Section: Internal Medicine

Original Research Article

A WOLF IN BUDD-CHIARI'S CLOTHING: PSEUDO-BUDD-CHIARI SYNDROME IN TUBERCULOSIS PERITONITIS

Hema K[1], Rajashekar V[1], Akshaya Kumar[1], Roshni Subramaniyan[2], Sahasyaa Adalarasan[2], Mukil S[3], Santhi T S[4]

[1]Junior Resident, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India.
[2]Medical Student, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India.
[3]Assistant Professor, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India.
[4]Professor, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, India.

Received : 13/02/2026
Received in revised form : 07/04/2026
Accepted : 25/04/2026

Keywords:
Pseudo Budd Chiari syndrome, PBCS, Pseudo-Budd Chiari Syndrome, Budd Chiari Syndrome, Tuberculosis peritonitis.

Corresponding Author:
Dr. Sahasyaa Adalarasan,
Email: surgeonsahasyaa@gmail.com

DOI: 10.47009/jamp.2026.8.3.5

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (3); 22-26

ABSTRACT

Budd-Chiari syndrome and decompensated chronic liver disease are two different disorders of the liver. However, they share clinical presentations that can mimic one another, some of which are hepatomegaly, portal hypertension, and ascites. We report a rare case of pseudo-Budd Chiari syndrome in a 55-year-old female caused by a tuberculosis (TB) infection (TB peritonitis). She presented with complaints of abdominal pain, distension, and breathlessness. Both a CECT of the abdomen and a slice MD CT demonstrate hepatomegaly, free fluid in the abdomen and pelvis, and narrowing of the inferior vena cava, without any event of thrombosis, ruling out Budd-Chiari syndrome. Blood investigations show decreased total protein levels, deranged bilirubin levels, and an elevated prothrombin time. An omentum biopsy and ascitic fluid tapping were also done. An IVC angioplasty was performed on the patient, after which she was managed on anti-tuberculosis therapy, then discharged.

INTRODUCTION

Budd-Chiari syndrome is a rare condition characterized by hepatic venous outflow obstruction at any level from the small hepatic veins to the junction of the inferior vena cava with the right atrium, which is not caused by cardiac or pericardial disease. This obstruction leads to hepatic congestion and hypertension, leading to hepatic injury.[1] This condition is classified into primary Budd-Chiari syndrome, which results from thrombosis or inflammation of the hepatic vein walls, and secondary Budd-Chiari syndrome, caused by external compression or invasion, often due to malignancies. The disease presents in acute, subacute, or chronic forms, with symptoms including abdominal pain, ascites, and hepatomegaly.[2]

Pseudo-Budd-Chiari Syndrome, however, is a clinical and radiological mimic of true Budd-Chiari syndrome, where a patient shows classic symptoms like an enlarged liver and fluid buildup, but the major hepatic veins remain physically open (patent) rather than blocked by a clot or tumor. The apparent impairment to the outflow can be caused by a diffuse compression by an enlarged liver, but it is never caused by a primary venous lesion.[3]

Tuberculosis (TB) peritonitis is a form of extrapulmonary tuberculosis affecting the peritoneum. Risk factors for peritoneal involvement include a concomitant HIV infection, peritoneal dialysis, or cirrhosis. Reactivation of tuberculous collections lying dormant in the peritoneum accounts for most cases. Direct spread may occur from the gastrointestinal tract, and dissemination through lymphatic or hematogenous means has also been described. The abdomen is the most common site of extrapulmonary tuberculosis, with peritoneal disease being the most common form within the abdomen 4.

The etiology for pseudo-Budd-Chiari syndrome is generally attributed to those that damage the liver, such as decompensated liver diseases, alcoholic steatohepatitis, liver-associated tumors, and others that occur due to extrahepatic causes, such as pericardial conditions and right-sided heart failure, and systemic conditions like systemic lupus erythematosus, antiphospholipid antibodies, myeloproliferative neoplasms,[1,5-7] etc.

