

Modern Radiology and Minimally Invasive Biliary Drainage in Benign Obstructive Jaundice: A Comparative Clinical Study

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ABSTRACT

Background: Benign obstructive jaundice constitutes approximately half of all biliary obstruction cases, carrying substantial morbidity risk from cholangitis, hepatic failure, and sepsis. Optimised diagnostic sequencing and selection among minimally invasive biliary decompression techniques remain active areas of clinical investigation, particularly in settings where multiparametric imaging is being integrated into routine hepatobiliary care.

Objective: To evaluate the diagnostic yield of a stepwise multimodal imaging algorithm and to compare outcomes of a differentiated minimally invasive decompression strategy against conventional open surgical management in patients with benign obstructive jaundice.

Methods: A prospective-retrospective comparative study enrolled 424 patients treated at Samarkand State Medical University (2015–2024). The study group (n=296, 2020–2024) received a differentiated, severity-stratified protocol incorporating Doppler ultrasonography, MRCP, and ERCP/PTBD-guided biliary drainage. The control group (n=128, 2016–2020) underwent standard management with conventional open surgery as the primary modality. Outcomes included postoperative complication rates, 30-day mortality, length of stay, and long-term favourable results.

Results: MRCP detected biliary obstruction with 85–95% sensitivity and >90% specificity; multiparametric imaging reduced diagnostic errors by 20–30%. Hepatic artery Doppler parameters correlated strongly ($r=0.886$, $p<0.001$) with jaundice severity. Minimally invasive procedures yielded favourable long-term outcomes in 88.7% versus 70.0% in conventional surgery. Postoperative complications fell from 26.6% to 19.5% and 30-day mortality from 6.5% to 4.05% ($p<0.05$). Mean hospital stay shortened by five bed-days per patient.

Conclusion: An algorithm-driven, severity-stratified approach centred on minimally invasive biliary decompression substantially reduces complications, mortality, and hospitalisation costs compared with conventional surgery in benign obstructive jaundice. Doppler haemodynamic indices serve as reliable early predictors of hepatic decompensation and should inform the timing of intervention.

Keywords: obstructive jaundice; biliary decompression; MRCP; minimally invasive surgery; Doppler ultrasonography; cholangitis; hepatic haemodynamics

1. INTRODUCTION

Benign obstructive jaundice — defined as biliary outflow obstruction attributable to non-malignant aetiology — represents one of the most demanding presentations in hepatobiliary surgery. The syndrome encompasses choledocholithiasis, benign biliary strictures, Mirizzi syndrome, parasitic involvement, and congenital biliary anomalies, and accounts for roughly half of all obstructive jaundice cases encountered in clinical practice [1–3]. Despite its non-neoplastic nature, the condition carries formidable pathophysiological sequelae: progressive hepatocyte injury from intrahepatic cholestasis, coagulopathy secondary to malabsorption of fat-soluble vitamins, ascending cholangitis, and, at its most severe, multiorgan failure attributable to biliary sepsis.

The hepatic response to biliary obstruction unfolds in a temporally predictable fashion. Early haemodynamic changes — detectable by Doppler ultrasonography before overt biochemical deterioration — include compensatory arterial dilatation and increased portal venous resistance. As jaundice deepens, hepatocellular synthetic function declines, coagulation factors become depleted, and the blood-brain barrier is breached by accumulating bile acids, precipitating hepatic

encephalopathy. At two weeks of complete obstruction, irreversible histological changes begin to consolidate, underscoring the urgency of timely biliary decompression.

Diagnostic sequencing has evolved considerably over the past two decades. Transabdominal ultrasonography remains the first-line investigation, reliably identifying biliary dilatation in 87–89% of cases depending on obstruction level. Where aetiology or anatomy remains uncertain, magnetic resonance cholangiopancreatography (MRCP) has largely displaced diagnostic endoscopic retrograde cholangiopancreatography (ERCP), offering equivalent sensitivity for stone detection without procedural risk. In centres with endoscopic expertise, endoscopic ultrasonography (EUS) provides submillimetre resolution of perampullary anatomy and enables simultaneous tissue sampling [4–6].

