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Original Research Article

A prospective study of clinical, biochemical and radiological features in pancreatitis

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ABSTRACT

Background: Pancreatitis is quite common problem, it may present either as abdominal emergency with fulminant course or as an indolent process leading to long-term medical as well as surgical complications often leading to poor prognosis if not intervened timely.
Methods: The present study conducted in the department of medicine, Dr. S. N. medical college, Jodhpur. Participants after understanding the study protocol and procedure, asked to give their written consent for the study. A cross sectional hospital based study in patients admitted in various medical wards of Dr. S. N. medical college, Jodhpur. 50 patients with acute and chronic pancreatitis were studied.
Results: In the present study, most common cause of acute pancreatitis is biliary disease (50%) followed by alcoholism (37.5%) and in chronic pancreatitis is alcoholism (80%). Acute pancreatitis was more common in males (62.5%, 25 males) whereas chronic pancreatitis in males (80%, 8 male). Abdominal pain is the most common symptom (97.5%) followed by nausea-vomiting (92.5%) in acute pancreatitis. The history of previous abdominal pain in 100% of cases followed by epigastria pain in 90% of cases, in chronic pancreatitis. The amylase and lipase were elevated in 90% of cases. The amylase and lipase levels did not correlate with the severity. 66.6% of patients had severe pancreatitis with a positive predictive value of 66.6%. The ultrasonography imaging of pancreas was helpful in 70% and 100% in acute and chronic pancreatitis respectively. The CT scan was a better imaging modality as compared to ultrasonography in acute pancreatitis, where as it scored over ultrasonography imaging in chronic pancreatitis with complications.
Conclusions: Relevant clinical history, ultrasonography and computed tomography scan of pancreas are helpful in diagnosis of pancreatitis. The computed tomography scan was a better imaging modality as compared to ultrasonography.

Keywords: Computed tomography scan, Pancreatitis, Ultrasonography

INTRODUCTION

Pancreatitis defined as the inflammation of the pancreas and is always, associated with acinar cell injury. (Singer et al) Acute pancreatitis is clinically characterized by acute onset of abdominal pain and a rise in the activity of pancreatic enzymes in the blood and urine.[1] Most attacks have a benign course but severe attacks may lead to shock, renal failure, respiratory failure and death. Chronic pancreatitis is characterized clinically by recurrent or persistent episodes of abdominal pain. Although, in some cases, chronic pancreatitis occurs without pain, evidences of functional insufficiency such as steatorrhoea or diabetes is often seen. Clinically, the first manifestation

of alcoholic chronic pancreatitis can closely resemble acute pancreatitis. (Steer ML et al).[2] Various aetiological factors have been known to cause pancreatitis which include: gall stones, (Gorelick FS et al.), structural lesions like Stenosis or spasm of sphincter of oddi, pancreas divisum, traumatic, microlithiasis, toxins, alcohol, drugs, (frusemide tetracycline), infection (mumps, Coxsackie B-virus, viral hepatitis, HIV, salmonella, shigella, ascariasis lumbricoides), Metabolic (hyper-lipidemia, hypercalcemia), vascular (atherosclerosis, vasculitis, SLE, Wegener's disease. Behcet's disease) iatrogenic (ERCP, endoscopic sphincterotomy, coronary artery bypass) cystic fibrosis etc.[3]

Various studies have been undertaken in India and abroad about pancreatitis, its presentation and role of various laboratory and imaging techniques for diagnosis and prognostication. We have undertaken this study to know various clinical, laboratory and imaging features of acute and chronic pancreatitis in our region

METHODS

We have conducted a study on 50 cases of pancreatitis, out of which 40 cases comprised of acute pancreatitis and rest of chronic pancreatitis, admitted at department of medicine, Dr. S.N. medical college, Jodhpur in various wards. Diagnosis of acute pancreatitis/chronic pancreatitis was based on clinical findings and biochemical markers. This diagnosis was confirmed by ultra-sonographic examination and CT' Scan (computed tomography scan) abdomen.

Thorough physical examination of all the patients was done. All the patients were thoroughly investigated, which included routine investigations (Hb, TLC, and DLC), blood sugar, blood urea, S. creatinine, X-ray chest PA view, ECG, plain X-ray abdomen and special investigations like liver function test, serum calcium, serum LDH, serum protein, lipid profile, serum alkaline phosphatase and serum electrolytes. Apart from these serum amylase and serum lipase levels were measured. All patients were under gone USG and CT scanning.

