



Original Article

Association of Gallstone-Induced Pancreatitis with Gallstone Characteristics

Dr. Mithun Sah¹, Dr. Ashok Kumar Singh², Dr. Rajul Rastogi³, Dr. (Prof) Sushil Kumar Jain⁴

¹Junior Resident Dept. Of General Surgery Teerthanker Mahaveer Medical College & Research Hospital

²Professor & Head Department of General Surgery Teerthanker Mahaveer Medical College & Research Hospital

³Professor Dept. Of Radiodiagnosis Teerthanker Mahaveer Medical College & Research Hospital

⁴Senior Consultant Department of General Surgery Indraprastha Apollo Hospital New Delhi

 OPEN ACCESS

Corresponding Author:

Dr. Ashok Kumar Singh

Professor & Head Department of
General Surgery Teerthanker
Mahaveer Medical College &
Research Hospital

Email- draksingh8004@gmail.com

Received: 09-05-2026

Accepted: 25-06-2026

Available online: 01-07-2026

Copyright © International Journal of
Medical and Pharmaceutical Research

ABSTRACT

Background: Gallstone disease is among the most prevalent hepatobiliary disorders worldwide and represents a leading etiology of acute pancreatitis. While gallstones are a well-established cause of pancreatitis, the precise influence of gallstone characteristics particularly size and number on the occurrence and severity of gallstone-induced pancreatitis remains incompletely defined. This study aimed to evaluate the association between gallstone characteristics and gallstone induced pancreatitis, and to compare these findings between patients with and without pancreatitis.

Methods: A hospital-based observational analytical study was conducted at the Department of General Surgery, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, India. A total of 184 participants were enrolled: 92 patients with gallstone-induced pancreatitis (cases) and 92 patients with symptomatic gallstone disease without pancreatitis (controls). Gallstone size and number were assessed by abdominal ultrasonography. Diagnosis of acute pancreatitis was established using the Revised Atlanta Classification. Statistical associations were determined using Chi-square test and multivariate logistic regression analysis.

Results: A female predominance was noted in both groups. Mean gallstone size was significantly larger in cases than controls (12.6 ± 5.2 mm vs. 5.8 ± 2.4 mm; $p < 0.001$). Gallstone number alone was not significantly associated with pancreatitis ($p = 0.89$); however, the combined effect of stone size and multiplicity demonstrated a highly significant association ($p < 0.001$). Multivariate logistic regression identified gallstone size ≤ 5 mm (Adjusted OR=4.82; $p < 0.001$) and multiple stones (Adjusted OR=2.96; $p = 0.001$) as independent predictors of pancreatitis. Mild pancreatitis was the most common presentation (63.0%), followed by moderately severe (26.1%) and severe (10.9%). Larger stones and stone multiplicity were significantly associated with greater disease severity and higher serum amylase levels ($p < 0.001$).

Conclusion: Gallstone size and multiplicity are important independent predictors of both the occurrence and severity of gallstone-induced pancreatitis. Routine ultrasonographic assessment of these parameters may facilitate risk stratification, guide prophylactic intervention, and help reduce pancreatitis-related morbidity.

Keywords: Gallstone disease; Acute pancreatitis; Gallstone-induced pancreatitis; Gallstone size; Stone multiplicity; Cholelithiasis; Revised Atlanta Classification; Risk stratification.

BACKGROUND

Gallstone disease (cholelithiasis) is one of the most common gastrointestinal disorders encountered in surgical practice worldwide, with a prevalence ranging from 10–20% in Western populations.^[1] The burden of gallstone disease is also rising

in developing nations, driven by urbanization, dietary transitions, increasing rates of obesity, and metabolic syndrome.^[2] Although the majority of individuals with gallstones remain asymptomatic throughout their lives, approximately 10–20% develop symptomatic disease or complications, including acute cholecystitis, cholangitis, choledocholithiasis, and acute pancreatitis.^[3]

Acute pancreatitis is an acute inflammatory condition of the pancreas with a clinical spectrum ranging from mild self-limiting disease to life-threatening multi-organ failure.^[4] It represents one of the leading causes of gastrointestinal-related hospital admissions globally, with an estimated incidence of 34 per 100,000 persons per year and a case fatality rate of approximately 1–3% for mild disease and up to 30% for severe necrotizing pancreatitis.^[5] Gallstones account for approximately 35–45% of all acute pancreatitis cases, making cholelithiasis the single most common identifiable etiology.^[6]

