

## Budd–Chiari Syndrome Secondary to Burkitt’s Lymphoma in a 5-Year-Old Child

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### Abstract

**Summary:** We report a rare case of Budd–Chiari syndrome secondary to Burkitt’s lymphoma in a 5-year-old male presenting with jaundice and abdominal pain. The case highlights the diagnostic challenges in pediatric patients presenting with hepatomegaly and cholestatic jaundice, and emphasizes the importance of considering underlying malignancy in atypical cases of hepatic venous outflow obstruction.

**Keywords:** Burkitt lymphoma, Budd Chiari syndrome, venous thromboembolism/ etiology.

### Introduction

Burkitt’s lymphoma (BL) is a highly aggressive non-Hodgkin lymphoma (NHL) of B-cell origin, characterized by the translocation and deregulation of the MYC gene.<sup>1,2</sup> It accounts for 6–8% of childhood malignancies, with a median age of presentation around 10 years. Pediatric NHLs are predominantly high-grade, and BL is known for its rapid progression and frequent extranodal involvement, particularly of the abdomen, bone marrow, and central nervous system.<sup>1</sup>

Budd–Chiari syndrome (BCS) is a rare disorder caused by obstruction of hepatic venous outflow, which may be primary (due to venous abnormalities) or secondary (due to external compression or invasion by tumors).<sup>3</sup> Association of BL with BCS is extremely rare and can lead to diagnostic confusion, as both conditions may initially present with hepatomegaly and jaundice.

### Case Presentation

A 5-year-old male, resident of Bahria Town, Rawalpindi, presented to the emergency department with complaints of abdominal pain, anorexia, and yellow discoloration of the skin and sclera for 20 days. The pain was gradual in onset, intermittent, and generalized. There was no history of itching, clay-colored stools initially, fever, or bleeding tendencies.

#### Past and Family History

He had no history of previous transfusions, injections, or chronic illnesses. He was a product of normal vaginal delivery with normal developmental milestones. His immunization status was up to date. No family history of similar illnesses was reported.

#### Examination

The child appeared unwell and deeply icteric. Vitals were stable. Anthropometric measurements were between the 10th and 25th percentiles. There was no pallor, lymphadenopathy, or edema.

Abdominal examination revealed hepatomegaly (4 cm below the costal margin, firm, 13 cm span) and splenomegaly (1 cm below the costal margin). Ascites was clinically present, with shifting dullness positive.

#### Investigations

CBC: Hb 10.3 g/dL, WBC 9500/μL, Platelets 531,000/μL

LFTs: Total bilirubin 9.9 mg/dL (Direct 8.2 mg/dL), ALT 182 U/L, ALP 120.9 U/L

Serology: Hepatitis A, B, C, and E negative

Ultrasound: Mild hepatosplenomegaly with mild ascites

During hospitalization, jaundice worsened, and the child developed abdominal distension and tender hepatomegaly. Clay-colored stools and low-grade fever appeared.

#### Advanced Imaging

CT abdomen and pelvis revealed multifocal thrombosis of the inferior vena cava with hepatomegaly, nutmeg appearance of liver, and flip-flop pattern suggestive of Budd–Chiari syndrome, along with acute pancreatitis and right renal vein thrombosis.

A triphasic CT scan later showed periportal hypodensities, hepatic and renal hypodense lesions, and mesenteric lymphoid masses, suggesting a lymphoproliferative disorder.

#### Ascitic Fluid Analysis:

Protein 49 g/L, LDH 2296 U/L, ADA 34.3 U/L, with 80% lymphocytes and a few atypical cells.

Histopathology: Liver biopsy revealed sheets of monotonous intermediate-sized lymphoid cells with a “starry-sky” appearance, confirming Burkitt’s lymphoma.

#### Final Diagnosis:

Budd–Chiari syndrome secondary to hepatic infiltration by Burkitt’s lymphoma.

### Discussion

This case demonstrates a rare presentation of BL manifesting as Budd–Chiari syndrome. Burkitt’s lymphoma can involve the liver either by direct infiltration or secondary thrombosis due to

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SI AH AA MA - Conception, Design

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SI - Drafting

AH AA MS JZ- Critical Review

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None to report

#### Institutional Review Board

##### Approval

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hypercoagulability and venous obstruction.<sup>4</sup> Pediatric lymphomas are known to induce a prothrombotic state, and studies report venous thromboembolism in up to 10–15% of cases within the first year of diagnosis.

In this patient, hepatic venous thrombosis and IVC obstruction likely resulted from lymphomatous invasion. The diagnosis was exigent due to overlapping symptoms of hepatic illness and the scarcity of such a presentation in children. Imaging and biopsy were crucial investigations that led to the diagnosis.

The gold standard for the diagnosis of lymphoma is Histopathology, which shows hyperchromatic monomorphic lymphoid cells with phagocytic histiocytes—the classic “starry sky” pattern.<sup>5</sup>

#### Management and Outcome

Burkitt’s lymphoma management includes chemotherapy or chemoimmunotherapy, depending upon the disease stage. Prophylaxis for tumor lysis syndrome, febrile neutropenia, and monitoring for thrombotic complications remains the main supportive treatment.

The child was then referred to the pediatric oncology unit for further management and initiation of chemotherapy after the diagnosis was confirmed

## Conclusion

This case highlights the importance of considering unusual diagnosis like malignancy in pediatric patients presenting with unexplained hepatic venous obstruction. Imaging and histological evaluation early in the course of disease are essential for making a timely diagnosis and management of rare diseases like Budd–Chiari syndrome secondary to Burkitt’s lymphoma.

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