Each case was analyzed and divided into mild or severe depending upon the clinical, biochemical data (according prognostic criteria) and CT severity index findings. A severe attack of acute pancreatitis was defined, when an attack is accompanied or followed by any of following clinical finding. Attack followed by shock, pseudo pancreatic cyst, pancreatic abscess and death. (Michael J. Mc Mohan et al.) Rest of the cases were defined as mild.[4]

All of these studies, included clinical exam were done at the time of admission and follow-up studies were not performed in any case. On the basis of factor scoring system the severity of acute pancreatitis was predicted as described by modified glasgow prognostic criteria. These factors are age, WBC counts, serum blood glucose, serum albumin, serum calcium, arterial p02 and blood urea. (Garelick FS).[3]

Those having < 3 prognostic criteria were considered as mild and those > 3 prognostic criteria were treated as severe attack. The CT scan severity index 3 to 6 is mild and more than (Greenberger NJ).[5]

Table 1: CT severity index (prognostic scoring).

	Grades of acute pancreatitis based on	Score
A	Normal	0
B	Pancreatic enlargement	1
C	Inflammation confined to pancreas and peri-pancreatic fat	2
D	One peri-pancreatic fluid collection	3
E	Two or more fluid collection	4

Inclusion criteria: patients presenting with history and clinical features suggestive of acute or chronic pancreatitis, and later proved by serology and imaging were included in study group.

Table 2: Degree of necrosis (to be assessed by CT scan).

No necrosis	
Necrosis of 1/3 of pancreas	2
Necrosis of 1/2 pancreas	4
Necrosis of more than 1/2 of pancreas	6

Test for amylase

This test is based on the principle that amylase hydrolyses the α-1, 4 - glucan link in polysaccharides of three or more α - 1, 4 - linked D - glucose units. The procedure we employed uses substrate P-nitrophenol- α-D- maltoheptaoside, in which the terminal glucose unit has been blocked by an ethylidene group to protect the substrate from cleavage by the auxiliary enzyme α-glucosidase. The hydrolytic action of amylase on substrate (P-nitrophenol - α - D - maltoheptaoside) results in release of smaller oligosaccharides bound to P - nitrophenol. The rate of increase in the colour at 405 nm from the liberated P -nitrophenol is proportional to the amylase activity in the sample. The reference range is 60 - 180 IU/L at 37°C.

Test for lipase

This test is based on the principle that lipase catalyzes the hydrolysis of triglycerides sequentially to monoglyceride and two fatty acids. The most commonly used assay for lipase involves measuring the clearing of substrate emulsion by the action of lipase. Measurement can be made by either nephelometry or turbimetry.

Triolein + Lipase > Monoglyceride + 2 Oleic acids.
(Cloudy solution) (clear solution)

The optimum pH for this reaction is 8.8. In this assay both lipase anc.[4] lipoprotein lipase arc measured however, if colipase and a bile salt such as sodium deoxy cholate are included, the reaction rate and analytical sensitivity of pancreatic lipase in increased, while that of lipoprotein lipase is eliminated.

Colipase, aided by the addition of bile salts binds to lipase to form a complex. This association produces a conformational change in lipase, such that latter can now more efficiently bind to the substrate, the reference range for lipase depends on the substrate and whether or not colipase is used the upper reference limit is 0 - 160 IU/L at 37°C, when triolenc is used as substrate in the presence of colipase and bile salts.

RESULTS

In present study total 50 cases were enrolled out of them 40 cases were of acute pancreatitis and rest were chronic. Out of 40 cases of acute pancreatitis Maximum cases were of age group 30 - 39 years (32.5%).

Followed by 25% in the age group of 50 -59. None of patients was below 18 years of age and only one being above 70 years. In chronic pancreatitis group, maximum (40%) patient was of age group 40 - 49 years and 30% in the 30-39 years group. Male female ratio in acute pancreatitis was 1.7:1 (25 males, 15 females) and chronic pancreatitis 4: 1 (8 males, 2 females).

Table 3: Aetiological correlation of acute and chronic pancreatitis.

