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RESEARCH ARTICLE

Diagnosis and Treatment of Non-Foreign Body-Associated Secondary Dilatation of The Common Bile Duct

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ABSTRACT

Objective: To explore the etiology, diagnosis and treatment of non-foreign body secondary choledochectasis.

Methods: The clinical data of 162 cases admitted from January 1994 to December 2021 were retrospectively studied.

Results: The causes and diagnosis of non-foreign body secondary dilatation were accurately identified by careful history, physical examination, imaging and laboratory tests into 10 categories: inflammation; sphincter of Oddi dysfunction; post gastric bariatric surgery; compression; compensation; Bile duct injury; duodenal disease; other factors.

Conclusion: The etiology of non-foreign body secondary choledochectasis is complex. Effective treatment should be selected according to different etiologies.

Keywords: Non-foreign body-associated, dilatation, common bile duct, etiology, diagnosis, treatment

Introduction

Common bile duct (CBD) dilatation in adults is defined as the largest diameter measuring ≥8 mm.A diameter of 8–12 mm is defined as "mild", 12–16 mm as "moderate", 16–20 mm as "severe", and >20 mm as "very severe" dilatation. It is classified into congenital[1] and secondary[2] forms. Congenital bile duct dilatation and secondary bile duct dilatation have significant differences in etiology, dilation nature, lesion location and pathology. Congenital bile duct dilatation is a congenital malformation in which the normal tissue structure is lost, the submucosa is significantly thickened and occupied by collagen fibers, and the malignant transformation rate is 8%-15%. Secondary bile duct dilatation is caused by acquired factors and is a full-course dilation. The bile duct lesions are located distally, and the duct wall structure is basically normal. Carcinogenesis generally does not occur unless the primary disease is malignancy or is invaded by cancer[3].Secondary dilatation of the CBD can be divided into foreign body (FB)-associated and non-FB-associated types. FB-associated dilatation is caused by FBs such as stones, biliary roundworms, and cholangiocarcinoma. This type of dilatation has a clear cause and is easy to diagnose. Non-FB-associated dilatation is secondary dilatation where FBs (e.g., stones, parasites, tumors, or prostheses such as stents and balloons) are not found in the CBD. Non-FB-associated dilatation is not uncommon in clinical practice. However, it is sometimes difficult for clinicians to diagnose and treat it due to the unclear cause. To this end, we investigated 162 cases of non-FB-related secondary expansion of CBD, with the aim of investigating their etiology, diagnosis and treatment.

Clinical data

Data were collected from 162 patients (54 males and 108 females; mean age = 45 years; age range, 10–65 years) with non-FB-associated secondary dilatation of the CBD treated by our research team. Of the 162 patients, 90% had pain, tenderness or discomfort in the epigastrium, 84.6% (137/162) had gallstones, 62.3% (101/162) had an increased level of alkaline phosphatase (ALP), 48.2% (78/162) had preoperative jaundice, 25.9% (42/162) had total serum bilirubin >68.4 μmol/L (4 mg/dL), and 32.1% (52/162) had serum and urine levels of amylase >128 Winslow units.

Results

All patients underwent surgical exploration and treatment. Intraoperative measurements of the CBD diameter are shown in Table 1. The passage of Bakes' dilators through the distal CBD by etiology is summarized in Table 2.

Table 1. CBD diameter

Etiology	Type	CBD dilatation (mm)		Total number of patients	%
		8–12	>12		
Pancreatitis	I	0	32	32	32.1 (N = 52)
	II	10	8	18	
	III	1	2	2	
Stenosing papillitis	0	0	8	8	4.9
Bile-duct injury	0	5	7	12	7.4
After cholecystectomy	0	9	6	15	9.3
SO dysfunction	Strained	8	9	17	22.8 (N = 37)
	Relaxed	12	8	20	
Stones passed into the small intestine	0	9	3	12	7.4
Parapapillary diverticulitis	0	4	2	6	3.7
PSC	0	3	2	5	3.1
Others	0	9	6	15	9.3

Note: SO = Sphincter of Oddi; PSC = primary sclerosing cholangitis.