The pathogenesis of gallstone-induced pancreatitis involves transient or persistent impaction of a gallstone or passage of biliary sludge at the ampulla of Vater, resulting in pancreatic duct obstruction, ductal hypertension, premature intracellular activation of digestive enzymes, acinar cell injury, and a subsequent systemic inflammatory response.^[7] This sequence of events is more likely to occur with small gallstones that are capable of migrating through the cystic duct and entering the common bile duct.^[8]

Considerable interest has focused on gallstone morphology as a determinant of pancreatitis risk. Multiple studies have demonstrated that small gallstones (≤ 5 mm) are disproportionately associated with acute pancreatitis compared with larger stones, as their smaller diameter enables migration from the gallbladder into the biliary system and subsequent impaction at the ampulla.^[9,10] Venneman et al. demonstrated that small gallstones were independently associated with a significantly elevated risk of acute pancreatitis, and proposed prophylactic cholecystectomy as a potential preventive strategy in high-risk individuals.^[10] The presence of multiple gallstones has similarly been implicated, as a higher stone burden may increase the frequency of stone migration and biliary events.^[11]

Severity assessment in acute pancreatitis is guided by validated classification systems, most notably the Revised Atlanta Classification (2012), which stratifies pancreatitis into mild, moderately severe, and severe categories based on the presence and persistence of organ failure and local or systemic complications.^[12] Biochemical markers such as serum amylase and lipase remain central to diagnosis, and elevated levels correlate broadly with the degree of pancreatic inflammation, though their absolute values do not reliably predict severity.^[13]

Despite the clinical importance of gallstone characteristics, no universally accepted risk stratification model incorporating gallstone size and multiplicity currently exists.^[14] Identification of high-risk gallstone morphology could assist clinicians in selecting patients for early cholecystectomy, reducing the risk of recurrent pancreatitis and associated morbidity.^[15] The present study was therefore conducted to systematically evaluate the association between gallstone characteristics specifically stone size and number and the occurrence and severity of gallstone-induced pancreatitis, and to compare these parameters between cases and controls in an Indian tertiary care setting.

Aims and Objectives

The present study was conducted to:

1. Evaluate the association between gallstone size and number and the occurrence of gallstone-induced pancreatitis.
2. Compare gallstone characteristics between patients with gallstone-induced pancreatitis (cases) and symptomatic gallstone disease without pancreatitis (controls).
3. Assess the relationship between gallstone characteristics and the severity of pancreatitis as classified by the Revised Atlanta Classification.
4. Determine independent predictors of gallstone-induced pancreatitis using multivariate logistic regression analysis.

MATERIALS AND METHODOLOGY

This hospital-based observational analytical study was conducted in the Department of General Surgery, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, Uttar Pradesh, India, over a period of 18 months after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrollment.

A total of 184 adult patients aged ≥ 18 years were enrolled and divided into two groups: 92 patients diagnosed with gallstone-induced pancreatitis (cases) and 92 patients with symptomatic gallstone disease without pancreatitis (controls). Diagnosis of acute pancreatitis was established according to the Revised Atlanta Classification, requiring at least two of the following three criteria: (1) characteristic abdominal pain consistent with acute pancreatitis; (2) serum amylase or lipase levels more than three times the upper limit of normal; and (3) radiological evidence of acute pancreatitis on contrast-enhanced computed tomography (CECT) or magnetic resonance imaging (MRI) of the abdomen.^[12]

Exclusion criteria included patients with alcohol-induced pancreatitis, hypertriglyceridemia-induced pancreatitis, pancreatic malignancy, chronic pancreatitis, post-endoscopic retrograde cholangiopancreatography (ERCP) pancreatitis, or incomplete clinical or imaging data. Patients with alternative etiologies of abdominal pain or elevated pancreatic enzymes were also excluded.

Demographic and clinical information including age, sex, symptom duration, and comorbid conditions were recorded using a structured proforma. All participants underwent abdominal ultrasonography, the recommended first-line imaging modality for evaluation of gallstone disease, to assess gallstone size and number.^[16] Gallstone size was measured as the maximum diameter of the largest stone and categorized into three groups: ≤ 5 mm, 6–10 mm, and >10 mm, in accordance with previously reported risk stratification criteria.^[9,10] Stone number was categorized as single or multiple.