Parallel advances in therapeutic endoscopy and interventional radiology have transformed the management landscape. Endoscopic papillosphincterotomy with stone extraction succeeds in 85–90% of choledocholithiasis cases. For proximal or complex strictures inaccessible to the endoscope, percutaneous transhepatic biliary drainage (PTBD) under ultrasound or fluoroscopic guidance restores bile flow with technical success exceeding 90% [7,8]. The accumulated evidence suggests that minimally invasive decompression — whether endoscopic or percutaneous — reduces postoperative morbidity two- to three-fold relative to open biliary surgery, shortens hospitalisation, and achieves long-term patency comparable to surgical bypass in selected patients [9,10].

Nevertheless, critical questions persist. First, the optimal severity-stratified algorithm for selecting among endoscopic sphincterotomy, single-stage PTBD, two-stage decompression followed by definitive repair, and primary open surgery has not been prospectively validated in a large Central Asian cohort. Second, the utility of Doppler haemodynamic indices as early predictors of hepatic decompensation — and hence as objective triggers for expedited intervention — merits systematic evaluation. Third, the long-term outcomes of a fully protocolised minimally invasive approach have not been compared head-to-head against conventional surgical management in this population.

The present study was designed to address these gaps. Our objectives were: (1) to determine the diagnostic performance of a stepwise multimodal imaging algorithm incorporating Doppler ultrasonography, MRCP, ERCP, and PTBD cholangiography; (2) to identify Doppler parameters predictive of early hepatic failure across three severity classes; and (3) to compare postoperative complication rates, 30-day mortality, length of hospital stay, and long-term favourable outcomes between a severity-stratified minimally invasive protocol and conventional management.

2. MATERIALS AND METHODS

2.1 Study design and setting

A comparative prospective–retrospective cohort study was conducted at the Department of Surgery and the Department of Radiology, Samarkand State Medical University (SamSMU) Multiprofile Clinic between January 2015 and December 2024. Ethical approval was obtained from the SamSMU Institutional Scientific Review Board, and the study was conducted in conformity with the Declaration of Helsinki.

2.2 Patient selection

Consecutive adult patients admitted with clinically and biochemically confirmed benign obstructive jaundice (serum total bilirubin above 34 $\mu\text{mol/L}$ with biliary dilatation on imaging and no radiological evidence of malignancy) were eligible for inclusion. Patients with histologically confirmed malignant biliary obstruction, concurrent hepatocellular carcinoma, prior liver transplantation, or unavailable follow-up data were excluded.

The 296 patients admitted between 2020 and 2024 constituted the study group, managed under a prospectively implemented severity-stratified minimally invasive protocol. The 128 patients admitted between 2016 and 2020 constituted the historical control group, treated with conventional management that prioritised open biliary surgery as the primary intervention. This periodisation

minimised contemporaneous contamination between groups while allowing direct outcome comparisons within the same institutional setting.

2.3 Severity classification

All patients were stratified at admission into three severity classes according to the modified Galperin–Gulov classification: Class A (mild; total bilirubin $<60 \mu\text{mol/L}$ or $60\text{--}200 \mu\text{mol/L}$ without cholangitis or secondary complications, $n=183$, 43.1%); Class B (moderate; bilirubin $60\text{--}200 \mu\text{mol/L}$ with cholangitis and/or a secondary complication such as SIRS, coagulopathy, or incipient hepatic failure, $n=157$, 37.0%); and Class C (severe; bilirubin $>200 \mu\text{mol/L}$ with sepsis, hepatic encephalopathy, or single/multiorgan failure, $n=84$, 19.9%). Classification incorporated clinical, biochemical, haemodynamic, and imaging data evaluated at the time of hospital admission.

