

Lemmel Syndrome as a Cause of Obstructive Jaundice: Case Report

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ABSTRACT

Lemmel syndrome is a rare cause of obstructive jaundice, it was first described in 1934 by Dr. Gerhard Lemmel. It is caused by a periampullary duodenal diverticulum and only 5% are symptomatic. The diagnosis of this entity should be considered in a patient with obstructive jaundice and in the absence of choledocholithiasis or neoplasia. Here we present an 83-year-old male case successfully treated with ERCP.

KEYWORDS: Lemmel syndrome, jaundice, diverticula.

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INTRODUCTION

First described in 1934 by Dr. Gerhard Lemmel, Lemmel syndrome is a rare cause of obstructive jaundice secondary to periampullary duodenal diverticulum in the absence of choledocholithiasis or pancreatobiliary neoplasms.^{1,4}

CASE PRESENTATION

This is an 83-year-old male patient, with history of systemic arterial hypertension of 10 years of evolution under management with Losartan 50 mg every 24 hours with good control, as well as laparoscopic cholecystectomy 10 years prior without complications.

His condition began 8 days prior to his evaluation with the presence of jaundice that initially limited to the sclera and subsequently generalized, associated with colic pain in the right hypochondrium after eating cholecystokinetics, adding generalized pruritus, which is why he came for evaluation at our medical unit.

Upon arrival, laboratories were requested, which reported glucose 90 mg/dl, BUN 22 mg/dl, urea 63 mg/dl, creatinine 0.7 mg/dl, total bilirubin 9.2 mg/dl, direct bilirubin 7.4 mg/dl, bilirubin indirect 1.8 mg/dl, alkaline phosphatase 297 U/L, TGO 20 U/L, TGP 30 U/L, sodium 136 mg/dl, potassium 3.5 mg/dl, magnesium 1.8 mg/dl, phosphorus 1.5 mg/dl, calcium 7.7 mg/dl, cholesterol 127 mg/dl mg/dl, triglycerides 113 mg/dl, leukocytes 9.3 x10³, neutrophils 10.9 x10³, hemoglobin 16.2 g/dl, hematocrit 48.6%, mean corpuscular volume 95.1 fl, mean corpuscular hemoglobin 31.9 pg, platelets 192,000 x10³.

An ultrasound of the liver and bile ducts was performed and reported the surgical absence of the gallbladder and common bile duct measuring 9 mm, with no evidence of stone inside. An R factor of 0.41 is calculated, corroborating a cholestatic pattern. Patient who is staged as high risk according to the 2019 guidelines of the American Society of Gastrointestinal Endoscopy for choledocholithiasis, which is why it was decided to send him to ERCP.

In the ERCP report they indicate the presence of duodenal diverticula with intradiverticular ampulla of Vater, dilation of the bile duct of 15 mm and filling defects in the distal common bile duct, performing sphincterotomy and balloon sweeps witnessing the release of thick bile without stones, verifying adequate cleaning of biliary material at the end of the study (figures 1, 2 and 3).

48 hours after the study, a laboratory control was performed showing a decrease in total bilirubin to 6.5 mg/dl, direct bilirubin 3.7 mg/dl, and alkaline phosphatase 248 U/L. Patient whose course was asymptomatic, without developing any complications, for which reason his discharge was decided.



Figure 1. Cannulation.

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Figure 2. Sphincterotomy.



Figure 3. Balloon probe.

DISCUSSION

The majority of diverticula are located in the second portion of the duodenum, close to the ampulla of Vater (juxtapapillary), due to the weakness of the wall in this area, as well as the one presented by our patient.¹ Only 5% are symptomatic.^{1,2}

Complications include jaundice, cholangitis, or acute pancreatitis, and in rarer cases include diverticulitis, hemorrhage, perforation, or fistula.^{3,4}

The diagnosis of this entity should be considered in a patient with obstructive jaundice and in the absence of choledocholithiasis or neoplasia; We must also remember that the treatment of choice is endoscopic sphincterotomy, and it is only offered to symptomatic patients; Surgery is only reserved for those patients with severe symptoms or repeated symptoms, and there are currently various techniques that include simple diverticulectomy (associated or not with a choledochojejunostomy), gastroenteric diversion, diverticular inversion, transduodenal sphincteroplasty or pancreaticoduodenectomy.³⁻⁵

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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INFORMED CONSENT STATEMENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A written copy is available upon request.

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