International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Imaging Diagnosis of Budd-Chiari Syndrome in a Middle-Aged Female: A Case Report

Pintu Biswas[1], Dr. Harshith R[2], Dr. Kanak Choudhury[3], Dr. Nahid Niaj[4], Dr. Shubhajit Debnath[5], Dr. Md Imran Ahmed[6]

[1]Senior Resident, Department of Radiodiagnosis, Agartala Government Medical College & Gb Pant Hospital, Agartala, West Tripura
[2,4,5,6]Post Graduate Student, Department of Radiodiagnosis, Agartala Government Medical College & Gb Pant Hospital, Agartala, West Tripura
[3]Assistant Professor, Department of Medicine, Agartala Government Medical College & Gb Pant Hospital, Agartala, West Tripura

Abstract
Budd-Chiari Syndrome (BCS) is a rare disorder characterized by hepatic venous outflow obstruction, leading to liver congestion, ischemia, and cirrhosis. A 35 years old female presented with nausea, vomiting, reduced appetite and progressive abdominal distension. Clinical examination revealed mild icterus and pedal edema. Ultrasonography (USG) with Doppler showed caudate lobe hypertrophy, ascites, and absent hepatic veins, raising suspicion of BCS. Contrast-enhanced computed tomography (CECT) confirmed the diagnosis, revealing hepatic vein thrombosis, caudate lobe hypertrophy, peripheral liver atrophy, and gastroesophageal varices. Imaging played a crucial role in establishing the diagnosis, while the patient was lost to follow-up before further management. This case highlights the role of USG and CECT in the early diagnosis of BCS and emphasizes the need for prompt treatment, including diuretics, anticoagulation, and advanced interventions like TIPS or liver transplantation in severe cases, to prevent complications such as cirrhosis and portal hypertension.

Keywords: Ascites, Budd-Chiari Syndrome, cirrhosis, computed tomography, hepatic venous outflow obstruction, ultrasonography.

Introduction

Budd-Chiari Syndrome (BCS) is a rare and potentially life-threatening disorder characterized by obstruction of hepatic venous outflow, leading to hepatic congestion, ischemia, and subsequent liver damage. The obstruction can occur at the level of the hepatic veins, the inferior vena cava, or both. The etiology is often multifactorial, including prothrombotic conditions, connective tissue disorders, and external compression by tumors or abscesses. BCS is classified into primary (intrinsic venous abnormalities such as thrombosis) and secondary (extrinsic compression or invasion) types [1,2].

Patients with BCS typically present with nonspecific symptoms such as abdominal pain, hepatomegaly, ascites, or signs of portal hypertension. In some cases, the disease progresses silently until the development of complications such as liver cirrhosis or hepatocellular carcinoma [3,4].

The diagnosis of BCS relies heavily on imaging modalities. Doppler ultrasonography is the first-line inv-

estigation, revealing absent or reversed flow in the hepatic veins or inferior vena cava. Contrast-enhanced computed tomography (CECT) and magnetic resonance imaging (MRI) provide detailed visualization of hepatic vascular anatomy, obstruction, and secondary parenchymal changes [5,6].
This case report discusses a middle-aged female presenting with ascites and liver cirrhosis, diagnosed with BCS using ultrasonography and CECT, highlighting the importance of imaging in the diagnostic process and providing an overview of current management strategies.

Case report

A 35 years old female presented with nausea, vomiting, reduced appetite and gradual abdominal distension. She had mild icterus and edema of the foot, rest of the physical examination was unremarkable. She was advised USG abdomen, which revealed features of chronic liver parenchymal disease with enlarged caudate lobe & ascites. On Doppler interrogation the IVC was seen narrowed and there was non-visualization of hepatic veins (Fig. 1 & 2), hence making provisional diagnosis of Budd Chiari syndrome. Later she underwent CECT whole abdomen which confirmed the USG findings. There was narrow IVC with non-visualization of hepatic veins and the liver showed differential attenuation pattern characteristic of BCS (Fig. 3 & 4). There was mild splenomegaly and ascites. However the patient was lost to follow-up for further radiological investigation.

Representative images of this article:

