Acute Budd-Chiari Syndrome: A Unique and Severe Complication of Crohn's Disease

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INTRODUCTION

Crohn's disease (CD) is an inflammatory condition of the intestines that can affect any part of the gastrointestinal tract. Additionally, it may cause systemic inflammation in other organs, including the joints, eyes, liver, skin, and blood system. Treatment can address rare arterial and venous thrombosis, such as those in the hepatic and intracapsular veins.^{1,2} This article presents a case of Budd-Chiari syndrome (BCS) in a patient with CD, who also developed hepatic vein thrombosis (HVT). HVT was an unusual complication that significantly contributed to the progression of cirrhosis but did not respond to treatment.

CASE REPORT

A 33-year-old man was diagnosed with Crohn's disease during a colonoscopy. He was referred to the hospital for appropriate management of his condition. Subsequent liver function tests and an abdominal computed tomography (CT) scan with contrast revealed acute BCS, characterized by the new onset of abdominal ascites secondary to ischemic events. After an unsuccessful attempt at interventional radiology, treatment with low molecular weight heparin (LMWH) at a dosage of 0.6 IU subcutaneously twice daily was initiated.

INVESTIGATIONS

The following values were obtained: Urea: 28 mg/dL, Creatinine: 0.87 mg/dL, Aspartate aminotransferase: 728 U/L, Alanine aminotransferase: 639 U/L, Alkaline phosphatase: 122 U/L, Gamma-glutamyl transferase: 76 U/L, C-reactive protein: 25 mg/dL, White blood cell count: 10.7 x 10^9/L, Neutrophils: 6.7 x 10^9/L, Hemoglobin: 16.2 g/dL, Platelets: 457 x 10^9/L, Albumin: 38 g/dL, and Lactate Dehydrogenase. An acute case of BCS associated with ischemic events leading to abdominal ascites was identified. This patient had a serum alanine aminotransferase level of 866 U/L. Diagnosis was supported by contrast enzyme assay and abdominal CT. Thrombophilia tests were negative, ruling out other causes. After unsuccessful intervention by a radiologist, the patient was treated with subcutaneous LMWH at 0.6 IU twice daily.

The patient continued follow-up care, confirming that the BCS presentation correlated with CD. The diagnosis was further validated by a colonoscopy and double-balloon enteroscopy conducted at an external facility five years prior.

DISCUSSION

Inflammatory bowel disease (IBD) is associated with a variety of complications, including the risk of venous thromboembolic events, liver disease, cancer, and bone disease.³ Hallmarks of IBD include systemic venous thrombosis and focal micro-thrombi in the vasculature of the inflamed colon. The pathophysiology of IBD involves a combination of factors. Portal vein thrombosis, significantly affecting the gastrointestinal tract, may complicate the condition.⁴ Symptoms such as ascites, abdominal discomfort, and hepatomegaly are common. Patients with IBD also exhibit a higher incidence of venous thromboembolism, potentially due to proinflammatory cytokines counteracting natural anticoagulants and creating a hypercoagulable state. Additionally, IBD can lead to BCS.

In the case discussed, the patient had active CD and a family history of myeloproliferative disorders. Despite unsuccessful interventional radiology attempts, treatment with LMWH was initiated. Following a decline in liver function observed during follow-up, preparations for liver transplantation began.

BCS results from the obstruction of hepatic venous outflow, leading to necrosis, congestion, fibrosis, and progressive liver cirrhosis. In some cases, liver function may deteriorate rapidly, leading to acute liver failure.⁵⁻⁷ This condition, evidenced by elevated liver function tests and extended prothrombin times requiring intensive care, necessitates further investigation as other causes have been excluded.

In conclusion, BCS is a rare but critical condition in the context of IBD with thrombophilia, abdominal pain, and abnormal liver function tests, requiring aggressive treatment.

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