CASE REPORT

An Unusual Presentation of Splenic Rupture in a Hemodialysis Patient

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ABSTRACT

Splenic rupture, although rare in dialysis patients, can be life-threatening. In this case, a 45-year-old man with kidney failure on hemodialysis experienced sudden and severe abdominal pain in the left flank, without any history of trauma. He displayed symptoms of hypovolemic shock, characterized by pallor, hypotension, and tachycardia. Additionally, he had abdominal distension and tenderness. An abdominal CT scan revealed a splenic hematoma and intra-abdominal hemorrhage. The patient required a splenectomy to address the ongoing bleeding, but unfortunately, he succumbed to post-splenectomy sepsis 2 weeks later.

Keywords: Atraumatic, splenic rupture, hemodialysis, platelet dysfunction, kidney failure

Atraumatic splenic rupture is less common than trauma-related cases[1]. However, in dialysis patients, platelet dysfunction significantly increases the risk of bleeding from internal organs like the spleen[1]. Nontraumatic splenic rupture can also occur due to factors such as heparin use, infections, splenic infarction, amyloidosis, and portal hypertension[1-4].

Spontaneous splenic rupture is a potentially life-threatening condition and it is important to maintain a high level of suspicion in kidney failure patients experiencing abdominal pain and bleeding. In cases of hemodynamic instability, splenectomy may be necessary. However, it carries a risk of post-splenectomy sepsis. In this case, a patient with kidney failure, on hemodialysis, experienced spontaneous splenic rupture, but unfortunately developed post-splenectomy sepsis and passed away.

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CASE PRESENTATION

Medical History

A 45-year-old man with kidney failure due to chronic glomerulonephritis began hemodialysis in January 2024. He visited the emergency department with severe abdominal pain that had been ongoing for 8 hours. The pain started suddenly, was sharp, and rated as 10 on a scale of 1 to 10.

It was present throughout his abdomen, particularly severe on the left side, and worsened with movement. He had no prior history of left chest wall or shoulder pain, no previous instances of similar pain, and no abdominal injuries. He mentioned passing loose, watery stool before coming to the hospital but had not experienced vomiting, increased abdominal swelling, or a fever. About 1 month ago, he was treated for an anterior chest wall abscess, which had developed at the point of previous insertion of a subclavian venous catheter.

He had an incision and drainage and was treated with antibiotics in the outpatient clinic but did not return for a follow-up. He had been consistently receiving hemodialysis twice a week along with intradialytic doses of unfractionated heparin (5,000 IU).

For the past 2 months, he had been producing limited urine and developed ascites, requiring occasional abdominal paracentesis guided by ultrasound. Four days before his hospital visit, his packed cell volume was measured at 28% before his routine dialysis

session. He did not have diabetes and had not recently undergone surgery.

His regular medications included nifedipine, torsemide, hydralazine, metoprolol, iron sucrose, vitamins, and erythropoietin.

On examination, he was sweaty, pale, and in shock with pulse rate and blood pressure of 110 bpm and 97/54 mmHg, respectively. His pulse was weak but equal on both sides. The abdomen was distended and generalized abdominal tenderness, maximal in the left hypochondriac region, was present. There were no significant findings in other systems.

Initial differential diagnosis:

- Suspected abdominal aortic dissection
- Splenic rupture
- Spontaneous bacterial peritonitis.

Treatment

He was admitted to the high-dependency unit and started on intravenous noradrenaline. A blood transfusion of 4 pints of blood and 1 unit of fresh frozen plasma was urgently requested. He was also administered intravenous antibiotics (meropenem and metronidazole) and the general surgical team was promptly consulted for further assessment.

Oxygen therapy was initiated and he was advised nil per os. He underwent laparotomy, on Day 1 of admission which revealed a ruptured spleen with a subcapsular hematoma of approximately 2 liters and 2.5 liters of blood in his abdomen (hemoperitoneum) necessitating a splenectomy. He received an additional 5 units of blood and 2 units of fresh frozen plasma due to significant blood loss during surgery.

While on inotropic support, his pulse rate was 102 bpm and his blood pressure was 130/60 mmHg. He was not given heparin for his subsequent dialysis. He had pneumococcal and meningococcal vaccination on the 7th day of surgery as vital signs were stable and results were normal.

Outcome and Follow-Up

On the 13th day of surgery, he developed a massive upper gastrointestinal bleed and blood parameters were consistent with a disseminated intravascular coagulopathy from sepsis (see Table 1).

Thereafter, he received 4 units of fresh frozen plasma with blood and but bleeding did not stop. He succumbed to the illness on 15th day of surgery.