2.4 Diagnostic protocol

All patients underwent a structured diagnostic algorithm proceeding from non-invasive to invasive modalities according to the 'simple to complex' principle. Transabdominal ultrasonography (US) was the initial investigation in every patient, followed by colour-flow Doppler assessment of the common hepatic artery (CHA) and portal vein (PV) in patients undergoing detailed haemodynamic evaluation ($n=74$, distributed across severity classes: Class A $n=20$, Class B $n=20$, Class C $n=20$, intraoperative $n=14$). Where biliary obstruction level or aetiology remained uncertain after US, MRCP was performed ($n=68$); multislice CT (MSCT) was reserved for cases where pancreatic pathology required three-dimensional assessment. Therapeutic ERCP with papillosphincterotomy was performed in 102 patients in the study group where endoscopic decompression was the planned intervention; percutaneous transhepatic cholangiography (PTCD/PTBD) was used in 11 patients with post-surgical anatomy precluding endoscopic access. Statistical processing employed SPSS v25.0; non-parametric data were analysed with the Mann–Whitney U test (two-group comparisons) and Kruskal–Wallis H test (three or more groups), with $p<0.05$ considered statistically significant. Spearman rank correlation was used to quantify associations between continuous variables.

2.5 Treatment protocol in the study group

In the study group, decompression strategy was individualised by severity class. Class A patients with favourable anatomy underwent primary single-stage minimally invasive decompression (endoscopic or percutaneous), followed by definitive repair under elective conditions where indicated. Class B patients with cholangitis and haemodynamic instability received urgent biliary decompression within 24 hours, with radical intervention deferred until systemic stabilisation. Class C patients — those with overt sepsis, encephalopathy, or organ failure — underwent emergency percutaneous or endoscopic drainage as bridging therapy, followed by staged radical surgery once physiological reserve permitted. Two-stage management (primary decompression then definitive repair) was applied in 116 of 218 patients undergoing minimally invasive procedures (53.3%). Conservative adjuncts — intravenous antibiotics, hepatoprotective agents, fresh frozen plasma, and vitamin K — were employed across all classes as part of perioperative preparation.

3. RESULTS

3.1 Patient demographics and aetiology

Of 424 enrolled patients, 287 (67.8%) were female and 137 (32.2%) were male ($p<0.001$), reflecting the known female predominance in gallstone-related biliary disease. The predominance persisted across both groups: 190 women (64.8%) and 106 men (35.2%) in the study group versus 97 women (75.8%) and 31 men (24.2%) in controls ($p<0.001$ within each group). The working-age cohort (30–59 years) comprised the largest demographic stratum: 220 patients overall, with 144 in the study group and 76 in controls. The duration of jaundice before admission ranged from 1 to 21 days across the entire cohort, without statistically significant intergroup differences ($p>0.05$).

The most prevalent aetiology was cholelithiasis with choledocholithiasis (203 patients, 47.8%), followed by stenosis or papillitis of the major duodenal papilla (77 patients, 18.1%), hepatic echinococcosis with biliary rupture (47 patients, 11.1%), post-inflammatory and iatrogenic biliary strictures (30 patients, 7.0%), biliary pancreatitis (18 patients, 4.2%), Mirizzi syndrome (17 patients, 3.8%), congenital choledochal cyst (10 patients, 2.4%), pseudotumorous pancreatitis (7 patients, 1.6%), and penetrating duodenal ulceration (4 patients, 0.9%). Obstruction was localised to the distal bile duct in 214 patients (50.5%), to the proximal biliary segment in 181 (42.7%), and at intrahepatic level in 29 (6.8%).

3.2 Diagnostic imaging performance

Transabdominal ultrasonography identified biliary dilatation in 89% of distal and 87% of proximal obstruction cases. Among 68 patients undergoing MRCP, biliary obstruction was characterised with 85–95% sensitivity and specificity exceeding 90%. The combined multiparametric algorithm (US + MRCP + laboratory markers) reduced overall diagnostic error by 20–30% relative to single-modality assessment. ERCP in 102 study-group patients demonstrated common bile duct dilatation in 90.1% and intrahepatic ductal dilatation in 87.2%; common bile duct diameter measured 11–20 mm in 59.6% of patients, >20 mm in 26.9%, and <10 mm in 13.5%. Choledocholithiasis was visualised in 82.4% of ERCP examinations, with multiple stones present in 61.5% of affected patients. In 11 patients where ERCP was anatomically precluded, PTCD provided complete cholangiographic mapping and served as both diagnostic and therapeutic access.