It is because of the significant overlap in the clinical presentations that accurate differentiation between true and pseudo-Budd-Chiari syndrome relies heavily on investigations such as various forms of imaging (CT/CECT scans, Ultrasound,

ISSN (O): 2687-5365; ISSN (P): 2753-6556

angiography/venography) as well as liver biopsies, to better determine the differential diagnosis.

Case Presentation

A 55-year-old female presented with features of abdominal pain and distension for a week, along with breathlessness for a period of 5 days. On examination of the abdomen, it was found to be soft, and free fluid could be felt. Vitals were normal, with a pulse rate of 86/min and BP of 130/70 mm Hg. She has had a history of Diabetes mellitus for the past 15 years, and is in the early stages of diabetic neuropathy with complete rectal prolapse.

Blood investigations showed a slight decrease in hematocrit, total protein, and albumin levels. There was a substantial decrease in the percentage of lymphocytes. However, there were significantly elevated levels of fasting blood glucose, prothrombin time, and bilirubin. Urine showed the presence of sugar, protein, and albumin. In contrast, liver enzyme test results were mostly within the normal ranges.

Table 1: Blood investigation results

Lab Parameters	Values	Reference ranges
Total leucocyte count	7500 cells/mm^3	4000-11000 cells/mm^3
Differential count (PMNL %/lymphocytes %)	80/7	40-70/20-40
MCV	86.6 fL	80-100 fL
MCH	27 pg/cell	27-33 pg/cell
MCHC	31.1	32-36 g/dl
Hematocrit	33.2%	36-48% for women
Platelet count	162 × 10^9 cells/L	150-450 × 10^9 cells/L
Prothrombin Time	21.22 seconds	10-13.5 seconds
APTT	34.52 seconds	25-35 seconds
urea	25 mg/dl	6 – 24 mg/dl
creatinine	0.53 mg/dl	0.7 – 1.3 mg/dl
Random blood sugar	99 mg/dl	<140 mg/dl
Fasting blood sugar	110 mg/dl	70-99 mg/dl
INR	1.77	0.8-1.2

(PMNL- Polymorphonuclear leucocytes, MCV- mean corpuscular volume, MCH- mean corpuscular haemoglobin, MCHC – mean corpuscular haemoglobin concentration, APTT- activated partial thromboplastin time, INR – International Normalized Ratio)

Table 2: Liver enzyme values on days 1, 2, 5, and 6

Day	1	2	5	6	Reference range
TB/DB (mg/dl)	2.24/1.18	1.98/0.88	1.3/0.6	2/0.4	0.1-0.2/0.0-0.3
SGOT/SGPT (U/L)	29/14	39/10	75/23	56/19	5-40/7-56
ALP (IU/L)	102	132	116	113	44-147
Total protein (g/dl)	7.4	6.4	8.1	6.3	6.0-8.3
Albumin (g/dl)	2.5	3.4	2.8	2.5	3.5-5

(TB- total bilirubin, DB – direct bilirubin, SGOT - serum glutamic-oxaloacetic transaminase, SGPT - Serum Glutamic Pyruvic Transaminase, ALP- alkaline phosphatase).

Table 3: Urinalysis results

Urine sugar	1+
albumin	2+
blood	1+

Moreover, tests for anticardiolipin, Beta -2 glycoprotein I (to rule out Antiphospholipid antibody syndrome), HbSAg (Hepatitis B surface Antigen), HCV (Hepatitis C virus), and HIV (Human immunodeficiency virus), anti-nuclear antibodies (to determine SLE- Systemic Lupus erythematosus), and JAK2V617F mutation (seen in myeloproliferative neoplasms) all came out to be negative.

An Ultrasound of the abdomen showed liver with coarse echoes, splenomegaly (spleen size of 13.8), free fluid in the abdomen and pelvis indicating moderate ascites, and renal collaterals in the pelvis indicating portosystemic collaterals due to portal hypertension. A slice MD CT of the abdomen revealed nodular surface of the liver (IMAGE 1,2,3), as well as the splenomegaly and free fluid in the abdomen and pelvis. The CT angiogram of the abdomen (IMAGE 4) showed multiple perisplenic and splenorenal varices, recanalized paraumbilical vein with perigastric and coronary collaterals, tortuous left gonadal vein, a few tiny calcified granulomas in the right lobe of the liver, and intrahepatic narrowing of the inferior vena cava noted secondary to external compression by hypertrophied segments of liver (IMAGE 4). A CECT of the abdomen also shows the same features.

