BUDD-CHIARI SYNDROME: A RARE AND LIFE-TREATHENING COMPLICATION OF CROHN’S DISEASE
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BACKGROUND

• Budd-Chiari Syndrome (BCS) is characterized by obstruction of hepatic venous outflow and may be triggered by the pro-thrombotic state that occurs with inflammatory bowel disease (IBD), most commonly ulcerative colitis.

• Crohn’s disease (CD) as a precipitant of BCS, although recognized, is extremely rare.

• To our knowledge, only eight papers reporting the association between BCS and CD have been published in the English literature to date.

CASE REPORT

• Our patient is a 27 year old male who reported intermittent hematochezia, diarrhea and abdominal pain for a year. He underwent colonoscopy in an outside hospital one year prior to presentation, which was suggestive of IBD, but was lost to follow up and remained untreated.

• At admission, cachexia, anasarca with ascites and tender hepatomegaly were noted. Severe anemia, leukocytosis, elevated liver enzymes and prothrombin time, and low albumin were present.

• Stool studies ruled out gastrointestinal infection. Paracentesis showed no evidence of spontaneous bacterial peritonitis and there was evidence of portal hypertension. Esophagogastroduodenoscopy & colonoscopy revealed gastrroduodenitis and pancolitis, respectively.

• Abdominal CT scan showed evidence of venous thrombosis with hepatomegaly.

• Liver biopsy (LBx) revealed severe perivenular sinusoidal dilation with areas of confluent hepatocyte dropout, bands of hepatocyte atrophy, and centrilobular fibrosis, consistent with chronic outflow obstruction/BCS.

RESULTS

• Our patient was transfused and hemodynamically stabilized.

• His thrombosis was treated with heparin infusion and he was transitioned to apixaban.

• His anasarca improved with diuretics and CD was treated with prednisone and mesalamine.

• The hematochezia resolved and diarrhea, anemia, and volume status improved.

• CT scan of the abdomen post treatment showed resolution of his inferior vena cava thrombus.

### Table: Blood tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Pre-Tx</th>
<th>Post-Tx (6mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hgb (g/dl)</td>
<td>5.1</td>
<td>8.6</td>
</tr>
<tr>
<td>PT (sec.)</td>
<td>18.6</td>
<td>16.8</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>2.2</td>
<td>4.3</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>83</td>
<td>25</td>
</tr>
<tr>
<td>AST(U/L)</td>
<td>81</td>
<td>20</td>
</tr>
<tr>
<td>ALK PHOS (U/L)</td>
<td>138</td>
<td>88</td>
</tr>
</tbody>
</table>

DISCUSSION

• BCS associated with CD is a rare event.

• The precise mechanism responsible for increased risk of thromboembolism in IBD remains unclear and is likely multifactorial.

• Notably, several studies have demonstrated that proinflammatory cytokines can counteract natural anticoagulant activity leading to a hypercoagulable state.

• Anticoagulants are mainstays of therapy, even in the setting of gastrointestinal bleeding.

• LBx can be diagnostic in some acute and subacute cases, such as ours.

• Early recognition and immediate treatment of thrombotic complications of CD is critical to prevent hepatocyte injury, liver failure, and fatal complications like pulmonary embolism or liver failure.

REFERENCES


