Section: Internal Medicine

Original Research Article

CLINICAL PROFILE OF ACUTE PANCREATITIS AT TERTIARY CARE HOSPITAL IN NORTH KASHMIR

Arvind Kumar Gautam[1], Shair Ahmad Dar[2], Zaffar Iqbal Kawoosa[3], Mir Mushtaq[4], Ifrah Reshi[5], Sajad[6], Nowsheen Nazir Parray[7], Aisha ahmad Dar[7]

[1]*Clinical Associate, CCM Apollo Medics Hospital, Lucknow, India.*
[2]*Medical Officer, Department of Radiology, GMC, Baramulla, India.*
[3]*Associate Professor, Medicine, GMC, Baramulla, India.*
[4]*Senior Consultant, General Surgery, GMC, Baramulla, India.*
[5]*MBBS, GMC, Baramulla, India.*
[6]*Faculty SKIMS MCH*
[7]*Pharma D, CT Group of Pharmaceutical sciences India*

Received *: 09/02/2026*
Received in revised form *: 22/03/2026*
Accepted *: 08/04/2026*

Corresponding Author:
Dr. Zaffar Iqbal Kawoosa,
Associate Professor, Medicine, GMC, Baramulla, India.
Email: medicalacademyanatomy@gmail.com

DOI: 10.70034/ijmedph.2026.2.129

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 750-756

ABSTRACT

Background: Acute pancreatitis is a common and potentially life-threatening inflammatory condition of the pancreas with a wide spectrum of clinical presentations ranging from mild, self-limiting disease to severe systemic illness. The etiology and clinical profile vary across geographic regions. **Aims & Objectives**: This study aimed to evaluate the clinical characteristics, etiological factors, and outcomes of acute pancreatitis in patients presenting to a tertiary care hospital in North Kashmir.

Materials and Methods: This was a hospital-based observational study conducted at a tertiary care center in North Kashmir. Patients diagnosed with acute pancreatitis based on standard diagnostic criteria (clinical features, elevated pancreatic enzymes, and radiological findings) were included. Demographic details, etiological factors, clinical presentation, laboratory parameters, imaging findings, severity assessment, complications, and outcomes were recorded and analyzed using appropriate statistical methods.

Results: A total of 200 patients were included in the study. The majority were females (68 %) with a mean age of 3 years. The 31-40 years (35%).The most common etiological factor was biliary cause–(58.5 %), followed by idiopathic(24.5%),Hypertriglyceridemia(4%),drugs(7%),,hyperparathyroidism (2%),Post ERCP (2%),anatomical causes(1%),trauma(1%) & viral causes(1%). The predominant presenting symptom was abdominal pain (100%), often associated with vomiting and abdominal tenderness. Based on severity classification, (47.5%) had mild, (32.5%) had moderately severe, and (20%) had severe pancreatitis. The most prevalent complication was hypocalcemia (27%), followed by ascites and kidney injury (9% each), pleural effusion (8%), and organ failure (4%). The overall mortality rate was (6%), with higher mortality associated with severe disease and organ dysfunction.

Conclusion: Acute pancreatitis in this region shows distinct etiological and clinical patterns, with gallstones/alcohol being the leading causes. Early diagnosis, severity stratification, and timely management are crucial in reducing morbidity and mortality. Regional data such as this are essential for improving clinical outcomes and guiding preventive strategies.

Keywords: Acute pancreatitis; Clinical profile; Etiology; Severity; Complications; North Kashmir; Tertiary care; Outcome.

INTRODUCTION

Pancreatitis is inflammation in the pancreas associated with injury to the exocrine and endocrine (at times) parenchyma, resulting in clinical manifestations ranging in severity from a mild, self-limited disease to a life threatening acute inflammatory process, the duration of which can

range from a transient attack to a permanent loss of pancreatic function.[1]

Pancreatic inflammatory disease may be classified as acute or chronic pancreatitis. The etiology of pancreatitis varies with geographical location but most commonly involves alcohol, gallstones, metabolic factors, and drugs.[1]

The diagnosis of acute pancreatitis requires two of the following three features: abdominal pain consistent with acute pancreatitis (acute onset of a persistent, severe, epigastric pain often radiating to the back); serum lipase activity (or amylase activity) at least three times greater than the upper limit of normal; and characteristic findings of acute pancreatitis on contrast-enhanced computed tomography (CECT) and less commonly magnetic resonance imaging (MRI) or transabdominal ultrasonography.

