

Case report

Atraumatic spleen rupture presenting as acute abdominal pain – a case report

Abstract:

Splenic rupture is a potentially life-threatening condition associated with acute abdominal pain. Although rare, atraumatic spleen rupture (ASR) has been reported. It is not usually considered in the differential diagnosis of acute abdomen so often missed in emergency leading to high morbidity and mortality. We present the case of a 38-year-old male who presented with fever and acute abdominal pain, had atraumatic splenic rupture which was diagnosed early resulting in better outcome of patient.

Keywords: Atraumatic spleen rupture, Viral infection, splenectomy

Introduction:

Splenic rupture, a potential life-threatening condition generally associated with trauma carries risk of significantly high morbidity and mortality. However, rare cases of ASR have been reported. High index of clinical suspicion is required to diagnose ASR in cases of acute abdominal pain, especially without any trauma history, which makes it challenging to diagnose and is often missed leading to high morbidity and mortality. We present the case of a 38-year-old male who presented with acute abdominal pain was detected to have atraumatic splenic rupture and was managed with splenectomy.

Case Report:

A 38-year-old male, came to ER with complaints of high grade, intermittent fever since 4 days, body ache and retro orbital pain since 3 days. He also had pain in the left upper quadrant of abdomen for 1 day. Abdominal pain was insidious in onset, dull aching, continuous, initially occurring in the left upper quadrant, gradually becoming generalized, associated with episodic vomiting. There was no history of chest pain, breathlessness, burning micturition, rash or eschar, or any neck stiffness. There was no history of any trauma, injury or any accident.

Patient was conscious, cooperative and oriented to time, place and person. General examination revealed mild pallor. There was no icterus, cyanosis, edema, clubbing, or any significant lymphadenopathy. His pulse rate was 92/min, regular, low volume, no radio radial or radio femoral delay, all peripheral pulses were palpable, Blood pressure was 80/56 mmHg, left arm supine posture, Respiratory rate was 20/min, Temperature was 99.3°F, SpO₂ was 98% with room air. Systemic examination revealed mildly distended abdomen, superficial tenderness in the left hypochondrium along with presence of rebound tenderness. On percussion there was dull note and no shifting dullness was present.

Routine investigation revealed low Hb (10.3 mg/dl) with low platelet count ($120 \times 10^9/L$) and normal leucocyte count (TLC $5.98 \times 10^9/L$). Liver function test showed elevated transaminases

(SGOT 963, SGPT- 813, ALP 67, GGT 89). Renal profile was normal (urea 34.3 mg/dl, creatinine 1.0 mg/dl). Further workup including fever profile (Typhidot, dengue NS1 and serology, Malaria antigen and smear, Leptospira antibody, Scrub typhus IgM) and viral markers were inconclusive. Blood and urine culture were sterile and serum procalcitonin was normal. Chest X Ray view was normal. USG whole abdomen revealed increased echogenicity of liver, splenomegaly, moderate amount of free fluid with internal echoes and few septations in the peritoneal cavity in peri splenic and perihepatic region. He was started on injectable ceftriaxone, antipyretics, iv fluids and other supportive measures. Repeat blood count was done next day and there was drop in His hemoglobin dropped to 5.3 gm/dl next day for which PRBC transfusion was done, and CT triple phase angiography abdomen was suggestive of active extravasation of contrast from superolateral aspect of spleen suggestive of active bleed with peri splenic hematoma and hemoperitoneum (Figure 2, 3, 4, 5). Exploratory laparotomy with urgent Splenectomy was done under general anesthesia revealing frank blood in the abdominal cavity along with large blood clots with a large wedge-shaped clot of size 6 cm x 4 cm x 4 cm observed in the peri splenic fossa and a large curved lacerated wound of approximately 10 cm over the posterior surface of spleen (figure 1).

Post operatively, he received PRBC and FFP transfusion. Haemophilus influenzae b (HIB), Pneumococcal and meningococcal Vaccination were done. He improved gradually and was discharged. He was stable and doing good on his last follow up 3 months after surgery.



Figure 1: a large curved lacerated wound of approximately 10 cm over the posterior surface of spleen



Figure 2: Active contrast extravasation noted on venous phase images from upper pole



Figure 3: Coronal MIP images (arterial phase): Loculated perisplenic haematoma noted. Hyperdense fluid noted in perihepatic region suggestive of haemoperitoneum



Figure 4: Arterial phase images (Axial): Heterogeneous, perisplenic haematoma noted, with layered hyperdense content

