Clinical Outcomes of Patients with Acute Pancreatitis at a Tertiary Care Centre: Dhulikhel Hospital

Shrestha NM, Pathak S, Poudel P, Sapkota V, KC Pukar, Neupane S, Gurung RB

Department of Internal Medicine,
Dhulikhel Hospital, Kathmandu University Hospital,
Kathmandu University School of Medical Sciences,
Dhulikhel, Kavre, Nepal.

Corresponding Author

Nikesh Mani Shrestha
Department of Internal Medicine,
Dhulikhel Hospital, Kathmandu University Hospital,
Kathmandu University School of Medical Sciences,
Dhulikhel, Kavre, Nepal.
E-mail: drnikestha@gmail.com

Citation

Shrestha NM, Pathak S, Poudel P, Sapkota V, KC Pukar, Neupane S, et al. Clinical Outcomes of Patients with Acute Pancreatitis at a Tertiary Care Centre: Dhulikhel Hospital. *Kathmandu Univ Med J.* 2025; 93(5): 89-94. (Special Issue)

ABSTRACT

Background

Acute pancreatitis (AP) is an inflammatory condition of the pancreas with a wide spectrum of severity, ranging from mild, self-limiting illness to severe disease with systemic complications and high morbidity.

Objective

To evaluate the clinical profile, etiology, severity, outcomes, and risk factors for complications in patients with acute pancreatitis admitted to a tertiary care center in Nepal.

Method

An observational cross-sectional study was conducted among 76 patients with acute pancreatitis admitted at Dhulikhel Hospital from December 2023 to November 2024 AD. Diagnosis were made based on the most recent revised Atlanta criteria 2012. Clinical, laboratory, and radiological data were collected. Severity classification, predictors of complications, and outcome measures including hospital stay, mortality, and late complications were analyzed using descriptive statistics for baseline characteristics, Chi-square test were applied to compare categorical and continuous variables, respectively. A p-value < 0.05 was considered statistically significant.

Result

The mean age was 47.72 ± 17.26 years, with a male predominance (53.9%). Alcohol (46%) and biliary causes (38%) were the most common etiologies. Based on the severity, 43.42% had mild AP, 46.05% had moderately severe acute pancreatitis and 10.52% had severe acute pancreatitis. C-Reactive Protein (CRP) was the most reliable severity marker (sensitivity 77.78%, specificity 96.88%). Acute Respiratory Distress Syndrome (ARDS) occurred in 87.5% of severe acute pancreatitis cases. Acute Kidney Injury (AKI) among acute pancreatitis was significantly associated with prolonged hospital stay ($p < 0.001$). Among 22 patients with peri-pancreatic fluid collection, 60% of those with severe acute pancreatitis developed late complications. Risk of developing late complications was higher in patients with Bedside Index for Severity in Acute Pancreatitis (BISAP) ≥ 2, hematocrit ≥ 44%, Body Mass Index (BMI) ≥ 25 kg per square meter, and Acute Kidney Injury.

Conclusion

Alcohol and biliary causes are the most common causes of acute pancreatitis in our population. C-Reactive Protein is a strong early marker of severity, while Acute Kidney Injury is a key determinant of hospital stay. Patients with severe acute pancreatitis, high Bedside Index for Severity in Acute Pancreatitis scores, or elevated hematocrit are more likely to develop late complications. Early risk stratification using simple markers can guide timely intervention and improve outcomes.

KEY WORDS

Acute pancreatitis, Outcome measures, Tertiary care center

INTRODUCTION

Acute pancreatitis (AP) is defined as the inflammation of the pancreas, characterized by parenchymal edema and necrosis caused by auto digestion by its own enzyme, involving regional tissues or remote organ system, leading to multi organ failure or death.[1] The two most common etiology of AP are alcohol and gall stones, contributing 80% of all cause, and gall stones being most common cause.[2] The severity of acute pancreatitis can be accessed using various scales; some of the widely used being Atlanta criteria, Bedside Index for Severity in Acute Pancreatitis (BISAP) score, Ranson Criteria, CT severity index and modified Glasgow score. Severity is an important indicator of mortality and facilitates management decisions for intensive care, nutritional support, urgent surgical intervention and antibiotic use.[3,4]