Table 2. Passage of Bakes' dilators through the distal CBD

Etiology	Size (#) of Bakes' dilators			Total number of patients	%
	≥3	4–9	≥10		
Pancreatitis	32	18	2	52	32.1
Stenosing papillitis	8	0	0	8	4.9
Bile-duct injury	8	4	0	12	7.4
After cholecystectomy	0	13	2	15	9.3
SO dysfunction	0	17	20	37	22.8
Stones passed into the small intestine	0	8	4	12	7.4
Parapapillary diverticulitis	0	4	2	6	3.7
PSC	0	5	0	5	3.1
Others	1	11	3	15	9.3

Note: #1 = 1 mm in diameter, et cetera; SO = sphincter of Oddi; PSC = primary sclerosing cholangitis.

An enlarged and hard pancreas was observed in 52 patients. Pathology was undertaken for some of these patients, which confirmed an intraoperative diagnosis of chronic pancreatitis. Moreover, 24 of them had evidence of acute pancreatitis (intraoperative incision of the pancreatic capsule was undertaken for drainage). Bile duct cholangiography revealed that there were two types of distal CBD stenosis caused by pancreatitis. Type I (n = 32) was fixed annular stenosis, which

had a length of 30–35 mm, mostly with a smooth surface. It was seen commonly in patients with the third segment of the CBD running through the pancreatic parenchyma. Among them, 28 underwent proximal dilated CBD-jejunum Roux-*en*-Y anastomosis (hereafter referred to as "biliary-enteric drainage"), two underwent pancreaticoduodenectomy, and two underwent sphincteroplasty, for whom biliary-enteric drainage was done 3-months later due to postoperative jaundice. In type II (n = 18), the anterior wall of the distal CBD was compressed and adherent, and the lumen was relatively narrow and projected backwards. It was seen commonly in patients with no or little coverage of pancreatic tissue at the posterior wall of the CBD and non-severe fibrosis. After adhesiolysis, a Bakes' dilator (> #6) could pass through the distal CBD, and no other treatment was given.

Eight patients with stenosing papillitis and 12 patients with bile-duct injury (one following pancreatic sphincterotomy and stone removal, three following radical gastrectomy for cancer, and eight following laparoscopic cholecystectomy) underwent biliary-enteric drainage.

Fifteen patients with compensatory dilatation of the bile duct caused by cholecystectomy did not receive special treatment because the bile-duct diameter was ≤13 mm.

Thirty-seven patients had sphincter of Oddi (SO) dysfunction (SOD). Among them, 17 patients were diagnosed as having "strained" SOD by endoscopic measurement of SO pressure >40 mmHg; 15 of them underwent sphincteroplasty and two underwent biliary-enteric drainage. The other 20 patients (12.3%) were diagnosed as having "relaxed" SOD, in whom a preoperative SO pressure <10 mmHg was documented and a large (approximately 25–30 mm in diameter) and fish mouth-shaped SO opening was observed upon intraoperative choledochoscopy; and all of them underwent biliary-enteric drainage.

Twelve patients had stones expelled into the duodenum (with no stones in the CBD) and six had parapapillary diverticulitis. No other treatment was undertaken intraoperatively. Postoperative antibiotic treatment was administered.

Five patients had primary sclerosing cholangitis (PSC) and were treated with endoscopic stents. One patient had a poor outcome and underwent biliary-enteric drainage 3-months later.

All patients recovered after undergoing the specific treatment described above, and were discharged from hospital. A total of 101 patients (62.3%) were followed up without serious complications for 3–5 years, but 61 patients were lost to follow-up.

Discussion

Diagnosis

Combination of four examinations

In recent years, there has been a tendency to emphasize imaging, but overlooking the importance of medical history and physical examinations, which results in inadequate accuracy in the diagnosis. For difficult and complex cases,we advocate that medical history, physical examination, imaging, and laboratory tests are all indispensable for an accurate diagnosis.

Medical history: Patients should be asked about the cause of the onset, nature, time, and location of pain, prior history of similar conditions, previous examinations and treatments, prior history of surgery and trauma, and the relationship between them.

