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Atraumatic Splenic Rupture And Severe Intraperitoneal Haemorrhage In Acute Pancreatitis In A Patient With Peptic Ulcer Disease: Experience At American Cancer Hospital Iikeduru Imo State Nigeria.

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Abstract
Splenic hemorrhage is a rare complication of acute pancreatitis.
Diagnosis may be extremely difficult especially in individuals with preexisting morbidities that could lead to complications that might result in acute abdomen.
In this article, we present a 44 year-old man, a case of spontaneous splenic rupture with massive hemoperitoneum as a complication of acute pancreatitis in a known peptic ulcer patient. Clinical findings, laboratory and imaging reports only were not sufficient to make correct diagnosis. Definitive diagnosis could only be reached at laparotomy.
The purpose of this study is to emphasize the importance of high index suspicion in the diagnosis of acute pancreatitis in a patient with other underlying conditions that could lead to acute abdomen, in this case peptic ulcer disease and the importance of laparotomy as both a diagnostic and therapeutic procedure in complications of acute pancreatitis and in acute pancreatitis patients with normal serum amylase and lipase levels.
Key-Words: *Splenic rupture, pancreatitis, hemoperitonium, laparotomy, lipase, amylase.*

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I. Introduction

Atraumatic splenic rupture [ASR] is a rare complication of acute pancreatitis that is seldomly reported, [1) Although there has been scanty report of traumatic splenic injuries in Nigeria, there is none such report on atraumatic splenic injury associated with pancreatitis. Approximately 10% of atraumatic splenic rupture result from inflammatory processes. (2)ASR can develop in a diseased spleen. According to Renzulli et al, neoplasia [30.3%], infection [27.3%], inflammatory and non-infectious causes [20.0%] are frequently reported pathologies in ASR1. With respect to infection, malaria is the single most important cause of ASR globally, [3]

Other infections that infrequently result in ASR include babeosis caused by Babesia microti, transmitted mainly through the bite of infected Ixodes scapularis ticks [4]; infectious mononucleosis, caused by Epstein-Barr virus [5].

On the other hand, ASR can occur in a normal-appearing spleen without predisposing factors. This is referred to as" idiopathic" ASR [Tonolini et al]3.The anatomical proximity of the pancreatic tail and splenic hilum is responsible for the susceptibility of the spleen to inflammatory processes involving the pancreas.

The pathophysiological mechanisms leading to splenic lesions in acute pancreatitis are not fully understood. Nevertheless, a number of theories have been postulated, and these include: direct spleen erosion caused by pancreatic pseudocysts, pancreatic enzyme extravasation, perisplenic adhesions caused by recurrent inflammation of the pancreas, and spleen congestion following splenic vein thrombosis,[6].Other possible splenic complications of pancreatitis include arterial pseudoaneurysm, perisplenic / intrasplenic pancreatic pseudocyst, infarction, splenic subscapsular infection, haematoma and necrosis,[7].

Penetration of the wall of the stomach by a benign ulcer may result in free perforation into the greater or lesser peritoneal cavities. A" confined perforation" occurs when a penetrating ulcer is walled off by an adjacent structure or organ with the commonest sites of secondary involvement being pancreas, gastrohepatic omentum, liver, biliary tract, colon and mesocolon. Penetration into the spleen by benign gastric ulcers is rare [8]. Few cases of splenic abscess caused by penetrating gastric ulcer have been reported[9].According to Glick et al, penetration of the spleen by benign gastric ulcer is rare, but when it occurs there is high probability of haemorrhage(10).

Splenic hemorrhage is a surgical emergency. Although, haemodynamically stale patients can be managed conservatively with percutaneous drainage and splenic artery embolization [11], surgical intervention with splenectomy is required for hemodynamically unstable patients []12). Consequently, high index suspicion and urgent surgical intervention –laparotomy with splenectomy - are required for optimal patient outcomes.

II. Case Report

A 44 year-old man who presented with severe abdominal pain, vomiting, chest pain, cough, inability to stand or walk. Abdominal pain was generalized, severe, peppery, continuous, not related to meals, non-radiating with no known relieving or aggravating factor, and associated with loss of appetite and vomiting which was non-projectile, non-bilious and contained recently ingested food.

