



Risk factors for acute pancreatitis post percutaneous transhepatic biliary stenting in patients with distal malignant obstruction

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Background: Percutaneous transhepatic biliary stenting (PTBS) is an effective treatment for distal malignant biliary obstruction (MBO). Postoperative acute pancreatitis (AP) is a dangerous complication of this procedure. This study sought to investigate the risk factors for AP after PTBS.

Methods: A total of 463 patients who underwent PTBS to treat suspected MBO from October 2012 to October 2021 were enrolled in this retrospective study. Among them, 26 individuals met the diagnostic criteria for postoperative pancreatitis following PTBS. The incidence of AP at 1 month postoperatively was recorded and analyzed. Several risk factors for AP were analyzed, and the odds ratios (ORs) were calculated by univariate and multivariate logistic analyses.

Results: The incidence of AP after PTBS was 10.88% (26/239). The results of the multivariate analyses showed that repeated bile duct hemorrhage (OR =14.370, P=0.0001), intraoperative dilation (OR =7.848, P=0.0003), an operation time >50 min (OR =5.783, P=0.0009), and previous endoscopic intervention (OR =5.468, P=0.0021) were correlated with a high incidence of AP, while sex, age, time to biliary obstruction, body mass index, Eastern Cooperative Oncology Group score, previous anticancer treatments, forceps biopsy, obstruction length, stent size, contrast volume, operators, 125I strand placement, and blood parameters were not significantly correlated with AP (all P>0.05).

Conclusions: A long operation time, intraoperative dilation, repeated bile duct hemorrhage, and previous endoscopic intervention were independent risk factors for AP. These factors should be considered by clinicians in future practice.

Keywords: Malignant biliary obstruction (MBO); acute pancreatitis (AP); biliary stenting; risk factor

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Introduction

Distal malignant biliary obstruction (MBO) is most commonly caused by periampullary carcinomas, such as ampullary carcinoma, cholangial carcinoma, pancreatic carcinoma, and duodenal papillary carcinoma, which are often accompanied by clinical manifestations, such as skin

yellowing, biliary bleeding, and steatorrhea (1). Most distal periampullary tumors lack typical symptoms in the early stage; thus, patients are usually diagnosed with advanced tumors and have a surgical resection rate of less than 20% (2). The palliative treatment of patients with MBO who are not amenable to surgical intervention to reduce patient jaundice

is key to prolonging patient survival. To address biliary stasis, biliary metal stenting is widely used in the palliative treatment of MBO due to its ability to adequately dilate the obstructed bile duct and restore the physiological flow of bile (3). In addition, preoperative biliary stent placement greatly improves the outcome of resectable MBO (4).

Metal biliary stents are implanted using one of two main approaches: endoscopic retrograde cholangiopancreatography (ERCP), and percutaneous transhepatic biliary stenting (PTBS). The former is completed under the joint guidance of endoscopy and fluoroscopy, and has a high technical success rate and low radiation dose, but a long operation time and a high cost. The latter is completed under the guidance of fluoroscopy, and has the same technical success rate as ERCP and a short operation route, but it increases the radiation dose to the operator (5). The surgical access of ERCP is advantageous for distal malignant obstruction. Percutaneous transhepatic cholangial drainage (PTCD) is widely used as the preferred option after ERCP failure. However, studies on the complications associated with PTCD are not as comprehensive as those on the complications associated with ERCP.

Acute pancreatitis (AP) is one of the most dangerous complications following percutaneous transhepatic biliary stenting (PTBS). Yang *et al.* and Sugawara *et al.* reported (6,7) that the incidence of AP after PTBS ranges from 4.6% to 24.2%, but their findings were not identical, and the data are subject to bias due to the small sample sizes of the studies. Severe postoperative AP may lead to peripancreatic fluid accumulation, pancreatic fat necrosis, or multiorgan failure, and it may even be life-threatening. For various reasons, the early activation of pancreatic enzymes during PTBS may lead to the autodigestion of the pancreas and consequently AP. The abdominal pain caused by postoperative pancreatitis after PTBS also needs to be distinguished from that caused by biliary stenting. Thus, the identification and prevention of postoperative pancreatitis by distal MBO PTBS has important clinical significance for the prognosis of patients.

