left inguinal region. A computed tomography (CT) scan did not reveal the left testis in its normal position; instead, the testis was identified in the left inguinal region. Scrotal Doppler Ultrasound was performed, revealing normal blood flow. The left testis was manually manipulated and descended into the left scrotum by Urologist. The patient was discharged with a prescription. TTD is a rare complication of blunt scrotal trauma. Even in the presence of multiple injuries, a thorough examination of the testicles is always recommended. Ultrasound (USG) and Doppler USG are the most useful diagnostic tools for TTD, but CT can also be helpful in cases of complex trauma. While TTD is not a life-threatening condition, a careful plan is recommended for the repositioning of the testis.

Keywords: Emergency Medicine,, Trauma, Testicular Dislocation

Introduction

Testicular traumas can lead to the production of antisperm antibodies, resulting in hypogonadism, which can ultimately lead to infertility (1). Traumatic Testicular Dislocation (TTD) is a rare form of scrotal trauma, involving the displacement of the scrotum directly towards the surrounding tissues due to direct compression, often occurring towards the inguinal area (2,3). TTD can occur either in isolation or in conjunction with blunt abdominopelvic trauma (4). While TTD is more commonly diagnosed immediately after trauma, delayed diagnoses have been reported in a minority of cases.

Ultrasonography (USG), color Doppler USG, and computed tomography (CT) are the primary diagnostic tools (5). Early diagnosis and treatment are recommended to preserve testicular function and minimize the risk of malignant transformation (2).

In this report, we present a case of TTD in an adult patient involved in a motor vehicle accident and provide a brief review of this rare condition.

Case

An 18-year-old male patient was brought to our emergency department following a motorcycle accident. Upon arrival, his vital signs were stable, and the Glasgow Coma Scale (GCS) was determined to be 15. Deformity and crepitation on palpation were noted in the patient's right wrist region. The patient also reported a new swelling in his left groin. There was no history of undescended testis or testicular surgery in the patient's medical history. On physical examination, the left side of the scrotum was found to be empty, without the presence of hematoma. A swelling in the described area was palpated in the left inguinal region. Rectal tone was normal, and no blood was detected in the urine test. A CT scan, conducted due to trauma, did not reveal the left testis in its normal position; instead, the testis was identified in the left inguinal region (Figure-1 and 2). Scrotal Doppler Ultrasound was performed, revealing normal blood flow. The patient was consulted to the urology department. It was determined that the left testis had displaced to the proximal part of the left inguinal canal. The left testis was manually manipulated and descended into the left scrotum. A repeat scrotal Doppler ultrasound conducted one hour later showed no scrotal pathology. Since no urological intervention was deemed necessary, the patient was discharged with a prescription for NSAIDs and advised to follow up at the urology outpatient clinic.

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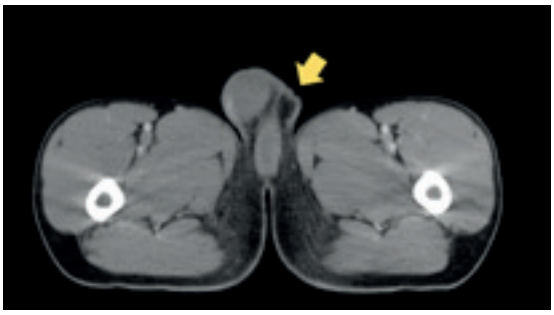

Figure 1. The left testis, which is not in its normal position on the CT scan (arrow head)

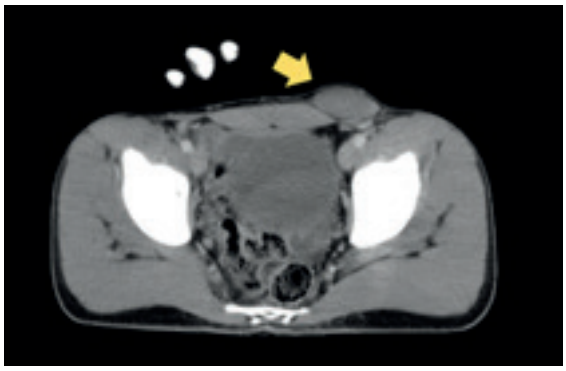

Figure 2. Identified left testis in the left inguinal region on the CT scan (arrow head)

Discussion

Testicles are organs that are less affected by traumas due to their location and mobility, accounting for less than 1% of all injuries. Approximately 85% of testicular injuries are due to blunt mechanisms, with motorcycle accidents also present in the etiology (6).

TTD, also known as traumatic dislocation of the testis, was first reported in 1818 by Clauby in a case where an individual fell from a wagon (7). The true incidence of TTD remains unknown, likely due to its rarity, leading to underreporting or oversight (4). In a case presentation reported by Zavras et al., 47 cases were identified in the literature, with the majority occurring after a motorcycle accident. The average age of these cases was 25.09 (6-62), and almost half of them were unilateral (49.5%).

The primary mechanism of TTD involves the rupture of spermatic cord fascias (external, cremasteric, and internal fascias) due to a direct force pushing the testis outside the scrotum (2). The most common area where the scrotum migrates is the superficial inguinal pouch, observed in half of the cases. Other areas include pubic, penile, canalicular, and entirely abdominal, perineal, acetabular, and crural regions.

During physical examination, a palpable mass in the migrated area is detected along with an empty hemiscrotum. However, it can often be overlooked during the initial assessment due to concurrent severe injuries (4). In our case, there was also associated extremity trauma. Undescended testis or undiagnosed cryptorchidism should always be questioned in these cases.

