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Etiological Classification of Bile Duct Dilatation and Proportion of Each Cause in 1430 Patients

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***Abstract*— Objective: To investigate the etiology of bile duct dilatation and proportion of each cause. Methods: A retrospective study was performed by classification on clinical data collected from 1430 patients with bile duct dilatation in our hospital from January 2000 to December 2013. Results: There were 90 patients with congenital bile duct cysts (6.3%) and 1340 with secondary bile duct dilatation (93.7%). In total, 22 categories of causes were identified. Among them, the top five were bile duct stones (n=476, 33.3%), pancreatic head carcinoma (n=179, 12.5%), periampullary carcinoma (n=159, 11.9%), cholangiocarcinoma (n=89, 6.2%), and chronic pancreatitis or pancreatic head cyst (n=80, 5.6%). Conclusion: The causes of bile duct dilatation can be classified into congenital and secondary categories. The former accounted for approximately 6.0% and the latter for approximately 94%. The most common causes were bile duct stones, pancreatic head carcinoma and periampullary carcinoma.**

***Index Terms*— Bile Duct Dilatation; Etiology; Classification; Proportion.**

I. Introduction

Normal diameter of common bile duct is <8 mm. A common bile duct diameter ≥8 mm at its widest point is called common bile duct dilatation. Normally, the diameter of right hepatic duct is 3.5 mm and that of left hepatic duct is 3.3 mm. The condition with a diameter of left or right hepatic duct exceeding the normal range is called intrahepatic bile duct dilatation [1]. Generally, but not absolutely, extrahepatic bile duct dilatation occurs before intrahepatic bile duct dilatation. Hilar or intrahepatic lesions can cause intrahepatic bile duct dilatation without extrahepatic bile duct dilatation.

Bile duct dilatation is common in clinical practice. Many patients come to the hospital because of bile duct dilatation. Some causes are obvious and some are subtle. In order to fully understand and investigate the causes of bile duct dilatation and proportion of each cause, develop a comprehensive and accurate diagnosis strategy, and identify effective treatment options in a timely manner, clinical data collected from 1430 patients with bile duct dilatation in our hospital from January 2000 to December 2013 were analyzed by classification. The results are summarized as follows.

II. Clinical Data

The 1430 patients enrolled included 876 males and 554 females, with a male to female ratio of 1.6: 1, and were aged 14-85 years, with a mean age of 56 years. All the patients underwent B ultrasound, CT or MRI, 233 (16.3%) underwent endoscopic retrograde cholangiopancreatography (ERCP), and 80 (5.6%) underwent upper gastrointestinal barium meal examination and endoscopy. Bile duct dilatation was found in 1315 patients (92.0%) in preoperative diagnosis and in 115 patients (8.0%) during intraoperative exploration. All patients underwent surgery. Surgical procedures were selected according to the specific cause. For example, patients with stones underwent choledocholithotomy and T-tube drainage; those with cholangiocarcinoma underwent resection of cholangiocarcinoma; and those with pancreatic head carcinoma underwent pancreaticoduodenectomy.

III. Results

The causes of bile duct dilatation and proportion of each cause in 1430 patients are shown in Table 1. 90 patients (6.3%) had congenital bile duct cysts, with the maximum cyst size of 100 mm × 150 mm and the minimum size of 10 mm × 15 mm. 1340 patients (93.7%) had secondary bile duct dilatation, of which 374 patients (27.90%) had a common bile duct diameter of 8 mm-12 mm, 478 (35.6%) of 12 mm - 16 mm, 240 (17.9%) of 16 mm - 20 mm, and 248 (18.5%) of >20 mm. Degree of bile duct dilatation caused by different causes is shown in Table 2. The relationship between the degree of secondary common bile duct dilatation from the top five causes and the serum total bilirubin is shown in Table 3.

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Table 1 Causes of bile duct dilatation in 1430 patients

Cause	Case number	%	Cause	Case number	%
Congenital bile duct cysts	90	6.3	Oddi sphincter relaxation	6	0.4
Choledocholithiasis	476	33.3	Bilioenteric anastomotic stricture	15	1.0
After cholecystectomy	51	3.6	Chronic cholecystitis with impacted stones in the neck of gallbladder	28	2.0
Biliary parasites	51	3.6	Gallbladder carcinoma	30	2.1
Cholangitis	53	3.7	Periampullary carcinoma	159	11.1
Bile duct stricture	14	1.0	Pancreatic head carcinoma	179	12.5
Extrahepatic cholangiocarcinoma	89	6.2	Hilar metastases	17	1.2
Mirizzi Syndrome	10	0.7	Chronic pancreatitis or pancreatic head cysts	80	5.6
Hemobilia	10	0.7	Duodenal bulb ulcers	16	1.1
Constrictive papillitis	9	0.6	Parapapillary diverticulitis	4	0.3
Duodenal papillary carcinoma	40	2.8	Duodenal obstruction	3	0.2

Table 2 Causes and degree (mm) of secondary bile duct dilatation in 1340 patients