Physical examination: There were more female patients than male patients. Ninety percent had epigastric pain or discomfort. The pain was more intense if accompanied by gallstones or pancreatitis. Some patients even had severe pain, jaundice, and fever. A positive Murphy's sign is suggestive of

gallstones and cholecystitis. Tenderness in the left upper quadrant is suggestive of pancreatic disease.

Imaging: B-ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), and endoscopic retrograde cholangiopancreatography (ERCP) not only provide a complete and accurate picture of bile-duct dilatation, they also identify the causes of bile-duct dilatation in most cases. They are essential examinations for patients with biliary dilatation.[4–6] In recent years, widespread use of magnetic resonance cholangiopancreatography (MRCP), endoscopic ultrasonography,[7] endoscopic retrograde cholangiopancreatography, and percutaneous transhepatic cholangiography has increased the prevalence of the diagnosis significantly. MRCP has replaced invasive methods as the "gold standard" for the diagnosis of bile-duct dilatation.[8]

Laboratory tests: Laboratory tests should be undertaken according to the possible causes. More than 60% of these patients had an increased ALP level, 25% had an increased level of total serum bilirubin and abnormal liver function, and 32% had increased levels of amylase in serum and urine. Tests for tumor markers should be conducted if necessary to facilitate the diagnosis and differential diagnosis.

Etiological diagnosis

There are several causes of non-FB-associated secondary CBD dilatation.[9] They can be classified into 10 categories.

Inflammation

Inflammation could be subdivided into two categories. The first type is stenosing papillitis. Choledochoscopy and ERCP revealed that the papilla was enlarged and smooth, with a narrowed, red, and mildly erosive orifice. Only Bakes' dilators of number ≤3 could pass through the distal CBD. The proximal bile duct was dilated significantly. Stenosing papillitis is often secondary to chronic discharge of stones in the biliary duct or pancreatic duct, chronic pancreatitis, sclerosing cholangitis, reflux cholangitis, peptic ulcer disease, or inflammation and fibrosis caused by the onset of cholesterolosis.[10] Transduodenal sphincteroplasty to expand the orifice relieves pain in some patients.[11] The second type is PSC. PSC is a chronic cholestatic liver disease of unknown etiology. It is characterized by persistent inflammation, destruction, as well as fibrosis of intrahepatic and extrahepatic bile ducts. Irregular narrowing and dilatation of the biliary system results in a typical "bead-like" cholangiogram.[12] The most common complications are ulcerative colitis (80%) and chronic pancreatitis.[13] Patients with PSC are susceptible to hepatobiliary and intestinal malignancies, such as cholangiocarcinoma, gallbladder cancer, hepatocellular carcinoma, or colorectal cancer.[14]

SO dysfunction (SOD)

The SO is the smooth muscle valve between the distal CBD and the opening of the pancreatic duct. It not only regulates the flow of bile and pancreatic juice into the duodenum, it also prevents the reflux of duodenal juice into the bile duct and pancreatic duct. It was first described in 1887 by the Italian anatomist Ruggero Oddi.[15] Due to its location, SOD can be divided into biliary (which is more common), pancreatic, and mixed SOD.[16] Biliary SOD can be subdivided further into strained SOD and relaxed SOD. Strained SOD can be diagnosed by endoscopic manometry.[17] Jeffrey and colleagues suggested that SOD can be diagnosed by a mean basal sphincter pressure >40 mmHg.[18,19] Manometry is not accurate due to relaxation of the SO under anesthesia. Therefore, endoscopic manometry should be carried out before or after surgery. Relaxed SOD can be diagnosed if one of the following findings is present: (a) a low mean basal sphincter pressure (<10 mmHg); (b) passage of a Bakes' dilator of size ≥10 (≥10 mm) during intraoperative exploration (a Bakes' dilator of size >10 cannot pass through the strained SO even

if it is relaxed under anesthesia); (c) Intraoperative choledochoscopy showed that the opening of the sphincter of Oddi was fish-mouthed, with a diameter of about 25-30 mm, and the biliary lens could easily enter the duodenum; (d) On the premise of excluding duodenal obstruction, the duodenum Angiography shows reflux of barium into the bile duct; (e) Gas is visible in the bile duct; (F) In relaxed SOD, the large papillary orifice results in the reflux of small-intestinal fluid, which often causes retrograde infection and inflammation of the biliary tract, thereby leading to bile-duct dilatation.Two or more conditions must be met to diagnose relaxed SOD.