USG is a very important imaging modality in the diagnosis of testicular traumas and determining the need for surgery (6). The essential diagnostic tools for diagnosis of TTD include USG and Doppler USG. Additionally, abdominal and pelvic CT is recommended to assess the presence of pelvic and scrotal traumas associated with intraabdominal dislocation (4).

Manual reduction or surgical exploration are the treatment options for these cases. Manual reduction can be attempted within the first 3-4 days after trauma, before the resolution of edema and without the development of adhesions. However, manual reduction is successful in only about 15% of cases (2). In our case, manual manipulation was recommended by the urology consultant. Surgical intervention has advantages, such as requiring a relatively minor approach, carrying low morbidity, and the potential to detect accompanying testicular torsion or testicular trauma (4). In our case, surgical intervention was not recommended, the performed manual manipulation was successful, and no complications were observed during the one-hour follow-up.

Regardless of the chosen treatment, early diagnosis and prompt intervention are crucial. Delayed diagnosis, particularly after the fourth month, has been reported to show histological changes.

Conclusion

TTD is a rare complication of blunt scrotal trauma. Even in the presence of multiple injuries, a thorough examination of the testicles is always recommended. Ultrasound (USG) and Doppler USG are the most useful diagnostic tools for TTD, but CT can also be helpful in cases of complex trauma. While TTD is not a life-threatening condition, a careful plan is recommended for the repositioning of the testis.

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Case Report

Journal of Emergency Medicine Case Reports

Ischemic Stroke After Bee Sting: A Case Report

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Abstract

The global impact of a bee sting extends beyond common perceptions, showing various indications that may range from local reactions to more severe complication. This report details a compelling case involving a 63-year-old male who developed neurological deficits merely six hours after a bee sting. A 63 -year-old male patient was brought to the emergency department because of widespread redness, pain and itching at the sting area after a bee sting while pruning roses in the garden. During the follow-up in the emergency room, the patient developed weakness in his right lower/upper extremities. However, the diffusion magnetic resonance imaging of the patient showed an infarction detected in the area adjacent to the left lateral ventricle.Various clinical presentations after a bee sting have been described in the literature. Bee sting cases are generally associated with mild and transient local symptoms. Sometimes life-threatening allergic reactions may occur. The scarcity of the reported after-sting strokes emphasizes the need for further exploration into this uncharted territory to better comprehend and manage these exceptional indications. This case is presented for its rarity, underscoring the imperative for increased attention and research on these complications.

Keywords: Bee sting, ischemic, stroke

Introduction

The global impact of a bee sting extends beyond common perceptions, showing various indications that may range from local reactions to more severe complications such as vomiting, diarrhea, dyspnea, generalized edema, acute renal failure, hypotension, and collapse (1). The uncommon but documented after-sting complications such as vasculitis, serum sickness, neuritis, and encephalitis often develop within days to weeks following the initial incident (2).

Amid these considerations, the foremost concern is the potential occurrence of anaphylaxis life-threatening reaction with mortality implications. The documented incidence of anaphylaxis (3) resulting from bee stings varies within the range of 0.4% and 5%. Notably, severe allergic reactions leading to mortality typically surface before the age of 20, with a twofold prevalence observed in the male population (4).

While classical local allergic reactions following a bee sting are commonplace, their diagnosis relies heavily on a thorough patient history. In the current case, the medical history and physical examination findings are consistent, pointing to a presentation consistent with a local allergic reaction. Recognizing and understanding such reactions are crucial for prompt and effective medical intervention.

In addition to the complexity, the neurologic symptoms following bee stings are infrequently documented in the literature, with cases primarily emphasizing encephalitis and acute disseminated encephalomyelitis. The occurrence of a stroke directly related to a bee sting is even rarer, with the precise etiology remaining elusive. This report details a compelling case involving a 63-year-old male who developed neurological deficits merely six hours after a bee sting. The scarcity of the reported after-sting strokes emphasizes the need for further exploration into this uncharted territory to better comprehend and manage these exceptional indications. This case is presented for its rarity, underscoring the imperative for increased attention and research on these complications.

Case

A 63 -year-old male patient was brought to the emergency department because of widespread redness, pain and itching at the sting area after a bee sting while pruning roses in the garden, with a medical history of hypertension, type 2 diabetes, and coronary artery disease. He denied alcohol or tobacco use.

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Upon arrival for the ER, the vital signs of the patientwere as follows: blood pressure (BP):130/80 mmHg, heart rate (HR) 75 beats/min, respiratory rate (RR) 14/min, fever: 36.4 C° oxygen saturation: 98% in room air. His general condition was alert and oriented, the neurologic examination and, the mental-status examination was normal, GlasgowComa Scale (GCS) was 15 (E4, V5, M6)., In the other physical examinations, there was observed no tachycardia, no tachypnea and no murmur-rubbing-galloping. His respiratory system examination was clear according to the bilateral auscultation. The remainder of his physical examination was normal. After the physical examination, the laboratory tests were requested, and the symptomatic treatment was initiated. During the follow-up in the emergency room, the patient developed weakness in his right lower/upper extremities. In the control neurological examination, his mental status examination was normal, and the GCS score was 15. The patient developed right central facial paralysis, a 3/5 loss of strength in his right upper/lower extremities, and a positive Babinski sign on the right side. As the current clinical presentation changed, a non-contrast brain CT scan was performed, and no acute pathology was detected. However, the diffusion magnetic resonance imaging of the patient showed an infarction detected in the area adjacent to the left lateral ventricle (Figure-1). In response to the detection of an acute ischemic lesion in the imaging tests, rt-PA (recombinant human tissue-type plasminogen activator) was administered in accordance with the recommendations of the neurology consultant physician. The patient's relatives were informed about the treatment. However, since it was not accepted, the patient was administered oral (PO) medication, which included 300 mg of acetylsalicylic acid and 0.6 mg of LMWH (Low Molecular Weight Heparin) Enoxaparin., The patient was admitted to the neurology clinic and hospitalized. However, the patient was discharged on the 5th day of the hospital stay.

