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CASE REPORT

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A rare presentation of spontaneous rupture of splenic vein aneurysm as cardiac arrest: A case report

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Abstract

Splenic vein aneurysm (SVA) is a rare condition associated primarily with portal hypertension. This case highlights a potentially fatal, rare SVA complication in a young patient who presented in cardiac arrest, creating a diagnostic enigma in the emergency department until a POCUS revealed a possible underlying etiology.

KEYWORDS

aneurysm, cardiac arrest, spleen, splenic vein rupture

1 | INTRODUCTION

Portal vein aneurysms (PVAs) are the most common, yet rarely clinically encountered type of visceral venous aneurysm.[1] PVAs were found in 0.43 percent of the 4186 consecutive patients who underwent routine abdominal contrast-enhanced multidetector computed tomography (MDCT) from 2004 to 2006.[2] These are anatomically classified as intrahepatic or extrahepatic PVAs, with extrahepatic accounting for nearly 63 percent of all cases. Young and old have been equally affected, with an average age of 53.4 years. Aneurysm sizes range from 19 to 50 mm, with a median of 28.4 mm.[2] The majority of aneurysms occur in: The portal vein's main trunk, the hepatic hilus, or the junction of the splenic vein and the superior mesenteric vein.[3] Splenic vein aneurysm first identified in 1953,[4] is a type of extrahepatic PVA.[1,5] SVA can be caused by a congenital weakness in the vessel walls or an acquired cause such as: portal hypertension, liver disease, pancreatitis, or trauma.[1]

Intra-abdominal hemorrhage is a medical emergency that can cause hypovolemic shock, cardiac arrest, and even death if not treated immediately. The most common cause of a life-threatening intra-abdominal bleed is blunt and penetrating abdominal trauma. Nontraumatic causes such as: bleeding from hepatocellular carcinomas, rupture of arterial or venous aneurysms, or spleen rupture are uncommon.[6–9]

We present an intriguing and unusual case of spontaneous rupture of a splenic vein aneurysm, which resulted in hypovolemic shock and cardiac arrest.

2 | CASE PRESENTATION

A 19-year-old boy was brought to our emergency department by emergency medical service (EMS) as a case of cardiac arrest. Further history from the family revealed that the patient had been suffering from abdominal pain for the previous 8 h. The pain was sudden in onset and was generalized in location. It was not radiating or shifting anywhere, and there were no reported aggravating or relieving factors. Initially, the pain was milder with no associated symptoms such as: nausea, vomiting, loose stools, constipation, or fever. After nearly 8 h, the pain became more intense, and the patient began to feel dizzy, prompting him to seek medical attention.

The patient was found unconscious with no palpable pulse when the EMS arrived. Cardiopulmonary

resuscitation (CPR) was commenced following the advanced life support (ALS) protocol. The cardiac monitor's initial rhythm was pulseless electrical activity (PEA). Prior to arriving at the hospital, the patient had a return of spontaneous circulation (ROSC), subsequent to three cycles of CPR in 10 min.

In the ED, the patient's airway was protected with a laryngeal tube, and he received two boluses of 0.9% sodium chloride. The patient had equal chest rise and good air entry bilaterally. Three minutes after his presentation to the ED, he had another PEA cardiac arrest. CPR restarted, and ROSC was achieved after two cycles. A definitive airway was secured with an 8.0 mm endotracheal tube. The patient's skin was cold and clammy, with no apparent external bleeding or penetrating trauma.

A bedside POCUS scan revealed free fluid with echogenic contents in both the abdomen's right and left upper quadrants (Figure 1). Central venous access was obtained in the right femoral vein, and another peripheral IV line was inserted to aid fluid resuscitation. Blood and blood products were arranged, and a general surgeon was consulted for a potentially life-saving exploratory laparotomy. Under ultrasonic guidance, 15 ml of fluid was aspirated, revealing a gross hemoperitoneum. According to the hospital's local guidelines, a massive transfusion protocol was activated. The patient was transferred to the operating room while maintaining an acceptable mean arterial pressure of 63 mmHg.