He was a known peptic ulcer disease patient, so he took anti-ulcer drugs when he developed symptoms but he did not experience any relief thereafter. He denied any history of trauma prior to abdominal pain.

He had previous surgical history of herniorrhaphy and appendectomy no history of hypertension or diabetes but he was an alcohol addict and a heavy cigarette smoker. On examination, he was afebrile, pale, anicteric, dehydrated with cold and clammy extremities, hypotensive, tachycardic with tattoos and incisional scars on the abdomen, severe epigastric and left upper quadrant tenderness. Subsequent examination after about 24 hours from presentation revealed worsening abdominal tenderness and distension.

Abdominal paracentesis yielded copious amount of bloody aspirate. Preliminary diagnosis was acute abdomen secondary to exacerbation of peptic ulcer disease r/o perforated viscus. Double intravenous access was secured and blood samples were collected for laboratory investigations which included full blood count, metabolic panel, serology, lipid profile, lactate dehydrogenase, serum lipase. Serum and urine amylase were also requested.

He was then infused with 2L of crystalloids and subsequently transfused with 2L of screened, group compatible fresh whole blood.

Results of laboratory investigations were as follows: WBC total - 11520/cumm [Ref - 4000 – 11000], neutrophils - 57% [Ref – 45 - 70], lymphocytes - 5% [Ref – 25 - 40], monocytes - 23% [Ref – 2 - 8], eosinophils -13% [Ref -1 - 6], basophil - 2% [Ref -0 - 1], platelet – 181000/cumm [Ref – 150000 - 400000], PCV - 60% [Ref – 36 - 50], Hb - 20g/dl [Ref – 12 – 16], malaria parasite - ++, widal - 320 for Salmonella typhi D O and H antigen [Ref - significant titre -> 1:80], RBS - 86mg/dl [Ref-80-140], HBsAg - non-reactive [Reactive / non-reactive], HCV - non-reactive [Ref - reactive / non - reactive], HIV – non –reactive [Reactive/non-reactive]. LFT: AST - 48U/L [Ref – 0 - 45], ALT-86U/L [Ref – 0 - 45], ALP - 201U/L [Ref -80-306], total bilirubin-15mg/dl [Ref-0.2-1.2], direct bilirubin - 0.9mg/dl [Ref - 0.0 - 0.2], total protein - 5.4g/dl [Ref - 6.6 - 8.8], albumin - 3g/dl [Ref-3.5-5.2g], E/U/C: sodium - 138mm/L [Ref – 135 - 155], potassium - 3.5mmol/L [Ref - 3.5 - 5.5], chloride - 104mmol/L [Ref – 96 - 108], urea - 45mg/dl [Ref – 13 - 45], creatinine - 2.2mg/dl [Ref - 0.6 - 1.4], serum lactate dehydrogenase - 838U/L [Ref – 0 - 248], serum amylase - 69iu/L [Ref – 25 - 96], serum lipase 1.1u/L [Ref – 0 - 10], urine amylase -155u/L [Ref – 50 - 650], stool m/c/s - no ova, larvae or parasite seen, no organism isolated after 24 hours of incubation at 37 degree Celsius, serum calcium - 7.5mg/dl [Ref - 8.4 - 10.2].

Abdominal ultrasound revealed [i] sonographic features in keeping with large bowel obstruction r/o early perforated bowel [ii] hepatomegaly with fluid collection in Morison's pouch.

Consequently, he was counseled on urgent surgery and wheeled to the operating theatre after obtaining his consent, and placed in supine position. Routine cleaning with antiseptic solution and draping was done, a midline incision was made on the anterior abdominal wall to gain access into the abdominal cavity.

The findings included massive hemoperitoneum [800ml of blood], enlarged spleen with ruptured capsule in the upper portion, bleeding from splenic vessels, hematoma, necrotic tail of pancreas, multiple subperitoneal necrosis. Stomach was immobilized at the greater curvature, the spleen and necrotic pancreatic tail were resected and bleeding vessels ligatured. Abdominal cavity was washed with normal saline. Two drainages were inserted into the abdominal cavity and abdomen was closed in layers with absorbable sutures with nylon to skin. Hemostasis was maintained. The procedure was well tolerated and post-op condition was satisfactory.

