

## A Case Study on Budd Chiari Syndrome

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### Abstract

Budd-Chiari syndrome is a rare but life-threatening condition that requires rapid diagnosis and appropriate treatment. It is an uncommon condition induced by thrombotic or non thrombotic obstruction to hepatic venous outflow. It occurs in 1 out of 100,000 individuals and is more common in females. Some 10–20% also has obstruction of the portal vein. Budd-Chiari syndrome is a potentially fatal disorder, if untreated. The syndrome occurs in persons of all races. I present a rare case of Budd-Chiari syndrome in a male with mild abdominal pain since one year. Early recognition of Budd-Chiari syndrome is important in preventing possible morbidity and mortality for this patient.

**Keywords:** Budd-Chiari syndrome; Hepatomegaly; Ascites; Endophlebitis.

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### Introduction

Budd-Chiari syndrome is an uncommon condition induced by thrombotic or nonthrombotic obstruction to hepatic venous outflow. Budd described it in 1845, and Chiari added the first pathologic description of a liver with “obliterating endophlebitis of the hepatic veins” in 1899. Hepatomegaly, ascites, and abdominal pain characterize Budd-Chiari syndrome. It occurs in 1 out of 100,000 individuals and is more common in females. It is a potentially fatal disorder, if untreated. The syndrome occurs in persons of all races. The syndrome is equally present in both sexes.<sup>1-7</sup>

Here, I present a case of Budd-Chiari syndrome in a male with mild abdominal pain since one year. Early recognition of Budd-Chiari syndrome is important in preventing possible morbidity and mortality for this patient.

### Case Report

Mr. X 43-year-old male came with the complaints of gradually progressive abdominal distension with pain since 8–10 days. After taking meals his abdominal pain is severe for 1–2 hours. He had a Past history of Mild abdominal pain since 1 year on & off which was usually subsided by taking medicines. On presentation he was conscious, oriented, afebrile, Pulse: 88/min., Respiration: 24/min, BP: 130/80 mm of Hg.

On observation his abdomen is tender, spleen palpable and mild ascitis. Laboratory studies revealed low haemoglobin (8.0 gm %), decreased RBCs (2.7 million/cmm), decreased platelet (1.0 lac), decreased total leucocyte count (2,400/cmm), PTINR is raised (1.24) and hypoalbuminemia (2.6 g/dL). His colour doppler of upper abdomen showed

- Mild hepatomegaly with 2 small echogenic nodular in left lobe ? Angiomas.

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- Abrupt narrowing of hepatic veins and IVC at their confluence? Web? Stricture.
- Mild secondary portal hypertension.

And Gastroduodenos copy report revealed Grade II esophageal varices which confirm the diagnosis of Budd-Chiari syndrome.

The etiology in patient was Secondary (25%): compression of the hepatic vein by an outside structure (e.g. a tumor); two small echogenic nodular in left lobe and narrowing of hepatic veins and inferior vena cava at their confluence found in color Doppler.

He was showed the sign and symptoms of mild hepatomegaly, hypersplenism, abdominal distension, abdominal pain, mild ascitis and mild portal hypertension.

Medications given as prescribed by the consultant Inj. Zostum 1 gm 12 hrly. IV; Inj. Kaplin 10 mg OD IV; Inj. Pantodac 40 mg OD IV; Inj. Tramazac 2cc in

each pint IV and Inj. Dolonex 2cc IM OD.

Patient is posted for spleenectomy with central lienorenal shunt. Intravenous fluid is started 10% dextrose 2 pints with Inj. MVI (multivitamin) 10 cc, Inj. Tramazac 1cc IM, Tab. Aciloc 150 mg BD. Postoperative orders are carried out that includes-Nasal oxygen 4-6 litre/min., nil by mouth till further order, ryles tube aspiration continuously and strict Total Parenteral Nutrition.

On 1<sup>st</sup> postoperative Day, ryles tube was removed and sips orally started. ICD was 100 cc. IInd postoperative day, ICD was removed. Liquids orally started. IIIrd postoperative Day, patient was ambulated. And on IVrth postoperative Day, Foley's catheter is removed and patient was discharged. Hence, prognosis is good.

He had several complications of Low platelet count, Grade II esophageal varices, Portal hypertension and Prothrombin time (PTINR) is raised.

### *Nursing Care Plan*

Nursing Diagnosis	Goal	Interventions
Pain and discomfort related to distended abdomen and ascitis.	Increased level of comfort.	<ul style="list-style-type: none"> <li>• Maintain bed rest when patient experiences abdominal discomfort.</li> <li>• Observe, record and report presence and character of pain and discomfort.</li> <li>• Administer analgesics or antispasmodic as prescribed.</li> </ul>
Increased risk for gastrointestinal bleeding and hemorrhage related to esophageal varices.	The patient will develop no episodes of G I bleeding and hemorrhage.	<ul style="list-style-type: none"> <li>• Assess patient for GI Bleed.</li> <li>• Avoid activities that increase intra abdominal pressure (straining and turning).</li> </ul>
Ineffective individual coping related to fear and anxiety before going for operation.	The patient will develop coping strategies and feel good.	<ul style="list-style-type: none"> <li>• Provide psychological support to the patient.</li> <li>• Give information's regarding the complications of the disease if not operate.</li> </ul>
Knowledge deficit related to home care management.	Verbalization of understanding of the home care remedies.	<ul style="list-style-type: none"> <li>• Advice to adhere completely to the therapeutic regimen.</li> <li>• Take a well balanced diet.</li> <li>• The patient and family are also instructed about the possibility of bleeding tendencies and easy susceptibility to infection.</li> </ul>

### **Discussion**

Mr. X is a case of Budd Chiari syndrome which shows symptoms of abdominal distention with pain since 8-10 days and signs of mild hepatomegaly, spleenomegaly, mild ascitis, portal hypertension and Grade II esophageal varices, treated surgically.

Classic clinical symptoms of Budd-Chiari syndrome include severe upper abdominal

pain, jaundice, hepatomegaly (enlarged liver), ascites, elevated liver enzymes and eventual encephalopathy.

The cause cannot be found in about half of the patients. The cause may be Secondary (25%): compression of the hepatic vein by an outside structure (e.g. a tumor), Congenital venous webs, Primary (75%): thrombosis of the hepatic vein, Pregnancy and oral contraceptive use and inferior vena caval stenosis.

A minority of patients can be treated medically with sodium restriction, diuretics to control ascites, anticoagulants such as heparin and warfarin, and general symptomatic management. Milder forms of Budd-Chiari may be treated with surgical shunts to divert blood flow around the obstruction or the liver itself. Patients with stenosis or vena caval obstruction may benefit from angioplasty. Liver transplantation is an effective treatment for Budd-Chiari. It is generally reserved for patients with fulminant hepatic failure, failure of shunts, or progression of cirrhosis that reduces the life expectancy to 1 year.

Several studies have attempted to predict the survival of patients with Budd-Chiari syndrome. In general, nearly 2/3 of patients with Budd-Chiari survive 10 years. The prognosis is poor in patients with Budd-Chiari syndrome who remain untreated.

- Pieter Martens in his Review Article: Budd-Chiari syndrome recommended that continued effort needs to be made in pooling patients and deciding which patients are good candidates for certain therapies. Efforts should be made in addressing the definition of disease progression under certain therapies.

## Conclusion

As the prognosis of Budd Chiari Syndrome is poor, if not treated in time. But if it is diagnosed early and treated surgically, prognosis will be good.

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