

# CLINICAL STUDY OF ACUTE PANCREATITIS AND ITS MANAGEMENT

1. DR AKSHATHA H S – Senior Resident
2. DR PRAMOD T\* – Associate Professor
3. DR ANOOP S RAO – Senior Resident

\*corresponding author

Email – [pramodthejoram@gmail.com](mailto:pramodthejoram@gmail.com)

Mobile – 9844558073

Mailing address – No 48, 31<sup>st</sup> B cross, 4<sup>th</sup> T block, Corporation layout, Jayanagar, Bangalore 560041

Institution – KODAGU INSTITUTE OF MEDICAL SCIENCES, MADIKERI, KARNATAKA

## Abstract

### Background

Acute pancreatitis includes a wide spectrum of disease, from mild self-limiting symptoms to a fulminant process with multiple organ failure and high mortality. Alcohol and gallstones are the most common causes. Most patients can be managed conservatively. This study analyses the incidence, clinical presentation and management of acute pancreatitis.

### Methods

In our study 50 patients with acute pancreatitis were studied and data collected. These were analysed, tabulated and compared.

### Results

The peak incidence was in the 4<sup>th</sup> decade in males (40%) and 5<sup>th</sup> decade in females (50%). The mean age group in our study is 40.72 years. 80% were male patients and alcohol was the cause in 72% patients. Most of the patients had pain abdomen and vomiting. Amylase and lipase were elevated in most patients. CT scan was diagnostic in 90% patients and ascites was seen in 60% patients. 64% patients had mild pancreatitis and 88% patients were managed conservatively. Mean hospital stay in our study was 4.75 days. More than 90% patients improved.

### Conclusion

Alcohol is the main cause, seen in the 4<sup>th</sup> decade in males. CT scan is diagnostic, lipase is elevated in most cases. Most cases are mild disease and managed conservatively.

### Key words

Acute pancreatitis, alcohol, gallstones, serum lipase

## INTRODUCTION

Acute pancreatitis is a common acute clinical condition requiring emergent care. The etiological factors are varied and may include biliary stones, alcoholism, trauma, drugs, and metabolic and idiopathic causes. Most patients have acute edematous pancreatitis with interstitial inflammation and favorable clinical outcome. Acute

pancreatitis includes a wide spectrum of disease, from mild self-limiting symptoms to a fulminant process with multiple organ failure and high mortality.<sup>1</sup>

Some patients suffer acute necrotizing pancreatitis with severe parenchymal necrosis and rapid deterioration to multiple organ failure frequently requiring organ support. Several scoring systems are available involving clinical and laboratory data, which can differentiate mild from severe pancreatitis.<sup>2</sup>

Acute pancreatitis has been recognized since antiquity<sup>3</sup> but the importance of pancreas and the severity of its inflammatory disorders were realized only in middle of 19th century.<sup>4</sup>

The nature of disease was recognized way back in 1925 when Moynihan described acute pancreatitis as –The most terrible of all the calamities that occur in connection with abdominal viscera<sup>5</sup> –but even today with technical advantage in medical and surgical field acute pancreatitis remains a major cause of morbidity and mortality.<sup>6,7</sup>

Acute pancreatitis is related to alcohol or biliary tract stone disease in 80% of Cases. The remaining 10% is related to metabolic factor, drugs and other condition and 10% are idiopathic.<sup>4,7</sup>

Acute pancreatitis has been classified way back in 1882 in to apoplectic and sub-acute forms. Later in 1963 in a symposium at Marseilles a classifications based on the morphology was accepted until 1993 when it was realized that the earlier classification was possible only after surgery or during autopsy, so a classification system was very much needed at the outset of the disease for a working diagnosis. Finally a classification system, which is clinically based, was established in the Atlanta symposium in 1993.<sup>8</sup>

Acute pancreatitis is a pathological broad spectrum of disease ranging from parenchymal edema to severe necrotizing pancreatitis. Clinical presentations vary from mild abdominal discomfort to hypotension, metabolic derangement, sepsis, fluid sequestration, multiple organ failure and death. 90% experience mild to moderate course and self-limited, and 10% experience a severe life threatening form of acute pancreatitis. Based on the above it is presently classified into mild acute pancreatitis associated with minimal organ dysfunctions and uneventful recovery, and severe acute pancreatitis associated with organ failure and/or local complications such as necrosis, abscess or pseudo cyst.

