

Ikeda Type I Peripapillary Choledochoduodenal Fistula: A Case Report

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ABSTRACT

Despite their low incidence, choledochoduodenal fistulas pose a diagnostic challenge because clinical presentations range from an asymptomatic condition identified incidentally to acute cholangitis, while neoplastic disease may not be readily excluded during the initial evaluation. The increasing use of endoscopic retrograde cholangiopancreatography (ERCP) has provided greater insight into this entity. We report a case of an Ikeda type I peripapillary choledochoduodenal fistula in a patient who recovered uneventfully after ERCP, and we review the literature regarding its management and follow-up.

KEYWORDS: fistula; peripapillary; choledochoduodenal; Ikeda; type I

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INTRODUCTION

Biliary fistulas are classically defined as abnormal communications between the biliary tract and an adjacent organ or the external environment. The former are classified as internal fistulas, whereas those communicating with the skin are classified as external fistulas. Internal biliary fistulas are uncommon; an incidence of 0.22% was reported in a series of 66,340 autopsies (1).

The occurrence of these fistulas has declined substantially with the advent of ERCP and newer biliary technologies (2). They are more common in women, with reported female-to-male ratios of 3:1 and 12:9 (3,10). Most patients present between 60 and 80 years of age; however, when the fistula results from nonbiliary disease, age at presentation generally reflects the age distribution of the underlying disorder (4,10). According to etiology, biliary fistulas are classified as spontaneous, iatrogenic, postoperative, or post-traumatic (3). Most spontaneous fistulas arise as complications of gallstone disease, followed by malignancies of the gallbladder, common bile duct, duodenum, or pancreas; less commonly, they result from perforation associated with duodenal Crohn disease, duodenal peptic ulceration, or paraduodenal abscesses (3,4). Based on the organs involved, bilioenteric fistulas account for most cases and include cholecystoduodenal, cholecystocolic, cholecystogastric, choledochoduodenal (2%-5%), and combined fistulas (2%-3%). Less common subtypes include biliobiliary, bronchobiliary, pleurobiliary, and biliovascular fistulas (3,5-7). Communications with the urinary bladder,

gravid uterus, appendix, pericardial cavity, and vagina have also been reported (8). Clinical manifestations are highly variable, and some patients remain asymptomatic (3). The natural history is frequently characterized by complications, most commonly cholangitis. Less frequently, passage of gallstones into the gastrointestinal tract may cause intestinal obstruction, including gallstone ileus due to impaction at the ileocecal valve (3,9) or Bouveret syndrome due to duodenal obstruction (11).

Periampullary choledochoduodenal fistulas are divided into two types (10,12,15).

Type I fistulas are usually located on the longitudinal fold in close proximity to the major papilla and are also referred to as parapapillary choledochoduodenal fistulas. They are generally small and originate from the intramural portion of the common bile duct. Nearly all parapapillary fistulas have been attributed to spontaneous extrusion of common bile duct stones (13).

This type of fistula develops when a stone small enough to enter the intramural portion of the common bile duct becomes impacted at that site. Increased intraductal pressure leads to ulceration and ultimately to stone extrusion, with subsequent fistula formation (14).

Passage of dilators into the duodenum during common bile duct exploration may also cause a parapapillary choledochoduodenal fistula. Such fistulas are usually distal and communicate with the duodenum within the distal 2 cm of the common bile duct (10).

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Type II fistulas are located in the duodenal mucosa adjacent to the longitudinal fold and may result from larger stones, penetration by a duodenal ulcer, stones impacted in the cystic duct, or complications of laparoscopic cholecystectomy. Unlike type I fistulas, type II fistulas are large enough to permit passage of larger stones. They are also located more proximally and allow drainage of the biliary system into the duodenum approximately 2 cm proximal to the junction of the common bile duct and the major papilla. The combined frequency of both types is 2.53% in endemic areas; in nonendemic areas, it has been reported to be three to four times higher (10,12). Reported causes include cholelithiasis, choledochostomy, periampullary tumor invasion, pancreatitis, open or laparoscopic cholecystectomy, perforated duodenal ulcer, and papillitis (10,16,17).

