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A case of acute calculous cholecystitis with sudden deterioration in the absence of signs of peritonitis: A case report

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Abstract
Acute cholecystitis is inflammation of the gallbladder. It usually occurs when a gallstone blocks the cystic duct. The main complications of acute cholecystitis like gangrenous cholecystitis, which can cause a serious infection that could spread throughout the body. Perforated gallbladder which can spread the infection (peritonitis) or lead to a build-up of pus (abscess). Without appropriate treatment, acute cholecystitis can sometimes lead to potentially life-threatening complications. In this case report we are discussing Portal vein thrombosis a rare complication of acute cholecystitis in a 71-year-old gentleman, which underwent sudden deterioration in absence of signs of peritonitis and the offending cause was removed surgically with laparoscopic cholecystectomy and was started on anticoagulants immediately after surgery which resulted in improvement in patient condition with recanalization of portal vein on follow up.

Keywords: acute cholecystitis, portal vein thrombosis, septic pylephlebitis

Introduction

Acute cholecystitis develops when a gallstone(s) lodges in the gallbladder neck (infundibulum) obstructing bile flow into the common bile duct (CBD). This leads to biliary stasis, gallbladder wall edema, venous obstruction, and in extreme cases, arterial obstruction with necrosis of the gallbladder wall. Incidence of portal vein thrombosis with acute cholecystitis is rare and portal vein thrombosis occurrence when cirrhosis is not present with an incidence of 0.7/100,000 [1].

There is paucity of literature to support portal vein thrombosis with acute cholecystitis, and through this case we would like to highlight the varied presentation of acute cholecystitis and role of timely intervention. Here, we present a case of acute cholecystitis which was initially being managed conservatively. However, during the course, he suddenly deteriorated and developed new onset acute left portal vein thrombosis. High index of suspicion along with timely surgical and medical intervention helped in successful management of the patient and an important learning from the case.

Case Report

A 71-year-old gentleman, presented to the emergency department with dull aching right upper quadrant pain, vomiting and fever for 4 days. He had no other complaints and no co-morbidities. On examination, he was febrile at 101'F with tachycardia (102beats/min). Abdominal examination revealed mild generalized distension with minimal tenderness in right hypochondrium. Blood investigations revealed raised infective markers {Leukocytosis; 19960 x 10^9/L, C-reactive protein (CRP) 4.5mg/dl}. Rest of Blood parameters including liver and kidney function tests were normal. Abdominal X-ray showed few small bowel air-fluid levels. Abdominal Computed Tomography (CT) was done. It was suggestive of acute calculus cholecystitis with an impacted calculus at the junction of the gall bladder neck and proximal cystic duct along with few dilated small bowel loops with free flow of contrast.

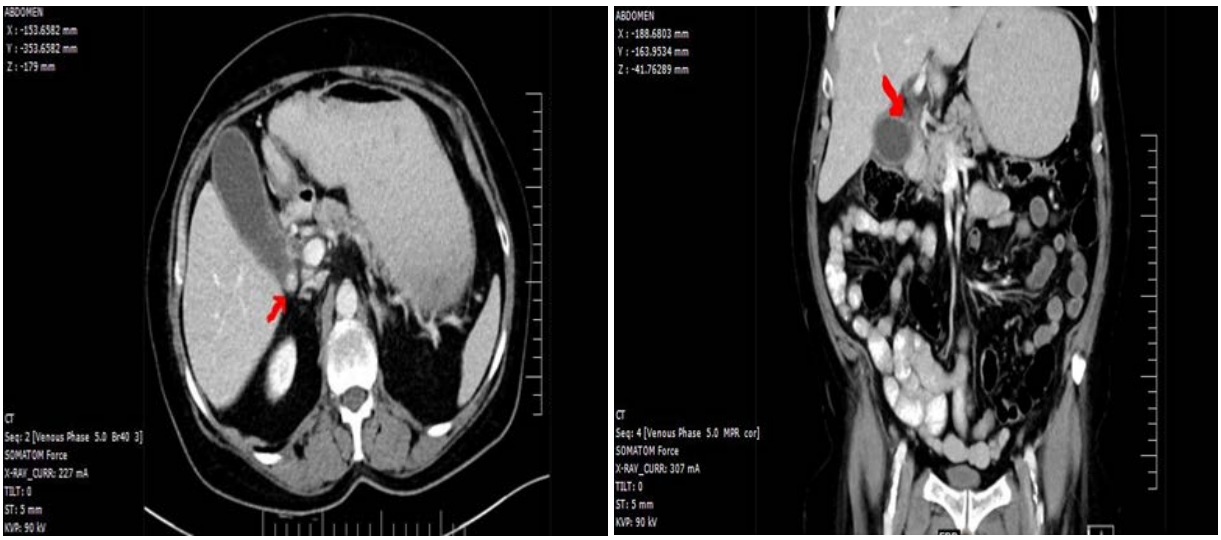

Fig 1

(Red arrow - Impacted calculus at the junction of the gall bladder neck and proximal cystic duct)