Diagnosis remains clinical and can be supported by 1.5 – 2 fold increase above the upper limit of normal of serum amylase. But an estimation of serum lipase, trypsinogen or isoamylase assay are confirmatory<sup>9</sup> and will increase the diagnostic yield. Supportive radiological procedures are sonography, computed tomography and MRI. Currently CECT is the imaging modality of choice where areas of hypo perfusion correlate with necrosis.<sup>10</sup>

The treatment of acute pancreatitis is largely supportive. Patient with mild disease are treated by eliminating oral intakes, instituting intravenous hydration and providing frequent parenteral analgesia. Use of antibiotics and drugs, which reduce the pancreatic secretion, have been studied extensively. In the surgical management there are various diagnostic, prophylactic and therapeutic options available for both

the disease process and its complication but none of them have shown to improve the outcome in acute pancreatitis.

An increased mortality rate associated with the disease is due to inability to assess the severity of the disease at the outset. Various prognostic scoring systems have been developed involving multiple factor and single factor. The drawback with the current severity scoring system is that they are cumbersome and time consuming and lack sensitivity and specificity. In fact their necessity has been questioned.<sup>11</sup>

The incidence of acute pancreatitis (AP) has been rising over the years in western countries<sup>12, 13, 14</sup> and, in fact, this disease represents a significant cause of morbidity and mortality regardless of its etiology.<sup>14</sup>

This increasing incidence seems to be related to several factors. On the one hand, life expectancy has risen, and the mean age of the first episode reaches his peak around the sixth decade. On the other hand, in this century, we face the obesity epidemic, and as it is known, overweight is an individual risk factor for biliary gallstones, one of the main causes of acute pancreatitis<sup>12</sup>

Due to change in classification system, lack of statistics in our country and lack of accuracy of scoring system, a better sensitive, specific, severity scoring system which can predict at the outset of the disease is very much needed at present.

The aim of this prospective study is to analyse data concerning incidence, etiology and management of acute pancreatitis.

## **MATERIALS & METHODS**

The study group has evaluated 50 consecutive patients with acute pancreatitis admitted to NMCH & RC, Raichur between November 2015 to October 2017. It is a prospective Hospital base study.

### **Inclusion criteria:**

- Patients of both the sex.
- Age above 18Years

### **Exclusion criteria:**

- Blunt Injury Abdomen cases
- Post-operative cases
- Post ERCP pancreatitis
- Malignancy

### **Method of collection of data**

All the patients were evaluated thoroughly at the time of admission and frequently in those showed deterioration their clinical status to find out associated local/systemic complication.

Serum amylase and lipase was investigated immediately on presentation. Preliminary USG of

Abdomen and Pelvis was done on the same day of presentation. CECT was done after 48 hours

in all patients except in persistent ARF.

In the absence of gallstones and / or history of significant history of alcohol use, a serum triglyceride levels done (>1000mg/dl taken as diagnostic). After doing all available investigation if no cause was found, considered as idiopathic pancreatitis. Severity assessment done with Atlanta classification. All patients were put on conservative line of management.

Patients were followed up daily clinically and serum amylase was repeated on the 3rd day. Repeat USG/CT/MRI abdomen & pelvis was done if patient's condition remained same or deteriorated. If the patient developed any of the above mentioned complications, such patients were evaluated for medical/surgical management of the same complications. Patients were informed about any surgical procedure and consent was taken for the same.

Patient data collected regarding age, gender, complaints, aetiology, history of alcoholism, calculus cholecystitis, trauma to abdomen etc. were evaluated. Complications if developed during the course of treatment and later on were assessed in detail. Management of these complications was assessed in detail and the patients were followed up regularly.

Initial conservative management consists of nasogastric suction, intravenous administration of fluid, antibiotic and supportive care in all patients.