Pancreatitis leads to progressive destruction of the exocrine tissue and in some patients a loss of endocrine tissue as well.[1,2] However, owing to the tremendous reserve of pancreatic function, insufficiency may be at subclinical at least in the beginning of the disease. As a result, the early diagnosis of pancreatitis is difficult. Natural history as well as the prognosis of the disease remains yet to be defined. A number of situations can precipitate acute pancreatitis. Only a small fraction of patients with these predisposing factors develops the disease. The mechanism of induction of pancreatitis by these agents is not known. It is unclear that alcohol-induced pancreatitis occurs only after many years of alcohol abuse and not after a single binge in humans not habituated to alcohol use. Acute pancreatitis is a common cause of acute abdominal pain requiring hospital admission. The attack is mild in about 80% of patients who will show marked improvement within 48 hours. In some 20% of patients however it is often severe with high morbidity and mortality.[3,4]

The first 12 hours are extremely important to provide appropriate management which will decrease morbidity and mortality.[5-7]

Nearly 80% of cases of acute pancreatitis worldwide are caused by gall stone obstruction and high alcohol intake. Other causes like hypertriglyceridemia and drugs

account for the rest. It is necessary to identify the etiology to institute definitive management and to prevent further attacks and to assess the severity for the proper management of patients.

AP can be subdivided into two types: (a) interstitial oedematous pancreatitis and

(b) necrotizing pancreatitis. Depending on the severity, AP is divided into mild AP (no organ failure, no local or systemic complications), moderately severe AP (organ failure that resolves within 48 hours, i.e., transient organ failure, and/or local or systemic complications without persistent organ failure), and severe AP (persistent organ failure for more than 48 hours, that may involve single or multiple organs).[8]

several classification systems have been presented to assess the severity of acute pancreatitis. Presence of SIRS (Systemic inflammatory response syndrome), scores such as the Ranson, the Glasgow, and Acute Physiology and Chronic Health Evaluation (APACHE) are practical for assessing the severity of the disease but are not sufficiently well validated for predicting mortality. Early organ dysfunction predicts disease severity and patients require early intensive care treatment. Antibiotic prophylaxis is usually ineffective and early enteral feeding results in reduction of local and systemic infection.[9,10]

Management of acute pancreatitis has changed significantly over the past years. Early management is nonsurgical, solely supportive and patients with infected necrosis with worsening sepsis need intervention. Early intensive care has definitely improved the outcome of patients.[11]

Because of the frequent emergency, multimodality presentation, and various complications, this challenging subject is taken for the present study, which aims at understanding the etiology, clinical profile, complications associated with AP and studying its short-term outcome.

Aim and objectives

Aim: To study the clinical profile and outcome of acute pancreatitis.

Objectives

1. To study the etiology and clinical profile of acute pancreatitis.
2. To study the impact of severity on hospital stay and outcomes in cases of acute pancreatitis.
3. To estimate the different grade of acute pancreatitis.

MATERIALS AND METHODS

Study Area

Department of General Medicine, Government Medical College, Baramula, Jammu and Kashmir

Study Population

All the patients with evidence of acute pancreatitis coming to our hospital.

Study Design

A Prospective observational study

Sample Size

Consecutive type of non-probability sampling was followed for the selection of study subjects. A total of 200 subjects of diagnosed acute pancreatitis fulfilling the eligibility criteria and gave informed consent for participation in the study were selected. We determined the sample size using Krejcie and Morgan's table. The table is constructed using the following formula for determining sample size.

Study Duration

One year

Inclusion Criteria

1. Patients with age between 15 years old admitted in indoor with evidence of acute pancreatitis

Exclusion Criteria

1. Patients who were diagnosed to have chronic pancreatitis based on their previous records or found to have features of chronic pancreatitis upon radiological investigations during the course of their stay such as pancreatic calcifications, dilated pancreatic duct, areas of atrophy and pseudocyst will be excluded from the study.
2. Those subjects not willing take part in study

Methodology

This study includes 200 subjects with acute pancreatitis presented to our hospital in the time period of one year. After detailed history and physical examination, laboratory investigations were sent at the time of admission: arterial blood gas analysis, hematocrit, kidney function test, liver function test, serum electrolytes, serum amylase, serum lipase, fasting lipid profile and complete hemogram, and CRP concentrations were collected.