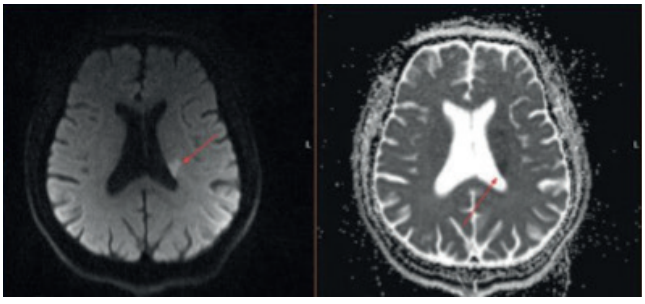

Figure 1. Diffusion magnetic resonance imaging of the patient showed an infarction detected in the area adjacent to the left lateral ventricle (red arrow)

Discussion

Various clinical presentations after a bee sting have been described in the literature. Bee sting cases are generally associated with mild and transient local symptoms. Sometimes life-threatening allergic reactions may occur. Classical local allergic reactions occur after bee sting, and the diagnosis depends on the medical history. In our case, the medical history and physical examination findings are consistent with the local allergic reactions. Many bites may cause urticaria and systemic toxicity including cardiovascular and neurologic problems (5). The reactions by bee sting are frequently mediated by IgE, but these reactions may also occur by IgG antibody and IgG venom complex triggered complement activation. The symptoms usually occur within the first few hours, but they can be observed even after hours. The connection between bee stings and strokes is an interesting topic, and there are potential mechanisms through which bee venom might influence the risk of stroke. However, it is important to note that research in this area may be limited, and the information available might not be conclusive.

Pathophysiologically, it is said that several mechanism scan cause this condition. Bee stings may trigger severe allergic reactions (anaphylaxis) in susceptible individuals. Anaphylaxis may lead to hypotension (low blood pressure), which, in turn, may affect cerebral blood flow and increase the risk of ischemic events such as strokes.Bee venom contains various inflammatory mediators, including histamine, thromboxane, and leukotrienes.These mediators may contribute to a hypercoagulable state and potentially promote conditions favorable to strokes.Vasoactive substances in bee venom may influence blood vessel tone and reactivity.Changes in blood vessel dynamics may impact the risk of stroke, particularly if there is a predisposition to vascular events (6-8).According to many studies, patients should be observed for a period of at least 6 hours (9,10). In our case, reactions were observed after the first hour and minimized through the treatment by the medical teams, but at the sixth hour of the follow-up, neurologic symptoms were developed.

Our patient had no previous medical history of epilepsy or cerebrovascular disease and here, we present ischemic stroke after bee sting, which is an extremely rare neurological involvement due to bee sting.

Conclusion

The scarcity of the reported after-sting strokes emphasizes the need for further exploration into this uncharted territory

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to better comprehend and manage these exceptional indications. This case is presented for its rarity, underscoring the imperative for increased attention and research on these complications.

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Case Report

Journal of Emergency Medicine Case Reports

A Case of Pulmonary Embolism after Receiving a Dose of Sildenafil

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Abstract

Several studies have posited the potential of PDE5Is to precipitate arterial and venous thrombotic or embolic incidents, albeit rare occurrences. The present investigation documents a case of pulmonary thromboembolism (PTE) in an elderly male patient, aged 81, following the consumption of sildenafil. An 81-year-old male patient who was previously diagnosed with syncope was admitted to our Emergency Department. He reported symptoms of dyspnea and vertigo. Approximately two hours before admission, there was a history of accidentally ingesting a total dose of 100 milligrams of sildenafil. The occurrence of PTE after sildenafil administration in this case, albeit rare, underscores the significance of meticulous evaluation of medications during patient history-taking. This case report, the importance of thorough efforts to rule out the diagnosis of PTE and to address even the slightest suspicion.

Keywords: Pulmonary embolism, phosphodiesterase 5 inhibitors, adverse effect

Introduction

On a globalscale, phosphodiesterase-5 inhibitors (PDE5Is) like sildenafil and tadalafil have garnered extensive prescriptions for the management of male erectile dysfunction. Sildenafil augments the actions of nitricoxide (NO) through the inhibition of phosphodiesterasetype 5 (PDE5), an enzyme responsible for the breakdown of cyclic guanosine monophosphate (cGMP) with in the corpus cavernosum. Nevertheless, it is recognized that vasodilation is not confined solely to the corpuscavernosum (1-3).

Several studies have posited the potential of PDE5Is to precipitate arterial and venous thrombotic or embolic incidents, albeit rare occurrences (4,5). The present investigation documents a case of pulmonary thromboembolism (PTE) in an elderly male patient, aged 81, following the consumption of sildenafil.

Case Report

An 81-year-old male patient who was previously diagnosed with syncope was admitted to our Emergency Department. He reported symptoms of dyspnea and vertigo. Approximately two hours before admission, there was a history of accidentally ingesting a total dose of 100 milligrams of sildenafil.

As per his family's account, the patient suffered a sudden loss of consciousness while seated on the couch, followed by a recovery to consciousness within a span of two to three minutes. Initial physiological assessments revealed a blood pressure of 115/75 mmHg, a heart rate of 86 beats per minute, a respiratory rate of 18 breaths per minute, a body temperature of 36.7 °C, an oxygen saturation level of 91%, and a finger stick blood glucose level of 215 mg/dL. The cognitive state was characterized by a heightened level of concentration. The findings of an auscultatory examination indicated the absence of any cardiac murmur and the presence of typical respiratory sounds.

