Medical Sciences (CIOMS).[6] DILI generally occurs between 5 and 90 days after drug ingestion.[6] R value in our patient was >5 suggesting hepatocellular type of injury. Liver injury is likely to be more severe in hepatocellular type than in cholestatic/ mixed type. Patients with serious liver injury have a mortality rate ranging from 0.7 to 1.3/100 000 individuals receiving a given drug.[6]

Previous studies have shown albendazole-induced hepatotoxicity was mixed type.[3,4] In the largest published series of prolonged albendazole use in hydatid disease, incidence of hepatotoxicity was 5%; the dose used was 400 mg twice a day for 28 days.[3] In an earlier report of recurrent hepatitis due to albendazole in an adult, the liver biopsy showed periportal invasion of inflammatory cells, cytotoxic necrosis and varying degree of steatosis.[7] In our patient, hepatitis occurred after a single dose of albendazole; in all the previously reported cases, except in one case,[7] the patients had received the drug over several days before they developed evidence of hepatic injury.

The exact mechanism of albendazole-induced hepatitis is not known. Our patient had symptom onset within 7 days of drug ingestion, hence a possibility of immune-mediated injury could be considered. The possible antigen may be a component of the benzimidazole group, possibly the sulphur group. Another possibility may be a single nucleotide polymorphism involving CYP3A4 enzyme, which can lead to idiosyncratic drug reaction.

In conclusion, albendazole-induced hepatitis can occur even after a single dose of the drug. Awareness about this condition is essential because anti-parasitic medication is taken frequently, often empirically.

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Chronic pancreatitis causing thrombotic occlusion of IVC and renal veins

Introduction

Pancreatitis is an inflammatory disease of pancreas which may lead to a variety of vascular complications. Common complications are hemorrhage into a pseudocyst, erosion of arteries adjacent to inflamed pancreas leading to frank intraperitoneal bleed or pseudoaneurysm formation and thrombosis of the portal venous system.[1] Inferior vena cava (IVC) thrombosis is a very rare condition associated with pancreatitis and only few case reports are there in the literature describing this condition.[2-5] Here we report a case of chronic pancreatitis having IVC thrombus extending into bilateral renal veins.

Case Report

A 35 year old man, known alcoholic, with previous episode of acute pancreatitis 5 years back presented with acute abdomen to the casualty. The patient gave history of similar episodes in the past. On examination, abdomen was distended and tender with presence of guarding and rigidity. He had mild tachycardia but blood pressure was normal. Blood examination revealed mildly elevated serum amylase level. Based on history and clinical findings diagnosis of acute on chronic pancreatitis was made and contrast enhanced CT scan of abdomen was advised.

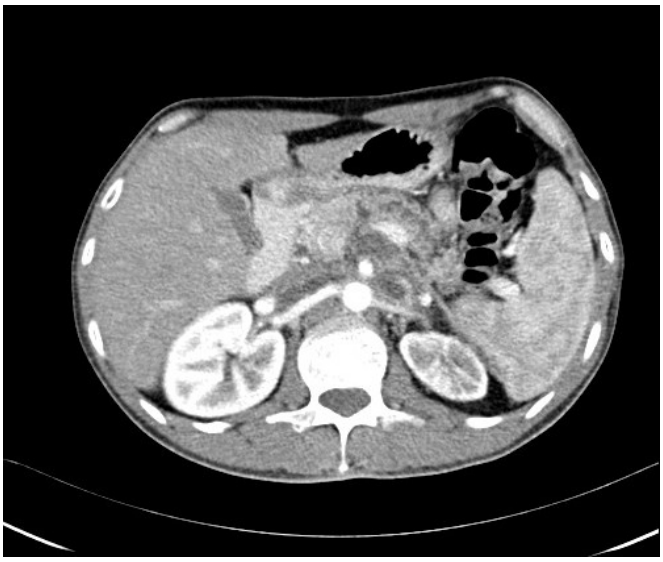

Figure 1: CT image showing mildly atrophic pancreas with dilated main pancreatic duct

CT showed pancreas to be mildly atrophic with dilated main pancreatic duct (**Figure 1**) consistent with chronic pancreatitis. No parenchymal calcification or intraductal calculi were seen. Unusual finding noted was a large intraluminal thrombus within the IVC extending from the level of renal hilum upto the intrahepatic IVC (**Figure 2,3**). Thrombus was also seen extending into bilateral renal veins (**Figure 2**). Celiac artery and its branches were normal in course and caliber. Spleno-portal axis was also normal. Based on imaging, diagnosis of chronic pancreatitis with large IVC thrombus extending into bilateral renal veins was made.

