Journal of Cardiovascular Disease Research

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

A COMPARATIVE STUDY OF GALL STONE PANCREATITIS AND ALCOHOLIC PANCREATITIS IN RELATION TO DIFFERENT BIOCHEMICAL MARKERS

Dr. Nirmali Barua[1], Dr. Bismita Doley[1], Prof. Dr. Bijoyananda Das[2], Prof. Dr. Jishan Ahmed[3]

[1]Senior Resident (Department of General Surgery), Lakhimpur Medical College and Hospital

[1]PGT (Department of General Surgery), Assam Medical College and Hospital

[2]Professor (Department of General Surgery), Assam Medical College and Hospital

[3]Professor (Department of General Surgery), Assam Medical College and Hospital

Corresponding author: Prof. Dr. Bijoyananda Das, Professor (Department of General Surgery), Assam Medical College and Hospital, Dibrugarh, Assam

Email id- bijoyananda4321@gmail.com , Ph no.- 6001188218

ORCID ID- 0009000665279526

Abstract:

Acute Pancreatitis (AP) is a common condition with an overall mortality rate of 6-15%. Internationally, gall stones and alcohol are the most common causes of pancreatitis. Amylase estimation is technically simple, inexpensive, and sensitive but it has low specificity. Lipase has a greater sensitivity in acute alcoholic pancreatitis and in patients who present late to the emergency room and remains elevated longer than amylase. The presence of elevated liver enzymes usually indicates liver damage. Alanine Transaminase (ALT) is more specific to the liver than Aspartate Transaminase (AST). The present study was conducted to see whether changes in specific biochemical markers are significant enough to act as a diagnostic tool to differentiate between alcoholic pancreatitis and gallstone pancreatitis. Thorough history (especially of alcohol abuse, >4-5 drinks/day) and clinical examination were recorded. Severity grading of all patients was done based on BISAP scoring. Abdominal ultrasound was used for screening for gall stones, common bile duct stones and pancreatitis. Patients diagnosed with gall stone on abdominal ultrasound were tested for liver and pancreatic enzyme levels. A total of 140 cases (70 of each group) was analyzed in this study. There was no statistically significant co-relation of serum amylase, lipase, AST, ALT, ALKP with the severity of pancreatitis. However, a direct co-relation was seen between raised

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

TLC, BUN and severity of pancreatitis. The average serum amylase level was significantly higher for gallstone pancreatitis as compared to alcoholic pancreatitis at presentation. The serum AST, serum ALT, serum bilirubin and serum ALKP levels in gallstone pancreatitis were higher as compared to alcoholic pancreatitis**.** However, a direct co-relation was seen between raised TLC, BUN, and severity of pancreatitis. Raised biochemical markers like AST, ALT, amylase, lipase, ALP and serum bilirubin can be used as good markers for diagnosis of gallstone pancreatitis. However, a normal or low serum level of these markers does not rule out possibility of gallstones in the aetiology of pancreatitis and may require further evaluation.

Introduction

Acute Pancreatitis (AP) is a common condition with an overall mortality rate of 6-15%. It is defined as "an inflammatory process of pancreas with possible peripancreatic tissue involvement and multi-organ dysfunction with increasing mortality rate".[1] Internationally, gall stones and alcohol are the most common causes of pancreatitis.[2] The incidence of gallstone pancreatitis is increased among patients with small gallstones (less than 5 mm in diameter) or microlithiasis.[3] Excessive alcohol use as a cause of pancreatitis is more common among men than women. The link between alcohol and acute pancreatitis is complicated, but it appears to be dose-dependent. Other causes of AP include metabolic abnormalities (e.g., hypertriglyceridemia), duct obstruction (e.g., from a tumour or pancreas divisum), medications (e.g., azathioprine, thiazides, and estrogens), and trauma.[4] Pancreatitis caused by gall stone disease is caused by transient blockage of the ampulla of Vater by a migrating stone. However, it has been seen that only 4 – 8 % of patients with cholelithiasis develop acute pancreatitis. This shows that there must be other factors responsible for predisposing a patient to gallstone pancreatitis.[3] There are various theories for the mechanism of pancreatitis–Refluxed bile damages the pancreatic duct mucosal barrier, high pancreatic duct pressure secondary to obstruction at ampulla causes direct damage to the epithelium, reflux of duodenal contents with bile salts and enterokinase into pancreatic duct may lead to premature activation of enzymes.[3] Activation of the enzymes by co-localisation of zymogen granules and lysosomes results in auto-digestion of pancreas. In response to the initial insult, acinar cells release proinflammatory cytokines, such as TNF a, IL-1, IL-2 and IL-6, and anti-inflammatory mediators such as IL-10 and IL-1 receptor antagonists.[5] Serum amylase and lipase continue to be important tests in the diagnosis of AP. Amylase estimation is technically simple,

