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Case Report

Journal of Emergency Medicine Case Reports

From Symptom to Diagnosis: A Case of Organophosphate Poisoning in an Unconscious Paediatric Patient

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Abstract

Organophosphates are used as pesticides in agriculture and poisoning is widespread all over the world. Organophosphates inhibit the enzyme cholinesterase which breaks down acetylcholine in the nervous system. As a result of this inhibition, acetylcholine accumulates in the central nervous system, autonomic ganglia and nerves. This leads to cholinergic discharge. The clinical picture depends on cholinergic discharge and varies from nonspecific symptoms to coma. Mortality and morbidity are high. A 3.5-year-old male patient was brought to the emergency department unconscious. On initial evaluation, he had a Glasgow coma scale of 10 and concomitant myosis and bradycardia. Haemogram, blood electrolytes, blood glucose, brain tomography and electrocardiogram were normal. In his medical history, it was learnt that he had contact with organophosphate inhalation. Procedure and treatment were planned. The diagnosis of organophosphate poisoning is based on medical history and clinical findings and there is no specific laboratory test. Therefore, it is vital to make a differential diagnosis in patients presenting with coma and confusion. In our patient, the diagnosis of organophosphate poisoning was understood after a detailed medical history and we would like to emphasise the importance of medical history in the differential diagnosis.

Keywords: Bradycardia, child, poisioning, unconscious

Introduction

Organophosphates are generally used as pesticides in agricultural fields (1). Although organophosphates have many advantages in increasing agricultural yield, they are potentially dangerous for humans and other living organisms in the environment (1). Poisoning is observed as a result of suicidal use and accidental exposure. In these cases, mortality and morbidity rates are high (1,2).

Although it is difficult to determine the actual incidence of organophosphate poisoning due to the difficulties in data collection, it is estimated that pesticide poisoning causes 250.000 - 350.000 deaths per year globally (3). Organophosphate pesticides are recognised as the largest group of pesticides used globally for agriculture, animal husbandry and other commercial purposes (1,2).

The mortality rate in organophosphate poisoning is variable. It has been reported to be 3-25% on average depending on the substance ingested, amount, previous health status of the patient, factors related with respiratory support, intubation and weaning from the ventilator (2,3).

Starting appropriate treatment by making a differential diagnosis is very important for patient mortality (3,4). As in all paediatric patients, medical history and examination findings are very valuable in the diagnosis in patients in whom intoxication is considered. Here, organophosphate intoxication detected by detailed medical history interrogation in a paediatric patient who was brought to the emergency department in an unconscious state will be presented.

Case Report

A 3.5-year-old male patient who was known to be healthy was brought to the emergency department unconscious. It was learnt that he suddenly started to fall asleep while his hair was being cut at the barber just before his admission and then did not respond to sounds. The patient's medical history revealed that he had no fever, had eaten at home with his family 1 hour ago, and had no history of trauma or infection in the last 1 month. It was learnt that neuromotor development was compatible with his age, there was no history of convulsion, no consanguineous marriage, no known diabetes, cardiac or neurological disease in the family.

In the initial evaluation of the patient; peak heart rate was 70/min, SPO was 98%, respiratory rate was 28/min and blood pressure was 85/65 mm/Hg. On neurological

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examination, Glasgow Coma Scalewas 10 and bilateral myosis was found in the pupils. Lateralisation findings and nuchal rigidity were not detected. Other system examinations were evaluated as normal.

Blood tests revealed fingerstick blood glucose 95 mg/dl, creatinine 0.26 mg/dl, sodium: 138 potassium: 3.9 AST: 28 ALT: 11 Hb: 12,4 leucocyte: 9000 platelet: 478.000 and was evaluated as normal. No acidosis, alkalosis or lactate elevation was found in blood gas. Brain tomography was found to be normal. Electrocardiogram was compatible with sinus bradycardia.

Intoxication was considered because of pupillary miosis and sinus bradycardia accompanying loss of consciousness. When he was evaluated with toxidrome findings, his medical history of organophosphate poisoning was questioned again. It was learnt that the patient sat next to the beans at his father's workplace just before going to the barber, tried to put them in his mouth once, but his father prevented him from doing so, and he put them in his mouth and took them out immediately. It was learnt that the beans were sprayed with tebuconazole-containing organophospate pesticide.

During this period, the patient, who had spontaneous breathing and was monitored, regressed his tendency to sleep and his Glasgow Coma Score was evaluated as 15.

The patient was consulted to 114 National Poison Advisory Centre. It was recommended to follow-up for 24 hours in terms of cholinergic symptoms. Cholinesterase level could not be evaluated in our hospital. In the ward follow-up, no acute pathology was detected and the patient was discharged with healing. Outpatient follow-up is continued due to the risk of possible chronic effects.

Discussion

Acute organophosphate poisonings which occur as a result of suicide or accident are frequently observed in our country, especially in rural areas, as in the whole world (3).

The mechanism of action of organophosphates is through inhibition of cholinesterase enzyme in the nervous system (3,4,5). Acetylcholine is the main neurotransmitter in the autonomic and somatic nervous system (4). Cholinesterase hydrolyses acetylcholine to choline and acetic acid. Inhibition of cholinesterase by organophosphate absorption leads to acetylcholine accumulation and excessive stimulation. This leads to cholinergic discharge in the central nervous system, autonomic ganglia, parasympathetic and sympathetic nerves. Symptoms caused by cholinergic discharge in the central nervous system include anxiety, insomnia, emotional lability, tremor, headache, dizziness, mental confusion, delirium and hallucinations. With nicotinic (sympathomimetic) action, muscle fasciculations, muscle weakness, hypertension and tachycardia may occur. The muscarinic effect causes smooth muscle contractions in all organs (e.g. lungs, gastrointestinal tract, eyes, bladder, secretory glands). This may result in salivation, lacrimation, sweating, myosis, urinary incontinence, and bradyarrhythmia, or it may cause weakening of sinus node and AV node conduction resulting in ventricular dysrhythmias. Symptoms and signs develop depending on the balance between nicotinic and muscarinic receptors (4,5). In our patient, myosis due to muscarinic effect, bradycardia and loss of consciousness due to central nervous system cholinergic discharge were observed.

The clinical evoliotion depends on the agents used, the amount of absorption and the mode of exposure (3,4,5). Onset of symptoms is very rapid with inhalation and slowest with transdermal absorption (3,4,5). In our patient, absorption occurred both by inhalation and gastrointestinal route and symptoms appeared in approximately 1 hour.

Most patients start to show symptoms within 8-24 hours after ingestion, depending on the amount ingested (3,4,5). Patients should be monitored in terms of coma, seizure, respiratory failure, excessive secretions or severe bronchospasm (5,6). The need for endotracheal intubation may arise due to bronchospasm (5,6).

The treatment process after poisoning consists of decontamination, prevention of absorption, general support and intensive respiratory support (5,6). Atropine forms the basis of treatment in organophosphate poisoning because of its anticholinergic properties. Atropine was not administered in our patient because bradycardia did not persist. Antidotes are administered according to the degree of intoxication. As an antidote, pralidoxime (PAM) is a cholinesterase reactivator which helps to reverse bronchospasm and muscle fasciculation by accelerating the restoration of enzyme activity in the neuromuscular junction (5,6). No antidote was administered in our patient because of the absence of bronchospasm findings.

Exposure to organophosphate may cause free radical production and consequently lipid peroxidation, which may lead to DNA damage (7,8). For children, organophosphate exposure has been associated with developmental problems such as decreased IQ level and attention deficit hyperactivity disorder (7,8). In a study by Chbara et al. the risk of organophosphate-induced delayed neurotoxicity was reported (9). Delayed neurotoxicity is a rare condition that may occur weeks after the first exposure and is a distal axonopathy affecting both central and peripheral nervous system. It manifests as motor-sensory polyneuropathy in the peripheral nervous system and myelopathic symptoms in the central nervous system (9). In organophosphate poisoning, management of chronic findings as well as acute findings is important in terms of morbidity (9). Our patient was followed up in terms of possible chronic symptoms.

The differential diagnoses of intoxication in unconscious paediatric patients include endocrine and metabolic causes, trauma, seizure, vascular pathologies, cardiac diseases, infections and psychiatric convulsions (10).

Organophosphate intoxication must be kept in mind in

the differential diagnosis in patients who present with altered consciousness and in whom anamnesis cannot be obtained, if there are findings related with nicotinic and muscarinic effects.

Conclusion

Organophosphate poisoning is a public health problem with high mortality and morbidity. It is vital to make a differential diagnosis and to provide appropriate intervention in the early period. Clinically specific findings may not always be observed in patients. Especially in patients who are brought to the emergency department unconscious as in this case, it may be more difficult to make a differential diagnosis. For this reason, diagnostic medical history and examination findings are very valuable in patients with intoxication as in all paediatric patients. In our patient, organophosphate poisoning was determined by taking a detailed medical history.

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Case Report

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A Giant Bilateral Hydronephrosis Case Saved Despite the Patient's Negligence

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Abstract

Hydronephrosis due to nephrolithiasis is common, but giant hydronephrosis is rare in adults. Most of the cases previously identified in the literature are unilateral. In the study, we presented bilateral giant hydronephrosis. A 40-year-old male patient stated that he had stones in both kidneys and took at least 2-3 tablets of various painkillers daily but did not come to urology check-ups due to the pandemic. The examinations detected bilateral giant hydronephrosis, and the parenchyma was severely thinned. The patient underwent bilateral ureterorenoscopic lithotripsy and inserted a double J catheter. Seven liters of fluid were drained from the right and 8 liters from the left. It was determined that the size of both kidneys of the patient, who was followed for two years, decreased by half. Bilateral giant hydronephrosis cases are rarely seen in the literature. With the findings in this study, we tried to show that damaged both kidneys are not life-threatening, and the COVID-19 pandemic has affected our health directly and indirectly.

Keywords: Adult, COVID-19, giant, hydronephrosis

Introduction

Giant hydronephrosis is defined as a hydronephrotic kidney containing more than 1 liter of urine. Palpable mass and abdominal distension findings on examination may be mistakenly interpreted as a mass and confused with polycystic kidney on imaging.

Hydronephrosis is generally unilateral and frequently detected prenatally in the pediatric patient group (1). In adults, hydronephrosis mainly occurs due to ureteral stones, pregnancy, pelvic ureteric junction stenosis and obstruction in the bladder tract (2, 3).

Renal calculus causes 10-15% of hydronephrosis in adult patients (4). In post-mortem studies, the incidence of hydronephrosis in adults was found to be 3.1%, but the literature remains unclear about the incidence of bilateral hydronephrosis in adults (5).

Hydronephrosis due to nephrolithiasis is common, but giant hydronephrosis is a rare entity in adults. Most of the cases previously identified in the literature are unilateral. This study will try to emphasize a bilateral giant hydronephrosis case and show that it heals with appropriate and rapid treatment without posing a life-threatening risk.

Case Report

A 40-year-old male patient applied to the emergency room with complaints of increasing abdominal pain, nausea, vomiting, burning sensation in urination, and frequent urination for 1 week. He had a familial history of hypertension and is being treated due to hypertension. He has been using lercanidipine hydrochloride. He smoked 1 pack/day of cigarettes and did not report chronic alcohol use. He stated that he was diagnosed with bilateral ureteral stones a year ago, and Extracorporeal Shock Wave Lithotripsy (ESWL) treatment was recommended. Still, he did not come for a urology treatment due to the COVID-19 pandemic and took at least 2-3 tablets of various analgesics daily. Lung examination was normal, and there was tenderness in both the abdomen's upper quadrants and costovertebral angles. Also, there was a palpable mass in both costovertebral angles. There was no pretibial edema. Blood pressure was 120/70 mmHg, heart rate was 100/minute, and electrocardiography showed normal sinus rhythm (no T peak). In blood tests taken, leukocyte count was 10.7 109/L, urea 424 mg/dl, creatinine 12.7 mg/dl, C-Reactive protein (C-RP) 1.4 mg/dl, glomerular filtration rate (GFR) 4 ml/minute, pH 7.17,

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pCO2 27 mmHg, HCO3 9.6 mg/dl, base deficit -17 mmol/L was observed. Normal yellow urine came from the urinary catheter. In the urine test, pH and density were normal, and 12% leukocytes and 10% erythrocytes were detected.

