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Case Report

A Case Report on Decompensated Chronic Liver Disease with Budd–Chiari Syndrome

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ABSTRACT

The rare illness, known as Budd-Chiari Syndrome (BCS), is characterized by hepatic venous outflow tract obstruction and can arise from the right atrium, inferior vena cava, or the tiny hepatic veins within the liver. It is an uncommon liver condition in which obstructions, typically caused by blood clots, restrict the hepatic veins, preventing blood from exiting the liver and returning to the heart. As a result, the liver enlarges (hepatomegaly), accumulates fluid (ascites), and causes abdominal pain. We present a rare case of a young female diagnosed with decompensated chronic liver disease secondary to Budd–Chiari syndrome, who came with the features of abdominal distension and weight loss, along with a Model for end stage liver disease Na score of 20 and splenomegaly with portal hypertension. The diagnosis was confirmed by Doppler ultrasonography and contrast-enhanced CT of the abdomen, supported by abnormal laboratory parameters including prolonged prothrombin time with elevated INR, decreased serum albumin and total protein levels, and elevated WBC count, lymphocytes, GGT, and bilirubin. Management included anticoagulants, antibiotics, statins, proton pump inhibitors, β blockers, hepatoprotective agents, diuretics, and other supportive care, with a step-up approach planned for hepatic venous outflow restoration to prevent disease progression.

Keywords: Budd-Chairi syndrome, Ascities, Hepatomegaly, Liver, Endoluminal venous lesion.

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INTRODUCTION

Budd–Chiari syndrome (BCS) is a rare but severe form of hepatic vascular disease caused by the partial or complete obstruction of the hepatic venous outflow tract in the absence of constrictive pericarditis or right-sided heart failure. Available meta-analyses have shown that the incidence and prevalence of BCS are low in the normal population. The incidence of BCS is approximately 1 new case per million inhabitants annually (Gavriilidis *et al*., 2022). It predominantly affects women aged 19–49 years. The incidence of BCS has been reported to range from 0.2–4.1 per million annually. The prevalence of the disease has been estimated to range between 2.4 and 7.7 per million in Asian countries (Li *et al*., 2019; Porrello *et al*., 2023) and 1.4–4.0 per million in the Western population (Ki *et al*., 2016). Primary Budd–Chiari syndrome is caused by the obstruction of the hepatic venous outflow tract due to an endoluminal venous lesion, which is thrombosis or a venous web caused by a prothrombotic state or an unknown cause (Ollivier-Hourmand *et al*., 2018). Secondary Budd–Chiari syndrome is caused by extrinsic compression of the hepatic veins due to space-occupying lesions like cysts, abscesses, or benign or malignant tumors. It is also caused by the obstruction of the hepatic veins due to an invasive lesion like a malignant tumor or parasitic disease. Adults differ from pediatric patients in the higher prevalence of secondary causes. The pathophysiology of Budd–Chiari syndrome is the obstruction of the hepatic venous outflow. This causes hepatic congestion with increased sinusoidal pressure and hepatomegaly (Samanta *et al*., 2023). BCS is a potential diagnosis in many of the acute and chronic liver illnesses since clinical symptoms can vary widely, from abrupt liver failure to complete asymptomatic people. Only a tiny percentage of people with BCS present with a fulminant form of the disease, while most patients have a chronic presentation. The typical signs and symptoms include hepatomegaly, splenomegaly, ascites, GI bleeding, jaundice, and dilated abdominal veins. The presence of hepatic vein or Inferior Vena Cava (IVC) blockage is necessary for the diagnosis of BCS (Porrello *et al*., 2023). However, invasive venography is still the gold standard; it is carried out during an endovascular intervention technique (Sharma *et al*., 2016). As a consequence, Doppler ultrasound has become the primary imaging technique with a diagnostic accuracy rate higher than 90%. The technique assesses hepatic, portal, and inferior vena cava patency, determines the location and duration

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of vascular obstruction, and detects liver parenchymal changes that include intrahepatic comma-shaped collaterals and caudate lobe hypertrophy. Most patients are now treated with systemic anticoagulant medication and endovascular procedures, which have significantly increased survival (Thuluvath *et al.*, 2021). Ten to twenty percent of the patients will be considered for liver transplantation, typically as of result is acute liver failure or the failure of all other treatments (Bansal *et al.*, 2018). BCS develops into fibrosis-related liver failure and ultimately mortality if treatment is not received (Aktas *et al.*, 2022).

