Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Radiology

Complications of Periampullary Duodenal Diverticula: A Report of Two Cases

Zakaria Zouaki[1]*, Khalid Gharbi[2]

[1]Department of Radiology, Hassan II Hospital, Laayoune, Morocco
[2]Department of Gastroenterology, Hassan II Hospital, Laayoune, Morocco

DOI: https://doi.org/10.36347/sjmcr.2026.v14i07.033 | **Received:** 10.05.2026 | **Accepted:** 17.06.2026 | **Published:** 31.07.2026

***Corresponding author:** Zakaria Zouaki
Department of Radiology, Hassan II Hospital, Laayoune, Morocco

Abstract **Case Report**

Periampullary duodenal diverticula (PADD) are acquired mucosal outpouchings arising from weak points in the duodenal wall in close proximity to the ampulla of Vater. Although the vast majority remain asymptomatic, their anatomical relationship with the distal common bile duct and the main pancreatic duct may give rise to potentially life-threatening complications, including obstructive jaundice, acute cholangitis, and recurrent acute pancreatitis. We report two cases illustrating the clinico-radiological spectrum of PADD-related complications. The first is a 68-year-old male with acute cholangitis in the context of Lemmel syndrome: MRI revealed a 4 cm periampullary diverticulum causing extrinsic compression of the distal common bile duct. The second is a 55-year-old female with two episodes of recurrent acute pancreatitis of initially unexplained origin; MRI performed during remission showed a 17 mm air-containing diverticulum on the medial wall of D2, with no ductal dilation in the intercritical period. Both cases highlight the pivotal role of MRI cholangiopancreatography (MRCP) in the non-invasive diagnosis and characterization of PADD, and the importance of tailored multidisciplinary management.

Keywords: periampullary duodenal diverticulum; Lemmel syndrome; acute pancreatitis; cholangitis; MRCP.

1. INTRODUCTION

Duodenal diverticula are the second most common gastrointestinal diverticula after colonic diverticula, with a reported prevalence ranging from 5% to 22% depending on diagnostic methodology [1,2]. They develop predominantly along the medial wall of the second and third duodenal portions (D2–D3), in close proximity to the ampulla of Vater — hence the term "periampullary" or "juxtapapillary" diverticula [3]. The vast majority are asymptomatic; however, their anatomical relationship with the distal common bile duct (CBD) and the main pancreatic duct (MPD) confers a significant pathogenic potential in a minority of cases.

Recognized complications include extrinsic biliary obstruction (Lemmel syndrome), recurrent acute pancreatitis, ascending cholangitis, diverticular perforation, and hemorrhage [4,5]. Proposed mechanisms encompass direct mechanical compression of the biliopancreatic ducts, sphincteric dysfunction from ampullary distortion, intraluminal bacterial overgrowth with enterobiliary reflux, and impaired periampullary motility [6]. MRI cholangiopancreatography (MRCP) has emerged as the gold-standard imaging modality for non-invasive morphological assessment of the biliopancreatic system and adjacent diverticula [7].

We report two cases illustrating distinct clinical presentations of PADD-related complications, with emphasis on the role of MRI and the principles of management.

2. CASE REPORTS

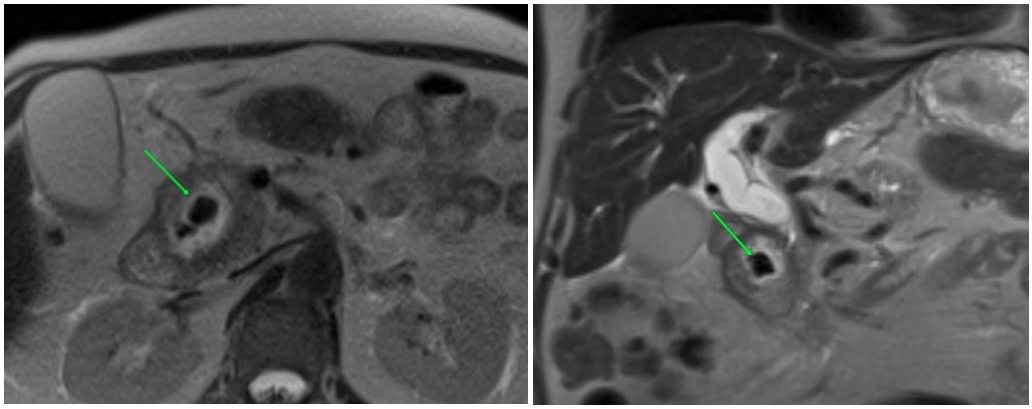
2.1. Case 1 — Lemmel Syndrome Complicated by Acute Cholangitis

