

Introduction

Lemmel syndrome (LS) is a rare cause of obstructive jaundice occurring in the absence of pancreaticobiliary tumors or choledocolithiasis and commonly caused by periampullary diverticulum (PAD) with an incidence of 1-27%. They are typically asymptomatic and underreported leading to complications in ~5% of cases. Herein, we report a case of septic shock due to acute cholangitis secondary to Lemmel Syndrome.

Case Presentation

A 58-year-old male with a medical history significant for hypertension who presented to the ER with right upper quadrant abdominal pain, jaundice, and multiple episodes of nonbloody-nonbilious emesis. On admission, the patient was febrile, tachycardic and hypotensive. Initial laboratory tests are shown in Table 1 below. CT of the abdomen showed cholelithiasis and diffuse gallbladder thickening with no intra or extrahepatic biliary duct dilation. He developed septic shock requiring vasopressors. Blood cultures were positive for *E. coli*, and antibiotics were administered. Emergent ERCP was performed due to the presence of acute cholangitis. He was found to have a large diverticulum located around the major ampulla. Double wire technique was performed to access the ventral pancreatic duct (VPD) and common bile duct (CBD). Fluoroscopic images revealed biliary tapering in the distal third but no visualized stones. Plastic stents were placed into the VPD and CBD and biliary sphincterotomy was performed. He also underwent laparoscopic cholecystectomy. The patient was discharged with resolution of symptoms.

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An Unusual Cause of Septic Shock Secondary to Lemmel Syndrome Mohammad Nabil Rayad, MD¹; Noreen Mirza, MD¹; Dema Shamoon, MD¹; Fatima Kamal, MD²; Raed Atiyat, MD¹; Yatinder Bains, MD¹

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Blood Chemistry		Reference Range
Sodium	137 mmol/L	136 - 145 mmo
Potassium	3.8 mmol/L	3.5 - 5.3 mmol
Blood urea nitrogen (BUN)	10.0 mg/dL	6 - 24 mg/dL
Creatinine	1.0 mg/dL	0.6 - 1.2 mg/dl
Lactic acid	5.3 mmol/L	0 – 2.0 mmol/l
C-reactive protein	21.8 mg/dL	0.0-0.8 mg/dL
тѕн	1.1180 µIU/mL	0.400 - 4.500 µ
Total bilirubin	3.3 mg/dL	0.2–1.2 mg/dL
Direct bilirubin	1.94 mg/dL	0.0-0.3 mg/dL
AST	487 U/L	10-36 U/L
ΔΙΤ		0.4611/1
Alkaling phosphataso		9-40 U/L
Alkaline phosphatase	155 U/L	40-115 U/L
Complete blood count		
White blood cell (WBC)	4.3x10 3/ uL	4.4 - 11 × 103/
Hemoglobin	12.2 g/dL	13.5 - 17.5 g/d
Platelet	107 x103 /uL	150 - 450 × 103

Table 1. Initial Laboratory Values.





Discussion

The pathophysiology of LS is due to direct mechanical irritation of the diverticulum causing papillary fibrosis. Secondly, the diverticulum may cause sphincter of Oddi dysfunction. Thirdly, the distal CBD may be directly compressed by the periampullary diverticulum. The imaging modalities of choice to confirm the diagnosis include enhanced contrast CT and MRCP. ERCP allows for direct visualization of the duodenal diverticulum. Treatment of these entities is required if the patient becomes symptomatic. The mainstay of treatment is endoscopic sphincterotomy with stent placement or papillary balloon dilation. Due to the high recurrence rate, if endoscopic treatment fails then surgical approach via diverticulectomy is performed. It is important physicians remain vigilant regarding this condition since misdiagnosis leads to delay in therapy. The incidence of septic shock in LS is unknown and to our knowledge, there has only been one other reported case in literature.



A: Cholangiogram of biliary tree with no stones B: Cholangiogram with diverticulum, stent in place and no obvious stones C: Periampullary diverticulum after placement of CBD and pancreatic stent D: CT abdomen showing CBD stent in place with gallbladder sludge.