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

inexpensive, and sensitive. However, its main disadvantage is its lack of specificity. Patients with AP secondary to hyperlipidaemia, acute exacerbation of chronic pancreatitis, or delayed estimation of amylase in the course of the disease may present with normal amylase levels. Lipase has a greater sensitivity in acute alcoholic pancreatitis and in patients who present late to the emergency room, usually days after the onset of the disease, because it remains elevated longer than amylase.[6] Liver produces transaminases to synthesize/ breakdown amino acids for storage and energy production. Though normal blood levels of these enzymes are low, if a hepatocyte is damaged, the cell membrane becomes more permeable, and some enzymes leak out into the blood stream. Thus, the presence of elevated liver enzymes usually indicates liver damage. Alanine Transaminase (ALT) is more specific to the liver, whereas Aspartate Transaminase (AST) is also found in significant amounts in heart and muscle cells and thus, not specific. Most diseases cause ALT to rise more than AST. However, AST levels may double or triple compared to ALT in alcoholic liver diseases.

Aim and Objectives

The aim of the present study is to assess the changes in specific biochemical markers (AST, ALT, amylase, lipase) in alcoholic pancreatitis and gallstone pancreatitis and draw a comparison between them. The objectives of the study are:

- To assess the liver enzymes and pancreatic enzymes in alcoholic pancreatitis and in gallstone pancreatitis.
- To study whether the here is a difference in level of liver enzymes and pancreatic enzymes between alcoholic and gallstone pancreatitis and its relation to severity

Materials and Methods

This study was done in the Department of General Surgery, Assam Medical College and Hospital from 1st June 2022 to 31st May 2023. This is a Hospital based cross sectional study. Patients diagnosed with pancreatitis by clinical, biochemical and radiological methods who were hospitalized or attended Out Patient Department and Casualty of The Department of Surgery and The Department of Medicine, Assam Medical College and Hospital. All cases of Gall stone pancreatitis and Alcoholic pancreatitis meeting the inclusion criteria were taken into the study. A

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

written and informed consent was taken from the participants for conducting the study.

Inclusion Criteria:

All cases of more than 12 years of age admitted to the Department of Surgery and Department of Internal Medicine, Assam Medical College, Dibrugarh (Assam) with pancreatitis as provisional clinical diagnosis and confirmed by abdominal ultrasound.

Exclusion Criteria:

- Children (age < 12 years)
- Any patient with gallstone pancreatitis with a history of chronic alcoholism.
- Any patient with a history of intake of anti-tubercular drugs or hepatotoxic drugs
- Any patient with chronic liver disease or primary /secondary hepatic or pancreatic carcinoma
- Patients with a history of hepatobiliary or pancreatic surgery
- Patients not giving consent for inclusion in the study.

