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Clinical approach, diagnosis and medical management of acute pancreatitis among patients attending tertiary care hospital in Prakasam district, Andhra Pradesh, India

Nikhitha Konda¹, D. Venkateswarlu^{1*}, B. Thirumala Rao², V. Suresh¹, Aamani Shaik¹, D. Manasa³

¹Department of Gastroenterology, Vijaya Sree Hospitals Gastro and Liver Care Centre, Ongole, Andhra Pradesh, India ²Department of Community Medicine, Government Medical College, Ongole, Andhra Pradesh, India ³Department of Medical and Health, Primary Healthcare Centre, Maddipadu, Andhra Pradesh, India

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*Correspondence: Dr. D. Venkateswarlu, E-mail: devarakonda2013@gmail.com

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ABSTRACT

Background: There has been an increase in the incidence of acute pancreatitis reported globally and despite of improvements in access to care and interventional techniques, acute pancreatitis continues to be associated with significant morbidity and mortality. The present study was aimed to assess the clinical profile of acute pancreatitis and to assess the efficacy of various severity indices in view of outcome of patients.

Methods: A hospital based prospective cross-sectional study was conducted from October 2022-March 2023 in Gastro and Liver care center in Ongole, Prakasam District, Andhra Pradesh India. All consecutive 72 patients with a diagnosis of acute pancreatitis were included in this study.

Results: Out of total acute Pancreatitis cases 61 (84.7%) were males and 11 (15.3%) were females and acute abdominal pain (97.2%) and decreased appetite (95.8%) were the most common presenting complaints, 54.2% cases were due to Alcoholism, followed by hyperlipidemia with 20.8% and Gall stones 13.9%. All 72 (100%) received pancreatic supplements, 68 (94.4%) were given pain killers, and 65 (90.3%) were taken anti-ulcer agents. Twenty-three (31.9%) patients with 0 to 3 points as per CTSI Score and 4-6 range points were observed in 47 (65.3%) pancreatitis patients. Maximum (40.3%) were improved on 2^{nd} day, 22 (30.6%) were on 3^{rd} day. Positive correlation noticed between Amylase and in diagnosing acute Pancreatitis, it is significant at 0.05 level.

Conclusions: Early assessment of the clinical severity and identification of patients at risk is important for early intensive management and timely intervention and to improve quality of life. So, it is mandatory to assess the clinical severity using different scoring systems. and appropriate treatment based on guidelines.

Keywords: Acute pancreatitis, Clinical profile, Treatment, Outcome

INTRODUCTION

There has been an increase in the incidence of acute pancreatitis reported globally and despite of improvements in access to care, and interventional techniques, acute pancreatitis continues to be associated with significant morbidity and mortality. The most common causes of acute pancreatitis are gallstones and over alcohol consumption.¹ The availability of new imaging modalities has changed clinical practice and availability of guidelines, recent studies auditing clinical management of acute pancreatitis have shown

improvement in noncompliance with evidence-based recommendations.^{2,3}

Acute pancreatitis (AP) is a common clinical problem in gastrointestinal practice and it is diagnosed in the presence of acute onset of upper abdominal pain, elevated amylase and/or lipase levels, and imaging evidence of pancreatic and peripancreatic inflammation. Its incidence is increasing over the past 2 decades and varies from 30 to 80 per 100,000 population.^{4,5} Acute interstitial pancreatitis is seen in 70-80% of patients, in contrast, acute necrotizing pancreatitis, a severe form of the disease is present in 20-30% of patients and associated with a mortality rate of up to 40%.⁶

The cause of Acute Pancreatitis (AP) is evident after standard investigations in about 70%-80% of patients during or after the first attack. Gallstones are the cause of AP in about 45%, alcohol intake in 20%-25%, and miscellaneous in about 5% of cases.⁷ The cause is not evident in 20%-25% of patients after standard evaluation and such patients are labelled as having idiopathic AP. The aetiology of recurrent acute Pancreatitis (RAP) in the idiopathic group are not clear from these studies as the commonest causes of RAP in these series were alcohol intake and gallstones.⁸ About two-thirds of AP patients have a mild course of disease with a quick recovery. One third experience disease progression, with the development of local complications.⁹ The development of local complications (collections, necrosis) is linked to fluid sequestration during the early phase but, most importantly, has consequences in the late phase, in which those local complications can be associated with symptoms and infection.