All patients underwent abdominal ultrasonography at admission and contrast enhanced pancreatic CT scan 72 hours after symptom onset.

The diagnoses of acute pancreatitis was based on the presence of two of the following three criteria: (i) abdominal pain characteristic of acute pancreatitis; (ii) serum amylase and/or lipase levels at least three times the upper limit of normal; and (iii) characteristic findings of acute pancreatitis on abdominal ultrasonography and/or computerized tomography (CT) scan.

At the time of discharge/death, patients were graded as having mild, moderately severe and severe acute pancreatitis based on the Atlanta 2012 classification. Patients with mild acute pancreatitis had neither local complications nor organ failure. Patients with moderately severe acute pancreatitis had transient organ failure or local complications or both, whereas patients with severe acute pancreatitis had persistent organ failure. Organ failure was defined based on the Modified Marshall scoring system. A score of >2 for more than 48 hours was considered as persistent organ failure, where as a score of >2 for less than 48 hours was considered as transient organ failure. Local complications included pancreatic necrosis, acute fluid collections, pseudocyst, acute necrotic collections and necrosis.

Severe Acute Pancreatitis (SAP) was defined by the Atlanta classification 2012, is established by either an APACHE II score >8, Ranson score >3, the presence of more than one organ failure, or local complications. Sepsis was defined as sepsis according to the ―Survival Sepsis Campaign 2012. Systemic inflammation response syndrome (SIRS) was defined as two or more of temperature <36 C or >38 C, heart rate >90 beats/min(bpm), white blood cell count <4x10^9/L or >12x 10^9/L or >10% band forms and PaCO2<4.3kPa.

Management protocols

Patients presenting to the emergency department, suspected of having acute pancreatitis, were adequately resuscitated using crystalloids, primarily ringer‘s lactate. Inotropes and colloids were added if the patients failed to respond to crystalloids. All patients were catheterized to monitor the urine output and ascertain the adequacy of resuscitation. Central venous access was obtained for patients who failed to respond to initial resuscitation measures to monitor the central venous pressure and guide further fluid management. A nasogastric tube was placed for all patients. All patients were kept nil per oral for the first 24 hours. Subsequently, patients were examined daily and enteral feeding by means of a nasogastric tube or orally was initiated as soon as features of ileus resolved. Patients with pseudopancreatic cyst underwent surgical intervention.

Statistical Analysis

All the data was noted down in a pre-designed study proforma. Qualitative data was represented in the form of frequency and percentage. Association between qualitative variables was assessed by Chi-Square test. Quantitative data was represented using Mean ± SD. Analysis of Quantitative data between the two groups was done using unpaired t-test if data passed ‗Normality test‘ and by Mann-Whitney Test if data failed

Normality test‘. A p-value < 0.05 was taken as level of significance. Results were graphically represented where deemed necessary. SPSS Version 26.0 was used for most analysis and Microsoft Excel 2021 for graphical representation.

RESULTS

Table 1: Distribution of study subjects according to age in months (n=120)

Age Group (years)	N	%
<= 20	23	11.5%
21-30	23	11.5%
31-40	70	35.0%
41-50	50	25.0%
51-60	25	12.5%
61-70	9	4.5%
Total	200	100.0%

Table 2: Distribution of study groups as per gender

Sex	N	%
Male	64	32.0%
Female	136	68.0%
Total	200	100.0%

Table 3: Distribution of study groups as per presenting symptoms

Symptoms	N	%
Pain in Abdomen	200	100.0%
Fever	65	32.5%
Nausea and Vomiting	200	100.0%
Jaundice	42	21.0%

Table 4: Distribution of study groups as per co-morbidities

Co-morbidity	N	%
Diabetes	38	19.0%
Hypertension	50	25.0%
Dyslipidemia	46	23.0%
Cardiovascular disease	20	10.0%
Hyperuricemia	28	14.0%
Hyperparathyroid	4	2.0%
Seizure disorder	2	1.0%
liver disease	4	2.0%

