Etiology and surgical treatment of hilar bile duct stricture

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OBJECTIVE: To improve the surgical effects of hilar duct stricture.

METHODS: The clinical data of 76 patients with hilar bile duct stricture treated at our hospital from 1990 to 2000 were analyzed. The diagnosis was determined by triad signs of cholangitis, increase of ALP and γ-GGT levels, dilation of intrahepatic and extrahepatic bile ducts confirmed by ultrasonography (US), computed tomography (CT), percutaneous transhepatic cholangiography (PTC) or endoscopic retrograde cholangiopancreatography (ERCP). The location of stricture was divided according to the Bismuth classification standard.

RESULTS: Among the 76 patients, 46 (60.5%) suffered from injurious stricture, including 13% of Bismuth type Ⅰ, 39% of type Ⅱ, 19.4% of type Ⅲ, and 28.2% of type Ⅳ. Inflammatory stricture was found in 28 patients, locating in the left hepatic duct (LHD) 46.4% (13/28), the right hepatic duct (RHD) 35.7% (10/28), and the common hepatic duct (CHD) 17.9% (5/28), respectively. The percentages of patients with stricture due to Mirizzi's syndrome, bile duct cyst, and sclerosing cholangitis were 9.2%, 3.9% and 2.6%, respectively. Bile duct repair procedures included biliary reconstruction with pedicled umbilical vein graft for 9.2% of the patients, and proximal cholangiojejunostomy combined with LHD and RHD plasticity for 92.2%. Seventy of the 76 patients were followed up for 2–10 years, and the excellent outcome rate was 94.7%.

CONCLUSIONS: Injurious stricture is the major type of hilar bile duct stricture. Inflammatory stricture is mainly composed of RHD. Hilar bile duct stricture should be treated surgically according to various etiological features and technical principles of biliary repair.

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Key words: stricture; bile duct; operation; repair

Introduction

Hilar bile ducts stricture remains a major challenge to hepatobiliary surgeons. Hilar bile duct stricture include benign and malignant lesions; but malignant lesions mainly comprises of hilar cholangiocarcinoma and metastasis from gallbladder cancer. Benign lesions are common cause of biliary stricture, including chronic fibrostic or erosive inflammation, sclerosing cholangitis and granulomas, inflammatory strictures secondary to bile duct injuries, which took place in cholecystectomy, common bile duct exploration, abdominal trauma, cholangiojejunostomy and other operations. 49.1% of strictures occur in the upper 1/3 of the extrahepatic bile duct,[1-3] and many factors affect its surgical outcome, such as etiology, sites of stricture, times of biliary repair, reconstructive procedures,

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Table 1. Symptoms of patients with hilar bile duct strictures

	Fever with chills+jaundice+ abdominal pain	Abdominal pain+ jaundice	Jaundice+fever with chills	Abdominal pain	Jaundice	Fever with chills
n	36	19	5	5	3	2
%	47.4	25.0	6.6	6.6	3.9	2.6

Table 2. Physical signs of patients with hilar bile duct strictures

	Tenderness+ jaundice	Jaundice	Tenderness	Symptom-free
n	24	21	13	18
%	31.6	27.6	17.1	23.7

and stent-time. We, therefore, analyzed the clinical data of 76 patients with hilar bile duct stricture treated at our hospital from 1990 to 2000 retrospectively, attempting to improve the surgical results and reduce the rate of reoperation.[4]

Methods

Patients

A total of 76 patients with hilar bile duct stricture were admitted to Southwest Hospital, Chongqing, China during a 10-year period from 1990 to 2000. Of these 76 patients, 34 were men and 42 women aged from 22 to 73 years. The age of 70% male patients and 78.5% female patients ranged from 30 to 60 years. Clinical symptoms and physical signs of patients with hilar bile duct stricture are shown in Tables 1 and 2.