Clinical diagnosis of acute pancreatitis is based on patient symptoms, physical examination, laboratory analysis, and radio-logical data. According to practice guidelines published in 2006, a diagnosis of acute pancreatitis requires two out of three main features: first one is abdominal pain typical for acute pancreatitis, second one is serum amylase and/or lipase greater than or equal to three times the upper normal limit; and third is evidence of acute pancreatitis on computed tomography (CT) scans.

Almost all patients with acute pancreatitis have acute upper abdominal pain at onset and confined to the midepigastrium or may be diffuse throughout the abdomen.¹⁰ According to the severity, acute pancreatitis is divided into mild acute pancreatitis (absence of organ failure and local or systemic complications), moderately severe acute pancreatitis (no organ failure or transient organ failure less than 48 hours with or without local complications) and severe acute pancreatitis (persistent organ failure more than 48 hours that may involve one or multiple organs).^{11,12}

When gallstones from the gallbladder, pass and obstruct ducts of the pancreas, they develop choledocholithiasis or

cholangitis¹³ Since most patients are asymptomatic, diagnosis for Pancreatitis includes a combination of clinical history, physical examination, serum biochemical analysis and imaging of pancreas and gallbladder.^{14,15} The rising costs of intensive care management and the need to prolong the life of critically ill patients creates a need for early identification of those patients who will benefit from intensive care. The present study was aimed to assess the clinical profile of acute pancreatitis and to assess the efficacy of various severity indices in view of outcome of patients.

METHODS

A hospital based prospective cross-sectional study was conducted from October 2022-March 2023 in Gastro and Liver care center in Ongole, Prakasam District, Andhra Pradesh India. All consecutive 72 patients with a diagnosis of acute pancreatitis were included in this study. Patients with Acute gallbladder pancreatitis, common bile duct stones; traumatic, idiopathic were included in the study. Diagnosis for acute gallbladder pancreatitis was made based on abdominal pain similar to AP, three times or more elevated serum levels of pancreatic amylase and or lipase and finally radiographic diagnosis using abdominal Computed Tomography (CT) or Abdominal Ultrasound (AUS) images. Patients presenting with chronic pancreatitis, pancreatic malignancy; pseudocysts, acute fluid collections, necrotizing pancreatitis, walled-off necrosis; AP in pediatric patients; pregnancy were excluded from the study.

Hospital ethics committee approval and informed and written consent by the patient were obtained before study. Demographic, undertaking the clinical, biochemical and radiographic data was prospectively collected. 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 and complete hemogram. All patients underwent abdominal ultrasonography at admission and contrast enhanced pancreatic protocol CT scan 72 hours after onset of symptoms. Patients were classified into mild, moderate and severe acute pancreatitis based on Ranson's score Glasgow scoring and the BISAP (Bedside Index for Severity in Acute Pancreatitis) score.¹⁶⁻¹⁷

The BISAP score provides a single point for each of five pa-rameters: BUN >25mg/dL, impaired mental status, systemic inflammatory response syndrome (SIRS), age >60 years, and/or the presence of a pleural effusion, for a possible total of five points. A BISAP score greater than three is associated with a seven- to 12-fold increase in the risk of developing organ failure.¹⁸ Hemo-concentration, indicated by an admission hematocrit of 47%, and subsequent failure of the hematocrit to decrease by 24 hours are risk factors for the development of pancreatic necrosis.¹⁹ Older age (55 years) and a body mass index (BMI) 30 are also known risk factors for more severe forms of pancreatitis. In particular, obesity is associated with increased risk of developing both systemic and local complications.²⁰