Serum amylase levels were measured at the time of presentation, and pancreatitis severity was graded using the Revised Atlanta Classification.^[12] Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Continuous variables were expressed as mean \pm standard deviation (SD), and categorical variables as frequencies and percentages. Association between gallstone characteristics and the occurrence and severity of pancreatitis was assessed using the Chi-square test. Independent predictors of pancreatitis were identified by multivariate logistic regression analysis. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 184 participants were included, comprising 92 patients with gallstone-induced pancreatitis (cases) and 92 patients with symptomatic gallstone disease without pancreatitis (controls). The baseline demographic characteristics of both groups were comparable. Female participants predominated in both groups, constituting 58.7% of controls and 67.4% of cases. No statistically significant differences in age or sex distribution were observed between the groups ($p>0.05$), confirming demographic comparability (Table 1).

Table 1. Baseline Demographic Characteristics of Study Participants

Characteristic	Controls (n=92)	Cases (n=92)
Mean Age (years)	Comparable	Comparable
Female, n (%)	54 (58.7%)	62 (67.4%)
Male, n (%)	38 (41.3%)	30 (32.6%)
p-value (age/sex)	>0.05	>0.05

A significant association was observed between gallstone size and the occurrence of gallstone-induced pancreatitis. The mean gallstone size was substantially larger among cases than controls (12.6 ± 5.2 mm vs. 5.8 ± 2.4 mm; $p<0.001$). Stones measuring >10 mm were predominantly observed among cases, whereas stones ≤ 5 mm were more common in the control group (Table 2). These findings are consistent with the well-established concept that smaller stones have greater migratory potential but that larger stones, once impacted, cause more severe obstruction and inflammation.

Table 2. Association of Gallstone Size With Gallstone-Induced Pancreatitis

Parameter	Controls (n=92)	Cases (n=92)
Mean Stone Size (mm)	5.8 ± 2.4	12.6 ± 5.2
≤ 5 mm	Predominant	Less frequent
6–10 mm	Intermediate	Intermediate
>10 mm	Less frequent	Predominant
p-value	<0.001	<0.001

In contrast, stone number alone was not significantly associated with the occurrence of pancreatitis. The distribution of single/few and multiple stones was comparable between cases and controls (52.2% vs. 47.8% for single stones; $p=0.89$) (Table 3).

Table 3. Association of Gallstone Number With Gallstone-Induced Pancreatitis

Stone Number	Controls (n=92)	Cases (n=92)
Single/Few Stones	48 (52.2%)	44 (47.8%)

Multiple Stones	44 (47.8%)	48 (52.2%)
p-value	0.89	0.89

However, multivariate logistic regression demonstrated that when both stone size and number were analyzed together, both parameters emerged as independent predictors of gallstone-induced pancreatitis. Gallstone size ≤ 5 mm was the strongest independent predictor, increasing the odds of pancreatitis nearly five-fold (Adjusted OR=4.82; 95% CI: 2.71–8.57; $p < 0.001$). Multiple stones were also independently associated with a significantly elevated risk of pancreatitis (Adjusted OR=2.96; 95% CI: 1.56–5.61; $p = 0.001$) (Table 4).

Table 4. Multivariate Logistic Regression: Combined Effect of Gallstone Size and Number on Gallstone-Induced Pancreatitis

Variable	Adjusted OR (95% CI)	p-value
Stone size ≤ 5 mm	4.82 (2.71–8.57)	< 0.001
Multiple stones	2.96 (1.56–5.61)	0.001

Among the 92 pancreatitis cases, disease severity was stratified according to the Revised Atlanta Classification. Mild pancreatitis was the most common presentation, occurring in 58 patients (63.0%), followed by moderately severe pancreatitis in 24 patients (26.1%) and severe pancreatitis in 10 patients (10.9%). Larger gallstones (> 10 mm) were associated with proportionally more moderate and severe pancreatitis cases ($p = 0.003$), whereas stones ≤ 5 mm were predominantly associated with mild disease. Multiple stones were similarly associated with a greater proportion of moderate-to-severe presentations ($p = 0.002$) (Table 5).

Serum amylase levels demonstrated a significant progressive increase with increasing stone size: 880 ± 270 IU/L for stones ≤ 5 mm, 1240 ± 310 IU/L for stones 6–10 mm, and 1680 ± 420 IU/L for stones > 10 mm ($p < 0.001$), suggesting a relationship between stone size, degree of ductal obstruction, and extent of pancreatic inflammation.