In view of acute cholecystitis, he was managed conservatively with intravenous antibiotics and analgesics. On the 4th day of admission, the patient had a repeat fever spike of 103'F associated with chills and rigors. Blood culture were sent which grew gram negative bacilli. Antibiotics were changed to cover a broader spectrum of micro-organisms. However, on day 6, patient had new onset sudden, severe epigastric discomfort along with high grade fever and tachycardia. Abdominal examination at this time revealed no tenderness, guarding or distension. Hematological parameters showed leukocytosis (23,000 x 10^9/L) with CRP increased to 29.4 mg/dl. Liver function tests showed direct hyperbilirubinemia with raised gamma glutamyl transferase and alkaline phosphatase levels.

Repeat CT scan of the abdomen was done which showed further distension of gall bladder with irregularity along the superior gall bladder wall with reactive mural thickening of the common hepatic duct extending into the primary hepatic confluence. It also showed a thrombotic occlusion of the entire length of the left portal vein and its intrahepatic segmental branches resulting in perfusional anomaly in left lobe of liver. The main portal vein and the right portal vein were patent with good luminal contrastation in right lobar segmental branches.

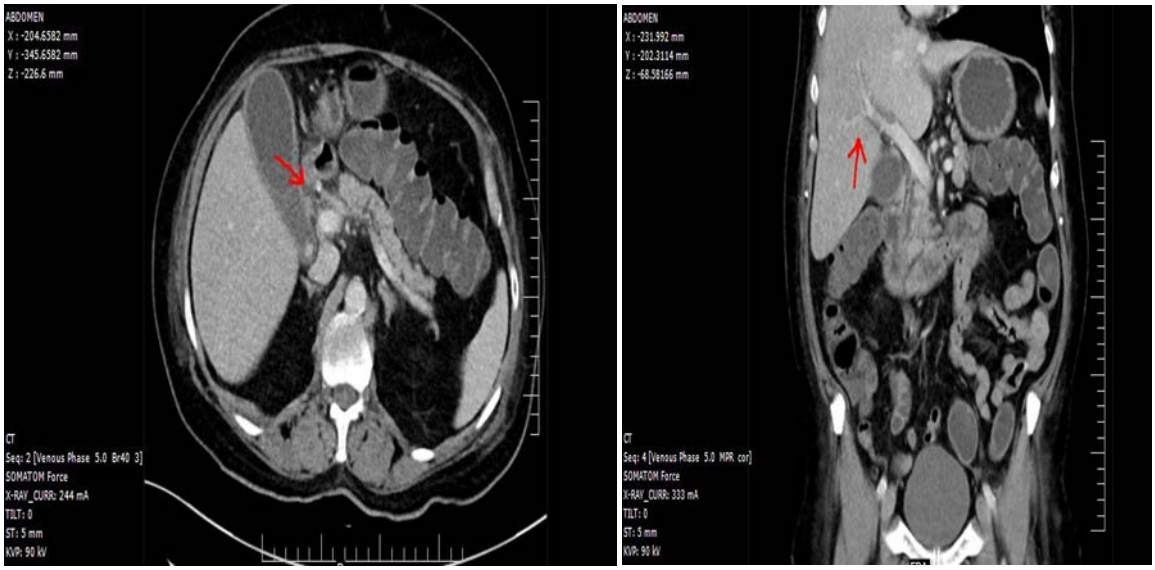

Fig 2

(Distended gall bladder with reactive mural thickening of the common hepatic duct extending into the primary hepatic confluence. Red arrow showing thrombotic occlusion of the entire length of the left portal vein and its intrahepatic segmental branches.)

In view of above scan findings, patient was posted for surgical exploration. Diagnostic laparoscopy revealed acutely inflamed, thickened intrahepatic gall bladder with localized collection. Meticulous dissection was done and laparoscopic cholecystectomy accomplished. Post –operatively, low molecular weight heparin was started after 24 hrs. of surgery and were changed to oral coumarin anticoagulants at time of discharge.

Prior to discharge, doppler portal vein was done which showed a heterogenous content, consistent with sub-acute thrombosis in the left branch of portal vein. Patient was put on anticoagulants for 3 months and repeat doppler of portal venous system revealed recanalization of the left portal vein with good hepatopetal flow. Oral anticoagulants were then stopped on advice of hematologist. At 12 months of follow up, patient was asymptomatic.