In 2019, the European Society of Gastrointestinal Endoscopy (ESGE) provided a clear definition of post-ERCP pancreatitis (PEP) (8) and listed the definite risk factors and likely risk factors associated with PEP. However, the risk factors associated with PTBS, which provides an alternative biliary stenting route for postoperative pancreatitis, have not been fully evaluated, and several risks are still debated. This study sought to help clinicians to promptly identify post-PTBS pancreatitis to intervene as early as possible to reduce the risk of its occurrence and

to analyze the incidence and risk factors for postoperative AP in patients who underwent PTBS. We present this article in accordance with the STROCSS reporting checklist (available at <https://qims.amegroups.com/article/view/10.21037/qims-24-431/rc>).

Methods

General information

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of the First Affiliated Hospital of Zhengzhou University (No. 2022-KY-217). Informed consent was obtained from all the individual participants included in the study. The clinical data of 463 patients treated with PTBS at Department of Interventional Radiology, The First Affiliated Hospital of Zhengzhou University from October 2012 to October 2021 were collected. Among them, 224 were excluded for the following reasons: (I) internal and external drainage tube placement; (II) incomplete data; (III) combined hyperamylasemia before stenting; (IV) benign biliary obstruction; and (V) a previous surgery to resect tumors history. Ultimately, 239 patients were included in the study (*Figure 1*). The included patients were diagnosed with distal MBO by pathology or imaging. The basic characteristics of the patients are set out in *Table 1*.

Preoperative preparation

Following preoperative fasting for 12 h, abdominal enhancement computed tomography (CT) or magnetic resonance cholangiopancreatography was used to assess the degree of biliary stricture, stricture location, tumor size, and planned puncture route. For patients with a bleeding tendency and low platelet level, the blood volume was replenished, and the platelet volume was increased to at least $50 \times 10^9/L$. The normal serum amylase level was recorded 2 h before surgery to identify increases in serum amylase after surgery.

Procedures

Each patient lay flat on the digital subtraction angiography examination bed (Artis Zeego, Siemens, Germany). Due to individual differences in the location of the liver and biliary tract, a suitable puncture site was selected under

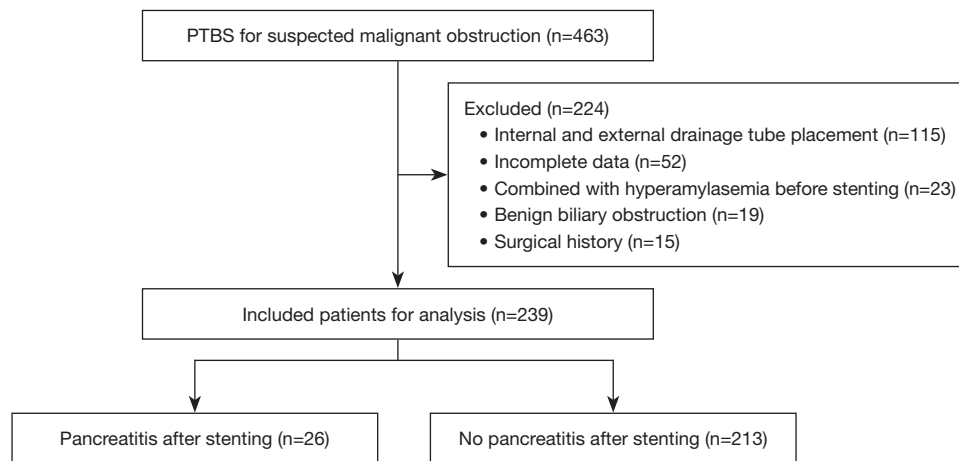


Figure 1 Study workflow for exclusion and inclusion of patients. PTBS, percutaneous transhepatic biliary stenting.