Urinary ultrasound (USG) was performed, and bilateral cystic hydronephrosis was detected. Abdominal computer tomography (CT) showed severe thinning in kidney parenchyma and bilateral giant hydronephrosis (Rad-V Radiology hydronephrosis grading system). Kidney dimensions (Length/AP thickness/Width) were measured as 29x24x18.5 cm on the right and 31x24x12.5 cm on the left. A 17x8 mm diameter stone was detected in the right proximal ureter, and a 23x9 mm diameter stone was detected in the left proximal ureter (Figure-1). The diameter of the right proximal ureter was measured as 16 mm, and the diameter of the left proximal ureter was measured as 32 mm. The ureters distal to the stone were observed within normal limits.

Considering the patient's acute renal failure (ARF), internal medicine and urology consultation was performed. The internal medicine physician did not consider emergency dialysis because there was urine output. The urologist planned an operation due to bilateral ureteral stones.

The patient underwent bilateral ureterorenoscopic lithotripsy and was inserted into a double J catheter. Seven liters of liquid were drained from the right and 8 liters from the left kidney. In blood tests taken 10 days after the operation, leukocyte 8.47 109/L, C-RP 0.96 mg/dl, urea 58 mg/dl, creatinine 2.29 mg/dl, C-RP 0.92 mg/dl, GFR 33 ml/min, pH 7.33, pCO2 27 mmHg, HCO3 14.3 mg/dl, base deficit -10.1 mmol/L. The patient was discharged with the recommendation to come 1 month later to the urology outpatient clinic.

The control CT scan taken 1 month later showed that the kidney dimensions (Length/AP thickness/Width) were 17x15x9 cm on the right and 20.5x17x9.5 cm on the left (Figure-2).

In the follow-up abdominal magnetic resonance imaging (MRI) obtained twenty months later, kidney dimensions (Length/AP thickness/Width) were seen to be 12.6x12x7.5 cm on the right and 15x11.7x6.4 cm on the left (Figure-3).

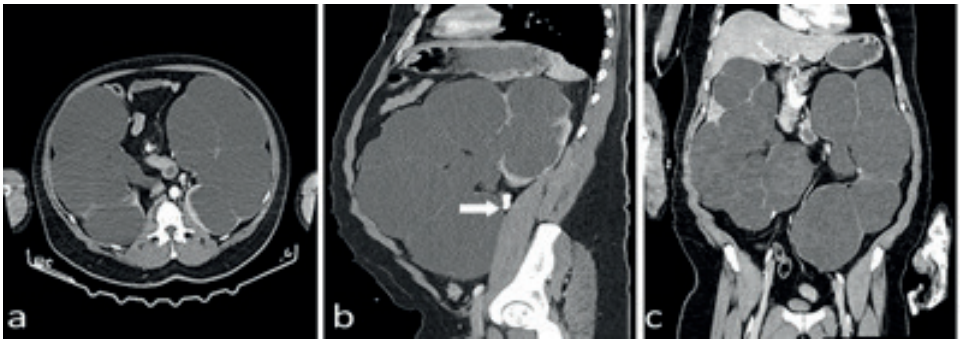

Figure 1 (a-b-c). Axial, sagittal, and coronal contrast-enhanced abdominal CT images. Bilateral giant hydronephrosis, stone in the left proximal ureter (b: white arrow)

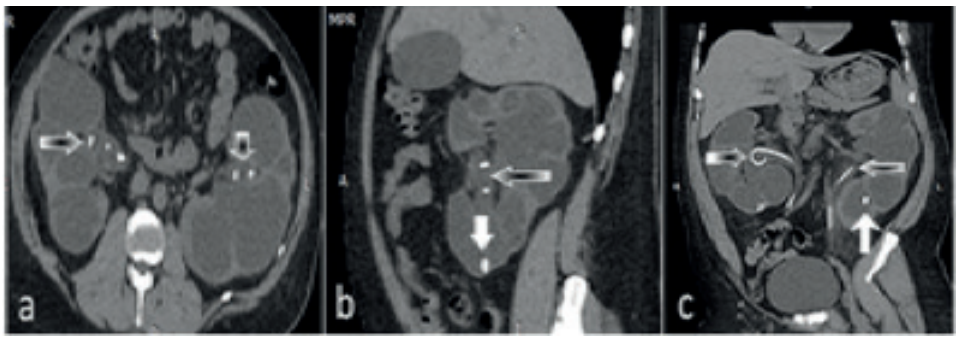

Figure 2 (a-b-c). Axial, sagittal, and coronal non-contrast abdominal CT images. Bilateral ureteric stents on control images in 1 month after admission to the hospital (a-b-c: hollow arrows indicate double J catheter, b-c: White arrows indicate renal calculus)

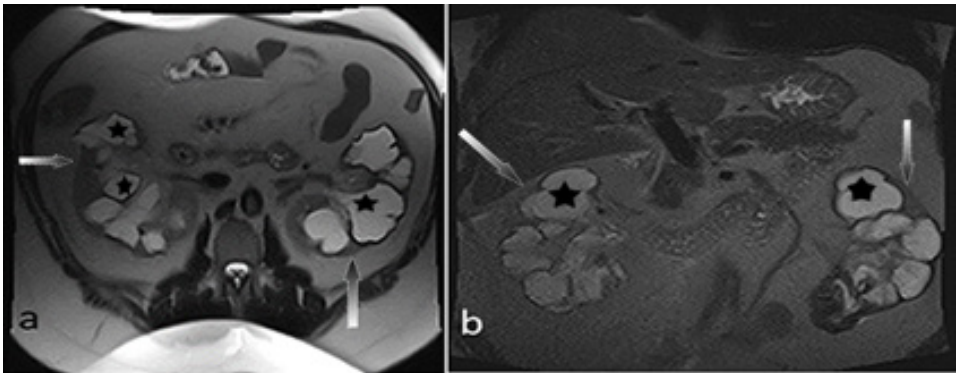

Figure 3 (a-b). Control images 20 months after admission to the hospital. Axial and coronal T2W MRI images show dilated hyperintense calyces (black stars) and markedly thinned hypointense renal cortex (dotted black/white arrows) bilaterally.

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Discussion

Cases of giant hydronephrosis are rarely seen in the literature. There is usually an obstructive cause, such as a stone or an intra-abdominal mass. In the case report by Gölcük et al., giant hydronephrosis was observed on the right side, and the causative factor was determined to be nephrolithiasis (6). Our patient also developed hydronephrosis after stone-related obstruction, but unlike the literature, bilateral giant hydronephrosis was detected.

It is difficult to diagnose giant hydronephrosis. Because it can be confused with abdominal mass or fluid. Since our patient had a known history of bilateral nephrolithiasis and chronic flank pain, we focused on the kidneys. That's why diagnosis and treatment could be done quickly.

Nephrostomy, pyeloplasty, and calicourethorostomy can be applied to treat hydronephrosis. In cases of advanced hydronephrosis, nephrectomy may be the only solution if kidney functions are impaired. In the case of Hassen et al., a nephrectomy was performed on the giant left kidney due to the structural defect in the retroperitoneum and massive pelvic dilatation (7). In their study by Sallami et al., nephrectomy was performed in patients who did not respond to kidney-protective treatment (8). In our case, since the blood tests and clinical findings improved after the lithotripsy operation, the patient did not need additional surgery. This is a rare case in the literature because both kidneys were spared.

Although it may be difficult to identify hydronephrosis clinically, it can be diagnosed and monitored easily with imaging studies.In our patient, the known history of nephrolithiasis and the developing symptoms were guiding.

Conclusion

Patients with nephrolithiasis frequently visit the emergency room, especially with complaints of pain. In this way, urology and radiology follow-up can be performed, but our patient had his treatment (long-term analgesic use) at home due to fear ofCOVID-19 infection. In addition to the deterioration of renal functions due to the existing obstruction and uncontrolled analgesic use and abuse, the development of renal failure was inevitable. Cases of bilateral giant hydronephrosis are rarely seen in the literature. With the findings in this study, we tried to show that damage to both kidneys is reversible, and the COVID-19 pandemic has affected our health directly and indirectly.

Consent was obtained from the patient for this study.

The authors declared no conflict of interest.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Emergency Diagnosis: Pancreatic Ductal Rupture

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Abstract

Chronic pancreatitis is a condition that results from episodes of acute pancreatitis. It is difficult to diagnose chronic pancreatitis in the early stages and to recognize complications. Recognizing duct rupture, one of the complications of chronic pancreatitis, is important for initiating treatment. A 75-year-old woman presented to the emergency department with abdominal pain. Amylase and lipase levels were 65 u/L and 75 u/L, respectively. Computed tomography performed in the emergency department revealed ductal rupture and the patient was hospitalized in the gastroenterology service. Treatment included endoscopic examination followed by pancreatic rest, hydration and proton pump inhibitors. Pancreatic duct rupture, which is rarely encountered in the emergency department, should be considered as an alternative diagnosis in patients presenting with abdominal pain and a previous pancreatitis attack.

Keywords: Chronic pancreatitis, complication, ductal rupture

Introduction

Chronic pancreatitis is an inflammatory disease that causes permanent structural changes in the pancreas and can lead to impaired exocrine and endocrine functions. Chronic pancreatitis can result from attacks of acute pancreatitis due to any reason (1).Pseudocysts, biliary obstruction, pancreatic diabetes, vascular complications may occur as a result of chronic pancreatitis. Pseudocysts, one of the complications of chronic pancreatitis develop in about 10 percent of patients with chronic pancreatitis. They may be triggered by an acute exacerbation of pancreatitis or occur as a result of ductal rupture.

Pancreatic duct (DP) rupture and leakage of pancreatic fluid into the abdomen can occur as a complication of acute or chronic pancreatitis (2). The other causes are pancreatic malignancy, abdominal surgery or abdominal trauma (3). DP rupture may occur in the main pancreatic duct or in one of its small branches. The clinical consequences of a DP rupture may depend on the etiology, location, and extent of the rupture. Clinical results occur depending on the rate of pancreatic fluid secretion, the location of the leak relative to anatomical tissue regions, the systemic inflammatory response ability to control the leakage, and the obstruction of the DP with stones or strictures. A small leak of pancreatic fluid from one of the lateral branches may resolve on its own. A persistent leakage from the main DP rupture may present with pseudocyst formation or be complicated by external pancreatic fistulas or internal fistula formation causing pleural effusion or ascites(4). Early recognition of complications is important for early initiation of treatment.

The diagnosis of ductal rupture is rarely made in the emergency department because pancreatic enzymes are not severely elevated or do not present with severe clinical signs. therefore, suspicion will help in the path to diagnosis. Here, we aimed to discuss our patient who was diagnosed with pancreatic ductal rupture, a rare complication of chronic pancreatitis, in the emergency department.

Case Report

A 75-year-old woman presented to the emergency department with nausea, vomiting and abdominal pain. Her medical history included abdominal pain in the epigastric region for the past 15 days, aggravated after meals, and accompanied by occasional nausea and vomiting. Her medical history included hypertension, diabetes mellitus, nasal squamous cell carcinoma, and previous ischemic stroke. She was taking metformin, ramipril, clopidogrel, and escitalopram. On physical examination, general condition

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was good, consciousness was clear, oriented and coherent. Vital signs were arterial blood pressure: 130/80 mm/hg, pulse rate: 98 beats/minute, fever was 36.7°C. There was no incisional scar on abdominal examination. Bowel sounds were normoactive, tenderness and rebound were present in the epigastric region on palpation. When the patient's previous admissions were analyzed, it was found that she had pancreatitis approximately 1 month and 6 months ago. The patient had no subsequent hospital admissions. Biochemical tests showed amylase 65 u/L and lipase 75 u/L. C-reactive protein was 226 mg/L, white blood cell count was 12100 µL, and other parameters including liver function tests were within normal limits. Contrast-enhanced abdominal computed tomography (CCT) was performed to rule out other causes of acute abdomen due to severe pain, epigastric rebound tenderness, borderline elevated pancreatic enzymes, and elevated acute phase reactants. On CCT, the gallbladder was within normal limits, the pancreatic duct was 5 mm in size, a focal area of defect in the anterior part of the pancreatic corpus and a diffuse heterogeneous inflammatory appearance in the mesenteric adipose tissue in the adjacent peripancreatic area were observed. All these findings were consistent with acute pancreatitis and ductal rupture in the background of chronic pancreatitis (Figure-1). The patient was consulted to the gastroenterology and general surgery clinics. Urgent surgical intervention was not considered and the patient was admitted to the gastroenterology clinic for furthermore evaluation. Intravenous (IV) fluid therapy, proton pump inhibitor, and ceftriaxone were administered during hospitalization.Magnetic resonance cholangiopancreatography (MRCP) was planned in the gastroenterology service but could not be performed because the patient had claustrophobia. The patient followed a stable clinical course and was discharged at the end of treatment with a follow-up visit after 3 months. CCT and Endoscopic retrograde cholangiopancreatography (ERCP) were planned after 3 months for control. The control CCT scan showed a 2.5 cm diameter lobule contoured cystic formation at the level of the pancreatic head (Figure-2). In ERCP, the papillary head was not observed in normal localization and the duct could not be entered. The patient was referred to an advanced center for further investigation.The patient died pending further investigation.