Severe AP occurred in 20% patients with AP and has mortality rates of 10-30%.[5] The morbidity increases significantly with the development of local complication and systemic complication involving single or multiorgan failure.[6]

CRP is a useful marker for predicting severity of disease only when used after 48 hrs of onset of acute symptoms. Hematocrit has been routinely assessed in every AP cases at the time of admission and may constitute a good marker for severity of AP.[7] Obesity has been identified as a negative prognostic factor in acute pancreatitis.[8]

In this background, the present study was done to look for the outcome of AP in terms of length of hospital stay, development of complications and mortality, as well as to look for the markers to predict severe AP and mortality in AP.

METHODS

This prospective cross-sectional study was conducted among patients presented to Dhulikhel hospital with the complain of pain abdomen and diagnosed with acute pancreatitis and admitted to the medical ward or ICU of Dhulikhel Hospital from December 2023 to November 2024. Patients aged > 18 years meeting at least two of the Revised Atlanta 2012 criteria for acute pancreatitis were included.[1] (i) Abdominal pain characteristic of AP, (ii) serum amylase and/or lipase levels at least three times the upper limit of normal, and (iii) characteristic findings of AP on abdominal ultrasonography and/or CT scan. Patients who were suffering from chronic pancreatitis based on their hospital records or had radiological findings of pancreatic calcifications, dilated pancreatic duct, areas of atrophy, and pseudo cysts and referred cases from other centers were excluded.

Data on demographics, clinical presentation, laboratory parameters (CBC, LFT, RFT, CRP, Ca++, FLP, etc.), and radiological findings were collected using a structured proforma. The severity of acute pancreatitis was assessed using the Modified Atlanta Classification, and organ failure was evaluated with the Marshall scoring system. Patients with mild AP had neither local complications nor organ failure. Patients with moderately severe AP had transient organ failure or local complications or both, whereas patients with severe AP had persistent organ failure. Organ failure was defined based on the Modified Marshall scoring system. A score of ≥ 2 for more than 48 h was considered as persistent organ failure, whereas a score of ≥ 2 for < 48 h was considered transient organ failure. Local complications included pancreatic necrosis, acute fluid collections, pseudo cyst, acute necrotic collections, and walled off necrosis. All the patients were managed as per the standard protocol.

Total Population sampling was used, and data were collected over a one-year period. Data were entered in Microsoft Excel and analyzed using SPSS version 25.0. Descriptive statistics were used for baseline characteristics. The Chi-square test and Student's t-test were applied to compare categorical and continuous variables, respectively. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 76 patients with acute pancreatitis were included in the study. The mean age of the patients was 47.72 ± 17.26 years, with a slight male predominance (53.94% male, 46.06% female). In terms of disease severity, 43.42% of patients had mild acute pancreatitis (AP), 46.05% had moderately severe AP, and 10.52% had severe AP based on Modified Atlanta 2012 classification. The mean body mass index (BMI) of the cohort was 24.94 ± 3.72 kg/m², with no significant variation across severity groups (Mild: 24.84 ± 4.16, moderately severe: 25.19 ± 3.39, Severe: 24.32 ± 3.45). The average duration of hospital stay was 5.03 ± 3.29 days, which increased with disease severity (Mild: 4.6 ± 3.49, moderately severe: 4.83 ± 2.50, Severe: 7.63 ± 4.66). Regarding patient outcomes, 97.36% (n=74) of patients showed clinical improvement. Two patients left against medical advice (LAMA), and there was no mortality (Table 1).

The most common causes of acute pancreatitis were Alcohol (46.05%) followed by biliary causes (38.16%) and ERCP-related (9.21%). Less common causes included hypertriglyceridemia, drug-induced (thiazide), idiopathic, and viral etiologies. Alcohol and biliary causes were evenly distributed across all severity groups, whereas ERCP was more associated with severe cases (Table 2).

