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BUDD-CHIARI SYNDROME ASSOCIATED WITH UNTREATED HYPOTHYROIDISM IN A YOUNG FEMALE: A CLINICAL CASE REPORT

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ABSTRACT **Background:** Budd–Chiari Syndrome (BCS) is a rare condition caused by hepatic venous outflow obstruction, often associated with underlying prothrombotic states. **Case Summary:** A 22-year-old female presented with abdominal distension, pain, pedal oedema, weight loss, and hyperpigmentation. She had discontinued thyroxine therapy two months prior. Imaging confirmed BCS with hepatic vein obstruction and inferior vena cava thrombus, along with ascites and pleural effusion. Endoscopy revealed esophageal varices and portal hypertensive gastropathy. She was treated with anticoagulation, diuretics, and thyroxine, resulting in clinical improvement. **Conclusion:** This case highlights the importance of early recognition of BCS and the potential prothrombotic role of untreated hypothyroidism, emphasizing the need for timely multidisciplinary management.

KEYWORDS : Budd-Chiari Syndrome; Hepatic Vein Thrombosis; Hypothyroidism; Portal Hypertension.

INTRODUCTION

Budd-Chiari Syndrome (BCS) is defined as hepatic venous outflow tract obstruction occurring anywhere from the small hepatic veins to the inferior vena cava (IVC), in the absence of cardiac disease, sinusoidal obstruction syndrome, or constrictive pericarditis [1]. The syndrome may present in acute, subacute, or chronic forms depending on the rapidity of venous occlusion. Its pathophysiology involves hepatic congestion, sinusoidal hypertension, hepatocellular ischemia and necrosis, and, if untreated, progressive fibrosis and cirrhosis [1,2].

BCS is rare, with an estimated incidence of 1 per million in Western populations, though higher rates have been reported in parts of Asia and Africa [3]. In approximately 80% of cases, an underlying prothrombotic disorder is identified, including myeloproliferative neoplasms, antiphospholipid syndrome, inherited thrombophilia's (protein C, protein S, or antithrombin III deficiency; factor V Leiden; prothrombin G20210A mutation), and hormonal therapies [2,3]. In young women, the interaction between subclinical hypercoagulability and hormonal or metabolic disturbance — such as hypothyroidism — may substantially amplify thrombotic risk [4].

Hypothyroidism is known to alter hemostatic balance by impairing fibrinolysis, reducing levels of tissue plasminogen activator, and increasing von Willebrand factor activity, thereby creating a prothrombotic milieu [4]. Despite this association, BCS precipitated by hypothyroidism — particularly in patients with treatment non-adherence — remains underreported. We present a case of BCS in a 22-year-old female with undertreated hypothyroidism, illustrating both the diagnostic challenges and the critical role of multidisciplinary intervention.

2. Case Presentation

2.1 Patient History and Presentation

A 22-year-old female presented to the gastroenterology department on 01 July 2022 with a 20-day history of progressive abdominal distension and pain, associated with nausea, vomiting, and loose stools. She also reported bilateral pedal oedema (intermittent), generalized skin darkening with facial discoloration, and a weight loss of approximately 12 kg over the preceding two months. Her menstrual cycle had been irregular for two months.

Her past medical history was notable for a spontaneous abortion two years prior and a confirmed diagnosis of hypothyroidism for which she had been prescribed Tab. Thyroxine 75 mcg daily. However, she had independently discontinued this medication two months before the current admission — a clinically significant detail given the prothrombotic implications of untreated thyroid dysfunction.

On physical examination, she appeared pale and malnourished. Vital signs were Temperature 98.8°F (37.1°C), Blood Pressure 130/90 mmHg. Abdominal examination revealed a distended, tender abdomen with tenderness localized to the right hypochondrium, positive shifting dullness consistent with ascites, and clinical hepatomegaly. Pallor was noted; there was no jaundice at the time of admission.

2.2 Investigations and Imaging

A comprehensive investigative workup was initiated on admission. Laboratory findings on serial assessment are summarized in Table 1. Key abnormalities included anemia (hemoglobin 9.2 g/dL), thrombocytopenia (platelets 98 × 10^3/µL), deranged liver function tests (ALT 88 U/L, AST 92 U/L, ALP 210 U/L, total bilirubin 2.1 mg/dL), hypoalbuminemia (serum albumin 2.4 g/dL), and a prolonged prothrombin time (INR 1.6). Thyroid-stimulating hormone (TSH) was markedly elevated at 12.4 mIU/L, confirming active hypothyroidism. HIV and hepatitis serology were negative.