During emergency monitoring, the patient experienced a syncope episode lasting one minute. After a period of bed rest, consciousness was restored. Subsequent blood pressure measurements recorded 90/60 mmHg initially and 120/80 mmHg after two hours of recumbency. The laboratory tests conducted upon admission yielded the following results: white blood cell count of 7.2 x 10^3/mm^3, hemoglobin level of 15.5 g/dL, platelet count of 130,000/uL, urea level of 68 mg/dL, creatinine level of 1.55 mg/dL, and d-Dimer level of 5.9 mg/L (with a normal range of 0-2 mg/L). The findings of the cerebral magnetic resonance imaging and computed tomography imaging (CTI) scans were unremarkable. Echocardiography revealed the presence of right ventricular

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dilatation and since the d-dimer value is also outside there reference range prompting the performance of chest CTI due to suspected acute PTE. A thrombus was observed within the major pulmonary artery. PTE was diagnosed through CTI evaluation (Figure-1).

An 80 mg dose of enoxaparin was administered via subcutaneous injection in the emergency department. Subsequently, the patient was admitted to the hospital under the care of the chest disorders clinic. Bilateral Doppler ultrasonography of the lower limbs revealed unremarkable findings.

After a 13-day hospitalization period, the patient was discharged with a follow-up care plan in place.

Discussion

The administration of sildenafil and other PDE5Is is frequently linked with adverse effects, including headache, dyspepsia, lumbago, myalgia, flushing, and rhinorrhea or nasal congestion. There is no established association between the use of PDE5Is and hypercoagulable states. However, some studies have suggested that in rare instances, PDE5I usage may be implicated in arterial and venous thrombotic or embolicevents. A hemodynamic impact of sildenafil has been observed to result in a decrease of approximately 8-10 mm Hg in systolic and diastolic blood pressure, as demonstrated in previous studies (5-7).

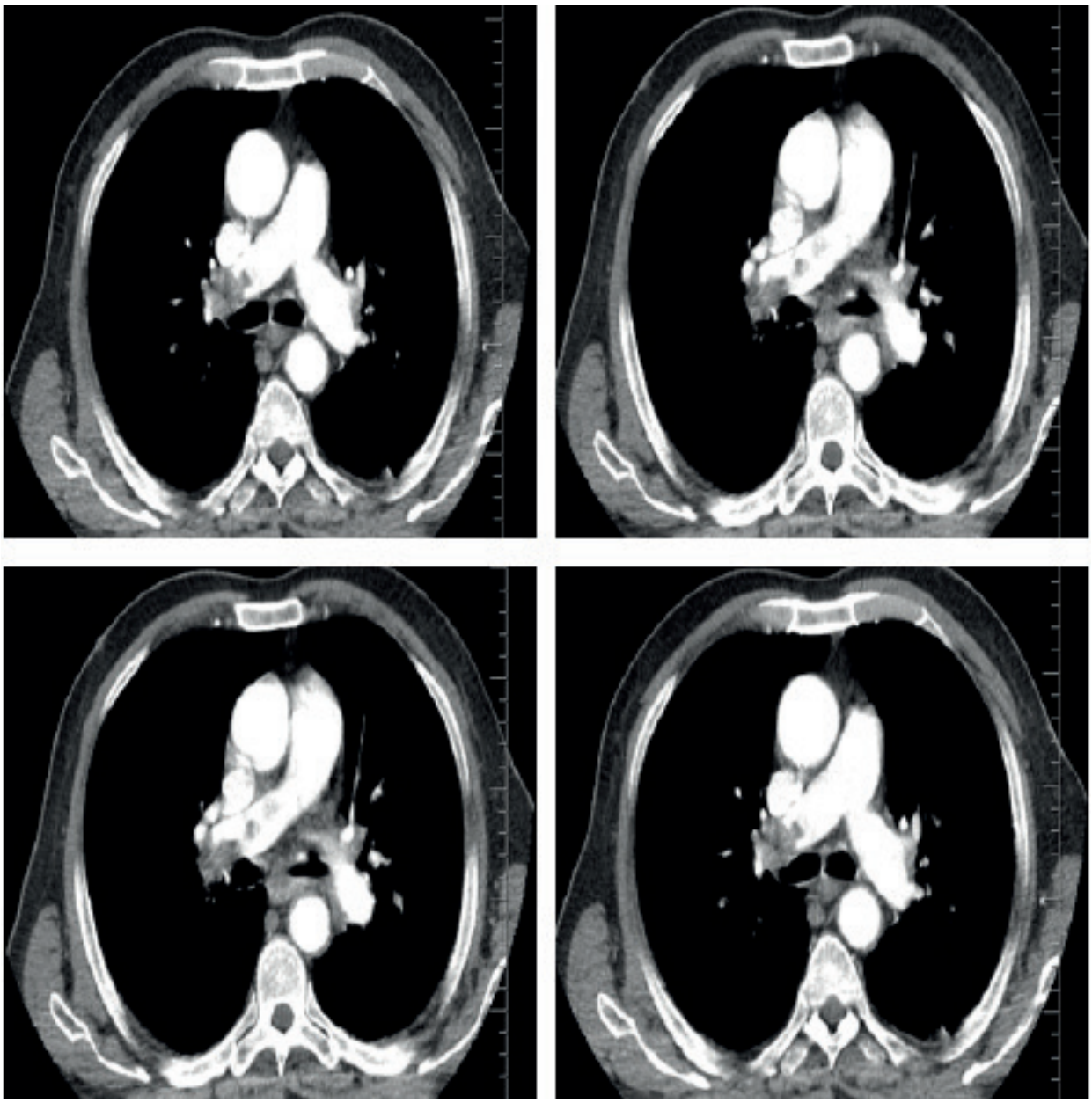

Figure 1. CTI Imaging of the Patient

The hypotension observed in this case was likely initially attributed to using sildenafil. Furthermore, the low D-dimer value under scores the importance of meticulous case evaluation and the judicious selection of diagnostic methods to avoid over looking the PTE, which can pose a significant risk to life (8). Rare occurrences of PTE have been documented in correlation with the usage of sildenafil. In a scientific article by Chi Chen, a case of PTE following the administration of tadalafil 20 mg to manage erectile dysfunction in a 56-year-old male devoid of prior coronary artery disease history is reported (9).

