

Lemmel's Syndrome as a Cause of Acute Cholangitis: A Case Report with Successful Endoscopic Management

Dra. Ethel Cassandra Ramírez Ortiz^{*1}, Dra. Alexandra Espinoza Romo², Dra. Aranza Guevara Bastida³, Dr. José Alfredo Peñúñuri Domínguez⁴, Dr. Isidoro Wiener Carrillo⁵

¹Instituto Tecnológico y de Estudios Superiores de Monterrey campus Monterrey, México.

²Instituto de Estudios Superiores de Tamaulipas red Anáhuac, México.

³Universidad La Salle Facultad Mexicana de Medicina, México.

⁴Universidad Anáhuac México, México.

⁵Cirugía General, México.

ABSTRACT

ARTICLE DETAILS

Background: Amyand's hernia is a rare condition characterized by the presence of the vermiform appendix within an inguinal hernia sac. Its incidence is low, and its association with acute appendicitis is even rarer, often leading to diagnostic difficulty. Most cases are diagnosed intraoperatively, particularly when presenting as an incarcerated or strangulated hernia.

Case Presentation: We report the case of a 66-year-old man with a 30-year history of untreated right inguinoscrotal hernia who presented with acute scrotal pain and signs of intestinal obstruction. Physical examination revealed a painful, non-reducible right inguinoscrotal mass. Laboratory studies showed elevated inflammatory markers. Computed tomography demonstrated an incarcerated right inguinal hernia containing bowel loops. Emergency surgical exploration was performed. Intraoperatively, an inflamed appendix with a periappendiceal abscess was identified within the hernia sac. Appendectomy, abscess drainage, and herniorrhaphy were successfully performed. The postoperative course was uneventful, and the patient was discharged in good clinical condition. Histopathological examination confirmed acute nonspecific appendicitis without perforation.

Conclusion: Amyand's hernia complicated by acute appendicitis is an uncommon surgical finding that may mimic an incarcerated inguinal hernia. Early surgical intervention and individualized management based on intraoperative findings are essential to achieve favorable outcomes.

KEYWORDS: Amyand's hernia; Acute appendicitis; Inguinoscrotal hernia; Case report; Emergency surgery

Published On:
27 March 2026

Available on:
<https://ijmscrs.com>

I. INTRODUCTION

Lemmel's syndrome is a rare clinical entity, first described in 1934. It is defined as obstructive jaundice caused by a periampullary duodenal diverticulum in the absence of choledocholithiasis or neoplasia. Currently, only a limited number of cases have been published, and its etiopathogenesis has not been fully established (1).

The pathophysiology of this syndrome is based on three main mechanisms:

- Direct mechanical irritation by the periampullary diverticulum, which leads to chronic inflammation of the ampulla and subsequent fibrosis.
- Dysfunction of the sphincter of Oddi induced by periampullary diverticula, promoting biliary stasis and reflux from the duodenum into the common bile duct.
- Mechanical compression of the distal portion of the common bile duct or the ampulla by the periampullary diverticulum.

Lemmel's Syndrome as a Cause of Acute Cholangitis: A Case Report with Successful Endoscopic Management

These three processes facilitate the reflux of duodenal contents, bacterial overgrowth, biliary stasis, and the formation of gallstones (2).

The incidence of duodenal diverticula ranges from 5% to 10% and increases with age, reaching up to 20% in individuals aged 80 years (2). Most are extraluminal and acquired. They are most commonly located in the second portion of the duodenum, within a 3 cm radius of the ampulla of Vater (juxtapapillary), due to anatomical weakness in this area caused by the entry of biliary and pancreatic structures (1, 4). Approximately 6% of duodenal diverticula occur in the first portion, 53–75% in the second portion, 12% in the third portion, and 24% in the fourth portion (2). These diverticula may be pseudodiverticula, consisting of mucosal pouches lacking a muscular layer, or true (congenital) diverticula, with the former being far more common (2).

Congenital diverticula are typically located on the medial wall of the second or third portion of the duodenum (5).

Only about 5% are symptomatic, often representing an incidental finding during evaluation for another condition or during upper gastrointestinal endoscopy (1). Choledocholithiasis occurs in 20–40% of patients with periampullary duodenal diverticula (2).