The Esophagogastroduodenoscopy revealed pan-gastritis.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

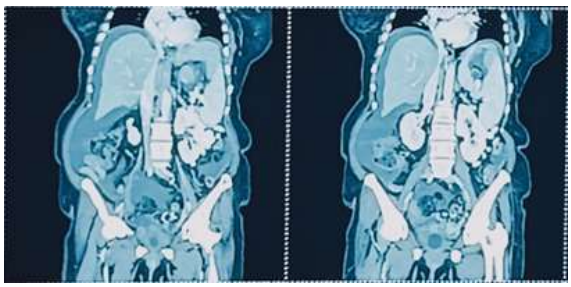

IMAGE 1

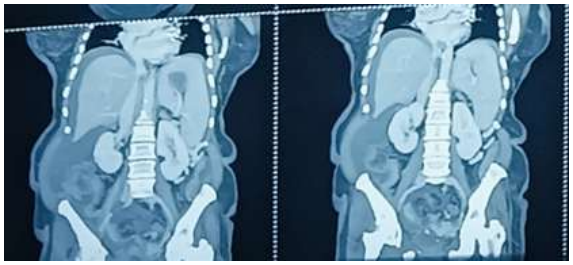

IMAGE 2

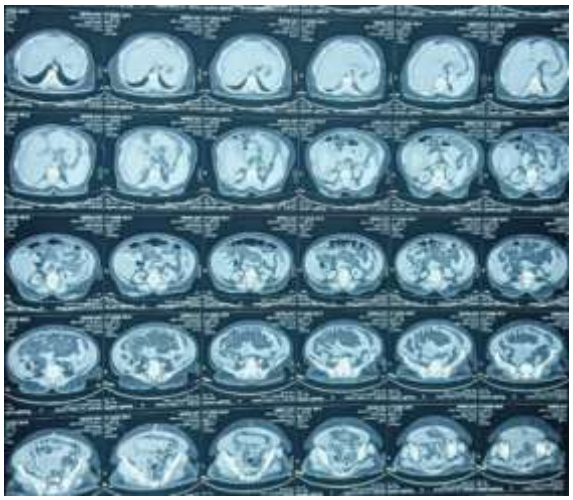

IMAGE 3

Image 1,2,3: CT abdomen showing nodular surface of liver along with splenomegaly and moderate free fluid in the abdomen and pelvis.

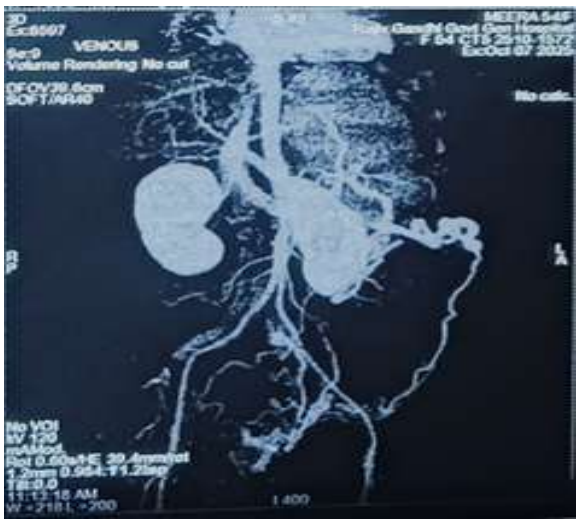

Image 4: CT angiogram of the abdomen revealing collaterals and intrahepatic narrowing of the inferior vena cava

Omentum Biopsy with haematoxylin and eosin staining was also performed, revealing caseating granulomatous lesions, which suggests a tuberculosis infection. Additionally, a fine needle aspiration cytology was done from the pericardiac lymph node, from which reactive lymphadenitis was interpreted. Finally, an ascitic fluid tapping was done, which showed a lymphocytic effusion, indicating the decreased number of lymphocytes in the blood investigations.