Aetiology	Acute pancreatitis				Chronic pancreatitis			
	Male	Female	Total	%	Male	Female	Total	
Gall Stones	5	15	20	50	0	0	0	
Alcoholism	15	0	15	37.5	8	0	8	0
Post-operative	0	0	0	0	0	0	0	
Traumatic	1	0	1	2.5	0	0	0	
Idiopathic	4	0	4	10	0	2	2	0

Most common aetiology was biliary tract disease in 50% of cases, followed by alcoholism in 37.5% of cases. Traumatic and idiopathic were 2.5% and 10% respectively. The most common aetiological factor in chronic pancreatitis was alcoholism in 80% followed by idiopathic in 20% of cases (Table 3). 20% (5) of males and 100% (15) of females had biliary tract disease as etiology of acute pancreatitis. The 60% (15) of males were found to be alcoholic, whereas alcoholic aetiology was not found in females. Other causes were traumatic 4% (1) and idiopathic 16% (4) in males. In chronic pancreatitis case 100% of males had history of alcoholism as aetiology and while 100% of females has idiopathic aetiology.

Table 4: Clinical presentation of cases of acute and chronic pancreatitis.

Symptom/sign	Acute pancreatitis		Chronic pancreatitis	
	Total	%	Total	%
Epigastric pain	39	97.5	9	90
Nausea vomiting	37	92.5	8	80
Pain radiating back	24	60	1	10
Previous history of acute abdominal pain	12	30	10	100
Weight loss	0	0	6	60
Diarrhoea	0	0	3	30
Diabetes	2	5	2	20
Trauma	1	2.5	2	20
Fever	10	25	3	30
Tachycardia	8	20	0	0
Abdominal tenderness	24	60	0	0
Abdominal lump	3	7.5	0	0
Pulmonary rales	3	7.5	0	0
Hypotension	2	5	0	0

Most common clinical presentation in acute pancreatitis, was epigastric pain (97.5%) followed by nausea-vomiting (92.5%), pain radiating to the back (60%), abdominal tenderness (60%) and previous history of abdominal pain (30%) of cases. Some of the cases of acute pancreatitis showed fever (25%), tachycardia (20%), abdominal lump (7.5%), pulmonary rales (7.5%) and hypotension in (5%) of cases (Table 4). While in chronic pancreatitis was previous history of abdominal pain in all cases, followed by epigastric pain (90%), nausea-vomiting (80%), weight loss (60%), fever (30%), diarrhoea (30%) and diabetes (20%) of cases. 92.5% (37) of cases were mild and 7.5% (3) were severe acute pancreatitis.

Table 5: Serum enzyme levels in pancreatitis.

Enzyme	Total no.	%	Mean value		SD		t	P
			Mild	Severe	Mild	severe		
Serum amylase	36	90	1388.7	948.6	655.16	971.7	1.532	>.05
Serum lipase	36	90	649.7	795.0	389.98	506.9	0.884	>.05

In mild cases mean value of serum amylase was 1338.0 IU/L (range 105-9150 IU/L), while in severe cases it was 948.6 IU/L (range 78-1664 IU/L). Serum lipase in mild cases had mean value of 649.7 IU/L (ranges 100-1794 IU/L) and in severe cases 795.0 IU/L (range 379-1205 IU/L). Statistical analysis of these data show that serum amylase and lipase levels were not significant enough to differentiate between mild and severe pancreatitis (P>0.05) (Table 5).

Table 6: Ultrasonography finding in acute and chronic pancreatitis.

Ultra-sonographic finding	Acute pancreatitis		Chronic pancreatitis	
	Total	%	Total	%
Visualization of pancreas	28	70.0	10	100
Enlargement of pancreas	26	65.0	5	50
Pancreatic calcification	0	0	10	100
Pancreatic pseudocyst	4	10.0	2	20
Gall stone	15	37.5	0	0
Pleural effusion	3	7.5	0	0
Ascites	4	10.0	0	0

Table 7: Computed tomography scan finding in acute and chronic pancreatitis.

CT scan finding	Acute pancreatitis		Chronic pancreatitis	
	Total	%	Total	%
Visualization of pancreas	40	100	10	100
Enlargement of pancreas	39	97.5	6	60
Necrosis of pancreas	10	25	0	0
Abscess of pancreas	0	0	0	0
Pancreatic haemorrhage	0	0	0	0
Pancreatic pseudocyst	6	15	3	30
Pancreatic calcification	0	0	10	100
Gall stone	16	40	0	0
Ascites	4	10	0	0
Pleural effusion	3	7.5	0	0

In ultra-sonographic findings visualization of pancreas in 70% of cases, pancreatic enlargement in 65% of cases, gall stone in 37.5% of cases, pseudocyst and ascites each in 10% of cases and pleural effusion in 7.5% cases of acute pancreatitis. While pancreatic calcification and visualization of pancreas were found each in 100% cases of chronic pancreatitis, followed by pancreatic enlargement in 50% and pseudocyst in 20% of cases (Table 6).

