"A COMPARISON BETWEEN CLINICAL SCORES AND RADIOLOGICAL EVALUATION IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS - A PROSPECTIVE STUDY."

By

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Dissertation submitted to

In partial fulfillment for the degree of

MASTER OF SURGERY

IN

GENERAL SURGERY

Under the guidance of

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2017

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"A COMPARISON BETWEEN CLINICAL SCORES AND RADIOLOGICAL EVALUATION IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS - A PROSPECTIVE STUDY"** *is a bonafide and genuine research work carried out by me under the guidance of* ***DR. BALASAHEB B METAN,*** *Professor, Department of General Surgery, Shri. B.M. Patil Medical College, Hospital and Research centre, Vijayapur.*

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LIST OF ABBREVIATIONS

AP	Acute Pancreatitis
BISAP	Bed side index severity of acute pancreatitis.
APFC	Acute Peripancreatic Fluid Collection
ANC	Acute Necrotic Collection
WON	Walled Off Necrosis
CTSI	Computed tomography severity index.
CECT	Contrast Enhanced Computed Tomography
USG	Ultrasonography
SIRS	Systemic Inflammatory Resoponse Syndrome
ARDS	Adult Respiratory Distress Syndrome

ABSTRACT

Introduction :

Acute pancreatitis is a common acute medical condition requiring emergent care. The disease manifests in a wide range of severity, ranging from the mild peripancreatic edema to the potentially life threatening infected necrotizing and hemorrhagic pancreatitis.

Radiological evaluation using the Balthazar radiological CT severity index is being increasingly used to identify infected necrosis as well as to determine the severity of pancreatitis and also clinical scores like BISAP'S to assess the organ failure.

Aims and Objective :

To compare the accuracy of Ranson's score, BISAP's score and Balthazar's CT severity index scores in assessing severity of acute pancreatitis with respect to clinical outcomes.

Materials and methods :

All patients diagnosed to have Acute Pancreatitis and admitted in Shri B M Patil Medical College, Hospital and Research Center, Bijapur between October 2014 and May 2016.

All patients were subjected to thorough clinical examination & appropriate investigations.

Results

Of 50 cases AP 45 were male and 5 were female; mean age 37.6 who were evaluated for Balthazar CTSI (CECT abdomen), Ranson's and BISAP's clinical scoring.

45 patients underwent CECT abdomen showing Balthazar CTSI is significant for complications (P value of 0.026) in Pearson's correlation associated with acute pancreatitis. ROC analysis of Balthazar CTSI is significant for prediction of complications (P value of 0.049).

Conclusion :

Balthazar CTSI score was more accurate than BISAP's and Ranson's score, with BISAP's score being more easy to assess organ failure.

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INTRODUCTION

The anatomical basis was first created in the 17th century when the pancreatic duct was discovered (J.C. Wirsung 1642) and the duodenal papilla was described (J.K. Brunner 1683, C.B. Holdefreund 1713 and A. Vater 1750) .

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

Acute pancreatitis is a common acute medical condition requiring emergent care. There are two major causes of acute pancreatitis - alcohol and biliary disease which accounts 50-70% of total cases.

The disease manifests in a wide range of severity, ranging from the mild peripancreatic edema to the potentially life threatening infected necrotizing and hemorrhagic pancreatitis.

Ranson's clinical score and BISAP's clinical score are widely used in assessing the severity of acute pancreatitis. Radiological evaluation using the Balthazar radiological CT severity index is being increasingly used to identify infected necrosis as well as to determine the severity of pancreatitis.

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, is confirmatory and will increase the diagnostic yield. Supportive radiological

procedures are sonography, computed tomography. Currently CECT is the imaging modality of choice where areas of hypo perfusion correlate with necrosis.

AIM AND OBJECTIVE OF THE STUDY

To compare the accuracy of Ranson's score, BISAP's score and Balthazar's CT severity index scores in assessing severity of acute pancreatitis with respect to clinical outcomes.

REVIEW OF LITERATURE

Constantinos C et al. In their study in November 2002, compared Ranson's, APACHE II and APACHE III Scoring Systems in Acute Pancreatitis. All three scores correlated the length of stay with disease severity. The Ranson's score achieved the highest sensitivity and the lowest false-negative rate. They concluded that the APACHE III offered little advantage over the APACHE II score and that the Ranson's criteria proved to be as powerful a prognostic model as the more complicated APACHE II and III scoring systems. However, they noted a delay of 24 hours in assessing the severity of pancreatitis using the Ranson's score[2].

Singh VK et alEvaluated the Bedside index for severity in acute pancreatitis (BISAP) score and assessed mortality. There was a statistically significant increasing mortality with increasing BISAP score. A BISAP score 3 or more was associated with an increased risk of developing organ failure, persistent organ failure, and pancreatic necrosis. They concluded that BISAP score represents a simple way to identify patients at risk of increased mortality and the development of intermediate markers of severity within 24 hour of presentation[3].

Muddana V et al Compared BISAP, Ranson's and CTSI Scores in Predicting Organ Failure in Acute Pancreatitis. The number of patients with a they concluded that the BISAP score is an accurate means for risk stratification in patients with acute pancreatitis. Its components are clinically relevant and easy to obtain simple scoring system that may reach the maximal utility and novel models are needed to further improve predictive accuracy[4].

Chatzicostas et al concluded in all outcome measures the APACHE scores generate small and of similar extent changes in probability. The Balthazar score is superior to other scoring systems in predicting acute pancreatitis severity and pancreatic necrosis.

However, the Ranson and APACHE scores perform slightly better with respect to organ failure prediction[5]

Georgios I P et alconcluded that the BISAP score is an accurate means for risk stratification in patients with AP. Its components are clinically relevant and easy to obtain. The prognostic accuracy of BISAP is like those of the other scoring systems. We conclude that simple scoring systems may have reached their maximal utility and novel models are needed to further improve predictive accuracy[6].

Leung TK et alCTSI Ranson criteria, and APACHE II scoring system in AP concluded CTSI is a useful tool in assessing the severity and outcome of AP and the CTSI is an index in our study. Although Ranson score and APACHE II score also are choices to be the predictors for complications, mortality and the length of stay of AP, the sensitivity of them are lower than CTSI[7].

Erik J Simchuk et alComputed tomography severity index is a predictor of outcomes for severe pancreatitis concluded that CTSI >5 significantly correlated with death (P = 0.0005), prolonged hospital stay (P<0.0001), and need for necrosectomy (P<0.0001). Patients with a CTSI >5 were 8 times more likely to die, 17 times more likely to have a prolonged hospital course, and 10 times more likely to undergo necrosectomy than their counterparts with CT scores <5.These data show that the CTSI is an applicable and comparable predictor of outcomes in severe pancreatitis[8].

Lautz TB1, Turkel G, Radhakrishnan J, Wyers M, Chin ACUtility of the computed tomography severity index (Balthazar score) in children with acute pancreatitis Pediatr Surg. 2012 Jun;47(6):1185-91. doi: 10.1016/j.jpedsurg.2012.03.023 concluded TheCTSI is superior to clinical scoring systems for identifying children with acute pancreatitis at heightened risk for developing serious complications[9].

Bollen TL1, Singh VK, Maurer R, Repas K, van Es HW, Banks PA, Mortele KJA comparative evaluation of radiologic and clinical scoring systems in the early prediction of severity in acute pancreatitis Am J Gastroenterol. 2012 Apr;107(4):612-9. doi: 10.1038/ajg.2011.438. Epub 2011 Dec 20 concluded that the predictive accuracy of CT scoring systems for severity of AP is like clinical scoring systems. Hence, a CT on admission solely for severity assessment in AP is not recommended[10].

HISTORY

The earliest description of pancreas dates to 300 BC, given by Herophilus of Chalkaidon. During 100 AD Rufus of Ephesus thought that pancreas acts as cushion for stomach & named it as “PANCREAS” meaning “all flesh”.

The anatomical basis was first created in the 17th century when the pancreatic duct was discovered by J.C. Wirsung in 1642 and the duodenal papilla was described by J.K. Brunner in 1683.

The prognostication of acute pancreatitis was done for the time in 1974 by John HC Ranson when he was at New York university medical center, New York. Born in Bangalore, India in 1938 John Ranson rose to International prominence in medicine in the field of pancreatic diseases, & particularly acute pancreatitis. He contributed profound knowledge of about nonsurgical & surgical management of acute pancreatitis & his contributions in the field are fundamental to our present understanding of the disease & its clinical management. He was the recipient of many honors & is accepted as a leader in field of acute pancreatitis.

Emil J Balthazar, Professor of radiology, Bellevue Medical Center, New York in 1989, gave the CT grading of acute pancreatitis, & emphasized the role of CT in initial process of diagnosis, as an early predictive indicator of disease severity, & in detecting the complications associated with acute pancreatitis. There were various ill-defined terminologies with regards to acute pancreatitis. This lead to the symposium at Atlanta where in a university accepted, clinically based classification system for acute pancreatitis was developed.

ANATOMY OF PANCREAS[1]:-

The name ‘pancreas’ is derived from the Greek ‘pan’ (all) and ‘kreas’ (flesh). For a long time, its glandular function was notunderstood, and it was thought to act as a cushion for thestomach. The pancreas is situated in the retroperitoneum. It isdivided into a head, which occupies 30% of the gland by mass,and a body and tail, which together constitute 70%. The head lieswithin the curve of the duodenum, overlying the body of thesecond lumbar vertebra and the vena cava. The aorta and thesuperior mesenteric vessels lie behind the neck of the gland.Coming off the side of the pancreatic head and passing to the leftand behind the superior mesenteric vein is the uncinate processof the pancreas. Behind the neck of the pancreas, near its upperborder, the superior mesenteric vein joins the splenic vein to formthe portal vein. The tip of the pancreatic tailextends up to the splenic hilum.The pancreas weighs approximately 80 g. Of this, 80–90% iscomposed of exocrine acinar tissue, which is organised into lobules.The main pancreatic duct branches into interlobular andintralobular ducts, ductules and, finally, acini. The main duct islined by columnar epithelium, which becomes cuboidal in theductules. Acinar cells are clumped around a central lumen, whichcommunicates with the duct system. Clusters of endocrine cells,known as islets of Langerhans, are distributed throughout thepancreas. Islet cells consist of differing cell types: 75% are B cells(producing insulin); 20% are A cells (producing glucagon); and the remainder are D cells (producing somatostatin) and a smallnumber of pancreatic polypeptide cells. Within an islet, the Bcells form an inner core surrounded by the other cells. Capillariesdraining the islet cells drain into the portal vein, forming a pancreaticportal system.

