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General Surgery

A RARE CASE OF HEMOPERITONEUM DUE TO SPONTANEOUS SPLENIC LACERATION

KEY WORDS:
Hemoperitoneum, spontaneous splenic laceration.

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ABSTRACT

A rare case of hemoperitoneum due to spontaneous splenic laceration is reported.

INTRODUCTION:

Acute abdomen due to spontaneous hemoperitoneum is a serious condition demanding sound knowledge, high index of suspicion and proper patient management. We report a rare case of a 22 years old female with hemoperitoneum due to spontaneous splenic laceration.

CASE REPORT:

A 22- year old female patient attended the surgical emergency department with upper abdominal pain for 3 days. It is associated with nausea and 2 episodes of non-bilious, non-projectile vomiting but is not associated with either loose stools or constipation or bleeding per rectum or bleeding from any other site or urinary complaints or fever. History of trauma is conspicuously absent. Patient was not on any anticoagulant drugs but only on anticonvulsants. Her last menstrual period was 4 months back but the urine pregnancy test was negative.

5 months back the patient had a single episode of convulsion. It was investigated and MRI Brain showed acute intraparenchymal haemorrhage with perilesional edema in left frontal lobe with midline shift of 4 mm with minimal Subarachnoid hemorrhage in left frontal lobe for which anticonvulsants was started.

On arrival patient had tachycardia, hypotension and tachypnoea. Abdomen was diffusely tender with no guarding or rigidity.

Hemogram showed haemoglobin of 4.70 gm/dl and a haematocrit of 15%. Rest all routine blood investigations were within normal limits.

Ultrasonography showed approximately 48 x 42 mm sized heterogeneously hyperechoic lesion with linear hypoechoic area in the upper pole of spleen possibility of laceration. Peripheral smear was unremarkable. All hemolytic and coagulation profile like Direct coombs test and Indirect coombs test, Factor 8, Factor 9, Sickling test, Bleeding time, Clotting time, Prothrombin time with INR, Activated Partial Thromboplastin Time (APTT) were within normal limits. Dengue, Widal and chikungunya tests were also negative. Antinuclear antibody test was also negative.

CECT Abdomen and pelvis showed approximately 42 x 59 x 52 mm sized irregular, non-enhancing area in upper and mid portion of spleen extending up to the posterolateral surface suggestive of laceration with contusion (Grade III American Association for the Surgery of Trauma {AAST} injury) with mild hemoperitoneum with bilateral moderate to gross pleural effusion with underlying consolidation. Chest X-ray revealed consolidation in bilateral mid and lower zone and cardiomegaly. 2D echo showed an ejection fraction of 55%, severe mitral regurgitation with possibility of chordae rupture.

Patient was shifted to BiPAP mode of ventilation in the ICU and inotropic support was started. Patient was transfused 5 units of PCV, 8 units of Fresh frozen plasma and 4 units of platelets. Intercostal Drainage (ICD) tube was placed bilaterally and an intra-abdominal drain was inserted under local anesthesia. There was around 200 cc serohemorrhagic output in both the ICD and about 10 cc hemorrhagic output from the intra-abdominal drain.

Patient gradually improved symptomatically. Abdominal drain was removed after 4 days, right sided ICD was removed after 10 days and left sided ICD was removed after 12 days. Patient was shifted on ventimask 3 days after admission and inotropic support (noradrenaline) was gradually tapered over 2 weeks. NCCT Brain was done which showed the hypodense area in the left frontal lobe which is suggestive of gliosis/ oedema. MRI Brain showed focal non enhancing area of FLAIR hyperintense gliosis with volume loss and cystic encephalomalacia involving left frontal lobe without surrounding hemosiderin deposition. There was no evidence of acute infarct. Cerebral venous sinuses appear patent.

Patient was discharged after 20 days. At 1 month after follow up, the patient was asymptomatic.

DISCUSSION:

Idiopathic spontaneous intraperitoneal hemorrhage was first reported by Barber in 1909 and was later termed "Abdominal apoplexy" by Green and Powers in 1931.

The various causes of spontaneous hemoperitoneum are classified into following bases on the source of bleeding.[1]

CAUSES OF SPONTANEOUS HEMOPERITONEUM:

- HEPATIC
  - Rupture of hepatic adenoma/ hepatic adenomatosis/ hemangioma/ FNH

- Rupture of hepatocellular carcinoma/ primary angiosarcoma
- Rupture of metastatic lesion (colon, lung, renal cell carcinoma, testicular, Wilms, choriocarcinoma)
- Benign infiltrative disease of liver, Amyloidosis
- Liver rupture (HELLP Syndrome)
- Cirrhosis with portal hypertension and intraperitoneal rupture of varices
- Spontaneous bacterial peritonitis leading to hemoperitoneum.