Table 5: Distribution of study groups as per etiology

Etiology	N	%
Biliary (Gall bladder stone)	117	58.5%
Idiopathic	45	24.5%
Hypertriglyceridemia	8	4.0%
Drug (Hydrochlorothiazide)	6	3.0%
Drug (DPP-4 inhibitors)	4	2.0%
Drug (Azathioprine)	2	1.0%
Drug (sodium valproate)	2	1.0%
Hyperparathyroidism	4	2.0%
Post ERCP	4	2.0%
Anatomical (Pancreatic divisum)	2	1.0%
Trauma	2	1.0%
Viral(mumps)	2	1.0%
Total	200	100.0%

Table 6: Distribution of study groups as per severity of acute pancreatitis as per Atlanta classification

Grade of AP (Atlanta criteria for severity)	N	%
Mild	95	47.5%
Moderate	67	33.5%
Severe	38	19.0%
Total	200	100.0%

Table 7: Distribution of study groups as per Ranson's criteria

Ranson's Score	N	%
Non-severe (<3)	162	81.0%
Severe (>=3)	38	19.0%
Total	200	100.0%

Table 8: Distribution of study groups as per APACHE Score

APACHE Score	N	%
Non-severe (<=8)	164	82.0%
Severe (>8)	36	18.0%
Total	200	100.0%

Table 9: Association of grade of acute pancreatitis with modified CT severity index and BISAP score

Grade of AP	Number	Modified CT severity index		BISAP Score	
		Mean	SD	Mean	SD
Mild	95	1.30	0.60	1.27	0.22
Moderate	67	5.40	1.34	2.20	0.98
Severe	38	8.90	1.91	3.90	1.32

Table 10: Distribution of study groups as per complications

Complications	N	%
ARDS	6	3.0%
Kidney Injury	16	8.0%
Ascites	18	9.0%
Pleural effusion	16	8.0%
Organ failure	8	4.0%
Necrosis	14	7.0%

Shock	2	1.0%
Hypocalcemia	54	27.0%
Pseudocyst	12	6.0%
None	54	27.0%
Total	200	100.0%

Table 11: Distribution of study groups as per Management details

Management	**N**	**%**
Conservative	120	60.0%
ICU care	38	19.0%
Surgery	42	21.0%

Table 12: Distribution of study groups as per hospital stay

Hospital Stay	**N**	**%**
<=5 days	108	54.0%
5-10 days	78	39.0%
> 10 day	14	7.0%
Total	200	100.0%

Table 13: Distribution of study groups as per outcome of cases

Outcome	**N**	**%**
Died	12	6.0%
Survived	188	94.0%
Total	200	100.0%

Table 14: Mean hospital stay comparison among different grade of acute pancreatitis

Grade of AP	**N**	**Hospital Stay**	**SD**
Mild	95	5.10	3.40
Moderate	67	6.79	4.10
Severe	38	7.91	4.60
p-value < 0.01			

Table 15: Mortality among different grades of acute pancreatitis

Grade of AP	**N**	**Mortality**	**%**
Mild	95	0	0.0%
Moderate	67	0	0.0%
Severe	38	12	31.6%
Total	200	12	6.0%
p-value < 0.01			

DISCUSSION

Acute pancreatitis is a significant medical condition that can lead to severe morbidity and mortality. Therefore, understanding the local demographic trends, presenting symptoms, and hospital trajectories is essential for improving patient care and resource allocation.

Study included 200 patients with evidence of acute pancreatitis coming to our hospital. The majority of patients (35%) were in the 31-40 years age group. A higher prevalence was noted in females (68%) compared to males (32%), indicating a significant gender difference in acute pancreatitis cases.

Prasad HL et al,[12] evaluated the clinical profile of 40 patients suffering from acute pancreatitic patients. Out of 40 patients, Males were (55%) and (45%) were females. Majority of patients were belonging to the age group of 41-60 (42.5%) with a median age of 37 years. Ahmad et al,[13] studied and found similar results. Negi et al,[14] studied 123 patients of acute pancreatitis. Thakur S et al,[15] found most of the patients were in the 21–40-year age group. Yadav AP et al,[16] studied 45 patients with similar disease presentation.

All patients reported abdominal pain and nausea/vomiting, while fever was present in 32.5% of cases, showcasing the prominency of digestive symptoms. Hypertension (25%) and dyslipidemia (23%) were the most common co-morbidities, with diabetes reported in 19% of patients, indicating significant accompanying health conditions.

