Pyogenic hepatic abscesses secondary to choledocholithiasis eight years post-cholecystectomy: A case report

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A R T I C L E  I N F O
Keywords:
Hepatic abscess
Choledocholithiasis
Cholecystectomy

A B S T R A C T
Cholecystectomy is the gold standard surgical treatment for acute calculous cholecystitis. Only approximately 0.4 % of these patients subsequently develop choledocholithiasis. The incidence of hepatic abscesses in these patients is unknown, but is likely low, considering there are approximately 2–15 cases of hepatic abscesses per 100,000 people in the US. The authors report the case of a 62-year-old man whose CT scan revealed hepatic abscesses secondary to choledocholithiasis, eight years after a cholecystectomy.

1. Introduction
Cholecystectomy is the gold standard surgical treatment for acute calculous cholecystitis [1–3]. Post-cholecystectomy syndrome refers to the development of symptoms such as abdominal pain, fever, and jaundice after surgery, which may be due to both biliary (e.g. retained or recurrent calculi, injury or scarring to the biliary tree) and non-biliary (e.g. psychosomatic or gastrointestinal) etiologies [4]. This syndrome has an incidence reportedly as high as 40 % of patients who undergo cholecystectomy, with a postoperative onset ranging from 2 days to 25 years [5]. However, post-cholecystectomy choledocholithiasis is relatively rarer, with an approximate incidence of 0.4 % [6].

An uncommon complication of choledocholithiasis is the development of pyogenic hepatic abscesses [7]. These abscesses often involve polymicrobial infection, usually with E. coli and Klebsiella [8,9]. If left untreated, these abscesses can progress to peritonitis or sepsis, but when treated with antibiotics and percutaneous drainage, the mortality is reduced to approximately 10–30 % [10].

There is a lack of data on the incidence of post-cholecystectomy choledocholithiasis leading to hepatic abscesses. However, it is estimated that there are 2–15 cases of hepatic abscesses per 100,000 people in the US [8–10], mostly of bacterial etiology, and approximately half of these are caused by cholangitis [11]. Given the infrequency of cholangitis-induced pyogenic hepatic abscesses, as well as the 0.4 % incidence of choledocholithiasis in post-cholecystectomy patients [6], pyogenic hepatic abscesses secondary to post-cholecystectomy choledocholithiasis comprise a rare entity.

2. Case presentation
The patient is a 62-year-old male who presented to ER with confusion, jaundice, and abdominal pain. His past medical history was significant for alcohol use disorder and symptomatic cholelithiasis with a cholecystectomy eight years ago. Two years ago, the patient underwent endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy for symptomatic choledocholithiasis. However, the procedure was terminated early due to patient agitation, and confirmation of complete stone removal was not possible. A week later, the patient presented with sepsis and a new hypodense liver lesion was noted on CT, suggesting abscess formation. He was treated non-operatively and improved on antibiotics. Follow-up CT showed resolution of the abscess and mild residual intrahepatic duct dilatation (Fig. 1). A subsequent ERCP only showed mild residual biliary dilatation.

At the time of the current presentation, the patient was tachycardic at 121 beats per minute, febrile at 38.3 °C, and had elevated leukocytes (25.7 × 109/L), alkaline phosphatase (1048 U/L), alanine aminotransferase (144 U/L), and total bilirubin (302 μmol/L), with mildly elevated lipase (180 U/L). Blood cultures were drawn, and he was empirically started on piperacillin-tazobactam.

Since there was limited availability of ultrasound imaging overnight, a contrast-enhanced abdominal CT was performed (Fig. 2). This showed increased intrahepatic biliary dilatation as well as multiple rounded

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https://doi.org/10.1016/j.ejro.2020.100292
Received 26 October 2020; Received in revised form 21 November 2020; Accepted 23 November 2020
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septated cystic structures measuring up to 5.6 cm with subtle peripheral enhancement and surrounding parenchymal edema, most consistent with hepatic abscesses. The common bile duct (CBD) was dilated up to 1.2 cm in the porta hepatis and contained a new ovoid hyperdense filling defect, in keeping with choledocholithiasis. By this point, the patient’s blood cultures had grown *E. coli*, so he was switched to ciprofloxacin and metronidazole.

ERCP was performed, with removal of a stone and placement of a plastic endobiliary stent (Fig. 3). The next day, the intrahepatic biliary dilatation had significantly improved on ultrasound, and the patient’s bilirubin dropped to 190. Post-ERCP CT confirmed removal of the stone and resolution of CBD dilation. However, there was diffuse edematous wall thickening of the CBD, consistent with cholangitis (Fig. 4). Follow-up CT one month later showed significant improvement in the hepatic abscesses on continued antibiotic therapy, with only mild residual intrahepatic biliary dilatation (Fig. 5).