- SPLENIC
  - Post trauma- delayed splenic rupture
  - Infectious mononucleosis, Cytomegalovirus, AIDS, EBV, Malaria, Bartonella
  - Hematological malignancy associated splenomegaly (leukemia/ lymphoma)
  - Tumor- hemangiopericytoma, Primary/ Secondary angiosarcoma
  - Splenic cyst/ abscess
  - Splenic infarcts (infective endocarditis)
  - Torsion of wandering splenic pedicle
  - Torsion of spleen
  - Portal hypertension and splenomegaly with liver cirrhosis
  - Sickle cell anemia
  - Infiltrative diseases (Amyloidosis, Gauchers disease)
- RENAL
  - Angiomyolipoma
  - Renal cell carcinoma
  - Coagulopathy
  - Vasculitis- PAN, Wegeners
- ADRENAL
  - Severe stress
  - Sepsis
  - Anticoagulation
- GASTROINTESTINAL
  - Diverticulum of sigmoid colon
  - Colonic/ intestinal/peptic perforation
  - Angiodysplasia/ AV Malformation of the gut
- GYNECOLOGICAL
  - Rupture of ovarian cyst
  - Rupture of ectopic pregnancy
  - Retrograde menstruation
  - Ectopic endometrial tissue
  - Metastatic disease like gestational trophoblastic tumour
  - Pregnancy/ Postpartum spontaneous hemorrhage (rupture of uterine vessel)
  - Hemorrhagic corpus luteum cyst torsion
  - HELLP Syndrome
  - Rupture of uterine leiomyoma
- VASCULAR

  ARTERIAL
  - Rupture of arteriosclerotic / mycotic/ congenital aneurysm
  - Splanchnic arterial aneurysm/ mesentric/ retroperitoneal vessel rupture
  - Erosion of vessel by neoplastic / inflammatory disorder (pancreatitis/ cholecystitis/ appendicitis/ Meckel's diverticulitis)

  VENOUS
  - Spontaneous intraabdominal variceal rupture
  - Rupture of uterine veins
  - Rupture of rectus hematoma into peritoneum

  A-V MALFORMATIONS
- ANTICOAGULATION
  - Hemophilia
  - Idiopathic thrombocytopenic purpura
  - Hepatic failure
  - Anticoagulant therapy (Warfarin/ Heparin)
  - SLE/ PAN
  - Blood dyscrasias
  - Myeloproliferative disorder (leukemia/ lymphoma)
  - Dengue
  - Disseminated Intravascular Coagulation
  - Severe celiac disease with vitamin K malabsorption
  - Rodenticide poisoning Brodifacoum (Superwarfarin)
  - Congenital factor X deficiency
- OTHER
  - CAPD (Continuous ambulatory peritoneal dialysis)
  - Hemodialysis dependent
- IDIOPATHIC

SPLENIC CAUSES

Delayed splenic rupture may occur following blunt abdominal trauma. Spontaneous splenic rupture mainly occurs in cases of marked splenomegaly because of underlying hematologic malignancies (acute leukemia or lymphoma) or infectious causes such as mononucleosis or Cytomegalovirus, malaria, EBV, Bartonella henselae, AIDS in young patients.[2] Few cases are reported citing spontaneous rupture of hyper vascular tumours like hemangiopericytoma leading to splenic hematoma. Wandering spleen is an unusual condition characterized by increased splenic motility due to laxity and its torsion can lead to spontaneous splenic rupture.[3]

Clinical presentation in such cases include acute abdominal pain and shoulder pain due to subdiaphragmatic irritation. Diagnosis at CT is suggested by the identification of a grossly abnormal spleen with peri splenic hemorrhage or clot in the organ. Treatment may be conservative, or surgical in the form of total or partial splenectomy, or transcatheter embolization, depending on the underlying disease.[4,5]

CONCLUSION:

Spontaneous hemoperitoneum should be considered in the differential diagnosis of patients who have acute abdomen with falling hematocrit and with signs and symptoms of hypovolemia with associated positive history of a predisposing condition to avoid a dangerous diagnostic delay and culmination into a catastrophe.

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