An indwelling urinary catheter was placed in most patient to allow close monitoring of urine output, and a CVP catheter was frequently introduced (in necessary cases). Most of the systemic complication was managed by conservative and supportive care including ICU.

## RESULTS

The peak incidence was in the 4<sup>th</sup> decade in males (40%) and 5<sup>th</sup> decade in females (50%). The mean age group in our study is 40.72 years.

**Table 1: Age distribution**

AGE GROUP IN YEARS	MALE		FEMALE		TOTAL	
	NO	%	NO	%	NO	%
21 – 30	11	22	1	2	12	24
31 – 40	13	26	2	4	15	30
41 – 50	10	20	4	8	14	28
51 – 60	5	10	1	2	6	12

>= 60	1	2	2	4	3	6
TOTAL	40	80	10	20	50	100

**Table 2: Sex Distribution**

SEX	NO. OF PATIENTS	PERCENTAGE
MALE	40	80
FEMALE	10	20
TOTAL	50	100

**Table 3: Etiology**

Etiology	No of patients	Percentage
Alcohol	36	72
Gall stones	6	12
Hypercalcemia	0	0
Hypertriglyceridemia	4	8
Idiopathic	4	8

**Table 4: Clinical Features**

CLINICAL FEATURE	NO. OF PATIENTS	PERCENTAGE
PAIN ABDOMEN	50	100
VOMITING	40	80
NAUSEA	8	16
FEVER	12	24
JAUNDICE	4	8
HEMATEMESIS	1	2
LOOSE STOOLS	1	2
ABDOMINAL DISTENSION	2	4

**Table 5: Laboratory Investigations**

INVESTIGATIONS	NO. OF PATIENTS	PERCENTAGE
S. AMYLASE (>240 U/L)	42	84
S. LIPASE (>320 U/L )	49	98

**Table 6: USG Examination**

USG EXAMINATION	NO. OF PATIENTS	PERCENTAGE
DIAGNOSTIC	38	76
NON-DIAGNOSTIC	12	24

**Table 7: CECT**

CECT EXAMINATION	NO. OF PATIENTS	PERCENTAGE
DIAGNOSTIC	45	90
NON-DIAGNOSTIC	3	6

**Table 8: Complications**

COMPLICATIONS	NO. OF PATIENTS	PERCENTAGE
LOCAL	30	60
SYSTEMIC	14	28

**Table 9: Local Complications**

LOCAL COMPLICATIONS	NO. OF PATIENTS	PERCENTAGE
PANCREATIC ASCITES	30	60
PANCREATIC NECROSIS	6	12
PLUERAL EFFUSION	20	40

**Table 10: Systemic complications**

SYSTEMIC COMPLICATIONS	NO. OF PATIENTS	PERCENTAGE
HYPERKALAEMIA	8	16
HYPOCALCAEMIA	4	8
HYPERGLYCAEMIA	6	12
ACUTE RENAL FAILURE	6	12
ARDS	8	16
UPPER GI BLEEDING	2	4
SEPTICAEMIA	2	4

This classification defines three degrees of severity: mild acute pancreatitis, moderately severe acute pancreatitis, and severe acute pancreatitis. Transient organ failure is organ failure that is present for <48 h. Persistent organ failure is defined as organ failure that persists for >48 h.

- 1) **Mild acute pancreatitis**-Mild acute pancreatitis is characterized by the absence of organ failure and the absence of local or systemic complications.
- 2) **Moderately severe acute pancreatitis**-Moderately severe acute pancreatitis is characterized by the presence of transient organ failure or local or systemic complications in the absence of persistent organ failure.
- 3) **Severe acute pancreatitis**- Severe acute pancreatitis is characterized by persistent organ failure. Persistent organ failure may be single or multiple organ failure.

**Table 11: Severity**

SEVERITY	NO. OF PATIENTS	PERCENTAGE
MILD	32	64
MODERATELY SEVERE	14	28

SEVERE	4	8
--------	---	---

Out of 50 patients, 44(88%) were managed conservatively, 4(8%) patients underwent cholecystectomy on follow-up, 2(4%) were referred to higher center in view of complications and 2(4%) died due to multiorgan failure (1 patient) and ARDS (1 patient).