3.3 Hepatic Doppler haemodynamics

Hepatic Doppler evaluation was performed in 74 patients (plus 14 intraoperative controls in Class B and C patients). Hepatic artery peak systolic velocity (Vmax) in the CHA exceeded control values by 10.8% in Class A, 51.7% in Class B, and 55.1% in Class C ($p < 0.001$ for Classes B and C versus control). CHA diameter increased by 32% in Classes B and C combined. The pulsatility index in Class C patients was 6.26 ± 0.05 , representing a 5.26-fold elevation above the control value of 1.90 ± 0.02 ($p < 0.001$). Portal venous Vmax declined progressively: values in Classes B and C were 14.5% and 18.0% below Class A, respectively. At bilirubin concentrations above 100 $\mu\text{mol/L}$, portal Vmax was 25% lower than in patients with bilirubin below this threshold. A strong positive correlation was demonstrated between jaundice severity and CHA diameter ($r = 0.886$, $p < 0.001$) and a strong inverse correlation between portal Vmax and CHA Vmax across Classes B and C ($r = -0.70$, $p < 0.001$). These Doppler derangements preceded overt biochemical deterioration, supporting their role as early haemodynamic predictors of hepatic failure.

Table 1 presents detailed Doppler parameters across severity classes. Footnote: HAC, hepatic artery common; * $p < 0.05$ vs. control (Mann–Whitney U test).

Doppler parameter	Control (n=14)	Class A (n=20)	Class B (n=20)	Class C (n=20)	p
HAC Vmax (cm/s)	83.7 ± 1.2	$92.7 \pm 1.8^*$	$127.0 \pm 1.3^*$	$129.6 \pm 1.1^*$	<0.001
HAC Vmin (cm/s)	22.0 ± 0.3	22.1 ± 0.5	$26.6 \pm 0.8^*$	$27.7 \pm 0.5^*$	<0.05
HAC diameter (cm)	0.50 ± 0.002	0.58 ± 0.007	$0.64 \pm 0.001^*$	$0.66 \pm 0.007^*$	<0.05
Resistance index (RI)	0.73 ± 0.004	0.76 ± 0.003	0.77 ± 0.006	0.78 ± 0.003	>0.05
Pulsatility index (PI)	1.90 ± 0.02	$1.23 \pm 0.01^*$	$1.35 \pm 0.05^*$	$6.26 \pm 0.05^*$	<0.001
Portal vein Vmax (cm/s)	16.8 ± 0.9	16.5 ± 0.7	$12.7 \pm 0.5^*$	$11.3 \pm 0.6^*$	<0.001

Table 1. Doppler haemodynamic parameters of the common hepatic artery and portal vein according to obstructive jaundice severity class.

Serum biochemical profiles mirrored the Doppler gradient. Total bilirubin increased from 64.5 $\mu\text{mol/L}$ (Class A) to 220.6 $\mu\text{mol/L}$ (Class B) and 369.9 $\mu\text{mol/L}$ (Class C). ALT rose from 63.9 to 137.5 and 210.0 U/L; alkaline phosphatase from 56.9 to 339.3 and 466.7 U/L. Total protein declined from 67.9 to 59.4 g/L, and the coagulation index increased from 1.00 to 2.10 across the same gradient, consistent with progressive hepatocellular synthetic failure. Medium-molecular-weight toxin index (MSM) increased from 0.494 to 0.709 conv. units. In Class C, erythrocyte antioxidant capacity was 30.4% lower than in Class A controls ($p<0.05$). Table 3 (see below) presents the full biochemical matrix.

3.4 Treatment outcomes

In the study group, minimally invasive procedures were deployed in 218 of 296 patients (73.6%): endoscopic transpapillary decompression alone in 102 (46.7%), percutaneous transhepatic biliary drainage alone in 9 (4.1%), and two-stage combined endoscopic/percutaneous approaches in 116 (53.3%). The remaining study-group patients underwent open surgery either as a primary procedure (complex anatomy, failed endoscopy) or as the definitive second stage following initial decompression.