The patient presented with symptoms of hemoptysis, dyspnea, and mild left chest discomfort. The patient was transported to the emergency medical facility, where his vital signs, including blood pressure, body temperature, and pulse, were observed to be with in the standard range. However, tachycardia was detected. The electrocardiogram displayed the S1Q3T3 pattern, which was observed unintentionally (10,11).

The lack of similar findings in our case complicates the diagnosis and adds valuable insight to the literature, emphasizing the importance of thorough exclusion of pulmonary thromboembolism (PTE) in cases involving sildenafil usage. Treatment approaches for patients with pulmonary thromboembolism (PTE) differ based on the patient's clinical stability. In cases of stable patients, the primary treatment strategy involves the administration of anticoagulants. For unstable patients, treatment may necessitate thrombolytic therapy for reperfusion or interventions such as surgical thrombectomy and extra corporeal membrane oxygenation. Additionally, preventive measures to avert embolism recurrence and identification of the embolics ource are crucial aspects of management (11,12). In our case, a method by these treatment strategies was followed and investigations were performed to identify the source of the embolism to prevent recurrence.

Conclusion

In contrast to other cases, the individual under scrutiny was of advanced age and consumed sildenafil inadvertently, rather than intentionally for the treatment of erectile dysfunction. Additionally, unlike other instances, our subject presented with symptoms of syncope and mild chest pain, and their electrocardiogram did not display the S1Q3T3 pattern typically associated with pulmonary embolism.

The occurrence of PTE after sildenafil administration in this case, albeit rare, under scores the significance of meticulous evaluation of medications during patient history-taking. It also highlights that PTE may manifest with nonspecific symptoms and sometimes seemingly benign conditions. This under scores the importance of thorough efforts to rule out the diagnosis of PTE and to address even the slightest suspicion.

The case report has been composed with anonymized characteristics, ensuring that sensitive and detailed patient data have been omitted. However, these data remain accessible to editors and reviewers for evaluation and verification purposes. This practice is supported by the editorial and review process, there by maintaining the confidentiality of the patient while allowing for thorough scrutiny of the report.

There is noc onflict of interest between the authors.

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Case Report

Journal of Emergency Medicine Case Reports

Neuroleptic Malignant Syndrome in Puerperalwomen – Frozen in Summer

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Abstract

Neuroleptic malignant syndrome is an rare event, even more so among patient from puerperal period. The incidence risk is of 0.11%. It is often occurs when a patient is prescribed on antipsychotic for long duration of time. We report a case of a female patient, two weeks in post natal period presented with altered behaviour at home. She has prior admission being treated for post partum psychosis with anti psychotic. However antipschotic was withheld as she developed extrapyramidal symptoms while in ward. At home, she developed fever, altered sensorium and fitting-like-episode. On arrival patient was unresponsive and with features of impaired airway patency. She had unstable vital sign with elevated blood pressure, heart rate and temperature. Brain imaging came back normal, excluding brain pathology. She was diagnosed as neuroleptic malignant syndrome as fulfilled features of elevated creatine kinase. She was treated symptomatic by providing first aid to reduce temperature and started on benzodiazepines. She was subsequently admitted to intensive ward and responded well to treatment. Physician must be familiar and have high index of suspicious to identify and treat neuroleptic malignant syndrome. Without prompt treatment, it is highly fatal.

Keywords: Neuroleptic malignant syndrom, puerperal, anti psychotic, withdrawal

Introduction

"Syndrome malin des neuroleptiques," also known as neuroleptic malignant syndrome (NMS), was first described by Delay in 1960. NMS is an idiosyncratic adverse reaction related to the usage of antipsychotics (1). Antipsychotics have been widely used in recent days to treat psychiatric illness. First-generation antipsychotic (FGA)is well known to cause neuroleptic malignant syndrome (NMS), a rare adverse event. Thus, most psychiatrists opt for second-generation antipsychotics (SGA). The prevalence of NMS is extremely low in the general population, with an incidence risk of 0.11% (2). In the post-partum period, female patients have an increased risk of developing NMS (3). We discuss the challenges and limitations in managing a patient within a puerperal period presented with altered mental status.

Case

A 21-year-old female, para one, 15 days postpartum, with a history of prior admission for puerperal psychosis. On previous admission, she was started on tablet risperidone, to which she developed extrapyramidal symptoms after ten days of medication. Subsequent risperidone was withheld, and she was started on artane. Later, the patient was discharged well against medical advice. Three days later, the patient was brought in by her husband with the complaint of reduced consciousness, fever and stiffing of limb for two days, worsening on the day of presentation.

On arrival, the patient was unresponsive with a blank stare (E4V2M1), the presence of stridor and increased oral secretion but saturating well on high flow mask oxygen. Her heart rate was 180bpm, showing extreme sinus tachycardia with normal blood pressure (121/59mmHg), and her hyperpyrexia with temperature of 42.8 degrees Celsius. Neurological examination reveals areflexia with limb tonic rigidity and non reactive pupil of 6mm bilaterally. She was intubated uneventfully for airway protection and maintenance. Initially, the impression was neuroleptic malignant syndrome, given her history of being started on antipsychotics.