**Table 12: Management**

MANAGEMENT	NO. OF PATIENTS	PERCENTAGE
CONSERVATIVE	44	88
SURGICAL	4	8
REFERAL TO HIGHER CENTRE	2	4

**Table 13: Hospital Stay**

HOSPITAL STAY (NO. OF DAYS)	NO. OF PATIENTS	PERCENTAGE
0 - 3 DAYS	14	28
4 - 6 DAYS	14	28
7 - 9 DAYS	16	32
> 10 DAYS	6	12
TOTAL	50	100

Mean hospital stay in our study was 4.75 days. Out of 50 patients, 46 patients improved, 2 were referred to higher center and 2 patients died. Out of 2, 1 patient died due to multi organ failure and 1 due to ARDS.

**Table 14: Outcome**

OUTCOME	NO. OF PATIENTS	PERCENTAGE
IMPROVED	46	92



REFERRED TO HIGHER CENTRE	2	4
DEATH	2	4
TOTAL	50	100

**DISCUSSION**

The mean age of presentation in our study was 40.72 years and is comparable to the studies by Vipul V Nandu et al and Satyanarayana Rao et al.

**Table 15: Comparison of mean age**

Mean	Satyanarayana Rao SV et al <sup>15</sup>	Kashid A et al <sup>16</sup>	Vipul V Nandu et al <sup>17</sup>	Choudhuri Get al <sup>18</sup>	Our study (n=50)
Age in years	36.2	35	38.94	44.89	40.72

Peak incidence is in 4<sup>th</sup> decade in males(40%) and 5<sup>th</sup> decade in females(50%). It is comparable to the study by Satyanarayana Rao SV et al.

**Table 16: Comparison of age**

Age Group	Satyanarayana Rao SV et al <sup>15</sup>		Our study		Satyanarayana Rao SV et al <sup>15</sup>		Our study		Satyanarayana Rao SV et al <sup>15</sup>		Our study	
	Male(n=52)		Male(n=40)		Female(n=8)		Female(n=10)		Total(n=60)		Total(n=50)	
	No	%	No	%	No	%	No	%	No	%	No	%
11-20	4	7.5	0	0	0	0	0	0	4	6.66	0	0
21-30	10	18.55	11	22	2	25	1	2	12	20	12	24
31-40	22	42.3	13	26	3	37.5	2	4	25	41.6	15	30
41-50	8	14.8	10	20	2	25	4	8	10	16.6	14	28
51-60	8	14.8	5	10	1	12.5	1	2	9	15	6	12
>60	0	0	1	2	0	0	2	4	0	0	3	5

Total	52	86.6	40	80	8	13.3	10	20	60	100	50	100
-------	----	------	----	----	---	------	----	----	----	-----	----	-----

There was male predominance in our study with males accounting for 80% of patients with M: F ratio 4:1. Out of 50 patients 40 (80%) were males and 10 (20%) were females. The other studies also had a higher percentage of males.

**Table 17: Comparison of sex**

Sex	Kashid A et al <sup>16</sup>	Choudhuri G et al <sup>18</sup>	Satyanarayana Rao SV et al <sup>15</sup>	Our study (n=50)
Male (%)	70.91	66.6	86.66	80%
Female (%)	29.09	33.4	13.33	20%

**Table 18: Comparison of aetiology**

AETIOLOGY	Satyanarayana Rao SV et al <sup>15</sup> (n=60)	Vipul V Nandu et al <sup>17</sup> (n=142)	Pupelis G et al <sup>19</sup> (n=274)	Our study (n=50)
Alcohol (%)	76.6	78.17	54	72
Gallstone (%)	5	4.93	19	12
Miscellaneous(%)	18.3	16.9	27	8

Alcohol was the main etiological factor in our study and present in 72% of patients. This was comparable to the studies by Vipul V Nandu et al and Satyanarayana Rao et al.