CASE PRESENTATION

A 41-year-old woman had a history of hypothyroidism diagnosed at 16 years of age and treated with levothyroxine. Beginning in early 2025, she developed biliary colic followed by fever and jaundice, without reported weight loss. During that admission, she was diagnosed with choledocholithiasis. Her pain improved and the fever resolved during hospitalization. In February 2025, she underwent laparoscopic cholecystectomy. The gallbladder measured 8 × 6 × 4 cm and contained multiple calculi; the operative findings were classified as Parkland grade IV. No bile duct dilatation was observed intraoperatively. Postoperatively, she tolerated oral intake, although postprandial pain did not completely resolve after resumption of a full diet. She was discharged and subsequently required five emergency department admissions. The most recent admission occurred in June 2026 for abdominal pain, tachycardia, vomiting, fever up to 39 °C, jaundice, and dark urine. Laboratory results were as follows: leukocyte count, $18.3 \times 10^3/\mu\text{L}$; hemoglobin, 11.3

g/dL; platelet count, $216 \times 10^3/\mu\text{L}$; glucose, 144 mg/dL; urea, 19.2 mg/dL; blood urea nitrogen, 9.0 mg/dL; creatinine, 0.98 mg/dL; sodium, 138 mmol/L; potassium, 3.9 mmol/L; chloride, 101 mmol/L; calcium, 8.4 mg/dL; phosphorus, 3.24 mg/dL; magnesium, 1.27 mg/dL; albumin, 3.7 g/dL; total bilirubin, 4.7 mg/dL; direct bilirubin, 3.58 mg/dL; indirect bilirubin, 1.1 mg/dL; alkaline phosphatase, 499 U/L; gamma-glutamyl transferase, 623 U/L; amylase, 55 U/L; lipase, 35 U/L; prothrombin time, 19.5 seconds; international normalized ratio, 1.50; and activated partial thromboplastin time, 31.5 seconds. Hepatobiliary ultrasonography showed an absent gallbladder consistent with prior surgery, preserved caliber of the intrahepatic bile ducts, and diffuse extrahepatic bile duct dilatation measuring up to 11.2 mm in the distal third and 12.9 mm in the middle third. No intraductal stones were visualized at the time of examination. Mild hepatic steatosis was also noted.

After prophylactic rectal indomethacin, ERCP was performed during the same hospitalization. A Haralldsson type III major papilla and an Ikeda type I periampullary choledochoduodenal fistula were identified (Figures 1 and 2). The bile duct was cannulated with a sphincterotome on the first attempt. Cholangiography demonstrated a 17.6-mm bile duct with a 10-mm filling defect in the distal third. An extended biliary sphincterotomy was performed (Figure 3), followed by balloon extraction of a single stone (Figure 4). Completion cholangiography showed adequate contrast drainage, and the procedure was concluded (Figures 5 and 6).

The patient was observed for 36 hours. During this period, she showed no clinical evidence of cholangitis or pancreatitis, remained afebrile and without tachycardia, then tolerated oral intake. She ambulated with assistance and had no recurrence of jaundice or dark urine; she was discharged home in stable condition.



Figure 1. Peripapillary choledochoduodenal fistula and biliary cannulation.