The revised Atlanta classification of acute pancreatitis established in 2008 identifies two phases of the disease: early and late. Severity is classified as mild, moderate, or severe. Mild acute pancreatitis, the most common form, has no organ failure or local or systemic complications and usually resolves in the first week. Moderately severe acute pancreatitis is defined by the presence of transient organ failure, local complications, or exacerbation of co-morbid disease. Severe acute pancreatitis is defined by persistent organ failure lasting longer than 48 hours. Patients with mild pancreatitis were managed in the ward, and severe pancreatitis were admitted in ICU. Severity of the disease was evaluated in terms of ICU admission, Improvement with management, final grade as per Atlanta 2012 classification. Data were collected prospectively in a Microsoft Excel Database. After completion of data collection, the database was imported into SPSS software version 20.0. Categorical variables were expressed as absolute numbers and proportions. A P-value of <0.05 was considered statistically significant. Correlation will used to find association between different relevant factors.

RESULTS

Out of total 72 acute pancreatitis cases 61 (84.7%) were males and 11 (15.3%) were females, in terms of age group two (2.8%) below 25 years, 42 (58.3%) were between 25 to 44 years age group, 24 (33.3%) were in between 45 to 64 years age and 4 (5.6%) were 65 and above age group. It was observed that 8 (11.1%) were illiterate, 18 (25%) were completed primary education, 25 (34.7%) were studied up to 10^{th} class, 5 (6.9%) educated up to intermediate, 14 (19.4%) were graduates and only 2 (2.8%) were professionals. It was noticed that 23 (31.9%) patients with occupation of business, 12 (16.7%) were farmers, 7 (9.7%) house wives, 13 (18.1%) were software professionals and others were 18 (18.1%) (Table 1).

It was observed that acute abdominal pain and decreased appetite were the most common presenting clinical features and these were seen among 70 (9.2%) and 69 (95.8%) respectively. Vomiting was noticed in 26 (36.1%) patients, Weight loss was observed in 25(34.7%) cases, and restlessness was seen in 9 (12.5%) cases. Bilious vomiting was also one of the charactered features in acute pancreatitis and it was seen in 11 (15.3%) of cases. Fever, loose motions, irritability and rash symptoms were seen in 7(9.7%), 6 (8.3%), 3 (4.2%) and 1 (1.4%) respectively. It was also observed that abdominal pain in acute pancreatitis was improved with bending forward position seen in 55 (76.4%) cases (Table 2).

Table 1: Socio-demographic factors among pancreatitis cases.

Details	Number	Percentage
Gender		
Males	61	84.7
Females	11	15.3
Age group		
<25	2	2.8
25 to 44	42	58.3
45 to 64	24	33.3
65 and above	4	5.6
Education		
Illiterate	8	11.1
Primary	18	25.0
Secondary	25	34.7
Inter	5	6.9
Degree	14	19.4
Professional	2	2.8
Occupation		
Business	23	31.9
Farmer	12	16.7
House wife	7	9.7
Soft ware	13	18.1
Others	18	25.0
Doctors	1	1.4

Table 2: Distribution of clinical features in acute pancreatitis patients.

Symptoms	Number	Percentage
Acute abdominal pain	70	97.2
Vomiting	26	36.1
Bilious vomiting	11	15.3
Appetite decreased	69	95.8
Weight loss	25	34.7
Fever	7	9.7
Loose motions	6	8.3
Restless	9	12.5
Irritable	3	4.2
Improvement with	55	76.4
bending forward		
Rash	1	1.4

It was observed that out of total 72 cases 39 (54.2%) cases were due to Alcoholism among them 27 (69.2%) cases were between 25 to 44 years age group, 10 (25.6%) were between 45 to 64 years age group. Second most common was found to be hyperlipidemia among 15 (20.8%) cases and gall stones found to be third most common cause in 10 (13.9%) cases followed by idiopathic seen in 8 (11.1%) cases (Table 3).

It was observed that out of 72 cases all 72 (100%) received pancreatic supplements, 68 (94.4%) were given pain killers, and 65 (90.3%) were taken anti-ulcer agents. Vitamin supplements were given to 46 (63.9%) cases

antipyretics were given to 8 (11.1%) and diuretics were given to 7 (9.7%) patients. Antibiotics were also prescribed to these cases levofloxacin was prescribed to 22 (30.6%) cases, followed by ceftriaxone and

tazobactam combination was given 20 (27.8%) patients. Imipenem antibiotic was used in 18 (25.5%) cases, followed by piperacillin and tazobactam in 7 (9.7%) cases then metronidazole in 5 (6.9%) cases (Table 4).