Visualization and enlargement of pancreas by computed tomography scan respectively was found in 100% and 97.5% in acute pancreatitis. Associated findings in computed tomography scan-gall stone in 40% of cases, pancreatic necrosis in 25% cases, and ascites in 10% cases and pleural effusion in 7.5% of cases. Calcification and visualization of pancreas by computed tomography scan, each was found in 100% cases of chronic pancreatitis, followed by pancreatic enlargement in 60% of cases and pseudocyst in 30% of cases (Table 7).

Table 8: Computed tomography scan severity index in acute pancreatitis- a grade of pancreatitis.

Category / severity index	CT grading No.	No. of cases	%
Normal pancreas	0	1	2.5
Pancreatic enlargement alone	1	4	10
Involvement limited to pancreatic fat	2	25	62.5
One peri pancreatic fluid collection	3	10	25.0
More than two peri pancreatic fluid collection	4	0	0

Computed tomography scan severity index revealed pancreatic enlargement and peri pancreatic fat involvement (grade 1-2) in 72.5% of cases, peri pancreatic fluid collection in 25% and normal pancreas in 2.5% of cases in acute pancreatitis (Table 8).

Table 9: Degree of pancreatic necrosis.

CT scan Finding	Total	%
No necrosis	30	75
Necrosis 1/3 of pancreas	9	22.5
Necrosis ½ of pancreas	1	2.5
Necrosis > ½ of pancreas	0	0

Pancreatic necrosis was found in 25% of cases (1/3 necrosis of pancreas 22.5 % cases, ½ necrosis of pancreas 2.5 % of cases) (Table 9).

Table 10: Comparison of the pancreatic visualization in ultrasonography and computed tomography scan in acute and chronic pancreatitis.

Category	Acute pancreatitis		Chronic pancreatitis	
	Total	%	Total	%
Ultrasonography	28	0	8	00
CT scan	40	00	8	00

Ultrasonography was helpful in the diagnosis of acute pancreatitis in 70% of cases and chronic pancreatitis in 100% cases. The computed tomography scan was equally important in diagnosis of both acute and chronic pancreatitis in 100% of cases (Table 10).

The patient who had 4 or more factors was either all had severe pancreatitis but if total number of factors 3 or less than 3, the disease is mild (decided by modify Glasgow system) this indicate the predictive value of scoring system was 100% (Table 11).

Table 11: Predictive value of the original scoring system in acute pancreatitis.

No. of factors present	No. of patients	Mild disease	Severe disease	Severity %
0	5	5	0	0
1	7	7	0	0
2	6	16	0	0
3	5	4	1	20
4	3	0	3	100
5	1	0	1	100
6	3	0	3	100
7	0	0	0	0
8	0	0	0	0

DISCUSSION

In acute pancreatitis, out of the 40 patients studied under this diagnosis 32.5% were in the age group (30-39 years), followed by 25% in the (50-59) years age group. None of the patients were below 18 years of age and only one patient was above 70 years of age. The age of the patients varies between 18-75 years, with mean age for mild cases being 42.2 years and that of severe cases being 44.2 years. Study findings were in correlation with that of Mc Entee et al [6] where the mean age was 42.4 years (range 20-69 years) whereas the study by Corfield et al have shown the mean age to be 60 years (range 3-94 years).[7]

In chronic pancreatitis, the mean age of the patients were 45.4 years (range 30-69 years) which differs from the study by Balaji LN and Tandon RK where the mean age was 23.9 years.[8] In acute pancreatitis case sex ratio was 1.7:1 (25 males, 15 females). This was slightly higher than the study by S.R. Thomson et al., where sex ratio was 1.05:1. But almost similar to the study by Gillespie WJ et al where it was 2:1.[9,10] In chronic pancreatitis male: female ratio was 4: 1 (8 males, 2 females) which was contrary to the study by Balaji LN and Tandon RK (where the sex-ratio was 1:1.8.[8] The difference is

probably due to small number of patient in present study group.

In acute pancreatitis, gall stones were the major aetiological factor in 50% of cases, whereas alcoholism comprised 37.5% of cases and idiopathic aetiology in 10% of cases. These observations were almost similar to those in the study by Imrie CW and Whyte AS where the biliary disease was found in (51 %) of cases, alcoholism in (26%) of cases and idiopathic in (13%) of cases as aetiological factors.[11]

Another study by Blarney SL et al have shown gall stone as aetiological factor in 44% of cases, while alcohol accounted for 33% of cases and rest 24% being idiopathic.[12] In contrast Park et al showed that biliary causes were responsible for 35% of cases and idiopathic aetiology in 30% of cases.[13] While the study by Jacob ML et al shown biliary disease in 47% of cases and alcoholism in 31% of cases.[14] These variations may be because of different culture socioeconomic group and less occurrence of gall stones in those places.