A 68-year-old male with no prior digestive history was admitted as an emergency with Charcot's triad: fever (39.5°C), jaundice, and right upper quadrant pain. Laboratory investigations revealed a systemic inflammatory response (CRP 142 mg/L, leukocytosis 14,000/mm³) and mixed cholestatic pattern (total bilirubin 85 µmol/L, alkaline phosphatase 3× ULN, GGT 5× ULN, AST and ALT 2× ULN). Emergency ultrasound demonstrated intra- and extrahepatic biliary dilation without identifiable choledocholithiasis.

Complementary bili-pancreatic MRI identified, on the medial wall of D2, a large periampullary

diverticulum measuring 4 cm in diameter with mixed fluid-air content. It exerted direct extrinsic compression of the distal CBD, resulting in upstream dilation to 17 mm with intrahepatic biliary involvement. The main pancreatic duct was simultaneously enlarged to 6 mm, indicating a compressive effect on the pancreatic ductal system. The overall picture was consistent with Lemmel syndrome complicated by acute ascending cholangitis.

Management included emergency broad-spectrum intravenous antibiotics. ERCP was attempted but proved technically challenging given the juxtapapillary diverticular location and resulting ampullary distortion. Urgent percutaneous transhepatic biliary drainage (PTBD) was performed, achieving effective decompression and resolution of the infectious syndrome. Following stabilization, elective laparoscopic diverticulectomy with papillary sphincteroplasty was performed with an uneventful postoperative course.



A B

Figure 1. Axial (A) and coronal (B) T2-weighted MRI — Case 1: A 4 cm periampullary duodenal diverticulum (green arrow) causing extrinsic compression of the distal CBD, with upstream biliary dilation to 17 mm. Lemmel syndrome complicated by acute cholangitis.

2.2. Case 2 — Recurrent Acute Pancreatitis of Diverticular Origin

A 55-year-old female with no history of alcohol use or known cholelithiasis was referred for etiological work-up after two episodes of acute pancreatitis six months apart. Both episodes were confirmed biochemically (serum lipase > 3× ULN) and radiologically (CT: pancreatic enlargement, peripancreatic fluid, no necrosis). Repeated biliary ultrasound, lipid profile, and investigations for toxic, metabolic, and autoimmune causes were all negative.

Bili-pancreatic MRI performed during remission identified a periampullary diverticulum on the medial wall of D2, at the superior genu, in close proximity to the ampulla of Vater. It measured 17 × 8 mm and contained predominantly air, consistent with a communicating diverticulum. Unlike Case 1, there was no biliary ductal dilation (CBD 5 mm) and no pancreatic ductal dilation (MPD 2 mm) in the intercritical period. Pancreatic parenchymal morphology was normal.

Following systematic exclusion of all alternative etiologies, the diagnosis of PADD-related recurrent pancreatitis was established. The proposed mechanism involved intermittent mechanical obstruction and functional disruption of the papillary region, leading to transient episodes of pancreatic ductal hypertension. The patient underwent surgical diverticulectomy with intraoperative papillary assessment. At 18 months follow-up, no further pancreatitis episode had occurred.

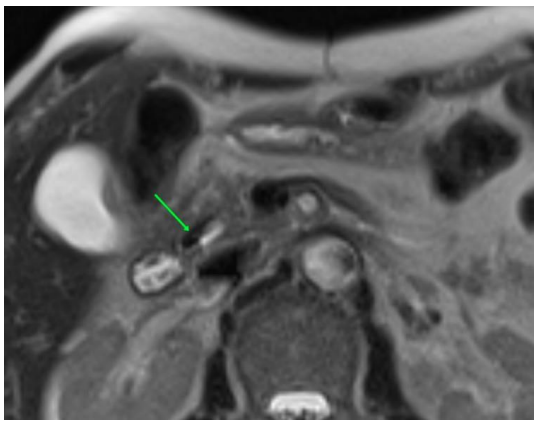


Figure 2: Axial T2-weighted MRI — Case 2: A 17 × 8 mm air-containing periampullary duodenal diverticulum (green arrow) on the medial wall of D2, in close proximity to the ampulla of Vater. No biliary or pancreatic ductal dilation in the intercritical period

