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CASE REPORT

Spontaneous hemoperitoneum, due to bleeding from retroperitoneal varices, in a cirrhotic patient: a case report

Ahmad Abutaka[1], Renol Mathew Koshy[1], Abdulrahman Abu Sabeib[1], Adriana Toro[2] & Isidoro Di Carlo[1,3]

[1]Department of General Surgery, Hamad General Hospital, Al Rayyan Road, 3050, Doha Qatar
[2]Department of Surgery, Barone Romeo Hospital, via Mazzini 14, 98066 Patti, Italy
[3]Department of Surgical Sciences and Advanced Technologies "G.F. Ingrassia", University of Catania, via Santa Sofia 78, 95100 Catania, Italy

Correspondence
Renol Mathew Koshy, Department of General Surgery, Hamad General Hospital, Al Rayyan Road, 3050 Doha, Qatar.
Tel: +974 55748825;
E-mail: renolkoshy@gmail.com

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Key Clinical Message

Hemoperitoneum from retroperitoneal varices in cirrhotic is very rare. This condition should be taken into account based on anamnesis, clinical features, and laboratory findings; but due to the unstable presentation, diagnosis remains a challenge. Emergency laparotomy could be effective treatment, but the prognosis remains poor related to the hepatic reserve.

Keywords

Cirrhosis, hemoperitoneum, hemorrhagic shock, portal hypertension, varices.

Introduction

Spontaneous hemoperitoneum is a rare and catastrophic complication of portal hypertension [1], mainly affecting patients with liver cirrhosis. Rupture of retroperitoneal varices can lead to hemoperitoneum. The worldwide mortality rate of patients with ruptured retroperitoneal varices causing hemoperitoneum is 65.7%, with surgical intervention reducing the mortality rate to 57.1% [1]. Only 35 cases of spontaneous rupture of retroperitoneal varices causing hemoperitoneum have been reported in the literature [2]. This case report describes the first such patient with this condition in Qatar.

Case Report

A 47-year-old alcoholic male was brought to the Emergency Department (ED) of the Hamad General Hospital in Doha, Qatar. The patient was unresponsive, with no history of trauma. On examination, his blood pressure was 60/35 mmHg, his heart rate was 121 beats/min, his respiratory rate was 16 breaths/min, his body temperature was 35.4°C, his Glasgow coma scale score was 3/15, and his body had a strong smell of alcohol. The patient was intubated and ventilated. His abdomen was found to be distended and tense, with an everted umbilicus; his bowel sounds were negative and a digital rectal examination showed no blood or masses. A nasogastric tube drained bilious fluid, and ultrasonography and a diagnostic peritoneal lavage (DPL) revealed blood. His MELD score was 68.8 and his Child–Pugh grade was C-11. His unresponsiveness may have been due to alcohol intoxication or encephalopathy.

Laboratory investigations showed a red blood cell (RBC) count of $2.6 \times 10^6/\mu L$, a hemoglobin (Hb) concentration of 7.0 g/dL, a hematocrit of 23.7%, a platelet count of $70 \times 10^3/\mu L$, and a white blood cell (WBC) count of $7.3 \times 10^3/\mu L$, creatinine was 133 mmol/L, albumin was 13 g/L, total bilirubin was 17.6 μmol/L, AST was 130 U/L, ALT was 37 U/L, ALP was 194 U/L, lactic acid was 17.9, INR was 1.5, ammonia was 166 mmol/L, ethanol was 35 mmol/L, pH was 6.93, P_{CO_2} was 70.9 mmHg, P_{O_2} was 199 mmHg, BE was −17.0 mmol/L, and HCO3 was 9 mmol/L.

A massive transfusion protocol, consisting of 5 units of packed RBCs and 4 units of fresh frozen plasma, was

initiated and an emergency exploratory laparotomy was performed. Intraoperatively, around 3 L of blood and clots were evacuated and a bleeding retroperitoneal varix, in the left lower quadrant, was identified and ligated (Fig. 1). The liver was cirrhotic and the spleen was enlarged. Following this damage control procedure, the abdomen was left open with a Bogota bag as a laparotomy, for a relook if required. The patient, still intubated, was transferred to the surgical intensive care unit for postoperative care. He continued to ooze serous fluid from the laparotomy, but there was no more bleeding. His hemoglobin stabilized, with an RBC count of $3.1 \times 10^6/\mu L$, Hb 9.1 g/dL, hematocrit 27.1%, platelet count $147 \times 10^3/\mu L$, WBC count $4.4 \times 10^3/\mu L$, creatinine 169 mmol/L, albumin 22 g/L, total bilirubin 30 μmol/L, AST 1347 U/L, ALT 336 U/L, ALP 47 U/L, lactic acid 29.7 mmol/L, and INR 1.4.

However, the patient developed multiple organ dysfunction syndrome (MODS) and died 48 h after the operation.