Postoperative complications occurred in 19.5% of study-group patients versus 26.6% in controls — a 26.7% relative reduction ($p<0.05$). Thirty-day mortality was 4.05% in the study group versus 6.5% in controls ($p<0.05$), representing a 37.7% relative mortality reduction. Favourable long-term outcomes were documented in 88.7% of patients managed by single-stage minimally invasive intervention, 78.0% by two-stage combined approach, and 70.0% by conventional open surgery. Hospital length of stay was reduced by a mean of five bed-days per patient in the study group. The technical success rate of ultrasound- and fluoroscopy-guided biliary drainage exceeded 90%. Risk of hepatic failure development was reduced by 25–40% in the study group relative to controls, with a corresponding shortening of intensive care unit stay.

Table 2 summarises the primary comparative outcome data.

Outcome measure	Study group (n=296)	Control group (n=128)
Overall postoperative complications (%)	19.5%	26.6%
30-day mortality (%)	4.05%	6.5%
Favourable long-term outcome – minimally invasive (%)	88.7%	70.0%
Favourable long-term outcome – two-stage combined (%)	78.0%	—
Mean hospital stay reduction (bed-days)	–5.0	Reference
Technical success of US/fluoroscopy-guided drainage (%)	>90%	—
Risk reduction for liver failure development	25–40% lower	Reference

Table 2. Primary clinical outcomes: study group versus control group.

Liver failure developed in 205 patients overall (48.3%): compensated in 79 (18.6%), subcompensated in 90 (21.2%), and decompensated in 36 (8.5%). Hepatic encephalopathy was documented in 27 patients (6.3%). Within the study group, the differentiated decompression

protocol substantially reduced progression to decompensated failure, as reflected in the lower overall mortality. Intraoperative Doppler assessment in 36 emergency operations confirmed preoperative haemodynamic findings: portal and hepatic artery waveform patterns in Class B and C patients mirrored preoperative recordings, validating their predictive utility.

Table 3 presents the biochemical severity matrix underlying the classification system.

Biochemical parameter	Class A (n=183)	Class B (n=157)	Class C (n=84)
Total bilirubin (μmol/L)	64.5 [48.6–79.2]	220.6 [202.5–250.6]	369.9 [318.0–434.4]
ALT (U/L)	63.9	137.5	210.0
Alkaline phosphatase (U/L)	56.9	339.3	466.7
Total protein (g/L)	67.9	63.0	59.4
Clotting index (INR-equivalent)	1.00	1.60	2.10
Medium-MW toxin index (MSM, conv. units)	0.494	0.631	0.709

Table 3. Serum biochemical parameters by obstructive jaundice severity class on admission. Values expressed as mean or median [IQR] where applicable.

4. DISCUSSION

The results of this study reinforce an emerging consensus that severity-stratified, minimally invasive biliary decompression represents the optimal management framework for benign obstructive jaundice, reducing postoperative complications by more than a quarter and 30-day mortality by over a third relative to conventional surgical approaches. These findings are broadly consistent with multicentre data from European and Asian centres, which report two- to three-fold reductions in complication rates when endoscopic or percutaneous drainage replaces primary open biliary surgery [11,12].

The diagnostic algorithm developed in this study demonstrated MRCP sensitivity of 85–95% for biliary obstruction characterisation — figures aligned with meta-analytic estimates of 90–95% in the recent literature [5,13]. Critically, the multiparametric strategy reduced diagnostic errors by 20–30%, which translated into more precise selection of the decompression modality and location of the intervention. This gain in diagnostic precision carries direct therapeutic consequences: misclassification of obstruction level remains a leading cause of technical failure at ERCP and misplaced PTBD access routes.

The Doppler haemodynamic findings merit specific comment. The progressive escalation of CHA pulsatility index — rising more than fivefold from control values in Class C patients — alongside the inverse correlation between portal flow velocity and bilirubin concentration ($r=-0.70$, $p<0.001$), corroborates experimental and clinical observations that arterial hyperperfusion compensates for falling portal venous inflow as parenchymal resistance increases with cholestatic injury [14,15]. The strong association between CHA diameter and jaundice severity ($r=0.886$) suggests that Doppler vascular parameters capture pathophysiological derangements before they are reflected in serum biochemistry, providing a quantitative trigger for earlier decompression. Implementation of these parameters as decision criteria in Class B patients resulted in timely intervention that contributed to reduced progression to Class C failure.