3. DISCUSSION

Duodenal diverticula have a prevalence of 5–22% depending on the diagnostic modality, making them the most common small bowel diverticula [1,2]. Acquired in nature, they arise at mural weak points corresponding to vascular and ductal entry sites. The periampullary location — within 2–3 cm of the ampulla — is the most frequent and clinically significant topography [3,8]. PADD develop mainly along the medial wall of the inferior genu of D2 and superior genu of D3, in the region of biliopancreatic ductal convergence, explaining their potential to obstruct the terminal biliopancreatic system.

First described by Georg Lemmel in 1934, Lemmel syndrome refers to obstructive jaundice caused by a juxtapapillary duodenal diverticulum without choledocholithiasis or malignancy [9]. It remains underdiagnosed due to its rarity and limited clinical awareness. Pathophysiological mechanisms include direct extrinsic CBD compression by a distended diverticulum, sphincteric dysfunction from ampullary distortion, and bacterial overgrowth promoting ascending cholangitis through enterobiliary reflux [10]. In our first case, the 4 cm diverticulum caused manifest CBD compression (17 mm dilation) and MPD enlargement (6 mm), indicating obstruction of the entire sphincteric segment. The differential diagnosis with choledocholithiasis and cholangiocarcinoma must be rigorously excluded, justifying MRCP as the first-line morphological investigation [7,11].

The association between PADD and acute pancreatitis is well documented, though the mechanisms remain debated [12,13]. The leading hypothesis is intermittent MPD obstruction by the diverticulum, particularly during postprandial air-filled distension. Secondary sphincteric dysfunction and bacterial colonization promoting local pancreatic inflammation are also proposed. As PADD-related pancreatitis is a diagnosis of exclusion, thorough systematic investigation is mandatory. The absence of ductal dilation between episodes — as in our Case 2 — should not exclude this diagnosis, as obstruction may be intermittent and fully reversible between attacks [14,15].

MRCP is the gold-standard modality for biliopancreatic evaluation [7,16], outperforming ultrasound (limited by duodenal meteorism) and CT (insufficient ductal resolution). Its advantages in PADD assessment include direct diverticulum visualization, morphological ductal assessment, detection of compression signs, and characterization of diverticular content. Air content — as in Case 2 — is a specific sign of a communicating diverticulum and argues against tumor or infection [17].

Management of complicated PADD must be individualized based on clinical presentation, patient condition, diverticulum morphology, and complication type. No international consensus guidelines currently exist. In acute settings (severe cholangitis, complete biliary obstruction), emergency biliary decompression is the priority, achieved via ERCP, PTBD, or surgery depending on feasibility [18,19]. ERCP is frequently challenging due to ampullary anatomical distortion and increased perforation risk. Definitive treatment relies on surgical diverticulectomy with papillary sphincteroplasty, ensuring durable decompression and preventing recurrence [20]. For recurrent pancreatitis without chronic ductal obstruction, conservative surveillance may be considered, reserving surgery for recurrent or endoscopy-refractory cases [21].

4. CONCLUSION

Periampullary duodenal diverticula are a common and usually benign anatomical finding whose rare complications — biliary obstruction, acute cholangitis, and recurrent pancreatitis — can be life-threatening and must be promptly recognized. The two cases reported here illustrate the clinico-radiological heterogeneity of these complications and the importance of rigorous diagnostic evaluation.

Bili-pancreatic MRI plays a central role, enabling precise characterization of the diverticulum and its impact on adjacent ductal structures. Its systematic use in unexplained biliopancreatic obstruction should reduce diagnostic delay and optimize therapeutic management. A multidisciplinary approach associating gastroenterologists, interventional radiologists, and surgeons is essential. Surgical diverticulectomy remains the reference treatment for documented symptomatic forms, with excellent long-term outcomes.

Conflicts of Interest

The authors declare no conflicts of interest.