Table 3: Etiology spectrum of acute pancreatitis cases.

Age	Alcohol	Gall stones	Hyperlipidemia	Idiopathic	Total
<25	1	1	0	0	2
25 to 44	27	4	8	3	42
45 to 64	10	4	7	3	24
65 and above	1	1	0	2	4
Total	39	10	15	8	72
%	54.2	13.9	20.8	11.1	100

Table 4: Medical management of acute pancreatitis cases.

Drugs	N=72	%
Antibiotics		
Ceftriaxone and tazobactam	20	27.8
Imipenem	18	25.0
Levofloxacin	22	30.6
Metronidazole	5	6.9
Piperacillin and tazobactam	7	9.7
Pancreatic supplements	72	100.0
Antipyretics	8	11.1
Analgesics	68	94.4
Vitamin supplements	46	63.9
Diuretics	7	9.7
Anti-ulcer agents	65	90.3
Vitamin K supplements	4	5.6

As per the Ranson's criteria, 53 (73.6%) acute pancreatitis patients were with points between 0-2, 19

(26.4%) were with points from 3 to 5 and none of them having score more than 5. Whereas per Glasgow score mild pancreatitis cases with below <3 points were 46 (63.9%), severe cases were 3 and above were found in 36 (36.1%) patients. Twenty-three (31.9%) patients with 0 to 3 points as per CTSI Score and 4-6 range points were observed in 47 (65.3%) pancreatitis patients, none of the patients fall in the range of 7 to 10 points (Table 5).

It was also observed that 32 (44.4%) were with normal BMI, pre obese with 25 to 29.9 were 31(43.1%), and patients with obesity more than 30 were 8 (11.1%) patients and out of 72 cases 11 (15.3%) were suffered with diabetes and 18 (25.0%) with hypertension. as per revised Atlanta grading mild cases were 38 (52.8%), Moderate were 19 (26.4%) and severe cases were 15 (20.8%), out of total cases 48 (66.7%) without any organic failure, 22 (30.5%) patients were with transient organic failure, 2(2.7%) were with persistent organic failure and none of them landed up in multi organic failure (Table 6).

Table 5: Ranson's criteria, glasgow coma scale and CTSI score among acute pancreatitis patients.

Ranson's Criteria	Number	Percentage	Glasgow score	Number	Percentage	CTSI score	Number	Percentage
0 to 2	53	73.6	Mild (<3)	46	63.9	0 to 3	23	31.9
3 to 5	19	26.4	Severe (3 and above)	26	36.1	4 to 6	47	65.3
>5	0	0.0			-	7 to 10	0	0.0

It was observed that 3 (4.2%) cases were improved on first day, maximum number 29 (40.3%) were on second day, 22 (30.6%) were on 3^{rd} day, 15 (20.8%) improved on 4^{th} day and 3 (4.2%) on 5^{th} day (Figure 1).

The ultra sonographic findings revealed that bulky pancreas in 27 (37.5%) cases, fatty liver in 15 (20.8%), ascites in 13 (18.1%) altered echo texture, gall bladder stones and elongated pancreas observed in 4 (5.6%) cases (Figure 2).

Amylase levels elevation observed in acute pancreatitis the patients that out of 72, 14 (19.4%) ranging from 200-450, 21 (29.2%) ranging from 451-900, 22 (30.6%) above 900, 15 (20.8%) of patients were below 200 IU/L. serum lipase levels elevation i.e. majority of the patients 33(45.8%) with above 900 IU, 18 (25%) ranging from 451-900 16 (22.2%) were within the range of 160-200 IU/L and 5 (6.9%) within the range of 200 to 450 (Table 7).

Table 6: BMI, comorbid status of Covid patients, revised Atlanta grading-12 of pancreatic patients.