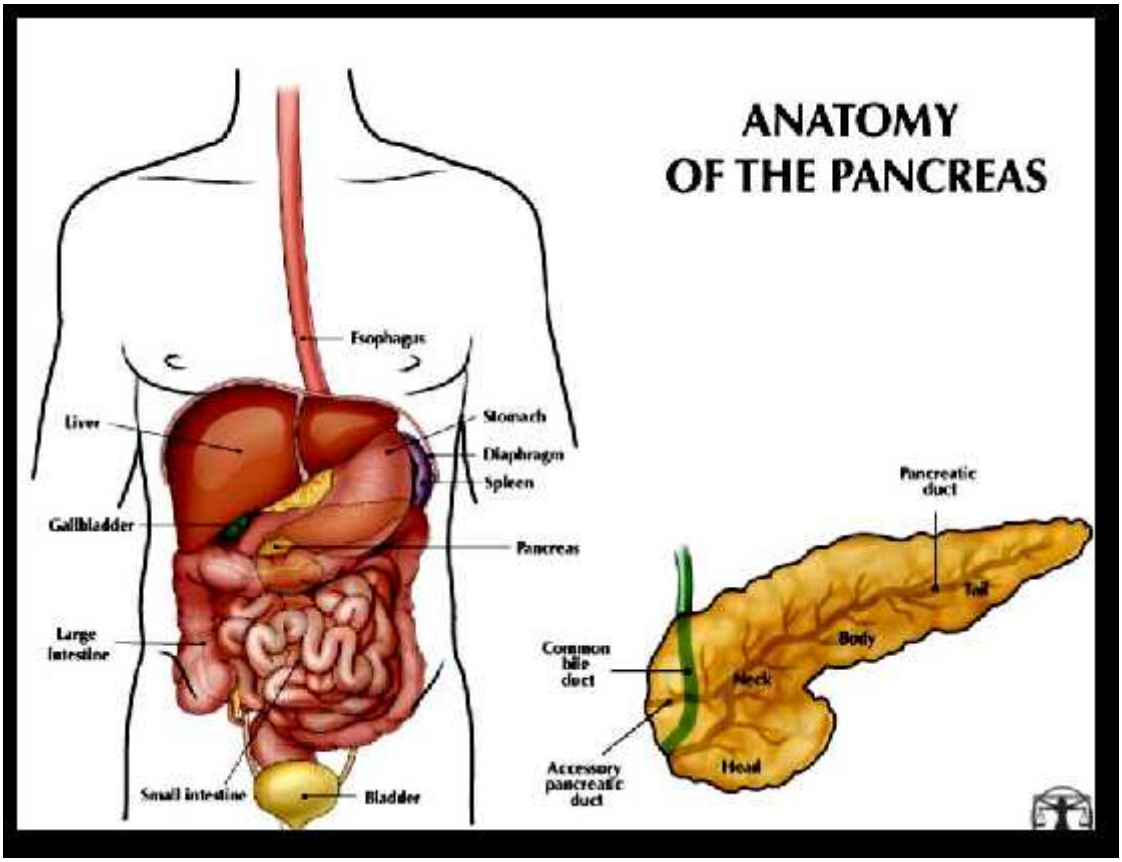


Fig 1: ANATOMY OF PANCREAS

ETIOLOGY ACUTE PANCREATITIS[1]

Alcohol intake and biliary tract disease account for majority of the cases (90%) Relative frequency depends on the patient population and prevalence of alcoholism in the population studied. In United States alcohol abuse is the main cause.

OBSTRUCTION

- Choledocholithiasis.
- Ampullary or pancreatic tumour.
- Worms or foreign bodies obstructing the papilla.
- Pancreas divisum with accessory duct obstruction.
- Choledochocele.
- Peri ampullary duodenal diverticula.
- Hypertensive sphincter of Oddi.

TOXIN OR DRUGS

- TOXIN- Ethylalcohol, Methylalcohol, scorpion venom, organophosphorus, insecticides.
- DRUGS - Azathioprine Mercaptopurin, Valproic acid, Estrogens, Tetracycline, Metronidazole, Nitrofurantoin, Furosemide, Sulfonamide, Methyldopa, Cimetidine, Ranitidine, Didanosine, Acetaminophen, erythromycin.

TRAUMA

- Accidental - Blunt trauma to the abdomen.
- Iatrogenic - postoperative trauma, ERCP, Endoscopic sphincterotomy.

METABOLIC ABNORMALITIES

- Hyper triglyceridemia
- Hypercalcemia

HEREDITARY PANCREATITIS

INFECTION

- Parasitic- Ascariasis, Clonorchiasis
- Viral - Mumps, Rubella, Hepatitis A, B, non-A, non-B, Coxsackie Virus-B, Echo virus, adenovirus, cytomegalovirus, varicella, Epstein bar virus, Human Immunodeficiency virus.
- Bacterial- Mycoplasma, Campylobacter jejuni,
- Mycobacteriumtuberculosis, Mycobacterium avium complex, Legionella, Leptospirosis.

VASCULAR ABNORMALITIES

- ISCHEMIA – Hypo perfusion, Atherosclerotic emboli.
- VASCULITIS - SLE, PAN, Malignant hypertension.

MISCELLANEOUS CONDITIONS

- Penetrating peptic ulcer.
- Crohn‘s disease.
- Reye‘s syndrome,
- Cystic fibrosis.
- Hypothermia.
- Pregnancy.

IDIOPATHIC CAUSE

DIAGNOSTIC WORK UP

1) Routine Blood Tests

- Pancreatitis can induce a diffuse capillary leak syndrome that, when combined with vomiting, can result in significant fluid losses. The resulting hypovolemia can be marked. It usually leads to an increased haematocrit, haemoglobin, blood urea nitrogen, and creatinine.
- Serum albumin levels may be markedly depressed, particularly if fluid lossesare corrected by administration of albumin-free crystalloid solutions.
- The serum electrolytes may be normal, but with significant vomiting, ahypochloremic metabolic alkalosis can develop.
- The white blood cell count is usually elevated with an associated left shift inthe differential count.
- Blood glucose may be elevated either due to associated diabetes mellitus or because of increased glucagon and catecholamine release combined withdiminished insulin release.
- Hyperbilirubinemia is relatively common during the early stages ofpancreatitis. It can be caused by either a biliary tract stone or by the inflamed(and possibly fibrotic) pancreas causing bile duct obstruction, and in thissetting, cholangitis with positive blood cultures can be superimposed on the pancreatitis. On the other hand, the hyperbilirubinemia of pancreatitis can alsoreflect the non-obstructive cholestasis that often accompanies any severeillness. Elevation of ALT, ALP and GTT also significant.
- Hypertriglyceridemia is routinely noted in patients who have hyperlipidaemiainduced pancreatitis. Hypertriglyceridemia can also be induced by exposure toethanol, and therefore, the diagnosis of pancreatitis is

always suspected when lactate serum is found when evaluating an alcoholic patient with abdominalpain. A serum triglyceride should be obtained and considered the aetiology if1,000 mg / dl.

- Many patients with pancreatitis appear to have hypocalcaemia, but for themost part, that hypocalcaemia can be explained by the hypoalbuminemia thataccompanies pancreatitis. Occasionally, however, patients with severepancreatitis have a reduction in their free, ionized calcium that is not areflection of hypoalbuminemia. This type of hypocalcaemia is associated witha poor prognosis. Some of these patients manifest tetany and carpopedalspasm, making treatment with calcium mandatory.
- In those cases, thrombocytopenia, elevated levels of fibrin degradationproducts, a decreased fibrinogen level, prolonged partial thromboplastin time,and a prolonged prothrombin time can be observed.

2) Amylase Measurement

- The elevation of serum amylase (normal 60-180U/L) is observed within 24hours of the onset of symptoms and gradually returns to normal in thesubsequent weeks.
- Serum amylase greater than three times the upper limit of normal wassignificant.
- Persistent elevated serum amylase beyond initial week of illness reflects ongoing pancreatic inflammation or development of pancreatic complication,pseudo cyst, phlegmon or necrosis.

- Serum amylase determination has high sensitivity (>95%) but overallspecificity is low (70%), since elevated serum level occur in many condition(intra-abdominal and extra abdominal).
- S-Type isoenzyme is seen in ruptured ectopicpregnancy, salivary gland disorder and salphingitis etc.
- Urinary amylase excretion (normal 4-400 U/L) is more sensitive index ofacute pancreatitis though not diagnostic.

4) Serum lipase

- Serum lipase elevation is a more specific indicator of acute pancreatitis than serum amylase because lipase circulating in the serum is mostly of pancreatic Origin. Lipase is elevated for longer period and hence useful in patient who present late. But, serum lipase is not most specific for acute pancreatitis, as it can be raised in perforated peptic ulcer, acute cholecystitis and intestinal ischemia.

RADIOLOGICAL PROCEDURE

1) RADIOGRAPH

Plain radiograph of the abdomen may reveal paralytic ileus, increased gastro colic separation, sentinel loop (dilated proximal jejunal), colon cut-off sign (distension of colon at the level of transverse colon with no gas in splenic flexure),cholelithiasis, obliteration of psoas margins.

Plain radiograph also rules out potential abdominal emergencies like perforation of hollow viscous or mesenteric ischemia. A chest radiograph may show left pleural effusion, elevated left hemi diaphragm, basal atelectasis and delineates other causes of plain abdomen likeleft lower lobe pneumonia or pneumoperitoneum. In multi organ failure if lung isaffected ARDS changes are seen on chest X –ray.

Upper GI contrast studies may show widening of -C ‘loop of duodenum, anterior displacement of stomach and duodenal mucosal abnormalities, but are notlonger favored as these finding are not specific.

2) ABDOMINAL ULTRASOUND

Abdominal ultrasound examination can be inconclusive and often misleading.

It is largely operator dependent and in 30-40% of patient‘s pancreas cannot be visualized due to air filled bowel loops. It is the also inaccurate in detecting pancreaticnecrosis and regional infection. Still, it can be used to detect pancreatic oedema,peripancreatic fluid collection, and gallstone causing pancreatitis, biliary sludge andalso pseudo cyst, ascites, portal or splenic vein thrombosis.

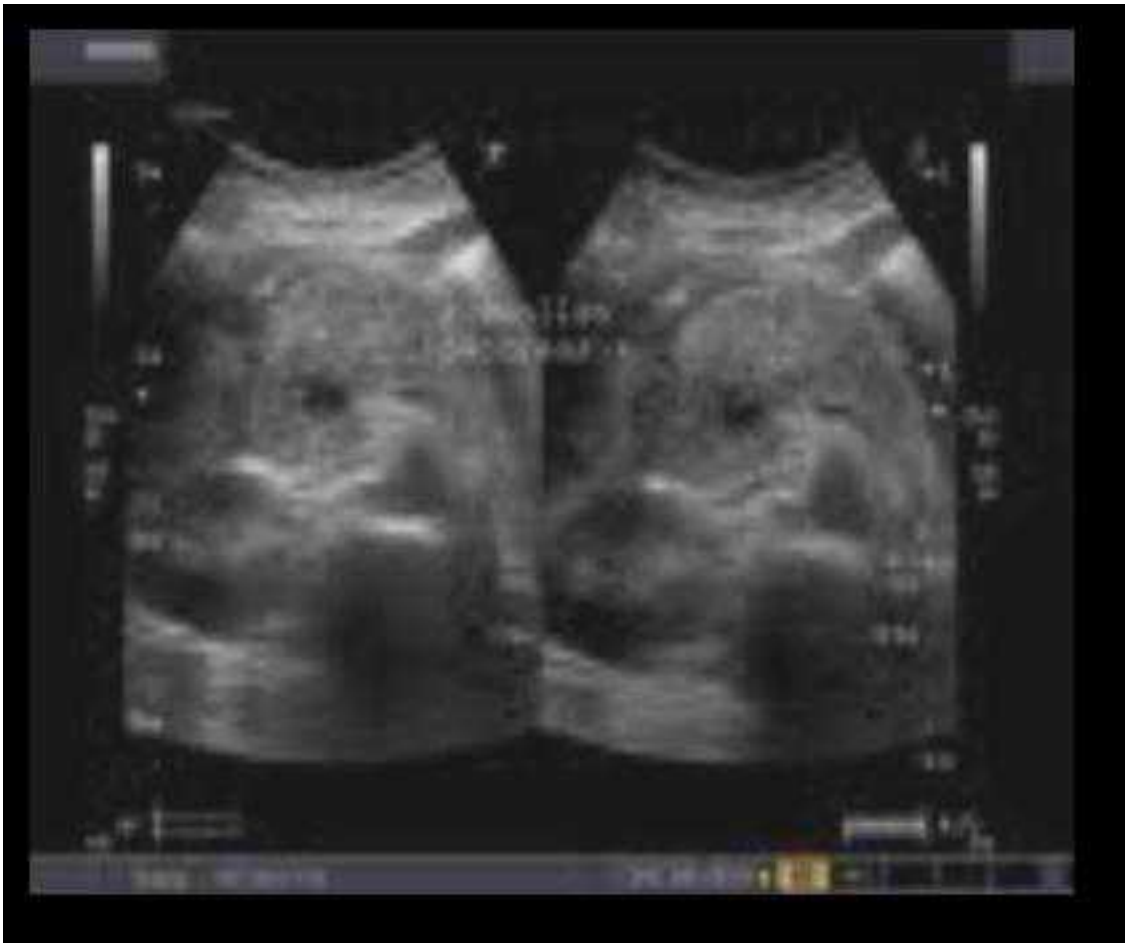


Fig 2: Showing bulky pancreas.