After gastric bariatric surgery

Roux-*en*-Y gastric bypass has been the type of bariatric surgery recommended for obese patients in recent years. Neal and coworkers discovered bile-duct dilatation of unknown cause in all 269 patients with normal endoscopic findings within the bile duct after bariatric surgery, regardless of whether they underwent cholecystectomy.[20] Patrick and collaborators reported that bile-duct dilatation after Roux-*en*-Y gastric bypass for obesity was associated with SOD.[21] The possible reason is that the hepatic and biliary vagus nerves are destroyed during separation after gastrectomy, which results in the failure of rhythmic relaxation and contraction of the bile duct,[22] thereby leading to SOD and bile-duct dilatation.

Compression

Compression can be subdivided into two categories. The first type is compression by pancreatic disease. This refers to compression outside the CBD wall due to pancreatic disease, including compression of the pancreatic bile-duct due to pancreatic-head cancer,[23] pancreatic cyst,[24] acute and chronic pancreatic pancreatitis (32%),[25,26] which leads to dilatation of the proximal CBD. Chronic pancreatitis causes three types of distal CBD stenosis that lead to proximal bile-duct dilatation.[27] Type I is fixed annular stenosis of the CBD. Type II is relative stenosis due to compression of the anterior wall of the CBD.[28] Type III is compression of the distal bile duct by pancreatic pseudocyst. The second type is compression due to other diseases. Cancer metastasis to the bile duct, peribiliary aneurysm,[29] parapapillary diverticulitis, lymphadenopathy, Ormond disease, neuroma, and hydronephrosis[30] can also compress the bile duct and lead to proximal bile-duct dilatation.

Compensation

Compensatory bile-duct dilatation occurs after cholecystectomy or due to chronic atrophic cholecystitis.[4]

Bile-duct injury

Bile-duct injury can be subdivided into four categories. The first type is traumatic injury. Bile-duct stenosis and adhesion to, and traction upon, surrounding tissues caused by trauma to the biliary tract can lead to proximal bile-duct dilatation.[31] The second type is surgical injury. Bile-duct injury can be caused by open surgery, incorrect ligation or transection of the CBD during laparoscopic or robotic surgery,[32] electrical burns, a too-short residual cystic duct, loose ligatures or metal clips, bile leakage, anastomotic leakage and stenosis,[33] inappropriate surgery timing, and an unskilled procedure. The third type is interventional injury. Wang and colleagues reported a prevalence of bile-duct injury of 2.1% (50/2340) after transcatheter arterial chemoembolization (TACE) for hepatic malignancies.[34]Moreover, the prevalence of bile-duct injury (8.6%) after TACE was significantly higher for metastatic malignancy than for hepatocellular carcinoma (0.5%). The complications of bile-duct injury include bile-duct stenosis, jaundice, biloma, and proximal bile-duct dilatation.[35,36] The fourth type is endoscopic injury. In endoscopic sphincterotomy and stone removal, a

too-long incision can lead to defect and stenosis of the duodenal wall, or complications (e.g., biliary-tract infection and bile-duct dilatation). Nevzat and colleagues reported that 6.8% of 261 patients who underwent sphincterotomy and stone removal had major complications, such as bile leakage, stenosis, and proximal bile-duct dilatation, due to anatomic abnormalities.[37]

Ectopic opening of the CBD

Ectopic opening of the CBD means that the opening of the CBD is not at the duodenal papilla, but instead at the stomach, pyloric duct, duodenal bulb, or the third or fourth segment of the duodenum.[38] Ulku and collaborators reported that ectopic opening of the CBD was a rare congenital anomaly that occurred at a prevalence of 2.5% (10/400), usually at the duodenal bulb.[39] It occurred more frequently in male than in female patients. Overall, 45% of patients had duodenal ulcers, 94% had significant dilatation of the CBD, 51% had CBD stones, and 33% had pancreatic-duct dilatation.[40] The main cause of CBD dilatation is a narrow opening. It is because the ectopic opening does not have a complete sphincter and allows only a crack, thereby causing stenosis. Clinical manifestations, including recurrent or refractory duodenal ulcers, CBD stones, and acute cholangitis or pancreatitis, are suggestive of an ectopic opening.[41]

