A study of etiological clinical biochemical and radiological profile of patients with acute pancreatitis in rural population

Shilpashree Channasandra Shekar¹, Suhas Narayana Swamy Gowda²*, Naveen Narayan³, Ajay Nagraj², Vishnu Venugopal², Raghunandan Manjappa Kanmani²

INTRODUCTION

"Acute pancreatitis is the most terrible of all the calamities that occur in connection with the abdominal viscera. The suddenness of its onset, the illimitable agony which accompanies it, and the mortality attendant upon it, all render it the most formidable of catastrophes" Lord Moynihan, in 1925. Pancreatitis has been recognized since antiquity but the importance of pancreas and the severity of its inflammatory disorders were realized only in middle of 19th century.¹ More than a century after its comprehensive description, Acute pancreatitis (AP) remains a common disorder with devastating consequences. AP is one of the most common gastrointestinal diseases requiring hospitalization worldwide, with a rising incidence ranging from 13 to 45 per 100,000 persons/year. AP includes a wide spectrum of disease from one with mild self-limiting symptoms to fulminant processes with multi-organ failure and high mortality. Although most episodes are mild and self-

ABSTRACT

Background: Pancreatitis has been recognized since antiquity. Acute pancreatitis is an acute inflammatory process of the pancreas with variable involvement of other tissues or remote organ systems, presenting with variable clinical and systemic manifestations, presenting with mild self-limiting disease to severe life-threatening multi-organ failure.

Methods: This was a prospective study of 60 patients, who were admitted with the diagnosis of acute pancreatitis (AP) during the period from December 2017 to June 2019. The data was collected from the all the patients who met the inclusion criteria, and recorded in the proforma prepared for the study.

Results: Out of 60 patients 86.7% were male and 13.3% were female. The highest incidence was noted in 40-51 years age group (35%). Alcohol was the most common cause (75% patients). Abdominal pain was the most common mode of presentation (100%), and epigastric tenderness was the most common sign (100%). More than 3-fold elevation of serum amylase and lipase was seen in 26.7% and 33.3% of patients respectively. USG and CT scan was diagnostic only in 58.5% and 76.7% of patients respectively. All patients were managed conservatively. There was no mortality.

Conclusions: In AP patients one should not only rely on enzyme level elevations for diagnosing AP. Patients with only a small increase in amylase and/or lipase levels or even with normal levels may also have or develop acute pancreatitis. High degree of suspicion is required; USG, CT scan and enzyme levels study are complimentary to the clinical suspicion.

Keywords: Acute pancreatitis, Serum amylase, Serum lipase
limiting, up to a fifth of patients develop a severe attack that can be fatal. The presentation of wide spectrum of symptoms gives the clinician a heartbreaking exercise to bring back the patient from the clutches of the disease process. The incidence of AP has been increased recently (5-11 cases per 100,000), representing the 3rd cause of admission in gastroenterology Units in the United States and the 5th cause of death due to non-malignant diseases.²

The hospital admission rate for AP is 9.8 per year per 100,000 population in the UK, although worldwide, the annual incidence may range from 5 to 50 per 100,000.³ AP can either resolve quickly, or cause a systematic inflammatory response leading to a multi-organ failure and death.⁴

Mortality rate estimated is 5%, which may be as high as 30% in the most severe cases. The course and severity of AP can fluctuate rapidly and unpredictably. Alcohol abuse and biliary tract diseases are etiological in most of the cases.⁵ Alcoholism ranks first as the etiological factor.

The diagnosis is made by clinical examination which is supported by laboratory investigations and imaging studies.⁶ AP should be suspected in a patient with history of biliary tract disease or alcoholics presenting with pain abdomen in the epigastrium radiating to the back, vomiting, and distension of the abdomen.

Serum amylase and lipase have been used as biochemical markers to diagnose AP.⁷ The current UK guidelines for the management of AP has emphasized the greater accuracy of serum lipase compared to amylase.⁸

Imaging studies such as ultrasonography and computed tomography (CT) supports the diagnosis in most of the cases. USG has become routine investigation for diagnosis of AP as it is non-invasive and cost-effective and is useful as baseline for sequential examination of pancreas. CT scan plays an important role in the prognostic information and identifying complications.