UNDER REVIEW

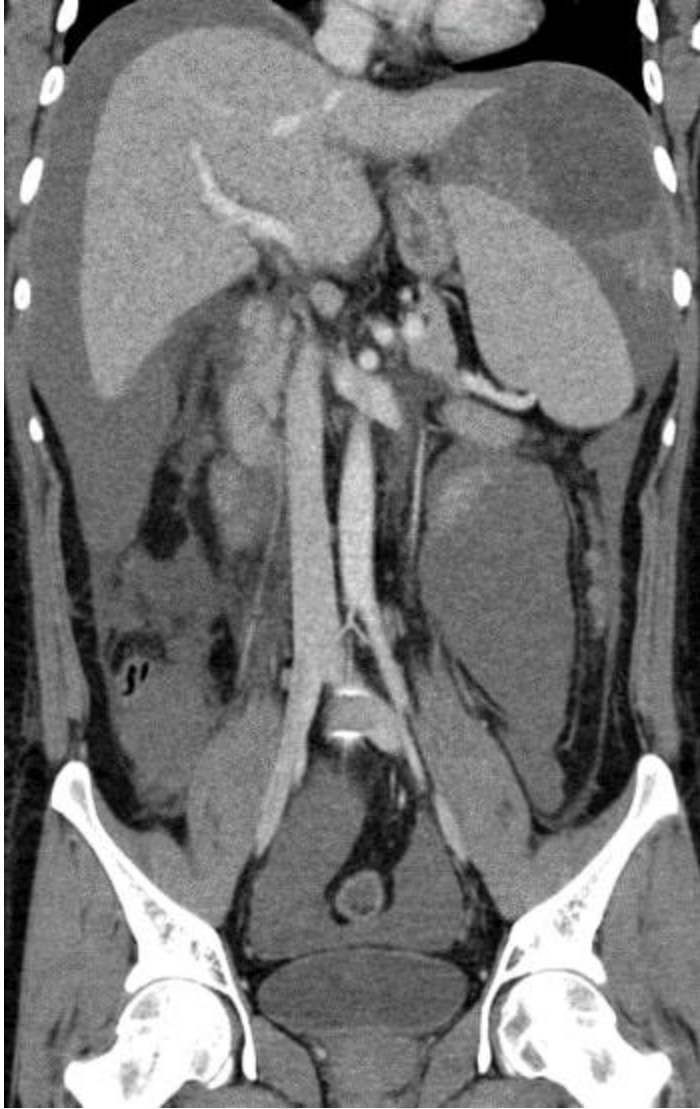


Figure 5: Coronal MIP images-extensive hemoperitoneum

Discussion:

Splenic rupture is a potentially life-threatening condition presenting as acute abdominal pain with high morbidity and mortality. Splenic rupture can be traumatic or ASR. ASR is a rare condition with incidence of 1% and mortality rate of 12.2%.^{1,2} ASR is a rare cause of acute abdominal pain and is rarely considered as differential diagnosis in absence of history of trauma. Rokitansky and Atkinson described first documented case of ASR in 1861 and 1874 and Weideman defined spontaneous splenic rupture.³ Knoblich differentiated non traumatic splenic rupture into pathological spleen and due to unknown etiology, with more than 90% cases being related to pathological spleen, which was renamed as atraumatic-pathological splenic rupture and atraumatic-idiopathic splenic rupture, according to etiology and pathological changes in spleen.^{2,4}

Exact mechanism of idiopathic atraumatic splenic rupture is not understood however, there are few hypothetical mechanisms proposed including parenchymal engorgement and vascular occlusion due to hyperplasia of intrasplenic cellular or reticuloendothelial cells or abdominal muscle compression during activities like sneezing, coughing or defecation.⁵

Orloff and Perkins gave diagnostic criteria for Idiopathic splenic rupture viz., 1) absence of any history of trauma 2) absence of any pre-existing splenic disease 3) absence of adhesions or scarring in the spleen, 4) grossly normal spleen, macroscopically and histologically,⁶ with addition of 5th criteria of full virological studies of acute phase and convalescent sera show no significant rise in viral antibody titre by Crate and Payne.⁷

Upper or left sided abdominal pain, tenderness was the most common initial presentation followed by hypovolemic shock and peritonitis in later stage.¹ Kehr's sign i.e., a sharp radiating pain to the left shoulder, is found in 20% cases.⁸ Diagnosis of ASR is a diagnosis of exclusion and CECT abdomen plays an important role.⁹

Many viral infections have been recognized which affect the spleen, but histological finding develops later. So, it is possible in our case that a subclinical viral infection was responsible for splenic rupture however, it is extremely difficult to test and the result obtained would have no change on the management of the patient.

Management is based on hemodynamic stability, amount of blood product required, degree of hemoperitoneum and splenic injury extent as classified by American Association for the surgery of Trauma (AAST) grades of splenic injury.¹⁰ Prompt surgical splenectomy is required to stabilize the patient with high grade injuries, similar to our case.

Conclusion:

Splenic rupture is a rare condition, and not a differential diagnosis for acute abdominal pain in cases with no history of trauma, so often missed in ER, which leads to significantly high morbidity and mortality. It should be kept as a differential diagnosis, even in absence of any history of trauma and other splenic pathology. CECT abdomen is essential for diagnosis and management is based on aggressive treatment of shock and Splenectomy, depending on hemodynamic status.

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