Smoking was more frequent among severe cases (50%) than mild (39.39%). Pleural effusion and ascites were present in 37.5% of severe cases. ARDS occurred in 87.5% of severe cases and 11.42% in moderately severe cases. CRP > 150 mg/dL was present in 87.5% of severe cases compared to only 6.06% of mild cases (Table 3), supporting its predictive value as shown in table 4. Despite some high-risk features,

Table 1. Demographic profile of patients with acute pancreatitis (N=76)

Characteristics	Number (%)
Age range (years)	47.72±17.26
Male	41 (53.94)
Female	35 (46.06)
Severity	
Mild AP	33 (43.42)
Moderately severe AP	35 (46.05)
Severe AP	8 (10.52)
Average BMI (kg/m^2)	24.94±3.72
Mild	24.84±4.16
Moderately severe	25.19±3.39
Severe	24.32±3.45
Duration of hospital stay (day) average	5.026±3.29
Mild AP	4.6±3.49
Moderately severe AP	4.83±2.50
Severe AP	7.63±4.66
Outcome	
Improved	74 (97.36)
LAMA	2

Table 2. Etiology of patients with acute pancreatitis

Etiology	Frequency n (%)	Mild AP n (%)	Moderately severe AP n (%)	Severe AP n (%)
Alcohol	35 (46.05)	15 (45.45)	17 (48.57)	3 (37.50)
Biliary	29 (38.16)	11 (33.33)	15 (42.85)	3 (37.50)
ERCP	7 (9.21)	4 (12.12)	1 (2.85)	2 (25.00)
Hypertriglyceri-demia	1 (1.32)	0	1 (2.85)	
Idiopathic	1 (1.32)	1 (3.03)	0	
Viral	1 (1.32)	1 (3.03)	0	
Drug (thiazide)	2 (2.63)	1 (3.03)	1 (2.85)	
Total	76	33	35	8

the recovery rate was 100% in both mild and moderately severe AP groups and 75% in the severe group, with two patients left against medical advice (Table 3).

C-reactive protein (CRP > 150 mg/dl) was the most reliable predictor with 77.78% sensitivity, 96.88% specificity, and a positive predictive value (PPV) of 87.5%. BMI ≥ 25 kg/m² showed low sensitivity (22.22%) and moderate specificity (82.61%). Hematocrit ≥ 44 mm had low predictive value (PPV: 12.5%) and poor sensitivity (12.5%), although its specificity was 78.79% (Table 4).

AKI (acute kidney injury) was significantly associated with prolonged hospitalization (p = <0.001). Other factors such as pleural effusion, ARDS, high CRP, peripancreatic fluid collection, high hematocrit, or elevated BMI did not show statistically significant associations with hospital stay duration (Table 5).

Table 3. Subgroup analysis of various parameters with severity in patients of acute pancreatitis

Parameters	Mild AP n (%)	Moderately severe AP n (%)	Severe AP n (%)
Smoking	13 (39.39)	16 (45.71)	4 (50)
Pleural effusion	0	6 (17.14)	3 (37.50)
Ascites	0	10 (28.57)	3 (37.50)
BMI ≥ 25 kg/m^2	14 (42.42)	18 (51.42)	4 (50.0)
ARDS	0	4 (11.42)	7 (87.50)
HCT ≥ 44 mm in 1st h	7 (21.21)	10 (28.57)	1 (12.50)
CRP > 150 mg/dl	2 (6.06)	13 (37.17)	7 (87.50)
Outcome			
Recovered	33 (100)	35 (100)	6 (75.00)
Mortality	0	0	0

Table 4. Comparison of body mass index, hematocrit, and C-reactive protein at admission to determine severity of acute pancreatitis

Parameters	BMI (%)	CRP (%)	HCT (%)
Sensitivity	22.22	77.78	12.50
Specificity	82.61	96.88	78.79
Positive predictive value	50.00	87.50	12.50
Negative predictive value	57.58	93.94	78.79

Table 5. Association of different parameters with hospital stay (N = 76)