REFERENCES

1. Lobo DN, Balfour TW, Iftikhar SY, Rowlands BJ. Periampullary diverticula and pancreaticobiliary disease. Br J Surg. 1999 ;86(5):588–597.
2. Egawa N, Tomioka T, Kashiwagi H, *et al.,*Duodenal diverticula affecting the extrahepatic biliary system and the pancreatic duct. Intern Med. 1994 ;33(8):461–466.
3. Shemesh E, Friedman E, Czerniak A, Bat L. The association of biliary and pancreatic anomalies with periampullary duodenal diverticula. Arch Surg. 1987 ;122(10):1055–1057.
4. Boix J, Lorenzo-Zuñiga V, Añaños F, *et al.,* Impact of periampullary duodenal diverticula at ERCP : a proposed classification. Surg Laparosc Endosc Percutan Tech. 2006 ;16(4):208–211.
5. Zoepf T, Zoepf DS, Arnold JC, *et al.,* The relationship between juxtapapillary duodenal diverticula and disorders of the biliopancreatic system : analysis of 350 patients. Gastrointest Endosc. 2001 ;54(1):56–61.

6. Kim MH, Myung SJ, Kim YS, *et al.,* Routine bile culture and antibiotic susceptibility testing in endoscopic biliary procedures. Gastrointest Endosc. 1998 ;47(2):147–153.
7. Becker CD, Grossholz M, Becker M, *et al.,* Choledocholithiasis and bile duct stenosis: diagnostic accuracy of MR cholangiopancreatography. Radiology. 1997 ;205(2):523–530.
8. Lemmel G. Die klinische Bedeutung der Duodenaldivertikel. Arch Verdauungskrankh. 1934;56 :59–70.
9. Mäkinen J, Isolauri J, Laurikka J, Tarkka M. Lemmel's syndrome : duodenal diverticulum compressing the common bile duct. HPB Surg. 1992 ;6(1):11–17.
10. Osnes M, Lootveit T, Larsen S, Aune S. Duodenal diverticula and their relationship to age, sex, and biliary calculi. Scand J Gastroenterol. 1981 ;16(1):103–107.
11. Karayiannakis AJ, Bolanaki H, Courcoutsakis N, *et al.,* A rare cause of obstructive jaundice: Lemmel's syndrome. Case Rep Gastroenterol. 2016 ;10(3):795–800.
12. Yildirgan MI, Başoğlu M, Yilmaz I, *et al.,* Periampullary duodenal diverticula causing pancreatic and biliary disease. J Hepatobiliary Pancreat Surg. 2001 ;8(5):443–445.
13. Inceoglu R, Dosluoglu HH, Kullu S, *et al.,* Diverticula of the duodenum and choledocholithiasis. Hepatogastroenterology. 1992 ;39(6):528–529.
14. Uomo G, Manes G, Ragozzino A, *et al.,*Periampullary extraluminal duodenal diverticula and acute pancreatitis: an underestimated etiological association. Am J Gastroenterol. 1996 ;91(6) :1186–1188.
15. Yin WY. Giant duodenal diverticulum: a review. Chang Gung Med J. 2007 ;30(2):105–117.
16. Adamek HE, Albert J, Breer H, *et al.,*Pancreatic cancer detection with MRCP and ERCP : a prospective controlled study. Lancet. 2000 ;356(9225):190–193.
17. Papalambros E, Sigala F, Felekouras E, *et al.,* Giant duodenal diverticulum: is it an unusual presentation of biliary colic? World J Gastroenterol. 2005 ;11(36):5724–5726.
18. Kim CW, Chang JH, Kim JH, *et al.,* Size and type of periampullary duodenal diverticula are associated with bile duct diameter and recurrence of biliary symptoms. J Gastroenterol Hepatol. 2013 ;28(5):893–898.
19. Tomita R, Tanjoh K, Fujisaki S. Surgical treatment for Lemmel's syndrome : case report and review of the literature. Hepatogastroenterology. 2000 ;47(33):623–627.
20. Economou M, Zissis M. Bacteriobilia: aetiology, diagnosis, and treatment. Ann Gastroenterol. 2003 ;16(4):318–323.
21. Wiesner W, Schöb O, Hauser RS, Hauser M. Acute colonic pseudo-obstruction and acute periampullary diverticulum in a patient with duodenal diverticulosis. AJR Am J Roentgenol. 2001 ;176(4):1060–1062.