III. Discussions

Splenic complications in acute and chronic pancreatitis are uncommon. Subscapular hematomas, pseudocysts, and splenic rupture are more common in chronic pancreatitis [11], while splenic infarctions and intrasplenic haemorrhage are seemingly more frequent in acute pancreatitis [13]

The anatomic proximity between pancreatic tail and the hilum of the spleen are partly responsible for the pathology of splenic complications [12].

Diagnosis of splenic complications poses a big challenge due to absence of specific symptoms. However, the presence of pain in the left upper quadrant is an indication. Imaging tests such as abdominal ultrasound scan, CT scan and MRI are valuable in identifying splenic complications. MRI allows for better characterization of the of various soft tissues vascular alterations vis-a-vis CT [14]. In the case presented here, due to circumstances beyond us, the patient could not do a CT.

Abdominal ultrasound scan revealed hepatomegaly with fluid collection in Morison's pouch and sonographic features in keeping with large bowel obstruction r/o early perforated bowel but both spleen and pancreas were unremarkable. Serum and urine amylase and serum lipase levels were within normal range but these results were only available after surgery. It was during laparotomy that the findings of acute/chronic pancreatitis-splenic rupture, intrasplenic hemorrhage, hematoma, necrotic tail of pancreas and multiple subperitoneal necrosis- were seen.

This underpins the critical need for laparotomy in arriving at correct diagnosis in addition to improving overall outcome in this patient.

Moreover, the case presented here suggests that worsening abdominal pain and distension, followed by anemia and hemodynamic instability, epigastric pain not relived by anti-ulcer drugs, finding of hemoperitoneum from diagnostic paracentesis are clinical indicators pointing to a ruptured viscus with hemorrhagic vessels. However, confirmation of pancreatitis - acute or chronic as being the cause of the rupture required laparotomy, which was also necessary to exclude other differentials of acute abdomen such as perforated gastric ulcer in this patient who was also suffering from peptic ulcer disease. Diagnosis of acute pancreatitis requires 2 of the following 3 features: [i] abdominal pain characteristic of acute pancreatitis,[ii]serum amylase and/or lipase greater or three times the upper limit of normal, and [iii] characteristic findings of acute pancreatitis on CT scan[15].In the case presented here, only one criterion was met prior surgery, which was abdominal pain characteristic of acute pancreatitis; serum amylase and lipase were normal and CT scan was not done, and during surgery there were typical findings of pancreatitis.

Lipase is more sensitive and specific than amylase in diagnosis of acute pancreatitis, with a negative predictive value 94 -100% [16]. Acute pancreatitis in the setting of normal amylase and lipase has been rarely reported in the literature. However, the absence of elevated lipase should not exclude its diagnosis [17].Omar Nadhem and Omar Salh in 2017 reported two cases of acute pancreatitis with atypical presentation- a case series of 2 patients who presented with epigastric abdominal pain and were found to have acute pancreatitis by abdominal CT scan despite having normal serum amylase and lipase levels. Factors that can lead to normal amylase and lipase values include: hypertriglyceridemia, extensive pancreatic necrosis [acute fulminant or acute-on-chronic pancreatitis], [18], or very early pancreatitis when inflammation has not resulted in extensive pancreatic acinar cell destruction yet [19]. Some of these factors were present in the patient presented here and could account for the normal serum amylase and lipase levels.

IV. Conclusion

Laparotomy is essential in the diagnosis of acute pancreatitis in (i) hemodynamic instability resulting from splenic rupture and/or hemorrhage; (ii) when laboratory or imaging results do not suggest acute pancreatitis but there is high index of clinical suspicion for acute pancreatitis or its complications; (iii) health facilities that lack modern state-of-the-art diagnostic machines such as CT, MRI. In such instances, laparotomy would not only be diagnostic but life-saving.

Abbreviations

R/o: rule out
CT: Computed tomography
MRI: Magnetic resonance imaging

Statement Of Ethics

There is no ethical approval at our institution for case reports.

Consent

Patient's consent was obtained for this publication.

Disclosure Statement

The author declares that he has no competing interest and has not received any fund or graft from any organization.

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