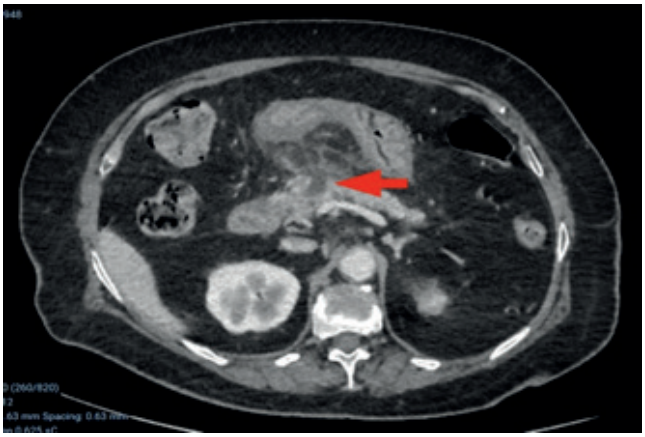

Figure 1. Anteriorly ruptured pancreatic duct

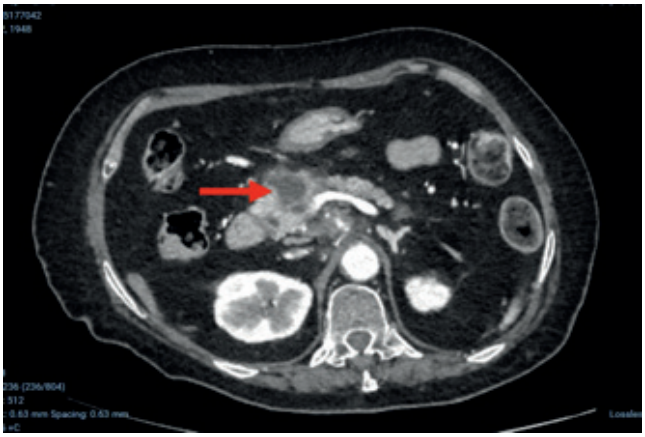

Figure 2. Cystic formation in the head of the pancreas

Discussion

Chronic pancreatitis is one of the causes of abdominal pain diagnosed in the emergency department (5). Chronic pancreatitis is most commonly seen in patients with frequent pancreatitis attacks. Acute pancreatitis can be considered as a status, chronic pancreatitis is the pathological ongoing response process to pancreatic damage (6,7). Acute and chronic pancreatitis are not two completely separate conditions. Can be thought of as a continuum and two parts of the same disease spectrum. Our patient also had chronic pancreatitis due to an acute pancreatitis attack.

Pancreatic fluid from a DP rupture may spread to the retroperitoneum, mediastinum, or if confined to the small sac, it is then surrounded by a well-formatted, non-epithelialized wall and becomes a pseudocyst over a period of 4 to 6 weeks. Pancreatic pseudocysts are the maturation of a phlegmonous inflammatory event and consist of an accumulation of pancreatic secretions and secondary inflammatory products resulting from pancreatic duct rupture (7). Pancreatic pseudocysts are seen in 10-20% of patients with acute pancreatitis and 20-60% of patients with chronic pancreatitis (8). Many of the clinical features of chronic pancreatitis may take time to develop and may not be present in the early stages of the disease. Chronic pancreatitis may be easier to diagnose when end-stage features develop. The clinical challenge is to make the correct diagnosis in the early cases of chronic pancreatitis, when interventions aimed at preventing disease progression may be effective (8). In our patient with a diagnosis of ductal rupture, no pseudocyst was detected at this stage.However later cystic formation developed at the site of rupture.

Internal pancreatic fistulas formed as a result of DP rupture may occasionally fistulate with other areas such as the pericardium or organs such as the stomach, bronchus and large or small intestine. Pancreatic fluid may also fistulate outward toward the skin surface, creating an external pancreatic fistula. Pancreatic ascites may develop due to anterior DP rupture and leakage of pancreatic fluid into the peritoneal cavity. Posterior DP rupture may cause pancreatic fluid to leak into the pleural space through the esophageal or aortic hiatus and cause pleural effusion. (4). In our patient, the fluid spread anteriorly and peritoneal contamination occurred due to fluid leakage around the pancreas.

ERCP has the highest accuracy in diagnosing pancreatic duct rupture because it provides detailed images of the pancreatic duct. It is also useful in determining the exact location (head, neck, trunk, or tail) and size of the rupture. However, ERCP is no longer used in the diagnosis of chronic pancreatitis due to the availability of alternative imaging methods and the risk of complications. In early disease, changes in the duct may be minimal and the diagnosis may not be obvious. Recent advances in imaging, such as multislice spiral contrast-enhanced computed tomography (CCT) and magnetic resonance imaging (MRI), may aid in the noninvasive detection of ductal rupture. CCT has been shown to be a useful technique for visualizing the pancreatic parenchyma and identifying and localizing pancreatic fluid collections (4). The site of fluid collections seen on CCT may be indicative of the location of DP rupture. In our patient, CCT performed in the emergency department helped us to diagnose the rupture.

Initial management of DP rupture or fistula complications (pleural effusions or pancreatic ascites) includes prolonged pancreatic rest and endoscopic placement of stents in the pancreatic duct (9). Conservative treatment includes recommendations such as adequate drainage of pancreatic secretions and optimal nutritional support during the recovery period to prevent or control sepsis. If the patient becomes septic, aggressive treatment and correction of electrolyte disturbances are required. Total parenteral nutrition therapy can be applied to rest the pancreas and reduce exocrine pancreatic secretions. Treatment may include total parenteral nutrition, jejunal feeding, or medications such as anticholinergics, carbonic anhydrase inhibitors, calcitonin, glucagon, octreotide or somatostatin(4). Our patient was treated conservatively with proton pump inhibitors, antibiotherapy, and intravenous fluid therapy.

Conclusion

The diagnosis of ruptured ductus pancreaticus is a rare entity in the emergency department. Early diagnosis may allow treatment before the patient's clinical condition becomes complicated. Epigastric pain and low level of enzyme elevation should be a warning in this regard. We thought that our case may be noteworthy for clinicians working in the emergency department because it allows early diagnosis and treatment.

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Case Report

Journal of Emergency Medicine Case Reports

A Rare Case of Occupational Aniline Poisoning: Management in a Limited-Facility Setting

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Abstract

Cyanosis along with altered mental should bring all the possible ethiologies into her mind in a minimum of time, based on the trauma, medical, environmental, and occupational history of the patient. Poisoning with methemoglobinemia inducing agents is one of the most important differentials especially when a history of occupational exposure to these agents is present.The patient, a 41-year-old healthy woman and engineer, experienced symptoms after exposure to aniline leading to dizziness and weakness, prompting a visit to the emergency department with cyanosis and low oxygen saturation. Initial vital signs showed decreased oxygen levels and elevated respiratory rate with normal ABG values despite ongoing cyanosis. Patient required intubation, mechanical ventilation, and was diagnosed with ARDS based on chest X-ray findings. Management included IV diuretics, 100% oxygen, and antioxidant treatment in the ICU. Despite treatment, oxygen saturation remained at 88% on the first day.This case is a reminder of the importance of taking occupational history and management of aniline toxicity in a setting where methylene blue, the drug of choice for aniline toxicity, is not available in the drug stock.

Keywords: Aniline, aniline poisoning, emergency medicine, occupational medicine, toxicology

Introduction

Aniline, introduced to the chemical industry in the late 1800s (1), is primarily used for industrial manufacturing of dyes, drugs, rubber accelerators, and other chemicals (2). It can enter the body through inhalation, ingestion, or dermal absorption (3). Early toxic effects include methemoglobin (MetHb) formation and damage to red blood cells (RBCs) (4). Phenylhydroxylamine, an active metabolite of aniline, catalyzes MetHb formation in vivo (5). Additionally, rat models show splenic toxicity related to nitrated proteins found only in aniline-exposed rats (6).

Aniline exposure can lead to either acute or chronic poisoning. Acute poisoning is characterized by bluish lips, Heinz's bodies in the blood, and para-aminophenol in urine. Chronic poisoning symptoms include headache, unsteadiness, memory loss, sleeplessness, increased knee-jerk reflex, and finger tremors (7).

Standard therapies for acute aniline poisoning include intravenous infusion of methylene blue (MB), glucose, ascorbic acid, and exchange transfusions, but responses vary among patients. Lubash reported successful treatment with hemodialysis in one case(8). This case report describes a patient with unintentionalpoisoning of aniline, who admitted with the symptoms and signs of acute respiratory distress (ARDS).

Case Report

Our patient was a 41-year-old healthy woman, an engineer at a car liquid manufacturing company. She developed symptoms after six hours of unprotected cutaneous contact and respiratory aspiration of pure aniline in the laboratory. Her first symptom was dizziness while driving from her workplace to home. However, within two hours, her symptoms progressed to weakness and drowsiness, prompting her to seek help. She was subsequently brought to the emergency department (ED) by EMS, presenting with a cyanotic appearance despite receiving nasal oxygen, with a Glasgow Coma Scale (GCS) of 15/15.

On arrival, her vital signs demonstrated a significantly decreased oxygen saturation of 60% and an elevated respiratory rate of 22/min, while other vital signs were within normal ranges. Notably, she exhibited pronounced peripheral cyanosis, characterized by a bluish discoloration of the lips, fingertips, and toes. This cyanosis was indicative of severe hypoxemia and poor peripheral perfusion. Despite being on supplemental oxygen, the cyanosis persisted, suggesting that the hypoxemia was due to a failure of oxygen exchange at the alveolar level, rather than simply inadequate oxygen delivery.

The first arterial blood gas (ABG) sampling showed normal values, but the persistent cyanosis and low oxygen

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saturation despite oxygen supplementation through a reservoir mask were concerning. This raised suspicion for an underlying condition impairing gas exchange. The patient was then intubated and placed on mechanical ventilation (MV). Urinary catheterization revealed the excretion of brownish urine, suggesting a hemolytic event possibly due to aniline toxicity.

A chest X-ray revealed blunting of the left costophrenic angle, consistent with early signs of pulmonary edema. This, coupled with the patient's hypoxemia and the clinical context, led to the diagnosis of Acute Respiratory Distress Syndrome (ARDS). In this case, ARDS has been characterized by acute onset of respiratory failure due to non-cardiogenic pulmonary edema, marked by bilateral infiltrates on chest imaging, likely developed secondary to chemical pneumonitis and direct pulmonary toxicity from the inhalation of aniline.

The patient was transferred to the intensive care unit (ICU) for further management. Despite being on a ventilator with 100% FiO2, her oxygen saturation remained critically low at 88% on the first day of admission, indicative of severe ARDS. Management continued with intravenous (IV) infusion of loop diuretics, aiming to reduce pulmonary edema, and the ventilator settings were adjusted to optimize oxygenation and reduce ventilator-induced lung injury.

In addition, antioxidant treatment, including IV infusion of vitamin C (2 g daily) and N-acetylcysteine (NAC) (7 g every 8 hours), was initiated upon ICU transfer to mitigate oxidative stress and potential lung injury caused by the chemical exposure. The IV infusion of furosemide continued at a dosage of 2 mg/hr to manage fluid balance and minimize pulmonary edema.