Discussion

The overall prevalence of Gall Stone disease in most developed nations, is between 10% and 20% [1]. The prevalence increases with age in both males and females. At the age of 65, about 30% of women have Gall Stones, and by the age of 80 years, 60% of both males and females have Gall Stones. The large majority of these (70–85%) are asymptomatic [2] but they can occasionally obstruct the cystic duct resulting in increased pressure within the gallbladder. As a consequence, bile is being forced across the mucosal membrane in causing an acute chemical inflammatory reaction. Transient obstruction precipitates acute biliary pain (biliary colic) whereas persistent obstruction can lead to acute cholecystitis or its subsequent complications. Bacteria predominantly Escherichia coli, Klebsiella, Enterobacter, and Bacteroides species [3] are cultured from the bile in approximately one-half of patients with gallstones, and unrelieved obstruction in the presence of this infected bile may produce an empyema.

The persistently obstructed gallbladder becomes intensely inflamed and oedematous, which cause an increase in transmural pressure in the gallbladder wall. It can result in venous ischaemia, leading to gangrene and or perforation. Perforation may be contained by the liver or surrounding viscera leading to localised abscess formation, like in our case or may result in biliary peritonitis.

The association of portal vein thrombosis with cholecystitis is considered relatively rare and has usually been published as case reports, fourteen case as per literature according to Muneer M. *et al* [4].

The pathogenesis of portal vein thrombosis is multifactorial and is usually associated with pancreatic or liver malignancies with a frequency of 21 to 24% [5]. Local thrombotic risk factors commonly observed are cholecystitis, cholangitis, pancreatitis, appendicitis and splenectomy [5].

The most commonly observed symptoms of acute portal vein thrombosis are abdominal pain, nausea, vomiting, diarrhea, and constipation [6] with nonspecific clinical signs are present such as abdominal pain and distension, like in our case. They often are associated with high grade fever and shock.

Presentation is different in different patients depending upon the site of thrombosis, acute or chronic, and collateral vein development. The consequences of portal vein thrombosis are related to the extension of the thrombus. Upstream (towards liver) from the thrombus, there is little effect on the intestine as long as the mesenteric venous arches remain patent. Ischemia results from extension of the thrombus into the mesenteric veins and the mesenteric venous arches [7] there by preventing collateral circulation. Alternatively, reflex arteriolar vasoconstriction might occur when the arches are thrombosed [8]. When ischemia is prolonged for several days, intestinal infarction may follow. In 20-50% of the cases, intestinal infarction is responsible for death due to peritonitis and multiple organ failure, even when resection of the infarcted gut is carried out.

Portal vein thrombosis post cholecystitis is rare and only a few cases have been reported in literature. This can be attributed to the subclinical course of the disease, which is usually detected incidentally during radiological examination. Previous authors observed no specific etiological factors associated with portal vein thrombosis and speculated that occurrence of portal vein thrombosis post cholecystitis could be due to inflammation or infectious process involving the cystic vein. Septic portal vein thrombosis, the so-called septic pylephlebitis, is usually related to appendicitis or diverticulitis. It is so strongly associated to Bacteroides bacteremia that Bacteroides bacteremia of unknown origin should prompt the search for portal or mesenteric vein thrombosis [8]. As suggested by F. Arthur *et al* [9], initial triggering factor for portal vein thrombosis in our case could be the intense inflammatory response caused by the stone in the cystic duct, which sits in close proximity to the draining cystic veins in Calot's triangle [8]. Cystic veins eventually drain into the right portal vein branch through which the thrombosis can be propagated into left portal vein, as in our case.

The aim of treatment of PVT is to prevent thrombus expansion and to attain portal vein patency. The treatment approach includes management of offending factor, use of antibiotics, hydration, anticoagulant therapy, and occasionally thrombolytic therapy or surgical embolectomy [10]. Time interval between thrombus formation and start of anticoagulant treatment dictates outcome of recanalization.

In our case, the offending cause was removed surgically with laparoscopic cholecystectomy and was started on anticoagulants immediately after surgery which resulted in improvement in patient condition with recanalization of portal vein on follow up.

Conclusion

Portal vein thrombosis is a rare complication of acute cholecystitis. Early recognition of this complication of cholecystitis with appropriate management can improve outcomes and shorten hospital stay and prevent life threatening complications like mesenteric and hepatic ischemia.

Cconservative treatment could be a feasible and safe approach for the treatment of portal venous thrombosis with acute cholecystitis, if treated at an early stage. The treatment duration is usually 6 months if a solely transient local cause is identified, but may have to be extended in absence of an identifiable local cause and/or identification of a persisting systemic cause [11]. The diagnosis of this condition requires a high index of suspicion and early management of underlying etiology to achieve better outcomes.

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