The two-stage decompression–radical repair strategy, applied in 53.3% of minimally invasive cases, reflects current international guidelines for moderate and severe biliary obstruction [16,17]. By stabilising systemic inflammation and restoring hepatocellular synthetic function before definitive

surgery, this approach ensures that open or laparoscopic reconstruction is performed under significantly more favourable physiological conditions. The 78.0% favourable long-term outcome rate with two-stage combined management — while lower than the 88.7% seen with single-stage minimally invasive procedures — nonetheless exceeds the 70.0% achieved by conventional open surgery, supporting the sequenced approach even for complex cases.

Several limitations warrant acknowledgement. The retrospective nature of the control group introduces the possibility of selection and documentation bias, though the use of a defined historical cohort from the same institution and applying the same severity classification system mitigates the most systematic confounders. The 10-year recruitment window spans significant evolution in endoscopic technology and operator experience; it is therefore possible that some of the observed outcome improvement reflects secular trends in institutional expertise rather than the protocol per se. Long-term follow-up beyond the immediate postoperative period was not uniformly available for all patients, limiting conclusions regarding the durability of favourable outcomes, particularly after single-stage endoscopic stone extraction where recurrence depends on cholecystectomy completion. Finally, intraoperative Doppler data were available only for emergency cases in Classes B and C, precluding a complete preoperative–intraoperative comparison across all severity strata.

5. CONCLUSION

A structured, severity-stratified minimally invasive approach to benign obstructive jaundice — anchored by a multimodal diagnostic algorithm and guided by hepatic Doppler haemodynamics — achieves substantial reductions in postoperative complications (26.6% to 19.5%), 30-day mortality (6.5% to 4.05%), and hospital length of stay compared with conventional open biliary surgery. Doppler parameters of the common hepatic artery and portal vein precede biochemical deterioration and provide objective, reproducible criteria for expedited biliary decompression in Class B and C patients. These findings support the broader adoption of protocol-driven minimally invasive biliary care within hepatobiliary surgical services in Central Asian healthcare settings.

REFERENCES

1. Attasaranya S, Fogel EL, Lehman GA. Choledocholithiasis, ascending cholangitis, and gallstone pancreatitis. *Med Clin North Am.* 2008;92(4):925–960.
2. Kimura Y, Takada T, Kawarada Y, et al. Definitions, pathophysiology, and epidemiology of acute cholangitis and cholecystitis: Tokyo Guidelines. *J Hepatobiliary Pancreat Surg.* 2007;14(1):15–26.
3. Dooley JS, Sherlock S. *Sherlock's Diseases of the Liver and Biliary System.* 13th ed. Oxford: Wiley-Blackwell; 2018.
4. Kaltenthaler E, Vergel YB, Chilcott J, et al. A systematic review and economic evaluation of magnetic resonance cholangiopancreatography compared with diagnostic endoscopic retrograde cholangiopancreatography. *Health Technol Assess.* 2004;8(10):1–89.
5. Romagnuolo J, Bardou M, Rahme E, Joseph L, Reinhold C, Barkun AN. Magnetic resonance cholangiopancreatography: a meta-analysis of test performance in suspected biliary disease. *Ann Intern Med.* 2003;139(7):547–557.
6. Tse F, Liu L, Barkun AN, Armstrong D, Moayyedi P. EUS: a meta-analysis of test performance in suspected choledocholithiasis. *Gastrointest Endosc.* 2008;67(2):235–244.
7. Williams EJ, Taylor S, Fairclough P, et al. Are we meeting the standards set for endoscopy? Results of a large-scale prospective survey of endoscopic retrograde cholangio-pancreatograph practice. *Gut.* 2007;56(6):821–829.
8. Weber A, Gaa J, Rosca B, et al. Complications of percutaneous transhepatic biliary drainage in patients with dilated and nondilated intrahepatic bile ducts. *Eur J Radiol.* 2009;72(3):412–417.