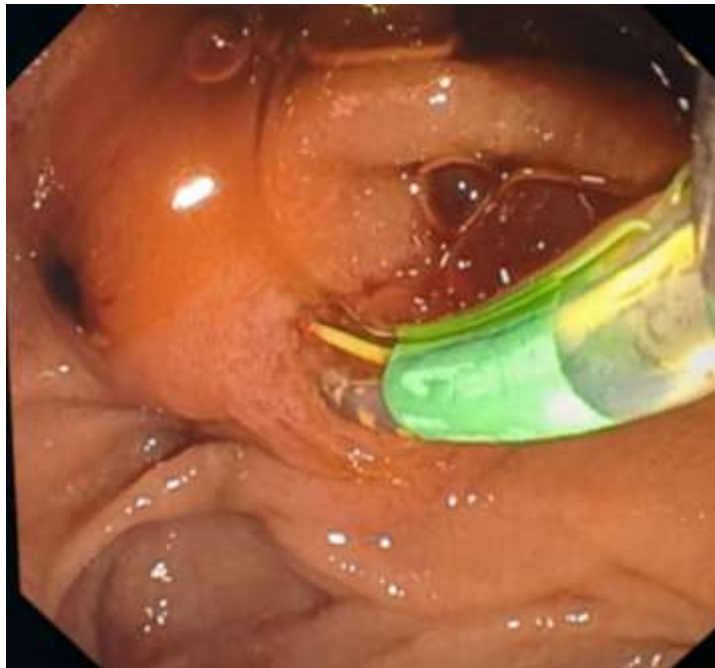


Figure 2. Close-up view of the peripapillary choledochoduodenal fistula and biliary cannulation.



Figure 3. Extended biliary sphincterotomy.



Figure 4. Extraction of the bile duct stone.

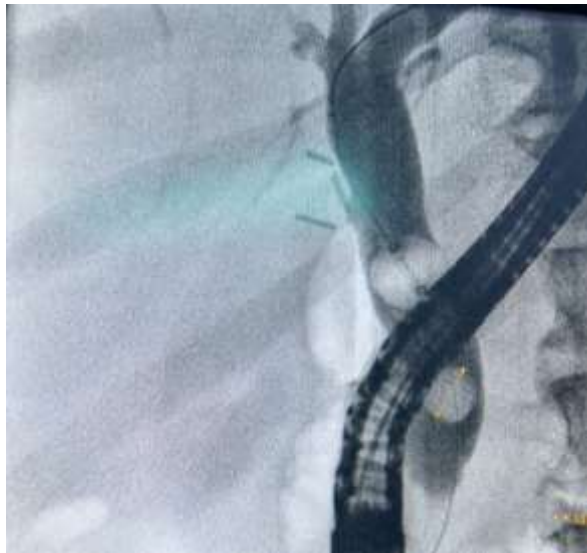


Figure 5. Cholangiogram before extraction of the 10-mm stone.



Figure 6. Cholangiogram after stone extraction.

DISCUSSION

Currently, the diagnosis of a peripapillary choledochoduodenal fistula is usually made intraprocedurally during ERCP because the initial clinical presentation overlaps with that of conventional acute cholangitis. The presence of cholangitis is particularly relevant because an additional communication with the main bile duct bypasses the sphincteric valve mechanism and may facilitate ascending biliary infection. Infection, in turn, may promote stone formation and has been associated with gallstone ileus (3,10). In patients with a fistula but no cholangitis, uncertainty remains as to whether the fistula should be followed with repeat ERCP or closed during the index procedure. The evidence is limited and management remains controversial. Available approaches range from surgical treatment in distal or complex cases such as Roux-en-Y choledochojejunostomy to initial endoscopic therapy with sphincterotomy and stent placement. The latter may promote fistula closure by decreasing biliary flow and pressure through transpapillary

decompression, followed by subsequent stent removal and reassessment (10,19). At present, management should be individualized according to each patient's clinical presentation. Nevertheless, recurrent cholangitis and biliary reflux should be regarded as potential stimuli for premalignant and malignant epithelial changes in the biliary tree and gallbladder and, consequently, for carcinogenesis (10,18,19).

CONCLUSION

There is currently no evidence-based consensus regarding the treatment that provides the greatest benefit for patients with peripapillary choledochoduodenal fistulas. This reflects the rarity of the condition and the fact that most historical descriptions involved duodenal ulcers, although choledocholithiasis appears to be a more frequent cause. In the present case, the documented bile duct stone was managed with endoscopic sphincterotomy without stent placement, which facilitated biliary drainage into the

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duodenum. In the setting of prior cholecystectomy, this approach may reduce the risk of recurrent choledocholithiasis, as suggested by the patient's favorable in-hospital course through discharge. Additional case reports with long-term follow-up are needed to define, with greater statistical power, the optimal management of these patients and their complications.

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