

Acute Biliary Peritonitis - A Rare Case Report of Isolated Gall Bladder Gangrene Secondary to Coeliac Artery Thrombus

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Abstract

Acute thrombosis of the celiac trunk is a very uncommon condition, unless diagnosed early can become a life threatening emergency. The clinical presentation is highly variable depending on the extent of the thrombus. We report a case of biliary peritonitis related to an acute thrombosis of the celiac trunk and isolated gangrene of the gall bladder. This case highlights the role of diagnostic laparoscopy in the diagnosis of acute upper abdominal pain.

Keywords: Pain Abdomen; Mesenteric Ischaemia; Laparoscopy; Gall Bladder

Introduction

Atherosclerotic vascular occlusion of abdominal arteries is considered as a dreaded disease with high mortality. In developing countries like ours, the presentation is different from those described in Western Reports.

We are hereby reporting a case of isolated gallbladder gangrene secondary to celiac trunk stenosis in a 46year old male patient, who was successfully managed by early surgical intervention.

Case report

A 46year old patient presented to emergency room with a 3-day history of pain abdomen Tenderness was noted in right subcostal region, suggestive of acute cholecystitis. Other examination findings were unremarkable.

Emergency CT-abdomen showed Atherosclerotic changes in the abdominal aorta, bilateral common and internal iliac arteries with extensive steno-occlusive disease involving the coeliac trunk, SMA, IMA, infra-renal abdominal aorta (Figures 1 & 2).

Routine hematological reports were within normal limits except for raised total leukocyte count (10.4 mg/dl), LFT was normal, coagulation profile showed prolonged prothrombin time. He was provisionally diagnosed with acute ischemic cholecystitis, and underwent emergency diagnostic laparoscopy. On diagnostic laparoscopy, (Figures 3 & 4) Omentum was adhered to gall bladder, gall bladder wall was gangrenous, necrosed with peri-cholecystic fluid collection. Rest of the visualised viscera was normal. Cholecystectomy was performed. He was stabilized, cardiology opinion was taken and discharge on post-operative day 3. HPE reports were followed up (Figure 5).



Figure 2: CT image showing gas shadow in the Gall Bladder



Figure 3: Laparoscopic view showing normal viscera



Figure 4: Laparoscopic view showing gangrenous gall bladder

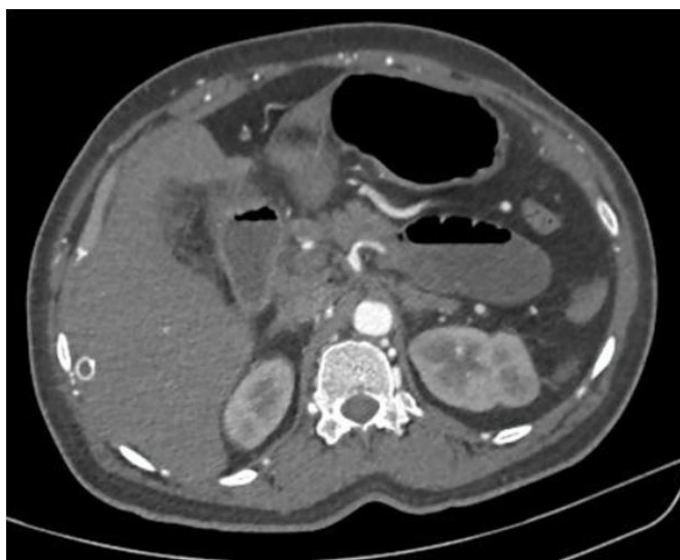


Figure 1: CT image with showing block in the Coeliac trunk

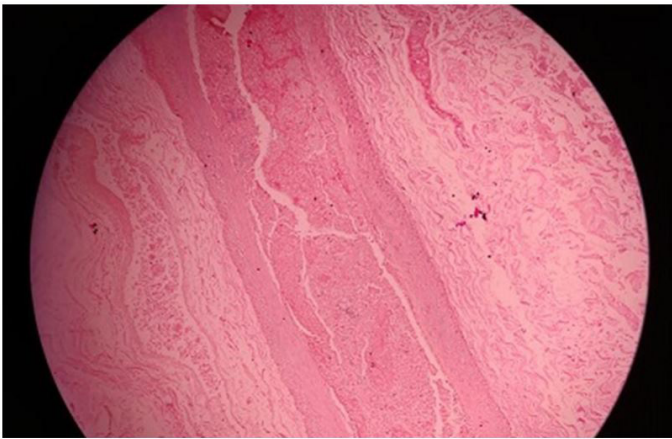


Figure 5: Microscopic photograph of ischaemic gall bladder wall with thrombosed arteriole wall