		8.0-12.0		12.0-16.0		16.0-20.0		>20.0	
Cause	Case number	N	%	N	%	N	%	N	%
Choledocholithiasis	476	91	19.1	201	42.2	145	30.5	39	8.2
After cholecystectomy	51	41	80.4	10	19.6	0		0	
Biliary parasites	51	43	84.3	8	15.7	0		0	
Cholangitis	53	8	15.1	23	43.4	13	24.5	9	17.0
Extrahepatic cholangiocarcinoma	89	29	32.6	18	20.2	17	19.1	25	28.1
Mirizzi Syndrome	10	1	10.0	5	50.0	3	30.0	1	10.0
Hemobilia	10	4	40.0	5	50.0	1	10.0	0	
Constrictive papillitis	9	3	33.3	4	44.5	2	22.2	0	
Duodenal papillary carcinoma	40	4	10.0	7	17.5	23	57.5	6	15.0
Oddi sphincter relaxation	6	3	50.0	2	33.3	1	16.7	0	
Bilioenteric anastomotic stricture	15	4	26.7	7	46.6	4	26.7	0	
Chronic cholecystitis	28	20	71.4	7	25.0	1	3.6	0	
Gallbladder carcinoma	30	10	33.3	12	40.0	5	16.7	3	10.0
Periampullary carcinoma	159	17	10.7	66	41.5	59	37.1	17	10.7
Pancreatic head carcinoma	179	42	23.5	69	38.5	41	22.9	27	15.1
Hilar metastasis	17	1	5.9	3	17.6	8	47.1	5	29.4

Chronic pancreatitis	80	40	50.0	20	25.0	14	17.5	6	7.5
Duodenal obstruction	3	2	66.7	1	33.3	0		0	
Parapapillary diverticulitis	4	3	75.0	1	25.0	0		0	
Duodenal bulb ulcer	16	10	62.5	5	31.3	1	6.2	0	
Bile duct stricture	14	4	28.6	5	35.7	3	21.4	2	14.3
Total	1340	380	28.4	479	35.7	341	25.4	140	10.5

Table 3 The relationship between the degree of common bile duct dilatation from the top five causes and the serum total bilirubin (μmol/L)

	Bile duct dilatation	<30		30.1-68.3		68.4-171		>171	
Cause	(mm)	N	%	N	%	N	%	N	%
Choledocholithiasis	8.0-12.0	12	2.5	20	4.2	20	4.1	40	8.4
	12.1-16.0	72	15.1	86	18.1	23	4.9	21	4.4
(n=476)	16.1-20.0	7	1.5	7	1.5	13	2.7	13	2.7
	>20.0	14	2.9	44	9.2	44	9.2	40	8.4
	Subtotal	105	22.1	157	33.0	100	21.0	114	23.9
Pancreatic	8.0-12.0	13	7.3 13	4	2.2	12	6.7	13	7.3
head	12.1-16.0	7.3 19	10.6	7	3.9	16	8.9	32	17.9
carcinoma	16.1-20.0	13	7.3	3	1.7	12	6.7	7	3.9
(n=179)	>20.0	58	32.4	3	1.7	8	4.5	4	2.2
	Subtotal			17	9.5	48	26.8	56	31.3
Periampullary carcinoma	8.0-12.0	8	5.0	0		0		9	4.9
	12.1-16.0	2	1.3	9	5.7	23	14.5	33	20.5
(n=159)	16.1-20.0	8	5.0	0		4	2.5	46	29.5
	>20.0	0		0		7	4.4	10	6.5
	Subtotal	18	11.3	9	5.7	34	21.4	98	61.6
Extrahepatic	8.0-12.0	12	13.50	0		5	5.6	12	13.5
cholangiocarcinoma	12.1-16.0	0		4	4.5	9	10.1	5	5.6
	16.1-20.0	0		2	2.2	1	1.1	15	16.9
(n=89)	>20.0	0		6	6.7	3	3.4	15	16.9
	Subtotal	12	13.5	12	13.5	18	20.2	47	52.8
Chronic	8.0-12.0	36	45.0	4	5.0	0		0	
pancreatitis	12.1-16.0	10	12.5	4	5.0	5	6.3	0	
(n=80)	16.1-20.0	10	12.5	2	2.5	2	2.5	0	
	>20.0	0		2	2.5	5	6.3	0	
	Subtotal	56	70.0	12	15.0	12	15.0	0	

IV. Discussion

Bile duct dilatation is not only a clinical symptom of many diseases, but also a sign. There are many conditions that can cause bile duct dilatation and they can be divided into congenital and secondary categories. Congenital biliary dilatation (cysts), which is congenital bile duct abnormalities [2], accounted for about 6% of the causes. It was first reported by German Abrahan veter in 1713, then supplemented and improved by F.Alonso-lej, Caroli, Longmire and other researchers, and finally classified into five types [3]. This classification is still regarded as a classic and the treatment is also very mature [4]. Secondary bile duct dilatation, also known as postnatal or acquired bile duct dilatation, is very common in clinical practice, accounting for 94% of the causes. It is secondary to some diseases or acquired factors.