In case of chronic pancreatitis alcoholism proved to be the major aetiology in 80% of cases and the rest 20% being idiopathic. These findings were in correlation with the study by Arenha GV et al where alcoholism was attributed in 78% of cases and rest 22% being idiopathic.[15] Johnson and Imrie also conducted study with similar results.[16] Thereby proving that alcoholism is the major cause of chronic pancreatitis.

In case of acute pancreatitis, we found that all the female patients had biliary tract disease as aetiological cause but only 20% male has biliary tract disease. Alcoholism as aetiological factor was found in 60% of males. While none of the females has alcoholism as aetiological factor. While idiopathic cause accounted for in 16% of males and none in the females. Traumatic aetiology was found in 4% of males' patients. The above study was in contrast to the study by Thomson SR et al where biliary tract disease was the major aetiological factor in 30% of males and 53% of females.[9]

Alcoholism accounted for 26.5% of males and 3% of females. While idiopathic aetiology was responsible in 19% of males and 22% of females. Hence the above correlation has proved the higher incidence of alcoholism as the major aetiological factor in Indian males possibly as a result of higher consumption of alcohol by the male community in India and more common occurrence of gall bladder disease in females in India is probably due to faulty dietary habits, in no case of alcoholism was aetiological factors in female, since very few females consume alcohol in India. While in cases of chronic pancreatitis alcoholism was the major cause in males (100%) while idiopathic aetiology was predominant in females (100%). These findings were similar to the study conducted by Sarles and Johnson, which concludes the male preponderance in chronic pancreatitis with alcohol, as a principle cause of chronic pancreatitis.[17]

In acute pancreatitis, epigastric pain was the predominant clinical presentation in 97.5% of cases, followed by nausea-vomiting in 92.5%, pain radiating to the back in 60%, abdominal tenderness in 60% and previous history of acute abdominal pain in 30% of the cases. While 25% of patients had fever, tachycardia was present in 20% of cases, pseudocyst in 7.5%, pulmonary rales in 7.5% and Hypotension in 5% of the cases. The above findings were slightly higher than the study by Albo R et al where there was nausea-vomiting in 83% of cases, epigastric pain in 60% and pain radiating to the back in 40% of the cases.[18] Another study conducted by Saxona A et al showed abdominal pain in 90% of cases and abdominal tenderness and fever in 86% of the cases.[19]

In current study, the clinical diagnosis of mild in 92.5% of cases and severe in 7.5% (3) of cases. In chronic pancreatitis, predominant clinical presentation was history of previous abdominal pain in 100% of cases. Followed by epigastric pain in 90%, nausea-vomiting in 80%, weight loss in 60 % of cases, fever in 30% of cases. Exocrine pancreatic insufficiency as evidenced by diarrhoea in 30% and diabetes mellitus in 20% of the cases. These findings are well correlated with the study by Rai RR, Acharya SK et al which reported epigastric pain in 83%, diabetes in 48%, recurrent abdominal pain in 30% and exocrine pancreatic dysfunction as diarrhoea in 9% of the cases.[20] Another study by Lankisch PG et al reported abdominal pain in 50% of alcoholic patients and 62% in non-alcoholic patients.[21]

In mild cases mean value of serum amylase was 1388.7 IU/L (range 105 -9150 IU/L) while in severe cases it was 948.6 IU/L (range 78-1664 IU/L). Serum lipase in mild cases had a mean value of 649.7 IU/L (range 100 IU/L - 1794 IU/L) and in severe cases 795.0 IU/L (range 379 - 1205 IU/L).