A diagnosis of Tuberculosis peritonitis causing pseudo- budd chiari syndrome was made. However, the Serum Ascites Albumin Gradient (SAAG), calculated from the ascitic fluid, was found to be greater than 1.1 g/dL, which is unusual, as the SAAG in tuberculosis is generally less than 1.1 g/dL 8. Following this, the patient underwent an inferior vena cava angioplasty due to severe stenosis of the inferior vena cava. There were no significant events postoperatively, and the patient was managed on anti-tuberculosis therapy, after which she was discharged.

DISCUSSION

Pseudo-Budd Chiari syndrome (PBCS) is a condition that is not well-known. The less than 20 reports found in the literature for PCBS (compared to thousands for true BCS) had patients with a mechanical compression of the hepatic outflow due to an enlarged liver and liver cirrhosis.[3,5,8]

Dhawan et al in 1978, described a patient with clinical and radiological findings of BCS and liver cirrhosis. A complete constriction of the right HV and a narrowed left HV were shown to be present due to the hypertrophy of the left lobe of the liver and a regenerative nodule. However, there were no thrombotic occlusions seen at the autopsy. This was the first paper to introduce the term pseudo-BCS.[3,10]

Another report was made by Janssen et al. It described three patients with alcoholic liver disease and BCS, without any thrombotic occlusion. These cases were denominated pseudo-BCS.[5]

Rector et al also reported a patient with liver cirrhosis, probably due to non-alcoholic steatohepatitis and BCS. There was a distortion of the IVC caused by cirrhosis and increased abdominal pressure, which suggested BCS due to membranous obstruction, but no thrombus or membranous occlusion was found.[9]

Some of the described patients had a good evolution after alcohol withdrawal.[3,5,10]

In this case however, PBCS is caused by a tuberculosis infection, i.e., TB peritonitis. While there are cases of TB peritonitis causing BCS,[11] this is the first documented case of a TB peritonitis causing PBCS.

The diagnosis of PBCS is difficult because clinical and radiological findings are extremely similar to BCS, making invasive methods necessary.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Mechanical compression of the hepatic outflow due to an edematous liver and liver cirrhosis can mimic the imaging of BCS. Those with an uncertain or false positive diagnosis require venography or arteriography, and/ or a liver biopsy to make the correct diagnosis.[12,13,14]

In essence, all the investigations were necessary to help narrow down the diagnosis; the ultrasound, which showed splenomegaly and coarse echoes of the liver, the CT and CECT of the abdomen revealing multiple varices and collaterals formed due to the portosystemic hypertension, and most importantly, the intrahepatic narrowing of the inferior vena cava. The omentum biopsy revealed the true cause of the disorder, i.e., a possible tuberculosis infection. The ascites tapping due to the presence of a moderate amount of free fluid in the abdomen and pelvis explained the decrease in the percentage of lymphocytes in the blood. Although the lymph nodes from the CECT scans were subcentimetric, the fluid needle aspiration cytology showed reactive lymphadenitis.

The possibility of Antiphospholipid antibody syndrome, systemic lupus erythematosus, HIV infections, Hepatitis B and C infections were ruled out, as they were some of the important etiologies of many liver diseases, including Budd-Chiari syndrome.

According to the 2016 European Association for the Study of the Liver (EASL) recommendations on Budd-Chiari syndrome, the first-line diagnostic study is Doppler ultrasonography; MRI and CT scanning are for diagnostic confirmation.[12,15]

Catheterization and venography can clearly delineate the nature and severity of an obstruction. Occasionally, therapeutic interventions can be undertaken at the same time, including balloon angioplasty, localized thrombolysis, and the placement of a stent or transjugular intrahepatic portacaval shunt.[12,16-19] Liver biopsy can be of prognostic assistance, particularly if liver transplantation is being considered, to establish the degree of hepatocellular damage, and also to provide information for differential diagnoses. However, in current practice, venography is rarely considered necessary for establishing a diagnosis.[10] For patients who consume alcohol, cessation of its consumption may cause reversibility of this disorder.[3,5,10-13]

A limitation of this report is that, while omentum biopsy reports, ascitic fluid tapping, and abdominal ultrasound were conducted, the corresponding images were unavailable.

