

Surgical resection of duodenal diverticulum due to Lemmel's syndrome: a case report

Abstract

Periampullary diverticulum is an extra-luminal structure found within 2-3cm from the ampulla of Vater. Those formations are mostly asymptomatic and might be found incidentally. However, PAD is the most common cause of Lemmel's syndrome, which is defined as obstructive jaundice caused in absence of choledocholithiasis or tumor. The present study describes a case of surgical resection of periampullary diverticulum a 56-year-old male with a past medical history significant for video laparoscopic cholecystectomy (performed 7years ago), presented to the emergency room with 8hours of sharp epigastric pain and laboratory results altered. MRCP showed mild dilatation of the intrahepatic biliary tract.

For symptomatic patients, endoscopic intervention and surgical resection are the most common techniques for treatment but the options vary based on symptomology and the pathophysiology of the subtype of Lemmel's syndrome. In this case, diverticulostomy and internal choledocho-duodenostomy with PDS separated suture was performed to the treatment. The follow-up laboratory values revealed normalization and the postoperative period had no complications.

Keywords: periampullary diverticulum, Lemmel's syndrome, cholangitis, surgical resection

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João Alfredo Diedrich Neto, Marcos Orestes Gonçalves, Vanessa Santos Andrade Cruz Vento, Bruno Alves Vento, Carolina Bittencourt Letzov, Vinicius Beck da Silveira, Eduarda Paitl Augustine, Arthur Freccia, Luize Cristine Dias, Gabriella Laís Braatz

Department of Surgery, Hospital Arquidiocesano Consul Carlos Renaux in Brusque- SC, Brazil

Correspondence: João Alfredo Diedrich Neto, Department of Surgery, Hospital Arquidiocesano Consul Carlos Renaux in Brusque- SC, Brazil, Email joaodiedrich@gmail.com

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Introduction

A duodenal diverticulum consists of outpouching of the duodenal mucosa.¹ These are classified in intramural and extramural diverticula, the majority of which are extra-luminal.²

Furthermore, there is an extra-luminal diverticulum, specifically found within a radius of 2-3cm from the ampulla of Vater, known as periampullary diverticulum (PAD).³ The incidence of periampullary diverticulum is 1%-27%³ and the prevalence has been described as high as 65% in elderly population.² PAD is the most common cause of Lemmel's syndrome, which is defined as obstructive jaundice caused in absence of choledocholithiasis or tumor. Patients with Lemmel's syndrome due to periampullary duodenal diverticulum usually present with jaundice and right upper quadrant discomfort.³ Along these lines, laboratory workup usually reveals leukocytosis, elevated erythrocyte sedimentation rate, C-reactive protein, direct and total bilirubin, liver enzymes, alkaline phosphatase, and elevated gamma-glutamyl transferase. Besides that, compression of the ampulla of Vater can increase pancreatic enzyme levels.³ Treatment options for Lemmel's syndrome include surgical resection, endoscopic intervention and conservative management.⁴

Case presentation

A 56-year-old male with a past medical history significant for video laparoscopic cholecystectomy (performed 7years ago) presented to the emergency department due to 8 hours of sharp epigastric pain. On physical examination, he had a heart rate of 114, normal blood pressure, and jaundice, negative Murphy's sign, positive Blumberg's sign.

Laboratory results demonstrated elevated lactate dehydrogenase (LDH) of 1160 UI/L, amylase of 663 UI/L, total bilirubin of 9,2mg/dL, direct bilirubin of 5,8mg/dL and indirect bilirubin of 3,4mg/dL, aspartate aminotransferase of 291U/L, alanine aminotransferase of 414U/L, PT of 30s, INR of 1.5 and albumine level of 3,8g/dL. Other

parameters were also considered, as hemoglobin of 15g/dL and WBC of 11480μL.

CT scan did not demonstrate bile duct dilatation. Despite that, MRCP showed mild dilatation of the intrahepatic biliary tract, more evident in the left lobe and extrahepatic biliary tract, associated with thickening and parietal enhancement, inferring an inflammatory process. The diameter of the common bile duct was 1.2 and a duodenal diverticulum, measuring 3.2 cm, was revealed in the second portion. The imaging indicated close relationship with the distal bile duct and expansion towards the pancreatic head.

