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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References

1. Mallick IH, Winslet MC. Vascular complications of pancreatitis. *JOP*. 2004;**5**:328–37.
2. Ma SK, Kim SW, Kim NH, Choi KC. Renal vein and inferior vena cava thrombosis associated with acute pancreatitis. *Nephron*. 2002;**92**:475–7.
3. Ohta H, Hachiya T. A case of inferior vena cava thrombosis and pulmonary embolism secondary to acute exacerbation of chronic pancreatitis: a rare finding in radionuclide venography. *Ann Nucl Med*. 2002;**16**:147–9.
4. Jones AL, Ojar D, Redhead D, Proudfoot AT. Case report: Use of an IVC filter in the management of IVC thrombosis occurring as a complication of acute pancreatitis. *Clin Radiol*. 1998;**53**:462–4.
5. Ohno T, Takabayashi A, Maki A, Usui Y, Takeuchi K, Kohno S. Complete obstruction of the inferior vena cava due to chronic relapsing pancreatitis: a case report. *Nippon Geka Hokan*. 1994;**63**:215–20.
6. Vujic I. Vascular complications of pancreatitis. *Radiol Clin North Am*. 1989;**27**:81–91.
7. Carr JA, Cho JS, Shepard AD, Nypaver TJ, Reddy DJ. Visceral pseudoaneurysms due to pancreatic pseudocysts: rare but lethal complications of pancreatitis. *J Vasc Surg*. 2000;**32**:722–30.
8. Bernades P, Baetz A, Levy P, Belghiti J, Menu Y, Fekete F. Splenic and portal venous obstruction in chronic pancreatitis. A prospective longitudinal study of a medical-surgical series of 266 patients. *Dig Dis Sci*. 1992;**37**:340–6.

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

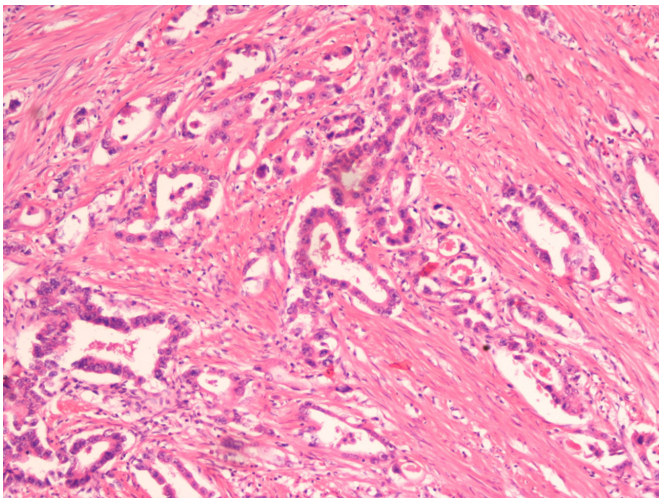

Figure 1: Represents the histopathological picture of colorectal cancer which shows adenocarcinoma cells (on hematoxylin and eosin stain) infiltrating the muscularis propria of the gut wall