Table 1: Laboratory test of first day of admission

Test	Result (reference values)
Complete blood count	
Red blood cells	3.51 cells/mcL (4.10-5.10)
Hemoglobin	9.4 g/dl (12.0-16.0)
Hematocrit	29.3% (39-50)
Platelets	245000 cells/mcL (150-450)
White blood cells	6400 cells/mcL , N: 70%, L:30% (4000-11000)
Arterial blood gas	
PH	7.44 (7.37-7.44)
pO_2	247.3 mmHg (80-100)
pCO_2	38.6 mmHg (35-45)
HCO_3	26.8 mmol/L (22-26)
Coagulation	
PT patient	13 sec
PT control	13 sec
PT activity	100% (70-100)
INR	1
PTT	30 sec(30-40)
Blood biochemistry	
Fasting blood glucose	82 mg/dl (70-115)
Blood urea Nitrogen	12 mmol/L (7-24)
Creatinine	0.9 mg/dl (0.6-1.3)
Aspartateaminotrans-	17 U/L (up to 31)
ferase Alanine amino-	11 U/L (up to 40)
transferase	110 U/L (80-306)
Alkaline phosphatase	142 mmol/L (135-145)
Sodium	3.8 mmol/L (3.5-5.1)
Potassium	7.9 mg/dl (8.5-10.5)
Calcium	4.3 mg/dl (2.6-4.5)
Phosphorus	

Discussion

Various conditions, including acute diarrhea, consumption of high-nitrate water and food, use of specific medications like topical anesthetics, silver nitrate, sulfonamides, phenacetine, and sodium valproate, or exposure to aniline, coloring compounds, or cleaning solutions, can potentially trigger acquired MET (9).

Aniline and its derivatives were among the earliest substances linked to MET. Safety measurements in industrial settings have reduced acute aniline poisoning cases compared to earlier decades (10).However, in our case, due to occupational exposure, aniline poisoningoccurred through inhalation and skin contact in a factory setting without proper protective measures.

MetHb is formed by a complex series of oxidation reactions. For aniline, it is through their metabolites in the body after biotransformation of aniline to phenylhydroxylamine by hepatic mixed-function oxidase enzymes. This metabolite then contributes to the formation of MetHbs (11).MET refers to the presence of greater than the average physiological concentration of 1 to 2% MetHb in RBCs (12).

In the setting of MET, the patient's condition is usually better than one would expect from the severity of cyanosis, but the cyanosis is unresponsive to oxygen therapy. For example, in our case, the patient's awareness was intact upon admission to ED. However, she was unresponsive to oxygen therapy via nasal cannula, and finally, the managing team decided to intubate her (13).

Pulse oximetry is not reliable, and ABG, as a complementary workup, often reveals normal partial pressures of oxygen (pO2) and carbon dioxide (pCO2), a normally calculated hemoglobin oxygen saturation, an increased MetHb concentration, and possibly metabolic acidosis(13). This pattern was also compatible with the ABG results drawn from the patient in the ED.

Intravenous MB is the preferred treatment for toxic MET. Although no controlled trials have been conducted, clinical experience suggests that MB can enhance the conversion of MetHb to hemoglobin by sixfold (14). However,there are reports of hemolytic anemia (HA) following IV administration of MB for treatment of aniline toxicity. This is attributed to the fact that both aniline and methylene blue can cause oxidative stress, which wrecks RBCs (15). Moreover, based on some case reports, MB sometimes cannot solve the problem alone, so more measurements, such as hemodialysis and exchange transfusion (ET), are

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required. For example, in a 4.5-year-old girl who accidentally was poisoned by aniline when she was given *cough syrup*, the treatment was done through two consecutive IV doses of MB. The therapy first reducedMetHbbut, after two hours, the MetHb levels rose again, which led to an alteration in cognitive level. Immediately, the ET was started, leading to a significant drop in MetHb level and clinical improvement (16). Another case report also described a patient who did not respond to MB infusion after a suicidal oral intake of 80 ml of aniline. She developed an episode of seizure during her transference to another medical facility for hemodialysis after 22.5 mg of IV MB and the exchange of 1000 ml of blood. After that, hemodialysis with re-administration of IV MB led to improvement of MET, skin discoloration, and level of consciousness. Another dose of MB was administered after hemodialysis, and the patient gradually regained consciousness 24 hours later (8).

However, in our case, due to the unavailability of MB, the treatment was established on symptomatic treatment, including intubation and intravenous furosemideto address ARDS, and systemic antioxidants (vitamin C and NAC). Fortunately, these actions and closemonitoring of the patient in the ICU resulted in the patient's full recovery. The patient was also in good health during follow-up visits.

In two cases of occupational exposure, similar to the presented case, inhalation of aniline fumes has been reported as the cause of fatal MET. In both of them, HA became apparent after treatment with MB, mirrored in a dropped number of RBCs (15). In our case, the RBC count was relatively stable, and there was no HA induced by aniline.

There is also one case of occupational poisoning of aniline with low levels of MetHb in which conservative management was performed only with supplementary O_2 and close monitoring of VS and laboratory tests (17). However, in another case of exposure to large amounts of aniline during unloading aniline from bulk trucks the management was done with supplementary O2 and IV administration of MB in 4 consecutive doses up to six hours. He presented to ED acutely ill and with cyanoderma on his entire body. He also developed skin lesions of primary burns and contact dermatitis,and cornea inflammation caused by aniline.The dermal and corneal lesions were treated by topical ointments and eye drops, respectively (17).

In conclusion, anilinepoisoning, aniline poisoning, by various routes, results in MetHb, which varies in severity. Management includes a range of measures, from simple supplementary O2 to endotracheal intubation, administration of MB to ET, and hemodialysis. To our best of knowledge, the presented case is the first case in which the IV furosemidewas part of the management and exclusively the MET was managed without MB, but with systemic administration of antioxidants. It is crucial to promptly identify the underlying cause of acquired MET to restore normal tissue oxygenation and metabolism, prevent long-term complications, and minimize the risk of recurrent episodes.

Conclusion

Aniline is an aromatic agent that has historically been used in chemical industries. Most of the reported incidences of aniline poisoning are between 1900-2000, which are related to industrial exposures. Our case was a young woman who suffered acute symptoms of aniline poisoning. Timely admission and therapeutical interventions avoided further damage that can be tissue ischemia due to prolonged MET. Aniline poisoning is a rare poisoning etiology, which often can be conferred from a thorough occupational history.

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Case Report

Journal of Emergency Medicine Case Reports

Morestin Syndrome in the Emergency Department: A Case Report

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Abstract

Morestin Syndrome is a rare condition that occurs as a result of sudden compressive trauma to the thoracoabdominal region. It presents with cervicofacial petechial rash, edema, subconjunctival hemorrhage, and varying degrees of neurological symptoms. It can also be seen in non-traumatic situations such as severe coughing, asthma attacks, seizures, or difficult childbirth.

We present a case of a 5-year-old trauma patient referred to us from an external center. The patient had cervicofacial petechial rash, subconjunctival hemorrhage, and altered consciousness, but no serious organ injury was detected. The laboratory tests showed elevated liver function tests. Imaging revealed a minimal pneumothorax that did not require intervention and a proximal right humerus fracture. The patient's consciousness improved during follow-up in the emergency critical care area. After the emergency diagnostic and treatment process, the patient was admitted to the pediatric surgery ward for observation and was discharged in good health after 4 days.

It is important for emergency physicians to recognize this rare case early. It provides insight into the severity of the trauma and guides further investigation. Although there is no specific treatment, head elevation and oxygen support can increase venous return and accelerate the recovery process.

Keywords: Compressive trauma, morestin syndrome, traumatic asphyxia

Introduction

Traumatic asphyxia is a rare condition referred to in the literature as Perthes Syndrome, Olivier Syndrome, Acute Thoracic Compression Syndrome, or Morestin Syndrome (1). It can also develop in non-traumatic situations such as difficult childbirth, epileptic seizures, severe coughing, or asthma attacks. Although a frequency of 1/18,500 traumatic cases has been reported in adult patients, the true pediatric incidence is unknown (2). This condition, typically seen in pediatric patients, occurs as a result of sudden compressive trauma to the thoracic and/or abdominal region. Clinical findings develop due to the direct and indirect effects of the trauma, along with the sudden and high-pressure reversal of venous drainage (3). Although characterized by facial edema, cyanosis, petechial rash, and subconjunctival hemorrhage, the clinical presentation may vary depending on the severity of the trauma and the duration of exposure (4). It is often accompanied by injuries such as pneumothorax, hemothorax, or pulmonary contusion (5). In critical cases, the presence of vital organ injury is the main factor that increases mortality.

Case Report

A 5-year-old girl with no known medical history was referred to us from a regional hospital following an out-of-vehicle traffic accident. The history revealed that she had been trapped between a slowly reversing vehicle and the curb for approximately 1 minute. Upon admission, her general condition was good, her consciousness was lethargic, and her Glasgow Coma Scale (GCS) score was 12 (E:3 V:4 M:5). Her heart rate was 108 beats per minute, temperature 36.9°C, blood pressure 110/67 mmHg, respiratory rate 18 breaths per minute, and oxygen saturation 95%. On inspection, she had widespread petechial rash on her face and neck, dermabrasion on her chin and nose, and bilateral subconjunctival hemorrhage (Figures 1-2). Thoracic examination was unremarkable, with no tenderness on palpation. In the abdomen, there was a 3 cm superficial laceration and a 5x10 cm abrasion in the right inguinal region, without tenderness. Movement of the right shoulder was painful. A 2 cm superficial laceration was observed on the right knee, but joint movements were normal. Laboratory findings showed a white blood cell

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count of 18,900/mm³, hemoglobin level of 11 g/dL, and platelet count of 383,000/mm³. Coagulation parameters were normal. Lactate dehydrogenase (LDH: 2197 U/L), aspartate aminotransferase (AST: 1251 U/L), and alanine aminotransferase (ALT: 798 U/L) levels were elevated.

Brain, thoracic, abdominal, and vertebral computed tomography (CT) scans, along with direct radiographs of the right shoulder and right knee, were requested for the patient. Thoracic CT revealed minimal pneumothorax in the right hemithorax, and no intervention was planned, with follow-up recommended using daily chest X-rays. A non-displaced fracture was observed in the right humerus at the greater tubercle, and no surgery was planned; a Velpeau bandage was recommended. Analgesics and prophylactic antibiotics were administered. During follow-up in the emergency critical care area, the patient's consciousness improved, there was no hemoglobin drop, and no free fluid was detected on abdominal ultrasound. The patient was admitted to the pediatric surgery ward for observation.