3) COMPUTED TOMOGRAPHY SCAN[14]

CT scan is currently the most sensitive non-invasive method to confirm the diagnosis for acute pancreatitis25. The specificity of an admission CT scan is found100% and sensitivity is 85%. Most episodes undetected by CT scan are mild and CTscan also provides alternate diagnosis in case with false positive elevation ofenzymes. Also, contrast enhancement differentiates between oedematous andnecrotizing pancreatitis.

CT FINDING IN ACUTE PANCREATITIS

A. PANCREATIC CHANGES

- Parenchymal enlargement-diffuse, focal
- Parenchymal oedema
- Necrosis

B. PERIPANCREATIC CHANGES

- Blurring of fat planes
- Thickening of fascial planes
- Presence of fluid collection

C. NON- SPECIFIC SIGNS

- Pleural effusion
- Bowel distension
- Mesenteric oedema

CT scan is also useful in demonstrating structural complication that develops during acute pancreatitis like pancreatic abscess, pseudo cyst or fluidcollection. Also, severity of acute pancreatitis can be graded using CT scan and hasbeen used in prediction of prognosis.

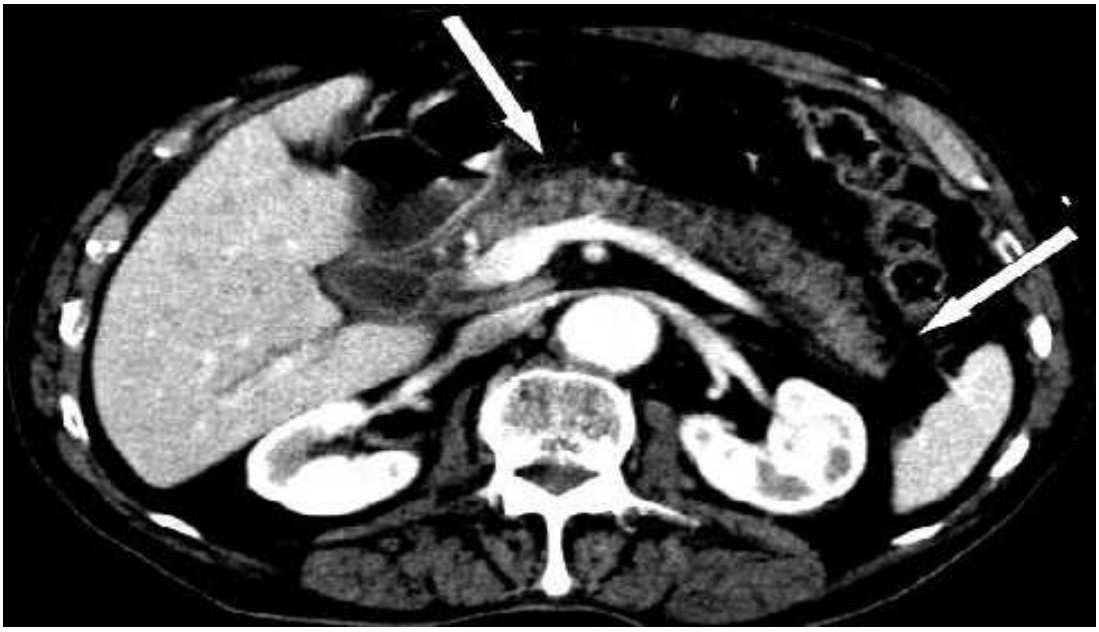


Fig. 3: CT image interstitial oedematous pancreatitis with peripancreatic fat stranding (arrows)