Table 1 Basic characteristics of patients

| Parameters | AP group (n=26) | Non-AP group (n=213) | $t/\chi^2/Z$ value | P value |
|---|---------------------|-----------------------|--------------------|---------|
| Characteristics before biliary stenting | | | | |
| Sex (male/female), No. (%) | 14 (53.8)/12 (46.2) | 119 (55.9)/94 (44.1) | 0.038 | 0.845 |
| Age (years), mean \pm SD | 56.15 \pm 7.69 | 58.68 \pm 9.86 | -1.316 | 0.188 |
| Time to biliary obstruction (days), mean \pm SD | 2.47 \pm 0.92 | 2.46 \pm 0.66 | 0.888 | 0.409 |
| BMI (kg/m ²), mean \pm SD | 26.50 \pm 3.92 | 25.99 \pm 2.89 | 0.812 | 0.527 |
| ECOG score (0–1/2–3), No. (%) | 13 (50.0)/13 (50.0) | 103 (48.4)/110 (51.6) | 0.025 | 0.874 |
| Blood parameters before biliary stenting, mean \pm SD | | | | |
| White blood cell count ($\times 10^{12}/L$) | 6.27 \pm 1.64 | 5.90 \pm 1.46 | 1.196 | 0.233 |
| Platelet ($\times 10^9/L$) | 170.88 \pm 23.87 | 161.92 \pm 26.50 | 1.645 | 0.101 |
| Hemoglobin (g/dL) | 126.65 \pm 16.67 | 122.82 \pm 11.63 | 1.139 | 0.264 |
| Glutamic pyruvic transaminase (U/L) | 90.04 \pm 39.60 | 88.56 \pm 28.44 | 0.184 | 0.855 |
| Total bilirubin ($\mu\text{mol/L}$) | 160.55 \pm 41.29 | 155.03 \pm 34.19 | 0.655 | 0.518 |
| CA19-9 (U/mL) | 552.60 \pm 157.88 | 496.95 \pm 137.93 | 1.082 | 0.192 |

AP, acute pancreatitis; SD, standard deviation; BMI, body mass index; ECOG, Eastern Cooperative Oncology Group; CA19-9, carbohydrate antigen 19-9.

fluoroscopy at the right quarter rib. Local anesthesia (2% lidocaine) was administered to the superior border of the ribs at the proposed puncture site, and the patient was asked to breathe calmly. Under the guidance of ultrasound or fluoroscopy, the appropriate intrahepatic bile duct branches were selected for puncture with a 21G puncture needle (Cook, USA), taking care to avoid the intercostal artery. After the successful extraction of dark green bile,

diluted contrast agent (300 mgI/100 mL, Hengrui Medical Instruments Co., Ltd., China) was injected for low-pressure cholangiography to observe the biliary obstruction and whether the pancreatic duct was inadvertently visualized; the puncture path was adjusted as needed. A 0.018-inch microguide wire was introduced, the puncture needle was withdrawn, and the 6F sheath was advanced along the microguide wire. With the help of a 5-F catheter (Cook,

USA), a 0.035-inch guidewire was manipulated through the biliary obstructed segment to the duodenum. The biliary stent release system was implanted via the above guidewire, and the stent release position was precisely determined by fluoroscopy. The appropriate metal stent (diameter: 8–10, length: 50–60 mm) was selected according to the length of the obstruction, ensuring that the stent completely covered the obstruction site. As all the patients had distal bile duct obstruction, the stent was placed across the papillary muscle and no more than 1 cm distal to the duodenum. For severe biliary obstruction that is difficult to pass with the stent releasing system, a balloon catheter (diameter: 10 mm, Bard Rival) was delivered along the guidewire for pre-dilatation for 2–3 min.

Serum amylase and bilirubin levels were monitored in all patients at 3 and 12 hours postoperatively to assist in the diagnosis of AP and to obtain information about biliary obstruction. If serum amylase was elevated and the patient complained of new abdominal pain or worsening abdominal pain, further investigations were required to ascertain if the patient had AP. If a patient's bilirubin level does not decrease, it can indicate poor biliary drainage. In such instances, a further CT scan was performed to evaluate the biliary stent and a secondary percutaneous intervention was performed if necessary in the specific case.