Parameters (n)	Hospital stay		P value
	≤ 7days	> 7 days	
Pleural effusion (9)	7	2	0.482
ARDS (11)	8	3	0.365
High CRP > 150 (22)	17	5	0.216
Low calcium (25)	22	3	0.668
Peripancreatic fluid (22)	17	5	0.218
BISAP ≥ 2 (23)	19	4	0.634
TLC > 1100 (39)	34	5	0.674
Hematocrit ≥ 44% (18)	16	2	0.643
BMI ≥ 25 (36)	32	4	0.377
AKI (3)	0	3	< 0.001

Out of 22 patients assessed for resolution of peripancreatic fluid collection or development of late complications (pseudocyst/Walled of Necrosis) in 4-6 weeks follow up, 17 had moderately severe acute pancreatitis (MSAP) and 5 had severe acute pancreatitis (SAP). Among moderately sever AP patients, 82.36% achieved resolution, while 17.64% developed late complication. In contrast, among patients with severe AP, 40% recovered, whereas 60% developed late complication, indicating a higher chance of developing late complication rate in severe disease, 18.18% patients with alcohol-induced pancreatitis, 22.22% patients with biliary pancreatitis, one patient with ERCP-induced pancreatitis (100%) and the one with hypertriglyceridemia-induced

pancreatitis (100%) developed late complication. 50% patients with BISAP scores ≥ 2 developed late complication. Similarly, 50% patients with hematocrit ≥ 44% developed late complication. Among those with BMI ≥ 25 kg/m² 36.36% developed late complication. 1 of 2 patients with acute kidney injury developed late complication (Table 6).

Table 6. Risk factor for development of late complication in patient with peri-pancreatic fluid collection (n=22)

Paramaters	Resolved (%)	Pseudocyst/WON
Moderately severe AP (17)	14 (82.36)	3 (17.64)
Severe AP (5)	2 (40)	3 (60)
Etiology		
Alcohol (11)	9 (81.82)	2 (18.18)
Biliary (9)	7 (77.78)	2 (22.22)
ERCP (1)	0	1 (100)
High triglyceridemia (1)	0	1 (100)
BISAP ≥ 2 (6)	3 (50)	3 (50)
Hematocrit ≥ 44 (6)	3 (50)	3 (50)
BMI ≥ 25 (11)	7 (63.64)	4 (36.36)
AKI (2)	1 (50)	1 (50)

DISCUSSIONS

This prospective study evaluated the demographic characteristics, etiological factors, clinical severity, laboratory findings, and outcomes of 76 patients diagnosed with acute pancreatitis (AP) at Dhulikhel Hospital. The results have been compared with existing literature, both national and international, to assess similarities, discrepancies, and implications for clinical practice.

The mean age of patients was 47.72 ± 17.26 years, which falls within the middle age group. This finding aligns with studies conducted by Bhattarai et al. who reported a similar mean age, reflecting the typical age group affected by acute pancreatitis in South Asia.[9] The gender distribution in our study showed a male predominance (53.94%), which is consistent with the study done by Mukherjee et al. and Gautam et al.[10,11] This gender pattern may be attributed to higher alcohol consumption among males, a key etiological factor for acute pancreatitis

In our study, alcohol (46.05%) was the most common cause of acute pancreatitis, followed by biliary cause (38.16%). These findings are comparable to those study conducted by Manandhar et al. and Rao et al. who also reported alcohol as the leading cause of AP.[12,13] Interestingly, ERCP-induced pancreatitis accounted for 9.21% of cases, with a higher proportion seen in severe acute pancreatitis. This may be due to the increasing use of ERCP for biliary and pancreatic pathologies and is supported by study done by Patel et al.[14] Less frequent causes included hypertriglyceridemia (1.32%), drug-induced (thiazide, 2.63%), idiopathic (1.32%), and viral causes (1.32%). These were similarly reported in other studies.[9,13]

Based on the Modified Atlanta 2012 classification, 43.42% had mild acute pancreatitis, 46.05% had moderately severe acute pancreatitis and 10.52% had severe acute pancreatitis. This pattern is comparable to that reported by Negi et al. and Gautam et al. where the majority of cases were either mild or moderately severe, reflecting timely diagnosis and supportive care.[2,10]