Laboratory examinations

The levels of alkaline phosphatease (ALP) and gamma glutamyl transferase (γ-GGT) increased significantly. The level of ALP increased 2 to 6 times in 94.3% patients (72/76), up to 1440 IU/L, and that of γ-GGT increased 2 to 12 times up to above 200 IU/L. Normal levels of ALP and γ-GGT were observed only in 7.2% and 4.1% of the patients. The total bilirubin (TB) level increased 2 to 10 times, up to 520 μmol/L in 68.4% of the patients, 35.5% patients presented with hypo-albuminemia. Because of hypovolemia due to jaundice and cholangitis, these blood parameters were higher than practical levels.

Imaging findings

Dilation of the intrahepatic and extrahepatic bile ducts above stricture sites was confirmed by ultrasonography (US), computed tomography (CT), and imaging changes of calculus, bile duct cyst, and free gas in the bile duct.

Surgical histories

Sixty-nine patients (90.8%) had undergone 1 to 4 operations previously: one in 51 patients (67.0%), 2 in 12 (15.8%), 3 in 5 (6.6%) and 4 in 1 (1.3%).

Methods of diagnosis

The diagnosis of hilar bile duct stricture was dependent on the symptoms of triad of acute cholangitis combined with increased levels of ALP, γ-GGT, TB as well as imaging findings of dilation of the intrahepatic bile duct revealed by US and CT, and eventually confirmed by percutaneous transhepatic cholangiography (PTC) or endoscopic retrograde cholangiopancreatography (ERCP) with interruption of contrast medium. The etiological factors and sites of stricture were ascertained by medical history and surgical exploration.

Statistical analysis

The data were analysed by χ^2 test. A P value of less than 0.05 was considered statistically significant.

Results

Etiology

Injurious stricture

In this group, 46 patients (60.5%) had injurious biliary stricture, one of the most predominant causes of the disease. Of these patients, 44 (94.7%) were due to cholecystectomy and biliary exploration, 1 patient (2.2%) with left hepatic duct

(LHD) stricture was attributable to right hepatectomy, and 1 had abdominal trauma and bile duct injury. According to Bismuth classification, 6 patients (13%) belonged to Bismuth type Ⅰ, 18 (39%) to type Ⅱ, 8 (17.4%) to type Ⅲ, and 13 (28.3%) to type Ⅳ.

Inflammatory biliary stricture

Eighteen patients (23.7%) had imflammatory biliary stricture secondary to hepatolithiasis and pyogenic cholangitis: 7 patients (38.9%) with LHD and right hepatic duct (RHD) stricture, 3 (16.7%) with LHD stricture, 3 with common hepatic duct stricture, 2 with LHD and common hepatic duct (CHD) stricture, 2 with RHD plus distal common bile duct (DCD) and right-posterior hepatic duct (RHDP) stricture respectively, and 1 with LHD, RHD and biliary stricture of the caudate lobe. As to sites of biliary stricture, the ratio of LHD:RHD:CHD was 13:10:5. The incidence of LHD stricture was not significantly different from that of RHD stricture ($\chi^2 = 0.39$, $P > 0.05$), but it was significantly higher than that of CHD stricture ($\chi^2 = 1$, $P < 0.05$).

Biliary stricture from Mirizzi's syndrome

Seven patients with biliary stricture (9.2%) suffered from Mirizzi's syndrome, all belonged to Bismuth types Ⅰ and Ⅱ.

Biliary stricture caused by bile duct cysts

Three patients with biliary stricture (3.9%) suffered from membranous stenosis at the junction of cysts and the hilar hepatic duct, or after intact cystojejunostomy, resulting in stoma stricture.

Biliary stricture caused by sclerosing cholangitis

In 2 patients (2.6%), one had biliary stricture secondary to choledochoduodenostomy, and the other had the disease secondary to hepatic hemangioma after sclerosing therapy.

Surgical repair

Biliary reconstruction with pedicled umbilical vein graft was performed in 7 patients (9.2%). It was only suitable for those patients (Bismuth type Ⅰ) with Mirizzi's syndrome.