An intrahepatic bile duct diameter of 5 mm is called mild dilatation of intrahepatic bile duct, 5-9 mm called moderate dilatation, and >9 mm called severe dilatation. A common bile duct diameter of 8-12 mm is called mild dilatation, accounting for approximately 28.4%, 12-16 mm called moderate dilatation, accounting for approximately 35.7%, 16-20 mm called severe dilatation, accounting for approximately 25.4%, and >20 mm called extremely severe dilatation, accounting for approximately 10.5% [1].

Mild to moderate biliary dilatation is commonly found in post-cholecystectomy patients, and patients with biliary parasites, chronic cholecystitis with impacted cervical stones, Oddi sphincter relaxation [5], traction and compression caused by duodenal bulb ulcers, pancreatic head carcinoma, chronic pancreatitis or pancreatic head cysts, and general common bile duct stones. Serious to extremely serious dilatation is mostly seen in patients with sand-like stones, cone-shaped stones or impacted stones in the lower segment of common bile duct, traumatic biliary stricture, and carcinoma, including cholangiocarcinoma, hilar metastasis, periampullary carcinoma, and pancreatic head carcinoma. The top five causes of secondary bile duct dilatation were bile duct stones (n=476, 33.3%), pancreatic head carcinoma (n=179, 12.5%), periampullary carcinoma (n=159, 11.9%), cholangiocarcinoma (n=89, 6.2%), and chronic pancreatitis or pancreatic head cyst (n=80, 5.6%).

There is a certain relationship between the top five causes and serum bilirubin. Generally, in bile duct dilatation caused by carcinoma, the more severe the bile duct dilatation, the higher the level of serum bilirubin. The absence of jaundice in bile duct dilatation caused by carcinoma is not only associated with early detection of lesions and incomplete obstruction of bile duct, but also associated with whether the carcinoma directly originates from the bile duct wall or just compresses the bile duct wall. For example, since cholangiocarcinoma directly originates from the bile duct wall, it tends to progress and cause bile duct stricture, and over 86% of patients with cholangiocarcinoma had jaundice [6], with a serum total bilirubin level of >30 μmol/L; pancreatic head carcinoma compress the bile duct to exert an indirect effect in most cases, and will not cause serious bile duct stricture if the contralateral bile duct is elastic, so that 1/3 of patients with pancreatic head carcinoma do no have jaundice. In addition, in some cases, although carcinoma is detected late, the flow of bile is not affected since necrosis has occurred at the center of carcinoma to form a tunnel, so that the patient may not have jaundice.

Bile duct dilatation caused by choledocholithiasis is complex. As shown in Figure 1, 22.1% of patients with common bile duct stones had almost no jaundice (serum total bilirubin< 30 μmol/L) in clinical practice. Of them, 14 patients had a common bile duct diameter of >20 mm, accounting for approximately 10% of all patients with a common bile duct diameter >20 mm (14/142). This indicated that the degree of common bile duct dilatation is not proportional to incidence of jaundice in these patients. Patients with common bile duct dilatation do not necessarily have jaundice. It may be because of the flow of bile through gaps between stones into the duodenum. The bile duct pressure is presumably not high. Bile duct dilatation caused by choledocholithiasis is the gradual expansion bile duct during stone formation [1]. The flow of bile through gaps between stones into the duodenum is affected by many factors, such as the size, shape, number, and location of stones. If there are cone-shaped stones or impacted stones in the terminal segment of common bile duct, the gaps between stones will disappear, and the bile can not flow into the duodenum. In this case, the degree of common bile duct dilatation is proportional to the incidence of jaundice. 70% of patients with bile duct dilatation caused by chronic pancreatitis did not have jaundice; and even in those with jaundice, serum total bilirubin did not exceed 171 μmol/L at most.

In summary, secondary bile duct dilatation can be divided into six types according to bile duct pressure and cause.

From the benign and malignant perspective, it can be divided into bile duct dilatation caused by benign lesions and that caused by malignant lesions. From the functional and organic perspective, it can be divided into bile duct dilatation caused by functional disturbance and that caused by organic lesions.

In addition, secondary bile duct dilatation can be divided into obstructive and non-obstructive types, depending on whether the bile duct is obstructive. The obstructive type includes bile duct dilatation caused by stones, parasites, polyps, tumors, ectopic pancreas or other obstructions; and the non-obstructive type includes bile duct dilatation caused by bile duct compression (e.g., caused by peribiliary aneurysm, acute and chronic pancreatitis, pancreatic pseudocyst, pancreatic head carcinoma, parapapillary diverticulitis, compression from metastasis, ectopic pancreas in duodenum partition, and duodenal postbulbar ulcers), bile duct stricture, constrictive papillitis, bilioenteric anastomotic stricture, and pancreatic and bile duct junction injuries [14].

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