The treatment of AP is largely supportive. Use of antibiotics, analgesics, IV fluids and drugs, which reduce the pancreatic secretion.

In 2009, the Healthcare Cost and Utilization Project National inpatient sample identified 274,119 individuals discharged from the hospital with AP, representing a 30% increase from 2000 and necessitating a median length of stay of 4 days. AP contributed to, and/or was responsible for, 8653 deaths in 2009, representing an underlying cause of death rate of 1 per 100,000 and ranking it as the 14th leading cause of gastrointestinal death with a cost of 2.6 billion dollars in inpatient expenses.⁹ Hence AP is an economic burden to patients and the health care system. A clinical description of AP was first presented in 1652 by the Dutch anatomist Nicholas Tulp, and nearly 350 years have passed, there continue to be many unanswered questions. Despite more than 100 years of experience and thousands of experimental and clinical studies, the management of AP still remains challenging and mortality rates remains 5%. In spite of technical advances in medical and surgical fields, AP remains a major cause of morbidity and mortality.¹⁰

The objective of this study was to determine the pattern and aetiology of AP in the rural population and to study the role of serum amylase, lipase and imaging studies in the diagnosis of AP.

METHODS

This prospective descriptive study was conducted at Sri Adichunchanagiri Hospital and Research Centre, B G Nagara, to study the pattern and aetiology of AP in the rural population and to determine the role of serum amylase, lipase and imaging studies in the diagnosis of AP.

Sampling method

At our centre all consecutive patients who fulfilled inclusion criteria were included in the study. We considered the aetiology to calculate the sample size. Among different aetiology in our context idiopathic pancreatitis was lowest with prevalence of 11.7%. Considering the prevalence at 80% power and 5% confidence level the estimated sample size was 70. The study group of 60 patients included those who were admitted with AP at our hospital, during the period from December 2017 to June 2019 over duration of 18 months. In our study 85% of the determined sample size was met in the stipulated study duration of 18 months.

The data were collected after taking informed written consent from the all the patients who met the inclusion criteria, and the study was designed to study the etiological, clinical, biochemical and radiological profile in patients of AP. Patients included were those with history suspicious of pancreatitis such as pain abdomen mainly in the epigastrium and/ umbilical region radiating to the back and on evaluation were diagnosed with AP.

All patients diagnosed of AP and acute on chronic pancreatitis between the age of 16 to 70 years, giving valid information and written consents, were included in the study. Those patients below 16 years of age and those above 70 years, those with traumatic pancreatitis, patients with severe pre-existing co-morbidities, immunocompromised patients, those associated with malignancies and patients not willing to participate in the study were excluded.

Statistical analysis

Descriptive statistics expressed in terms of means, proportions was carried out.

Institutional ethical committee clearance was obtained for the study.
**RESULTS**

The following observations were made from the study and results were analysed. The clinical data pertaining to the cases in respect of detailed history, symptomatology, signs, investigations and radiological assessment were performed and recorded in the tables respectively.

In our study, the youngest patient was 25 years old and the eldest being 65 years. The highest incidence was noted in the age group of 41-50 years accounting for 35% of the patients, followed by 25% in the age group of 31-40 years, 18.3% in age group of 51-60 years, 13.3% in age group of 21-30 years and least of 8.4% in 61-70 years age group. The mean age of presentation was 44±10 years.

In our study, male preponderance was seen with 86.7% (52 cases) of total patients and 31.6% (8 cases) were females, with male to female ratio of 6.5:1.

The duration of hospital stay in majority of cases (78.3%) was <5 days, followed by 5-10 days in 16.7% cases and 11-15 days in 5% of cases in study group.

Most of the study subjects were laborers accounting for 41.6% of total cases followed by farmers accounting to 31.7% cases and least among the businessmen and housewives accounting for 13.3% and 10% respectively.

Our study showed alcohol abuse was the main etiological factor in 45 cases and all were males accounting for 75% of cases and among 8 cases (7 in males, and one in female) cause was unknown accounting for 11.7% of patients.

In our study all the patients presented with pain abdomen (100%), 48 cases (80%) presented with radiation of pain to back, 33 cases (55%) with vomiting, fever was present in 10 cases (16.7%) and 3 cases (5%) with jaundice out of which 2 cases of jaundice in males were due to alcoholic pancreatitis, one in female due to biliary pancreatitis.