Proximal Roux-en-Y cholangiojejunostomy combined with LHD and/or RHD plasticity was performed in 55 patients (72.4%). The posterior anastomotic wall was shaped with connection of incised LHD, RHD and CHD, while the anterior wall was covered with free jejunal loop. A T-tube could be inserted to drain bile when necessary.

Incision of the CHD after Roux-en-Y side-to-side cholangiojejunostomy was made in 2 patients (15.8%).

Other surgical procedures performed simultaneously included hepatic quadrate lobectomy (1 patient), right hepatectomy (2), left-outer segmentectomy (1), stenting with U-type tube (5), and gastrojejunostomy (1), respectively.

Caval-mesentery vein shunt and subsequent two-stage biliary reconstructive procedures were performed in 1 patient (1.3%). T-tube stent after incision of the biliary tract was used in 1 patient (1.3%).

Follow-up

Seventy (92%) of 76 patients were followed up for 2–10 years. Seventy-two patients (94.7%) had satisfactory outcomes without symptoms of cholangitis and they lived a normal life. Only 4 patients had recurrent symptoms. One patient suffered from proximal bile duct injury and unknown right hepatic artery injury. After the middle hepatic artery was resected for exposure of the left hepatic duct, ischemia and liver abscess occurred, and biliary stricture was diagnosed 2 years later. In one patient who had had repair with pedicled umbilical vein graft, symptoms recurred 9 months after operation due to bile leakage and insufficient time for biliary stenting. In one patient with right hepatic hemangioma who had received injection of absolute ethanol, recurrent symptoms included jaundice, abdominal pain, massive gastric ulcer, enlargement of the gallbladder, dilation of the intrahepatic and extrahepatic bile ducts, stricture and restricture of the LHD and RHD 9 months after operation. Another patient had recurrence of symptoms because of bile reflux and consistent membra-

nous stricture of the hilar bile duct.

Discussion

Changes of causes for hilar bile duct stricture

In our study, injurious stricture was the major cause (60%, 46/76) for benign biliary stricture, and 94.7% cases were resulted from cholecystectomy and/or bile duct exploration. Comparison of the data on etiological factors for hilar biliary stricture treated at Southwest Hospital during 1975 – 1990 showed that bile duct injuries (second cause) accounted for 60% of the causes of hilar biliary stricture ($\chi^2=10.85$, $P<0.01$). With the increas-ed number of laparoscopic cholecystectomies performed, the occurrence of bile duct injuries increased 2–3 times. In Japan, the injury rate of the bile duct due to LC was 1.7% in 1999; however, the injury incidence of open cholecystectomy was only 0.61% in 1999.[5]

In contrast, the incidence of inflammatory biliary stricture at the porta hepatis due to hepatolithiasis reduced from 71.7% to 23.1% during the same period.[6] This change was attributable to the decreased incidence of hepatolithiasis and improvement of comprehensive treatment.[7] Following the extensive practice of left hepatectomy for hepatolithiasis, LHD stricture was treated consequently for the benefit of exposure and incision of the RHD. Therefore, the incidence of hilar biliary stricture reduced significantly.

RHD stricture is the prevailing problem of inflammatory biliary stricture. In 18 patients with inflammatory biliary stenosis in this group, the ratio of LHD to RHD and to CHD stricture was 13:10:5. The incidence of biliary stricture at the LHD and RHD was higher than that at the CHD. The scope of exposure showed that the CHD was located lower than the CHD or RHD when stricture occurred. Thus sufficient exposure of strictures could be obtained without the disturbance from hyperplasia of the quadrate lobe of the liver. Furthermore, patients were diagnosed and treated early because of presence of jaundice and physical signs of cholangitis resulted from obstruction of intrahepatic bile flow. Patients with LHD stricture were also cured after left hepatectomy. Compared to the LHD, the diagnosis and treatment of RHD stricture were more difficult. The median length of the RHD was 0.87 cm, and the right anterior or right posterior branches of the RHD were commonly involved in RHD stricture without symptoms for branch stricture, which was ignored intraoperatively.