Table 5. Relationship Between Gallstone Characteristics and Severity of Pancreatitis (Cases, n=92)

A. Stone Size and Severity of Pancreatitis

Stone Size	Mild	Moderately Severe	Severe
≤ 5 mm	10	2	0
6–10 mm	20	6	2
> 10 mm	28	16	8
p-value	0.003		

B. Stone Number and Severity of Pancreatitis

Variable	Association
Multiple stones	Greater proportion of moderate/severe pancreatitis; $p = 0.002$

C. Serum Amylase According to Stone Size

Stone Size	Mean Serum Amylase (IU/L)
≤ 5 mm	880 ± 270
6–10 mm	1240 ± 310
> 10 mm	1680 ± 420
p-value	< 0.001

DISCUSSION

The present study investigated the association between gallstone characteristics specifically stone size and number and the occurrence and severity of gallstone-induced acute pancreatitis in a tertiary care hospital in northern India. Our findings demonstrate that gallstone size is a significant and independent predictor of both the occurrence and severity of pancreatitis, and that the combined effect of small stone size and multiple stones confers the greatest risk.

The mean gallstone size was significantly larger in the pancreatitis group compared to the control group (12.6 ± 5.2 mm vs. 5.8 ± 2.4 mm; $p < 0.001$). Although counterintuitive, this finding is explained by the well-recognized paradox in gallstone-induced pancreatitis: while small gallstones (≤ 5 mm) are more likely to migrate through the cystic duct and cause transient ampullary obstruction, larger gallstones once they pass into the common bile duct tend to produce more sustained obstruction, greater ductal hypertension, and consequently more severe pancreatic inflammation.^[9] Our multivariate logistic regression confirmed gallstone size ≤ 5 mm as the strongest independent predictor of pancreatitis occurrence (Adjusted OR=4.82; $p < 0.001$), consistent with prior studies.^[10,11]

The observation that small gallstones carry the highest risk of pancreatitis is supported by robust epidemiological and physiological evidence. Venneman et al. demonstrated in a prospective cohort that small gallstones were independently associated with an increased risk of acute pancreatitis and hypothesized that their mobility, combined with preserved gallbladder motility and rapid cholesterol crystallization, facilitated repeated stone passage through the ampulla.^[10] A subsequent study by the same group identified fast crystallization and stone microstructure as additional determinants of migratory potential.^[11] Similarly, Sugiyama and Atomi reported stone size as the most significant risk factor for acute biliary pancreatitis among several evaluated parameters.^[17]

The role of gallstone number in pancreatitis risk is more complex and nuanced. In our study, stone number alone was not significantly associated with the occurrence of pancreatitis ($p = 0.89$), a finding that differs from some prior reports.^[18] However, when analyzed in combination with stone size through multivariate logistic regression, the presence of multiple gallstones emerged as a significant independent predictor (Adjusted OR=2.96; $p = 0.001$). This suggests that the effect of stone multiplicity is not independently sufficient but acts synergistically with small stone size to increase pancreatitis risk. A higher stone burden likely increases the probability of stone migration, recurrent ampullary obstruction, and biliary events, consistent with existing literature.^[17,19]

With respect to disease severity, our findings demonstrated that larger stones (> 10 mm) were associated with proportionally more cases of moderately severe and severe pancreatitis ($p = 0.003$), while stones ≤ 5 mm were associated predominantly with mild disease. This is consistent with the concept that larger biliary stones, once impacted at the ampulla, produce more prolonged obstruction of the pancreatic duct, sustained activation of pancreatic enzymes, and a more intense systemic inflammatory response.^[4,7] Correspondingly, serum amylase levels increased progressively with increasing stone size (880 ± 270 IU/L for ≤ 5 mm stones to 1680 ± 420 IU/L for > 10 mm stones; $p < 0.001$), suggesting a direct relationship between stone size, ductal obstruction severity, and the degree of pancreatic acinar cell injury.

Similarly, multiple stones were associated with a greater proportion of moderate-to-severe pancreatitis cases ($p = 0.002$). The increased likelihood of stone migration and recurrent biliary obstruction in patients with multiple stones may perpetuate pancreatic duct obstruction, amplify inflammatory cascades, and predispose to more severe and complicated disease courses.^[6,20]

The severity distribution in our cohort mild 63.0%, moderately severe 26.1%, and severe 10.9% is broadly consistent with population-level data from published series, which report mild pancreatitis in approximately 60–80% of cases.^[5,12] The Revised Atlanta Classification used in this study provides a clinically relevant and widely validated framework for severity stratification, incorporating organ failure and local complications as key determinants.^[12]

The predominance of female patients in both groups (58.7% controls, 67.4% cases) reflects the well-established higher prevalence of gallstone disease in women, attributed to female sex hormones, parity, and hormonal contraceptive use, which increase biliary cholesterol secretion and reduce gallbladder motility.^[1,2] The comparable age and sex distribution between groups strengthens the internal validity of our case-control comparison.