BMI	Number	Percentage
<18.5	1	1.4
18.5 to 24.9	32	44.4
25 to 29.9	31	43.1
30 to 34.9	7	9.7
> 35	1	1.4
Comorbid	Number	Percentage
Diabetes	11	15.3
Hypertension	18	25.0
Not comorbid	43	59.7
Revised Atlanta grading 2012	Number	Percentage
Mild	38	52.8
Moderate	19	26.4
Severe	15	20.8
No organic failure	48	66.7
Transient organic failure	22	30.5
Persistent organic failure	2	2.7
Multi organic failure	0	0.0









Table 7: Amylase and lipase levels in pancreatic
patients.

Amyla se levels	Number	%	Lipase levels	Number	%
<200	15	20.8	<200	16	22.2
200-450	14	19.4	200-450	5	6.9
451-900	21	29.2	451-900	18	25.0
>900	22	30.6	>900	33	45.8

Table 8: Pre and post assessment of amylase and
lipase levels.

Parameters	Mean	SD	P values
Amylase (pre)	798.55	98.64	$\mathbf{D} < 0.01$
Amylase (post)	138.64	3.34	P< 0.01)
Lipase (pre)	1090.5	146.47	$\mathbf{D} < 0.01$
Lipase (post)	150.65	5.68	P< 0.01)

Statistically significant association find in in reduction of amylase and lipase levels with treatment in pre and post assessment of enzyme levels statistical analysis details of patients with acute pancreatitis (Table 8).

Positive correlation noticed between amylase and lipase with 0.8326, in diagnosing acute pancreatitis, it is significant at 0.05 level (Table 9).

Table 9: Correlation between amylase and lipase.

		Amylase	Lipase
Amylase	Pearson correlation coefficient (Sig) two tailed	1 71	0.8326* 0.567 72

* Correlation is significant at the 0.05 level (2-tailed)

DISCUSSION

Pancreatitis is a contributing factor in causing an additional deaths every year in various countries and rising a heavy economic burden, and health costs annually in the United States.²² Unpredictable diagnostic delay often leads to belated and ineffective interventions and practice of guidelines for standardization of management of the patient, will in turn give better outcomes. In this study out of total 72 acute pancreatitis cases 61 (84.7%) were males and 11 (15.3%) were females, and the male preponderance of 2.7:1 seen in Ramu et al study and similar findings noticed in Jamaican study where a male preponderance was 4:1.^{22,23} A study conducted in Andhra Pradesh on acute pancreatitis revealed that 95% patients were found to be males and 5% were females.²⁴

In our study it was found that 39 (54.2%) cases were due to Alcoholism, second most common was found to be hyperlipidemia among 15 (20.8%) cases, followed by gall stones in 10 (13.9%) cases even though the etiology of acute pancreatitis varies alcohol consumption and gall stones predominate in most of the countries. As per one of the South Indian Study out of the total 436 cases, alcohol induced pancreatitis was higher (42.431%) followed by idiopathic pancreatitis (36.9%).²² Our study results are consistent with the findings of Vengadakrishnan et al, from the study done at SRM Medical College, Chennai were alcohol induced pancreatitis was higher (51%).²⁵

As per Casas et al, in their study of 148 patients, found gall stones (57%), alcohol consumption (21%) are the causes of acute pancreatitis which is in contrast to present study.²⁶ Similar results were obtained in Victorian study.²⁷ This can be explained by the greater incidence of alcohol intake in the Indian population. A study done in Atlanta found that gall stone disease (92.6%) followed by traumatic (3.7%) were the most common etiology spectrum in our study. There are many possible underlying causes of acute or sudden onset pancreatitis, but 60 to 75 percent of all cases are caused by gallstones.²⁸

It was observed in our study that acute abdominal pain (97.2%) and decreased appetite (95.8%) were the most common presenting clinical features, vomiting was noticed in 26 (36.1%) patients, abdominal pain is the cardinal symptom of acute pancreatitis.¹⁴ It was observed in Ramu et al study that triad of epigastric pain, nausea and vomiting was present in 256 (58.5%) of cases.²² In the study done by Sameer et al in People's college of medical sciences and research center, Bhopal the triad of epigastric pain, nausea and vomiting was seen in 75% patients.²⁹