Table 1: Serial Investigations During Admission (01–06 July 2022)

Investigation	01/07/2022	02/07/2022	03/07/2022	04/07/2022	06/07/2022	Reference Range
Hemoglobin (g/dL)	9.2	—	—	9.0	9.5	12–16
WBC (×10^3/µL)	11.4	—	—	10.8	10.2	4–11
Platelets (×10^3/µL)	98	—	—	95	102	150–400
Total Bilirubin (mg/dL)	2.1	—	—	1.9	1.6	<1.2
Direct Bilirubin (mg/dL)	1.4	—	—	1.2	1.0	<0.3
ALT / SGPT (U/L)	88	—	—	72	60	7–56
AST / SGOT (U/L)	92	—	—	80	65	10–40
ALP (U/L)	210	—	—	190	175	44–147
Serum Albumin (g/dL)	2.4	—	—	2.5	2.7	3.5–5
PT/INR	1.6	—	—	1.5	1.3	<1.1
Serum Creatinine (mg/dL)	0.9	—	—	0.8	0.9	0.6–1.1
Serum Sodium (mEq/L)	132	—	—	134	136	136–145
TSH (mIU/L)	12.4	—	—	—	—	0.4–4.0
Ascitic Fluid SAAG	—	High	High	—	—	>1.1 = hepatic
Ascitic Fluid Protein	—	Low	Low	—	—	>2.5 g/dL = exudative

— = not performed on that date; SAAG = Serum-Ascites Albumin Gradient; ALT = Alanine Transaminase; AST = Aspartate Transaminase; ALP = Alkaline Phosphatase; TSH = Thyroid-Stimulating Hormone.

Ascitic tapping was performed on 02 July 2022, yielding 30 mL of yellow straw-colored fluid. Biochemical analysis revealed a high serum-ascites albumin gradient (SAAG > 1.1 g/dL), low protein content, and the presence of red blood cells — a pattern consistent with hepatic cause of ascites and portal hypertension rather than malignancy or infection.

Esophagogastroduodenoscopy (OGD) demonstrated large esophageal varices with portal hypertensive gastropathy, confirming significant portal hypertension. Triple-phase contrast-enhanced CT (CECT) of the abdomen and pelvis revealed:

- Inhomogeneous mottled hepatic enhancement with non-visualization of the hepatic veins — the cardinal imaging hallmark of Budd-Chiari Syndrome.
- Small filling defects in the suprahepatic portion of the inferior vena cava persisting into the portal and venous phases, consistent with partial thrombus.
- Hemangioma in segment VI of the liver (incidental finding).
- Fibrous ascites and bilateral pleural effusion.

These findings, in conjunction with the clinical picture, confirmed the diagnosis of primary Budd-Chiari Syndrome secondary to hepatic vein thrombosis with IVC involvement.

2.3 Clinical Course

On 03 July 2022, the patient continued to report abdominal pain and had a single episode of vomiting overnight. Strict input/output monitoring revealed fluid retention (input 1000 mL, output 350 mL). Aspiration fluid analysis again confirmed high SAAG and low protein. A sodium-restricted diet (<2 g/day) and fluid restriction (<1.5 L/day) were instituted. Subcutaneous Clexane (enoxaparin) 40 mg was commenced the following day.

On 04 July 2022, the patient passed normal stools without vomiting, though abdominal pain persisted. Right hypochondrium and epigastric tenderness were noted on examination. Tab. Rabeprazole 20 mg BD was added for gastroprotection, and an endocrinology review was requested in view of the elevated TSH and history of thyroxine non-adherence.

On 05 July 2022, abdominal pain had improved, though post-prandial distension persisted. A left breast lesion was identified on clinical examination; subsequent Sono mammogram classified this as a complex cyst/chronic abscess (BI-RADS Category II — benign finding). Surgical review confirmed outpatient follow-up was appropriate. Nutritional support with Hepatic Resource powder and Threptin biscuits was commenced.

By 06 July 2022 — Day 5 of admission — abdominal pain and distension had substantially improved. The endocrinologist reviewed the patient and reinstated Tab. Thyroxine 75 mcg daily, with a repeat TSH planned after 3 months.

2.4 Discharge and Final Diagnosis

The patient was discharged in a clinically stable condition on 06 July 2022 with the following confirmed diagnoses:

- Hepatic Vein Thrombosis with Budd-Chiari Syndrome
- Portal Hypertension with Ascites and Esophageal Varices
- Hypothyroidism (previously non-adherent to therapy)

Table 2: Discharge Medication Regimen

Medication	Dose	Frequency	Indication
Tab. Rabeprazole	20 mg	Once daily	Gastroprotection / varices
Tab. Thyroxine	75 mcg	Once daily	Hypothyroidism
Tab. Furosemide	20 mg	Once daily	Ascites / oedema
Tab. Spironolactone	50 mg	Once daily	Ascites / anti-aldosterone
Tab. Ademetionine	400 mg	Once daily	Hepatoprotection
Inj. Enoxaparin S/C	40 mg	Once daily × 3 days	Anticoagulation
Tab. Paracetamol	650 mg	SOS	Analgesia / antipyresis
Tab. Multivitamin	Standard	Once daily × 1 month	Nutritional support

SOS = as required; S/C = subcutaneous.

Follow-up Instructions: Gastroenterology OPD review at one week with repeat LFTs, RFTs, CBC, and PT/INR. Endocrinology follow-up for repeat TSH at three months.