Patients with severe AP had higher rates of complications such as pleural effusion (37.5%), ascites (37.5%), and acute respiratory distress syndrome (ARDS, 87.5%). ARDS was observed more in the severe group, consistent with the findings of Reid et al. and Shah et al. who emphasized ARDS as a major contributor to morbidity and intensive care need in AP.[15,16]

CRP (> 150 mg/dL) demonstrated moderate sensitivity (77.78%) but high specificity (96.88%) and a PPV of 87.5% for predicting severe acute pancreatitis. These values suggest that while CRP is a useful adjunct in early risk stratification, it should be interpreted alongside other clinical and biochemical parameters rather than as a standalone strong predictor. This aligns with findings from Raghuwanshi et al. and Vengadakrishnan et al. which also noted its utility when used in combination with other indicators.[18,19] BMI ≥ 25 kg/m² showed low sensitivity (22.22%) but a moderate specificity (82.61%). Although obesity is a known risk factor for severe AP, our data doesn't suggest it as a strong predictor. Similar results were found in the study conducted by Vengadakrishnan et al.[19] Hematocrit ≥ 44 mm showed poor sensitivity (12.5%) and PPV (12.5%) but a fair specificity (78.79%). These values indicate that hematocrit can help during the early assessment of a patient, but on its own, it is not a strong or reliable marker for predicting how severe the disease will be. Similar results was found in the study done by Negi et al.[2]

The mean duration of hospital stay was 5.03 ± 3.29 days, with a clear trend of increasing length of stay in severe acute pancreatitis (7.63 ± 4.66 days) compared to mild cases (4.6 ± 3.49 days) and moderately severe cases (4.83 ± 2.50 days). This is in agreement with findings from studies done by Mukherjee et al. and Poudel et al.[10,20] Smoking was more common among patients with severe AP (50%), probably due to the pro-inflammatory effects of tobacco on pancreatic microvasculature. However, the association was not statistically significant.

Despite a high complication rate in severe AP, the overall recovery rate was excellent: 100% in mild and moderately severe AP, and in severe AP, there were no deaths; two patients left against medical advice (LAMA). These outcomes are significantly better than those reported in previous studies conducted by Bhattarai et al. and Gautam et al. possibly due to early intervention, improved

supportive care, and close monitoring at our center.[9,11]

Out of all the clinical and biochemical variables assessed, only acute kidney injury (AKI) showed a statistically significant association with prolonged hospital stay (p = < 0.001). While this suggests that AKI may be an important prognostic factor in AP, the number of patients with AKI in our cohort was small (n = 3), limiting the strength of this conclusion. This finding is, however, consistent with previous studies by Negi et al. and Rao et al., which have demonstrated a clear link between AKI and worse outcomes in AP. Larger, multicenter studies are needed to confirm this association in our population.[2,13] Other factors such as high CRP, pleural effusion, ARDS, and peripancreatic fluid collection were not statistically significant, which could be attributed to sample size limitations

The severity of AP emerged as a strong determinant for the development of late complication (pseudocyst/ walled off necrosis). Among patients with moderately severe acute pancreatitis, 82.36% had resolution of PFC, while only 17.64% developed late complication. Whereas, in patients with severe acute pancreatitis 60% developed late complication. This supports the notion that local complications are more prevalent in severe acute pancreatitis due to greater pancreatic necrosis and inflammatory injury. Similar patterns have been reported in previous prospective studies, conducted by Lee et al. and Kochhar et al.[21,22]

Alcohol-related AP was associated with an 18.18% rate of late complication, while biliary pancreatitis showed a slightly higher rate (22.22%). These rates are consistent with findings by Lee et al. who noted that alcohol-induced AP tends to produce necrosis and delayed resolution of PFC.[21] Notably, in our cohort, the single patient with ERCP-induced AP and the single patient with hypertriglyceridemia-induced AP each developed late complications. Given the sample size of one for each etiology, these observations are anecdotal and cannot be generalized. However, they are in line with prior reports suggesting that such etiologies may be associated with more severe disease or persistent inflammation.