The presence of a juxtapapillary diverticulum is associated with an increased incidence of biliopancreatic disease (1), due to extrinsic compression of the biliary tract by the diverticulum itself (Lemmel's syndrome), which compresses the common bile duct and causes retrograde dilatation of the intrahepatic and extrahepatic biliary tree (2). It may also promote bacterial overgrowth, leading to gallstone formation through beta-glucuronidase activity, as well as inducing sphincter of Oddi dysfunction that results in stasis and biliary reflux from the duodenum into the common bile duct.

The prevalence of bacteriobilia increases with age and is higher in the presence of a juxtapapillary diverticulum. The organisms involved in bacterial overgrowth are those commonly found in bile, including *Escherichia coli*, *Streptococcus faecalis* (now *Enterococcus faecalis*), *Proteus* spp., *Klebsiella* spp., and anaerobes (8).

Among the clinical manifestations, cholestatic jaundice (due to elevated direct bilirubin), abdominal pain, cholangitis, and presentations that mimic periampullary tumors stand out; cholangitis represents the most severe form (2).

Only 1% of duodenal diverticula develop complications, with cholangitis being one of them. This condition must be diagnosed and treated promptly due to its high morbidity and mortality rates. Lemmel's syndrome is considered an uncommon cause of acute cholangitis (3). Other associated complications include intestinal obstruction, perforation, and gastrointestinal bleeding. Malignant transformation is extremely rare and has only been described in association with the inclusion of heterotopic/ectopic pancreatic or gastric tissue (2).

There is insufficient data to firmly associate acute or chronic pancreatitis with the presence of a duodenal diverticulum.

Evidence remains limited regarding the diverticulum's contribution to the pathogenesis of pancreatitis. However, cases have been reported in elderly patients presenting with both pancreatitis and duodenal diverticulum, suggesting a possible relationship between these two conditions (8).

Laboratory studies may reveal leukocytosis, elevated bilirubin levels (direct and total), increased liver enzymes, gamma-glutamyl transferase (GGT), alkaline phosphatase, and inflammatory markers. Elevated pancreatic enzymes may be observed if the diverticulum compresses the ampulla of Vater (7).

Diagnostic imaging plays an essential role in the diagnosis of Lemmel's syndrome, helping to avoid therapeutic errors. On computed tomography (CT) and magnetic resonance imaging (MRI), the periampullary diverticulum appears as a thin-walled saccular structure on the medial wall of the second portion of the duodenum. In some cases, it may fill with fluid content and mimic a pancreatic abscess, a cystic-neoplastic lesion in the pancreatic head (5), or lymph node metastases; oral contrast administration optimizes its visualization.

Currently, endoscopic retrograde cholangiopancreatography (ERCP) is the gold standard for both diagnosis and treatment, as it allows exclusion of other possible causes such as choledocholithiasis and tumors, and enables performance of endoscopic sphincterotomy as definitive therapy (2, 9). A retrospective study demonstrated that ERCP achieves a high success rate with a low complication rate (10). It has also been shown that there is no increased risk of complications related to diverticulum size when performing endoscopic sphincterotomy (6).

Surgical treatment, whether by diverticulectomy or biliodigestive bypass, is reserved exclusively for complicated cases such as perforation or post-ERCP hemorrhage, or when endoscopic cannulation is impossible due to factors including stones larger than 20 mm, intradiverticular ampulla, technical inability to cannulate, lack of appropriate equipment, operator inexperience, impacted stone in the distal common bile duct, or panlithiasis.

II. CASE REPORT

A 74-year-old male patient with no relevant past medical history presented to the emergency department. The symptoms began 3 days prior to admission, consisting of generalized colicky abdominal pain with an intensity of 7/10 on the Numerical Rating Scale (NRS), without apparent aggravating or relieving factors, associated with constipation of 2 days' duration. The patient reported self-medication with antispasmodics, gastric mucosal protectants, and lactulose, without improvement.