Methodology

Patients meeting the study criteria were enrolled in the study. Thorough history (especially of alcohol abuse, >4-5 drinks/day) and clinical examination were recorded. Severity grading of all patients was done based on BISAP scoring. Abdominal ultrasound was used for screening for gall stones, common bile duct stones and pancreatitis, performed in the Department of Radiodiagnosis, Assam Medical College and Hospital and the findings were recorded. Patients diagnosed with gall stone on abdominal ultrasound were tested for liver and pancreatic enzyme levels in the Department of Biochemistry, Assam Medical College and Hospital and the findings were recorded. All patients underwent routine investigations. Special investigations (CT abdomen) were carried out if needed. According to the methods used in the Biochemistry lab of Assam Medical College and Hospital normal range of -

a) Serum Amylase (30-110 U/L) is done by 2-point rate enzymatic test
b) Serum Lipase (23 – 300 U/L) is done by 2-point rate enzymatic test

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

c) Serum ALT (13 - 69 U/L) done by multiple point rate enzymatic test

d) Serum AST (15 - 46 U/L) is done by multiple point rate enzymatic test

e) Serum ALP (38-126 U/L) is done by multiple point rate enzymatic test

f) Serum bilirubin (0.0 – 1.1 mg/dl) is done by end point colorimetric dual-wavelength test

g) Serum creatinine (0.5 – 1.2 mg/dl) is done by 2-point rate enzymatic test

h) Blood urea Nitrogen (7 – 20 mg/dl) is done by colorimetric test

However, for our study, the following values were taken as a reference cut-off based on similar studies as mentioned in the review:

AST- 90 U/L (2 times the laboratory cut-off value)[107]

ALT- 150 U/L (2 times the laboratory cut-off value)[111]

ALP- 250 U/L (2 times the laboratory cut-off value)[113]

The reference values for the other parameters were as per the laboratory cut-off.

Statistical Analysis

All recorded data were tabulated in Microsoft Excel. Categorical data were presented as counts (percentages) and mean +/- standard deviation. Statistical significance was tested using chi square test for categorical data and t-test for continuous data. A p-value of <0.05 was considered as significant. Analysis was done using SPSS.

Results

A total of 140 subjects were analyzed in this study of which 70 cases were of gallstone pancreatitis and the other 70 cases were of alcoholic pancreatitis. The different parameters are analyzed as below.

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

Table 1: Elevation of TLC in alcoholic and gallstone pancreatitis

	Group			p-Value
TLC (>11000)	**Alcoholic**	**Gallstone**	**Total**	
No	45(64.3%)	47(67%)	92(65.7%)	0.721
Yes	25(35.7%)	23(33%)	48(34.3%)	
Total	**70(100%)**	**70(100%)**	**140(100%)**	

In our study, TLC count was found to be raised in 35.7% patients of alcoholic pancreatitis and 33% of patients of gallstone pancreatitis. There was no significant difference noted between the two groups.

Table 2: Elevation of Serum amylase with time in alcoholic and gallstone pancreatitis

	Group		
Time from onset	**Alcoholic**	**Gallstone**	**p-Value**
At presentation	563.87 ± 383.92	908.9±347.61	<0.001
After 48 hrs	492.15 ± 282.04	616.83±317.26	0.015
After 72 hrs	299.34 ± 206.22	251.29±124.47	0.097
At >/=1week	99 ± 98.24	91.73±45.87	0.575

In our study, the average serum amylase level was significantly higher for gallstone pancreatitis as compared to alcoholic pancreatitis at presentation. However, this difference in mean enzyme elevation level became insignificant as the time progressed.

Table 3: Elevation of Serum lipase with time in alcoholic and gallstone pancreatitis

	Group		**p-Value**
Time from onset	**Alcoholic**	**Gallstone**	
At presentation	1108.82 ± 708.46	2005.56 ± 1805.89	0.002
After 48 hrs	1464.75 ± 901.67	1505.44 ± 1537.71	0.848
After 72 hrs	1049.17 ± 721.98	894.2 ± 904.25	0.264
At >/= 1week	612.19 ± 465.67	283.47 ± 160.82	<0.001

In our study, the average serum lipase for gallstone pancreatitis was significantly higher compared to alcoholic pancreatitis. There was no significant difference in the lipase: amylase ratio in either group.