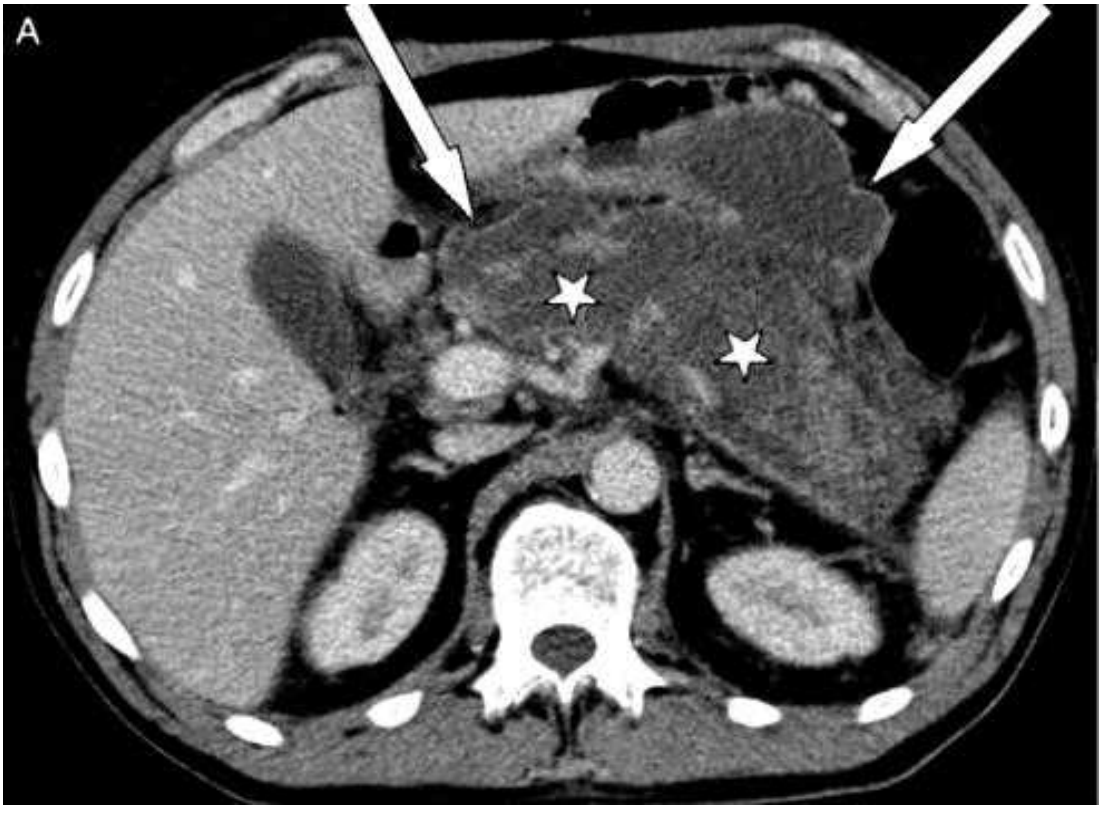


Fig. 4: CT image of Necrotising pancreatitis

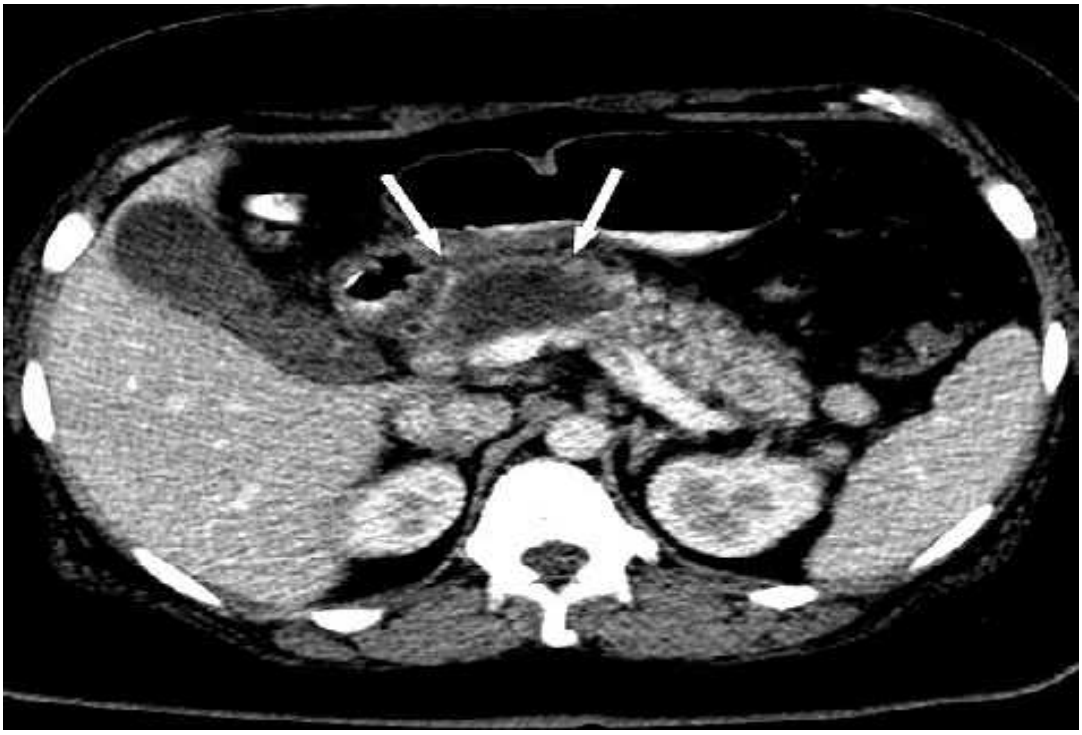


Fig. 5: CT image of acute necrotic collection

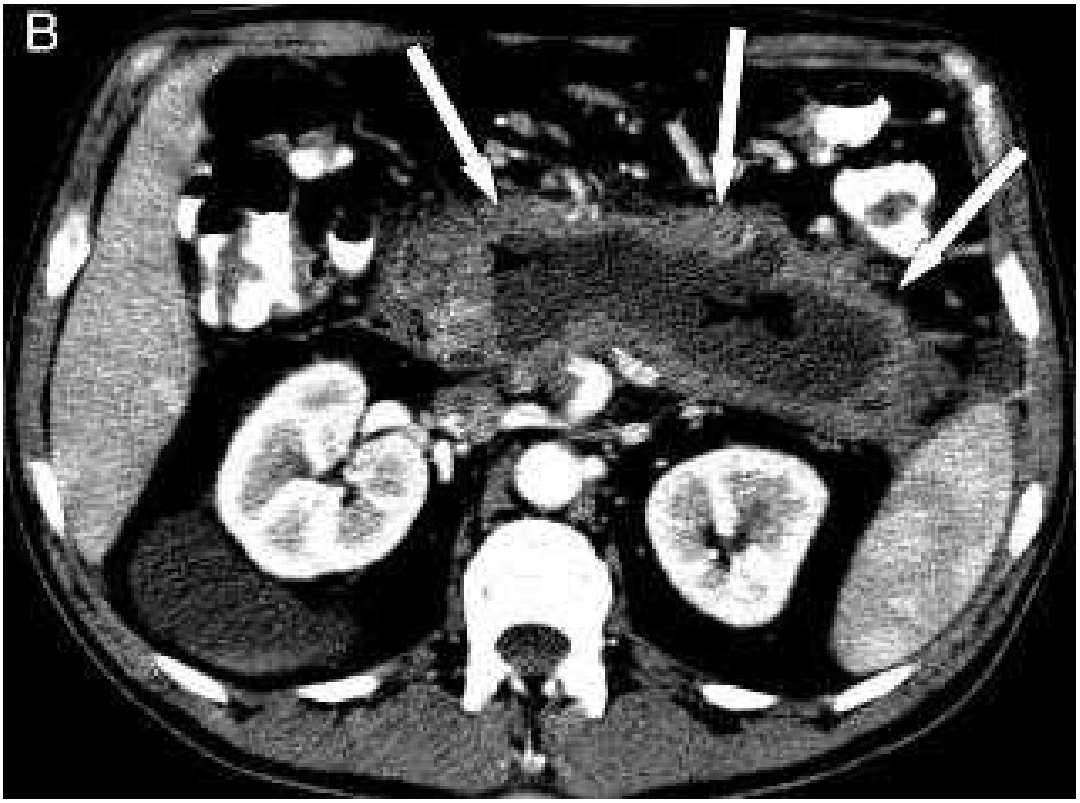


Fig. 6: CT image of walled-off necrosis

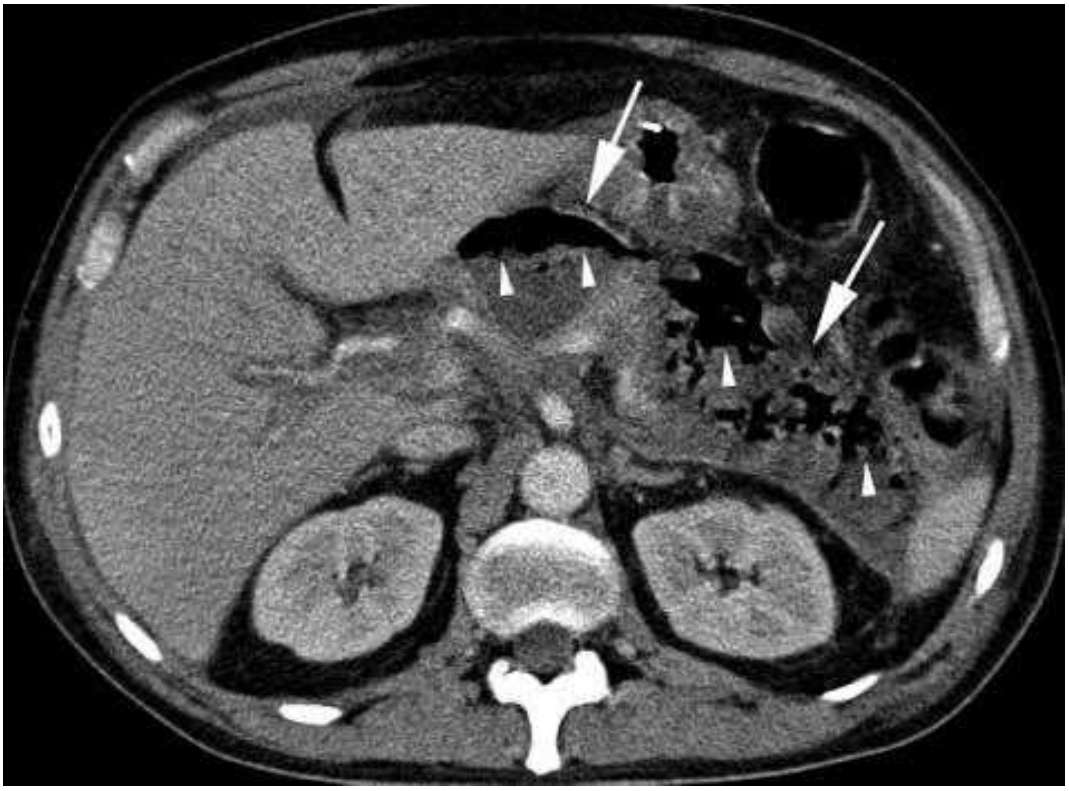


Fig. 7: CT image of infected pancreatic necrosis

MULTIFACTOR SCORING SYSTEM

One of the early systems for judging severity was developed by Ranson in 1936. It incorporates five feature measured at admission and six additional criteriadetermined during the initial 48 hours. The criteria were refined to create two similarsystems, one for alcoholic pancreatitis and for gallstone pancreatitis. Patient with zeroto two Ranson prognostic sign have essentially no mortality and do not requireanything more than simple supportive care. Patient with three or four signs have amortality of 15% and 40% of these patients require intensive care therapy. Patient withfive or six signs have a mortality rate of approximately 50% and almost universallyrequire support in an intensive care unit. Patient with seven or more prognostic signs have a predicted mortality of almost100%.

RANSON'S CRITERIA[1,]

A) Alcoholic Pancreatitis

- On admission to hospital
  - ✓ Age >55 years
  - ✓ White blood count> 16000/mm3
  - ✓ Blood Glucose level> 200mg/dl
  - ✓ Lactate dehydrogenage> 350 U/L
  - ✓ Aspartate aminotransferase>250 U/L
- Within 48 hours of admission
  - ✓ Decrease in hemotocrit>10 %
  - ✓ Increase in blood urea nitrogen > 5 mg/ dl
  - ✓ Serum calcium < 8 mg / dl
  - ✓ Arterial oxygen pressure< 60mm Hg
  - ✓ Base deficit >4mmol/L

- ✓ Fluid sequestration> 6Ltr

B) Gallstone Pancreatitis

- On admission to hospital
  - ✓ Age >70 year
  - ✓ White blood count>18,000
  - ✓ Blood Glucose level>220 mg/dl Lactate dehydrogenase>400U/L
  - ✓ Aspartate aminotransferase>250U/L
- Within 48 hours of admission
  - ✓ Decrease in haematocrit>10 %
  - ✓ Increase in blood urea nitrogen>2mg/dl
  - ✓ Serum calcium<8mg/dl
  - ✓ Fluid sequestration>4l
  - ✓ Base deficit>5mmol/l

Score of ≥3 indicates severe pancreatitis.

BISAP's (Bedside Index of Severity in Acute Pancreatitis)[1]

- ✓ Blood urea Nitrogen >25mg/dl.
- ✓ Impaired mental status.
- ✓ Development of SIRS (Systemic Inflammatory Response Syndrome).
- ✓ Age > 60 years
- ✓ Pleural effusion

Score of ≥3 indicates organ failure and pancreatic necrosis.

Further modification of this system in Glasgow by Imrie and his colleague in 1978 led to the Glasgow system where only 9 factor need to be assessed. A furtherrefinement of this system by Blamey and Imrie in 1984 led to Modified Glasgowsystem where only 8 factor need to be assessed0[41.]

MODIFIED GLASGOW (Imrie's) CRITERIA[41]

- Within 48 hours of admission
  - ✓ Age >55 years
  - ✓ White blood cell count>15000/mm3
  - ✓ Glucose> 180mg/dl
  - ✓ Blood urea nitrogen> 45 mg/dl
  - ✓ Lactate dehydrogenase > 600U/L
  - ✓ Albumin< 3.2gm/ dl
  - ✓ Arterial oxygen pressure < 60mm Hg
  - ✓ Serum calcium <8 mg/dl

Score of ≥3 indicates severe pancreatitis.

CT SEVERITY INDEX

The value of CT scan as an early predictive indicator of morbidity and mortality was first established by Sielgelmen et al in 1980 and Hill et al in 1982.

Balthazar in 1989 graded patient with acute pancreatitis into five categories based on CT scan finding[14]. He showed that patients without peripancreatic inflammation (grade A&B) have a mild uncomplicated course while those with one ormore peripancreatic collection (grade D&E) often exhibit a protracted clinical illnesswith a higher frequency of complication and death.

CT SEVERITY INDEX (BALTHAZAR 1990) SCORE:

Grading of pancreatitis 0 - 4

A. Normal pancreas - 0

B. Enlargement of pancreas - 1

C. Inflammatory changes in pancreas and peripancreatic fat - 2

D. Ill-defined single fluid collection - 3

E. Two or more poorly defined fluid collections – 4

Interpretation:

- **0 – 3 :**Mortality 3%, Morbidity 8%
- **4 – 6 :**Mortality 6%, Morbidity 35%
- **7 – 10 :** Mortality 17%, Morbidity 92%

Pancreatic necrosis grading

A. None - 0

B. Less than or equal to 30% - 2

C. 30-50 % - 4

D. More than 50% - 6

The maximum score that can be obtained is 10

MATERIALS AND METHODS

SOURCE OF DATA:

All patients diagnosed to have Acute Pancreatitis and admitted in Shri B M Patil Medical College, Hospital and Research Center, Bijapur between October 2014 and May 2016.

METHOD OF COLLECTION OF DATA:

- Patients of all age groups diagnosed to have acute pancreatitis in Shri B M Patil Medical College, Hospital and Research Center, from October 2014 to May 2016 will be included in this study.
- All patients were subjected to thorough clinical examination & appropriate investigations.

INCLUSION CRITERIA:

All patients diagnosed to have acute pancreatitis.

EXCLUSION CRITERIA:

- Acute on Chronic Pancreatitis
- Post ERCP Pancreatitis
- Acute pancreatitis in pregnancy
- HIV on ART
- Malignancy of pancreas and hepatobiliary tract
- Cirrhosis of liver

RESEARCH HYPOTHESIS: BISAP score is an easier evaluation tool to assess the severity of acute pancreatitis compared to Ranson's score & radiological scores.

TOTAL SAMPLE SIZE : 50

With 95% confidence limit,

Prevalence of Acute pancreatitis = 1.5%.[4]

Statistical analysis:

The Following formula is used to estimate the sample size for the comparative study between clinical scores and radiological assessment in acute pancreatitis.

$n = Z^2 P (1- P)/ d^2$

Where Z = 1.96 at 95% confidence limit

P = Prevalence

D =Desired precision

INVESTIGATIONS:

The following investigations and other ancillary investigations will be performed as deemed necessary for the individual cases.Necessary investigations will be repeated after 48 hours of admission.