He reported worsening of symptoms 24 hours prior to admission, with pain now localized in a "belt-like" distribution of a transfixing nature, radiating to the left scapula, with an intensity of 8/10 on the NRS. In the 7 hours prior to admission, he developed fever measured at 38.3°C (which subsided with antipyretics), holocranial headache of

Lemmel's Syndrome as a Cause of Acute Cholangitis: A Case Report with Successful Endoscopic Management

6/10 intensity, mucocutaneous jaundice, dark urine (choloria), anorexia, asthenia, and adynamia severe enough to prevent performance of daily activities.

On physical examination, the patient was neurologically intact, with no respiratory or cardiopulmonary findings. He presented with a pained facies, positive mucosal jaundice, and moderate mucocutaneous dehydration. The abdomen was flat, soft, and depressible, with increased peristalsis in both frequency and tone. It was tender to moderate and deep palpation throughout, with predominance in the epigastrium and both hypochondria. Generalized tympany was noted on percussion. Murphy's sign was positive, as was the rebound sign. Extremities showed adequate capillary refill. Weight was 60 kg, height 182 cm, and body mass index (BMI) 18.1 kg/m².

Laboratory studies were performed, including a complete blood count and blood chemistry panel, which revealed altered segmented neutrophils at 87%, and abnormal liver enzymes with the following values: aspartate aminotransferase (AST) 97 U/L, alanine aminotransferase (ALT) 301 U/L, alkaline phosphatase 371 U/L, lactate dehydrogenase (LDH) 158 U/L, gamma-glutamyl transferase

Endoscopic sphincterotomy was performed, followed by adequate outflow of the contrast medium.

The diagnosis was delayed contrast emptying secondary to edema and the presence of a diverticulum.

III. RESULTS

After performing endoscopic retrograde cholangiopancreatography (ERCP) for both diagnostic and therapeutic purposes, the presence of a periampullary duodenal diverticulum causing obstruction of biliary outflow was confirmed, resulting in cholestasis and abnormalities in the patient's liver function tests and bilirubin levels.

On the day following the endoscopic intervention, complete blood count and serum chemistry tests were repeated, demonstrating a notable improvement in parameters: segmented neutrophils decreased to 71%, aspartate aminotransferase (AST) to 45 U/L, alanine aminotransferase (ALT) to 120 U/L, alkaline phosphatase to 296 U/L, lactate dehydrogenase (LDH) to 105 U/L, gamma-glutamyl transpeptidase (GGT) to 292 U/L, direct bilirubin to 1.1 mg/dL, indirect bilirubin to 0.4 mg/dL, and total bilirubin to 1.5 mg/dL.

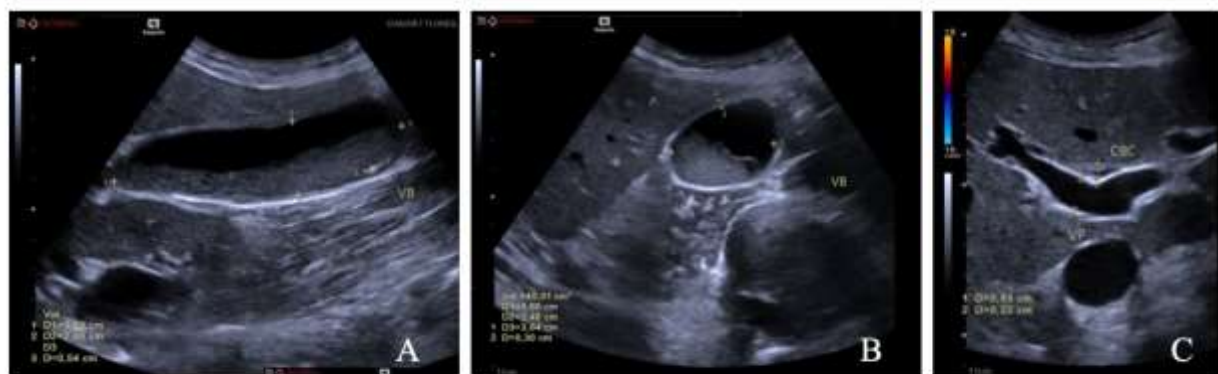


Figure 1. A) Gallbladder showing abundant biliary sediment within its lumen and a gallstone measuring 5.4 mm. B) Gallbladder with a wall thickness of 3 mm. C) Common bile duct with a diameter of 2.3 mm.