On physical examination, in our study, all the patient had upper abdominal tenderness (100%), followed by abdominal distension seen in 4 cases (6.7%), epigastric mass in 4 cases (6.7%) and icterus in 3 cases (5%). The number of patients with the diagnosis of AP and normal enzyme levels was 25 (41.7%) in amylase group and 18 (30%) in lipase group. Amylase and lipase levels in 10 (16.7%) and 12 (20%) patients respectively were between upper limit and 2-fold elevation. Amylase levels in 9 (15%) patients and lipase levels in 10 (16.7%) patients were between >2-fold elevation <3-fold elevation levels. The number of patients with more than 3-fold elevation of amylase levels was seen 16 patients (26.7%) lipase levels in 20 patients (33.3%) and among these in 2 patients’ amylase and lipase levels were >1000 IU/l (Table 1).

In our study plain X-ray abdomen was normal in 58 cases (86.7%) and multiple air fluid level was seen in only 2 cases (13.3%). In our study in 25 (41.7%) cases pancreas was sonographically normal. In 30 cases (50%) revealed bulky pancreas, peripancreatic fluid collection was seen in 5 cases (8.3%), pseudocyst was seen in 4 cases (6.7%) cholecystolithiasis were identified in 7 (11.7%) patients, choleodocholithiasis in 1 patient (1.7%), ascites were detected in 4 patients (6.7%) and pleural effusion was seen in 2 cases (3.3%).

In our study in 14 cases (23.3%) CT scan were normal, 46 cases (76.7%) showed pancreatic enlargement, pancreatic pseudocyst was identified in 4 cases (6.7%), GB stones was present 7 cases (11.7%), CBD stone was identified in one case (1.7%), ascites was seen in 4 cases (6.7%) and pleural effusion was identified in 2 cases (3.3%). Of the 60 patients included in this study, 53 cases (88.3%) were mild pancreatitis and 7 cases (11.7%) were moderate pancreatitis as per Ranson’s score, no cases of severe pancreatitis cases were present.

In our study, all patients required fluid resuscitation (100%). All patients required injectable non opioid analgesics (100%). 10 patients (16.7%) had been started on antibiotics. Nearly 50 patients (83.3%) required antiemetics. In our study, 7 patients diagnosed to have gall stone induced pancreatitis underwent laparoscopic cholecystectomy (10.5%) and one patient with CBD stone underwent ERCP and sphincterotomy. In our study 4 cases (6.7%) developed pancreatic pseudocyst and ascites and 2 cases (3.3%) had pleural effusion. All cases were managed conservatively. There was no mortality in our study (Table 2).

| Limits of enzyme level | Total no patients with amylase levels | Total no of patients with lipase levels |
|------------------------|--------------------------------------|---------------------------------------|
| Within-normal-limits amylase (0-86); lipase (0-64) | 25 | 18 |
| Between upper limit and 2 fold-elevation amylase (87-172); lipase (65-128) | 10 | 12 |
| >2 fold-elevation and <3 fold-elevation Amylase (173-258); lipase (129-192) | 9 | 10 |
| >3 fold-elevation amylase >258; lipase >192 | 16 | 20 |

Continued.
Table 2: Complications.

| Complications          | Frequency | %  |
|------------------------|-----------|----|
| Pseudocyst             | 4         | 6.7|
| Pleural effusion       | 2         | 3.3|
| Pancreatic ascites     | 4         | 6.7|

**DISCUSSION**

AP is a common emergency, accounting for 3% of all patients admitted with acute pain abdomen. AP is a disease that varies in severity ranging from a mild and self-limiting illness to a very severe and rapidly progressive condition leading to multiple organ failure and eventually to death.

An early diagnosis and identification of those who are at risk of development of severe disease and rapid institution of therapy might reduce the morbidity and mortality. If the etiological factors are known and can be eliminated, further attacks can be prevented. Frequent occurrence of serious complications has brought into fore the issues regarding management. While diagnosing a case of AP, a thorough history, a complete physical examination and biochemical tests are necessary. Imaging confirmation may be required.

This was a prospective descriptive study on 60 cases of AP admitted during the study period who have met the inclusion criteria in Adichunchanagiri hospital and research college attached to Adichunchanagiri institute of medical sciences were subjected to study.