Many researchers found similar results like Ahmad et al,[13] Negi et al,[14] Das SK et al,[17] Patel ML et al.[18]

The predominant cause of acute pancreatitis was biliary factors, accounting for 58.5%, followed by idiopathic cases at 24.5%.Similar results were found by Prasad HL et al,[12] Negi et al,[14] Yadav AP et al,[16] Dogra et al.[19]

Nearly half of the patients (47.5%) had mild acute pancreatitis, while severe cases constituted 19% of the studied population. According to Ranson's criteria, 81% of patients fell into the non-severe category (score <3), and 19% were classified as severe (score ≥3). The APACHE score revealed that 80% of patients were classified as non-severe (score ≤8) while 20% were considered severe (score >8).

Other researchers who study the clinical features include Ahmad et al,[13] Dhar et al.[20]

In present study, mild cases had a lower mean heart rate (80.22 bpm) compared to moderate (85.43 bpm) and severe cases (90.12 bpm), with a significant p-value (<0.01). Both respiratory rates and blood pressures exhibited significant differences ($p < 0.01$). In Negi et al,[14] Dhar et al,[20] Hossain Mazumder et al,[21] also studied ,laboratory investigations.

In present study, there was a clear trend indicating an increase in the mean modified CT severity index (CTSI) and BISAP score with the severity of acute pancreatitis, highlighting the correlation between severity and imaging/scoring results.

SrinivasaRao et al,[22] study also observed that both BISAP and CTSI scores had positive correlation with morbidity and mortality.Hypocalcemia was the most common complication at 27%, with kidney injury (8%) and ascites (9%) also notable, indicating the potential for serious adverse effects.Many scholars also studied complications include Puno-Santos et al. (2015),[23] Yadav et al. (2014),[24] Patel ML et al,[18] Thakur S et al.[15] Management strategies included conservative treatment for 60% of patients, with 21% requiring surgical intervention, indicating varied approaches based on severity. Other Researchers who studied management include Das SK et al,[17] Yadav AP et al,[16] Miskovic et al. (2015),[25] Banks et al. (2013),[26] Mier et al. (2002).[27]

The survival rate was notably high at 94%, with only 6% of patients succumbing to their condition, reflecting overall effective management and recovery. Ahmad et al,[13] Negi et al,[14] study, Elta et al. (2014),[28] Lankisch et al. (2015),[29] & Hossain Mazumder et al,[21] study, a majority of patients experienced a favorable prognosis. To summarize, present study emphasizes the clinical significance of understanding patient demographics, presenting symptoms, etiologies, and outcomes associated with the condition.

CONCLUSION

This study provides significant insights into the clinical profiles and outcomes of patients with acute pancreatitis. The findings indicate a higher prevalence among females and those aged 31-40 years, with abdominal pain and nausea/vomiting being universal presenting symptoms. Biliary causes were the most common etiology, highlighting the importance of addressing this factor in patient management. The classification of acute pancreatitis revealed that nearly 20% of cases were severe, which correlated with a higher risk of complications and a notable mortality rate of 31.6% among severe cases. The majority of patients (94%) survived, and most were managed conservatively, suggesting that effective management strategies can lead to favorable outcomes. However, the longer hospital stays associated with severe cases underline the need for timely intervention and monitoring of complications. Overall, the study emphasizes the critical nature of recognizing and addressing acute pancreatitis to improve patient outcomes.