3. Discussion

This case represents an unusual occurrence of choledocholithiasis and subsequent pyogenic hepatic abscesses in a patient who had undergone a cholecystectomy several years prior. Choledocholithiasis post-cholecystectomy is usually discovered within 4 years [5], rendering this case of hepatic abscess formation 8 years after a cholecystectomy an uncommon occurrence.

In our literature search, we found a few case reports of hepatic abscesses occurring years after biliary tree surgery. Similar to our case, Toshikuni et al. reported a case of pyogenic hepatic abscess 5 years after a cholecystectomy, which was treated with antibiotics and percutaneous drainage [11]. However, their patient had also undergone a choledochoduodenostomy for obstruction secondary to autoimmune

Fig. 1. Baseline contrast-enhanced CT from previous admission 2 years ago demonstrates mild intrahepatic biliary dilatation (arrow) and no evidence of hepatic abscess.

Fig. 2. Contrast-enhanced CT of the abdomen from the current presentation (a) shows multiple new fluid attenuation structures within the liver parenchyma, most consistent with hepatic abscesses (arrows). There is also increased intrahepatic biliary dilatation (circle). Narrowing the windows for evaluation of the liver parenchyma (b) better demonstrates ill-defined hypo-enhancing parenchyma around the cystic structures, supportive of abscesses and not simple cysts (arrows). A more inferior slice (c) demonstrates central intrahepatic biliary dilatation (arrow). Smaller scattered hypodense foci are in keeping with smaller abscesses. An image at the level of the common bile duct (d) demonstrates an ovoid intraluminal hyperdense filling defect (arrow), confirmed as choledocholithiasis on ERCP.
pancreatitis. Yu et al. also reported pyogenic hepatic abscesses occurring 7 years after a choledochojejunostomy [12].

Most often, multiple hepatic abscesses in the setting of biliary tree surgery develop shortly after the operation [13]. For example, Perali et al. describe a case of multiple hepatic abscesses a week after pancreaticoduodenectomy [14]. Güngör et al. report a hepatic abscess which developed eight days after laparoscopic cholecystectomy following ERCP and sphincterotomy [15], Valderrama et al. report a similar abscess two weeks after laparoscopic cholecystectomy following ERCP [16], and Qandeel et al. describe a hepatic abscess one week after a laparoscopic cholecystectomy [17].

Although these cases all involve different surgical procedures, timelines of infection, pathogens, and distributions of abscesses, they may be diagnosed and managed similarly. Most patients present with Charcot’s triad of jaundice, fever, and right upper quadrant abdominal pain [5]. They are diagnosed with the aid of ultrasound and/or CT, which have sensitivities of 85 % and 97 %, respectively [18].

On ultrasound, newly formed hepatic abscesses are usually poorly demarcated and hypoechoic, although echogenicity is variable [19]. At this stage, abscesses can be confused for solid neoplasms on ultrasound; however, they should not demonstrate internal vascularity or flow on Doppler [18]. Eventually, abscesses become more hypoechoic as central liquefaction progresses, until they appear as cystic lesions [19], with gas bubbles present in approximately 20 % of abscesses [20].

Hepatic abscesses also demonstrate this maturity-dependent density on contrast-enhanced CT, though in general, they appear as centrally hypodense lesions with peripheral enhancement, occasionally containing gas in the form of bubbles or air-fluid levels [18]. Some characteristic signs of pyogenic hepatic abscesses on contrast-enhanced CT include the “double target sign” and the “cluster sign”, which help distinguish them from simple cysts or other cystic neoplasms [20]. The “double target sign” consists of fluid-filled hypodense lesions surrounded by an inner rim, which represents the enhancing abscess membrane, and an outer ring, which consists of hypodense edematous parenchyma [20]. The “cluster sign” refers to multiple small locules forming a larger septated abscess cavity [20].

If there is suspicion for biliary obstruction, ERCP is generally indicated. If there is no evidence of biliary tree involvement, the pathogen may have originated in the bowels and ascended the portal vein, resulted from hematogenous spread from another source, or directly entered the liver by surgery or trauma [7]. However, if cholecodolithiasis is present, treatment mainly involves removing the stone during ERCP, along with antibiotic therapy, and percutaneous drainage for large abscesses [5].
While the treatment is well established, pyogenic hepatic abscesses secondary to choleclocholithiasis are still life-threatening if they are not detected and therapy initiated promptly [8]. This case shows how such complications can still occur many years after a cholecystectomy, furthering our understanding of this entity and emphasizing the importance of including it in our differential diagnoses.

Ethical Statement
Not applicable.

Funding Source
None.

Declaration of Competing Interest
The authors declare that they have no conflict of interest.

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