In this study, analysis of clinical presentation of AP was done. Relevant investigations were carried out and patients appropriately managed depending upon the aetiology and severity of AP. Studies on epidemiology of AP differ considerably in the incidence of AP, secondary to inadequate reporting, difficulty in making in the diagnosis, difference in patient populations, prevalence of alcohol intake, and tertiary care referral bias. In fact, because patients with mild pancreatitis may never be diagnosed, the true incidence of AP may be higher than that suggested in the literature.

**Age distribution**

Our study revealed incidence of AP is maximum in the age group of 41-50 years of 21 cases (35%), followed by 15 cases (25%) in the age of 31-40 years. Minimum age of the study group was 25 years and eldest was 65 years with mean age of distribution was 44±10 years (Table 3).

**Sex distribution**

There was a male predominance in our study with males accounting for 86.7% of patients and females accounting for 13.3% with male: female ratio of 6.5:1. The other studies also had a higher percentage of males. This again could be attributed to alcohol abuse which was the main etiologic agent (Table 4).

**Etiology**

**Alcoholic pancreatitis**

Excess alcohol intake is the most common aetiology of AP in males. Several studies suggest that it may be the most common aetiology of AP worldwide.

This study demonstrates that alcohol (75% of the total patients) was the most common cause of pancreatitis followed by gallstones (13.3%), whereas, remaining 11.7% patients were idiopathic i.e. there were no demonstrable cause identified. Most of the study subjects were from rural setup where more people are under-educated, hence people are prone to addictions leading to excessive alcohol consumption.

In our study 45 cases (75%) of alcoholic pancreatitis was seen in males and none of the in the females. Among 7
cases of GB stones (11.7%), 6 were seen in females and one was male and one case of CBD stone was present in female and among 7 cases of idiopathic pancreatitis (11.7%), 6 were males and one female. Above correlation has proved the higher incidence of alcoholism as the major etiological factor in Indian males possibly was due to higher consumption of alcohol by the male community and more common occurrence of gall bladder disease in females in India was probably due to faulty dietary habits, and in none of the females, alcoholism was etiological factors, since very few females consume alcohol in India.

Studies conducted by Thomson SR et al and Imrie CW et al have reported that alcoholic AP was more prevalent in men and younger patients.12,21 Our study was comparable to the following studies where alcohol abuse is the most common cause (Table 6).

Acute biliary pancreatitis

Gallstone pancreatitis is most common in women between 50-70 years of age.

In this study 7 cases out of 60 cases (11.6%) had gall stones as a cause of AP, among which 6 were females and one was male and one case in female was due to CBD stone.

Present study is contrary to the following studies by Choudhuri et al, Kumar et al, Nagesh et al, Kurrey et al and Sand J et al where biliary disease was the most common cause, but it is comparable to previous studies by Abruzzo et al, Imrie CW et al, Jacob ML et al and Kashid A et al where biliary disease was the most common cause, but it is comparable to previous studies by Choudhuri et al, Kumar et al, Nagesh et al, Kurrey et al and Sand J et al where biliary disease was the 2nd most common cause of AP (Table 7).12,13,21,22,24,25,27

Clinical presentations

In the present study all the 60 cases (100%) had pain abdomen, 48 cases (80%) of them had radiation of pain to the back. Vomiting was seen in 33 cases (55%), fever in 10 cases (16.7%) while jaundice was present in 3 cases (5%). Our study was comparable with the following studies (Table 8).

Physical examination

The usual findings on a physical examination are abdominal distension, tenderness, guarding and absent bowel sounds. Fever associated with AP is generally low grade. High grade temperature may indicate the development of infected pancreatic necrosis and associated fluid collection or cholangitis particularly if icterus is present. In our study all the 60 cases (100%) had with tenderness in the epigasitrus, abdominal distension was seen in 4 cases (6.7%) while epigastric mass was present in 4 cases (6.7%). Icterus was present in 3 cases (5%) (Table 9). Our study was comparable to the following study regarding signs of AP mentioned in Table 9.