- Complete Blood Count
- Urine examination
- Serum Amylase and Serum Lipase
- Blood Glucose level
- Serum Aspartate transaminase
- Serum Lactate dehydrogenase
- Serum calcium
- Hematocrit
- Liver function tests
- ABG

Radiological investigations

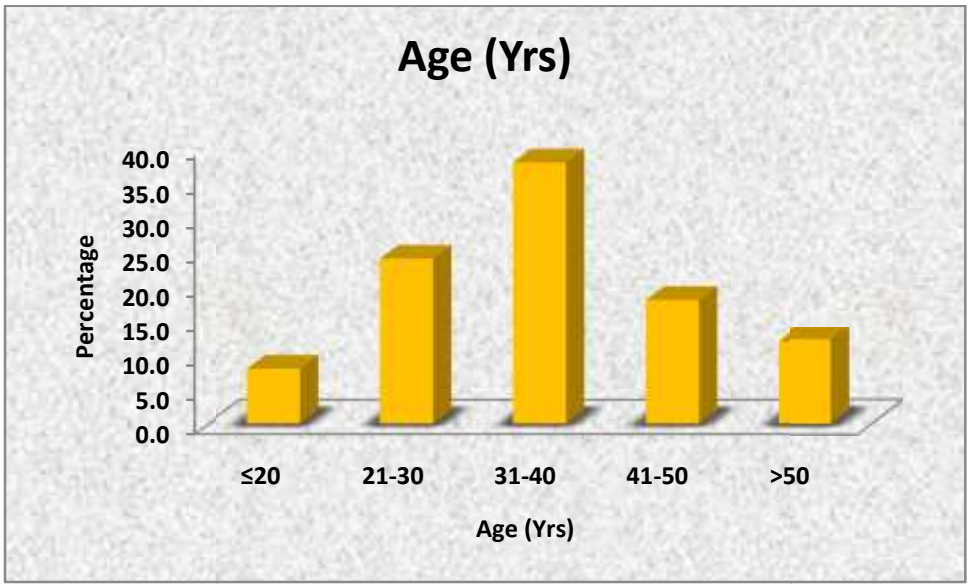
- Chest X ray
- Erect X ray abdomen
- Ultrasonography
- CECT Abdomen

RESULTS

Table1: Distribution of cases according to Age

Age (Yrs)	N	%
≤20	4	8.0
21-30	12	24.0
31-40	19	38.0
41-50	9	18.0
>50	6	12.0
Total	50	100.0
Mean±SD	37.6±15.6	

Graph 1: Distribution of cases according to Age

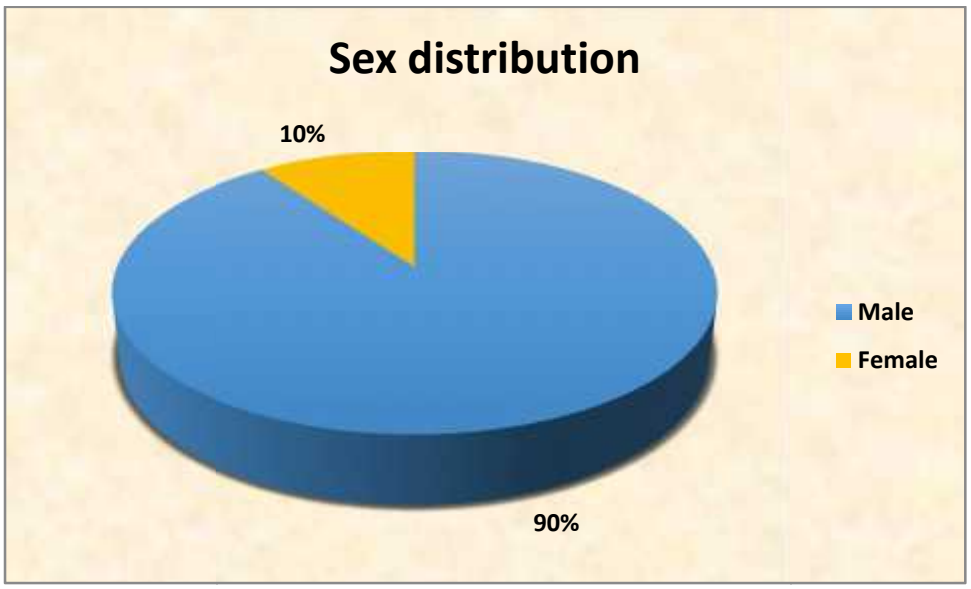


In present study it is observed that 19(38%) out of 50 patients were found in the age group of 31-40 yrs. The mean age group in the present study was 37.6yrs with standard deviation of 15.6

Table 2: Distribution of cases according toSex

Sex	N	%
Male	45	90.0
Fema1le	5	10.0
Total	50	100.0

Graph 2: Distribution of cases according toSex

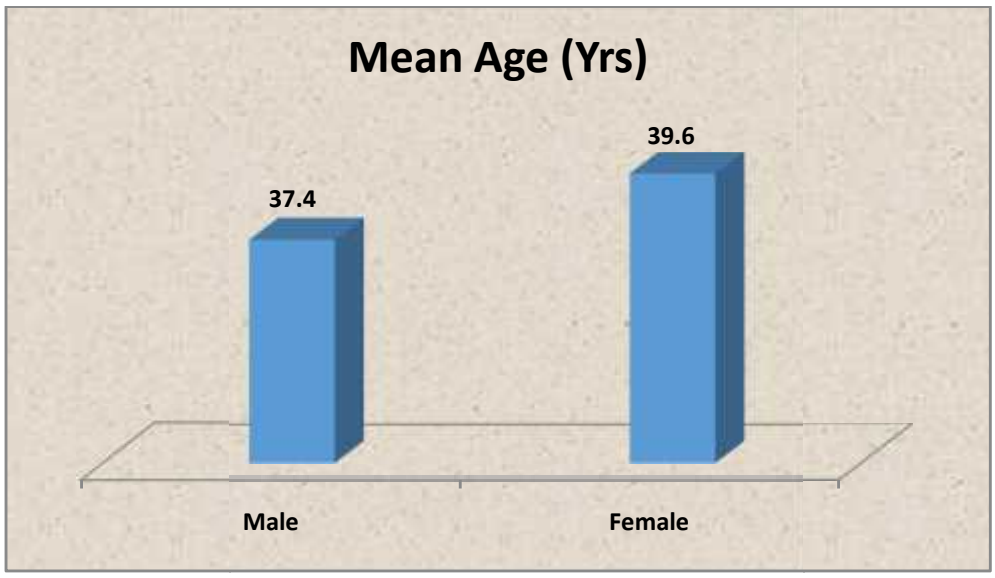


In present study it was observed that 45(90%) out of 50 patients were male and 5 patients were female.

Table3: Mean Age by Sex

Age (Yrs)	Male (N=45)		Female (N=5)		p value
	Mean±SD	Range	Mean±SD	Range	
	37.4±13.0	7-83	39.6±33.1	3-90	0.77

Graph 3: Mean Age by Sex

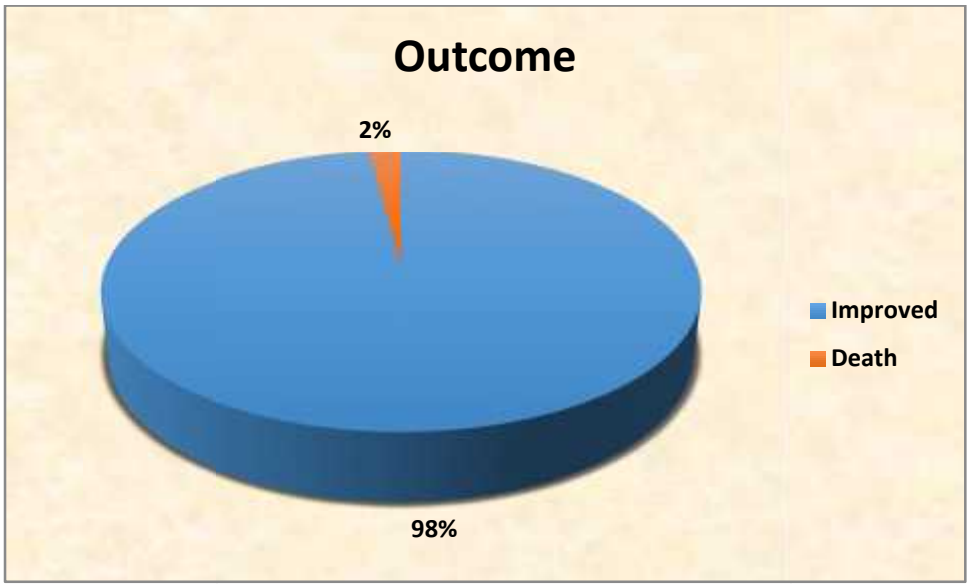


In present study it was observed that out of 45 male patients the mean age was 37.4yrs with sd of 13.0. And out of 5 female patients the mean age was 39.6yrs with sd of 33.1. The p value was found to be 0.77.

Table 4: Distribution of cases according to Outcome

Outcome	N	%
Improved	49	98
Death	1	2

Graph 4: Distribution of cases according to Outcome

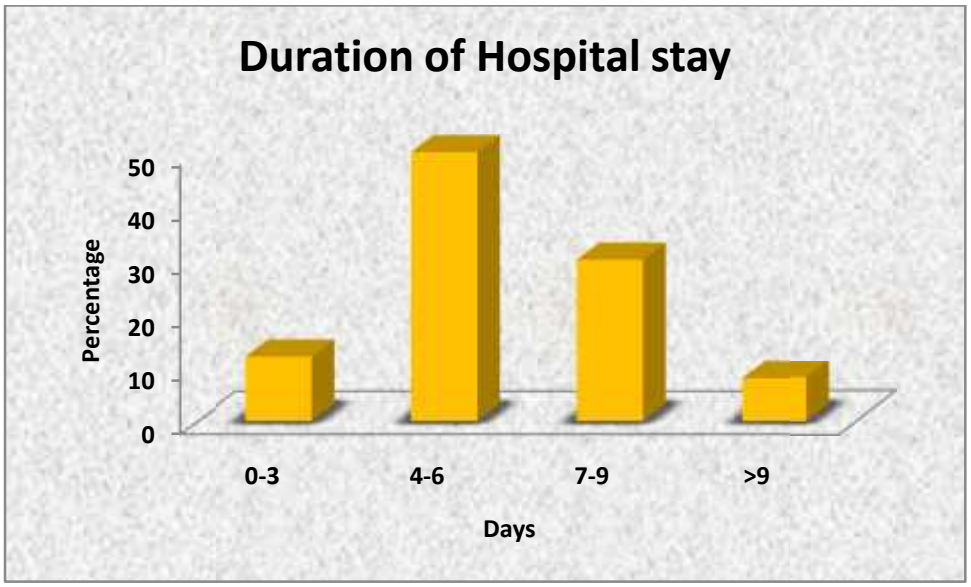


In present study it was observed that 49(98%) out of 50 patients improved and 1 patient died during hospital stay**.**

Table 5: Distribution of cases according to Duration of Hospital stay

Duration of Hospital stay(Days)	N	%
0-3	6	12
4-6	25	50
7-9	15	30
>9	4	8
Total	50	100
Mean±SD	6.1±2.9	
Range	1-16	

Graph 5: Distribution of cases according toDuration of Hospital stay



In present study it was observed that the duration of hospital stay was 4-6 days in 25(50%) out of 50 patients and 7-9 days in 15 patients. The mean value was 6.1±2.9.