From a clinical standpoint, our findings have *important practical implications*. Ultrasonographic detection of small gallstones (≤ 5 mm) or multiple stones in symptomatic patients should be regarded as a high-risk feature warranting close monitoring and consideration of early elective cholecystectomy. Guidelines from several surgical societies recommend early laparoscopic cholecystectomy following the index episode of gallstone-induced pancreatitis to prevent recurrence, and our data support extending this risk-based approach to the pre-pancreatitis period.^[15,16] Early intervention in high-risk patients particularly those with microlithiasis or multiple small stones may prevent the first episode of pancreatitis altogether, reducing associated morbidity, hospitalization costs, and potential complications.

The present study has *several limitations* that should be acknowledged. As a hospital-based observational study, selection bias cannot be entirely excluded. The sample size, while adequate for the primary analysis, may limit subgroup statistical power. Gallstone characteristics were assessed by ultrasonography, which may underestimate stone number and has known limitations in resolving microlithiasis compared to endoscopic ultrasound or magnetic resonance cholangiopancreatography (MRCP). Additionally, data on biliary sludge, gallbladder wall thickness, and post-cholecystectomy histology were not systematically collected and may have provided additional mechanistic insights.

Prospective multi-centre studies with larger sample sizes and more granular imaging data are recommended to validate and extend these findings.

CONCLUSION

Gallstone characteristics, particularly stone size and multiplicity, are significant and independent determinants of both the occurrence and severity of gallstone-induced acute pancreatitis. Small gallstones (≤ 5 mm) confer the greatest independent risk of pancreatitis, likely through their migratory potential and propensity for ampullary impaction, while larger stones are associated with more severe disease once pancreatitis develops. The combined presence of small size and multiple stones amplifies this risk. Serum amylase levels and clinical severity both escalate with increasing stone size, underscoring the role of stone burden in determining disease course. Routine ultrasonographic assessment of gallstone size and number should be incorporated into clinical risk stratification for all patients with symptomatic cholelithiasis. Identifying high-risk gallstone morphology small stones or multiple stones should prompt early surgical referral and consideration of prophylactic cholecystectomy to prevent the first or recurrent episode of gallstone-induced pancreatitis and its potentially serious complications.

Human Ethics: Ethical approval for this study was obtained from the Institutional Ethics Committee of Teerthanker Mahaveer Medical College and Research Centre, Moradabad, and written informed consent was obtained from all participants.

Animal Ethics: No animal subjects were involved in this study.

Data Availability Statement: The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Author Contributions: All authors contributed substantially to the study conception, design, data collection, analysis, manuscript preparation, and approved the final version of the manuscript.

Acknowledgements: The authors acknowledge the support of the Department of General Surgery and Department of Radiodiagnosis, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, and thank all study participants.

Conflicts of Interest: The authors declare no conflicts of interest.

Funding Statement: The authors received no financial support or external funding for this study.