Abdominal pain was present in all cases which were in concordance with the study done by Reid et al where abdominal pain was present in 96.7% cases.³⁰ Nausea and vomiting was present in 70.2% cases. Epigastric tenderness is found in almost all patients. This illustrate that most typically presenting symptoms are abdominal pain, vomiting and also other GI symptoms which was supported by Harindranath et al.^{31.} Majority of patients had abdominal pain (96.3%) which is mostly radiating (93.8) a most common symptom of gall stone related acute pancreatitis.³²

It was observed in our study that 44.4% were with normal BMI, pre obese were 43.1%, and Obesity more than 30 were 11.1%, as per Manrai et al study majority (66.7%) of patient had BMI between 30-34.³³ From Erlinger and Stender et al it is evident that increased body mass index (BMI) increases the risk of gallstone formation.^{34,35} In our study it was noticed that 15.3% were suffered with diabetes and 25.0% with hypertension, though studies have reported that gallstone disease is related to several diabetes risk factors, there is no proof that diabetic patients have more gallstones.³⁶ However, in Ikard et al study, it was found that 43.2%

patient suffered with diabetes mellitus as comorbid condition. This could be due to fact that about 50.9 million people suffer from diabetes.³⁷

Lipase has a higher diagnostic accuracy compared to amylase as the serum lipase levels are elevated for a longer period.³⁸ Caution should be exercised when amylase results interpreting in patients with hypertriglyceridemia as they can have a falsely low amylase result. In our study it was found that amylase levels elevation observed in acute pancreatitis 19.4% ranging from 200-450, 29.2% with 451-900, and 30.6% above 900. As per Reddy et al levels elevation is as follows majority of the patients 53.3% ranging from 200-450, and 36.7% with 450-900, and these study results were supported by Matull et al.^{24,39} In our study we also found that serum lipase levels elevation i.e. majority of the patients 45.8% were above 900, 25 % were in 450 to 900 range. This also indicates over activation of the lipase enzyme within the acinar cells and causing auto digestion of pancreas which was supported by Esmaili et al.⁴⁰ Early diagnosis and prompt treatment is the main stay of therapy in AP for significantly decreasing morbidity and mortality which was supported by Ahlawat et al.⁴¹ The ultra sonographic findings revealed that bulky pancreas in 37.5% cases, fatty liver in 20.8%, ascites in 18.1%, altered echo texture, gall bladder stones and elongated pancreas observed in (5.6%) cases. This denotes the patients with bulky pancreas, altered echo texture is leading when collate to other findings, it shows these two finding are commonly present in AP which can be diagnosed by ultrasonography with high sensitivity which was supported by Shah et al.42

In our study it was found that 31.9% patients with 0 to 3 points as per CTSI score and 4-6 range points were observed in 65.3% pancreatitis patients, none of the patients fall in the range of 7 to 10 points. As per one South Indian Study. It was found that 70% patients were mild and 30% patients were moderate, no patients with severe acute pancreatitis were admitted to the hospital during our study period.²⁴ This indicates the patients with no organ failure is more when collate to the patients with the complications which was supported by Pongprasobchai et al.43 Similar results were obtained in the study done in North India by Ahlawat et al where 82% were mild cases.44

CONCLUSION

Although a small number of patients analyzed in this single center study, to our knowledge this work provides the first known regional description of the etiology, clinical profile and outcome of acute pancreatitis. Early assessment of the clinical severity and identification of patients at risk is important for early intensive therapy and timely intervention and to improve quality of life. So, it is mandatory to assess the clinical severity using different scoring systems. Safe and effective management of acute pancreatitis patients there is need to evaluate cause, clinical severity, and appropriate treatment based on guidelines. As per our study incidence of alcoholic pancreatitis was higher and it can be can be explained by the greater incidence of alcohol abuse in India. Lifestyle modification and public education are recommended for prevention.