Table 6: Mean score per Parameter

Parameter	Mean ± SD	Range
RANSON's score	4.7±1.6	0-7
BISAP's score	1.5±1.3	0-4
BALTHAZAR's CT severity index	4.7±1.9	0-8

Table 7: Distribution of cases perDuration of Hospital stay and Different Scores

Duration of Hospital stay	RANSON's score		BISAP's score		BALTHAZAR's CT severity index	
	Mean	SD	Mean	SD	Mean	SD
0-3	4.67	1.63	1.50	1.52	4.17	2.14
4-6	4.84	1.68	1.52	1.12	4.76	1.81
7-9	4.80	1.66	1.60	1.45	4.93	2.09
>9	3.50	1.00	1.00	1.41	4.50	1.00
Total	4.70	1.62	1.50	1.27	4.72	1.85
ANOVA p value	0.50		0.88		0.86	

Graph 6: Distribution of cases according toDuration of Hospital stay and Different Scores

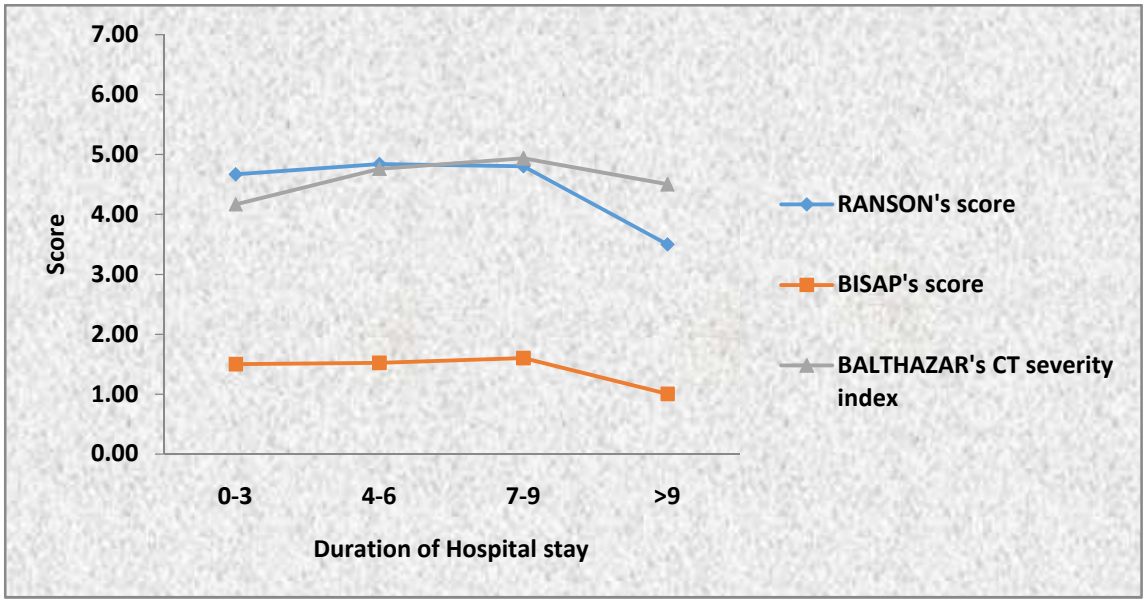


Table 8: Correlation of Different Scores with Duration of Hospital stay and Presence of Complications

Pearson Correlation	Duration of Hospital stay		Complications	
	r value	p value	r value	p value
RANSON's score	-0.194	0.176	0.006	0.969
BISAP's score	-0.044	0.762	0.036	0.806
BALTHAZAR's CT severity index	0.074	0.610	0.314	0.026 (Sig)

Table 9: Distribution of Complications according to BALTHAZAR's CT severity index.

BALTHAZAR's CT severity index	Total	No. of Complications	Cumulative no. of Complications	Cumulative Proportion of no. of Complications
0	4	0	0	0.00
2	1	0	0	0.00
4	18	5	5	0.38
5	7	0	5	0.38
6	15	6	11	0.85
7	3	1	12	0.92
8	2	1	13	1.00
Total	50	13		

Graph 7: Distribution of Complications according to BALTHAZAR's CT severity index

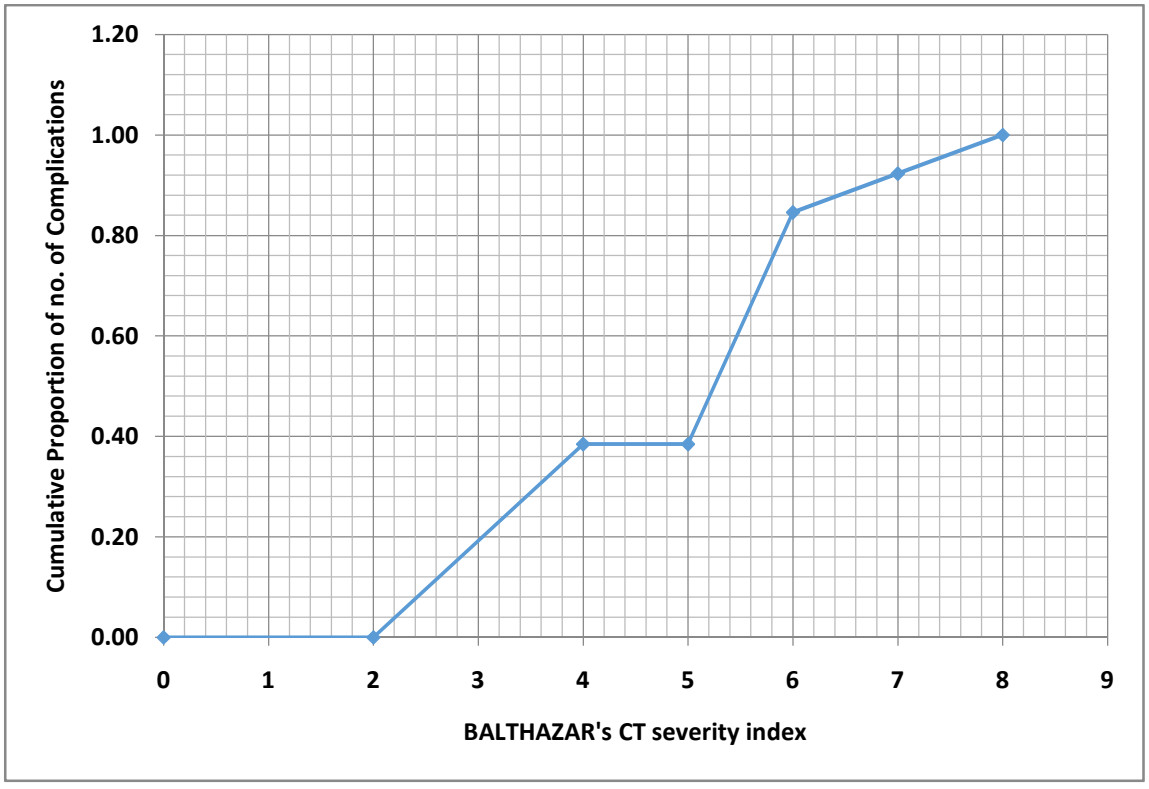


Table 10: Distribution of Complications according to BISAP's score

BISAP's score	Total	No. of Complications	Cumulative no. of Complications	Cumulative Proportion of no. of Complications
0	15	4	4	0.31
1	10	2	6	0.46
2	13	4	10	0.77
3	9	3	13	1.00
4	3	0	13	1.00
Total	50	13		

Graph 8: Distribution of Complications according to BISAP's score

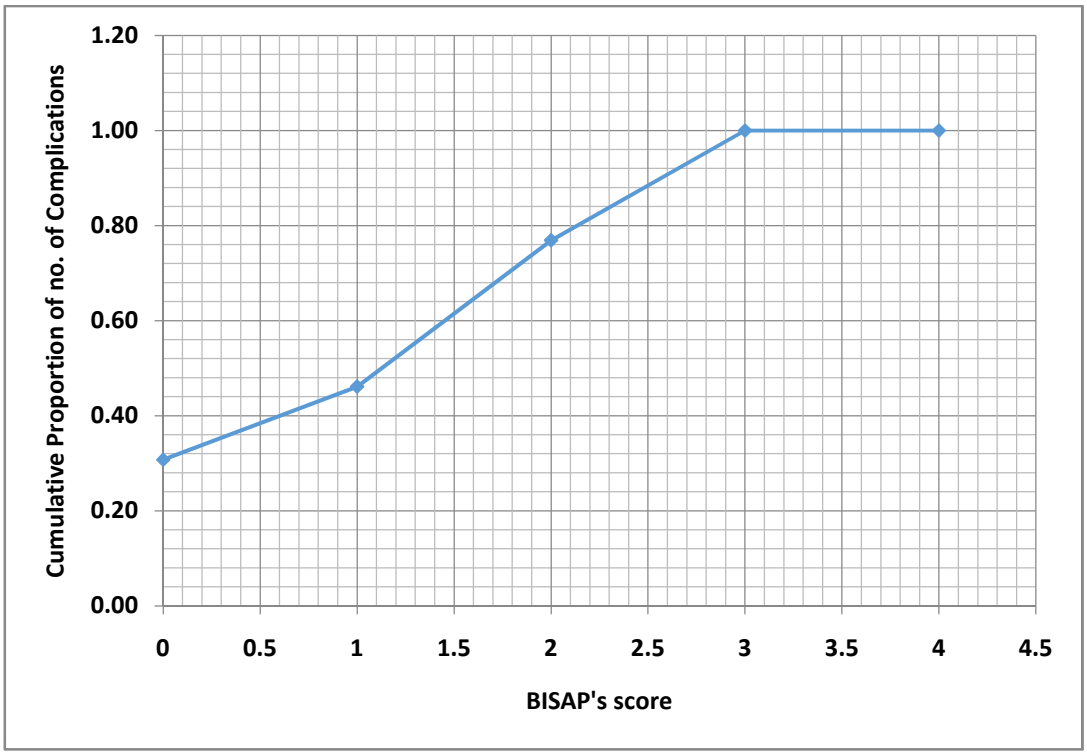


Table11: Distribution of Complications according to RANSON's score

RANSON's score	Total	No. of Complications	Cumulative no. of Complications	Cumulative Proportion of no. of Complications
0	1	0	0	0.00
2	1	0	0	0.00
3	12	3	3	0.23
4	9	4	7	0.54
5	9	2	9	0.69
6	10	1	10	0.77
7	8	3	13	1.00
Total	50	13		