Diagnostic criteria

Post-PTBS AP was diagnosed if at least two of the following criteria were met (9): (I) serum amylase exceeding 3 times the upper limit of the normal value after PTBS; (II) the acute onset of persistent abdominal pain, or the sudden worsening of abdominal pain; and/or (III) a diagnosis of AP on abdominal imaging.

The patient-related risk factors included sex, age, time to biliary obstruction, body mass index (BMI), Eastern Cooperative Oncology Group (ECOG) score, previous endoscopic intervention, diabetes, biliary stone, obstruction length, repeated bile duct hemorrhage, and routine hematology. The procedure-related risk factors included the procedure time, intraoperative contrast use, intraoperative balloon dilation, concurrent biopsy, stent diameter, operator, recurrent intraoperative biliary bleeding, and placement of ^{125}I seed strands.

Statistical analysis

SPSS 26.0 software (IBM, Chicago, IL, USA) was used for

the statistical analysis. All the data were expressed as the number (mean \pm standard deviation), percentage and range. All the parameters between both groups were compared by an independent samples *t*-test, χ^2 test, or Fisher's exact test. Univariate and multivariate logistic regression analyses were used to identify the independent risk factors associated with post-PTBS AP. A *P* value <0.05 was considered statistically significant.

Results

The success rate of the PTBS technique was 100% in the 239 patients (age: 58.66 ± 9.06 years). Of the 239 patients, 26 (26/239, 10.88%) developed post-PTBS AP, of whom 14 (53.8%) were male and 12 (46.2%) were female. The 239 patients were divided into the AP group ($n=26$) and the non-AP group ($n=213$). The baseline characteristics of the two groups before surgery were compared. The hematological parameters of the patients before biliary stent placement, including the white blood cell count, platelet count, hemoglobin count, glutamic pyruvic transaminase level, total bilirubin level, and carbohydrate antigen 19-9 (CA19-9) level, did not significantly differ between the groups ($P>0.05$) (Table 1). Therefore, the preoperative baseline information did not significantly differ between the AP group and the non-AP group.

A total of 25 risk factors, including 17 patient-related risk factors and eight procedure-related risk factors, were evaluated in the univariate analysis (Table 2). Of the 25 factors, six were found to be significantly associated with an increased risk of AP after PTBS. The patient-related risk factors that were significantly different included prior endoscopic intervention ($P=0.019$), diabetes ($P=0.017$), biliary stones ($P=0.027$), and repeated bile duct hemorrhage ($P<0.001$). The procedure-related risk factors that were found to significantly influence the risk of AP after PTBS included procedure time ($P=0.001$) and intraoperative dilation ($P=0.002$). The remaining 19 factors had no significant effect on increasing the risk of postoperative AP.

A multivariate logistic regression model (Table 3) was developed for the factors screened by the univariate analysis with a $P<0.10$. Six relevant risk factors were included in the regression model, including two procedure-related risk factors and four patient-related risk factors. The following four factors were confirmed to be independently associated with the occurrence of AP after PTBS: intraoperative dilation, total procedure time, repeated bile duct hemorrhage, and previous endoscopic intervention.