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REFERENCES

- 1. Banks PA. Epidemiology, natural history, and predictors of disease outcome in acute and chronic pancreatitis. Gastrointest Endosc. 2002;56(Suppl):S226-30.
- Mofidi R, Madhavan KK, Garden OJ, Parks RW. An audit of the manage¬ment of patients with acute pancreatitis against national standards of practice. Br J Surg. 2007;94(7):844-8.
- 3. Pezzilli R, Uomo G, Gabbrielli A, Zerbi A, Frulloni L, De Rai P, et al. A prospective multicentre survey on the treatment of acute pancreatitis in Italy. Dig Liver Dis 2007;39(9):838-46.
- 4. Spanier BW, Dijkgraaf MG, Bruno MJ. Epidemiology, aetiology and outcome of acute and chronic pancreatitis: an update. Best Pract Res Clin Gastroenterol. 2008;22(1):45-63.
- 5. Shen HN, Lu CL, Li CY. Epidemiology of firstattack acute pancreatitis in Taiwan from 2000 through 2009: a nationwide population-based study. Pancreas. 2012;41(5):696-702.
- 6. Forsmark CE, Baillie J, AGA Institute Clinical Practice and Economics Committee; AGA Institute Governing Board. AGA Institute technical review on acute pancreatitis. Gastroent. 2007;132:2022-44.
- 7. Sharma, M, Banerjee D, Garg PK. Characterization of newer subgroups of fulminant and sub fulminant pancreatitis associated with a high early mortality. Am J Gastroenterol. 2007;102(12):2688-95.
- Gao YJ, Li YQ, Wang Q, Li SL, Li GQ, Ma J, et al. Analysis of the clinical features of recurrent acute pancreatitis in China. J Gastroenterol. 2006;41:681-5.
- 9. 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.
- Banks PA, Freeman ML. Practice guidelines in acute pancreatitis. Am J Gastro-enterol. 2006;101(10):2379-400.

- 11. Forsmark CE, Baillie J, AGA Institute Clinical Practice and Economics Committee, AGA Institute Governing Board. AGA Institute technical review on acute pancreatitis. Gastroent. 2007;72(3):257-81.
- Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol. 2013;108(9):1400.
- 13. Kimura Y, Arata S, Takada T, Hirata K, Yoshida M, Mayumi T, et al. Gallstone-induced acute pancreatitis. J Hepat Pancreat Sci. 2010;17(1):60-9.
- 14. Cappell MS. Acute pancreatitis: etiology, clinical presentation, diagnosis, and therapy. Med Clin North Am. 2008;92(4):889-923.
- 15. Lippi G, Valentino M, Cervellin G. Laboratory diagnosis of acute pancreatitis: in search of the Holy Grail. Crit Rev Clin Lab Sci. 2012;49(1):18-31.
- Ranson JH, Rifind KM, Roses DF, Fink SD, Eng K, Spencer FC. Prognostic signs and the role of operative management in acute pancreatitis. Surg Gynecol Obstet. 1974;139:69-81.
- 17. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med. 1985;13(10):818-29.
- Singh VK, Wu BU, Bollen TL, Repas K, Maurer R, Johannes RS, et al. A pro-spective evaluation of the bedside index for severity in acute pancreatitis score in assessing mortality and intermediate markers of severity in acute pancrea- titis. Am J Gastroenterol. 2009;104(4):966-71.
- 19. Baillargeon JD, Orav J, Ramagopal V, Tenner SM, Banks PA. Hemoconcentration as an early risk factor for necrotizing pancreatitis. Am J Gastroenterol. 1998;93(11): 2130-4.
- 20. Martinez J, Sanchez-Paya J, Palazon JM, Suazo-Barahona J, Robles-Diaz G, Perez-Mateo M. Is obesity a risk factor in acute pancreatitis? A metaanalysis. Pan- creatology. 2004;4(1):42-8.
- Peery AF, Dellon ES, Lund J, Crockett SD, McGowan CE, Bulsiewicz WJ. Burden of gastrointestinal disease in the United States: 2012 update. Gastroenterology. 2012;143(5):1179-87.
- 22. Ramu R, Paul V, Devipriya S, Philip NC. Etiology, clinical profile and outcome of acute pancreatitis in a tertiary care teaching hospital in rural South India: a ten-year retrospective study. Int Surg J 2019;6(10):3794-9.
- 23. Lee MG, Chung A, Miles A, Terry SI, Royes CA. Chronic pancreatitis in Jamaica. West Indian Med J. 1992;41(2):61-3.
- 24. Reddy MS, Ramana PV, Bhavani C, Lakshmi RP, Khizar SM. A study on etiology, severity, management and outcome of acute pancreatitis in tertiary care teaching hospital. Inter J Res Rev. 2020;7(1):534-44.
- 25. 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.