Graph 9: Distribution of Complications according to RANSON's score

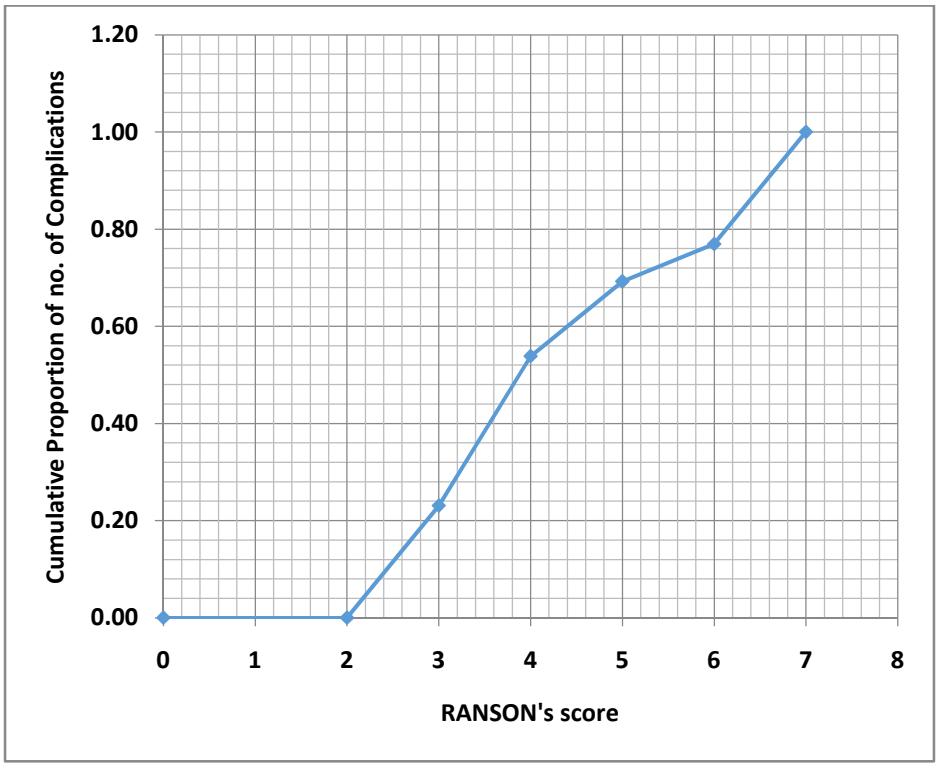
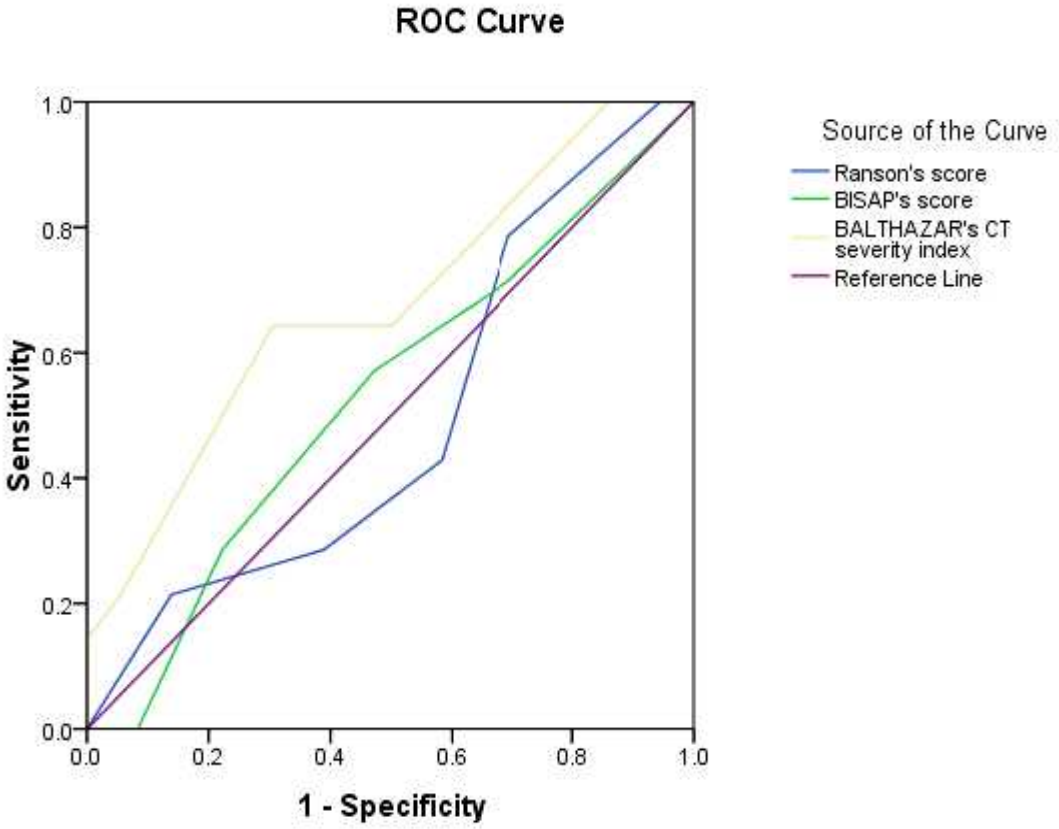


Table12 : ROC Analysis of RANSON's score, BISAP's score, BALTHAZAR's CT severity index in prediction of Complications

Scores	Area Under the Curve	p value	95% Confidence Interval	
RANSON's score	0.493	0.940	0.315	0.671
BISAP's score	0.532	0.730	0.353	0.711
BALTHAZAR's CT severity index	0.678	0.049 (Sig)	0.506	0.849



DISCUSSION

Acute pancreatitis is a common disease entity. The early identification of potentially severe acute pancreatitis enables the selection of patients who may require more intensive and invasive method of management than are appropriate in mild pancreatitis.

While diagnosing a case of acute pancreatitis, a through history, a complete physical examination and biochemical tests are necessary. Radiological conformation may require. In this study, analysis of clinical presentation of acute pancreatitis was done. Relevant investigations were carried out and appropriately managed depending upon the aetiology severity of acute pancreatitis.

COMPARISON OF AGE: -

The mean age of presentation in our study was 37.6 years and is comparable to the study by Kashid A et al.Other studies had late presentation in the 5th and 6th decade. This is probably because alcohol was the main etiological factor in our study which presents usually in the younger age group.

Table 13: Comparison of age:

Mean Age	Kashid A et al[23]	Choudhuri G et al[24]	Pupelis G Et al[25](n=274)	Our study (n=50)
Mean age in Years	35	44.8	47	37.6

COMPARISON OF SEX: -

There was male predominance in our study with males accounting for 90%. Out of 50 patients 45 (90%) were male and 5 (10%)were female. The other studies also had a higher percentage of males. This could beattributed to alcohol which was the main etiologic agent in our society.

Table 14: Comparison of sex

Mean age	Kashid A et al[23]	Choudhuri G et al[24]	Pupelis G Et al[25](n=274)	Our study (n=50)
Male %	70.91	66.6	73.7	90
Female%	29.09	33.4	26.3	10

HOSPITAL STAY

Mean hospital stay in our study was 6.1 days; it was comparable to the study by Choudhuri G et al.

Table 15: Hospital stay

Mean Hospital stay	Kashid A et al[23]	Choudhuri G et al[24]	Our study (n=50)
In days	10	6.6	6.1

MORTALITY

Mortality in our study was 2%, it was comparable to the study by Buchler MW et al.

Table 16: Mortality

Mortality	Kashid A et al[23]	Choudhuri G et al[24]	Buchler MW et al[26](n=86)	Our study (n=50)
Percentage	5.45	6.5	3.4	2.0

CTSI AND OUTCOME PREDICTION

Table 17: CTSI outcome prediction

STUDIES	DEATH	LENGTH OF STAY (Mean in Days)	COMPLICATIONS
Ting -Kai leung et al[7] (n=85)	5.3%	5.6	5.9
Our study (n=50)	2.0%	6.1	4.5

Table 18

COMPARISON 0F 'P' VALUE OF CTSI, RANSON'S, BISAP'S SCORES

STUDIES	BISAP's	Ranson's	Balthazar CTSI
Singh VK et al[4](n=339)	5.3	-	-
Ting -Kai leung et al[7] (n=85)	-	-	0.05
Muddanna V et al[5] (185)	-	0.74	-
Our study (n=50)	0.80	0.96	0.026

- A 7y/M patient underwent pancretiac Necrosectomy for pancreatic necrosis.

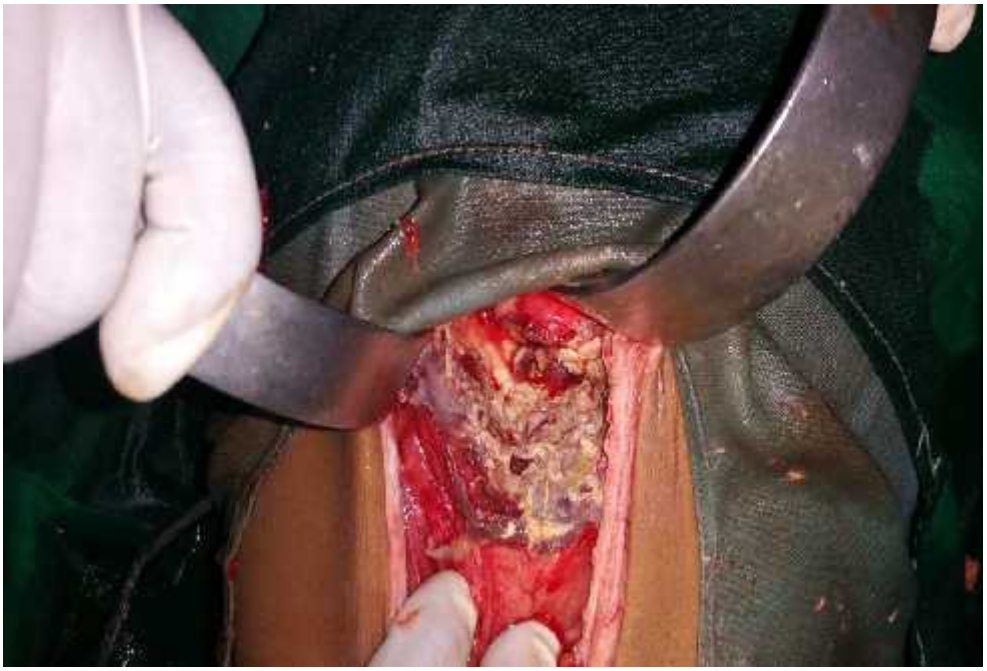


Fig 8: Necrosectomy

- A 16Y/M patient underwent Lateral pancreticojejunostomy for gallstone pancreatitis.