Blood panels were sent, and computed tomography (CT) of the brain was performed to exclude intracranial haemorrhage. Immediate first aid management was performed to decrease the temperature with ice packing and sponging. She was then started on boluses of midazolam and subsequently midazolam infusion with maintenance fluid. Her blood investigation reveals elevated creatinine kinase 4890 U/L and LDH 854 UL. Her other blood parameters and CT brain were normal. She also tested negative for NMDAR encephalitis.

She was admitted to the intensive care unit (ICU). Her condition improved tremendously upon being initiated

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on bromocriptine. She was warded in ICU for six days, subsequently extubated and discharged to general ward. We have obtained consent from the patient to publish the case.

Discussion

Risperidone has been closely associated with the highest cases of atypical antipsychotic causing NMS (1). Despite risperidone being an atypical antipsychotic, it causes NMS symptoms similar to typical antipsychotics. Previously, it was thought that(SGA)was safe from causing NMS; however, several cases have been reported recently (4). SGA is still strongly associated with causing NMS, with a threefold higher chance of developing (5). Interestingly, NMS caused by SGA lacks the usual cardinal features. This is a belief due to the different pharmacological properties of SGA (4). Risperidone is specifically known to cause marked extrapyramidal syndrome and is followed by NMS (6). This is true in our patients. A shorted duration of antipsychotic was observed among patients taking risperidone who developed NMS. Our patient was prescribed SGA for eight days. At that time, the patient was treated for psychosis. However she was not investigated further for anti-NMDAR encephalitis. An author summarised that anti-NMDAR encephalitis may present with psychiatric symptoms during the early phase (7,8). An author explained that as rare as NMS in pregnancy, it should be considered and investigated in depth among patients diagnosed with postpartum psychosis (9).

Classical features of NMS are muscle rigidity, hyperthermia, involvement of autonomic dysfunction and elevated creatinine kinase (7). The study, noted that hyperpyrexia and autonomic gastrointestinal symptoms such as sialorrhea arethe first symptoms for patients who developed NMS after a few days from initiating antipsychotics (7). Besides, most patients become symptomatic 1-3 days before formal diagnosis (6). Despite that, patients develop symptoms gradually, may become severe, and rapids deteriorate within a few hours (10). In the early stage of NMS, it is difficult to diagnoseas symptoms are often subtle (9).

In rare occurrence, NMS can occur among patients whose neuroleptic has been discontinued. The reported case involved a long-term neuroleptic patient, and NMS is seen as abrupt neuroleptic withdrawal (11). Uniquely, our patient was only on antipsychotics for a brief period. A similar case was described where a puerperal patient developed NMS after ten days on antipsychotic (12).

NMS is managed primarily consist of support therapy which requires temperature control with cooling device, fluids and electrolyte correction. Study also show that treatment with bromocriptine gives rapid recovery (13). After cooling therapy, benzodiazepine is recommended as part of initial therapy. However trial with bromocriptine can be done if patient does not respond well with initial therapy (14). In our patient, even though she responded with benzodiazepine, she was started on bromocriptine to achieve complete recovery.

A close differential diagnosis for NMS is anti-NMDA receptor encephalitis, which will present almost similar to NMS but without elevated CK (7). Any patient diagnosed with NMS should also be investigated for autoimmune encephalitis. There is debate about NMS being a feature of anti-NMDA receptor encephalitis (7).

Conclusion

Most NMS being life threatening case are diagnosed in inpatient and emergency settings. Thus, we should have a high index of suspicion among patientstaking antipsychotics and developing extrapyramidal syndrome. We highlight that NMS is uncommon, and diagnosis is often missed. This is because the initial presentation is subtle. Clinicians should always consider NMS as adifferential diagnosis in patients who develop altered mental status, hyperpyrexia and rigidity, especially if they are started on dopaminergic antagonists. Efforts should be taken to identify early features among patients coming for follow-up aclinic setting.

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Case Report

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A Case Report of non Hodgkin Lymphoma Presenting With Upper Gastrointestinal Tract Bleeding

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Abstract

Possible causes of upper gastrointestinal bleeding include peptic ulcer, esophageal ulcer, variceal bleeding and malignancies. A patient presenting with gastrointestinal bleeding should be evaluated as an emergency and treatment should be started as soon as possible. Hemodynamically stable patients should undergo endoscopy for both diagnostic and therapeutic purposes. In cases where bleeding cannot be stopped by endoscopic methods, invasive vascular methods and surgery may be tried. A 48-year-old male patient presented to the emergency department with complaints of nausea, vomiting in the form of coffee grounds and black stools. Gastrointestinal endoscopy revealed bleeding from a large, fragile mass in the stomach. Our patient was diagnosed as non-hodgkin lymphoma with early biopsy and the bleeding could not be stopped by endoscopic and surgical methods. With the chemotherapy given to our patient, the mass shrunk significantly and bleeding was under control. It should be kept in mind that early biopsy diagnosis and early chemotherapy may be a life-saving option in gastrointestinal bleeding caused by malignancy.

Keywords: Upper gastrointestinal tract bleeding, non hodgkin lymphoma, failure of medical treatment

Introduction

Initial evaluation of patients with acute gastrointestinal (GI) tract bleeding includes assessment of hemodynamic stability and resuscitation if necessary. Diagnostic studies (usually endoscopy) follow diagnostic goals and, when possible, treatment of the specific disorder(1). Non-Hodgkin's lymphomas (NHL) can have nodal or extranodal onset. NHL is seen in patients of all ages, races and socioeconomic levels(2)T cell progenitors, mature B cells, mature T cells, or (rarely. Patients with lymphoma with gastrointestinal system (GIS) involvement may present with anorexia, early satiety, weight loss, vomiting, ileus, perforation or bleeding. In this case report, we will try to present a case of lymphoma presenting with acute upper gastrointestinal tract bleeding refractory to treatment.