The surgeon reported a 1.5 cm splenic vein aneurysm tear at the pancreas' junction of the body and tail. This resulted in a massive hemoperitoneum which could have been the cause of the hypovolemic shock and cardiac arrest at presentation. Hemostasis was achieved by ligating both ends of the severed splenic vein. A diffuse ooze was also found in the adjacent pancreatic parenchyma, short gastric vessel area, and adjacent greater omentum. The absorbable hemostat material oxidized regenerated cellulose was used to control this.

Following surgery, the patient was transferred to the intensive care unit (ICU) for close monitoring. His ICU stay was complicated by multi-organ failure and persistent bleeding likely due to disseminated intravascular coagulation evident by multiple factor deficiencies, thrombocytopenia, low fibrinogen level, high d-dimers, and elevated prothrombin and activated partial thromboplastin times (Table 1). In accordance with the massive blood transfusion protocol, the patient received: packed red blood cells, fresh frozen plasma, ten units of cryoprecipitate, a dose of prothrombin complex, 6 g of fibrinogen, 500 ml of plasma protein fractions, and a 90 mcg/kg bolus of activated factor VII. Almost 3000 ml of blood was collected in the abdominal drain in 36 h postoperatively. After replacing intravascular volume, the patient was commenced on a noradrenaline infusion at 2 mcg/kg/min.

Despite all these measures, the patient persistently remained in shock, necessitating another exploratory laparotomy, which revealed venous congestion in some parts of the small bowel and the splenic flexure area of the colon. Intraoperatively, the patient suffered a further cardiac arrest resulting in death despite correcting hypothermia and prolonged CPR for over an hour.

3 | DISCUSSION

The SVA is a sporadic occurrence. Since Lowenthal and Jacob reported the first SVA in 1953,[4] very few cases have been documented. Though the etiology of SVA is still unknown, several contributing factors may lead to this condition including: trauma, portal hypertension, pancreatitis, and venous wall weakness.[1,10] Most SVA patients are asymptomatic or have mild-to-moderate abdominal or back pain. However, our patient presented with severe abdominal pain, which is usually an uncommon presentation. This is primarily due to aneurysmal rupture or splenic vein thrombosis.[1,11,12] These patient usually have hemodynamic instability. In 2009, Parpaglioni et al. described a case of spontaneous rupture of an SVA.[13] Although ultrasonography aids in diagnosing SVA, color doppler is now the preferred method. It is noninvasive and useful for monitoring.[14]

The SVA can also be detected using modern MDCT scanning. Still, it is essential to note that increased

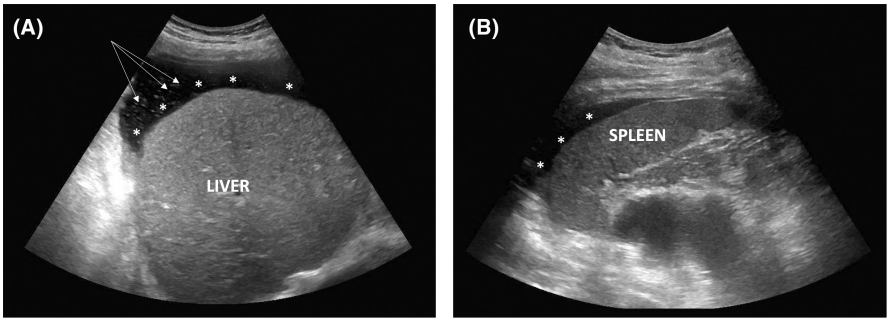

FIGURE 1 Selected images of Bedside POCUS (point of care ultrasound) showing free fluid in the abdomen shown by asterisks (*) both in the right upper quadrant (A) and left upper quadrant (B). Arrows in A are showing echogenic contents (debris) in the free fluid suggestive of hemoperitoneum.