Expulsion of stones into the duodenum

There is a small bend where the distal portion of the CBD enters the wall of the duodenum. This bend is a blind spot. Moreover, the small size of stones, deep positions, and interference by intestinal gas make it more difficult to detect stones at this bend by B-ultrasound or CT. The small stones at this bend can be flushed into the duodenum by bile due to relaxation of the SO under anesthesia and changes in body position. They can also be expelled into the duodenum if the CBD is explored with a choledochoscope, probe, or catheter. This condition occurred in 12 patients in our study.

Duodenal diseases

Crohn's disease below the duodenal papilla,[42] circular ulcers, various diseases that cause duodenal obstruction (and consequent increased duodenal pressure) can lead to reflux of intestinal juice into the stomach and CBD, thereby leading to CBD dilatation, especially if there is pre-existing reflux of duodenal juice into the bile duct.

Other factors

Biliary hemorrhage, heterotopic pancreas,[43] and sarcoidosis[44] can also cause CBD dilatation.

Treatment

Symptomatic treatment

Hepatoprotective, anti-inflammatory, and choleretic drugs can be used. Procoagulant drugs can be employed for coagulation disorders. Antibiotics can be used for biliary infection.

Etiological treatment

In general, the cause of CBD dilatation can be identified sooner or later for all patients. Etiological treatment should be considered wherever possible. Commonly used etiological treatments for CBD dilatation are summarized below.

Lesionectomy: Hemangioma, biloma, pancreatic cancer, pancreatic cysts, cancer metastasis, and lymphadenopathy can be removed surgically to relieve the pressure on the bile duct and allow unobstructed bile drainage. Cholecystectomy should also be undertaken for concurrent gallstones.

Adhesiolysis: CBD dilatation due to relative stenosis caused by compression of the anterior bile-duct wall by chronic pancreatitis (type-II bile-duct stenosis) and that due to adhesion can be treated by adhesiolysis.

Internal drainage: For patients with type-I bile-duct stenosis caused by chronic pancreatitis, biliary-enteric drainage should be undertaken due to the long stenosis. Roux-*en*-Y biliary-enteric anastomosis is first-line treatment for this condition.

Sphincterotomy should not be undertaken because it often fails due to the stenosis exceeding 30 mm.[45] Internal drainage can be used for stenosing papillitis. Strained SOD should be treated first with antispasmodic agents and neuromodulators. If symptoms persist, sphincterotomy or internal drainage can be undertaken.[46] In the present study, two patients underwent internal drainage and achieved a satisfactory outcome. Roux-*en*-Y biliary-enteric anastomosis can be carried out for relaxed SOD to reduce intestinal fluid reflux using the length of the intestinal loop. Bile-duct injury should be treated with bile-enteric drainage. Patients with gallstones passed into the bowel or parapapillary diverticulitis can be treated with antibiotics or traditional Chinese medicine formulations.

Endoscopic treatment: Endoscopic sphincterotomy is first-line treatment for strained SOD and stenosing papillitis.[45] Dilatation using an endoscopic balloon is first-line treatment for stenosis due to ectopic opening of the CBD. Biliary-enteric drainage can be undertaken if necessary. PSC can be treated first with endoscopic stents. If the outcome is poor, biliary-enteric drainage can be considered. Currently, the only curative treatment for PSC is liver transplantation.[47,48]

Other treatments: Interventional therapy or implantation of radioactive seeds can be used for advanced cancer that cannot be treated by resection.[49,50]

Authors' Contributions

Authors YFL, JFZ, JCW and ZSZ made equal contributions to this work. All the authors participated in the search and sorting of clinical data, referencing references and main papers. Specifically, YFL is mainly responsible for project design and manuscript writing. JFZ and JCW are mainly responsible for organizing clinical data and consulting related references. ZSZ was mainly responsible for table making and data statistics.

Declaration of Conflicting Interests

All authors declared no potential conflicts of interest with repect to the research, authorship,and/or publication of this article.

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