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Walled-Off Necrosis of the Pancreas Complicated by A Choledochopancreatic Fistula: A Case Report

K. Gharbi[1]*, H. Moustahfid[1], A.A. BAH[1], S. Soukrat[1], R. Akka[1], F. Fihri[2], S. Ben Elhnd[3]

[1]Department of Gastroenterology, Avicenne Military Hospital, Marrakech, Morocco
[2]Department of Visceral Surgery, Avicenne Military Hospital, Marrakech, Morocco
[3]Department of Radiology, Avicenne Military Hospital, Marrakech, Morocco

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***Corresponding author:** K. Gharbi
Department of Gastroenterology, Avicenne Military Hospital, Marrakech, Morocco

Abstract **Case Report**

In rare cases, acute pancreatitis (AP) may lead to severe, even life-threatening complications requiring surgical intervention. A biliopancreatic fistula may occur in this context and poses significant management challenges. We report the case of a 48-year-old male patient with a history of stage E acute pancreatitis. Six months later, the patient developed a large pancreatic necrotic collection (Walled-off necrosis, WON) complicated by a biliary fistula. The patient underwent a doublé biliary diversion with anastomosis. His postoperative course was uneventful, and his clinical condition was satisfactory after the procedure.

Keywords: Acute pancreatitis, walled-off necrosis, biliary fistula, surgery.

INTRODUCTION

The development of a biliopancreatic fistula in the context of acute pancreatitis (AP) is generally a consequence of necrotizing pancreatitis. It may result from necrosis and erosion of the walls of the pancreatic and biliary ducts, from communication between a pseudocyst and the biliary tract when the pseudocyst erodes into the adjacent common bile duct or gallbladder, or from infected necrosis [1]. The simultaneous occurrence of these severe complications is relatively rare and requires a multidisciplinary approach. We report a case illustrating the complex yet successful management of a biliopancreatic fistula secondary to acute necrotizing pancreatitis [2].

CASE REPORT

We present the case of a 48-year-old man with a history of gallstone-related acute pancreatitis classified as stage E. Six months after the initial episode, the patient presented to the emergency department with persistent abdominal pain associated with repeated episodes of vomiting. On admission, his general condition was significantly impaired. Clinical examination revealed cutaneous and mucosal jaundice along with a high-grade fever reaching 39°C, suggestive of an infectious process. Abdominal examination showed diffuse tenderness without guarding or rigidity, indicating an underlying intra-abdominal pathology requiring further investigation.

Laboratory tests performed on admission revealed a marked inflammatory syndrome, consistent with an ongoing infectious or inflammatory process. Serum lipase levels were within normal limits (60 IU/L), arguing against recurrent acute pancreatitis. In contrast, biochemical tests demonstrated a cholestatic syndrome, suggesting involvement or obstruction of the biliary tract. Renal function was preserved, with no significant abnormalities in renal parameters, which was reassuring in the overall assessment of the patient.

Contrast-enhanced abdominal computed tomography (CT), supplemented by magnetic resonance imaging (MRI), revealed a large, well-defined, encapsulated peripancreatic necrotic collection with thickened walls, measuring approximately 17 × 8.7 cm at its largest dimensions.

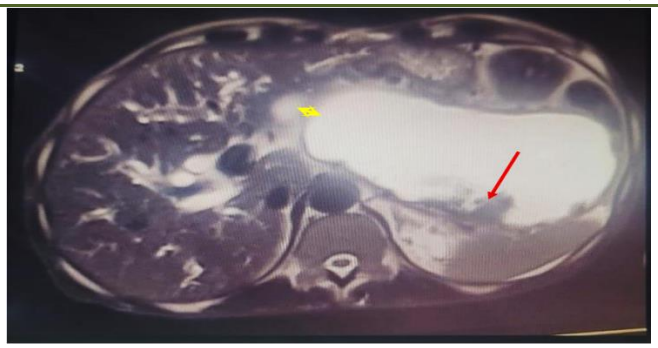

Figure 1: Abdominal MRI showing a large encapsulated peripancreatic necrotic collection with thickened walls measuring 17 × 8.7 cm (red arrow), associated with a fistulous communication to the common bile duct (yellow arrowhead), which is dilated to 18 mm, along with a sludge-filled distended gallbladder (hydrocholecyst)

The patient was started on intravenous antibiotic therapy with a third-generation cephalosporin combined with metronidazole.

An endoscopic retrograde cholangiopancreatography (ERCP) was attempted with the aim of placing a plastic biliary stent to bypass the fistula; however, cannulation of the common bile duct failed due to the presence of a type III papilla (prominent and pendulous), which was compressed inferiorly by the pancreatic collection.

Following the failure of the initial endoscopic management, a surgical approach was indicated.

A supra-umbilical midline laparotomy was performed, allowing exploration of the abdominal cavity. This enabled the creation of a doublé bypass consisting of a biliodigestive and a gastrojejunostomy, along with evacuation of the pseudocyst by percutaneous puncture (Figure 2).

Figure 2: Purulent fluid from the pancreatic pseudocyst

After complete evacuation of the purulent material and careful necrosectomy, the presence of bile within the cavity was identified, strongly suggesting the existence of a biliary fistula. This intraoperative finding was a key element in guiding subsequent therapeutic management.