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

Table 4: Elevation of Serum AST (SGOT) in alcoholic and gallstone pancreatitis

	Group			p-Value
AST(SGOT)>90	**Alcoholic**	**Gallstone**	**Total**	
No	55(78.6%)	37(52.9%)	92(65.7%)	0.001
Yes	15(21.4%)	33(47.1%)	48(34.3%)	
Total	**70(100%)**	**70(100%)**	**140(100%)**	

In our study, the serum AST levels were found to be elevated above 90 U/L in 21.4% of alcoholic pancreatitis cases. However, serum AST levels were elevated in 47.1% of gallstone pancreatitis cases.

Table 5: Elevation of Serum ALT (SGPT) in alcoholic and gallstone pancreatitis

	Group			p-Value
ALT (SGPT)>150	**Alcoholic**	**Gallstone**	**Total**	
No	59(84.3%)	29(41.4%)	88(62.9%)	<0.001
Yes	11(15.7%)	41(58.6%)	52(37.1%)	
Total	**70(100%)**	**70(100%)**	**140(100%)**	

In our study, serum ALT levels were found to be elevated in 58.6% cases of gallstone pancreatitis as compared to 15.7% of alcoholic pancreatitis. There was significant difference in the levels of Serum ALT in both types of pancreatitis with higher levels seen in gallstone pancreatitis. In 27.1% patients of alcoholic pancreatitis had serum ALKP levels >250 U/L compared to 55.7% of gallstone pancreatitis patients. This shows significant elevation with respect to etiology of acute pancreatitis.

Table 6: Elevation of Serum ALKP in alcoholic and gallstone pancreatitis

	Group			p-Value
ALKP>250	**Alcoholic**	**Gallstone**	**Total**	
No	51(72.9%)	31(44.3%)	82(58.6%)	0.001
Yes	19(27.1%)	39(55.7%)	58(41.4%)	
Total	**70(100%)**	**70(100%)**	**140(100%)**	

In our study, serum ALP was found to be elevated above normal in both groups of AP in varying

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

levels. However, it was found >250 U/L (above 2 times the laboratory cut off value) in 19 (27.1%) patients of alcoholic pancreatitis as compared to 39 (55.7%) patients of gallstone pancreatitis.

Table 6: Elevation of Serum Creatinine levels in alcoholic and gallstone pancreatitis

	Group			p-Value
Sr Creatinine>1.1	**Alcoholic**	**Gallstone**	**Total**	0.313
No	63(90%)	59(84.3%)	122(87.1%)	
Yes	7(10%)	11(15.7%)	18(12.9%)	
Total	**70(100%)**	**70(100%)**	**140(100%)**	

In our study, there was no significant difference in the incidence of elevated serum creatinine levels between alcoholic and gallstone pancreatitis (10% and 15.7%) respectively.

Table 7: Elevation of Serum Bilirubin in alcoholic and gallstone pancreatitis

	Group			p-Value
Sr. Bilirubin>1	**Alcoholic**	**Gallstone**	**Total**	0.001
No	58(82.9%)	25(35.7%)	83(59.3%)	
Yes	12(17.1%)	45(64.3%)	57(40.7%)	
Total	**70(100%)**	**70(100%)**	**140(100%)**	

In our study, about 17.1% of cases of alcoholic pancreatitis had raised total serum bilirubin levels compared to 64.3% cases of gallstone pancreatitis showing a difference which was statistically significant. Of the serum bilirubin fractions, direct bilirubin was raised more than indirect bilirubin in all cases of both alcoholic and gallstone pancreatitis.

Table 8: Elevation BUN in alcoholic and gallstone pancreatitis

	Group			p-Value
BUN>25mg/dl	**Alcoholic**	**Gallstone**	**Total**	0.771
No	63(90%)	64(91.4%)	127(90.7%)	
Yes	7(10%)	6(8.6%)	13(9.3%)	
Total	**70(100%)**	**70(100%)**	**140(100%)**	

In our study, the blood urea nitrogen was elevated above 25 mg/dl in only 7 (10%) patients of alcoholic pancreatitis and 6 (8.6%) of patients of gallstone pancreatitis. Though there was no

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

significant difference in the elevation of BUN in both groups but a direct co-relation of BUN with the severity of AP was seen.