Atrophy of the right lobe or right posterior lobe took place after long-time RHD stricture, and it was difficult to resect the lesion anatomically. The data from the Biliary Tract Group of the Chinese Society of Surgery revealed that right hepatectomy was made leastly in patients with hepatolithiasis. Our study revealed that the rate of right hepatectomy for patients with hepatolithiasis was 14.6% (94/644) in China, but the number of right hepatectomy was less than that of those who need resection.[8] Surgical treatment of RHD stricture has become a key problem that needs further study.

Surgical management of hilar biliary stricture

Bile duct injury caused strictures

The stump length of the CHD was >2 cm or <2 cm for patients with biliary stricture of Bismuth types Ⅰ and Ⅱ. The bile duct could be exposed clearly. Hilar plate separation to the lower porta hepatis and subtotal or total resection of the quadrate lobe of the liver were needed for biliary stricture of Bismuth types Ⅲ and Ⅳ. In addition to incision and plasticity of the LHD and RHD, the latter procedure was crucial to completion of cholangiojejunostomy,[9] side-to-side Roux-en-Y cholangiojejunostomy with a large stoma was indicated for biliary stricture of Bismuth types Ⅲ and Ⅳ. Some essential surgical techniques should be emphasized such as resection of stricture site, anastomosis to the healthy biliary duct, sufficient large stoma, maintainance of blood flow to the bile duct, no extension of anastomosis, use of 4–0 or 5–0 absorbable sutures, and drainage of all 3-hepatic ducts at the hilar site without missing any opened biliary tree. If it is inconvenient to use absorbable suture, interrupted suture with tying knots outside should be employed.

If bile duct injury is recognized at the time of initial operation, the surgeon should immediately consider his or her experience and ability to repair biliary stricture. End-to-end repair mostly can be finished when a bit of bile duct length is lost. Cholangiojejunostomy is generally used for those patients with bile injuries characterized by bile leakage or bile peritonitis. Calculus could form in the dilated bile duct above the stricture site, and could be taken easily; no hepatic lobectomy is needed for most patients because of minor pathologic alteration of bile trees. However, if stricture is caused by injury to the bile duct and hepatic artery, ischemic stricture always is followed by serious fibrous scar, extensive stenosis, liver abscess, and bile leakage after anastomosis. Hence stenting using an U-type tube for 1 to 1.5 years is extremely important. Relapse of biliary stricture could be avoided with the U-type tube because it is changeable and could be used for a longer period to obviate bile mud obstruction of the biliary tract. Restenosis may cause ischemic stricture easily.

Inflammatory biliary stricture at the porta hepatis

In our group, biliary stricture was mostly located at the left or right bile ducts (13/18, 73%), and stones were all present above the stricture. Classical treatment include combination of excision of the seriously lesioned sides of the liver, while maintaining healthy sides, stone removal under choledoscope, and cholangiojejunostomy. Left hepatectomy was commonly used in the past decades. In recent years, however, the incidence of atrophy of the right liver lobe with hypertrophy of the left lobe has been increasing, resection of the right liver or right posterior lobe seems to be an inevitable trend.

Additionally, intrahepatic stone and its carcinogenesis to the bile duct should be kept in mind because of inflammatory biliary stricture, and initial resection of liver segment with calculus is helpful to reduce the occurrence of cholangiocarcinoma.

Stoma stricture of cholangiojejunostomy after excision of the bile duct cyst

The definitive treatment of bile duct cyst is excision and Roux-en-Y hepaticojejunostomy. Postoperative biliary stricture usually occurs at the porta hepatis. Patients with bile duct cyst together with hepatolithiasis in addition to stoma stricture should be alert to the missed membranaceous or corded stenosis at the junction of cysts and the bifurcation of bilateral hepatic ducts.[10] In 56 patients who had undergone excision of biliary cysts and cholangiojejunostomy,[11] follow-up for 2 years showed occurrence of biliary stone in 5 patients, and calculus formation in 3 patients with distal stenosis. Only 1 of 24 patients with dilation of the intrahepatic duct and with no biliary stenosis had stone formation, and 1 of 29 patients without dilation of the intrahepatic duct and distal stenosis had stone.