CASE PRESENTATION

A 23-year-old female patient who is a known case of decompensated chronic liver disease with Budd–Chiari syndrome, Model for end stage liver disease Na Score-20, and splenomegaly with portal hypertension presented with abdominal distension and weight loss. On detailed history, she

Table 1: Relevant parameters of the case.

Sl. No.	Laboratory parameter	Measured value	Reference range
1	PCV	44.9	37–54%
2	MCV	61.4	80–100 fL
3	MCH	19.7	27–34 pg
4	Lymphocytes	19.3	20.0–40.0%
5	Serum. Protein	4.2	6.0–8.3 g/100 mL
6	Serum. Albumin	2.0	3.4–5.4 g/100 mL
7	Neutrophils	10	40–60%
8	Lymphocytes	60	20–40%
9	MCV	64.4	83–101 fL
10	MCH	23.3	27–32 pg
11	RDW-CV	18.0	11.6–14.0%
12	WBC absolute count	11.9	4.0–10.0 × 10^9/L
13	Absolute neutrophil count	8.3	2.0–7.0 × 10^9/L
14	Serum. Creatinine	0.54	0.57–1.11 mg/100 mL
15	Prothrombin time	25.0	10–15 s
16	PT-INR	2.35	0.9–1.2 s
17	MCV	64.8	83–101 fL
18	MCH	22.6	27–32 pg
19	WBC absolute count	12.6	4.0–10.0 × 10^9/L
20	Lymphocytes	18	20.0–40.0%
21	Prothrombin time	20.6	10–15 s
22	PT-INR	1.8	0.9–1.2 s
23	Protein S functional	19.50	63.50–149.00%
24	MCV	72.0	83–101 fL
25	MCH	23.4	27–32 pg
26	RDW-CV	14.8	11.6–14.0%
27	TLC	12.93	4–10 × 10^3/L
28	Absolute neutrophil count	8.23	2–8 × 10^3/L
29	Absolute eosinophil count	1.16	0.02–0.50 × 10^3/L
30	Bilirubin direct	0.6	0.10–0.39 mg/100 mL
31	Serum. Albumin	3.5	3.4–5.4 mg/100 mL
32	GGT	60.3	12–38 U/L
33	Serum. Creatinine	0.4	0.57–1.11 mg/100 mL
34	Prothrombin time	22.0	10–15 s

Table 2: Drug chart.

Sl. No.	Drug name	Generic name	Dose	ROA	FREQ
1	T. TAXIM-O	cefixime	200 mg	po	BD
2	T. SOMPRAZ	esomeprazole	40 mg	po	od
3	T. ELIQIS	apixaban	2.5 mg	po	BD
4	T. ECOSPRIN	aspirin	75 mg	po	od
5	T. DYTOR	torsemide	5 mg	po	BD
6	T. DOLO	paracetamol	650 mg	po	SOS
7	T. URSOCOL	ursodeoxycholic acid	300 mg	po	BD
8	T. ROSUVASTATIN + ASPIRIN	rosuvastatin + Aspirin	10/75 mg	po	od
9	T. HEPAMERZ	l-ornithine-l-aspartate	1 tab	po	od
10	INJ. CLEXANE	enoxaparin sodium	40 mg	S/C	od
11	T. CARDIVAS	carvedilol	3.125 mg	po	BD
12	T. RIFAGUT	Rifaximin	550 mg	po	BD
13	T. MOX CLAV	amoxicillin + potassium clavulanate	625 mg	po	BD

was not married, and no history of comorbidities, DM, HTN, or thyroid. No history of complaints like fever, scleral icterus, epistaxis, hematemesis, or hemorrhoids. The patient had a significant surgical history. IVC and right Inferior Hepatic Vein angioplasty (IHV), and Ascitic tapping was done, draining 1.5 L of ascitic fluid (2025). She underwent ultrasonography of the whole abdomen, which was abnormal with Cirrhosis of the liver with Budd–Chiari syndrome, canalized umbilical vein, moderate ascites, and gallbladder sludge. Her contrast-enhanced CT whole abdomen revealed that the liver had surface modularity and caudate lobe hypertrophy, chronic liver parenchymal change postbudd chiari, and splenomegaly. The histopathology report revealed that it was negative for malignancy. Laboratory findings are summarized in Table 1. Based on the clinical and investigative findings, a planned treatment was formulated. During the hospitalization, she was treated with anticoagulants, antibiotics, statins, proton pump inhibitors, β blockers, hepatoprotectives, diuretics and other supportive care (Table 2). The patient was planned to receive Physiological hepatic venous drainage, Dynamic International Prognostic Scoring System + IVC plasty/ stenting risk of procedure, lifelong anticoagulants, and prospects of further pregnancy.