Discussion

In the present study 70 cases of gallstone pancreatitis and 70 cases of alcoholic pancreatitis, who were admitted in the department of General Surgery, and department of Medicine, Assam Medical College and Hospital respectively, during the period 1st June 2022 to 31st May 2023 were included. The results of the present study are discussed below.

1. **Total Leucocyte Count**

   In our study, the TLC count was found to be raised in 35.7% patients of alcoholic pancreatitis and 33% of patients of gallstone pancreatitis.There was no significant difference noted between the two groups. This result was similar to the observations made by other studies like *Khan N.A* et al in 2021[7] , *Gungor B* et al in 2011[8]. *Khan N.A* et al also observed that the TLC was an independent predictor of the severity of pancreatitis.[7]

2. **Serum Amylase levels**

   In our study, the average serum amylase level at presentation for alcoholic pancreatitis was 563.87 ± 383.92 as compared to 908.9 ± 347.61 for gallstone pancreatitis. This shows a significant difference (p value <0.05). The 48-hour average for alcoholic pancreatitis was 492.15 ± 282.04 compared to 616.83 ± 317.26 for gallstone pancreatitis which is insignificant (p-value > 0.5). Our result co-relates well with other studies like *Pezzeli R* et al in 1993[13], *Manes G* et al in 1995[14], *Gungor B* et al 2011[8]. *Gungor B* et al in 2011 observed that specificity of serum amylase for biliary pancreatitis is 95%. However, sensitivity is 60%. In alcoholic pancreatitis, the sensitivity of serum amylase is thought to decrease secondary to pancreatic insufficiency due to chronic alcohol abuse.[8]

3. **Serum Lipase levels**

   In our study, the average serum lipase for alcoholic pancreatitis was1108.82 ± 708.46 as compared to 2005.56 ± 1805.89 for gallstone pancreatitis. This shows a significant difference (p-value < 0.05). However, the 48-hour lipase for alcoholic pancreatitis was 1464.75 ± 901.67 as compared to 1505.44 ± 1537.71 for gallstone pancreatitis which is insignificant (p-value > 0.05). The average lipase: amylase ratio for alcoholic pancreatitis

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

at presentation, 48 hours and 72 hours was 1.97, 2.98 and 3.50 respectively. The average lipase: amylase ratio for gallstone pancreatitis at presentation, 48 hours and 72 hours was 2.2, 2.4 and 3.3 respectively. Our result co-relates with *Pezzeli R* et al in 1993[13], *Singh HB* et al in 2020[9]. *Singh HB* et al in 2020 observed that the lipase: amylase ratio ≥3 is observed in alcoholic acute pancreatitis while biliary pancreatitis has ratio <3.[9]

4. **Serum AST levels**

In our study, the serum AST levels were found to be elevated in varying levels in both the groups. However, 21.4% of alcoholic pancreatitis cases had sr. AST above 90 U/L compared to 47.1% of gallstone pancreatitis cases. This shows significant difference (p value <0.05). Our results show similarity with *Lin G* et al in 1997[15], *Zhao Q* et al in 2018[10]. *Zhao Q* et al in 2018 noted that there were significant differences in the levels of ALT, AST, ALP between patients with MAP with between the biliary and the non-biliary etiology (all P < 0. 05) [10]

5. **Serum ALT levels**

In our study, serum ALT levels were found to be elevated above 150 U/L (more than 2 times our laboratory cut off value) in 58.6% cases of gallstone pancreatitis as compared to 15.7% of alcoholic pancreatitis. There was significant difference in the levels of Serum ALT in both types of pancreatitis with higher levels seen in gallstone pancreatitis. Our results were similar to the following studies- *Moolla* Z et al in 2012[2], *Zarnescu NO* et al in 2015[15]**.** *Moolla* Z et al in 2012 noted that Alanine transaminase was the only factor independent of gender to predict gallstone etiology, with activity >150 U/l having a specificity of 97 %. [2]

