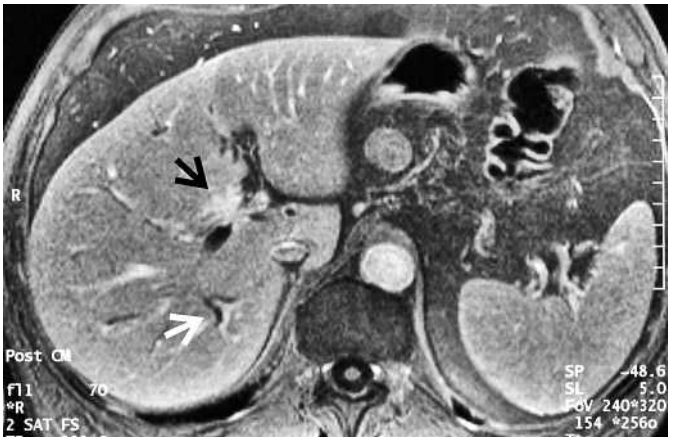


Figure **1** Abdominal magnetic resonance imaging (MRI) showed a hilar-located contrast-enhanced mass in the liver (20 mm diameter; black arrow), causing stricture of the ducti hepatici and intrahepatic cholestasis (white arrow).

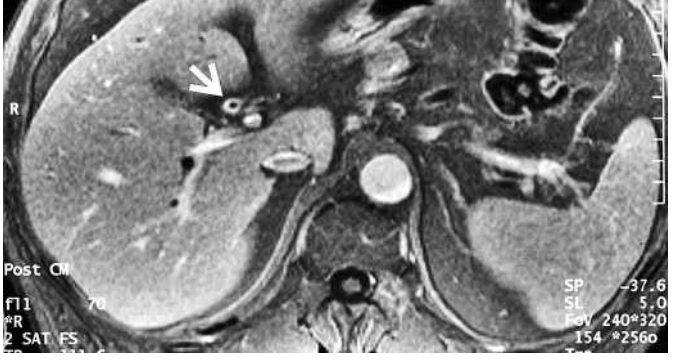


Figure **2** MRI view showing thickening and contrast enhancement of the slightly dilated (9 mm) common bile duct wall (arrow).

A 68-year-old man was admitted to hospital in May 2002 because of weight loss of 12 kg within 1 year. Blood tests showed elevated aspartate aminotransferase 224 U/l (normal range < 22), alanine aminotransferase 365 U/l (normal range < 24), alkaline phosphatase 513 U/l (normal range 60 – 170), γ-glutamyl transpeptidase 1.031 U/l (normal range < 28), total serum bilirubin 2.2 mg/dl (normal range < 1.0), direct serum bilirubin 1.8 mg/dl (normal range < 1), and serum angiotensin-converting enzyme 21 U/l (normal range < 20). There were no abnormalities regarding viral hepatitis markers, antibody profiles, or the CA 19 – 9 value. The patient had been diagnosed with sarcoidosis 5 years previously.

Abdominal magnetic resonance imaging (MRI) showed a hilar-located mass in the liver (20 mm diameter; Figure **1**) causing stricture of the ducti hepatici and intrahepatic cholestasis. Moreover, thickening along a length of 38 mm of the slightly dilated (9 mm) common bile duct wall was found (Figure **2**). Endoscopic retrograde cholangiopancreatography (ERCP) showed a hilar infiltration (Figure **3**) similar to cholangiocarcinoma. Biliary biopsy specimens did not exhibit malignancy. Liver biopsy revealed noncaseating epitheloid cell granulomas (Figure **4**), predominantly in the portal area, compatible with sarcoidosis. After dilation of the left hepatic duct stricture the patient was treated with oral prednisolone.

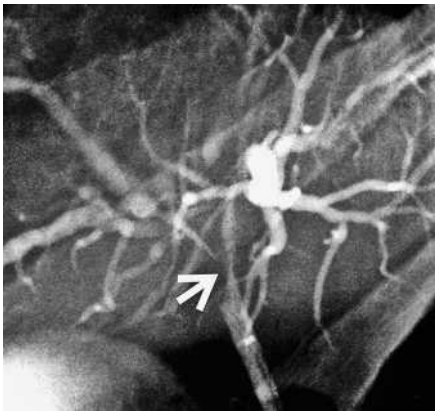

Figure **3** Cholangiogram showing strictures (arrow) of the ductus hepaticus communis (10 mm length), and both the right and left ducti hepatici (20 mm and 10 mm length, respectively). Note the dilation of the intrahepatic ducts.

At follow-up 6 months later, the pathologic findings from blood tests, MRI and ERCP had resolved (Figure **5**), and 47 months later the patient had no clinical signs of malignancy.

Overlap of sarcoidosis and primary biliary cirrhosis or primary sclerosing cholangitis has been described [1,2]. However, clinical signs of bile duct obstruction due to sarcoidosis alone have rarely been reported. Enlarged granulomatous lymph nodes or, as in our patient, masses, are suspected to contribute to biliary obstruction [3,4]. Although the combination of both a hilar-located sarcoidoma and an affection of the ductus hepatocholedochus due to sarcoidosis have not been previously reported, it seems likely that the rare feature of biliary obstruction is underreported in this setting. Awareness of sarcoidosis may facilitate its recognition in the differential diagnosis of painless obstructive jaundice.

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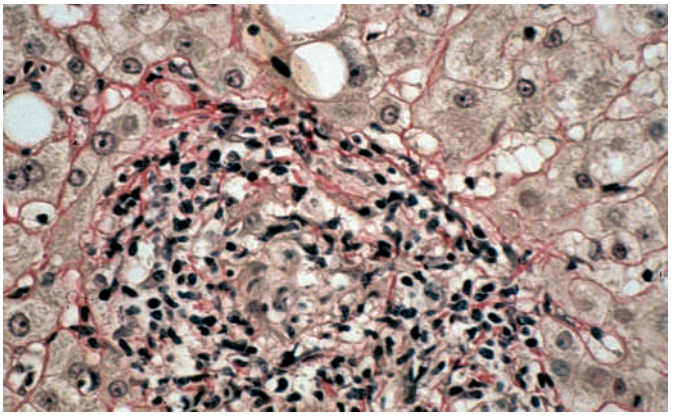

Figure **4** Histopathological examination of the liver biopsy specimen revealed a noncaseating epitheloid cell granuloma.

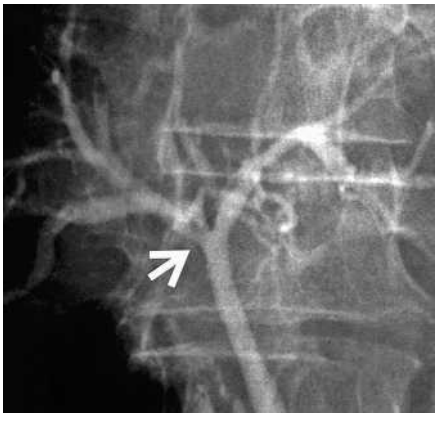

Figure **5** Cholangiography performed 6 months after initiation of treatment with corticosteroids revealed that the pathologic findings (arrow) had been resolved.

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