Serum amylase and lipase

In our study number of patients with the diagnosis of AP with normal serum amylase and lipase levels were seen 25 cases (41.7%) and 18 cases (30%) respectively. Amylase levels in 10 patients (16.7%) and lipase levels in 12 patients (20%) was between upper limit and 2-fold elevation. More than 2-fold elevation and >3-fold elevation in serum amylase was seen in 9 (15%) patients and serum lipase in 10 (16.7%) patients and more than 3-fold elevation in serum amylase was present in 16 patients (26.7%) and lipase in 20 (33.3%).

In this present study- (a) in majority of the cases there was no marked elevation seen in serum amylase and lipase levels. >3-fold elevation in serum amylase and lipase was seen 26.7% and 33.3% of cases respectively and in remaining cases <3-fold elevation was seen. Serum amylase in 41.7% of cases and serum lipase levels in 18% of cases were within normal limits; (b) serum amylase and lipase levels was helpful in making a diagnosis of AP only in 26.7% and 33.3% of cases respectively whereas serum amylase levels in 73.4% of cases and lipase in 66.7% of cases were not useful in diagnosis of AP; (c) serum amylase and lipase levels in two cases were >1000 IU/l, and were not a case of severe pancreatitis, hence serum amylase and lipase estimation has not correlated with the severity of the disease in our study. Traditionally, serum enzyme levels 3 times above the normal are considered to be diagnostic of AP.

Abruzzo et al found hypoamylasemia in patients with AP and severe destruction of the pancreas.32 Adams et al found an inverse relation between amylase and severe morphological changes.33 Our study clearly demonstrated that patients with even normal enzyme levels and/or with only mild elevations may have AP which was further confirmed by USG and CT scan. Clinical symptoms and imaging findings are not correlating with enzymatic levels, this may be due to delay in the presentation to the hospital.

Serum bilirubin was found to be within normal limits in 95% (57 cases), whereas it was raised in 5% (3 cases). Out of these 2 patients had AP due to alcoholism and in one patient due to gallbladder calculi. CRP was positive in 7 cases (11.6%) which were all moderate pancreatitis and was negative in 53 patients (88.3%) of mild pancreatitis.

Ultrasonography

In this study USG was normal in 25 cases (41.7%), 30 cases (50%) were reported of having bulky pancreas, peripancreatic collection was noted in 5 cases (8.3%) while pseudocyst and ascites were identified in 4 cases (6.7%), pleural effusion were seen in 2 cases (3.3%), GB stones were identified in 7 cases (11.7%) and CBD stone in one case (1.7%). Sensitivity of the ultrasonography is only 68%. Thus, a negative ultrasound does not rule out AP.
**CECT abdomen**

CT scan reveals all the features of AP and also complications such as necrotizing pancreatitis. Its greatest advantage is its utility when retroperitoneum cannot be visualized on USG due to bowel gas. In our study in 46 cases (76.7%) had pancreatic enlargement. Pseudocyst and ascites were identified in 4 cases (6.7%), pleural effusion was reported in 2 cases (3.3%) GB stone was present in 7 cases (11.7%), CBD stone in one case (1.67%). CT scan was normal in 14 cases (23.3%).

**Stratification according to Ranson’s score**

Early assessment and prediction of severity are of outstanding importance to avoid complications of pancreatitis and to decrease morbidity and mortality due to pancreatitis. Ranson’s scoring system is a first initial scoring system for the stratification of patients with pancreatitis. Present study showed that 88.3% of the total patients had mild pancreatitis, 11.7% of total patients had moderate pancreatitis, no cases of severe pancreatitis with no mortality in our study. Nagesh in his study of 49 patients at Bengaluru, demonstrated 77.4% had mild pancreatitis, whereas 22.6% patients had severe pancreatitis.

**Management modalities**

Intravenous antibiotics should be initiated in patients with documented infected pancreatic necrosis. in these cases, the choice of antibiotic is the best guided by the identification and sensitivity testing of the offending microorganism. The treatment of choice for infected necrosis is surgical debridement. This approach is based on clinical experience that infected necrosis is usually fatal without debridement, delay in surgery increases mortality, and at least half of the deaths in necrotizing pancreatitis are as a result of pancreatic infection. To date, no prospective randomized trial has been conducted that compares surgery with medical therapy and such a study will likely never be done. The anecdotal experience with radiological and endoscopic drainage is growing.