Table 1. Investigations

Test	Results
Full blood count	Hemoglobin – 3.3 g/L; Packed cell volume – 9.6%; Platelet – 2,67,000 cells/mm^3; White blood cell count – 12,000 cells/mm^3 (Neutrophil – 77.9%; Lymphocyte – 30%)
Abdominal CT scan	See Figure 1
Clotting profiles	
• At presentation	Prothrombin time (PT) – 10 seconds; Partial thromboplastin time with kaolin (PTTK) – 37 seconds
• At Day 14	PT – 16 seconds; PTTK – 50 seconds
Kidney function tests	
• At presentation	Sodium – 145 mmol/L; Potassium – 4.4 mmol/L; Urea – 21.3 mmol/L; Creatinine – 903 mmol/L
• At Day 14	Sodium – 138; Potassium – 3.9 mmol/L; Urea – 34.8 mmol/L; Creatinine – 385 mmol/L
	PT – 14 seconds; PTTK – 45 seconds, INR – 1
Splenic tissue histology	Normal splenic tissue with areas of cellular necrosis and inflammatory changes
D-dimer (Day 14)	3.67 µg/mL (0-0.5)
Peripheral blood film (Day 14)	Fragmented red blood cells with schistocytes and target cells. The white cells showed neutrophilia with toxic granulation. Platelets were normal, but reduced at 3-4 per high power field.

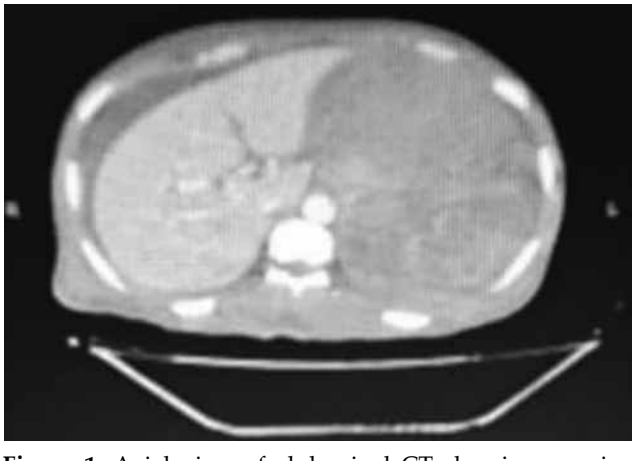

Figure 1. Axial view of abdominal CT showing massive splenic hematoma.

DISCUSSION

Atraumatic splenic rupture is a less common but potentially fatal cause of massive intra-abdominal hemorrhage. Splenic rupture can occur in a healthy spleen[5], although splenic injury from trauma is more prevalent. A systematic review of over 800 published cases identified the main causes of nontraumatic splenic rupture as malignancies, infections, and inflammatory diseases (30%, 27%, and 20%, respectively)[6,7].

Other causes included drugs (9%), mechanical causes such as pregnancy (7%), and unknown causes (7%)[6,7]. The mortality rate was 12%, with most deaths occurring in patients with splenomegaly, those over the age of 40, and those with malignancies[6,7]. Several mechanisms of splenic rupture have been postulated[8,9].

However, the most widely accepted explanations include raised intra-abdominal pressure due to contracting abdominal muscles during physical activities, intra-splenic cellular congestion, and increased fragility of splenic blood vessels resulting from infarctions and thromboses[8,9].

Spontaneous splenic rupture in dialysis patients is most frequently caused by uremic coagulopathy[8,10]. This condition is characterized mainly by platelet dysfunction, specifically a decrease in active integrin alpha IIb/beta 3 (formerly known as glycoprotein IIb/IIa), due to the accumulation of blood uremic toxins[11-13].

However, some other factors also contribute to this condition, for example, the use of antiplatelet medications, administration of heparin during dialysis, and anemia[13]. Splenic infarcts, thrombocytopenia, portal hypertension, and amyloidosis are also potential risk factors[14].

Conversely, the serum levels of pro-hemostatic clotting factors are generally normal or increased in chronic kidney disease, which may be prothrombotic[13].

Additionally, the reduced levels of naturally occurring anticoagulants such as protein C and S may also result in thrombosis in patients having nephrotic syndrome[13].

Therefore, strategies to prevent uremic bleeding should include optimization of hemodialysis, treatment of anemia, judicious use of medications such as anticoagulants and antiplatelets, and lastly administration of desmopressin before high-risk procedures[13,15].

Our patient had risk factors for uremic coagulopathy such as exposure to heparin, azotemia, and anemia. He had no other risk factor for splenic rupture. Due to the massive intraperitoneal hemorrhage and hemodynamic instability, he underwent splenectomy but developed post-splenectomy sepsis afterwards.

CONCLUSION

Splenic rupture should be considered in the differential diagnosis of abdominal pain with shock in a hemodialysis patient. Urgent investigation is needed for prompt surgical intervention.

Patients on hemodialysis who have had splenectomy are at risk of sepsis and have to be aggressively treated to prevent mortality.

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