TABLE 1 Important blood investigations

Lab investigations	Patient's value	Normal range
Hemoglobin (gm/dl)	6.5	13–17
White blood cells ($\times 10^3/\mu l$)	14.8	4–10
Platelets ($\times 10^3/\mu l$)	49	150–400
Prothrombin time (s)	20.7	9.7–11.8
Activated partial thromboplastin time (s)	58.5	24.6–31.2
International Normalized Ratio	2.0	-
D-dimer (mg/L)	>4.40	0.00–0.44
Fibrinogen (gm/L)	1.18	1.7–4.2
Factor II (%)	25.3	70–120
Factor V (%)	<5.3	70–120
Factor VII (%)	947.2	70–120
Factor VIII (%)	32.6	70–150
Factor IX (%)	25.6	70–120
Factor X (%)	48.5	70–120
Factor XI (%)	35.2	70–120
Factor XII (%)	33.8	70–150
Factor XIII (%)	70.4	70–140
Von Willebrand factor Antigen (%)	125.9	65.5–187.5
Von Willebrand factor Activity (%)	103.8	63.3–199.7
Urea (mmol/L)	10.30	2.76–8.07
Creatinine (μmol/L)	449	70–115
Alanine aminotransferase (U/L)	2516	0–40
Aspartate aminotransferase (U/L)	>5800	0–37
Troponin T (ng/L)	3116	0–14
Lactic acid (mmol/L)	18.2	0.5–2.2

radiation exposure and IV contrast reaction are shifting the trend toward magnetic resonance angiography (MRA).[15] Furthermore, magnetic resonance imaging aids in distinguishing SVA from other lesions, such as pancreatic tumors, reducing the risk of an incorrect biopsy—a potentially fatal error.[11]

The majority of studies on PVA address intervention options. Nevertheless, because SVA's have an uncertain course, the timing of definitive treatment is highly debated.[15] Torres et al. advocated prophylactic surgical intervention for abdominal venous aneurysms in patients with a small asymptomatic aneurysm or a low surgical risk.[1] High-risk patients, on the contrary, should be observed and closely monitored.[1,15,16] If the clinician chooses a conservative management approach, a close follow-up with ultrasonography, CT scan, or an MRA is required to regulate changes in luminal flow or size of the aneurysm.[15,17] Symptomatic aneurysms or those with associated complications such as: rupture, thrombosis or mass effect, on the contrary, should be treated surgically.[3,17]

Aneurysmectomy, splenectomy, splenorenal shunt, aneurysmorrhaphy, and distal pancreatectomy are surgical options. A 2016 publication by Kwon et al. reported the treatment of a traumatic SVA via a trans-splenic approach with a viabahn stent.[18] The transhepatic approach allows for greater access to the portal veins. It is commonly used for portosystemic collateral embolization, thrombosis or portal vein stenosis, and portal hypertension treatment via transjugular intrahepatic portosystemic shunts. In terms of hemorrhage risk, this approach is less risky than the transsplenic approach; keep in mind that the spleen is highly vascularized, and its vessels are very fragile. Complications may include: vasovagal reactions, pain at the site of puncture, bleeding, pseudoaneurysms, hemobilia, and infection.[19]

4 | CONCLUSION

SVA's and their treatment are controversial. An ongoing inflammatory process increases the risk of complications

such as SVA rupture. As a result, all patients with SVA's should be treated as soon as possible. Asymptomatic cases are best treated conservatively, whereas symptomatic or significant SVA's require surgical intervention. Furthermore, the use of POCUS in the emergency department, particularly in cardiac arrest settings, clearly makes a difference in management.

AUTHOR CONTRIBUTIONS

Hamid Ilyas: involved in data curation and wrote the original draft. Hina Akram: validated, wrote, reviewed, and edited the manuscript. Muhammad Abd Ur Rehman: conceptualized the study and did a literature review. Syed Haris Huda: wrote, reviewed, and edited the manuscript.

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CONFLICT OF INTEREST

In compliance with the ICMJE uniform disclosure form, all authors declared no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

ETHICAL APPROVAL

The initial manuscript of this article was submitted to the medical research center in the Hamad Medical Corporation, and an approval letter was obtained from the ethical committee as well as the institutional review board with approval number MRC-04-21-607.

CONSENT

Informed written consent was obtained from the patient's family to publish this report in accordance with the journal's patient consent policy, and we took approval from the online platform of Hamad Medical Corporation "www.abhath.hamad.qa" for publication of this article after anonymizing all the personal details of the patient.

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