In this study the main line of treatment was conservative in all cases (100%), as maximum cases were mild pancreatitis. Only 8 Cases (13.3%) required surgical interventions subsequently. Our study is comparable with a study conducted by Rehman et al in his study of 38 patients, demonstrated that 89.5% patients were managed with a conservative line of management while 10.5% patients required surgical interventions subsequently.

Out of 8 cases which required surgical interventions, 7 cases had GB stones all of them underwent laparoscopic cholecystectomy in the subsequent admissions and one patient with CBD stone was referred to gastroenterologist for ERCP and Sphincterotomy.

**Complications**

In our study, 4 cases (6.7%) developed pseudocyst and ascites and 2 cases (3.3%) had pleural effusion. All of them were managed conservatively. Pancreatic pseudocyst patients were followed up with serial USG and cyst regressed in size and hence managed conservatively.

In our study maximum cases were mild pancreatitis with duration of hospital stay of 5-10 days. All the cases were followed up for a period of 3 months and were all asymptomatic with no mortality. Alcoholic pancreatitis patients were counselled to stop consuming alcohol and de addiction was attempted with the help of psychiatrist in few cases. There was no subsequent admission for recurrence during the follow up period of 3 months. Relatively small study group and study included only the rural population mainly, are the limitations of this study.

---

### Table 3: Comparison of mean age distribution in different studies.

| Mean age in years | Buchler et al11 | Choudhuri et al12 | Kashid et al13 | Pupelis et al14 | Entee et al15 | Corfield et al16 |
|------------------|----------------|------------------|----------------|----------------|----------------|-----------------|
|                  | 55.1           | 44.89            | 35             | 47             | 42.4           | 60              |

### Table 4: Comparison of sex distribution in different studies.

| Sex             | Buchler et al11 | Choudhuri et al12 | Kashid et al13 | Pupelis et al14 | Present study |
|-----------------|-----------------|-------------------|----------------|----------------|---------------|
| Male (%)        | 61              | 66.6              | 70.91          | 73.7           | 86.7          |
| Female (%)      | 39              | 33.4              | 29.09          | 26.3           | 13.3          |

### Table 5: Comparison of male: female ratio distribution in different studies.

| Male: female ratio | Thomson et al17 | Gillespie et al18 | Bhimwal et al19 | Present study |
|-------------------|------------------|------------------|-----------------|---------------|
|                   | 1.05:1           | 2:1              | 1.7:1           | 6.5:1         |
Table 6: Comparison of etiological distribution in different studies.

| Etiology      | Choudhuri et al\textsuperscript{12} | Kumar et al\textsuperscript{22} | Nagesh et al\textsuperscript{23} | Kurrey et al\textsuperscript{24} | Sand et al\textsuperscript{25} | Present study |
|---------------|-------------------------------------|---------------------------------|---------------------------------|---------------------------------|-----------------|---------------|
| Alcohol (%)   | 45.83                               | 80                              | 81.1                            | 58                              | 70              | 75            |
| Biliary (%)   | 26.04                               | 16                              | 15.1                            | 28                              | 20              | 13.3          |
| Idiopathic (%)| 19.37                               | 4                               | 3.77                            | 14                              | 10              | 11.7          |

Table 7: Comparison of etiological distribution in different studies.

| Etiology      | Blarney et al\textsuperscript{27} | Imrie et al\textsuperscript{21} | Jacob et al\textsuperscript{26} | Buchler et al\textsuperscript{11} | Kashid et al\textsuperscript{13} | Present study |
|---------------|-----------------------------------|---------------------------------|---------------------------------|---------------------------------|----------------|---------------|
| Biliary (%)   | 44                                 | 51                              | 47                              | 45                              | 36.4           | 13.3          |
| Alcohol (%)   | 33                                 | 26                              | 13                              | 33                              | 29.1           | 75            |
| Idiopathic (%)| 24                                 | 13                              | 22                              | 14.5                            | 11.7           |               |

Table 8: Comparison of symptoms in different studies.