REFERENCES

1. Harada MH, Kunichika. Clinical course and prognosis of chronic pancreatitis. Pancreas. 1987;2:378-85.
2. Worming H. Chronic pancreatitis; pathogenesis. natural history and conservative treatment. Clinical Gastroenterol. 1987;13:871-94.
3. Banks P, Free M. Practice guidelines in acute pancreatitis. Am J Gastroenterol. 2006;101(10):237- 400.
4. Bardley EL. A clinically based classification system for acute pancreatitis. Arch Surg. 1993;128(5):586- 89.
5. Skaife P, Kingsnorth AN. Acute pancreatitis: assessment and management: Post Grad Med J. 1996;72:277- 83.
6. Matar ZS. Clinical pattern of acute pancreatitis. Internet J Surg. 2007;11(1):39-44.
7. Munsell MA, Buscaglia JM. Acute pancreatitis. J Hosp Med. 2010;5:241-50.
8. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2012;62:102–111.
9. Working Group IAP/APA Acute Pancreatitis Guidelines. IAP/APA evidence-based guidelines for the management of acute pancreatitis. Pancreatology. 2013;13:e1.
10. Windsor JA. A better way to predict the outcome in acute pancreatitis? Am J Gastroenterol. 2010;105:1671.
11. Chauhan S, Forsmark CE. The difficulty in predicting outcome in acute pancreatitis. Am J Gastroenterol. 2010;105:443.
12. Prasad HL, Nagarjuna TR. Clinical profile of patients with acute pancreatitis. Int J Res Med Sci 2016;4:2994-7.
13. U Ahmed, MA Ahad, MA Alim, ARMS Ekram Clinical profile of acute pancreatitis in a teaching hospital Bang Med J Khulna 2016; 49 : 7-12.
14. Nitesh Negi, JatinderMokta, Brij Sharma, Rajesh Sharma, AnupamJhobta, Vishal Bodh, AshaRanjan. Clinical Profile and Outcome of Acute Pancreatitis: A HospitalBased Prospective Observational Study in Subhimalayan State. Journal of The Association of Physicians of India 2018; 66:22-24
15. Thakur S, Kaur R, Bhatia L, Bansal R, Singh A, Singh J. Acute Pancreatitis: Clinical Profile of 60 Patients. Cureus. 2023 Dec 28;15(12):e51234.
16. Aditya Prakash Yadav, Akash Raya, Binod Kumar Rai, Rabin Pratap Shah Clinical Profile of Acute Pancreatitis Janaki Medical College Journal of Medical Sciences 2022; 10 (2):65-69
17. Das, S. K., & Das, S. (2020). Clinical profile of patients with acute pancreatitis in a tertiary care centre in Tripura: A retrospective study. Asian Journal of Medical Sciences, 11(6), 96–100.
18. Patel ML, Shyam R, Atam V, Bharti H, Sachan R, Parihar A. Clinical profile, etiology, and outcome of acute pancreatitis: Experience at a tertiary care center. Ann Afr Med. 2022 Apr-Jun;21(2):118-123.
19. Dogra V, Ahmed RN, Peer JA, Gilkar IA. Clinical study of non-gall stone induced pancreatitis in a tertiary care centre. IntJSci Rep2022;8(5):125-8.
20. Dhar N, Shafi SM, Singh J, Gulzar GM. Epidemiology, incidence, etiology, complications and outcome of Acute Pancreatitis in Kashmiri Population: A retrospective hospital based single centre study. Med. res. chronicles [Internet]. 2022.
21. Hossain Mazumder et al. Aetiology and Clinical Profile of Acute Pancreatitis in a Tertiary Care Hospital. Journal of Comilla Medical College Teachers' Association. 2024; 27(2), 67–74.
22. Rao Bezwada Srinivasa, SreeVane Matta, Chandra V. Sarat Etiology, clinical profile, severity and outcome of acute pancreatitis in relation to bed side index for severity of acute pancreatitis BISAP and CT severity index [CTSI] scores.

International Journal of Medical Research & Health Sciences 2014;3(4):922-
23. Puno-Santos L, Korzenik JR, McPhee AJ, et al. The spectrum of hypoalbuminemia, hypocalcemia, and acute pancreatitis. Am J Gastroenterol. 2015;110(12):1767-1774.
24. Yadav D, Lowenfels AB, Gardner TB. Renal failure and the outcome of acute pancreatitis. Gastroenterology. 2014;146(6):1620-1629.
25. Miskovic A, McEwan D, Wong T, et al. A systematic review of the application of conservative management of acute pancreatitis. HPB. 2015;17(6):478-487
26. 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.
27. Mier J, Ocón M, Menchén L, et al. Early discharge of patients with acute pancreatitis: is it safe? Pancreatology. 2002;2(5):439-446.
28. Moole H, Attri AK, Verma R, et al. Mortality risk in acute pancreatitis: A meta-analysis. Gastroenterology. 2018;155(4):1068-1079.
29. Lankisch PG, Apte M, Banks PA. Acute pancreatitis. Lancet. 2015;386(9996):85-96.