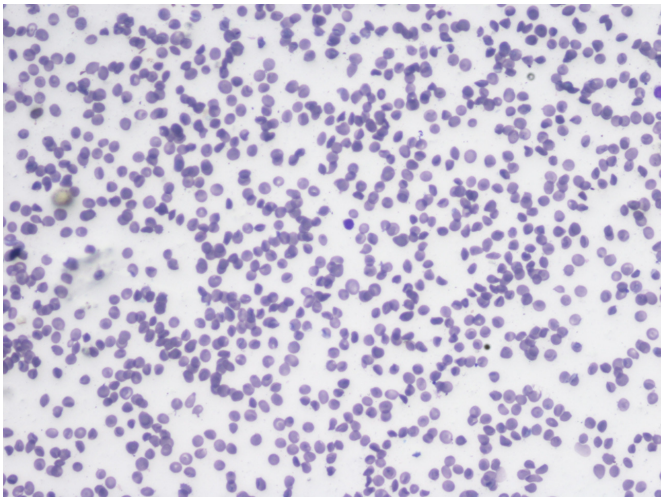

Figure 2: Represents the peripheral blood smear showing pancytopenia

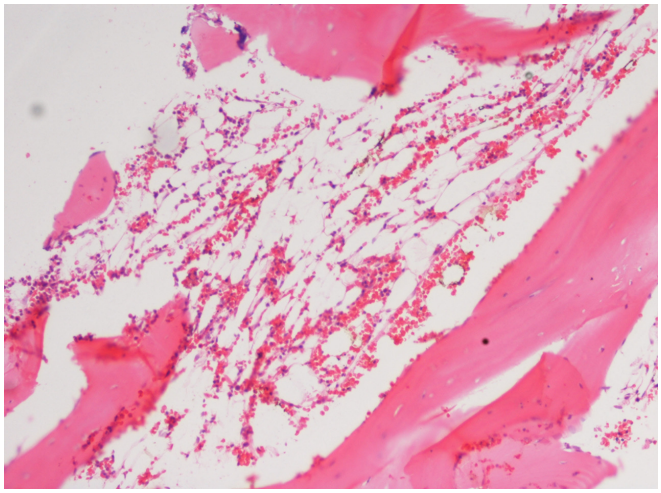

Figure 3: Represents the bone marrow picture showing mainly fatty marrow spaces and scant elements composed of stromal cells, lymphocytes and plasma cells (X100)

was 2400/mm^3, and platelet count was 30,000/mm^3. Peripheral smear described pancytopenia (**Figure 2**). Bone marrow biopsy suggested aplastic anemia (**Figure 3**). Computerized tomography showed a mass in the anorectal junction with no perirectal spread or nodes. There was no liver metastasis or ascites. He received preoperative short course radiotherapy of 25 Grays in 5 fractions followed by abdominoperineal resection. The patient's hemogram and platelet count normalized on transfusion of packed red cell and single donor platelet transfusion. The post-operative period was uneventful and the patient was discharged from the hospital 7 days later.

Discussion

Correlation of solid organ malignancy and hematological disease has rarely been reported in the literature. In the 2 published studies, on gastrointestinal malignancy, in patients with aplastic anemia, only sixteen patients have been reported.[1-3] In a review by Socie et al, 860 and 748 patients of aplastic anemia received immuosuppressive therapy and bone marrow transplantation respectively, 7 patients in each group had solid organ malignancy.[1] In this review six developed head and neck epidermoid carcinoma, 2 stomach adenocarcinoma, 4 hepatocellular carcinoma and 2 breast carcinoma. None of them had colorectal malignancy. The 10 year cumulative risks of developing solid organ cancers were 18.8% and 3.1% in patients who received immunosuppressive therapy and bone marrow transplantation respectively.[1]

A recent retrospective review by Kishida et al showed 14 colorectal cancer patients out of 734 patients with hematological disease.[2] In his review, the highest incidence of colorectal cancer was observed in patients with pure red cell aplasia (18.8%), followed by multiple myeloma (8%) and aplastic anemia (6.5%). Three of 46 patients (6.5%) with aplastic anemia had colorectal adenocarcinoma diagnosed on colonoscopy. All the three patients with colorectal malignancy died of aplastic anemia.[2]

Synchronous colorectal cancer in patients of aplastic anemia has not been reported in the literature. In this case the patient had an incidental aplastic anemia that was discovered during the work up for colorectal cancer. The pathophysiology behind the development of adenocarcinoma in patients with aplastic anemia has not been well studied.[4] Large-scale studies and reporting are needed to study the relationship between aplastic anemia and colorectal cancer.

In summary, survivors of aplastic anemia are at risk for development of solid organ malignancy following immunosuppressive therapy or bone marrow transplantation. Patients with aplastic anemia may present with synchronous colorectal adenocarcinoma even before receiving immnosuppressive treatment.

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References

1. Socié G, Henry-Amar M, Bacigalupo A, Hows J, Tichelli A, Ljungman P, et al. Malignant tumors occurring after treatment of aplastic anemia. *N Engl J Med*. 1993;**329**:1152–7.
2. Socié G, Henry-Amar M, Cosset JM, Devergie A, Girinsky T, Gluckman E. Increased incidence of solid malignant tumors after bone marrow transplantation for severe aplastic anemia. *Blood*. 1991;**78**:277–9.
3. Kishida T, Yonezawa M, Shibata Y, Tanaka S, Shinozawa I, Hoshino T, et al. Risk of colorectal cancer in patients with hematologic disease. *J Gastroenterol Hepatol*. 2000;**15**:1272–6.
4. Brodsky RA, Jones RJ. Aplastic anaemia. *Lancet*. 2005;**365**:1647–56.