The sites of the common hepatic duct severed are also correlated with anastomic stricture after cholangiojejunostomy. Biliary stones were seen in 5 of 18 patients whose common hepatic duct was cut off and anastomosis sites were below the porta hepatis, stone formation was observed in only 1 of 75 patients whose cut off sites were at the porta hepatis. This fact suggests that choosing the porta hepatis as the cut off site for bile duct cyst is reasonable. When insufficiency of blood inflow to the remnant cyst and the latter lacking epithelial tissue are considered, postoperative anastomotic stenosis is inevitable.[11] Fujii et al[12] reported that in 56 of 58 patients with hilar biliary stricture and cystic dilation, 38 patients had intrahepatic stones and 9 patients had cholangiocarcinoma. So it is important to relieve focal membraneous or corded stenosis and treat intrahepatic stones and carcinoma in patients with hilar biliary strictures, especially those with bile duct cysts.

Biliary stricture caused by Mirizzi's syndrome

Mirizzi's syndrome is an uncommon condition. Between the common hepatic duct and cystic duct, there is a parallel segment. The common hepatic duct is obstructed by stones in the neck of the gallbladder or the cystic duct, even cholecyst-

bile duct fistulae formed. It is clinically important to recognize the diagnosis before surgery. Since failure to appreciate the extraluminal obstructing process will result in unrewarding exploration of the common duct and postoperative persistence of obstruction, cholecystectomy is performed initially at the fundus of the gallbladder while retaining the inferior neck of the gallbladder to reconstruct the bile duct and repair the defect, and a T-tube is used to stent the repaired site for 3 months or repair with pedicled umbilical vein graft. Both methods need suturing carefully to avoid bile leak. If there are bile leakage, insufficient blood inflow to the bile duct, inadequate stenting time at the repaired site of the bile duct, the strictures could relapse (9.2%, 7 cases). If there is insufficiency of repair material, cholangiojejunostomy is the choice of operation.

Biliary stricture after duodenocholedochotomy

Duodenocholedochotomy used to be a classical operation for distal obstruction of the bile duct, but with strongly reversed peristalsis. The duodenal intestinal content consisting of various enzymes, especially pancreatic enzymes, could reversely enter into the bile duct. With a long-term inflammatory stimulation, secondary sclerosing cholangitis would be induced eventually with fibrosis formation in the bile duct; it is marked in patients with biliary infection or carcinoma sometimes. Clearly, cholangiojejunostomy is superior to duodenocholedochotomy in long-term outcomes.

Iatrogenic biliary stricture after interventional therapy of hepatic hemangioma

It is not a rare clinical biliary entity that occlusion of the hepatic artery or ectopic embolization of the bile ducts is induced by interventional therapy of hepatic hemangioma. Because the blood inflow of the bile duct is from the hepatic artery, occlusion of the hepatic artery could induce ischemic sclerosing stricture, even a longer length of stricture. This entity needs stenting with an U-type tube for up to 2 years.

Other causes of hilar biliary stricture also include primary sclerosing cholangitis and right-shift of the porta hepatis because of right-liver atrophy/left-liver hypertrophy complex. Scarring sclerosis of the bile duct is most serious at the porta hepatis for primary sclerosing cholangitis, and local resection of hilar slerosis and cholangiojejunostomy with an U-tube stent are effective. As to atrophy-hypertrophy complex, jaundice can be caused by enlargment of the gallbladder, which shifts up forward and its bile emptying is obstructed. Cholecystectomy and relief of biliary obstruction is the choice of treatment, but treatment of atrophic right liver lobe without intrahepatic stones is not necessary. Because hilar stricture may also be due to complications of cholecystectomy, right hepatic duct injury, and deep suturing to the bed of the gallbladder, occurrence of stricture and obstruction of the right hepatic duct are inevitable.

Competing interest

The author or authors do not choose to response to the statements listed in Instructions for Authors.

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