Discussion

Retroperitoneal bleeding is a very rare condition with an incidence of 0.1%, which increases six times in patients on anticoagulation [3]. The most common cause is trauma to the pelvic and lumbar regions, followed by iatrogenic complications of femoral artery cauterization. Rupture of aneurysms, commonly aortic and iliac [4–6], and surgeries and radiological interventions involving any of the retroperitoneal organs (pancreas, kidneys, and adrenals) also contribute to retroperitoneal hematomas. However, spontaneous hemorrhage is rarer and can be seen as a complication in patients on hemodialysis and anticoagulants, with hemorrhagic disorders and in cirrhotics with portal hypertension with retroperitoneal varices.

Dilated portosystemic collateral veins located in sites other than the gastroesophageal region can be defined as ectopic varices [2]. These sites may include the duodenum, jejunum and ileum, colon, rectum, stoma sites, and retroperitoneum, and, more rarely, the vagina and ovaries. Bleeding from ectopic varices represent 2–5% of variceal bleeds in the gastrointestinal tract [8]. The veins of Retzius connecting the superior and inferior mesenteric veins with the lumbar and lower intercostal veins represent the main sources of retroperitoneal bleeding from varices [9].

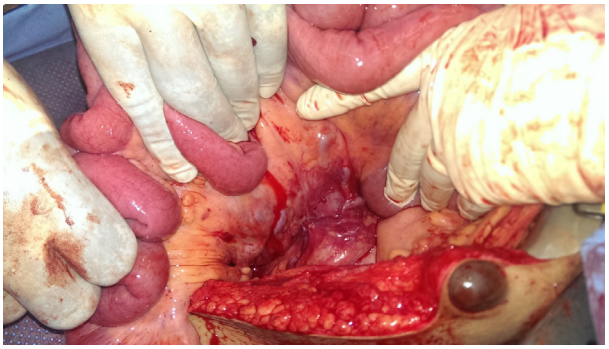

Figure 1. Retroperitoneal varices (after ligation).

The initial clinical manifestations in cirrhotic patients with rupture of retroperitoneal varices and hemoperitoneum include pain, abdominal distension, light headedness and syncope, with hypotension being the cause of all these symptoms [1]. As our patient was admitted to the ED unresponsive and in hemorrhagic shock, it was not possible to evaluate his symptoms. Of course, in patient like the present one, the anamnesis and the clinical features have to be considered to achieve the right diagnosis and the retroperitoneal rupture of varices have to be taken in account as one of the cause, also if rare.

In the presence of hemoperitoneum, abdominal pain is proportionate to the rate and volume of blood loss within the abdominal cavity. Hence, peritoneal signs may not be present [10]. Our patient presented with a massive and tense abdomen with an everted umbilicus and absent bowel sounds.

The first diagnostic step is abdominal ultrasound, which can acquire information about all the possible sites of bleeding (vessels or tumors) [1]. In women, measuring beta HCG level is a crucial diagnostic test. CT scan and magnetic resonance imaging (MRI) are valuable adjuncts in localizing the source of acute intraabdominal hemorrhage [11], but these methods can only be performed in well compensated and stable patients. In unstable cirrhotic patients, ultrasound and DPL would be diagnostic [12]. Our patient was hemodynamically unstable and therefore underwent a DPL before the emergency laparotomy.

Treatment of retroperitoneal bleeding is still not established as secured guidelines. All patients initially have to be treated in ICU with monitoring, fluid resuscitation, blood transfusion. In case of coagulation, disorders like hemophilia or warfarin administration, conservative management may be adopted [13].

Endovascular treatment in retroperitoneal hemorrhage became used even more in alternative to open surgery for arterial bleeding that can be blocked with embolization in critical and noncritical patients [13].

Open surgery is indicated in all patients who despite adequate resuscitation remain unstable. The role of surgery is to identify the source of bleeding and control it, while also evacuating the hematoma. The other indication for surgery is the presence of abdominal compartment syndrome [13].

Generally, management of variceal bleeding associated with portal hypertension includes treatment with a vasopressor to reduce portal pressure, as well as source control by endoscopic or interventional radiology techniques, with surgery considered the method of last resort. However, because of the rarity of such conditions, no certitudes have been established. A review of 34 cirrhotic patients with hemoperitoneum secondary to rupture of intraperitoneal varices found that surgical intervention was the only effective treatment to control bleeding [1]. Bleeding was halted in 26 (92.9%) of 28 patients who underwent ligation of the bleeding varix, with 12 (42.9%) of these patients surviving past the postoperative period [1]. In contrast, none of the patients managed nonsurgically survived.

Factors predictive of survival in cirrhotic patients with spontaneous hemoperitoneum secondary to retroperitoneal variceal rupture include functional hepatic reserve, the severity of hemorrhagic shock at presentation, and the time taken to control the bleeding [9]. All of these factors adversely affected our patient and contributed to his cause of death.

Conclusion

This clinical condition remains extremely rare and despite aggressive emergency management, the prognosis of this catastrophic complication of portal hypertension remains poor. Due to the unstable presentation in the ED, diagnosis is a challenge. Immediate emergency laparotomy remains the only effective treatment option.

Conflict of Interest

None declared.

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