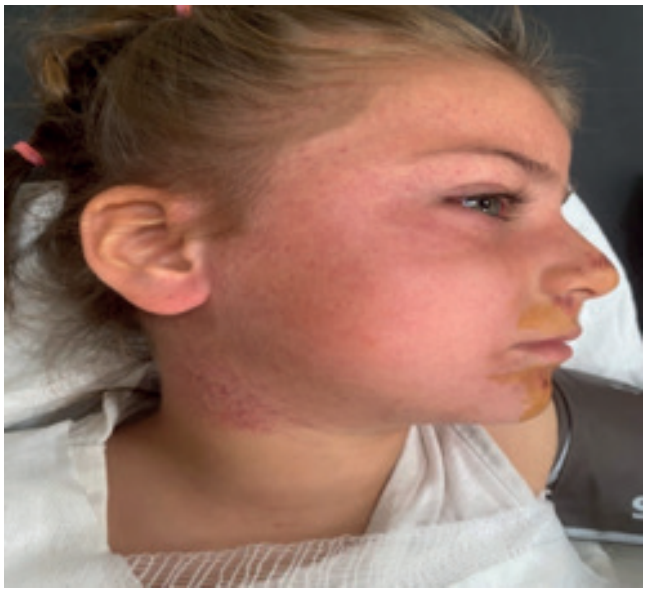

Figure 1. Cervicofacial diffuse petechial rash

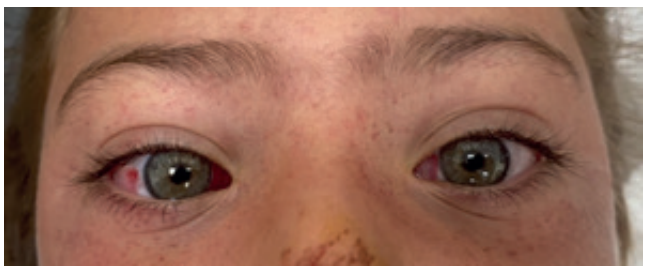

Figure 2. Bilateral subconjunctival hemorrhage
(Images were used with permission from the patient's parents)

Discussion

First noted by Olivier in 1837, this condition was later defined as traumatic asphyxia by Perthes in 1900, following an increasing number of case reports (6, 7). This phenomenon, observed during autopsies of individuals who died as a result of crushing in crowds, is more commonly encountered in the pediatric age group (8, 9). In all cases, there is a history of compressive trauma to the thoracoabdominal region. It is also seen, though more rarely, in non-traumatic situations such as severe crying, coughing, epileptic seizures, difficult childbirth, or asthma attacks. The pathophysiological mechanism involves glottic closure following deep inspiration due to a fear reaction, along with a sudden increase in thoracoabdominal pressure, leading to impaired venous drainage from the superior vena cava (2). The insufficient valvular support of the cervicofacial venous system, coupled with the Valsalva maneuver, explains why the lower venous system remains unaffected. The retrograde transmission of pressure results in venous stasis and capillary ruptures, which manifest as petechial rash, edema, cyanosis, and subconjunctival hemorrhage (3). The severity and duration of trauma closely influence the resulting lesions. Further worsening this condition is the development of neurological damage due to impaired cerebral blood flow and hypoxia (10). Altered consciousness, ranging from mild drowsiness to coma, may accompany these symptoms. Neurological recovery typically takes 1-2 days, but recovery from visual injuries may take longer.

This condition can provide insight into the severity of the trauma, as the valve system in the internal jugular vein can tolerate pressures up to 45 mmHg (11). While the external jugular vein drains the superficial tissues, the internal jugular vein is responsible for draining the deep neck tissues, airway, and cerebral circulation. This reality necessitates the investigation of patients with a dramatic appearance for potential critical organ injuries. Indeed, cases of vision loss due to retinal hemorrhage, hearing loss resulting from edema in the eustachian tubes, and hoarseness have been reported in the literature (12). Clinicians should anticipate difficult airway management due to edema.

In cases of traumatic asphyxia, injuries to the pulmonary, cardiac, or intra-abdominal organs are common due to the compressive force applied to the thoracoabdominal region (13). As in this case, minimal pneumothorax may be detected, though such injuries may not require surgical intervention. In the pediatric population, while the elastic structure of the thorax provides more resistance to pressure, the underdeveloped chest wall can transmit trauma energy to deeper tissues, leading to more severe internal organ damage (14). Additionally, neurological effects of traumatic asphyxia can range from brief consciousness impairment to coma. Some cases in the literature have resulted in permanent neurological damage, emphasizing the importance of long-term follow-up for patients exposed to significant trauma (10).Additionally, elevated liver function tests without imaging evidence suggest liver injury.

Treatment should begin with the immediate removal of the compression and trauma life support. Airway difficulty

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secondary to cervicofacial edema should be anticipated. Supplemental oxygen and head elevation may help increase venous drainage (15). The duration of trauma exposure and the severity of accompanying injuries are parameters that closely affect mortality (16). In our case, the short duration of exposure and the absence of serious organ injuries contributed to a favorable prognosis.

Conclusion

Early recognition of this uncommon condition and investigation of additional injuries closely affect the prognosis. The clinical course is quite good in patients without serious injuries.

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Case Report

Journal of Emergency Medicine Case Reports

Skin Rash Related to the Use of Wood Ash in Wound Healing, about A Case

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Abstract

The ash of wood plants is part of the ancestral remedies because of its physical and chemical properties. It has detergent, fertilizing, and antiseptic properties, which justifies its many uses. We report the case of a woman aged 50 years who presented with a generalized rash all over the body with painful macules a burning sensation and a fever. Symptoms appeared after taking wood ash from plants, for three days. The reason for use was the healing of Zona's wound that she contracted and treated before admission. The patient's general condition was preserved, and the drug treatment allowed a quick healing. Wood ash from plants with a high potassium hydroxyl content may be an irritant and partly explain the reaction observed. Traditional treatments should be used with great care to avoid adverse effects.

Keywords: Fever, skin rash, traditional medicine, wood ash

Introduction

Wood ash from plants has been used for centuries and has numerous applications. Ancestors used it for making soaps and toothpaste, to prevent gingivitis and aphthous ulcers (1). Due to the presence of sodium and potassium with an alkaline character, it was indicated for treating superficial wounds (2). It was also used by pre-Islamic Arab medicine as hemostatic (3). They also enabled local populations in Africa to make alkaline potash (in the form of crystals) and make preparations for healing (4). It was also noted that ash is added to sauces and cooking preparations to enhance the flavor of certain foods or reduce their acidity (4).

In daily practice, many adverse effects are reported following the use of medicinal plants or their derivatives. We describe a generalized rash with burning and fever following administration and local application of wood ash from plants.

Case Report

The case is a 50-year-old woman who weighs 53kg and measures 157 cm, and lives in a rural area and was admitted to the internal medicine department following the onset of a toxidermia and fever. She is immunocompromised who has been suffering from polymyositis for 4 years and contracted Zona before admission. The patient reported this adverse effect from oral (1 teaspoon in a glass of water) and local (powder applied) wood ashuse, for 4 days. She stated that she had not been taking any treatment associated with traditional preparation for 20 days. The reason for the use was the healing of a zonal wound; she claims that her wound healed, but at the end of the third day, there was an appearance of fever with pimples all over the body. The brief physical examination of admission to the internal medicine ward showed that the pulmonary, cardiovascular and neurological apparatus did not detect any abnormalities. Lymph node examination showed right and left axillary lymphadenopathy. The dermatological examination revealed a diffuse maculopapulic rash, hot to palpation, with no edema and oral, nasal or occular involvement. However, the purpuric erythema reached the blow and the extremities of the lower and upper limbs (Figure-1).Biological examination was correct except for three parameters which were high: C-reactive protein (CRP) which was 40.72g/L, white blood cells (GB): 28300/mm3 and sedimentation rate

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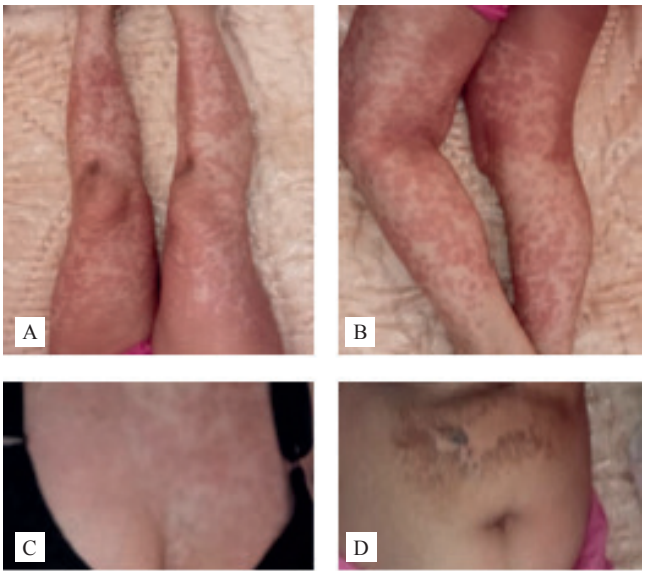

Figure 1. Appearance of the rash and macules on the lower limbs and bust.
A, lower limbs (face view), B, lower limbs (profile view), C, bust, D, Zona wound healing

(VS): 65nm (the first hour) and 130nm (the second hour). After drug management (corticosteroids and fusidic acid), his condition was improved without sequelae. Because of the lack of a direct cause and logical explanation for the occurrence of the rash in the patient, the adverse event was reported to the phyto vigilance unit. Oral and local use of ashes were heavily incriminated.

Discussion

Ash is the mineral residue obtained after the combustion of organic matter, most often a plant. They are complex in composition, varying according to the nature of the plant incinerated, the part (leaves, trunk, and branches), the season, and the soil or plant growth (5). Generally, ash contains calcium, silica, potassium, magnesium, and heavy metals. Their pH is alkaline (5).

Ash composition from the reported case could not be determined. Several elements can be incriminated; the wood of some plants may contain allergens and essences; it has been noted that benzo, naphto, and furano quinones are responsible for dermatitis (6) (7). A 20-year study at St John's Hospital for Skin Diseases, London, reported 83 cases of dermatitis caused by wood or wood derivatives (8). 300 botanical species and 28 toxic woods were identified (8). Irritant chemicals are often found in sap or latex and are characteristic of certain families such as Moraceae, Urticaceae, Euphorbiaceae, and Apocynaceae (8). Systemic symptoms may be caused by alkaloids or glycosides absorbed through the respiratory tract or food, or occasionally by skin abrasions (8). Ash from plant wood can accumulate heavy metals, such as chromium or arsenic. A study reported neurological and skin poisoning following seasonal exposure to arsenic from burning chromium-copper arsenate wood (9)

The ash contains large quantities of potash which is a powerful irritant if its concentration is right (4). In Turkey and Brazil, researchers have tested potassium hydroxyl solutions to treat Molluscum contagium in children; the studies showed that the solution was effective and may present a low-cost alternative, however, skin irritation and burns were observed at concentrations above 10% (10), (11). In the literature, a reported case confirms the hypothesis that the rash is related to the use of ash; a woman with HIV in Burkina Faso had necrotic epidermal detachments almost generalized following the use of a potash solution, prepared from the ashes of wood plants (4).

The pharmaceutical analysis, literature search, and imputability score calculation according to the French method of Bégaud (12), revealed that imputability was plausible with a score (I2). All reported data question the relationship between ash and the occurrence of the adverse effect cited. Despite the many reported cases in the literature cited above on the effects of wood ash, there is no pharmacological explanation for the effect report, investigations can provide conclusive answers.

Conclusion

Traditional treatments have been proving their effectiveness since ancient times, however misusing and not knowing the composition of preparations and their mechanism of action can lead to adverse effects.

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Case Report

Journal of Emergency Medicine Case Reports

A Different View of Minoca; A Rare Case of Coronary Cameral Fistula

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Abstract

Coronary cameral fistula (CCF) is defined as an abnormal connection between the coronary artery and the heart chambers. Although rare, most area symptomatic. It is less likely to be seen in older age. It usually starts from the right coronary artery and ends in the right ventricle or right atrium. Often detected incidentally, CCFs can also present with symptoms of heart failure or rarely with anginal symptoms. We report a case of coronary cameral fistula presenting with a prediagnosis of acute coronary syndrome in our clinic.We aimed to contribute to the literature by sharing a rare case of coronary cameral fistula presenting with a diagnosis of acute coronary syndrome especially in emergency departments.

Keywords: Angina pectoris, coronary angiography, coronary cameral fistula, stealing syndrome

Introduction

A coronary cameral fistula is defined as an abnormal connection between the coronary artery and the heart chambers (1). The most common coronary artery fistula is the fistula originating from the right coronary artery and spillingin to the right ventricle. Fistula sterminating in the left atrium or left ventricle are rare (2). In particular, CCFs originating from all three epicardial coronary arteries are rare but can be clinically significant. These cases may present with acute coronary syndrome causing steal syndrome. Since there is often no underlying obstructive lesion, these cases may be diagnosed as myocardial infarction with out obstructive coronary arteries (MINOCA) (3).

