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Lemmel's Syndrome: An Unusual Cause of Angiocholitis and Acute Pancreatitis, Successfully Treated with ERCP

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Abstract **Case Report**

Lemmel syndrome is a rare clinical entity characterised by the presence of a periampullary duodenal diverticulum leading to compression and dilatation of the common bile ducts, accompanied by obstructive jaundice. In a small percentage of cases, it may be associated with pancreaticobiliary complications such as cholangitis or acute pancreatitis, and there are no standardised approaches to its treatment. We present a case of Lemmel syndrome in a 53-year-old woman presenting with angiocholitis and acute pancreatitis successfully treated with ERCP.

Keywords: Lemmel syndrome, Duodenal diverticulum, jaundice, acute pancreatitis, ERCP.

INTRODUCTION

Lemmel's syndrome is a rare disorder characterised by extrinsic compression of the distal common bile duct by a periampullary diverticulum, causing obstructive jaundice in the absence of choledocholithiasis or pancreaticobiliary tumour. The duodenal diverticulum may also cause dysfunction of the sphincter of Oddi, leading to pancreatitis and cholangitis. To date, very few cases have been reported of this condition, which was first named in 1934 after Dr Gerhard Lemmel [1].

This syndrome can mimic several benign and malignant pathologies in the periampullary region. We report a case of a 53-year-old woman with obstructive jaundice due to Lemmel's syndrome secondary to a periampullary diverticulum complicated by acute pancreatitis successfully treated with ERCP.

CASE REPORT

A 53-year-old woman with a history of cholecystectomy was admitted to the emergency department with, epigastric pain and fever that had been present for 4 days, followed by jaundice of cholestatic appearance. Clinical examination in the emergency department revealed cutaneous and mucosal jaundice, fever of 38.3°C, good haemodynamic constants, and tenderness of the right hypochondrium with no defences.

A biological inflammatory syndrome CRP 80 mg/L, leucocyte 15000 /mm3, with a disturbed liver balance: bilirubin 55 mg/dL (normal, 0.2-1), aspartate aminotransferase 86 U/L (normal, 5-50), and serum alkaline phosphatase 530 U/L (normal, 25–125) elevated lipasemia to 1050IU/L.

An abdominal CT scan showed a mild cephalic interstitial pancreatitis (CTI SI = 2), with the main bile duct dilated to 10 mm, upstream of a duodenal diverticulum with no sign of diverticulitis, associated with an oedematous phenomenon of the duodenum by contiguity.

The patient was hospitalised in the gastroenterology department, undergoing intravenous hydration with rest of the digestive tract, as well as analgesic treatment with the need for stage three treatment with morphine. Antibiotic therapy was administered, followed by echo-endoscopy and endoscopic retrograde cholangiopancreatography, finding a duodenal diverticulum responsible for distal compression of the VBP with the presence of pus inside, a sphincterotomy, transpapillary dilatation and multiple scans were performed with significant pus discharge. The evolution was marked by the regression of the icterus, an improvement in his symptoms and his hepatic biological parameters, and the patient was discharged without complications.

Fig 1: a) CT scan of mild cephalic interstitial pancreatitis (CTI SI = 2). The duodenum is oedematous due to contiguity with a duodenal diverticulum; b) dilated main bile duct upstream of a duodenal diverticulum with no sign of diverticulitis

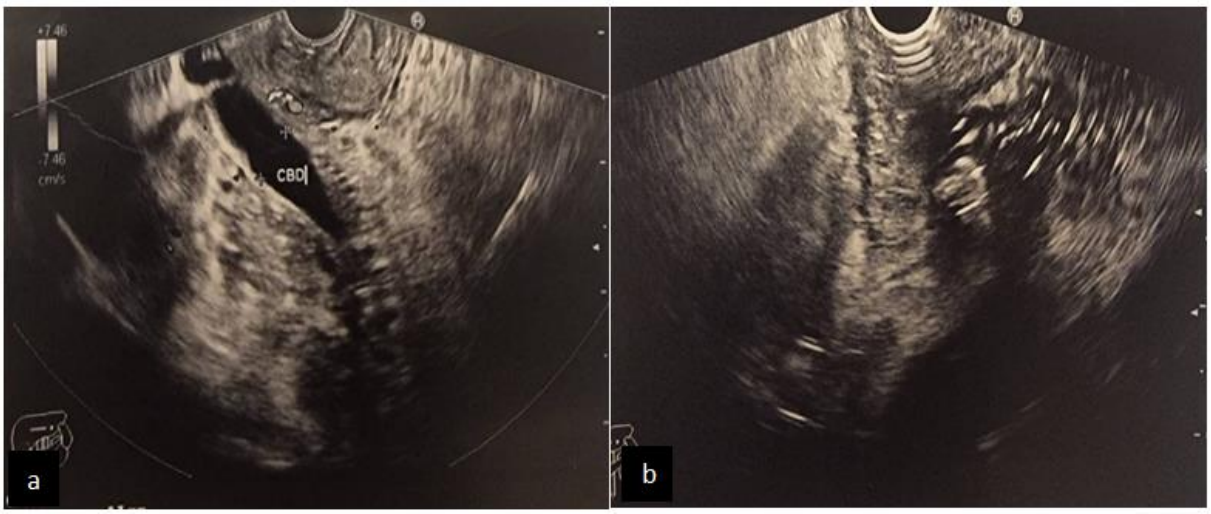

Fig 2: Endoscopic ultrasonography showing: a) Dilated common bile duct due to compression by diverticulum; b) Water along with air bubbles (echogenic foci) moving from the duodenal lumen into the diverticulum