**Table 19: Comparison of Clinical Feature**

Clinical feature	Kashid A et al <sup>16</sup>	Satyanarayana Rao SV et al <sup>15</sup>	Vipul V Nandu et al <sup>17</sup>	Our study (n=50)

Pain Abdomen (%)	92.73	100	100	100%
Vomiting, Nausea (%)	60	75	85.21	96

Pain abdomen was the presenting complaint in almost the entire 100% of patients. This was comparable to the studies by Satyanarayana Rao SV et al & Vipul V Nandu et al.

**Table 20: Comparison of Serum Amylase Sensitivity**

S. amylase	Kashid A et al <sup>16</sup>	Koizumi M et al <sup>20</sup>	Our study (n=50)
Sensitivity (%)	50.9	95.6	84%

**Table 21: Comparison of Serum lipase Sensitivity**

S. lipase	Koizumi M et al <sup>20</sup>	James.P.Corsetti Et al <sup>21</sup> (n=450)	Our study (n=50)
Sensitivity (%)	100	98%	98%

**Table 22: Comparison of Accuracy of USG Abdomen**

USG ABDOMEN	Kashid et al <sup>16</sup>	Satyanarayana Rao SV et al <sup>15</sup> (n=60)	Our Study (n=50)
Diagnostic (%)	66.67	81.6	76
Non Diagnostic (%)	33.33	18.3	24

**Table 23: Comparison of Accuracy of CECT scans**

CECT	Georgios I P et al <sup>22</sup> (n=185)	Gislason H et al <sup>23</sup> (n=181)	Our Study (n=50)
Diagnostic (%)	85.7	92	90
Non Diagnostic (%)	14.3	8	6

**Table 24: Comparison of Complications**

LOCAL COMPLICATIONS	Kashid A et al <sup>16</sup>	Choudhuri G et al <sup>18</sup>	Satyanarayana Rao SV et al <sup>15</sup>	Our Study (n=50)
Pancreatic Ascites (%)	34.5	40.5	11.6	60
Pancreatic Necrosis (%)	18.18	40.5	5	12
Pseudo cyst (%)	0	24.9	8.33	0
Plueral Effusion (%)	34.54	0	21.6	40
Organ failure (%)	29	40.5	11.6	12
UPPER GI Bleeding (%)	1.8	3.1	1.66	4

**Table 25: Comparison Severity of Acute Pancreatitis**

SEVERITY	Lee KJ et al <sup>24</sup> (n=146)	Our study (n=50)
Mild (%)	58.9	64%
Moderately severe (%)	29.5	28%
Severe (%)	11.6	8%

**Table 26: Comparison of Management**

MANAGEMENT	Vipul V Nandu et al <sup>17</sup> (n=142)	Our study (n=50)
CONSERVATIVE	80.28%	88%
SURGICAL	19.72%	8%
REFERRAL TO HIGHER CENTRE	0	4%

**Table 27: Comparison of Mean hospital stay**

Mean hospital stay(In days)	Choudhuri G et al <sup>18</sup>	Kashid A et al <sup>16</sup>	Satyanarayana Rao SV et al <sup>15</sup>	Our study
Mild disease	6.6	10	6.2	5.2
Severe disease	17.32	13.5	11.4	11.2

**Table 28: Comparison of Mortality**

Mortality	Choudhuri G et al <sup>18</sup>	Kashid A et al <sup>16</sup>	Satyanarayana Rao SV et al <sup>15</sup>	Our study
Percentage %	6.5	5.45	1.66	4%

Acute pancreatitis is a common disease entity and its frequent occurrence with its serious complications have brought into issues regarding management. It is an inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems. Severe acute pancreatitis is associated with organ failure with complications such as necrosis, abscess or pseudocyst.

While evaluating a patient suspected of having acute pancreatitis, 4 sequential steps need to be adopted.

- a) Establishing the diagnosis of pancreatitis excluding other abdominal conditions that have similar clinical features.
- b) Identify the presence of biliary tract disease excluding other possible etiologies of acute pancreatitis.