REFERENCES

1. Stinton LM, Shaffer EA. Epidemiology of gallbladder disease: cholelithiasis and cancer. *Gut Liver*. 2012;6(2):172–187. doi:10.5009/gnl.2012.6.2.172. PMID: 22570746.
2. Shaffer EA. Gallstone disease: Epidemiology of gallbladder stone disease. *Best Pract Res Clin Gastroenterol*. 2006;20(6):981–996. doi:10.1016/j.bpg.2006.05.004. PMID: 17127183.
3. Bates T, Harrison M, Lowe D, Lawson C, Padley N. Longitudinal study of gall bladder and biliary tract disease. *BMJ*. 1992;305(6863):1171–1175. doi:10.1136/bmj.305.6863.1171. PMID: 1467716.
4. Boxhoorn L, Voermans RP, Bouwense SAW, et al. Acute pancreatitis. *Lancet*. 2020;396(10252):726–734. doi:10.1016/S0140-6736(20)31310-6. PMID: 32891214.
5. Forsmark CE, Vege SS, Wilcox CM. Acute pancreatitis. *N Engl J Med*. 2016;375(20):1972–1981. doi:10.1056/NEJMra1505202. PMID: 27959604.
6. Toouli J, Brooke-Smith M, Bassi C, et al. Guidelines for the management of acute pancreatitis. *J Gastroenterol Hepatol*. 2002;17 Suppl:S15–39. doi:10.1046/j.1440-1746.17.s1.2.x. PMID: 12000591.
7. Lerch MM, Gorelick FS. Models of acute and chronic pancreatitis. *Gastroenterology*. 2013;144(6):1180–1193. doi:10.1053/j.gastro.2012.12.043. PMID: 23622126.
8. Lee SP, Nicholls JF, Park HZ. Biliary sludge as a cause of acute pancreatitis. *N Engl J Med*. 1992;326(9):589–593. doi:10.1056/NEJM199202273260902. PMID: 1734250.
9. Diehl AK, Holleman DR Jr, Chapman JB, Schwesinger WH, Kurtin WE. Gallstone size and risk of pancreatitis. *Arch Intern Med*. 1997;157(15):1674–1678. doi:10.1001/archinte.157.15.1674. PMID: 9250231.
10. Venneman NG, Buskens E, Besselink MG, et al. Small gallstones are associated with increased risk of acute pancreatitis: potential benefits of prophylactic cholecystectomy? *Am J Gastroenterol*. 2005;100(11):2540–2550. doi:10.1111/j.1572-0241.2005.00317.x. PMID: 16279912.
11. Venneman NG, Renooij W, Rehfeld JF, et al. Small gallstones, preserved gallbladder motility, and fast crystallization are associated with pancreatitis. *Hepatology*. 2005;41(4):738–746. doi:10.1002/hep.20616. PMID: 15793851.
12. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013;62(1):102–111. doi:10.1136/gutjnl-2012-302779. PMID: 23100216.

13. Tenner S, Baillie J, DeWitt J, Vege SS; American College of Gastroenterology. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol*. 2013;108(9):1400–1415. doi:10.1038/ajg.2013.218. PMID: 23896955.
14. Kundumadam S, Ratnayake CB, Windsor JA. Gallstone pancreatitis: general clinical approach and the role of endoscopic retrograde cholangiopancreatography. *ANZ J Surg*. 2020;90(9):1617–1623. doi:10.1111/ans.16000. PMID: 32474985.
15. da Costa DW, Bouwense SAW, Schepers NJ, et al. Same-admission versus interval cholecystectomy for mild gallstone pancreatitis (PONCHO): a multicentre randomised controlled trial. *Lancet*. 2015;386(10000):1261–1268. doi:10.1016/S0140-6736(15)00274-X. PMID: 26460661.
16. Mayumi T, Okamoto K, Takada T, et al. Tokyo Guidelines 2018: management bundles for acute cholangitis and cholecystitis. *J Hepatobiliary Pancreat Sci*. 2018;25(1):96–100. doi:10.1002/jhbp.519. PMID: 29098787.
17. Sugiyama M, Atomi Y. Risk factors for acute biliary pancreatitis. *Gastrointest Endosc*. 2004;60(2):210–212. doi:10.1016/S0016-5107(04)01544-2. PMID: 15278046.
18. Moreau JA, Zinsmeister AR, Melton LJ III, DiMagno EP. Gallstone pancreatitis and the effect of cholecystectomy: a population-based cohort study. *Mayo Clin Proc*. 1988;63(5):466–473. doi:10.1016/s0025-6196(12)64866-5. PMID: 3357292.
19. Ros E, Navarro S, Bru C, Garcia-Pugés A, Valderrama R. Occult microlithiasis in ‘idiopathic’ acute pancreatitis: prevention of relapses by cholecystectomy or ursodeoxycholic acid therapy. *Gastroenterology*. 1991;101(6):1701–1709. doi:10.1016/0016-5085(91)90410-y. PMID: 1955135.
20. Petrov MS, van Santvoort HC, Besselink MG, van der Heijden GJ, van Erpecum KJ, Gooszen HG. Early endoscopic retrograde cholangiopancreatography versus conservative management in acute biliary pancreatitis without cholangitis: a meta-analysis of randomized trials. *Ann Surg*. 2008;247(2):250–257. doi:10.1097/SLA.0b013e318153aca8. PMID: 18216530.