Case Report

A 51 years old male patient was admitted to the emergency room with a complaint of stabbing chest pain for the last one week. In his anamnesis, he described that the chest pain increased with exertion and radiated to the back. It was learned that he had no known disease and coronary history, was a 20 pack/year smoker, had no family history and was not taking any medication regularly. Vital signs in the emergency room revealed a blood pressure of 120/80 mmHg, pulse rate of 85 perminute and saturation was 95%. Electrocardiography (ECG) showed sinus rhythm V 1-6 with T negativity 85/min (Figure-1). Echocardiography revealed no wall motion defect and no major valve pathology. Laboratory findings showed renal function tests within normal range, C-reactive protein within normal range, haemoglobulin level 17.6 gr/dl, HS Troponin-T level 12.9 ng/l (upper limit 14 ng/l). At 3 hours, the control HS Troponin-T level was 69.2 ng/l and the patient was hospitalized in the coronary intensive care unit with a prediagnosis of acute coronary syndrome. Coronary angiography was performed during follow-up. Angiography showed dilatation in the main coronary artery (LMCA), ectasia in the left anterior descending artery (LAD) and circumflex artery (CX), and diffuse tortuosity in the coronaries. CCF formed by the LAD, CX and right coronary artery (RCA) together and spilledin to the left ventricle. Opaque filling was observed in the left ventricle (Figure-2). Although the patient had a low body mass index, dilatation of the coronary arteries was thought to be the effect of steal syndrome caused by multiple fistulas. The patient was admitted to the coronary intensive care unit with medical follow-up. The patient's medical treatment was adjusted as acetylsalicylicacid (ASA) 100 mg pill 1x1, metaprolol 50

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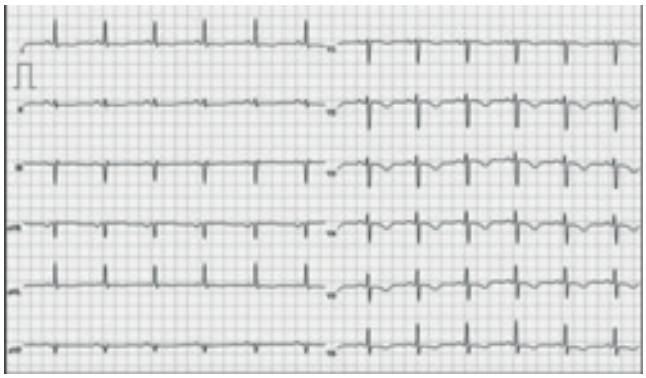

Figure 1. T negativity in leads V1-6 in normal sinus rhythm

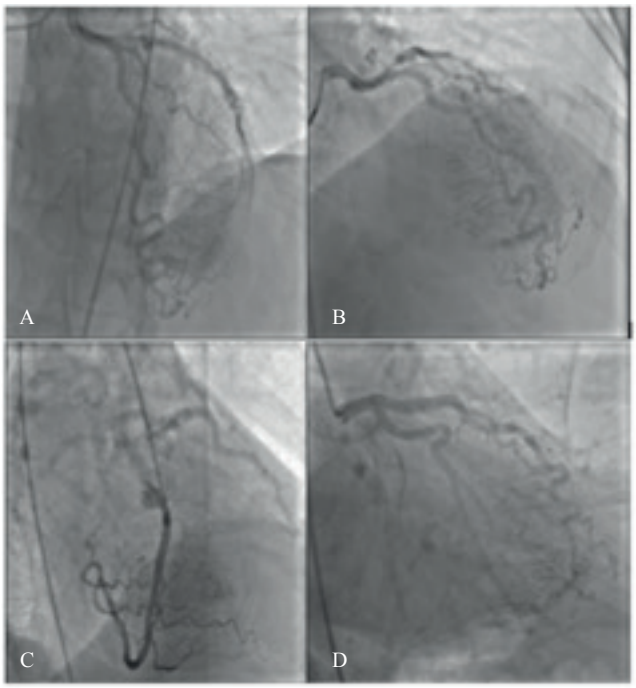

Figure 2. Coronary angiography images. Left anterior descending artery (A,B), right coronary artery (C) and circumflex artery (D) and left ventricular microfistulas in different views

mg pill 1x1, and rosuvastatin 20 mg pill 1x1 due to an LDL cholesterol level of 149 mg/dl. In the patient who currently had nosigns of heart failure, medical treatment was decided primarily in terms of CCF. The patient was discharged with the recommendation of out patient follow-up.

Discussion

CCFs are rare coronary anomalies. Especially CCFs opening into the left ventricle are less common in the literature (1). When we look at the etiology, the most common cause of CCFs is abnormal embryogenesis (4). While the diagnosis can be made at any age, the diagnosis is usually made in early childhood when a heart murmur occurs in an asymptomatic child or in a child with symptoms of heart failure. However, cases of CCF diagnosed at an advanced age with anginal complaints and signs of acute heart failure are seen in the literature (5). It may cause anginal symptoms by causing heart failure symptoms and steal syndrome. (6). When we look at the literature, we can see that patients with CCF also present with acute coronary syndrome (2, 4, 5). As in the case reported by Alsancak Y et al., CCFs originating from all three coronary arteries cause steal syndrome and are often diagnosed with a prediagnosis of acute coronary syndrome (7). The absence of an occlusive lesion after angiography in these patients with high troponin levels suggests atherosclerosis at the microvascular level and the diagnosis of MINOCA (3).

Conclusion

Angiography performed with suspicion of stenosis in the coronary arteries and finding a CHF instead of a stenosis may be considered lucky in these patients. The size of the fistula and the possibility that it may lead to heart failure in the follow-up will be the other side of the unlucky coin.

Consent statement: Written informed consent was obtained from the patient(s) for publication of this case report, including accompanying image.

Declaration of competing interest: The remaining authors declare that they have no competing interests.

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Case Report

Journal of Emergency Medicine Case Reports

Boerhaave Syndrome: A Case Report

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Abstract

Boerhaave syndrome is a spontaneous longitudinal transmural rupture of the esophagus, first described in 1724 by German physician Herman Boerhaave. Spontaneous ruptures constitute 15% of all esophageal ruptures, typically occurring after persistent vomiting that leads to a sudden increase in intraluminal esophageal pressure. The syndrome has a high mortality rate and presents with Mackler's triad: vomiting, mild chest pain, and subcutaneous emphysema. This case report describes a 63-year-old male who presented to the emergency department with severe chest and upper abdominal pain. Physical examination revealed tenderness in the upper quadrants and mild crepitus around the neck. A thoracoabdominal CT scan showed extraluminal air in the mid-lower esophageal area, leading to a diagnosis of Boerhaave syndrome. Emergency surgery included a right-sided thoracotomy, revealing a 3 cm esophageal perforation, which was repaired. Postoperatively, the patient was treated in the intensive care unit with expanded antibiotic therapy and managed for various complications. The patient was discharged on the 18th postoperative day. Early diagnosis and treatment of Boerhaave syndrome are critical for improving patient survival. Detailed patient history, recognition of clinical symptoms, and the use of appropriate diagnostic tools are essential for accurate diagnosis and timely surgical intervention.

Keywords: Boerhaave syndrome, esophageal perforation, esophageal repair, primary

Introduction

Boerhaave syndrome is a spontaneous, longitudinal, and transmural rupture of the esophagus, first described in 1724 by the German physician Hermann Boerhaave (1). While iatrogenic esophageal rupture may occur during endoscopic procedures conducted for diagnostic or therapeutic purposes, spontaneous ruptures are more commonly precipitated by persistent vomiting that causes a sudden increase in intraluminal esophageal pressure. Spontaneous ruptures account for approximately 15% of all esophageal ruptures (2). Given that Boerhaave syndrome is a rare clinical entity often identified postmortem, accurate assessments of incidence and mortality rates are challenging; however, high mortality remains inevitable (1,3,4).

Boerhaave syndrome occurs most frequently in patients aged 50-70 years (1). Its clinical presentation varies depending on the location of the rupture, the volume of leakage, and the time elapsed since the injury occurred. In approximately half of spontaneous rupture cases, Mackler's triad; comprising vomiting, mild chest pain, and subcutaneous emphysema is observed (3).

This study presents a case of Boerhaave syndrome that was surgically diagnosed and treated, accompanied by a comprehensive review of diagnostic and treatment approaches informed by current literature.

Case Report

The patient, a 63-year-old male, presented to the emergency department with severe chest and bilateral upper abdominal pain that began 3 hours prior. This chest discomfort and abdominal pain developed after the patient experienced a violent cough following water aspiration. Pain intensity fluctuated with position and respiration.

On physical examination, the patient was conscious, restless, and agitated, sitting in a wheelchair and breathing shallowly. Due to discomfort, he was unable to lie in a supine position. Abdominal examination while seated revealed tenderness, rebound, and involuntary guarding in the upper quadrants. The oropharynx appeared normal, with crepitus and tenderness noted on palpation in the anterior neck, which increased with swallowing. Vital signs included a temperature of 36.8 °C, blood pressure of 110/70 mmHg, and

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oxygen saturation of 96%. The electrocardiographyshowed sinus tachycardia at 120 beats per minute, and the patient was tachypneic at 24 breaths per minute. His overall condition was stable; he was conscious and oriented. On auscultation, bilateral lung sounds were normal.

The patient had no known history of surgery or chronic illness. Laboratory findings indicated a leukocyte count of 16,590 x 10^9/L, hemoglobin level of 15.6 g/dL, and venous blood gas lactate of 2.80 mmol/L. Although the patient reported no other comorbidities, his blood glucose level was elevated at 230 mg/dL.

Before admission to our hospital, the patient had visited another hospital, where a thoracoabdominal CT scan was performed. Examination of the abdominal CT images revealed extraluminal air in the mid-lower esophageal region, leading to a diagnosis of Boerhaave syndrome (Figure-1).

Since a thoracotomy was planned for emergency surgery, selective endotracheal intubation was performed, and the right lung was depressurized. The patient was positioned in the left lateral decubitus position, and a thoracotomy incision was made between the 6th and 7th ribs on the right hemithorax, followed by placement of a retractor. A 3 cm perforation in the esophagus was identified in the mediastinal region, and the esophagus was dissected from the surrounding tissue (Figure-2). No additional defects were found. The primary repair of the esophagus was performed since 5 hours had elapsed from the onset of the patient's complaints and there was no evidence of mediastinitis (Figure-3). A thoracic tube was inserted on the right side and positioned posterior to the esophagus. The operation concluded after layer closure.

Post-operatively, the intubated patient was transferred to the intensive care unit, where a midazolam infusion was initiated. The antibiotic regimen was broadened with the addition of piperacillin-tazobactam. Initial blood gas analysis revealed a lactate level of 3.27 mmol/L and a pH of 7.33.

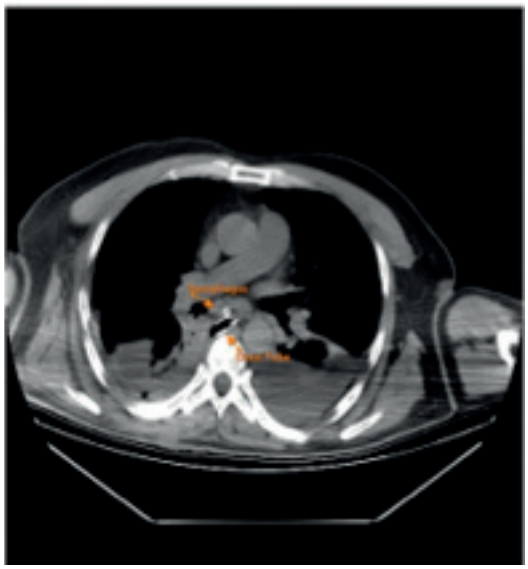

Figure 1. Perforated area in the esophagus on CT

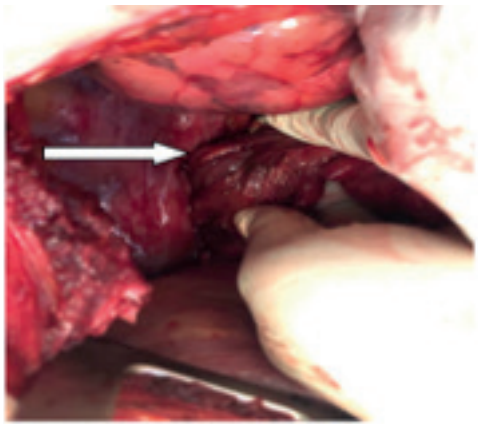

Figure 2. Perforated area in the esophagus

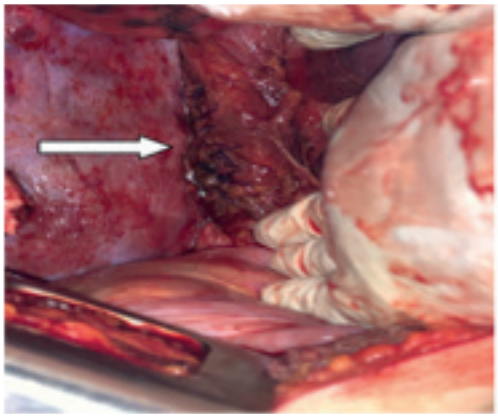

Figure 3. Appearance after primary repair

On the second post-operative day, pleural effusion was detected in the left hemithorax, prompting the placement of a pleurecan for drainage over two days. On the seventh post-operative day, due to persistently elevated CRP levels (150 mg/L), metronidazole was replaced with tigecycline. An anastomotic integrity check was performed using methylene blue administered via nasogastric tube, and no leakage was observed from the drains. Enteral nutrition through the nasogastric tube was initiated on the ninth post-operative day.

