

Primary Biliary Cholangitis in A Male Patient: A Case Report

M SAHA^a, MSAURKO^b, ATM R ISLAM^c, R GUPTA^d

Abstract

Primary biliary cholangitis (PBC) is a chronic autoimmune cholestatic liver disease characterized by progressive destruction of small intrahepatic bile ducts. It predominantly affects middle-aged women, and occurrence in males is uncommon. We report a case of a 53-year-old male presenting with long-standing pruritus, recent onset jaundice, and pedal edema. Laboratory evaluation revealed markedly elevated alkaline phosphatase, positive antinuclear antibody (ANA), and antimitochondrial antibody (AMA) with negative viral markers and no evidence of biliary obstruction on imaging.

Introduction

Primary biliary cholangitis (PBC), previously known as primary biliary cirrhosis, is an uncommon T-cell-mediated autoimmune cholestatic liver disease characterized by progressive destruction of small intrahepatic bile ducts^{1,2}. This leads to cholestasis, fibrosis, and eventually cirrhosis.

PBC predominantly affects females between 40 and 60 years of age, with a female-to-male ratio of approximately 10:3-5. The reported incidence and prevalence are approximately 1.76 and 14.6 per 100,000 population, respectively⁶.

Many patients are asymptomatic at diagnosis (>60%)w, while others present with fatigue and pruritus. Additional features include hyperlipidemia and osteopenia^{8,9}.

- Professor Madhusudan Saha, Professor of Gastroenterology, Sylhet Women's Medical College, Sylhet, Bangladesh
- Dr. Maithil Saha Aurko, Student 5th year MBBS, Dhaka Medical College, Dhaka, Bangladesh
- Dr. ATM Redwanul Islam, Medical Officer, Gastroenterology, Sylhet Women's Medical College, Sylhet, Bangladesh
- Dr. Rahul Gupta, Asst Registrar, Gastroenterology, Sylhet Women's Medical College, Sylhet, Bangladesh

Address of Correspondence: Dr. Madhusudan Saha, Professor of Gastroenterology, Sylhet Women's Medical College, Sylhet, Bangladesh, Email: madhunibedita@gmail.com ; Mobile: +8801711367847

Received: 02 Dec., 2025

Accepted: 08 March, 2026

Fibroscan suggested advanced fibrosis, and endoscopy revealed esophageal varices. A diagnosis of PBC with cirrhotic changes was made. The patient was treated with ursodeoxycholic acid along with supportive therapy. This case highlights the importance of considering PBC in male patients with cholestatic liver disease for early diagnosis and management.

Keywords: Primary biliary cholangitis, male patient, cholestasis, autoimmune liver disease

(J Bangladesh Coll Phys Surg 2026; 44: 138-139)

DOI: <https://doi.org/10.3329/jbcps.v44i2.89349>

Diagnosis is based on cholestatic liver enzyme abnormalities, positive autoimmune markers (ANA and AMA), and absence of biliary obstruction or viral hepatitis¹².

PBC in males is rare, and data from Bangladesh are limited. Here, we report a case of PBC in a male patient.

Case Presentation

A 53-year-old male, working in a Middle Eastern country, presented with generalized pruritus for more than two years, swelling of both legs for one month, and dark-colored urine for one week. There was no prior history of jaundice, alcohol intake, or hepatotoxic drug use.

On examination, the patient had mild icterus, anemia, and bilateral pedal edema. Multiple scratch marks with hyperpigmentation and keratinization were noted over the lower limbs. Abdominal examination revealed hepatomegaly.

Investigations

Laboratory findings showed:

- Total bilirubin: 6.3 mg/dL
- ALT: 85 IU/L
- AST: 128 IU/L
- Alkaline phosphatase: 1700 IU/L
- Serum albumin: 2.94 g/dL
- Prothrombin time: 17 seconds (control 12 sec)

Viral markers (HBsAg, Anti-HCV, Anti-HEV IgM, Anti-HAV IgM) were negative.