| Symptoms          | Kumar et al\textsuperscript{22} (%) | Forsmark et al\textsuperscript{28} (%) | Webster et al\textsuperscript{29} (%) | Albo et al\textsuperscript{30} (%) | Saxona et al\textsuperscript{31} (%) | Present study (%) |
|-------------------|-------------------------------------|----------------------------------------|------------------------------------|-----------------------------------|------------------|------------------|
| Pain abdomen      | 100                                 | 100                                    | 100                                | 94                                | 90               | 100              |
| Radiation of pain | 70                                  | 64                                     | 78                                 | 72                                | 66               | 80               |
| Vomiting          | 40                                  | 52                                     | 75                                 | 83                                | 48               | 33               |
| Fever             | 34                                  | 28                                     | 42                                 | 18                                | 86               | 16.7             |
| Jaundice          | 20                                  | 10                                     | 16                                 | 14                                | 28               | 5                |

Table 9: Comparison of signs in different studies.

| Signs             | Nagesh et al\textsuperscript{23} (%) | Present study (%) |
|-------------------|-------------------------------------|------------------|
| Abdominal tenderness | 100                                 | 100              |
| Abdominal distension | 33.96                               | 6.7              |
| Epigastric mass    | 3.77                                 | 6.7              |

Table 10: Comparison of complications in different studies.

| Complications         | Buchler et al\textsuperscript{11} (%) | Choudhuri et al\textsuperscript{12} | Kashid et al\textsuperscript{13} (%) | Present study (%) |
|-----------------------|---------------------------------------|------------------------------------|-----------------------------------|------------------|
| Acute fluid collection| -                                     | 40.5                               | 34.54                             | -                |
| Pseudocyst            | 2.45                                  | 24.9                               | -                                 | 6.7              |
| Ascites               | -                                     | -                                  | 34.54                             | 3.3              |
| Pleural effusion      | -                                     | -                                  | 40.5                              | 18.18            |
| Pancreatic necrosis   | 42.5                                  | 40.5                               | 0.5                               | 5.45             |
| Pancreatic abscess    | 0.5                                   | -                                  | -                                 | -                |