(GGT) 429 U/L, direct bilirubin 4.6 mg/dL, indirect bilirubin 1.6 mg/dL, and total bilirubin 6.2 mg/dL.

Abdominal computed tomography (CT) scan without and with intravenous contrast was performed, revealing an irregular image in the pancreatic head with partially defined borders measuring 10 × 13 mm. Additionally, an upper abdominal ultrasound showed intrahepatic biliary ducts within normal limits, a common bile duct diameter of 2.3 mm, a gallbladder with a wall thickness of 3 mm containing abundant biliary sludge and a single gallstone measuring 5.4 mm (Fig. 1).

The patient underwent endoscopic retrograde cholangiopancreatography (ERCP). The duodenoscopy was advanced to the second portion of the duodenum, where the major papilla was identified beneath an edematous duodenal fold and within a diverticulum. Biliary cannulation was achieved on the first attempt, and contrast medium was injected. The biliary tree appeared intact with a diameter of 6 mm but showed delayed emptying of the contrast.

The patient was discharged on postoperative day three due to favorable clinical progression and the gradual resolution of laboratory abnormalities. The endoscopic sphincterotomy was considered successful and effective in controlling the biliopancreatic obstruction secondary to Lemmel syndrome.

IV. DISCUSSION

Lemmel syndrome represents an uncommon but recognized cause of obstructive jaundice and acute cholangitis secondary to extrinsic compression of the distal common bile duct by a periampullary (juxtapapillary) duodenal diverticulum, in the absence of choledocholithiasis or neoplasms. As described in the literature, this entity predominantly affects elderly patients, with the incidence of duodenal diverticula increasing with age; however, only about 5% of these diverticula become symptomatic, and fewer than 1–5% develop severe complications such as cholangitis or persistent obstructive jaundice.

In the present case, the 74-year-old patient presented with a clinical picture typical of acute cholangitis (fever, abdominal

pain, jaundice, choloria, and cholestatic abnormalities with marked neutrophilic leukocytosis), accompanied by signs of systemic inflammation and physical findings suggestive of peritoneal irritation (positive Murphy's sign and rebound tenderness). Initial imaging findings (CT scan showing an irregular lesion in the pancreatic head and ultrasound revealing biliary sludge and small gallstones) initially raised suspicion of pancreatic pathology or choledocholithiasis, highlighting the importance of considering differential diagnoses in elderly patients with atypical presentations. ERCP confirmed the diagnosis by visualizing an intradiverticular papilla with edema and delayed biliary drainage, allowing for endoscopic sphincterotomy as the main therapeutic intervention.

Post-procedural evolution was favorable, with rapid biochemical improvement (significant decrease in bilirubin levels, transaminases, GGT, alkaline phosphatase, and neutrophils) and discharge on postoperative day three. This is consistent with current evidence supporting endoscopic sphincterotomy as the first-line treatment in most symptomatic cases of Lemmel syndrome.

Systematic reviews and case series report endoscopic success rates exceeding 90–95% in procedures with successful cannulation, with complete clinical and biochemical resolution in the vast majority of patients, low rates of early recurrence, and minimal complications when performed by experienced operators. In our patient, cannulation was achieved on the first attempt despite the intradiverticular location of the papilla, which is not always the case and may require a side-viewing duodenoscope or alternative techniques in more complex cases.

Although surgery (diverticulectomy or biliodigestive bypass) is reserved for severe complications (perforation, post-ERCP hemorrhage) or endoscopic failure (stones >20 mm, distal impaction, panlithiasis, or inaccessible ampulla), in current practice it represents a secondary option given the high efficacy and lower morbidity of the endoscopic approach.

V. CONCLUSION

This case reinforces that Lemmel syndrome should be included in the differential diagnosis of obstructive jaundice and cholangitis in elderly patients with juxtapapillary diverticula, especially when ERCP reveals delayed biliary emptying without evident lithiasis. Timely diagnosis through imaging (CT/MRI) and ERCP helps avoid unnecessary interventions and enables effective minimally invasive management. Although its association with pancreatitis remains controversial and supported by limited evidence, ampullary compression and duodenobiliary reflux justify close monitoring in recurrent cases.