Autoimmune markers revealed:

- ANA: >400 AU/mL
- AMA: Positive (1:40)

Ultrasonography showed hepatosplenomegaly and later mild ascites without biliary obstruction. Fibroscan demonstrated liver stiffness of 23.4 kPa, suggestive of advanced fibrosis. Upper GI endoscopy revealed grade II esophageal varices and portal hypertensive gastropathy.

Based on clinical, biochemical, and serological findings, a diagnosis of primary biliary cholangitis with cirrhotic changes was made.

The patient was treated with ursodeoxycholic acid (12–15 mg/kg/day) along with carvedilol, spiro-nolactone, furosemide, and human albumin infusion.

Discussion

Primary biliary cholangitis is a chronic autoimmune cholestatic liver disease that predominantly affects women, making this case in a male patient uncommon³⁻⁵.

The hallmark features of PBC include persistent elevation of alkaline phosphatase, presence of AMA, and absence of biliary obstruction¹². AMA is highly specific (~97%) and sensitive (~90%) for PBC¹².

Our patient presented with long-standing pruritus followed by jaundice and edema, indicating late presentation. The markedly elevated alkaline phosphatase and positive ANA and AMA strongly supported the diagnosis. Imaging excluded biliary obstruction, and Fibroscan findings suggested advanced fibrosis.

Cirrhosis at the time of diagnosis occurs in approximately 20–25% of patients¹³. The presence of esophageal varices further indicated portal hypertension.

Liver biopsy was not performed, as the diagnosis was established based on characteristic biochemical and serological findings.

The standard treatment of PBC is ursodeoxycholic acid (UDCA), which improves biochemical parameters and

delays disease progression¹⁴⁻¹⁵. Early diagnosis and treatment significantly improve transplant-free survival.

Conclusion

Primary biliary cholangitis, though rare in males, should be considered in patients presenting with cholestatic liver function abnormalities and pruritus. Early diagnosis using serological markers and prompt initiation of UDCA therapy can delay disease progression and improve outcomes.

References

1. Engel B, Taubert R, Jaeckel E, Manns MP. *Liver Int.* 2020;40(Suppl 1):149–153. doi:10.1111/liv.14378
2. Lleo A, et al. *Semin Liver Dis.* 2020;40(1):34–48. doi:10.1055/s-0039-1697617
3. Younossi ZM, et al. *Am J Gastroenterol.* 2019;114(1):48–63. doi:10.1038/s41395-018-0390-3
4. Lleo A, et al. *Autoimmun Rev.* 2008;7(8):626–630. doi:10.1016/j.autrev.2008.06.009
5. Lleo A, et al. *Sci Rep.* 2016;6:25906. doi:10.1038/srep25906
6. Trivella J, et al. *Hepatol Commun.* 2023;7(6):e0179. doi:10.1097/HC9.0000000000000179
7. Prince MI, et al. *Gut.* 2004;53(6):865–870. doi:10.1136/gut.2003.023937
8. Khanna A, et al. *Best Pract Res Clin Gastroenterol.* 2018;34–35:41–47. doi:10.1016/j.bpg.2018.06.007
9. Dyson JK, et al. *Aliment Pharmacol Ther.* 2016;44(10):1039–1050. doi:10.1111/apt.13794
10. Gershwin ME, Mackay IR. *Hepatology.* 2008;47(2):737–745. doi:10.1002/hep.22042
11. McNally RJ, et al. *Hepatology.* 2009;50(4):1169–1174. doi:10.1002/hep.23139
12. Nalbandian G, et al. *Am J Gastroenterol.* 1999;94(9):2482–2486. doi:10.1111/j.1572-0241.1999.01380.x
13. Beuers U, et al. *Gastroenterology.* 2015;149(6):1627–1629
14. Parés A. *Med Clin (Barc).* 2018;151:242–249. doi:10.1016/j.medcli.2017.12.021
15. Gulamhusein AF, Hirschfield GM. *Nat Rev Gastroenterol Hepatol.* 2020;17(2):93–110. doi:10.1038/s41575-019-0226-7