DISCUSSION

In this case study, a 23-year-old female patient with increased portal venous blood pressure, splenomegaly, and abdominal ascites was diagnosed with decompensated chronic liver disease with Budd–Chiari syndrome. Budd–Chiari syndrome is a rare hepatic vascular abnormality usually caused by blockage of outflow in hepatic veins, leading to congestive hepatopathy, sinusoidal hypertension, and progressive liver dysfunction. Budd–Chiari syndrome shows a higher prevalence rate in Asian people and predominantly affects young adults, as reported by Li Y *et al.*, and Ki M *et al.* In this study, the patient presented with abdominal distension and weight loss, which are common symptoms of Budd–Chiari syndrome. Imaging studies, such as ultrasonography and contrast-enhanced computed tomography performed a pivotal role in diagnosis confirmation by demonstrating cirrhotic liver morphology with caudate lobe hypertrophy, nodular liver surface, canalization of the umbilical vein, splenomegaly, and moderate ascites. Similar characteristics in the imaging studies have been described as main features of chronic hepatic venous outflow obstruction by Porrello *et al.*, and Bansal *et al.*, Laboratory tests have indicated impaired hepatic synthetic function, which revealed hypoalbuminemia, prolonged prothrombin time and elevated INR. These abnormalities are commonly observed in patients who have Budd–Chiari syndrome, as reported by Thuluvath *et al.* The presence of increased red cell distribution width in the patient may be interlinked to chronic liver disease, hypersplenism secondary to portal hypertension and possible nutritional deficiencies. According to Oliver *et al.*, despite prolonged coagulation parameters, people with Budd–Chiari syndrome remain in a hypercoagulable statement to the natural reduction of anticoagulant synthesis, such as protein C and protein S. Anticoagulation remains the cornerstone of treatment in Budd–Chiari syndrome and was appropriately initiated in this patient using low-molecular-weight heparin followed by oral anticoagulants. Long-term or lifelong anticoagulation is recommended to prevent recurrence of thrombosis and disease progression. Diuretics such as torsemide were used to manage ascites, while carvedilol was prescribed to lower portal pressure and reduce complications like variceal bleeding. Hepatoprotective agents, including ursodeoxycholic acid and l-ornithine l-aspartate, were used to support liver function and reduce ammonia-related complications. Rifaximin was administered to decrease the risk of hepatic encephalopathy by modulating gut ammonia production.

The patient's Model for end stage liver disease-Na score of 20 indicates moderate to severe liver dysfunction and is associated with increased mortality risk. Studies have shown that patients with higher Model for end stage liver disease scores often require interventional procedures such as transjugular intrahepatic portosystemic shunt or surgical decompression when medical therapy alone is insufficient. In this case, the patient was planned for physiological hepatic venous drainage and possible inferior vena cava plasty or stenting, which aligns with recommended step-up management strategies.

CONCLUSION

This case study emphasizes the importance of early detection of liver disorder in a young woman, where timely diagnosis was essential. Early confirmation of diagnosis using imaging studies like CT and Doppler ultrasonography and abnormal laboratory investigations leads to the prompt start of anticoagulation therapy and a step-by-step management strategy, which helps to stop the disease's progression and enhance clinical results.

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ABBREVIATIONS

BCS: Budd–Chiari Syndrome; **IVC:** Inferior Vena Cava; **IHV:** Inferior Hepatic Vein; **MELD-Na:** Model for End-Stage Liver Disease-Sodium; **PCV:** Packed Cell Volume; **MCV:** Mean Corpuscular Volume; **MCH:** Mean Corpuscular Hemoglobin; **RDW-CV:** Red Cell Distribution Width–Coefficient of Variation; **WBC:** White Blood Cell; **TLC:** Total Leukocyte Count; **PT:** Prothrombin Time; **INR:** International Normalized Ratio; **GGT:** Gamma-Glutamyl Transferase; **GI:** Gastrointestinal; **CT:** Computed Tomography; **DM:** Diabetes Mellitus; **HTN:** Hypertension; **ROA:** Route of Administration; **FREQ:** Frequency; **SOS:** As Needed (Si Opus Sit); **BD:** Twice Daily; **OD:** Once Daily; **S/C:** Subcutaneous.

CONFLICT OF INTEREST

The authors declare that there is conflict of interest.

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