Table 2 Univariate analysis of risk factors for post-PTBS pancreatitis

| Parameter | AP group (n=26) | Non-AP group (n=213) | OR (95% CI) | P value |
|---|-----------------|----------------------|----------------------|-----------|
| Patient-related risk factors | | | | |
| Sex (male/female) | 14/12 | 119/94 | 0.923 (0.407–2.086) | 0.845 |
| Age (≤ 60 / >60 years) | 7/19 | 92/121 | 0.485 (0.195–1.201) | 0.118 |
| Time to biliary obstruction (≤ 2.5 / >2.5 weeks) | 13/13 | 120/93 | 1.290 (0.571–2.915) | 0.540 |
| BMI (≤ 25 / >25 kg/m ²) | 7/19 | 81/132 | 1.666 (0.671–4.136) | 0.272 |
| ECOG score (0–1/2–3) | 13/13 | 103/110 | 0.936 (0.415–2.114) | 0.874 |
| Previous endoscopic intervention (yes/no) | 9/17 | 32/181 | 2.994 (1.228–7.300) | 0.019* |
| Diabetes (yes/no) | 5/21 | 12/201 | 3.988 (1.281–12.420) | 0.017* |
| Biliary stone (yes/no) | 8/18 | 29/184 | 2.820 (1.123–7.078) | 0.027* |
| Previous anti-cancer treatments (yes/no) | 6/20 | 59/154 | 0.783 (0.300–2.046) | 0.618 |
| Obstruction length (≤ 2 / >2 cm) | 18/8 | 139/74 | 0.835 (0.347–2.011) | 0.687 |
| Repeated bile duct hemorrhage (yes/no) | 8/18 | 13/200 | 6.838 (2.505–18.664) | 0.0002*** |
| White blood cell count [≤ 6.5 / >6.5] $\times 10^{12}$ /L] | 15/11 | 130/83 | 1.149 (0.503–2.622) | 0.742 |
| Glutamic pyruvic transaminase (≤ 80 / >80 U/L) | 13/13 | 98/115 | 0.852 (0.377–1.924) | 0.700 |
| Total bilirubin (≤ 150 / >150 μ mol/L) | 10/16 | 107/106 | 1.615 (0.701–3.721) | 0.260 |
| CA19-9 (≤ 500 / >500 U/mL) | 10/16 | 107/106 | 1.615 (0.701–3.721) | 0.260 |
| PLT [≤ 160 / >160] $\times 10^9$ /L] | 9/17 | 94/119 | 1.492 (0.636–3.498) | 0.357 |
| HG (≤ 125 / >125 g/L) | 15/11 | 129/84 | 1.126 (0.493–2.570) | 0.778 |
| Procedure-related risk factors | | | | |
| Simultaneous forceps biopsy (yes/no) | 7/19 | 44/169 | 1.415 (0.559–3.579) | 0.463 |
| Contrast volume (≤ 50 / >50 mL) | 21/5 | 171/42 | 0.969 (0.345–2.721) | 0.953 |
| Procedure time (≤ 50 / >50 min) | 13/13 | 171/42 | 4.071 (1.758–9.427) | 0.001** |
| Stent diameter (8 mm/10 mm) | 9/17 | 54/159 | 1.559 (0.656–3.702) | 0.314 |
| Stent length (50/60 mm) | 16/10 | 122/91 | 1.193 (0.518–2.752) | 0.678 |
| Operators (senior/intermediate operators) | 14/12 | 110/103 | 1.092 (0.483–2.472) | 0.832 |
| ¹²⁵ I stands placement simultaneous (yes/no) | 9/17 | 52/161 | 1.639 (0.689–3.898) | 0.264 |
| Intraoperative dilation (yes/no) | 10/16 | 29/184 | 3.966 (1.642–9.578) | 0.002** |

*, **, ***, stand for $P < 0.05$, $P < 0.01$, $P < 0.001$, respectively. PTBS, percutaneous transhepatic biliary stenting; AP, acute pancreatitis; OR, odds ratio; CI, confidence interval; BMI, body mass index; ECOG, Eastern Cooperative Oncology Group; CA19-9, carbohydrate antigen 19-9; PLT, platelet; HG, hemoglobin.

Discussion

Malignant obstruction of the distal biliary tract is often caused by periampullary cancer, of which pancreatic head cancer accounts for 60–70% of cases (10–12). It is predicted that pancreatic cancer will become the second leading cause of cancer death in the United States in the next

20–30 years (13). As distal MBO is most often diagnosed at an advanced stage, patients do not meet the standard for surgical resection. The placement of biliary stents to relieve biliary obstruction is the most effective way to improve patients' skin pruritus and reduce serum bilirubin. In addition, biliary stents combined with radioactive ¹²⁵I seed strand brachytherapy can also achieve therapeutic effects for