- c) Assess the severity of disease.
- d) Detect any complications and surgical intervention if required.

Ultrasonography of abdomen is an invaluable radiological study in acute pancreatitis with its sensitivity being 76%. The pancreatic changes noted are granular heterogeneity, hypoechogenicity, increased thickness of the gland and indistinct margins of the gland. It is also used for imaging the various complications such as pseudocyst, pancreatic ascites and abscess.

Dynamic contrast enhanced CT scan is the imaging modality used in acute pancreatitis for initial staging of severity of inflammatory process and for early detection of intra pancreatic and extra pancreatic complications. The current guidelines recommend DCT as a mandatory imaging procedure for patients with persistent organ failure, for those who develop SIRS or sepsis and for patients who do not improve within 6 to 10 days of conservative management.

The morphological severity of acute pancreatitis can be determined by using a CTSI that was developed by Balthazar and co.

In acute pancreatitis, amylase levels tend to be lower in alcoholic pancreatitis in comparison to biliary pancreatitis. It has a sensitivity of about 53.3% in our series. The advantage of this test is that it is quickly performed, simple and inexpensive.

The prime objective in the treatment of acute pancreatitis is conservative management with a good supportive and nutritional therapy and treat specific complications as well as to limit the severity of pancreatic inflammation and necrosis as well SIRS by specifically interrupting their pathogenicity. The role of surgery is limited to few complicated cases in acute severe pancreatitis.

In our study, 50 cases of acute pancreatitis are selected for clinical study including etiopathogenesis and management. Out of 50 cases, 44 cases were managed conservatively. 8% of the patients underwent cholecystectomy on followup(diagnosed with gallstone as etiology for acute pancreatitis)and 4% were referred to higher centre in view of complications.

This prospective study was undertaken since acute pancreatitis is a common disease with varied presentations and systemic complications such as Systemic inflammatory response syndrome, Multiorgan dysfunction syndrome and acute respiratory distress syndrome etc., and poses a great challenge to the surgeon in the management.

## CONCLUSION

Acute pancreatitis is a common acute abdominal condition. Most common in men. The peak incidence was 4<sup>th</sup> decade in males and 5<sup>th</sup> decade in females. Alcoholism is the most common etiological factor. Most common clinical manifestations are pain abdomen (100%) and vomiting (80%). Serum lipase assessment (sensitivity 98%) is the gold standard diagnostic test and is more sensitive than serum amylase (sensitivity 84%). USG is the initial

radiological investigation for acute pancreatitis. It is diagnostic in 76% of cases. CECT is diagnostic in 90% of cases. It is contraindicated in case of persistent acute renal failure. Disease stratification is most commonly done using Atlanta scoring system. Most of the cases were mild. Radiological assessment shows acute oedematous pancreatitis to be the predominant type. Complications are common with moderately severe and severe acute pancreatitis, pancreatic ascites being the most common cause. Multi organ failure is associated with high mortality rate. Alcoholism is associated in 20% of patients with recurrent acute pancreatitis. It is also concluded from this study that conservative treatment still holds the key in the management of acute pancreatitis and also in acute severe pancreatitis with or without complications in the initial stages of assessment.