Discussion

Vascular complications in pancreatitis are well documented. Nearly one fourth of patients having pancreatitis may develop vascular complications. Hemorrhage being one of the most common and most dreaded complications associated with it.[6] Formation of pseudoaneurysms secondary to pancreatitis is well known.[7] Pseudoaneurysms form due to adjacent inflammation necrotizing the vessel wall or due to release of pancreatic enzymes eroding the vessel. Another vascular complication associated with pancreatitis is thrombosis of spleno-portal axis. However splenic vein thrombosis is more common and may occur in about 10-40% of patients with chronic pancreatitis.[8]

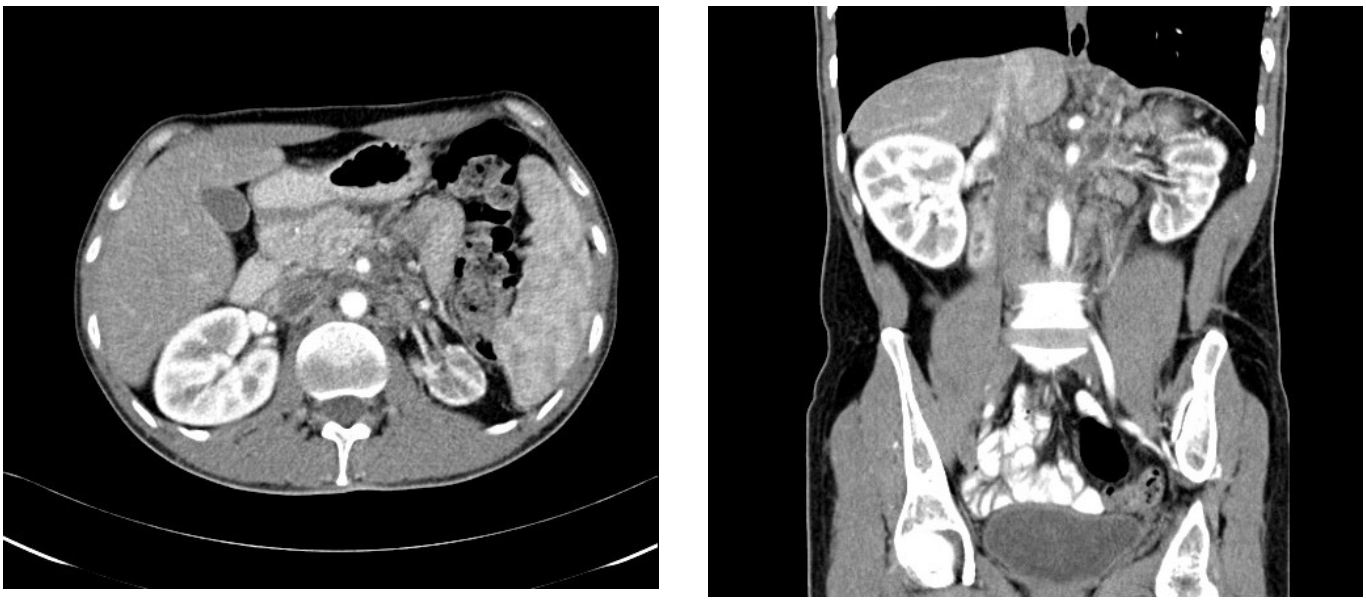

Figure 2: (a) Axial CT image and (b) coronal reformatted image, showing large IVC thrombus extending into bilateral renal veins

