
Hilar Tuberculous Lymphadenitis Presenting as Obstructive Jaundice in a Previously Cholecystectomized Patient: A Case Report

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ABSTRACT:

Background: Tuberculosis involving abdominal lymph nodes is an uncommon but recognized cause of obstructive jaundice. Because its clinical and radiological presentation may mimic malignancy of the biliary tract or pancreatic head, diagnosis is often challenging.

Case presentation: We report the case of a 40-year-old woman with a history of cholecystectomy performed four years earlier and followed for iron-deficiency anemia. She was admitted with a 20-day history of progressive cholestatic jaundice without digestive hemorrhage, associated with general deterioration. Clinical examination showed generalized mucocutaneous jaundice with stable hemodynamic status. Magnetic resonance cholangiography demonstrated a conglomerate of hilar and peri-pancreatic lymphadenopathy causing dilation of intrahepatic and extrahepatic bile ducts. Thoracic CT was normal. Diagnostic laparoscopy revealed multiple lymph nodes at the hepatic hilum, the largest measuring 2 cm, with purulent content. Biopsy with intraoperative frozen section revealed non-necrotizing tuberculoid granulomatous adenitis.

Conclusion: Tuberculous lymphadenitis of the hepatic hilum is a rare but important differential diagnosis of obstructive jaundice. Surgical exploration with histological confirmation remains crucial when imaging findings mimic malignancy. Early diagnosis allows appropriate antituberculous treatment and avoids unnecessary extensive surgery.

INTRODUCTION

Obstructive jaundice is most frequently caused by gallstones, biliary strictures, pancreatic tumors, or cholangiocarcinoma. However, rare etiologies such as infectious lymphadenitis may also produce biliary compression. Abdominal tuberculosis remains a major health problem in endemic regions and can involve the gastrointestinal tract, peritoneum, lymph nodes, and solid organs.

Tuberculous lymphadenitis located in the hepatoduodenal ligament or hepatic hilum is uncommon and may lead to extrinsic compression of the common bile duct. This clinical presentation is rare and often mimics malignant disease on imaging studies. Because of this similarity, diagnosis is frequently established only after surgical exploration and histopathological examination.

We report a case of hilar tuberculous lymphadenitis revealed by cholestatic jaundice in a 40-year-old woman, highlighting the diagnostic difficulties and reviewing the relevant literature.

METHODS

Clinical data were obtained from the patient's hospital records after informed consent. Laboratory findings, imaging studies, operative findings, and histopathological results were reviewed. The diagnostic strategy included abdominal MRI with biliary sequences (MRCP), thoracic CT scan, and exploratory laparoscopy with lymph node biopsy. Histological examination with frozen section analysis was performed intraoperatively to guide management.

RESULTS (CASE PRESENTATION)

A 40-year-old woman with a history of laparoscopic cholecystectomy performed four years earlier presented with progressive cholestatic jaundice evolving for 20 days. She had been followed for iron-deficiency anemia treated with oral iron supplementation. The clinical course was associated with deterioration of general condition without gastrointestinal bleeding.

At admission, the patient was conscious and hemodynamically stable. Physical examination revealed generalized cutaneous and mucosal jaundice. Abdominal examination showed healed trocar scars from the previous cholecystectomy, a soft abdomen without palpable mass, and no peripheral lymphadenopathy. Rectal examination revealed acholic stools and dark urine was reported by the patient. Gynecological examination was unremarkable.

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Laboratory investigations demonstrated hemoglobin of 10 g/dL, leukocyte count of 4200/mm³, and total bilirubin of 59 mg/L with disturbed liver function tests consistent with cholestasis. Coagulation parameters were within normal limits.

Magnetic resonance cholangiography performed on 02/02/2026 demonstrated a conglomerate of lymphadenopathy at the hepatic hilum with extension to pre- and retro-pancreatic regions. The largest node measured 27 mm and caused compression of the common bile duct with dilation of both intrahepatic and extrahepatic bile ducts.

Thoracic CT scan performed on 11 February showed no pulmonary or mediastinal abnormalities.

The patient underwent exploratory laparoscopy on 06/03/2026. Intraoperative exploration revealed multiple lymph nodes forming a mass at the hepatic hilum. The largest node measured approximately 2 cm. Upon manipulation, purulent material was evacuated. A biopsy of the lymph node was performed and sent for frozen section analysis. Histopathological examination demonstrated granulomatous lymphadenitis with tuberculoid features without necrosis.

Samples were sent for definitive histopathology and microbiological studies. The postoperative course was uneventful and antituberculous therapy was planned.



Figure 1: laparoscopic view of hepatic hilum nodule with derived from pus

DISCUSSION

Abdominal tuberculosis represents approximately 5–12% of extrapulmonary tuberculosis cases. Lymph node involvement is one of the most frequent manifestations and may occur in mesenteric, periportal, or peripancreatic stations. However, isolated hilar lymphadenitis causing obstructive jaundice remains rare.

The pathophysiology involves lymph node enlargement leading to extrinsic compression of the common bile duct or hepatic duct confluence. In some cases, inflammatory fibrosis may also contribute to biliary obstruction. Clinically, patients usually present with jaundice, abdominal pain, weight loss, and occasionally fever. These nonspecific symptoms overlap with those of malignant biliary obstruction.

Radiological findings are often misleading. On CT or MRI, tuberculous lymph nodes may appear as hypodense masses with peripheral enhancement or central necrosis. When located at the hepatic hilum or pancreatic head region, they may simulate cholangiocarcinoma, pancreatic carcinoma, or metastatic lymphadenopathy. Magnetic resonance cholangiopancreatography typically shows bile duct dilation upstream of the compressive lesion but does not reliably distinguish benign from malignant causes. Definitive diagnosis therefore relies on histopathological examination. The typical finding is granulomatous inflammation with epithelioid cells and Langhans giant cells, sometimes associated with caseous necrosis. However, non-necrotizing granulomas may also occur, particularly in early disease or partially treated cases. Microbiological confirmation by culture or PCR further supports the diagnosis but may not always be available.

Several diagnostic approaches have been described. Endoscopic ultrasound-guided fine needle aspiration has emerged as a minimally invasive method to sample periportal lymph nodes. Nevertheless, surgical exploration remains necessary when malignancy cannot be excluded or when tissue sampling is not feasible.

Management is primarily medical. Standard antituberculous therapy leads to regression of lymph node size and resolution of biliary obstruction in most cases. In selected patients with severe obstruction, temporary biliary drainage using endoscopic or percutaneous techniques may be required.

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Our observation illustrates the diagnostic challenge posed by this rare condition. In a patient presenting with obstructive jaundice and hilar lymphadenopathy in a tuberculosis-endemic area, tuberculous lymphadenitis should be considered in the differential diagnosis to avoid unnecessary radical surgery.

CONCLUSION

Tuberculous lymphadenitis of the hepatic hilum is a rare cause of obstructive jaundice that can mimic hepatobiliary malignancy. Imaging findings alone are insufficient for diagnosis. Histological confirmation remains essential, often obtained through minimally invasive procedures or surgical exploration. Early recognition allows prompt initiation of antituberculous therapy and favorable outcomes.

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