6. **Serum ALP levels**

In our study, 27.1% patients of alcoholic pancreatitis had serum ALKP levels >250 U/L (more than 2 times the laboratory cut off value) compared to 55.7% of gallstone pancreatitis patients. This shows significant elevation with respect to etiology of acute pancreatitis. Our results show similarity with *Zhao Q* et al in 2018[10] and *Davidson B* et al in 1988[17]**.** *Zhao Q* et al in 2018 noted that compared with the non-biliary AP group, the group with gallstone pancreatitis had a significantly higher incidence rate of an abnormal level of ALP, GGT, or ALP + GGT (all P < 0. 05) .[10]

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

7. **Blood Urea Nitrogen levels**

   In our study, 10% of cases of alcoholic pancreatitis had BUN>2 compared to 8.6% cases of gallstone pancreatitis. There was no co-relation of blood urea levels with the etiology of acute pancreatitis in our study. However, elevated blood urea levels proved to be good predictors of severe acute pancreatitis and increased morbidity and mortality. Similar studies include- *Shah SMA* et al in 2021[11], *Gungor B* et al in 2011[8]. *Shah SMA* et al in 2021 noted that BUN ≥25 mg/dl at 24 hours was found to be the optimal cut-off for determining the severity of acute pancreatitis with sensitivity of 95.7%.[11]

8. **Serum Creatinine levels**

   In our study, no significant difference was noted in the incidence of elevated serum creatinine levels between alcoholic and gallstone pancreatitis (10% and 15.7%) respectively. Our results show similarity with *Muddana V* et al in 2009[12]. *Muddana V* et al in 2009 noted that an increase in creatinine within the first 48 h is strongly associated with the development of Pancreatic Necrosis.[12]

9. **Serum Bilirubin levels**

   In our study, 17.1% of cases of alcoholic pancreatitis had raised total serum bilirubin levels compared to 64.3% cases of gallstone pancreatitis. Of the serum bilirubin fractions, direct bilirubin was raised more than indirect bilirubin in all cases of both alcoholic and gallstone pancreatitis. Our results showed similarity with the following studies- *Davidson B* et al in 1998[17], *Gungor B* et al in 2011[8]. *Gungor B* et al in 2011 noted that both total and direct bilirubin levels had the highest specificity, positive and negative predictive values among biochemical laboratory markers of biliary pancreatitis. The cut-off values of bilirubin were very low (1 and 0.21 mg/dL). This shows that very little obstruction of the common bile duct can precipitate an attack of AP. [8]

Conclusion

Acute Pancreatitis is one of the most common causes of acute abdomen in our country. Alcoholic pancreatitis and gallstone pancreatitis are the two of the commonest etiological factors leading to acute pancreatitis. The diagnosis is commonly based on radiological investigations. However, from our study, we have found that Serum AST, ALT and ALKP along with serum bilirubin can help to increase the suspicion of gallstone pancreatitis for the clinician. Our study consisted mostly

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

of patients presenting with acute abdominal pain but most had low scores on BISAP scoring (mild AP). BISAP scoring has proven to be a good indicator for prognosis and severity of acute pancreatitis. However, our study shows that liver enzyme levels do not help in prediction of severity of the disease. In conclusion, according to our study, raised biochemical markers AST, ALT, Amylase, Lipase as well as ALP and S. Bilirubin can be used as good markers for indication of gallstone pancreatitis. However, a normal or low serum level of these markers does not rule out the possibility of gallstones in the etiology of pancreatitis and may require investigation.