BISAP score ≥ 2 and hematocrit ≥ 44% were each associated with a 50% risk of late complication. Elevated hematocrit has been linked to hemoconcentration, which reflects early intravascular volume depletion and is a recognized predictor of pancreatic necrosis and local complications. 11 BMI ≥ 25 kg/m², was associated with a 36.36% risk of developing late complication in this study. Prior meta-analyses and clinical studies have identified obesity as a risk factor for both systemic complications and persistent local collections, likely due to increased peripancreatic fat and proinflammatory cytokine activity.[21] Additionally, acute kidney injury (AKI) was present in two patients, one of whom progressed to late complications. Although limited by small numbers, this supports findings from earlier studies indicating that AKI is a marker of systemic disease burden in AP and correlates with worse clinical outcomes.[23]

These findings suggest that severe AP, high BISAP scores, elevated hematocrit, obesity, and AKI should prompt clinicians to consider patients at higher risk of late complications. Early imaging follow-up and more aggressive supportive care may be warranted in such cases to reduce the likelihood of persistent fluid collections evolving into pseudocyst or WON.

Our study had few limitations. Firstly, this is a single-center study and the result may not be generalizable to general population and secondly, due to small number of severe patients, further larger studies are needed to further strengthen our results.

CONCLUSION

This study demonstrates that alcohol and biliary diseases are the primary causes of acute pancreatitis in our setting, with the majority of cases being mild to moderately severe. C-reactive protein (CRP) emerged as the most effective early marker for predicting severe disease. Although most patients recovered, complications such as ARDS and AKI significantly affected outcomes and hospitalization duration. AKI was the only parameter significantly associated with prolonged hospital stay. Furthermore, severe acute pancreatitis, high BISAP scores, elevated hematocrit, obesity, and AKI should prompt clinicians to consider patients at higher risk of late complications. Early imaging follow-up and more aggressive supportive care may be warranted in such cases to reduce the likelihood of persistent fluid collections evolving into pseudocyst or WON.

REFERENCES

1. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis 2012: revision of the Atlanta classification and definitions by international consensus. *Gut.* 2013;62(1):102–11. . Available from: https://doi.org/10.1136/gutjnl-2012-302779
2. Negi N, Mokta J, Sharma B, Sharma R, Jhobta A, Bodh V, et al. Clinical profile and outcome of acute pancreatitis: a hospital-based prospective observational study in Subhimalayan state. *J Assoc Physicians India*. 2018;66(3):22–4. Available from: PMID: 30341863.
3. Working Party of the British Society of Gastroenterology, Association of Surgeons of Great Britain and Ireland, Pancreatic Society of Great Britain and Ireland, Association of Upper GI Surgeons of Great Britain and Ireland. UK guidelines for the management of acute pancreatitis. Gut. 2005;54(Suppl 3):1–9. Available from: https://doi.org/10.1136/gut.2004.057026
4. Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol.* 2013;108(9):1400–15. Available from: https://doi.org/10.1038/ajg.2013.218

5. Frey CF, Zhou H, Harvey DJ, White RH. The incidence and case-fatality rates of acute biliary, alcoholic, and idiopathic pancreatitis in California, 1994–2001. *Pancreas*. 2006;33(4):336–44. Available from: https://doi.org/10.1097/01.MPA.0000236727.16370.99

6. Banks PA, Freeman ML, Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. *Am J Gastroenterol*. 2006;101(10):2379–400. Available from: https://doi.org/10.1111/j.1572-0241.2006.00856.x

7. Brown A, Orav J, Banks PA. Hemoconcentration is an early marker for organ failure and necrotizing pancreatitis. *Pancreas*. 2000;20(4):367–72. Available from: https://doi.org/10.1097/00006676-200005000-00005

8. Martínez J, Johnson CD, Sánchez-Payá J, de Madaria E, Robles-Díaz G, Pérez-Mateo M. Obesity is a definitive risk factor of severity and mortality in acute pancreatitis: an updated meta-analysis. *Pancreatology.* 2006;6(3):206–9. Available from: https://doi.org/10.1159/000092499

9. Bhattarai S, Gyawali M. Clinical profile and outcomes in patients with acute pancreatitis attending a teaching hospital at Gandaki Province, Nepal. *J Coll Med Sci Nepal.* 2020;16(3):175–80. Available from: https://doi.org/10.3126/jcmsn.v16i3.31299