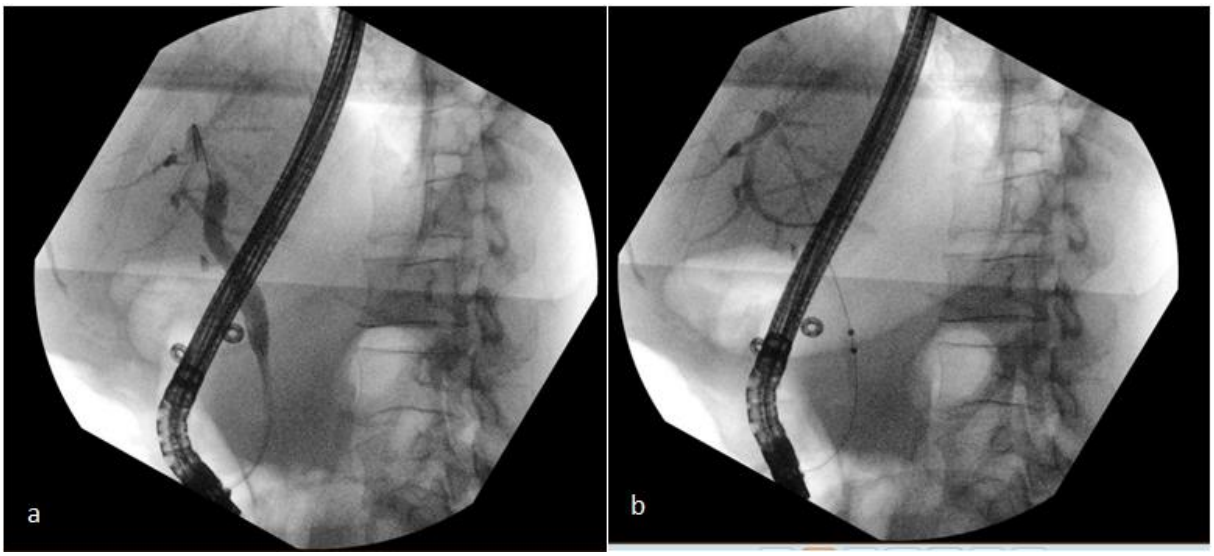

Fig 3: a et b) ERCP showing distal compression of the CBD, multiple scans are performed with significant pus oozing; c) Periampullary diverticulum oozing pus observed during ERCP

DISCUSSION

The incidence of duodenal diverticula is approximately 17%, and increases with age [2] Most are extraluminal and acquired. They are more frequently located in the second part of the duodenum, near the ampulla of Vater (juxtapapillary) because of the weakness of the wall in this area. Only 5% of diverticula are symptomatic [3] and they are usually discovered incidentally during the investigation of another disease or during endoscopy of the upper gastrointestinal tract. The existence of a juxtapapillary diverticulum is associated with a high incidence of biliopancreatic disease due, on the one hand, to extrinsic compression of the diverticulum itself on the biliary tract (Lemmel's syndrome).

Lemmel's syndrome is defined as extrinsic compression of the distal main bile duct by a periampullary diverticulum in the absence of biliary lithiasis or pancreaticobiliary tumour. There are few case reports of this condition, first named in 1934 by Dr Gerhard Lemmel [1].

Various pathogeneses of Lemmel's syndrome have been proposed, including the following. One possible pathogenesis involves chronic fibrosis of the papilla (chronic fibrous papillitis) caused by direct mechanical irritation due to chronic inflammation of the papilla or chronic periampullary diverticulitis. Secondly, a periampullary diverticulum can cause dysfunction of the sphincter of Oddi [2]. Thirdly, the CBD or ampulla may be mechanically compressed by the periampullary diverticulum, resulting in obstructive jaundice such as that seen in our patient.

The clinical symptoms of Lemmel syndrome are non-specific, manifesting mainly as right hypochondrial pain, recurrent attacks of acute abdominal pain and jaundice [1, 3].

The diagnosis is confirmed by imaging modalities, in particular CT scan, which is the reference imaging modality for rapid, non-invasive and specific assessment of periampullary duodenal diverticula, which appear as thin-walled cavitary lesions located in the 2nd part of the duodenum, compressing the intrapancreatic part of the common bile duct [4].

More recently, the diagnosis has been confirmed by EUS and ERCP, which show the distal common bile duct compressed laterally by the diverticulum, generally containing a bezoar, located on the wall of the second part of the duodenum [4, 5].

These examinations help to confirm the diagnosis, exclude other possible causes such as choledocolithiasis and tumours, and allow endoscopic sphincterotomy to be performed to free the obstruction of the common channel with placement of a biliary endoprosthesis [6].

The second therapeutic option is surgery by diverticulectomy [7]. Further research may be required to assess recurrence rates with sphincterotomy versus diverticulectomy.

Although Lemmel syndrome does not necessarily increase the risk of pancreaticobiliary malignancy, a missed diagnosis of Lemmel's syndrome may lead to recurrent pancreatitis and cholangitis, representing a risk of malignancy and increased morbidity and mortality [7].

Given the risk of complications and recurrence, it is important that clinicians diagnose and manage these patients promptly.

CONCLUSION

Lemmel syndrome is an unrecognised and rare cause of biliary tract obstruction. Because of the non-specificity of the clinical symptoms, and the risks of complications, careful analysis of cross-sectional imaging data is essential for rapid and specific diagnosis aided by EUS, and ERCP, in order to recognise and treat Lemmel syndrome rapidly.

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