References

1. Papachristou GI, Muddana V, Yadav D, O'connell M, Sanders MK, Slivka A, Whitcomb DC. Comparison of BISAP, Ranson's, APACHE-II, and CTSI scores in predicting organ failure, complications, and mortality in acute pancreatitis. American Journal of Gastroenterology. 2010 Feb 1;105(2):435-41.
2. Moolla Z, Anderson F, Thomson SR. Use of amylase and alanine transaminase to predict acute gallstone pancreatitis in a population with high HIV prevalence. World journal of surgery. 2013 Jan;37(1):156-61.
3. Patti MG, Pellegrini CA. Gallstone pancreatitis. Surgical Clinics of North America. 1990 Dec 1;70(6):1277-95.
4. Whitcomb DC. Acute pancreatitis. New England Journal of Medicine. 2006 May 18;354(20):2142-50.
5. Eric H. Jensen, Daniel Borja-Cacho, Waddah B. Al-Refaie, and Selwyn M. Vickers, sabiston textbook of Surgery, 2012
6. Yadav D, Agarwal N, Pitchumoni CS. A critical evaluation of laboratory tests in acute pancreatitis. The American journal of gastroenterology. 2002 Jun 1;97(6):1309-18.
7. Khan N A, Haider Kazmi S, Asghar M et al. Hematological Indices Predicting the Severity of Acute Pancreatitis Presenting to the Emergency Department: A Retrospective Analysis. Cureus 2021; 13(7): e16752.
8. B¨ulent G¨ung¨or, Kasım C¸a˘glayan, CaferPolat, Deniz S¸eren, Kenan Erzurumlu, Zafer Malazgirt.The Predictivity of Serum Biochemical Markers in Acute Biliary Pancreatitis. ISRN Gastroenterology 2010; 2011: 279607.

ISSN: 0975-3583, 0976-2833 VOL 15, ISSUE 07, 2024

9. Singh HB, Singh LJ, Shougrakpam P. Serum lipase amylase ratio in predicting the etiology of acute pancreatitis. J Med Soc 2020;34:96-100.
10. Zhao Qi, Wang Bing, Li ZhenFang, Fang ChunFang, Li Cong, Li SenLin. Clinical significance of abnormal liver function in determining the etiology and severity of acute pancreatitis[J]. *J Clin Hepatol*, 2018, 34(1): 152-156.
11. Shah SMA, Andrabi SAS, Quraishi AN, Kumar R, Khan TS, Shah AP, et al. Blood urea nitrogen as an early predictor of severity in acute pancreatitis. Int J Res Med Sci 2021;9:1015-9.
12. Muddana V, Whitcomb DC, Khalid A, Slivka A, Papachristou GI. Elevated serum creatinine as a marker of pancreatic necrosis in acute pancreatitis. *Am J Gastroenterol*. 2009;104(1):164-170.
13. Pezzilli R, Billi P, Miglioli M, Gullo L. Serum amylase and lipase concentrations and lipase/amylase ratio in assessment of etiology and severity of acute pancreatitis. *Dig Dis Sci*. 1993;38(7):1265-1269.
14. Manes G, Rabitti PG, Laccetti M, Pacelli L, Carraturo I, Uomo G. Early prediction of aetiology and severity of acute pancreatitis by serum amylase and lipase assays. *Minerva Gastroenterol Dietol*. 1995;41(3):211-215.
15. Lin G, Halevy A, Girtler O, Gold-Deutch R, Zisman A, Scapa E. The role of endoscopic retrograde cholangiopancreatography in management of patients recovering from acute biliary pancreatitis in the laparoscopic era. SurgEndosc. 1997;11(4):371-375.
16. Zarnescu NO, Costea R, ZarnescuVasiliu EC, Neagu S. Clinico-biochemical factors to early predict biliary etiology of acute pancreatitis: age, female gender, and ALT. J Med Life. 2015 Oct-Dec;8(4):523-6.
17. Davidson BR, Neoptolemos JP, Leese T, Carr-Locke DL. Biochemical prediction of gallstones in acute pancreatitis: a prospective study of three systems. British Journal of Surgery. 1988 Mar;75(3):213-5.