Discussion

Vascular abnormalities of the main arteries in the upper abdomen (including the celiac trunk and its axes or the mesenteric arteries) have been widely reported. In developing countries like ours, occlusion of major abdominal arteries the presentation is different from those described in Western Reports. A study published by Raghavendra Nagaraja et al showed that the patients here presented a decade earlier and venous obstruction was a more common cause. Further, the majority, are referred to tertiary medical centers at advanced stages of their disease once the features of peritonitis have developed [1]. However, according to western literature mesenteric artery (MAO) occlusion accounts for majority of cases. Schoots et al. found that MAO accounted for 71 % of cases, only 12 % were due to MVT and the remaining 17 % were due to NOMI [2]. Isolated gangrene of gall bladder with sparing of bowel has been reported by Rajiv Jain et al, however it was thrombosis involving the celiac axis and SMA, with an anomalous origin of Right Hepatic artery [3]. Different etiological forms of Acute mesenteric ischemia are: Arterial embolism (EAMI), Arterial thrombosis (TAMI), Venous thrombosis (VAMI) and Non-occlusive mesenteric ischemia (NOMI). Although they have different clinical and pathophysiological features, they do not facilitate in early diagnosis [4]. The origin of celiac trunk ranges between the 11th thoracic and the first lumbar vertebra. Most common causes of celiac trunk stenosis are median arcuate ligament syndrome, pancreatitis, malignant local invasion, atherosclerosis and idiopathic [13].

Twenty-five percent of the cardiac output goes to the splanchnic circulation at rest and increases to 35 % in the post-prandial state. Seventy percent of mesenteric blood flows to the mucosa and submucosa [5]. Therefore, microscopic changes of ischemia can be detected within minutes [6]. The gut can therefore survive a 75 % reduction in blood flow for up to twelve hours

without significant injury, irreversible bowel changes occurs within six hours of complete vascular occlusion [7].

Pathophysiology: An acute complete occlusion of an artery initially triggers a vascular spasm in the area of the ischemic bowel & results in hyperperistalsis and pain. At cellular level, energy loss with the formation of oxygen free radicals and the subsequent disintegration of mucosal cells occurs. After 3-6 hours, the intestinal peristalsis ceases and deceptive interval without pain begins, which results from the ischemia of the intramural pain receptors. In the final stage the mucosal damage becomes irreversible. Along with the infiltration of the intestinal wall by inflammatory cells, bacterial translocation takes place, resulting in intestinal gangrene [4,11].

Diagnosis: Ischemia secondary to an arterial embolism should be suspected in patients with atrial fibrillation who have a sudden onset of abdominal pain. Ischemia resulting from arterial thrombosis (TAMI) should be suspected in patients with evidence of generalized atherosclerotic disease particularly with a recent history of post-prandial syndrome. Ischemia due to venous thrombosis (VAMI) should be suspected in patients with hypercoagulable states. Non-occlusive mesenteric ischemia (NOMI) should be suspected in critically ill patients with an unexpected deterioration in their clinical condition [4]. Computed tomography angiography (CTA) should be performed as soon as possible for any patient with suspicion for abdominal artery steno-occlusive disease [8]. The diagnosis of gangrenous cholecystitis can be made with reasonable diagnostic accuracy when a markedly distended gallbladder with decreased gallbladder wall enhancement is seen on contrast-enhanced CT [9]. Tests like leukocytosis, elevated amylase level, or the development of high anion gap metabolic acidosis, are suggestive rather than pathognomonic of ischemia. D-dimer assay & time-dependent increase in its value has been found to increase as early as 30 minutes from the onset of intestinal ischemia or after ligation of the superior mesenteric artery in experimental animal models [10]. CT scanning is a diagnostic modality commonly utilized in patients who present with abdominal pain and has been reported to be sensitive in the diagnosis of abdominal artery occlusion. [10] Comprehensive biphasic CTA includes: a) Pre-contrast scans to detect vascular calcification, hyper-attenuating intravascular thrombus and intra-mural haemorrhage. b) Arterial and venous phases to demonstrate thrombus in the mesenteric arteries and veins, abnormal enhancement of the bowel wall, and the presence of embolism or infarction of other viscera. c) Multi-planar reconstructions (MPR) to assess the origin of the mesenteric arteries [8]. Multi-detector CT Imaging Findings of ischemia Bowel wall thickening caused by oedema, haemorrhage, or associated infection, with target or halo appearance, more prominent in cases with venous