9. Naitoh I, Ohara H, Nakazawa T, et al. Unilateral versus bilateral endoscopic metal stenting for malignant hilar biliary obstruction. *J Gastroenterol Hepatol*. 2009;24(4):552–557.
10. Ivshin VG, Kukushkin AV, Malova MN. Percutaneous transhepatic biliary interventions in patients with malignant obstructive jaundice. *Diagn Interv Radiol*. 2011;17(1):48–52.
11. Karpenko EV, Litvinov IV, Shapovalov AM. Minimally invasive technologies in the treatment of obstructive jaundice. *Sib Med J*. 2017;32(4):115–121.
12. Gulov MK. Classification and staged management of benign obstructive jaundice. *Vestnik Khirurgii*. 2022;181(3):44–51.
13. Aube C, Delorme B, Yzet T, et al. MR cholangiopancreatography versus endoscopic sonography in suspected common bile duct lithiasis: a prospective, comparative study. *AJR Am J Roentgenol*. 2005;184(1):55–62.
14. Lygidakis NJ, van der Heyde MN, Lubbers MJ. Evaluation of preoperative biliary drainage in the surgical management of pancreatic head carcinoma. *Acta Chir Scand*. 1987;153(11–12):665–668.
15. Sharma BC, Agarwal DK, Dhiman RK, Baijal SS, Choudhuri G, Saraswat VA. Bile lithogenicity and gallbladder emptying in patients with microlithiasis: effect of bile acid therapy. *Gastroenterology*. 1998;115(1):124–128.
16. Miura F, Okamoto K, Takada T, et al. Tokyo Guidelines 2018: initial management of acute biliary infection and flowchart for acute cholangitis. *J Hepatobiliary Pancreat Sci*. 2018;25(1):31–40.
17. Dumonceau JM, Tringali A, Papanikolaou IS, et al. Endoscopic biliary stenting: indications, choice of stents, and results (European Society of Gastrointestinal Endoscopy Clinical Guideline — updated 2017). *Endoscopy*. 2018;50(9):910–930.
18. Galperin EI, Vetshev PS. Obstructive Jaundice [in Russian]. Moscow: Vidar-M; 2012.
19. Mamoshkin AV. Antegrade percutaneous transhepatic interventions in complex forms of mechanical jaundice. *Surgery*. 2022;6:44–50.
20. Kononenko SN, Limonchikov SV. Comparative assessment of endoscopic and percutaneous methods of biliary decompression. *Endoscopy*. 2021;23(1):12–18.
21. Rerknimitr R, Angsuwatcharakon P, Ratanachu-ek T, et al. Asia-Pacific consensus recommendations for endoscopic and interventional management of hilar cholangiocarcinoma. *J Gastroenterol Hepatol*. 2013;28(4):593–607.
22. Manes G, Paspatis G, Aabakken L, et al. Endoscopic management of common bile duct stones: European Society of Gastrointestinal Endoscopy (ESGE) guideline. *Endoscopy*. 2019;51(5):472–491.
23. Soreide K, Korner H, Havnen J, Soreide JA. Bile duct cysts in adults. *Br J Surg*. 2004;91(12):1538–1548.
24. Baron TH, Kozarek RA. Endotherapy for organized pancreatic necrosis: perspectives after 20 years. *Clin Gastroenterol Hepatol*. 2012;10(11):1202–1207.
25. Gomi H, Solomkin JS, Schlossberg D, et al. Tokyo Guidelines 2018: antimicrobial therapy for acute cholangitis and cholecystitis. *J Hepatobiliary Pancreat Sci*. 2018;25(1):3–16.
26. Isayama H, Komatsu Y, Tsujino T, et al. A prospective randomised study of covered versus uncovered diamond stents for the management of distal malignant biliary obstruction. *Gut*. 2004;53(5):729–734.
27. Hamada T, Nakai Y, Isayama H, et al. Management of biliary complications after liver transplantation: clinical review. *J Hepatobiliary Pancreat Sci*. 2014;21(4):245–254.
28. Pawlik TM, Choti MA. Surgical therapy for colorectal metastases to the liver. *J Gastrointest Surg*. 2007;11(8):1057–1077.