Samples were sent for cytobacteriological, biochemical, and histopathological analysis (Table 1).

Table 1: Biochemical analysis results of the aspirated fluid from the collection.

Module	R/P	IDE	Test	Result	Interpretation	Annotation	Time
2	LB237/1	434/04	CA19-9	>1200.00 U/mL	EXP CNTRL >		05.02.2026 15:22
2	LB237/1	434/04	CEA	86.25 ng/mL	EXP CNTRL		05.02.2026 15:22
2	LB237/1	434/04	CA19-9	>1200.00 U/mL	EXP CNTRL >	R	05.02.2026 15:22
2	LB237/1	434/04	CEA	67.04 ng/mL	EXP CNTRL	R	05.02.2026 15:21
1	LB237/1	434/04	Lipase	>300.00 U/L	EXP CNTRL >		05.02.2026 15:03
1	LB237/1	434/04	Lipase	>300.00 U/L	EXP CNTRL >	R	05.02.2026 15:03
1	LB237/1	434/04	TBil2	87.2 µmol/L			05.02.2026 15:02
1	LB237/1	434/04	TBil2	86.8 µmol/L		R	05.02.2026 15:02
1	LB237/1	434/04	Glucose	8.86 mmol/L	CNTRL HIGH		05.02.2026 15:00
1	LB237/1	434/04	Glucose	8.88 mmol/L	CNTRL HIGH	R	05.02.2026 15:00

Biological analysis revealed a marked elevation of CA 19-9, with levels exceeding 1,200 U/mL. This significantly elevated value is particularly noteworthy, especially as the carcinoembryonic antigen (CEA) level, although above normal, showed only a moderate increase in comparison with CA 19-9.

In addition, serum lipase levels were significantly elevated, exceeding 300 U/L, consistent with ongoing pancreatic involvement or persistent pancreatic injury.

Analysis of the pseudocyst content also demonstrated the presence of bilirubin, with a measured level of 67 µmol/L. This finding strongly suggests the existence of a fistulous communication with the biliary tract, possibly secondary to the extension of the inflammatory process or erosion of a segment of the common bile duct.

DISCUSSION

Acute pancreatitis is a common condition, with 15–20% of cases progressing to necrotizing pancreatitis, which is associated with significant morbidity and mortality, particularly in the presence of infected necrosis [3,4]. According to the Revised Atlanta Classification, walled-off necrosis typically occurs after the fourth week of evolution and may be associated with severe local complications, as observed in our case.

Biliary complications in acute pancreatitis are usually related to transient inflammatory compression of the distal common bile duct. In contrast, biliary fistulization is an exceptional complication. The underlying pathophysiological mechanisms include direct erosion of the common bile duct by an adjacent necrotic collection, ischemic injury of the duct wall secondary to prolonged compression, or rupture of a pseudocyst into the main biliary tract [5,6].

The reported causes of pancreaticobiliary fistula include pancreatic pseudocyst [7,8], acute necrotizing pancreatitis, chronic pancreatitis and/or pancreatic lithiasis [9,10], intraductal papillary mucinous neoplasm [11], and pancreatic tuberculosis [12].

In our case, the large encapsulated necrotic collection measuring 17 × 8.7 cm, associated with dilation of the common bile duct up to 18 mm, suggests progressive inflammatory erosion leading to fistula formation. The presence of jaundice with fever and a biochemical cholestatic syndrome should raise suspicion not only for extrinsic biliary obstruction but also for an abnormal biliary communication.

Imaging plays a crucial role in diagnosis. Computed tomography (CT) allows assessment of the extent of necrosis, while magnetic resonance cholangiopancreatography (MRCP) is particularly useful for identifying biliary communication [13]. In the literature, most reported cases are diagnosed either intraoperatively or during endoscopic evaluation.

Endoscopic retrograde cholangiopancreatography (ERCP) theoretically represents the first-line treatment in cases of biliary fistula, enabling internal drainage through the placement of plastic or metallic stents [14]. This approach reduces intraductal pressure and promotes spontaneous fistula closure. However, in certain situations—particularly in the presence of significant anatomical distortion or extrinsic papillary compression—cannulation may be technically impossible, as observed in our case.

The management of infected necrotic collections currently follows a “step-up” approach, initially favoring minimally invasive drainage (endoscopic or percutaneous), followed, if necessary, by delayed necrosectomy [15]. However, the coexistence of a biliary fistula, sepsis, and failed endoscopic intervention constitutes a reasonable indication for upfront surgical management.

Surgery allows control of the septic source, necrosectomy, cholecystectomy in cases of lithiasis-related etiology, and temporary external biliary diversion (e.g., Kehr drain).

The favorable outcome observed in our patient confirms that, despite the potential severity of this rare complication, appropriate multidisciplinary management can achieve an excellent functional result.

CONCLUSION

Choledochopancreatic fistula complicating acute necrotizing pancreatitis is an exceptionally rare entity. Diagnosis relies on imaging, and the therapeutic strategy must be individualized. While ERCP represents the first-line treatment, surgery retains a key role in cases of endoscopic failure or when associated septic complications are present.

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