CONCLUSION

Pancreatitis is a common cause of acute abdomen in patients presenting to the surgery department. Most patients develop a mild and self-limiting course of the disease. The conclusion from this study was that in AP one should not only rely on enzyme level elevations for diagnosing AP. Patients with only a small increase in amylase and/or lipase levels or even with normal levels may also have or develop AP. Therefore, the clinician who makes the initial diagnosis of AP must evaluate the disease independently of the enzyme level elevations. High degree of suspicion is required; USG, CT, and enzyme levels study are collectively complimentary to the clinical suspicion. The incidence of AP in rural population is increasing nowadays probably due to alcohol abuse. Severity of pancreatitis can be determined early in the disease process. The most cases of AP have a self-limiting course. Alcoholism ranks first as the etiological factor. Among the males, alcoholism is the most common etiological factor. The management of the AP is mainly conservative and surgical interventions are required for biliary pancreatitis and for complications of AP. Alcohol is the most common etiological factor, social awareness should be created among the rural folks regarding the ill
effects of the alcohol abuse and they should be counselled for abstinence.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Fitz R. AP: a consideration of pancreatic haemorrhage, haemorrhagic, suppurative and gangrenous pancreatitis, and of disseminated fat necrosis. Boston Med Surg J. 1889;181.
2. Opie E. The aetiology of acute haemorrhagic pancreatitis. Bull John Hopkins Hospital. 1902;(74):398-401.
3. Peery AF, Dellon ES, Lund J, Crockett SD, Gowan CE, Bulsiewicz WJ, et al. Burden of gastrointestinal disease in the United States: 2012 update. Gastroenterology. 2012;143:1179-87.
4. Goldacre MJ, Roberts SE. Hospital admission for acute pancreatitis in an English population, 1963-98: database study of incidence and mortality. BMJ. 2004;328(7454):1466-9.
5. Quinlan JD. Acute pancreatitis. Am Fam Physician. 2014;90(9):632-9.
6. Steinberg W, Tenner S. Acute pancreatitis. N Engl J Med. 1994;330(17):1198-210.
7. Touuli J, Smith M, Bassi C, Locke D, Telford J, Freeny P, et al. Guidelines for the management of acute pancreatitis. J Gastroenterol Hepatol. 2002;17:15-39.
8. Matull WR, Pereira SP, O'Donohue JW. Biochemical markers of acute pancreatitis. J Clin Pathol. 2006;59(4):340-4.
9. 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(3):1-9.
10. Peery AF, Dellon ES, Lund J, Crockett SD, McGowan CE, Bulsiewicz WJ, et al. Burden of gastrointestinal disease in the United States: 2012 update. Gastroenterology. 2012;143(5):1179-87.
11. Büchler MW, Gloor B, Müller CA, Friess H, Seiler C, Uhl W. Acute necrotizing pancreatitis: treatment strategy according to the status of infection. Ann Surg. 2000;232(5):619-26.
12. Choudhuri G. AP Experience at Sanjay Gandhi PGI of Medical Sciences, Lucknow, Appendix 1-B, in Management of AP, Jaslok Hospital. Path Shodh. 2006;176-8.
13. Kashid A. AP Experience at Manipal Hospital, Bangalore. Appendix 1-A, in Management of AP, Path Shodh. 2006;173-5.
14. Pupelis G. Conservative approach in the management of severe AP: eight-year experience in a single institution. HPB. 2008;10:347-55.
15. Entee GP, Gillen P, Peel AL. Alcohol induced pancreatitis: social and surgical aspects. Br J Surg. 1987;74(5):402-4.
16. Corfield AP, Cooper MJ, Williamson RC. Acute pancreatitis: a lethal disease of increasing incidence. Gut. 1985;26(7):724-9.
17. Thomson SR, Hendry WS, McFarlane GA, Davidson AI. Epidemiology and outcome of acute pancreatitis. Br J Surg. 1987;74(5):398-401.
18. Gillespie WJ. Observation on AP. Br J Surg. 1973;60:63-5.
19. Bhimwal RK, Makwana M, Panwar RR, Lal K. A prospective study of clinical, biochemical and radiological features in pancreatitis. Int J Adv Med. 2017;4:1386-93.
20. Villanueva M, Hijona E, Bañales JM, Cosme A, Bujanda L. Alcohol consumption on pancreatic diseases. World J Gastroenterol. 2013;19(5):638-47.
21. Imrie CW. Observations on AP. Br J Surg. 1974;61:539-44.
22. Kumar A. Clinical Study, Management of Complications of AP, Bangalore, Karnataka: Bangalore Medical College and Research Institute. Gandhi University of Health Sciences; 2010.
23. Nagesh VR. Clinical Study of AP and its Management, Karnataka, Hubli. Rajiv Gandhi University of Health Sciences; 2011.
24. Kurrey LK, Patel V, Gaharwar APS, Jayant V, Pandre SK, Kumar S. Clinical Study of Pancreatitis and Its Management: A Prospective Study. IJSS J Med. 2017;3(3):27-33.
25. Sand J, Välíkoski A, Nordback I. Alcohol consumption in the country and hospitalizations for acute alcohol pancreatitis and liver cirrhosis during a 20-year period. Alcohol Alcohol. 2009;44(3):321-5.
26. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. Gastroenterology. 2013;144(6):1252-61.
27. Jacobs ML, Daggett WM, Civette JM, Vasu MA, Lawson DW, Warshaw AL, et al. AP: analysis of factors influencing survival. Ann Surg. 1977;185(1):43.
28. Forshmark CE, Baillie J, AGA Institute Clinical Practice and Economics Committee, AGA Institute Governing Board. AGA Institute technical review on acute pancreatitis. Gastroenterology. 2007;132(5):2022-44.
29. Webster PD, Spanhouri JB. Pathophysiology and management of AP, Hosp Pract. 1974;9:59-66.
30. Albo R, Silen W, Goldman L. A critical clinical analysis of AP. Arch Surg. 1963;86(6):1032-8.
31. Saxona A, Reynolds JT, Doolas A. Management of pancreatic abscess. Ann Surg. 1981;194:545-51.
32. Abruzzo JL, Homa M, Houck JC, Coffey RJ. Significance of the serum amylase determination. Ann Surg. 1958;147:921-30.
33. Adams JT, Libertino JA, Schwartz SI. Significance of an elevated serum amylase. Surgery 1968;63:877-84.

34. Rehaman SA, Chandrashekhar S, Reuben PJ. Clinical study of AP. J Evol Med Dent Sci. 2015;4:10142-55.

Cite this article as: Shekar SC, Gowda SNS, Narayan N, Nagraj A, Venugopal V, Kanmani RM. A study of etiological clinical biochemical and radiological profile of patients with acute pancreatitis in rural population. Int Surg J 2021;8:2624-32.