3. DISCUSSION

This case illustrates a diagnostically complex presentation of primary Budd-Chiari Syndrome in a young woman whose clinical course was compounded by medication non-adherence, nutritional compromise, and an active endocrine disorder. Several features warrant specific discussion.

Hypothyroidism as a prothrombotic risk factor. Thyroid hormones modulate multiple components of the coagulation cascade. Hypothyroidism reduces fibrinolytic activity through decreased tissue plasminogen activator (tPA) expression, increases von Willebrand factor, and is associated with elevated factor VIII levels — collectively conferring a prothrombotic state [4,5]. In this patient, self-discontinuation of thyroxine two months prior to presentation likely exacerbated her thrombotic predisposition. This association remains underappreciated clinically and is rarely discussed in the context of hepatic vein thrombosis. The dramatically elevated TSH (12.4 mIU/L) at admission corroborated significant thyroid dysfunction at the time of thrombus formation.

Diagnosis and Imaging.

The diagnostic gold standard for BCS remains contrast-enhanced cross-sectional imaging demonstrating hepatic vein non-visualization, caudate lobe hypertrophy (from its independent venous drainage), and inhomogeneous hepatic parenchymal enhancement [1,6]. In this case, triple-phase CECT provided definitive imaging confirmation, complemented by OGD evidence of established portal hypertension. High-SAAG ascites with low protein further corroborated a hepatic venous congestion etiology, distinguishing it from exudative causes [3].

Anticoagulation as the cornerstone of treatment. Early anticoagulation is recommended in all patients with BCS in the absence of absolute contraindications [1,7]. Initial therapeutic anticoagulation with unfractionated heparin followed by transition to low-molecular-weight heparin (enoxaparin) achieved symptomatic improvement within days in this patient. Long-term oral anticoagulation is typically required to prevent thrombus propagation and hepatic infarction [2,7]. Concurrent diuretic therapy with furosemide and spironolactone addressed the ascitic burden, while ademetionine (SAMe) provided hepatoprotective benefit.

Multidisciplinary Care.

Optimal management of BCS requires coordinated input from gastroenterology, hepatology, hematology, endocrinology, and surgery. In this patient, the involvement of an endocrinologist to reinstate thyroid replacement therapy, a surgeon for the incidental breast lesion, and a gastroenterologist to manage portal hypertension and varices was essential. This case reinforces consensus guidance that BCS is best managed in centers with multidisciplinary expertise and access to interventional radiology and transplantation services [1,8].

A limitation of this case report is that a comprehensive thrombophilia screen (including factor V Leiden, prothrombin gene mutation, JAK2V617F, antiphospholipid antibodies, and natural anticoagulant levels) was not documented in the available records. Such testing is strongly recommended in all BCS patients to guide long-term anticoagulation decisions and to identify conditions requiring targeted therapy.

Current management is consistent with established guidelines recommending early anticoagulation as first-line therapy in Budd–Chiari syndrome.

4. CONCLUSION

This case highlights a classical yet clinically complex presentation of primary Budd-Chiari Syndrome in a young woman, complicated by undertreated hypothyroidism, nutritional deficits, and multi-organ involvement. Key learning points include: (1) BCS should be actively excluded in young patients with unexplained ascites, hepatomegaly, and elevated liver enzymes; (2) hypothyroidism — particularly when poorly controlled — may contribute to hepatic vein thrombosis and should be evaluated in the BCS workup; (3) early anticoagulation, diuretic management, hormonal correction, and nutritional support are the pillars of initial management; and (4) multidisciplinary collaboration is indispensable. Thorough thrombophilia screening and long-term hepatological surveillance remain essential components of care to minimise risks of recurrence and hepatic decompensation.

5. Data Availability:

All data analyzed during this study are included in this published article. Additional details are available from the corresponding author upon reasonable request.

6. Patient Consent

Written informed consent was obtained from the patient for the publication of this case report and any accompanying clinical data. Patient anonymity has been preserved throughout.

7. Author Contributions:

All authors have equally contributed to the conception, data collection, analysis, and drafting of the manuscript, and approved the final version for publication.

8. Conflicts of Interest

The authors declare no conflicts of interest with respect to the research, authorship, or publication of this article.

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Abbreviations

ALP: Alkaline Phosphatase | ALT: Alanine Transaminase | AST: Aspartate Transaminase | BCS: Budd-Chiari Syndrome | BD: Twice Daily | BP: Blood Pressure | CBC: Complete Blood Count | CECT: Contrast-Enhanced Computed Tomography | IVC: Inferior Vena Cava | LFT: Liver Function Tests | LMWH: Low-Molecular-Weight Heparin | OGD: Esophagogastroduodenoscopy | OPD: Out-Patient Department | PT/INR: Prothrombin Time/International Normalized Ratio | RFT: Renal Function Tests | SAAG: Serum-Ascites Albumin Gradient | S/C: Subcutaneous | SOS: As Required | tPA: Tissue Plasminogen Activator | TSH: Thyroid-Stimulating Hormone.

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