10. Mukherjee D, Bhakta S, Lahiry S, Sinha R. Demographic profile of acute pancreatitis in Eastern India: a single centre experience. *Asian J Med Sci*. 2017;8(6):24–9. Available from: https://doi.org/10.3126/ajms.v8i6.17994

11. Gautam AK, Dewan KR, Shrestha R, KC V. Clinical profile and outcome of acute pancreatitis in a tertiary health care center of Nepal. *J Coll Med Sci Nepal.* 2023;19(3):313–9. Available from: https://doi.org/10.3126/jcmsn.v19i3.57808

12. Manandhar S, Giri S, Poudel P, Bhandari RS, Lakhey PJ, Vaidya P. Acute biliary pancreatitis: an experience in a tertiary level hospital of Nepal. *Indian J Surg.* 2013;75(6):449-53. Available from: https://doi.org/10.1007/s12262-012-0491-8

13. Rao BS, SreeVane M, Chandra VS. Etiology, clinical profile, severity and outcome of acute pancreatitis in relation to bedside index for severity of acute pancreatitis (BISAP) and CT severity index (CTSI) scores. *Int J Med Res Health Sci*. 2014;3(4):922–8. Available from: DOI: 10.5958/2319-5886.2014.00026.5

14. 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;21(2):118–23. Available from: https://doi.org/10.4103/aam.aam_86_21

15. Reid GP, Williams EW, Francis DK, Lee MG. Acute pancreatitis: a 7-year retrospective cohort study of the epidemiology, aetiology and outcome from a tertiary hospital in Jamaica. *Ann Med Surg (Lond)*. 2017;20:103–8. Available from: https://doi.org/10.1016/j.amsu.2017.06.039

16. Shah J, Rana SS. Acute respiratory distress syndrome in acute pancreatitis. *Indian J Gastroenterol.* 2020;39(2):123–32. Available from: https://doi.org/10.1007/s12664-020-01035-2

17. Gattinoni L, Caironi P, Valenza F, Carlesso E. The role of CT-scan studies for the diagnosis and therapy of acute respiratory distress syndrome. *Clin Chest Med.* 2006;27(4):559–70. Available from: https://doi.org/10.1016/j.ccm.2006.06.002

18. Raghuwanshi S, Gupta R, Vyas MM, Sharma R. CT evaluation of acute pancreatitis and its prognostic correlation with CT severity index. *J Clin Diagn Res.* 2016;10(6)–11. Available from: DOI: 10.7860/JCDR/2016/19849.7934

19. Vengadakrishnan K, Koushik AK. A study of the clinical profile of acute pancreatitis and its correlation with severity indices. *Int J Health Sci (Qassim).* 2015;9(4):410–7. Available from: https://doi.org/10.12816/0031233

20. Poudel R, Chandra K, Shah S, Mahasheth N, Mishra S, Paudel K. Prevalence of acute abdomen admission in surgery ward at tertiary care center of Nepal. *J Univ Coll Med Sci.* 2019;7(1):14–6. Available from: https://doi.org/10.3126/jucms.v7i1.24684

21. Lee DW, Kim HG, Cho CM, Jung MK, Heo J, Cho KB, et al. Natural course of early detected acute peripancreatic fluid collection in moderately severe or severe acute pancreatitis. *Medicina (Kaunas).* 2022;58(8):1131. Available from: https://doi.org/10.3390/medicina58081131

22. Kochhar R, Manrai M, Sidappa P, Medarapalem J, Yadav T, Khandelwal N, et al. Natural history of fluid collections following acute pancreatitis classified as per 2012 Atlanta revision. *Am J Gastroenterol.* 2013;108(Suppl 1). Available from: https://doi.org/10.14309/00000434-201310001-00246

23. Patra PS, Das K, Bhattacharyya A, Ray S, Hembram J, Sanyal S, et al. Natural resolution or intervention for fluid collections in acute severe pancreatitis. *Br J Surg*. 2014;101(13):1721–8. Available from: https://doi.org/10.1002/bjs.9665