Table 3 Multivariate analysis of post-PTBS pancreatitis

| Parameters | OR (95% CI) | P value |
|--|-----------------------|-----------|
| Intraoperative dilation (yes/no) | 7.848 (2.559–24.066) | 0.0003*** |
| Total procedure time (≤ 50 / > 50 min) | 5.783 (2.048–16.328) | 0.0009*** |
| Repeated bile duct hemorrhage (yes/no) | 14.370 (3.624–56.981) | 0.0001*** |
| Previous endoscopic intervention (yes/no) | 5.468 (1.849–16.164) | 0.0021** |
| Diabetes (yes/no) | 2.261 (0.486–10.527) | 0.2985 |
| Biliary stone (yes/no) | 1.428 (0.397–5.136) | 0.5856 |

, *, stand for $P < 0.01$, $P < 0.001$, respectively. PTBS, percutaneous transhepatic biliary stenting; OR, odds ratio; CI, confidence interval.

unresectable cholangiocarcinoma (14–16).

Adverse events, including biliary tract infection, biliary bleeding, biliary fistula, and pancreatitis, are inevitable post-PTBS (17). Previous studies at Department of Interventional Radiology, The First Affiliated Hospital of Zhengzhou University showed that biliary forceps biopsy may also cause postoperative bleeding (18,19); however, intraoperative forceps biopsy is not a risk factor for postoperative pancreatitis in PTBS. A 2019 meta-analysis by Facciorusso *et al.* (20) showed that the application of a new hemostatic powder effectively treated biliary bleeding. As AP due to PTBS is a challenging complication and relatively difficult to treat, it is essential to understand the risk factors associated with AP after PTBS.

AP is the most serious complication after biliary stent implantation. The ESGE has provided a clear definition of and defined the risk factors for AP after ERCP (8). Biliary stent placement via the PTBS route is still widely used; however, research on the complications of AP after PTBS is lacking, especially in relation to the stents used for distal malignant obstruction across the duodenal papilla (21–23). In this study, we retrospectively collected the clinical data of 239 patients with distal malignant obstruction and identified four risk factors associated with postoperative AP after PTBS.

The pathogenesis of AP after PTBS is unclear, and the onset of postoperative AP has been reported to be mainly related to mechanical and chemical injury (24). Guidewire and catheter abrasion of papillary muscles causes edema and spasms of the papillary muscle, which in turn leads to the premature activation of pancreatic duct obstruction by pancreatic protease, causing autodigestion of the pancreas. Injury to the pancreas causes the release of many cytokines, resulting in the activation of local or systemic inflammatory responses, which is also an important cause of single or

multiple organ failure (25).

In this study, the results of the multivariate logistic regression analysis revealed that a long operation time was an independent factor influencing AP after PTBS. This relationship may arise because the contact time between the guidewire and the duodenal papilla increases as the operation time increases. Prolonged friction and the excessive manipulation of the papillary muscle will inevitably increase damage to the papillary muscle and increase the risk of AP after PTBS in patients.

George *et al.* (26) concluded that the concentration and dose of intraoperative contrast agent is also a factor causing pancreatic injury, and that a high concentration and dose of contrast agent can lead to increased pressure in the biliopancreatic duct during PTBS. During the process, the pancreatic duct may be accidentally visualized, and the high concentration of contrast agent entering the pancreatic duct can damage the epithelial cells of the duct and trigger the development of pancreatitis. In this study, contrast agent was injected at doses of ≤ 50 and > 50 mL, but the dose of contrast agent was not an identified as an independent risk factor for postoperative AP in the univariate and multivariate analyses, which may be related to the fact that as much bile as possible was extracted to reduce bile duct pressure before contrast agent injection.

In this study, the multivariate analysis revealed that the patients who had undergone a previous ERCP intervention were more likely to develop AP after PTBS. We believe that the intubation, overdilation, and even the dissection of the duodenal papilla during ERCP cause some degree of damage to the papillary muscle. Postoperatively, it is difficult for the sphincter of Oddi (SOD) to return to the state it had before the ERCP intervention, and in severe cases, this may even lead to the dysfunction of the SOD, which increases the risk of AP (27). He *et al.* (28) reported

that 5.3% of patients developed AP after ERCP, and 18.3% of patients did not develop AP; however, these patients all had varying degrees of postoperative hyperserum amylasemia, which suggests that ERCP causes damage to the pancreas. When these patients underwent PTBS intervention again, the dysfunctional sphincter was more likely to obstruct the pancreatic duct or to cause pancreatic fluid reflux, making the previously damaged pancreas more prone to pancreatitis. Therefore, patients with a history of ERCP should be carefully monitored to detect the occurrence of postoperative pancreatitis after PTBS.