Statistical analysis of these dates show that serum amylase and lipase levels were not significant enough to differentiate between mild and severe pancreatitis ($P > 0.05$) hence are not counted as prognostic factors. Serum amylase and lipase were higher than normal in 90% of cases. The findings of this study were quite similar to the study by Reffaele et al who found serum amylase to be elevated in 97% of cases and lipase in 100% of cases.[22] Another study by Lifton LJ et al showed that serum amylase to be high in only 70% of cases and lipase in 63% of cases.[23] In one study by Patt H et al 97% of cases had higher levels of either serum amylase or lipase while 84% of the cases had elevated serum amylase levels.[24]

Ultra-sonographic findings in acute Pancreatitis, comprised of visualization of pancreas in 70% of cases, pancreatic enlargement in 65%, gall stones in 37.5%, pseudocyst and ascites in 10% of eases and pleural effusion in 7.5% of cases. These findings were similar to

that reported by Lawson TL et al which conclude an abnormal pancreas in 94% of cases.[25] Husband JE et al had ultrasonography visualization or pancreas in 75% of cases which was similar to present study.[26]

In another study by William Silverstein et al pancreatic visualization was found in 62% of cases which was also similar to present study While chronic pancreatitis calcification and visualization of pancreas was found in 100% of casts, Followed by pancreatic enlargement in 50% of cases and pseudocyst in 20% of cases.[27] These findings are almost similar to those by Ferrueci JT who had pancreatic calcification in 66% and pseudocyst in 25% of cases.[28] So, ultrasonography was helpful in the diagnosis of chronic pancreatitis in 100% of cases. The characteristic features were calcification of pancreas in 100% of the cases.

In Computed Tomography Scan in cases of acute pancreatitis, the visualization and pancreatic enlargement respectively was found in 100% and 97.5% of cases, associated findings were gall stones in 40% of cases, pancreatic necrosis in 25%, pseudocyst in 25%, ascites in 10% and pleural effusion in 7.5% of cases. These findings were quite similar to those in the study by Silverstain W et al who showed abnormal pancreatic (visualization of pancreas) imaging in 98% of cases and pseudocyst in 10% of cases.[27] Another study by Janet T. Husband et al showed pancreatic visualization in 87.5% of the cases.[26]

According to CT scan severity index, pancreatic enlargement and peri pancreatic fat involvement (grade 1 - 2) was found in 72.5% of the cases, peri pancreatic fluid collection in 25% of cases, normal pancreas in 2.5% of cases and in pancreatic necrosis 25% of the cases in present study. These findings are well correlated to those by Michael C. Hill et al who had inflammation limited to pancreas in 61% of cases and normal pancreas in 28% of the cases.[29] While Block S, Maier W et al reported pancreatic necrosis in 90% of cases in acute severe pancreatitis and 79% of the cases of mild pancreatitis which was dissimilar to present study.[30]

In chronic pancreatitis, pancreatic calcification and visualization of pancreas are seen in 100% of cases. Followed by pancreatic enlargement in 60% of cases and pseudocyst in 30% of cases. These were similar to the study reported by Michael C. Hill et al who found pancreatic calcification in 70% of cases but was contrary to that reported by Ferrucci JTet al who found calcification in 36% of cases, pancreatic enlargement in 36% of cases and pseudocyst in 15% of cases. [28,29] In the present study computed tomography scan was equally important in the diagnosis of both acute and chronic pancreatitis (100%). These observations were similar than that by Silverstein W et al (98%) and but significantly higher than Joseph T. Ferrucci (56%).[28]

In the present study severity of pancreatitis was based on the compiling scoring system which incorporate eight factors. Accordingly, patients with three or more positive factors were defined as severe pancreatitis of patients. It was found that 66.6% of patients had severe pancreatitis with a positive predictive value of 66.6%, which was similar to the study by Blarney SL, Imrie et al who had positive predictive value of 72% when nine factors were used in the compile scoring system and 79% when eight factors were used, within 48 hours of hospitalization of patients.[12] Michael J. Mc. Mohan had showed a positive predictive value of 82%, which was slightly higher than the present study.[4]

CONCLUSION

In the present study, most common cause of acute pancreatitis is biliary disease (50%) followed by alcoholism (37.5%) and in chronic pancreatitis is alcoholism (80%). Acute pancreatitis was more common in males (62.5%, 25 males) whereas chronic pancreatitis in males (80%, 8 male). Abdominal pain is the most common symptom (97.5%) followed by nausea-vomiting (92.5%) in acute pancreatitis.

The history of previous abdominal pain in 100% of cases followed by epigastria pain in 90% of cases, in chronic pancreatitis. The amylase and lipase were elevated in 90% of cases. The amylase and lipase levels did not correlate with the severity. 66.6% of patients had severe pancreatitis with a positive predictive value of 66.6%. The ultrasonography imaging of pancreas was helpful in 70% and 100% in acute and chronic pancreatitis respectively. The computed tomography scan was a better imaging modality as compared to ultrasonography in acute pancreatitis, where as it scored over ultrasonography imaging in chronic pancreatitis with complications.

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