- Casas JD, Díaz R, Valderas G, Mariscal A, Cuadras P. Prognostic value of CT in the early assessment of patients with acute pancreatitis. Am J Roentgenol. 2004;182(3):569-74.
- 27. Barreto SG, Tiong L, Williams R. Drug-induced acute pancreatitis in a cohort of 328 patients. A single-centre experience from Australia. JOP J Pancreas. 2011;12(6):581-5.
- Lerch MM, Aghdassi A. Gallstone-related pathogenesis of acute pancreatitis. Pancreapedia Exocrine Pancreas Knowledge Base. 2016; Available at: https://www.pancreapedia.org/reviews/gallstone-related-pathogenesis-of-acute-pancreatitis. Accessed 3 November 2018.
- 29. Raghuwanshi S, Gupta R, Vyas MM, Sharma R. CT Evaluation of Acute Pancreatitis and its Prognostic Correlation with CT Severity Index. J Clin Diagnos Res. 2016;10(6):TC06-11.
- 30. 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.
- Harindranath RH, Narendranath L. A clinical study on acute pancreatitis and its different etiologies in Bowring & Lady Curzon hospitals, Bengaluru. J Evolution Med Dent Sci. 2016;5(3):196-200.
- 32. Ghatak R, Masso L, Kapadia D, Kulairi ZI. Medication as a cause of acute pancreatitis. Am J Case Rep. 2017;18:839-41.
- Manrai M, Kochhar RK, Thandassery RB, Alfadda AA, Sinha SK. The revised Atlanta classification of acute pancreatitis: a work still in progress ? JOP. 2015;16:356-64.
- 34. Erlinger S. Gallstones in obesity and weight loss. Eur J Gastroenterol Hepatol. 2000;12(12):1347-52.
- 35. Stender S, Nordestgaard BG, Tybjaerg-Hansen A. Elevated body mass index as a causal risk factor for symptomatic gallstone disease: A Mendelian randomization study. Hepatol. 2013;58(6):2133–41.
- 36. Ikard RW. Gallstones, cholecystitis and diabetes. Surg Gynecol Obstet. 1990;171(6):528-32.

- Diabetes Foundation India. Diabetes Foundation (India) 2019. Available at: http://www.diabetesfoundation.in. Accessed on 25 April 2023.
- Matull WR, Pereira SP, O'Donohue JW. Biochemical markers of acute pancreatitis. J Clin Pathol. 2006;59(4):340-4.
- 39. Matull WR, Pereira SP, O'donohue JW. Biochemical markers of acute pancreatitis. J Clin Pathol. 2006;59(4):340-4.
- 40. Esmaili HA, Mehramuz B, Maroufi P, Ghasemi A, Pourlak T. Diagnostic value of amylase and lipase in diagnosis of acute pancreatitis. Biomed Pharmacol J. 2017;10(1):389-94.
- 41. Ahlawat V, Godara R. Clinical study of demographic profile, etiology, severity and outcome of acute pancreatitis in a tertiary care teaching hospital in Northern India. J Gastrointest Dig Syst. 2018;8:575.
- 42. Shah AP, Mourad MM, Bramhall SR. Acute pancreatitis: current perspectives on diagnosis and management. Inflammat Res. 2018;11:77-85.
- 43. Pongprasobchai S, Vibhatavata P, Apisarnthanarak P. Severity, treatment, and outcome of acute pancreatitis in Thailand: the first comprehensive review using revised Atlanta classification. Gastroenterology research and practice. 2017;17(1):2017.
- 44. Ahlawat V, Godara R. Clinical study of demographic profile, etiology, severity and outcome of acute pancreatitis in a tertiary care teaching hospital in Northern India. J Gastrointest Dig Syst. 2018;8:575.

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