## REFERENCES

- 1) Patel S, Patel T, Hada D, Suvera M, Parmar H. Clinical profile and outcome of acute pancreatitis and necrotizing pancreatitis. IAIM, 2015; 2(7): 116- 120.
- 2) Baig SJ1, Rahed A, Sen S. A prospective study of the etiology, severity and outcome of acute pancreatitis in Eastern India. Trop Gastroenterol. 2008 Jan- Mar; 29(1):20-2.
- 3) Reginald H. Fitz. Acute Pancreatitis: A consideration of pancreatic haemorrhage, haemorrhagic, suppurative and gangrenous pancreatitis, and of disseminated fat necrosis. Boston Med Surg J 1889; 70:181-7,205-7, 229- 35.
- 4) Opie EL. The etiology of acute hemorrhagic pancreatitis. Bull John Hopkins Hosp 1902; 12:182.
- 5) Moynihan B. Acute Pancreatitis. Ann Surg 1925; 81:132-42.
- 6) Baron TH, Morgan DE. Current concepts: Acute necrotizing pancreatitis. N Engl J Med 1999; 340: 18: 1412-17.
- 7) Steinberg W, Tenner S. Acute Pancreatitis. N Engl J Med 1994; 330:17:1198-1210.
- 8) Bradley EL III. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, GA, September 11 through 13, 1992. Arch Surg 1993; 128:586–90.
- 9) Steinberg WM, Stafford S, Goldstein, Davis ND, Shamma'a J, Anderson K. Diagnostic assays in acute pancreatitis: A study of sensitivity and specificity. Ann Intern Med 1985; 102:576-80.
- 10) Balthazar EJ. CT diagnosis and staging of acute pancreatitis. Radiol Clin North Am 1989; 27:19-37
- 11) De Bernandinis M, Violi V, Roncoroni L, Boselli AS, Gieunta A, Peracchia A. Discriminant power and information content of Ranson's prognostic signs in acute pancreatitis: A Meta analytic study. Crit Care Med 1999; 27:2272- 83.

- 12) Spanier BW, Dijkgraaf MG, Bruno MJ. Epidemiology, aetiology and outcome of acute and chronic pancreatitis: an update. *Best Pract Res Clin Gastroenterol.* 2008; 22:45–63.
- 13) American Gastroenterological Association (AGA) Institute on –Management of Acute Pancreatitis| Clinical Practice and Economics Committee; AGA Institute Governing Board AGA Institute medical position statement on acute pancreatitis. *Gastroenterology.* 2007; 132:2019–2021.
- 14) Lee JK, Enns R. Review of idiopathic pancreatitis. *World J Gastroenterol.* 2007; 13:6296–313.
- 15) Satyanarayana Rao S V, Babu Rao D, Krishna Rao S, Anvesh D. Acute Pancreatitis and its Clinical Study and Management in Amaravathi Region. *International Journal of Pharma Research & Review,* 2015; 4(11):43-49.
- 16) Kashid A, et al, acute pancreatitis experience at manipal hospital, Bangalore, Appendix 1-A, in management of acute pancreatitis, by Bhansali SK and Shah SC, Jaslok hospital 2006:173-175.
- 17) Vipul V. Nandu, Amol V. Deshpande. Clinical study of pancreatitis and its management. *Int Surg J.* 2016; 3(3): 1574-1579.
- 18) Choudhuri G, et al. Acute pancreatitis Experience at Sanjay Gandhi PGI of Medical Sciences, Lucknow, Appendix 1-B, in Management of Acute Pancreatitis, by Bhansali SK and Shah SC, Jaslok Hospital 2006. Pg. 176- 178.
- 19) Pupelis G, et al. conservative approach in the management of severe acute pancreatitis: eight-year experience in a single institution. *HPB* 2008; 10: 347-355.
- 20) Koizumi M, et al. JPN Guidelines for the management of acute pancreatitis: diagnostic criteria for acute pancreatitis. *J Hepatobiliary Pancreat Surg* 2006;13: 25-32.
- 21) James.P.Corsetti Christopher et al. Combined serum amylase and lipasedeterminations in acute pancreatitis. *Clin. Chem* 1993; 13.
- 22) Georgios I P, et al. Comparison of BISAP, Ranson's, APACHE-II, and CTSI Scores in Predicting Organ Failure, Complications, and Mortality in Acute Pancreatitis Comparison of BISAP, Ranson's, APACHE, and CTSI Scores. *Am J Gastroenterol* 2010; 105:435-441.
- 23) H. Gislason, A. Horn, D. Hoem et al. ACUTE PANCREATITIS IN BERGEN, NORWAY A study on incidence, etiology and severity. *Scandinavian Journal of Surgery.* 2004; 93: 29–33.
- 24) Lee KJ, Kim HM, Choi JS, et al. Comparison of Predictive Systems in Severe Acute Pancreatitis According to the Revised Atlanta Classification. *Pancreas* 2015; Sep 18.