Sugawara *et al.* (7) concluded that intraoperative balloon pre-dilatation of malignant obstruction sites that are difficult to pass by stent implanters is not a risk factor for the development of postoperative pancreatitis. Debate continues as to whether intraoperative balloon dilation is a risk factor for postoperative pancreatitis in PTBS. A few studies have examined this issue. Our study showed that intraoperative balloon dilatation for 2–3 min (diameter = 10 mm) is a risk factor for postoperative pancreatitis after PTBS, from a clinical perspective, this may be because the patients included in this study had long malignant obstructions of the distal biliary tract. Therefore, the stents used were mostly 10 mm × 60 mm, and while the larger size of the balloon provided sufficient space for the stent implantation device to pass through, the simultaneous compression of the balloon on the tumor might have caused some of the tumor tissues to be detached. The dislodged tumor tissues might have become lodged in the SOD and caused pancreatic duct obstruction, which increases the risk of pancreatitis after balloon dilation. Conversely, the present study primarily examined malignant obstruction of the distal bile duct mostly due to pancreatic head carcinoma. Pancreatic head cancer is characterized by hard tumors and a large amount of fibrous tissue. Balloon expansion can pull the pancreatic tissue, tearing the epithelial cells of the pancreatic duct. This tearing in turn leads to the release of cytokines and the early activation of trypsinogen in the pancreatic duct. Therefore, the intraoperative pre-dilatation of the obstruction site may be a risk factor for postoperative pancreatitis after PTBS, and decreasing the balloon diameter to 6–8 mm may decrease the risk of AP in clinical practice.

Recurrent intraoperative biliary bleeding is another concern. One study (29) reported that bacteria can induce the development of pancreatitis. Toxins in bacteria can cause the release of cytokines from immune cells, further inducing pancreatic cell injury. Repeated intraoperative

biliary bleeding in PTBS may be caused by poor or excessive manipulation. Lynn *et al.* (30) suggested that biliary bleeding is one of the important causes of bacterial entry into the biliary tract. In addition, leukocytes in the blood may also mediate the development of a local inflammatory response with the release of cytokines. Intraoperative repeated biliary bleeding forms blood clots, which are not easily fixed at the bleeding site due to the flow of bile. These blood clots flow with the bile to the duodenum abdomen, and as the blood clots are more viscous, they cause poor bile outflow, biliopancreatic duct pressure, and pancreatic fluid retention in the bile duct, after which pancreatitis may occur. At our center, we are of the view that the preoperative assessment of each patient's coagulation level and reasonable intraoperative operation could help to reduce intraoperative bleeding.

This study was subject to some limitations. First, it was a retrospective study and might have some selection bias due to the small sample size. Second, the type of stent placed in the PTBS was not clearly classified, and the reasons for the prolonged operative time need to be further investigated. Finally, all the malignant obstructions were distal, and the biliary stents all crossed the duodenal papillary muscle. Controversy remains as to whether the stent crossing the papillary muscle has an effect on the occurrence of postoperative AP.

Conclusions

Mild-to-moderate AP is a relatively common and serious complication after PTBS. Recurrent biliary bleeding, a history of ERCP intervention, a longer operative time, and intraoperative balloon dilation are independent risk factors for AP after PTBS.

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Footnote

Reporting Checklist: The authors have completed the STROCSS reporting checklist. Available at <https://qims.amegroups.com/article/view/10.21037/qims-24-431/rc>

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at <https://qims.amegroups.com/article/view/10.21037/qims-24-431/coif>).

The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of the First Affiliated Hospital of Zhengzhou University (No. 2022-KY-217). Informed consent was obtained from all the individual participants included in the study.

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