Further evidence is needed to standardize management and to better define the role of predisposing factors in this condition.

REFERENCES

- I. María Carmona Agúndez, Diego López Guerra, Juana Fernández Pérez, Gerardo Blanco Fernández, Síndrome de Lemmel: ictericia obstructiva secundaria a divertículo duodenal, *Cirugía Española*, Volume 95, Issue 9, 2017, Pages 550–551, ISSN 0009-739X, <https://doi.org/10.1016/j.ciresp.2017.02.003>. (<https://www.sciencedirect.com/science/article/pii/S0009739X17300349>)
- II. Dávila-Ruiz EO, García-Manzano RA, Barker-Antonio A, Martínez-Santiago NY, Isidoro-Hernández D, Sánchez-Guerrero RI. Ictericia obstructiva intermitente o síndrome de Lemmel: reporte de caso y revisión de la literatura. *Cir Cir*. 2020;88(Suppl 2):60-65. English. doi: 10.24875/CIRU.20000332. PMID: 33284277.
- III. León Moreno JF, Wong Nano CD, Dominguez Vega SJF. Síndrome de Lemmel: una causa infrecuente de colangitis aguda [Lemmel's Syndrome: A Rare Cause of Acute Cholangitis]. *Acta Gastroenterol Latinoam*. 2022 Mar 30;52(1):101-105. Spanish. doi: 10.52787/agl.v52i1.170. PMID: 41341773; PMCID: PMC12668449.
- IV. Cabrerizo García JL, Zalba Etayo B. Ictericia obstructiva intermitente secundaria a síndrome de Lemmel [Intermittent obstructive jaundice by Lemmel's syndrome]. *Gastroenterol Hepatol*. 2010 Feb;33(2):152-3. Spanish. doi: 10.1016/j.gastrohep.2009.10.007. Epub 2009 Dec 10. PMID: 20005013.
- V. Bittle MM, Gunn ML, Gross JA, Rohrmann CA. Imaging of duodenal diverticula and their complications. *Curr Probl Diagn Radiol*. 2012 Jan-Feb;41(1):20-9. doi:10.1067/j.cpradiol.2011.07.001.PMID: 22085659.
- VI. Chiang TH, Lee YC, Chiu HM, Huang SP, Lin JT, Wang HP. Endoscopic therapeutics for patients with cholangitis caused by the juxtapapillary duodenal diverticulum. *Hepatogastroenterology*. 2006 Jul-Aug;53(70):501-5. PMID: 16995449.
- VII. Volpe A, Risi C, Erra M, Cioffi A, Casella V, Fenza G. Lemmel's syndrome due to giant periampullary diverticulum: report of a case. *Radiol Case Rep*. 2021 Oct 4;16(12):3783-3786. doi:10.1016/j.radcr.2021.08.068. PMID: 34659600; PMCID: PMC8502835.
- VIII. Lobo DN, Balfour TW, Iftikhar SY, Rowlands BJ. Periampullary diverticula and pancreaticobiliary disease. *Br J Surg*. 1999 May;86(5):588-97. doi: 10.1046/j.1365-2168.1999.01121.x. PMID: 10361174.

Lemmel's Syndrome as a Cause of Acute Cholangitis: A Case Report with Successful Endoscopic Management

- IX. Grigoriu M, Palade R, Lutic C. Diverticulul duodenal periampular--implicații în patologia bilio-pancreatică [Periampullar diverticulum--related to bilio-pancreatic disorders]. Chirurgia (Bucur). 2010 Jan-Feb;105(1):37-43. Romanian. PMID: 20405678.
- X. Zippi M, Traversa G, Pica R, De Felici I, Cassieri C, Marzano C, Occhigrossi G, Paoluzi P. Efficacy and safety of endoscopic retrograde cholangiopancreatography (ERCP) performed in patients with Periampullary duodenal diverticula (PAD). Clin Ter. 2014;165(4):e291-4. doi: 10.7417/CT.2014.1745. PMID: 25203345.