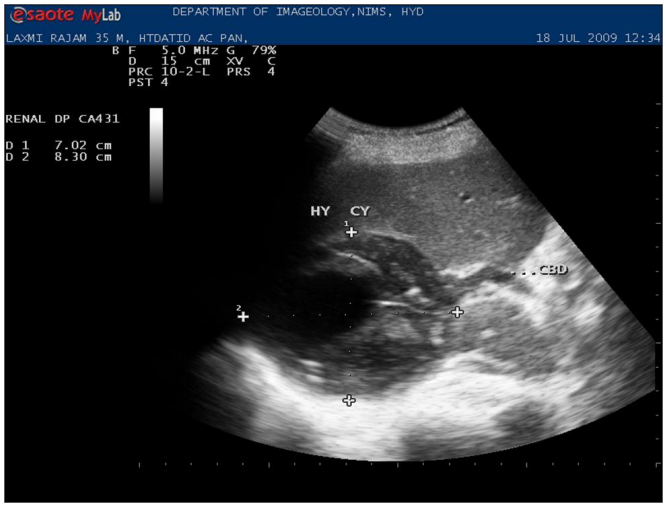

Figure 1: USG showing Hydatid cyst communicating with CBD with echogenic contents in CBD.

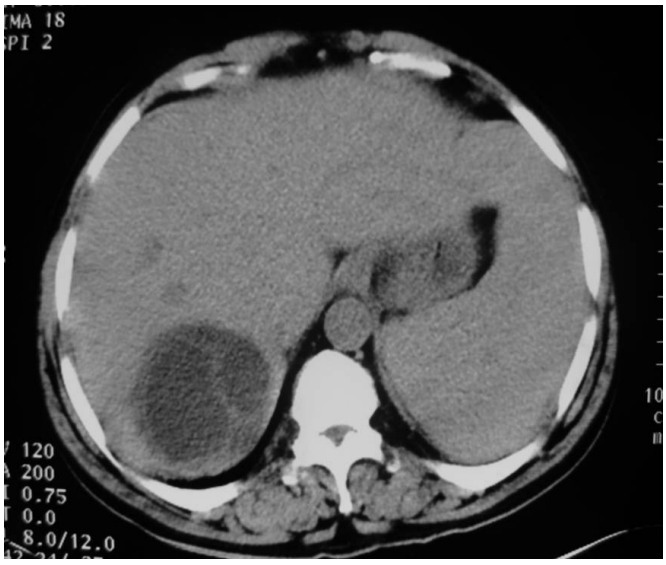

Figure 2: CT abdomen showing hydatid cyst in the liver with daughter cysts.

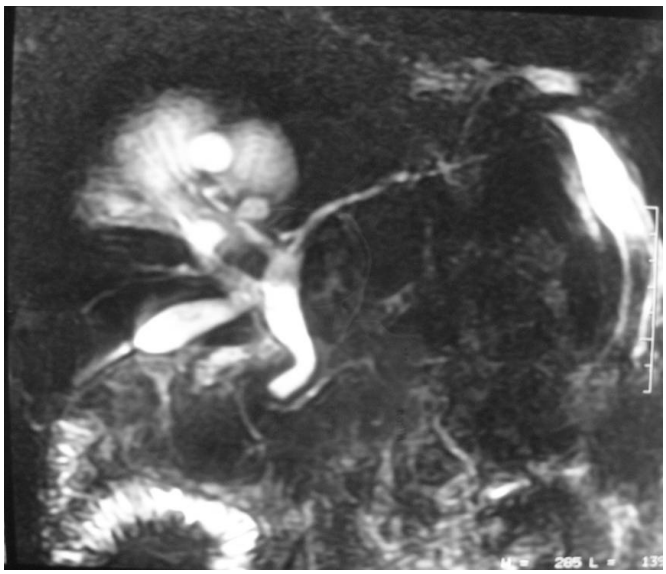

Figure 3: MRCP showing dilated CBD with echogenic contents & hydatid cyst in liver communicating with biliary tree.

Intrabiliary rupture of hepatic hydatid cyst with impacted hydatid membranes at ampulla of Vater presenting as acute pancreatitis

Introduction

Hydatid disease is endemic in Southern Australia. New Zealand, Africa, South America, Southern Europe, Middle East and the Far East.[1] Intrabiliary rupture is reportedly seen in 6-17% cases.[2-4] But rupture causing pancreatitis has been very rarely reported.

We report a case of acute pancreatitis due to impacted hydatid membranes at the ampulla secondary to rupture of hepatic hydatid cyst into the biliary tract, which was managed successfully with endoscopic sphincterotomy, basketing of membranes and biliary stenting.

Case Report

A 35-year old man was referred to our emergency room with pain abdomen of 1-week duration. The pain was located in the epigastric region, boring to the back, and was associated with nausea and vomiting, jaundice associated with itching, and fever with chills. He also suffered mild pain in the right upper quadrant for the 1 month prior to presentation for which he was self medicating.

His history was positive for significant alcohol consumption over 6 years. On examination he was icteric and per abdomen examination showed severe tenderness and guarding in the epigastric region with tender hepatomegaly. Laboratory data