In the future, one could incorporate existing imaging techniques with advanced modalities like Multiphase contrast-enhanced 3D MR angiography (3D CE-MRA) , which is a non-invasive imaging modality used to differentiate pseudo-Budd-Chiari syndrome (pseudo-BCS) from true Budd-Chiari syndrome (BCS).3D CE-MRA can distinguish between intrinsic obstruction (thrombosis, webs, or tumor invasion) seen in true BCS and extrinsic compression of hepatic veins/IVC caused by hepatomegaly or inflammatory masses, which is characteristic of pseudo-BCS.[20,21]

CONCLUSION

The diagnosis of pseudo-Budd Chiari Syndrome is difficult because clinical and radiological findings are extremely similar to BCS, making invasive methods necessary. Ultrasound, CECT, and CT scans, along with angiography/ venography, are crucial both for arriving at the correct diagnosis and for finding possible collateral channels. Tissue examination through omentum and liver biopsy, and ascitic fluid typing are equally essential in determining the cause and nature of the disorder at the cellular level.

Early recognition and treatment are crucial for both conditions - decompensated liver disease and pseudo-budd chiari syndrome - as they can significantly improve the patients' survival rate. Moreover, a possible reversibility with alcohol abstinence makes this clinical disorder a must for physicians to know.

Acknowledgements

There are no acknowledgements to be made.

Conflicts of Interest Statement

Authors declare that they have no financial interests or personal conflicts that may affect the study in this article.

REFERENCES

1. Frank H Miller, Case 295, The Teaching Files: Gastrointestinal https://www.sciencedirect.com/topics/medicine-and-dentistry/budd-chiari-syndrome#:~:text=35,the%20periphery%20(Fig%2013)
2. Hitawala AA, Goosenberg E. Budd-Chiari Syndrome. [Updated 2025 Apr 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan-. https://www.ncbi.nlm.nih.gov/books/NBK558941/
3. Robles-Medranda C, Lukashok H, Biccas B, Pannain VL, Fogaça HS. Budd-Chiari like syndrome in decompensated alcoholic steatohepatitis and liver cirrhosis. World J Gastroenterol 2006; 12(40): 6564-6566 [PMID: 17072994 DOI: 10.3748/wjg.v12.i40.6564]
4. Bickle I, Hartung M, Yap J, et al. Tuberculous peritonitis. Reference article, Radiopaedia.org (Accessed on 13 Apr 2026) https://doi.org/10.53347/rID-19978
5. Janssen, Harry L A et al. "Pseudo-Budd-Chiari Syndrome: decompensated alcoholic liver disease mimicking hepatic venous outflow obstruction." Hepato-gastroenterology vol. 49,45 (2002): 810-2 https://pubmed.ncbi.nlm.nih.gov/12063996/
6. Solela G, & Daba M (2023). Budd-Chiari Syndrome as an Initial Presentation of Systemic Lupus Erythematosus Associated with Antiphospholipid Syndrome: A Case Report with Review of the Literature. Open access rheumatology : research and reviews, 15, 139–143. https://doi.org/10.2147/OARRR.S425535
7. Pillay S, & Moffat N (2021). A rare case of constrictive pericarditis with Budd-Chiari syndrome due to right atrial thrombosis. SAGE open medical case reports, 9, 2050313X211032405. https://doi.org/10.1177/2050313X211032405
8. Medscape. Serum Ascites Albumin Gradient (SAAG) Interpretation https://emedicine.medscape.com/article/2172621-overview
9. Rector, W G Jr. "Pseudo-Budd-Chiari syndrome: extrinsic deformity of the intrahepatic inferior vena cava mimicking