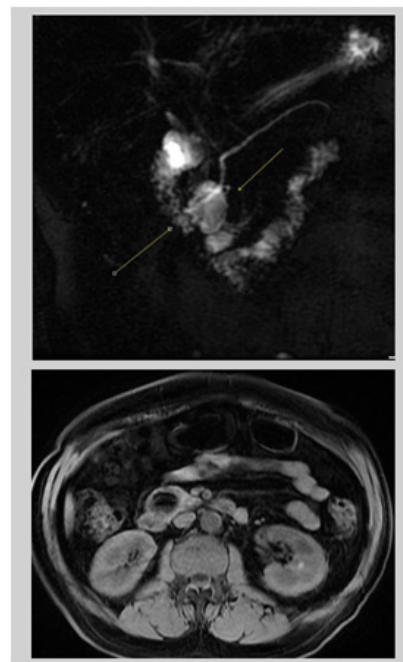


Figure 1 MRCP showed a duodenal 3.2cm. diverticulum measuring.



Figure 2 Diverticulostomy and catheterization of common bile duct.

Discussion

Lemmel's syndrome was first described in 1934, consisting of obstructive jaundice due to compression of the distal common bile duct by a PAD. Those formations are mostly asymptomatic³ and might be found incidentally in up to 22% of the population, although, less than 10% are symptomatic.² Complications occur in less than 5% of cases and might include bleeding, perforation, cholangitis, pancreatitis, bezoar or enterolith formation, and, rarely, obstructive jaundice.⁴

Specific mechanisms have been suggested as etiologic factors for Lemmel syndrome: first, diverticulitis or direct mechanical irritation of periampullary duodenal diverticulum resulting in chronic inflammation of ampulla which leads to fibrosis of papilla. Second, periampullary duodenal diverticulum may cause dysfunction of sphincter of Oddi. Third, the choledochus or the ampulla of Vater can be directly compressed by a distended PAD as a result of enterolith or bezoar.⁴

ERCP is considered the gold standard for diagnosis.³ Moreover, other modalities of imaging, such as a CT scan and magnetic resonance cholangiopancreatography (MRCP), demonstrate that PAD appear as thin-walled cavitory lesions of the second part of the duodenum. Some PAD can also be filled with fluid and that can lead to a misdiagnosis.³ The differential recognitions include pancreatic pseudocyst, infected necrotic collection, periampullary neoplasm, head of the pancreas neoplasm, and so on.³ Under diagnosis of Lemmel's syndrome, poor clinical condition due to cholangitis and evolution with worsening jaundice we performed surgical resection of PAD.

A Chevron incision was made. The duodenum was topical with few local adhesions from the previous cholecystectomy. The procedure included transverse duodenectomy. Diagnostic catheterization of the pancreatic duct was performed with a 4 French catheter. The catheterization of common bile duct, the papitomy was performed. After, an internal choledocho-duodenostomy with PDS separated suture was performed.

The postoperative period had no complications and the patient was discharged 20 days after surgery. The hospital stay was prolonged

due to sepsis of biliary origin caused by *Klebsiella pneumoniae* carbapenemase (KPC). The result of the anatomopathological exam showed simple cylindrical epithelium, without atypia or neoplastic alterations.

Conclusion

Lemmel's syndrome is specifically classified as obstructive jaundice in the absence of choledocholithiasis. The clinical picture can also demonstrate cholangitis due to mechanical compression of the terminal bile duct by diverticulum. When these formations are located within 2-3cm of the ampulla of Vater they are termed periampullary diverticula. PAD typically take place along the medial aspect of the second part of the duodenum and that can be more easily showed using a side viewing endoscope during ERCP. Notwithstanding, CT scan and MRI/MRCP can also characterize the biliary ductal dilation and show the diverticula. Treatment options vary based on symptomatology and the pathophysiology of the subtype of Lemmel's syndrome. For symptomatic patients, endoscopic intervention and surgical resection are the most common techniques.

In our case, surgical resection was performed and the follow-up laboratory values revealed normalization of previously elevated parameters.

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Conflicts of interest

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