occlusion. A high-attenuating bowel wall at non-contrast CT is due to haemorrhagic infarction, however hyper attenuating bowel wall at contrast- enhanced CT is due to congestion. Filling defects in the mesenteric arteries and veins indicate emboli or thrombi in the vessels. The absence of wall enhancement indicates absence of arterial flow. Mesenteric stranding and ascites are caused by congestive or reperfusion mesenteric oedema. If these manifest in cases without mesenteric congestion or reperfusion, they may indicate bowel perforation. Pneumatosis, Porto mesenteric venous gas and free peritoneal gas indicate trans-mural infarction of the bowel, with or without perforation [12].

Management: Fluid resuscitation immediately to enhance visceral perfusion. Electrolyte abnormalities should be corrected, and nasogastric drainage started. Broad-spectrum antibiotics should be administered immediately. Unless contraindicated, patients should be started on anticoagulation with intravenous unfractionated heparin. Prompt laparotomy should be done for patients with overt peritonitis [8]. The goal of surgical intervention includes re-establishment blood supply to the ischemic bowel and other abdominal viscera, resection of all non-viable regions, preservation of all viable bowel /viscera. Endovascular revascularization procedures can be attempted in partial arterial occlusion. Damage control surgery (DCS) is an important adjunct for patients who require intestinal resection and margins brought out of abdomen due to the necessity to reassess bowel viability and in patients with refractory sepsis. Diagnostic laparoscopy with planned re-laparotomy is an essential part of management [8]. ESTES guidelines recommend endovascular techniques as first line treatment when bowel integrity has not been compromised, the choice of vascular intervention will depend on available resources and expertise [4].

Prompt and goal-oriented diagnosis and consistent treatment within 4–6 h from the onset of symptoms can be decisive for the reduction of mortality [12].

Conclusion

Acute abdominal ischemic disease is a true emergency and in rare cases like this prompt emergency surgical management in order to reduce morbidity and mortality.

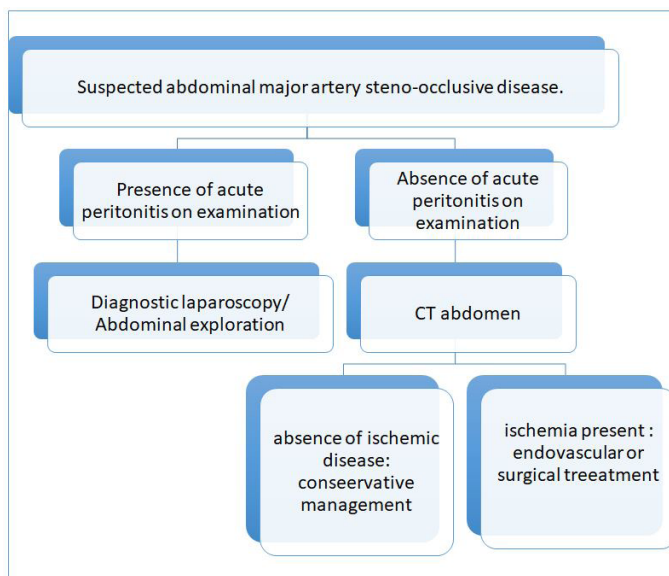


Figure 6: Diagnostic and treatment algorithm in patients with suspected acute abdominal steno-occlusive disease

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