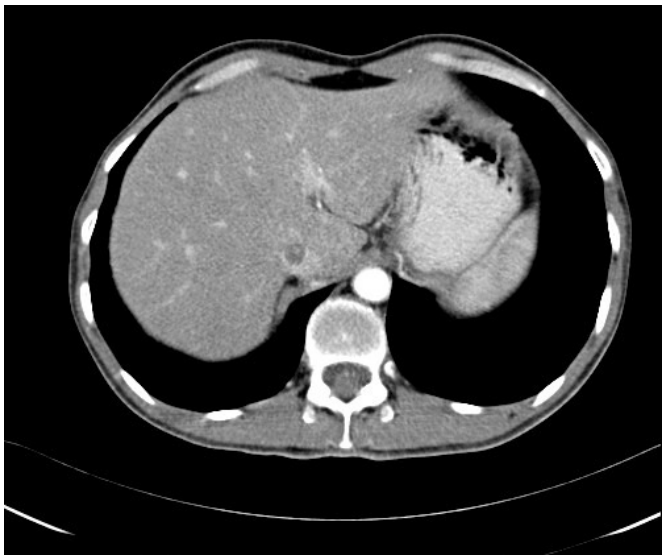

Figure 3: Thrombus seen within intrahepatic IVC

IVC thrombosis is commonly seen as an extension of deep vein thrombosis (DVT). However, variety of causes may lead to this condition. Important ones are renal cell carcinoma (RCC) and retroperitoneal tumors. Extrinsic compression may also lead to this condition, like compression produced by

aneurysms, abscesses, cysts, hematoma and or any dysfunction in coagulation system. Urgent management is required for this condition as it may further lead to pulmonary embolism. Standard treatment for DVT and IVC thrombosis remains systemic anticoagulation, however mechanical interruption of the pathway may be offered to patients who have contra-indication for anticoagulation by placing an IVC filters.

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Synchronous colorectal cancer with aplastic anemia

Introduction

Long-term survivors of patients with aplastic anemia may be at high risk for malignant disease. The most common amongst these are myelodysplastic syndromes and acute leukemia. They tend to occur following immunosuppressive therapy or bone marrow transplantation in patients with aplastic anemia. There are few published reports of metachronous solid organ malignancy in survivors of aplastic anemia who have received immunosuppressive therapy or bone marrow transplantation. The most common therapy-related solid organ malignancies that occur in aplastic anemia are head and neck cancers, hepatocellular carcinoma, breast cancer and colorectal cancer. Synchronous solid organ malignancy with aplastic anemia has not been reported in the literature. We report a case of colorectal adenocarcinoma with synchronous aplastic anemia and review the literature concerning the association of aplastic anemia with colorectal cancer.

Case Report

A 32-year-old man, presented with a 2-month history of bleeding per rectum. The patient gave no significant medical history, history of drug intake or any previous hospitalization. There was no family history of hematological illness or malignancy. Rectal examination revealed a circumferential ulcerative growth in the distal rectum, 3 cm from the anal verge up to 7 cm. Punch biopsy of the ulcerative growth revealed well-differentiated adenocarcinoma (**Figure 1**). On laboratory evaluation his hemoglobin was 5.5 g%, total leucocyte count

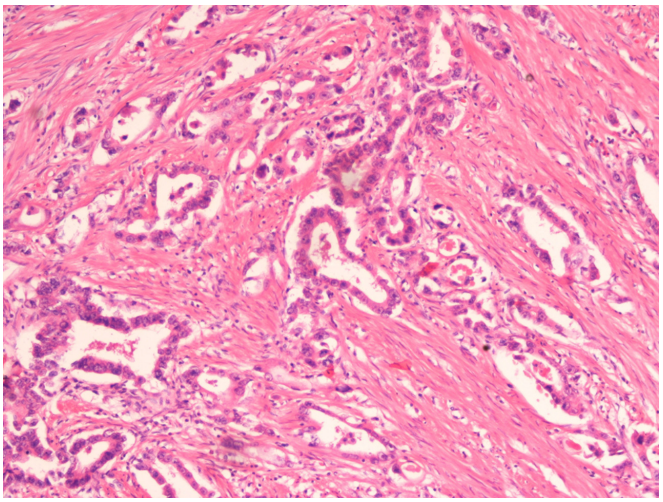

Figure 1: Represents the histopathological picture of colorectal cancer which shows adenocarcinoma cells (on hematoxylin and eosin stain) infiltrating the muscularis propria of the gut wall