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DOI

[10.1007/s002619900223](https://doi.org/10.1007/s002619900223)

Publication date

1997

Published in

Abdominal Imaging

[Link to publication](#)

Citation for published version (APA):

van Beek, E. J. R., Smits, N. J., & Reeders, J. W. A. J. (1997). Cat scratch disease as a rare cause of obstructive jaundice: a case report. *Abdominal Imaging*, 22, 418-420. <https://doi.org/10.1007/s002619900223>

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Cat scratch disease as a rare cause of obstructive jaundice: a case report

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Received: 20 March 1996/Accepted: 10 April 1996

Ultrasonography has become the first imaging modality in obstructive jaundice; it has the ability to define the cause and level of obstruction [1–3]. The most common causes depend on the population studied and include neoplasm (ranging from 18 to 86%) [1–3], calculous disease (varying from 5 to 48%) [1–3], and pancreatitis (in 2–27% of patients studied) [1–3]. Recently, we encountered a diagnostic challenge of a patient in whom silent obstructive jaundice developed, which proved to be of an etiology not previously reported.

Case report

A 56-year-old male pulmonologist presented with a 4-month history of progressive malaise, vague abdominal complaints of fasting pain radiating from the left upper abdomen to the back, anorexia, and weightloss. Within several days, obstructive jaundice had developed.

The previous medical history revealed Strongyloidiasis infection in 1946 and two B.C.G. vaccinations because the patient was at high-risk for tuberculosis infection. Pulmonary tuberculosis was excluded by chest radiography, which was otherwise unremarkable.

Ultrasonography (US) of the abdomen demonstrated dilatation of the common bile duct (CBD) caused by an extrapancreatic, multilobulated, hypoechogenic solid process (7.8×3.2 cm), which was located ventral to the inferior vena cava and cranialateral to the head of the pancreas (Fig. 1A). Doppler US showed signs of considerable narrowing of the portal venous system and encasement of the hepatic artery. Multiple US-guided cytological punctures were negative for malignant cells.

Computed tomography showed a lobulated hypodense mass with low-attenuation after intravenous contrast injection, which appeared to be situated in the pancreatic head, and subsequent severe narrowing of the portal vein (Fig. 1B). The splenic vein and the superior mesenteric vein were patent. The pancreas was normal.

Endoscopic retrograde cholangiopancreatography showed a normal pancreatic duct and marked, but smooth narrowing of the mid CBD. A polyethylene endoprosthesis was inserted for drainage (Fig. 1C).

At laparotomy the process was found to consist of enlarged lymph nodes located in the hepatoduodenal ligament and adjacent to the pancreas. A debulking procedure was performed: histopathology revealed granulomatous lymph nodes with central necrosis. Malignancy could be ruled out, and the endoprosthesis was removed. A ^{67}Ga gallium scintigram showed normal distribution without evidence of lymphadenopathy elsewhere.

In view of the histopathology (lymphadenitis with granulomatous lesions and central necrosis) and the occupation of the patient, a preliminary diagnosis of tuberculosis was assumed. However, all cultures for *Mycobacterium tuberculosis* remained negative, and the diagnosis was rejected.

Histopathology clearly excluded the possibility of malignancy or lymphoma, while sarcoidosis was less likely, since both the chest radiograph and ^{67}Ga gallium scintigraphy were normal.

Serology subsequently revealed that a recent infection with *Bartonella hensela*, which causes cat scratch disease, had taken place (positive polymerase chain reaction (PCR)). This was confirmed by a specific staining technique (IgM 25 negative and IgM 2379 positive) of the histological preparation of the lymph nodes, although no bacilli could be detected using Warthin–Starry silver impregnation staining. The patient was subsequently treated with claritromycin and made an uneventful recovery.

Discussion

The differential diagnosis of obstructive jaundice in the adult consists mainly of neoplasm (pancreatic and periampullary carcinoma, cholangiocarcinoma, lymphoma, metastasis), calculous disease, cholangitis (primary or AIDS related), and pancreatitis. Iatrogenic causes, such as postoperative strictures and inadvertent clipping of the CBD, are more frequently seen in this era of laparoscopic surgery. Finally, rarer causes include lymphadenopathy in the liver hilar region (benign as in tuberculosis, sarcoidosis, and toxoplasmosis or malignant) and lymphadenopathy in the hepatoduodenal ligament, which may cause obstruction of the CBD.

Cat scratch disease was first described in 1950, and is usually a benign, self-limiting illness, characterized

Fig. 1. **A** Oblique US scan: multilobulated hypoechoic well-delineated mass clearly extrapancreatic in origin. **B** Contrast-enhanced spiral CT shows multiple low attenuated lobulated lesions in the region of the pancreatic head suggesting intrapancreatic tumor. **C** ERCP showing a 4 cm long, smooth stenosis in the mid to distal CBD to just beyond the orifice of the cystic duct.

by chronic regional lymphadenopathy of more than 3 weeks duration [4]. A recent analysis estimated the number of patients discharged from hospital with this diagnosis at 0.77–0.86 per 100,000 [5]. In 1983, a small bacillus was discovered to cause the illness [6]. The bacillus is generally transmitted by cats (4–9% of family members scratched by the same cat may develop cat scratch disease) and usually results in a dermatologic reaction followed by lymphadenitis of the arms, axilla, or even generalized lymphadenopathy in severe cases, which may persist up to 24 months [4]. In approximately 2% of patients complications may arise, affecting the central nervous system, liver, spleen, lung, bone, and skin [7, 8]. To our knowledge, isolated lymphadenitis in the hepatoduodenal ligament has not been described before.

Cat scratch disease may be detected by the presence of three of four criteria [6]: (1) a history of animal (usually a cat) contact with presence of a scratch; (2) aspiration of sterile pus from the node or culture which excludes other etiology; (3) a positive cat scratch skin test and/or positive PCR serology; (4) a lymph node biopsy revealing histopathology consistent with cat scratch disease and/or demonstration of organisms by typical staining technique. In our patient, the first three criteria were

met. It should be realized that, with adequate antibiotic therapy, the prognosis is excellent.

In patients in whom US shows extrapancreatic tumour, lymphadenopathy must be suspected and further diagnostic work-up is mandatory. As shown in our case, this may result in compression of both the portal vein and the CBD. Tuberculosis, sarcoidosis, toxoplasmosis, and cat scratch need to be differentiated in granulomatous lymphadenitis. In patients who have not been vaccinated with BCG, skin testing for tuberculosis will often reveal a positive reaction, while in vaccinated patients chest radiography should be performed. Sarcoidosis is best diagnosed using chest radiography and ^{67}Ga gallium scintigraphy. Toxoplasmosis may be shown by positive Sabin–Feldman serology.

Generally, the more severe diagnoses will be regarded. If malignancy is excluded and sterile pus has been obtained, cat scratch disease must be considered. Although it is generally a self-limiting and benign disease, a more severe clinical course may be encountered, as demonstrated in our case. If lymphadenopathy is suspected as a cause of obstructive jaundice on the basis of a multilobulated mass on US or CT, cytological or histopathological investigation is always required. Furthermore, specific serological and

staining techniques need to be employed if no obvious etiology is discovered.

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