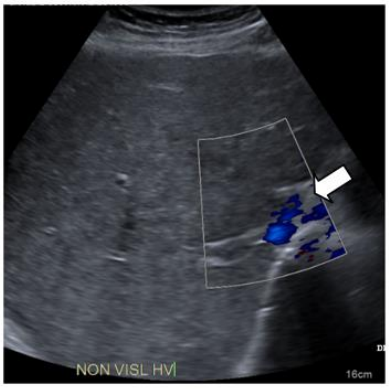

Fig 1. Doppler Usg showing narrow IVC and non visualization of hepatic veins

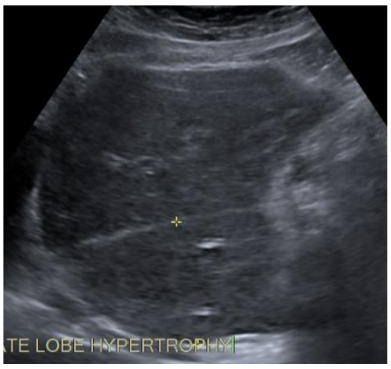

Fig 2. USG shows caudate lobe hypertrophy

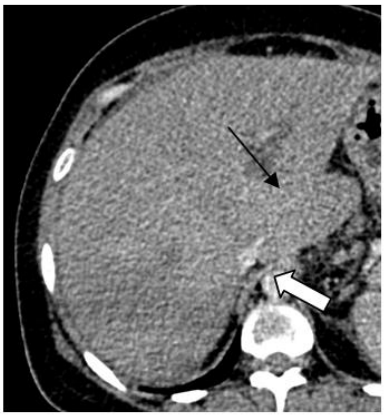

Fig 3. Axial CECT shows enlarged caudate lobe (black arrow), slit like IVC (white arrow) with non-visualization of hepatic veins and differential liver attenuation pattern.

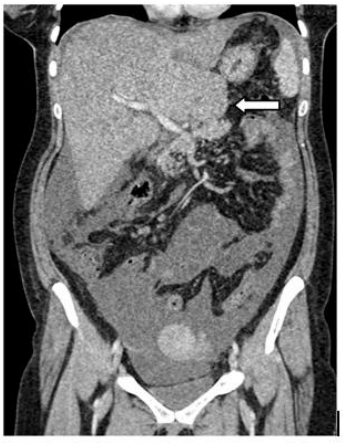

Fig 4. Coronal CECT shows caudate lobe hypertrophy and ascites

Discussion

Budd-Chiari Syndrome (BCS) is an uncommon cause of portal hypertension and liver dysfunction, with an estimated incidence of 1 in 100,000 individuals worldwide [7]. This case highlights the typical presentation of BCS in a middle-aged female with progressive ascites and liver cirrhosis. Ascites, a hallmark of BCS, occurs due to hepatic venous outflow obstruction, leading to increased sinusoidal pressure and fluid accumulation [8]. The progression to cirrhosis is indicative of a chronic course, where long-standing liver congestion and ischemia have resulted in irreversible architectural damage [9].

Imaging plays a pivotal role in the diagnosis and characterization of BCS.

Ultrasonography (USG) with Doppler is often the first-line investigation due to its wide availability and cost-effectiveness. The characteristic findings include absent, reversed, or turbulent flow in the hepatic veins, as well as intrahepatic collateral vessels. The caudate lobe often appears hypertrophied due to its independent venous drainage into the inferior vena cava (IVC), bypassing the obstructed hepatic veins [10]. Additionally, Doppler can identify IVC thrombosis or narrowing, further confirming the diagnosis.

Contrast-enhanced computed tomography (CECT) provides detailed anatomical information and is particularly useful in evaluating the extent of thrombosis or obstruction. In BCS, the liver may show inhomogeneous enhancement due to areas of congestion and ischemia, with delayed enhancement of the caudate lobe compared to the rest of the liver. Peripheral liver atrophy and caudate lobe hypertrophy are

hallmark features in chronic cases. Collateral vessels and varices are often identified, particularly in advanced disease stages. Additionally, CECT can exclude other causes of hepatic dysfunction, such as malignancy or portal vein thrombosis [11].

Magnetic resonance imaging (MRI) provides superior soft tissue contrast and having multiplanar imaging capabilities. T1-weighted images may show hypointense thrombi in the hepatic veins or IVC, while T2-weighted images highlight areas of congestion and edema. Dynamic contrast-enhanced sequences can demonstrate delayed and patchy enhancement of the liver parenchyma. MRI with MR angiography can also noninvasively map the venous anatomy, aiding in treatment planning [5].

In this case, ultrasonography (USG) served as the initial diagnostic modality, demonstrating absent flow in the hepatic veins and the presence of ascites. Doppler ultrasound is highly sensitive for detecting venous obstruction, making it an indispensable tool in the early evaluation of BCS [10]. CECT confirmed the diagnosis, revealing hepatic vein thrombosis and secondary changes such as hypertrophy of the caudate lobe and atrophy of the peripheral liver parenchyma. CECT also excluded other potential causes of liver dysfunction, such as malignancy or portal vein thrombosis, underlining its importance in delineating the anatomy and extent of vascular involvement [11].

Management of BCS is multimodal, tailored to the severity and underlying etiology. Initial treatment focuses on managing symptoms and complications, such as ascites, with sodium restriction, diuretics, and therapeutic paracentesis if necessary. Anticoagulation therapy is initiated in most patients to prevent thrombus propagation, provided there is no contraindication [12]. In cases of advanced liver dysfunction or portal hypertension, endovascular interventions such as angioplasty, stenting, or Trans jugular intrahepatic portosystemic shunt (TIPS) may be required. TIPS, in particular, is effective in relieving portal hypertension and improving hepatic outflow [13]. For patients with decompensated cirrhosis or those who fail to respond to other treatments, orthotopic liver transplantation remains the definitive option [14].

The prognosis of BCS depends on the extent of venous obstruction, liver function at diagnosis, and the effectiveness of therapeutic interventions. Early diagnosis through imaging and timely management are critical in improving outcomes in patients with BCS.

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