Case

A 48-year-old male patient was admitted to the emergency department with complaints of dizziness, intense weakness, vomiting in the form of coffee grounds and black diarrhea. On admission to the emergency room, blood pressure arterial: 80/55 mm-Hg, pulse rate: 130/min, temperature: 36.8 C°. Blood tests revealed hemoglobin (hb):8.5 gr/dl, ldh:642 U/L, hemostasis parameters were normal, urea 80 mg/dL, creatinine 1.3 mg/dl. The patient's rectal palpation was melena smear. An intravenous line was opened rapidly and saline (SF) infusion was started at 200 cc/h. 80 mg pantoprazole intravenous (IV) puff followed by infusion at 8 mg/h was started. The patient was ordered 3 units of erythrocyte suspension and hospitalized in the intensive care unit. The patient was admitted to the internal medicine outpatient clinic about 2 weeks ago with complaints of nausea, vomiting and abdominal pain. Blood tests performed at the outpatient clinic revealed hb:13.2 gr/dl and no pathologic values in biochemistry parameters. The patient had no special history. There was no history of regular medication and gastrointestinal surgery. Abdominal tomography (CT) was ordered. In the abdominal CT scan, it was reported as; "a lobulated contoured space-occupying solid mass lesion starting from the fundus level of the stomach and extending to the antrum, filling the lumen of the stomach almost completely at these levels and exceeding the stomach wall at the level of the great curvature, showing

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heterogeneous contrast uptake, measuring approximately 125x91 mm in the coronal plane at its widest point". During upper GI endoscopy, multiple biopsies were taken from the mass at the level of the great curvature. Although the patient was advised to continue hospitalization for the mass that allowed partial passage of the expected pathology result, the patient was discharged voluntarily. After admission to the emergency department, the patient was hospitalized in the intensive care unit, a central venous catheter was inserted and 1 unit of erythrocyte suspension was given without complications. During this period, the patient's arterial blood pressure and tachycardia improved slightly and urgent upper GI endoscopy was performed. It was observed that the patient had diffuse bleeding from the mass starting from the fundus and extending to the antrum. Endoscopic intervention (such as sclerotherapy, band ligation) could not be performed. The patient was then referred to oncologic surgery. The patient was taken to emergency surgery and was told that no surgical procedure could be performed for the mass that disrupted hemodynamics. The previous biopsy result of the patient was plasmablastic lymphoma and the Hb value was 7.7 g/dl in the control examinations. The remaining 2 units of erythrocyte suspension were also administered. Tranexamic acid was started 3x250 milligram IV. Since the hemostasis panel was normal, TDP (fresh frozen plasma) or cryoprecipitate was not given. The patient's relatives were interviewed. All possible risks including gastric perforation were explained. Consent for chemotherapy was obtained. DA-EPOCH chemotherapy protocol was started. With the treatment, the patient's GI bleeding stopped, her melena decreased, her vital signs improved and the need for erythrocyte suspension replacement disappeared. The patient, whose general condition improved, was transferred to the hematology service approximately 2 weeks later.

Discussion

Gastrointestinal bleeding originating proximal to the ligament of Treitz is called upper gastrointestinal bleeding and bleeding originating distal to the ligament of Treitz is called lower gastrointestinal bleeding. The initial evaluation of a patient with suspected clinically significant acute upper gastrointestinal bleeding includes history, physical examination and laboratory tests. The goal of the assessment is to determine the severity of the bleeding, its potential sources, and the presence of conditions that may affect subsequent management. The information collected as part of the initial assessment is used to guide decisions regarding triage, resuscitation, empirical medical treatment and diagnostic testing(1). Hematemesis is suggestive of upper GI bleeding. The majority of melena is also caused by upper GI bleeding (90%) but can also be caused by bleeding from the oropharynx or nasopharynx, small bowel or colon(3). Possible causes of upper gastrointestinal bleeding include peptic ulcer, esophageal ulcer, Mallory-Weiss tear, variceal bleeding, portal hypertensive gastropathy and malignancy. Physical examination is an important component in the assessment of hemodynamic stability. Tachycardia, orthostatic hypotension, and hypotension in the supine position are among the signs of hypovolemia(1). Our patient presented to the emergency department with hematemesis and melena. These findings suggested that the bleeding probably originated from the upper gastrointestinal tract. Hypotension, tachycardia and dizziness were findings suggesting the severity of bleeding. Laboratory tests that should be obtained in patients with acute upper gastrointestinal bleeding include complete blood count, serum chemistry, liver tests and coagulation studies(1). The initial hemoglobin level in patients with acute upper GI bleeding may be at baseline because the patient has lost whole blood. Over time, the hemoglobin level will decrease due to the entry of extravascular fluid into the vascular cavity and dilution of the blood due to the fluid administered during resuscitation. The hemoglobin level should initially be monitored every two to eight hours depending on the severity of bleeding(1). Because blood is absorbed as it passes through the small intestine and patients may have reduced renal perfusion, patients with acute upper GI bleeding typically have a high blood urea nitrogen (BUN)-creatinine or urea-creatinine ratio (values >30:1 or >100:1, respectively) (4). The higher the ratio, the more likely the bleeding is from an upper GI source (5). At the time of diagnosis, hb was 8.5 g/dl, urea was 80 mg/dL, creatinine was 1.3 mg/dl and hemostasis panel was normal. The urea/creatinine ratio was also high. Although the principles are similar in the treatment of all patients with upper GI bleeding, there are some special considerations when it comes to patients presenting with hemodynamic instability (shock, orthostatic hypotension) (1). Adequate peripheral access should be provided with two 18-gauge or larger intravenous catheters and/or a large-diameter, single-lumen central cordis.