Weaning from ventilation began on the ninth day, and sedation was gradually reduced over the following 48 hours. On the tenth post-operative day, the patient was extubated, and on the eleventh day, he was transferred to the general ward.

In the ward, daily chest x-rays were conducted, and the patient received intensive respiratory physiotherapy. The chest tube was removed after resolution of the effusion and pneumothorax. The patient progressed to enteral feeding without complications while in the ward. With no signs of dyspnea, tachycardia, or effusion, the patient was discharged on the eighteenth post-operative day.

Discussion

Boerhaave syndrome is a spontaneous esophageal rupture that occurs due to a sudden and significant increase in intraluminal esophageal pressure, typically as a result of

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vomiting. While rupture can also be attributed to iatrogenic, surgical, or traumatic causes, it most commonly originates from the posterolateral wall, approximately 2-3 cm proximal to the gastroesophageal junction (6). Besides vomiting, this syndrome can occur after childbirth, during epileptic seizures, following severe coughing or hiccupping, while lifting heavy weights, running long distances, or swallowing hard substances. It predominantly affects men aged 40 to 60 years (7,8). In our patient, we believe that the onset of Boerhaave syndrome was precipitated by a forceful cough that occurred after water aspiration.

Boerhaave syndrome is a rare condition with a highly lethal progression. The primary cause of mortality is related to infections in the mediastinum, pericardium, and lungs, which can lead to sepsis. The overall mortality rate for esophageal perforation is approximately 10%; however, if diagnosis is delayed, this rate can rise to as much as 50%. The mortality rate doubles for cases that are untreated within the first 24 hours (6-8). Therefore, prompt diagnosis and treatment are critical; in our case, the patient was diagnosed and operated on within the first six hours.

The classic presentation is described by Mackler's triad, which includes vomiting, lower thoracic pain, and subcutaneous emphysema. Other common findings may include pleural effusion, abdominal rigidity, and tachypnea (9). A posteroanterior chest X-ray can reveal pleural effusion, pneumothorax, pneumomediastinum, and subcutaneous emphysema. For esophagography, water-soluble contrast agents (such as Gastrografin) are preferred over insoluble agents like barium due to their lower inflammatory potential. Thoracoabdominal CT is considered the gold standard for diagnosis, exhibiting a sensitivity of 92-100%, and typically reveals pneumomediastinum. While upper gastrointestinal endoscopy has 100% sensitivity and 92% specificity, its use remains controversial, as it may exacerbate injury and increase the risk of mediastinal contamination (10).

Primary repair is the recommended treatment for esophageal perforations in all patients without esophageal malignancy or extensive mediastinal necrosis, including those who present more than 24 hours after perforation (2).

Conclusion

In conclusion, obtaining a detailed patient history, identifying significant clinical symptoms and physical examination findings, utilizing appropriate diagnostic tools, and maintaining a high level of clinical suspicion are essential for diagnosing Boerhaave syndrome and initiating early surgical intervention. This comprehensive approach is critical in maximizing the chances of survival for affected patients.

Ethical Declarations

Informed Consent: The patient signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed. Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: A conflict of interest has not been declared by the author.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Case Report

Journal of Emergency Medicine Case Reports

Acute Lipodermatosclerosis-Like Eruption and Deep Vein Thrombosis due to Gemcitabine Use Concomittant Side Effects of Gemcitabine

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Abstract

Gemcitabine is a chemotherapy agent commonly used in the treatment of various solid tumors. Its cutaneous side effects are less well-documented, and its venous thromboembolic side effects are controversial. Here, we aim to present a case diagnosed with an acute lipodermatosclerosis-like eruption and deep vein thrombosis following gemcitabine treatment. A 66-year-old male patient presented with new-onset eruptions and swelling in the right lower extremity, four days after the first dose of gemcitabine treatment for metastatic lung cancer. On examination, there were purple-red plaques and petechiae on the right lower extremity and 2+ edema edema was detected. By superficial ultrasound (USG) diagnosis of DVT was made. The patient was discharged with recommendations for subcutaneous enoxaparin, leg elevation, and follow-up in the outpatient clinic on the fifth day with continued antibiotic therapy. Acute lipodermatosclerosis-like rash, a rare but potential cutaneous side effect in patients receiving gemcitabine treatment, should be well recognized and managed. This ensures the continuity or need for revision of treatment. Additionally, vigilance for possible venous thromboembolic events should be maintained in patients receiving gemcitabine treatment, and the possibility of concurrent occurrences with cutaneous side effects should not be overlooked.

Keywords: Acute lipodermatosclerosis-like eruption, deep vein thrombosis, emergency medicine, gemicitabine, side effect

Introduction

Gemcitabine is a chemotherapy agent commonly used in the treatment of various solid tumors such as sarcomas and hematological malignancies. While its side effects, such as bone marrow suppression, are well-defined, its cutaneous side effects are less well-documented, and its venous thromboembolic side effects are controversial (1).

Cutaneous reactions definitively associated with gemcitabine are described under the term pseudocellulitis and include lipodermatosclerosis-like and erysipeloid reactions, as well as radiation recall events (1,2). Additionally, these reactions are often confused with infectious cellulitis due to antibiotics, hospitalizations, and chemotherapy-related infections (3).

Gemcitabine has also been associated with increased arterial and venous thromboembolic events; however, this risk has not been clearly established. As the oncological indications for its use expand, the recognition and characterization of these complications become crucial (4).

In this case report, we aim to present a case diagnosed with an acute lipodermatosclerosis-like rash and deep vein thrombosis in the right lower extremity following gemcitabine treatment.

Case Report

A 66-year-old male patient presented to our emergency department with new-onset eruptions and swelling in the right lower extremity, four days after the first dose of gemcitabine treatment for metastatic lung cancer. The patient had a history of hypertension but no other comorbidities. On examination, there were purple-red, non-blanching, raised plaques and petechiae predominantly on the anterior and medial aspects of the right lower extremity (Figure-1). Additionally, 2+ edema edema was detected around the right ankle, particularly around the medial malleolus. The patient was afebrile, and a complete blood count revealed thrombocytopenia (platelet count of 149,000), but the white blood cell count was normal. The patient had been referred to dermatology by his oncologist for this rash and was started on antibiotics for suspected cellulitis. However, as his symptoms did not improve, he presented to our emergency department with the same complaints. A superficial ultrasound (USG) was requested due to possible deep vein thrombosis (DVT) and cellulitis. The USG showed intraluminal thrombosis in the distal segment of the right superficial femoral vein and the popliteal vein. There was no response to compression

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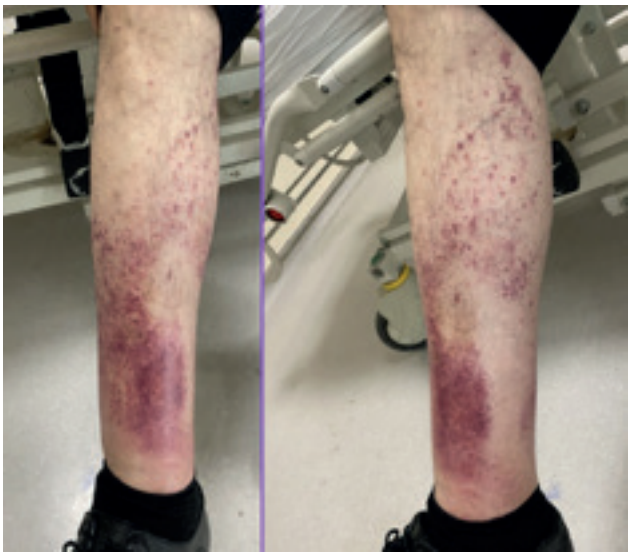

Figure 1. Tender, erythematous plaques on the bilateral lower extremities with overlying petechiae

and augmentation, and a diagnosis of DVT was made. The patient was consulted with Cardiovascular Surgery. He was discharged with recommendations for subcutaneous enoxaparin, leg elevation, and follow-up in the outpatient clinic on the fifth day with continued antibiotic therapy.

Discussion

Gemcitabine is a pyrimidine analog that blocks the cell cycle at the G1/S phase (5). It is used in various solid organ malignancies, including breast, ovarian, non-small cell lung, transitional cell bladder, pancreatic, and biliary tract cancers. Like other chemotherapeutic agents, it can be associated with alopecia, mucositis, and cutaneous hypersensitivity reactions. Additionally, it can be associated with rarer skin reactions (4).

Acute lipodermatosclerosis-like eruption associated with gemcitabine is a rare condition that is often treated as cellulitis. The differential diagnosis includes infectious cellulitis, drug hypersensitivity, toxic erythema secondary to chemotherapy, and other panniculitides such as erythema nodosum. It typically presents as sudden-onset erythematous and tender plaques, frequently on the lower extremities and often bilaterally. In our case, only the right lower extremity was affected. Additionally, patients often complain of lower extremity edema, as was observed with our patient's right ankle edema. Unlike cellulitis, these cases typically do not present with fever and leukocytosis; however, it should be noted that gemcitabine can cause drug-related fever and myelosuppression, complicating the diagnosis. The diagnosis of acute lipodermatosclerosis-like rash related to gemcitabine is primarily based on etiology and clinical findings. This side effect may necessitate the revision or discontinuation of treatment, as continuing therapy without dose reduction can lead to the persistence of the rash (4).

Classically, lipodermatosclerosis or sclerosing panniculitis develops on a background of venous insufficiency. The pathogenesis involves venous hypertension and increased vascular permeability leading to decreased fibrinolytic activity. In the acute phase, painful and erythematous plaques develop over the medial malleolus and other areas, followed by hyperpigmentation due to hemosiderin deposition. Diagnosis is primarily clinical and biopsy is not routinely performed (5,6).

In similar cases reported in the literature, bilateral involvement was observed in 85% of cases. In our case, involvement was unilateral, affecting only the right lower extremity. Additionally, edema was detected in 50% of cases in the literature, and our case also presented with edema. This edema is believed to be related to the accumulation of the drug's metabolites in the interstitial fluid and the pharmacokinetics of gemcitabine (5).

Cutaneous rashes typically occur 2 to 5 days after gemcitabine infusion. In the literature, these rashes were observed within the first 48 hours in 52% of cases (4). In our case, the rash appeared on the 4th day.

Treatment is conservative and includes high-potency topical steroids, compression therapy, leg elevation, and anti-inflammatory therapy (4).

Venous and arterial thromboembolic events are major causes of mortality and morbidity in cancer patients (7-9). Approximately 20% of cancer patients develop these events, and those who do are reported to be at increased risk of poor prognosis and increased mortality. Gemcitabine-based chemotherapy regimens have been reported to carry a significantly increased risk of thromboembolic complications (4). However, this relationship is mostly based on case reports.

In a prospective study involving 108 patients treated with gemcitabine and cisplatin for non-small cell lung cancer, thromboembolic events were reported in 17.6% of cases (10).

In a meta-analysis by Qi et al., arterial and venous thromboembolic events were investigated in patients receiving gemcitabine treatment. As a result, they found that gemcitabine use did not increase the frequency of these thromboembolic events compared to other chemotherapeutic treatments (4).

The pathogenesis of gemcitabine-associated thrombogenicity is not fully understood. It is believed that gemcitabine-associated thrombocytopenia and thrombocytosis may directly increase the risk of thromboembolic events. Additionally, due to the effects of gemcitabine on the coagulation cascade and potential endothelial damage, the frequency of these events may also increase (4).