ISSN (O): 2687-5365; ISSN (P): 2753-6556

membranous obstruction." Journal of clinical gastroenterology vol. 11,1 (1989): 88-91. https://pubmed.ncbi.nlm.nih.gov/2921498/
10. Dhawan, V M et al. "Pseudo-Budd-Chiari syndrome." Clinical nuclear medicine vol. 3,1 (1978): 30-1. doi:10.1097/00003072-197801000-00009
11. Baray F, Noori M B, Aram M M, & Hamidi H. (2022). Misdiagnosis of Budd Chiari syndrome, a case report from Afghanistan. Annals of medicine and surgery (2012), 73, 103218. https://doi.org/10.1016/j.amsu.2021.103218
12. Medscape. Budd-Chiari syndrome: Background, Pathophysiology, Etiology. eMedicine [Internet]. 2024 Jun 10 [cited 2024 Jul 12]. Available from: https://emedicine.medscape.com/article/184430-overview
13. Gosai, Falgun MD; Sarma, Alinda MD; Muthukuru, Sujit MD, et al. Psuedo Budd-Chiari Syndrome: An Uncommon Presentation of Very Common Disease: 2333. American Journal of Gastroenterology 112():p S1273-S1274, October 2017. https://www.ovid.com/jnls/ajg/abstract/00000434-201710001-02334~psuedo-budd-chiari-syndrome-an-uncommon-presentation-of-very?redirectionsource=fulltextview
14. Pagan, Andrea S MD; Arroyo, Yadis MD; Bertran-Rodriguez, Carlos MD. S2743 Pseudo Budd-Chiari Syndrome in Decompensated Alcoholic Hepatitis and Liver Cirrhosis. The American Journal of Gastroenterology 115():p S1433-S1434, October 2020. | DOI: 10.14309/01.ajg.0000713020.26001.b1
15. EASL Clinical Practice Guidelines: Vascular diseases of the liver Journal of Hepatology, 2015; 64, 179-202 https://doi.org/10.1016/j.jhep.2015.07.040
16. Virmani, V, Khandelwal, N, Kang, M et al. MDCT venography in the evaluation of inferior vena cava in Budd-Chiari syndrome. Indian J Gastroenterol 28, 17–23 (2009). https://doi.org/10.1007/s12664-009-0004-5
17. Juan Carlos Garcia–Pagán, Mathis Heydtmann, Sebastian Raffa, Aurélie Plessier, Sarwa Murad, Federica Fabris et al, TIPS for Budd-Chiari Syndrome: Long-Term Results and Prognostics Factors in 124 Patients, Gastroenterology,Volume 135, Issue 3,2008,Pages 808-815,ISSN 0016-5085,https://doi.org/10.1053/j.gastro.2008.05.051.
18. Hasija, R P, Nagral, A., Marar, S. et al. Transjugular intrahepatic portosystemic shunt (TIPSS) for Budd Chiari syndrome. Indian Pediatr 47, 527–528 (2010). https://doi.org/10.1007/s13312-010-0081-7
19. Fitsiori K, Tsitskari M, Kelekis A et al. Transjugular Intrahepatic Portosystemic Shunt for the Treatment of Budd–Chiari Syndrome Patients: Results From a Single Center. Cardiovasc Intervent Radiol 37, 691–697 (2014). https://doi.org/10.1007/s00270-013-0697-9
20. Brancatelli, Giuseppe and Vilgrain, Valerie and Federle, et al. Budd-Chiari Syndrome: Spectrum of Imaging Findings,American Journal of Roentgenology,188,2,W168-W176,2007,doi = 10.2214/AJR.05.0168 https://doi.org/10.2214/AJR.05.0168
21. Jimmy Daza, Isabella Wiest, Xin Wu Cui, Bo Zhang, Yunhao Chen, Yi Dong et al; Ultrasound Characteristics of Budd-Chiari Syndrome: A Multicenter Analysis. Dig Dis 2026; https://doi.org/10.1159/000549007
22. Porrello G, Mamone G, & Miraglia, R. (2023). Budd-Chiari Syndrome Imaging Diagnosis: State of the Art and Future Perspectives. Diagnostics (Basel, Switzerland), 13(13), 2256. https://doi.org/10.3390/diagnostics13132256.