For patients with active/live bleeding and hypovolemia, transfusion should be guided by hemodynamic parameters (e.g. pulse and blood pressure) rather than serial parameters, the rate of bleeding, estimated blood loss and ability to stop bleeding. If the initial hemoglobin level is low (<7 g/dL), transfusions should be initiated (6). However, transfusion support should not be delayed while waiting for laboratory test results in a patient with acute bleeding. Patients admitted to the hospital with acute upper GI bleeding are typically treated with a proton pump inhibitor (PPI)(1). After 80 milligrams of PPI, pantoprazole infusion was started at 8 mg/h. A wide vascular access was opened and iv SF infusion was started. Since the patient had active bleeding and hemodynamic instability, erythrocyte suspension replacement was performed even though the patient's hospitalization Hb value was <7 g/dl. Since the

hemostasis panel was normal, no blood or blood products were given for the hemostasis system. Upper endoscopy is the preferred diagnostic method for acute upper GI bleeding (7). Endoscopy has high sensitivity and specificity for detecting and identifying bleeding lesions in the upper gastrointestinal tract. In addition, once a bleeding lesion is identified, therapeutic endoscopy can provide acute hemostasis and prevent recurrent bleeding in most patients. Early endoscopy (within 24 hours) is recommended for most patients with acute upper GI bleeding. Endoscopy is recommended within 12 hours of presentation for patients with suspected variceal bleeding (1). Endoscopic therapy is indicated for the treatment of most ulcers with signs of new bleeding that increase the risk of recurrent bleeding. With appropriate treatment, high-risk lesions have recurrent bleeding rates of 5 to 20%, depending on the endoscopic appearance of the ulcer. On the other hand, ulcers with a clear base or flat pigmented spot have a low risk of recurrent bleeding (1). Surgery in peptic ulcer disease is the basis for emergency treatment of life-threatening complications such as bleeding, perforation, obstruction, and suspected malignancy, as well as disease refractory to medical therapy. Surgical options for peptic ulcer disease range from local therapies that only manage ulcer-related complications (ie, bleeding, perforation, or obstruction) to definitive ulcer operations. Generally, the least morbid procedure that will adequately manage the patient's problem should be used in each instance. Definitive ulcer operations may be directed largely or solely at reducing acid secretion (which impairs mechanisms of healing), or they may include strategies for managing the susceptibility of the ulcer bed to recurrent injury. Reconstruction is necessary following partial gastrectomy to reestablish gastrointestinal continuity. The Billroth I, Billroth II, and Roux-en-Y reconstruction techniques are the most common (8). After our case was partially stabilized, endoscopy of the upper gastrointestinal tract was performed. Although local methods (sclerotherapy) were tried because the mass was very large and fragile, they were not effective. The patient was then taken to emergency surgery and the gastrointestinal system surgeon reported that a palliative therapeutic procedure could not be performed because the mass was too large and invaded many vascular neural structures. The clinical presentation of NHL varies according to histologic subtype and sites of involvement. While some subtypes of NHL show variable lymphadenopathy for years (indolent), others are highly aggressive and can lead to death within weeks or even days if left untreated. Even within a specific NHL subtype, the clinical picture can vary greatly between individual patients (2). Aggressive lymphomas often present subacutely or acutely with a rapidly growing mass; they may present with structural symptoms such as fever, night sweats or weight loss; tumor lysis syndrome, ileus or bleeding. Examples of aggressive NHL include diffuse large B-cell lymphoma, Burkitt lymphoma, precursor B and T lymphoblastic leukemia/lymphoma, adult T-cell leukemia/lymphoma and plasmablastic lymphoma. Indolent lymphomas are usually insidious, presenting with lymphadenopathy, hepatomegaly, splenomegaly and/or cytopenias that grow slowly or increase and decrease over months or years. Examples of slow NHL include follicular lymphoma, chronic lymphocytic leukemia/small lymphocytic lymphoma and splenic marginal zone lymphoma (2). A minority of patients initially present with extranodal lymphoma (primary extranodal NHL), but many more patients develop extranodal disease (secondary extranodal NHL) during the course of their disease. Examples of extranodal manifestations of NHL include Patients with primary gastrointestinal tract lymphoma may present with anorexia, early satiety, weight loss, vomiting, ileus, perforation or bleeding (2). The most common site of primary extranodal disease is the gastrointestinal tract. Plasmablastic lymphoma (PBL) is a rare but highly aggressive lymphoma usually observed in the setting of HIV disease (9). Due to the rarity of this disease, there is no accepted evidence-based systemic treatment for PBL. CHOP remains a widely used treatment regimen, especially in resource-limited settings. However, more intensive regimens are recommended in PBL. The preferred chemotherapy regimen is the dose-adjusted (DA)-EPOCH protocol (cyclophosphamide and prednisone doses plus etoposide, vincristine and doxorubicin) (10). The HIV serology of our case was negative. All possible complications associated with chemotherapy were explained to our patient, including gastric perforation and vascular rupture. Accepting the possible risks, the patient was started on the da-epoch kt protocol. The treatment resulted in clinical improvement, hemodynamic stability, reduction and disappearance of the need for erythroid trasnfusion and improvement in laboratory values. The patient, whose general condition also improved and no longer needed intensive care, was transferred to the hematology service.

Conclusion

Aggressive non-hodgkin lymphoma may be among the etiologies of upper gastrointestinal bleeding. In cases where endoscopic and surgical methods are inadequate, chemotherapy may be a life-saving option considering the possible risks.

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