Current guidelines do not recommend routine VTE prophylaxis for patients receiving outpatient chemotherapy. However, some new data suggest low molecular weight

heparin therapy to reduce the risk of VTE in patients with certain types of cancer (4).

Conclusion

Acute lipodermatosclerosis-like rash, a rare but potential cutaneous side effect in patients receiving gemcitabine treatment, should be well recognized and managed. This ensures the continuity or need for revision of treatment. Additionally, vigilance for possible venous thromboembolic events should be maintained in patients receiving gemcitabine treatment, and the possibility of concurrent occurrences with cutaneous side effects should not be overlooked.

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Case Report

Journal of Emergency Medicine Case Reports

Spontaneous Rupture of Splenic Artery Aneurysm: A Case Report and Review of the Literature

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Abstract

Splenic artery aneurysms are uncommon and typically asymptomatic. However, they pose a high risk of mortality if they rupture. This case report describes the occurrence of a spontaneous rupture of a splenic artery aneurysm in a 55-year-old man with no known previous diseases, no trauma history, and no risk factors for developing an aneurysm who applied to the emergency department with abdominal pain. The diagnosis was determined with an urgent abdominal computed tomography angiography scan. The patient received urgent surgical intervention consisting of splenectomy and excision of the splenic artery aneurysm. The rupture of a spontaneous splenic artery aneurysm is a rare and fatal disease that necessitates urgent diagnosis and treatment. Spontaneous splenic artery aneurysm usually develops without any noticeable symptoms and is frequently discovered by chance. In the event of a rupture, it can lead to abrupt abdominal pain and hypovolemic shock and the mortality rate is high. Ultrasound, Computed Tomography, Magnetic Resonance Imaging, and abdominal aortic arteriography are the methods of imaging that are used to diagnose splenic artery aneurysms. The key elements in patient management include prompt resuscitation, diagnostic imaging, surgical consultation, and subsequently performing a laparotomy.

Keywords: Aneurysm, emergency medicine, rupture, splenic artery

Introduction

Splanchnic artery aneurysms are a rare but potentially life-threatening condition. Splenic artery aneurysms (SAA) are the third most prevalent intra-abdominal aneurysm, following aortic and iliac artery aneurysms, and account for 60% of all splanchnic artery aneurysms. SAA is described as an abnormal dilatation of the splenic artery, with a diameter above 1 cm. The prevalence of SAA in the whole population ranges from 0.1% to 10.4%. SAAs have a 4:1 female to male ratio, yet men are at greater risk than women to rupture (1). The exact cause of this condition is not completely known, but factors that increase the risk include trauma, hormonal and local hemodynamic events during pregnancy, portal hypertension, arterial degeneration and atherosclerosis (2,3). The majority of SAAs do not exhibit any symptoms before rupture and are typically discovered by chance during medical imaging procedures. Nevertheless, patients may exhibit many nonspecific gastrointestinal symptoms, with the most prevalent being indistinct or sharp epigastric or left upper quadrant pain that may radiate to the left shoulder (4). SAA is an uncommon and potentially life-threatening condition that is associated with a certain risk of rupture, particularly when it exceeds 2 cm in diameter. The symptoms consist of acute presentation with hypovolemic shock and acute abdomen as a result of intra-abdominal hemorrhage caused by rupture. The imaging methods used to diagnose splenic artery aneurysm include ultrasound, pulsed Doppler, Computed Tomography (CT), Magnetic Resonance Imaging (MRI), and the gold standard, abdominal aortic arteriography. There are multiple treatment options for splenic artery aneurysms, such as open, laparoscopic, and endovascular surgery. Surgeons will assess the most secure approach based on the urgency of the case (5, 6). This article discusses a case of spontaneous rupture of a splenic artery aneurysm (SAA) without any known risk factors.

Case Report

A 55-year-old male patient presented to the emergency room with a one-day history of abdominal pain. The patient reported no history of trauma and claimed to be in good overall health. The patient reported no known medical conditions or regular medication use in his medical history. The patient's vital signs

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indicated a blood pressure of 120/75 mmHg, heart rate of 80 bpm, oxygen saturation of 96% on room air, and body temperature of 36.8°C. Physical examination identified widespread abdominal tenderness, with no signs of guarding or rebound tenderness detected. The rectum was found to be empty during the rectal examination. His additional physical evaluation yielded no notable findings. The electrocardiogram revealed a normal sinus rhythm without any signs of acute ischemia. The results of a complete blood count revealed a hemoglobin level of 11.5 g/dL, a white cell count of 17.3×109/L, and a platelet count of 250×109/L. All other laboratory tests were within the normal range. An abdominal Computed Tomography Angiography (CTA) scan showed an 18X14 mm aneurysm in the distal portion of the splenic artery, located 3.5-4 cm from its origin. The scan also revealed extravasation of intravenous contrast, along with intravenous contrast extravasation consisting of a large amount of free intraperitoneal fluid and intraperitoneal bleeding (Figure-1). The patient was taken for emergency surgery by the general surgery department after obtaining informed consent. A splenectomy was performed involving the excision of the aneurysmal sac. The surgical report indicated a widespread organized hematoma within the abdomen and noted the presence of leakage-type hemorrhage surrounding the splenic artery aneurysm. The surgical pathology results showed a bleeding area adjacent to the capsule in the sections of the splenectomy specimen, with no additional pathological findings observed. On the 6th postoperative day, the patient was discharged without any postoperative complaints.

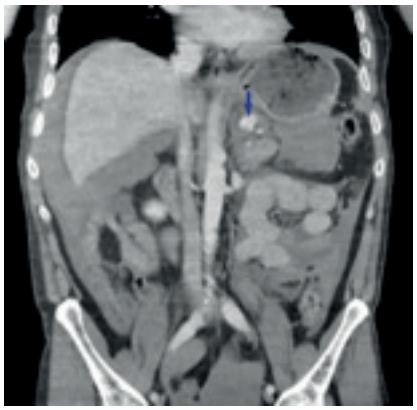

Figure 1. Splenic artery aneurysm rupture is observed at the location indicated by the arrow sign

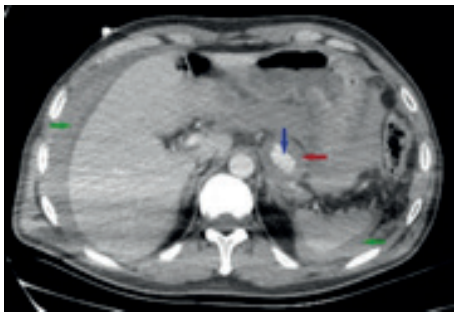

Figure 2. The hyperdense area at the tip of the blue arrow represents a ruptured aneurysmal appearance in the distal segment of the splenic artery, and there is a hypodense hematoma area around it indicated by the red arrow. The areas marked with green arrows represent perihepatic and perisplenic hemorrhagic fluid

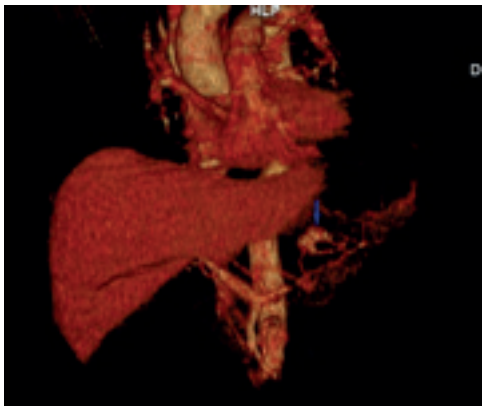

Figure 3. A three-dimensional CT image illustrates the splenic artery aneurysm indicated by the arrow

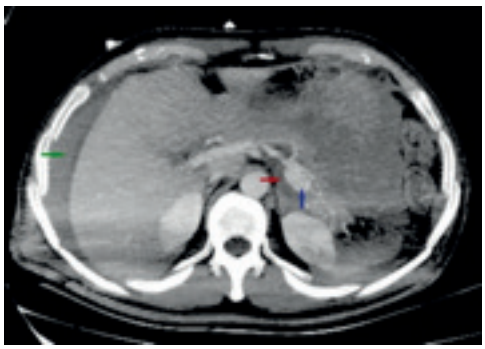

Figure 4. In another horizontal CT section, the blue arrow indicates the splenic artery aneurysm, the red arrow highlights the hematoma surrounding the aneurysm, and the green arrow shows the perihepatic hemorrhagic fluid

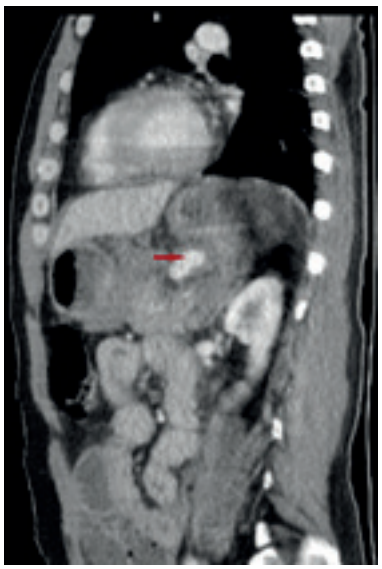

Figure 5. A sagittal CT section showing the splenic artery aneurysm, indicated by the arrow

Discussion

SAA is a rare but seriously life-threatening vascular disease due to the potential risk of rupture and bleeding, which occurs in 3-10% of cases. Rupture and bleeding result in sudden abdominal pain and can lead to rapidly progressing hypovolemic shock. The mortality rate might increase up to 75% after an aneurysm rupture in these patients. Therefore,

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it is crucial to make every effort to diagnose and treat it early (5, 7). CTA is the preferred initial diagnostic method for SAAs. Alternatively, magnetic resonance angiography can be employed to confirm the diagnosis in individuals with suspected SAA and renal insufficiency that limits the utilization of iodinated contrast medium. Its pathogenesis is not completely known, however factors that increase the risk include fibromuscular dysplasia, collagen vascular diseases, being female, having a history of multiple pregnancies, and having portal hypertension (2). The natural process of SAA is similar to other intra-abdominal aneurysms. Ultimately, rupture may occur as a consequence of a progressive increase in size. Approximately 80% of splenic artery aneurysms are asymptomatic and are typically discovered by chance during medical imaging examinations. Patients with symptomatic SAA (20%) may have abdominal pain in the epigastrium or left upper quadrant. Other symptoms may include anorexia, nausea, or vomiting and are often attributed to concomitant hiatal hernia or other pathologies such as gallstones and peptic ulcer disease. Rarely, a pulsatile or other mass may be identified during a clinical examination. The choice of treatment typically relies on the etiology, anatomical location, and sizes of the aneurysm. The conventional treatment method for treating SAA involves either open or laparoscopic surgery. Typically, when dealing with proximal SAAs, the preferred approach is aneurysmectomy and reconstruction while preservation of the spleen. On the other hand, for distal SAAs, aneurysmectomy with splenectomy is required (1, 2, 8). The main intervention for ruptured SAAs primarily consists of fluid resuscitation and hemodynamic support, however, urgent surgical intervention is necessary irrespective of the stability of the patient's hemodynamics. Furthermore, it is advised that all symptomatic SAAs be treated urgently (9,10). The literature contains a few cases of splenic artery aneurysm rupture in patients who did not have substantial risk factors or an identifiable etiology. In our clinical case, a splenic artery aneurysm ruptured spontaneously in a healthy male patient who had no history of trauma, pregnancy, or other known risk factors. In our case, aneurysmectomy was performed along with splenectomy as surgical treatment. In our case, the urgent radiological diagnosis, prompt fluid resuscitation, and timely surgical intervention played a very crucial role in his survival.

Conclusion

In conclusion, even in the absence of identifiable risk factors or in patients considered to be at low risk, the possibility of a spontaneous rupture of a splenic artery aneurysm should be considered in the differential diagnosis of abdominal pain. Advanced imaging techniques and rapid and appropriate life-saving interventions are crucial for the early diagnosis of life-threatening SAA rupture.

Ethics and approval to participate: Since this is a case report, there is no requirement for approval from the local ethics committee.

Consent for publication: The patient has provided signed consent for the release of their data, which includes personal details and images.

Conflict of interest: There are no conflicts of interest to declare.

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