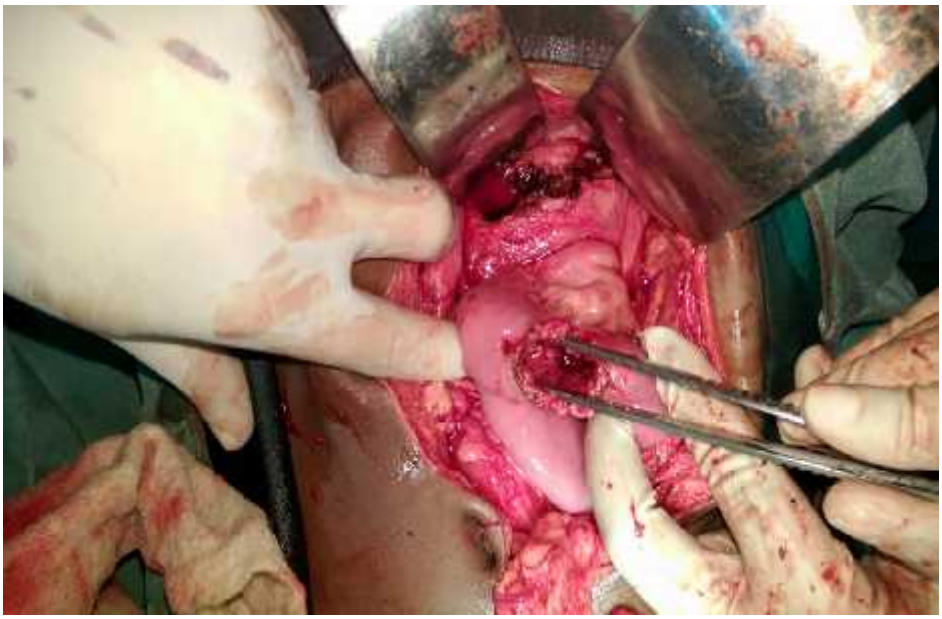


Fig 9: Ileal resection for anastomosis of Pancreatic duct

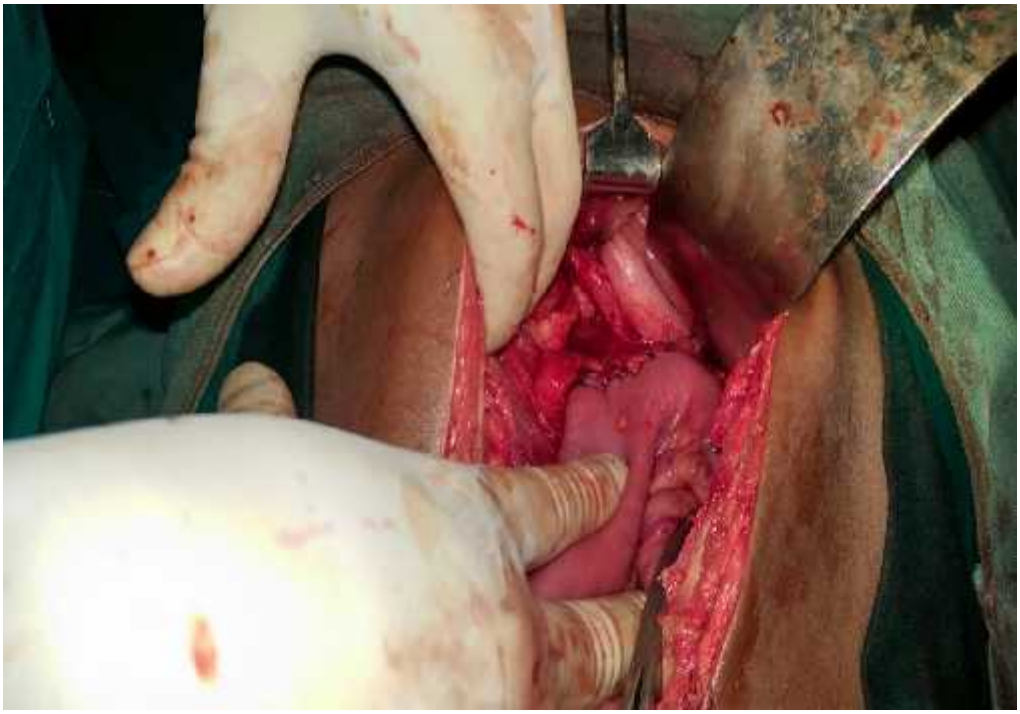


Fig 10: Lateral pancreatico jejunostomy

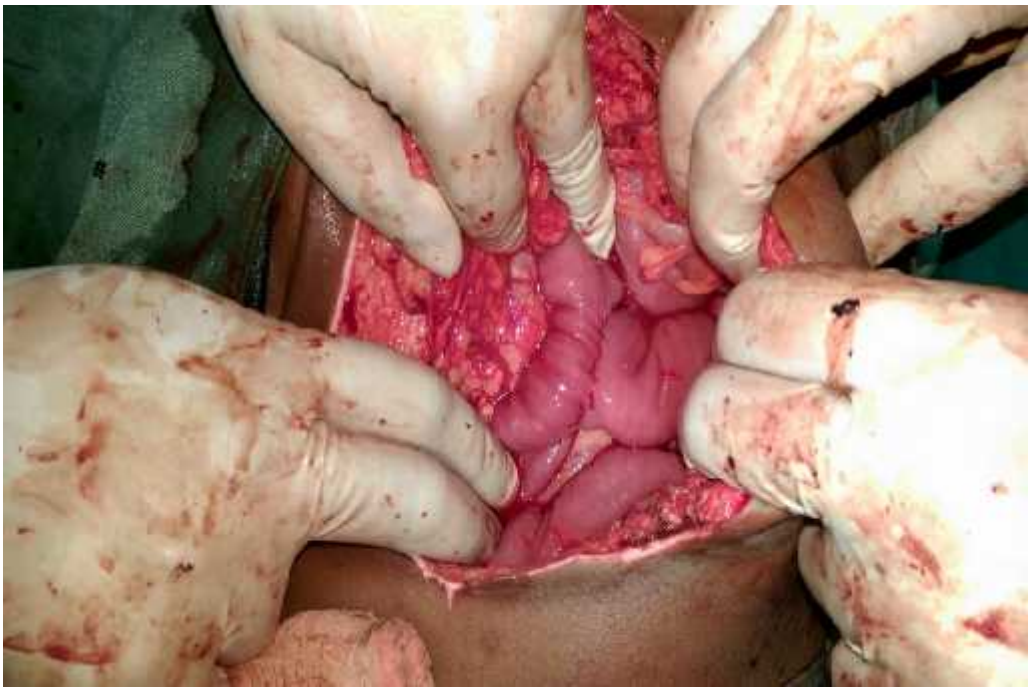


Fig 11: Roux en Y

CONCLUSION

- Most common in men.
- The peak incidence was 4th decade in both sexes.
- Serum lipase assessment (sensitivity 98%) is the gold standard diagnostic test than serum amylase (sensitivity 78%).
- USG is the initial radiological investigation for acute pancreatitis.
- CECT abdomen was more accurate in identifying the severity of acute pancreatitis.
- **Balthazar CTSI score was more accurate than BISAP's and Ranson's score, with BISAP's score being more easy to assess organ failure.**
- Complications are common with mild and severe acute pancreatitis.
- Most of the patients treated with conservative management.

SUMMARY

- The study includes a total of 50 patients of acute pancreatitis. 45 males and 5 females.
- The peak incidence in male and in female 4th decade in life.
- All the patients were investigated to find out complications (systemic/ local).
- Systemic complications were diagnosed by routine blood investigation, RFT, LFT, serum calcium and chest X ray.
- Local complications were diagnosed by USG abdomen and CT scan.
- 3 patients underwent surgery 2Frey's procedure & 1 Pancreatic Necrosectomy.
- Local complications were managed with conservative and operative procedure.
- All the patients were admitted in ICU and managedconservatively.
- There were 6 patients with multi organ failure was the most common cause of death.

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ANNEXURE – I

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 22-11-2014 at 3.30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title A comparison between clinical scores and Radiological evaluation in assessing the severity of acute pancreatitis- A prospective study

Name of P.G. student Dr. Anup Kussad
Dept of Surgery

Name of Guide/Co-investigator Dr Balasaheb B Metan.
Prof of Surgery.

for DR.TEJASWINI VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE – II

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT:	**A Comparison Between Clinical Scores and Radiological Evaluation in Assessing the Severity of Acute Pancreatitis A Prospective Study**
PRINCIPAL INVESTIGATOR:	**Dr. Anup Kubsad** Department of General Surgery Email: anupkubsad@gmail.com
PG GUIDE:	**Dr. Balasaheb B. Metan** Professor of Surgery B.L.D.E. University's Shri B.M. Patil Medical College & Hospital & Research Centre, Sholapur Road, VIJAYAPUR – 586103.

PURPOSE OF RESEARCH:

I have been informed that this study is A Comparison Between Clinical Scores and Radiological Evaluation in Assessing the Severity of Acute Pancreatitis - A Prospective Study

I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I understand that I will undergo detailed history, clinical examination,and laboratory.

BENEFITS:

Prevention of complications and to improve quality of life.

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator’s research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr.Anup Kubsad is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with mean that a copy of this consent form will be given to me to keep it and for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Anup Kubsad will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language

Date: Dr. BALASAHEB B. METAN (Guide) Dr.Anup Kubsad (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Anup Kubsad has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore, I agree to give my consent to participate as a subject in this research project.

_____

_____

(Participant)

Date

_____

_____

(Witness to above signature)

Date

ANNEXURE – III

CASE PROFORMA

1) Name	:	CASE NO	:
2) Age/sex	:	IP NO	:
3) Religion	:	Date of admission	:
		Date of surgery	:
4) Occupation	:	Date of discharge	:
5) Residence	:	Unit	:
		Diagnosis	:

6) Chief complaints:

7) History of presenting illness:

8) PastHistory:

9) Treatment history – Any surgery:

Drug intake:

10) History of trauma:

10) Personal History – Diet – Habits -

Appetite -

Bowel/Bladder -

Sleep -

11) Family History -

12) General Physical Examination -

Built	Nourishment
Pulse rate	Pallor:
Blood pressure	Icterus

Respiratory rate	Cyanosis
Temperature	Clubbing
Generalized lymphadenopathy	Pedal edema

13) Other systemic examination

- Abdominal system
- Respiratory system:
- Cardiovascular system:
- Central nervous system:

14) INVESTIGATIONS UNDERGONE BY PATIENT:

The following investigations and other ancillary investigations will be performed as deemed necessary for the individual cases and necessary will be repeated after 48 hours of admission if required.

On admission:

- Complete Blood Count
- Urine examination
- Serum Amylase
- Serum Lipase
- Blood Glucose level
- Serum Aspartate transaminase
- Serum Lactate dehydrogenase
- Serum calcium
- Liver function tests

Radiological investigations

- Chest X ray
- USG abdomen
- CECT

After 48 hours:

- Leucocytecount :
- Haematocrit fall:
- Blood Glucose level:
- Serum calcium:
- Serum Aspartate transaminase:
- PaO2:
- Serum Lactate dehydrogenase:
- Blood urea nitrogen

after IVF hydration :

BISAP's score:

Ranson's score:

Balthazar's CTSI score:

FINAL DIAGNOSIS :

Surgical Intervention:

Date:

Operative Finding:

Post-operative Management

Improved Unchanged Expired

Duration of hospital stay:

GUIDE BIO-DATA

1. **Name** **:** Dr. Balasaheb Metan
2. **Age (in years)** **:** 64 years
3. **Date of birth** : 12/03/1952
4. **Educational Qualification:**

Degree	Name of the College	Name of the University	Year of Passing
M.B.B.S	Dr. V.M Medical College, Solapur	Shivaji University, Kolhapur	1974
M.S General Surgery	Dr. V.M Medical College, Solapur	Shivaji University, Kolhapur	1980

5. **Present position** **:** Professor
6. **K.M.C Registration No** **:** 39414 September 1994
7. **Teaching Experience** : 14 years
8. **Publications** **:** 4
9. **Research projects** **:** 2

MASTER CHART

Sl no.	Name	IP NO/unit	Age/Sex	DOA	Diagnosis	DOD	Ranson's score	BISAP's score	BALTHAZAR's CT severity index	Treatment	Remarks
1	Shashidhar Badiger	35873 surg 3rd	39 M	11/26/2014	acute pancreatitis	12/1/2014	7	2	6	conservative	Chronic Pancretitis
2	Sangamesh Arenad	36933	31 M	12/7/2014	acute on chronic pancreatitis	1/8/2015	3	2	4	conservative	Improved
3	Umesh Navi	37012 surg 4h	34 M	12/31/2014	acute pancreatitis	1/8/2015	2	0	Not done	conservative	Improved
4	Sagar Sarwad	712 med 2nd	23 M	1/8/2015	acute pancreatitis	1/19/2015	2	0	2	conservative	Improved
5	Somanath Kambale	228 surg 5th	45 M	1/2/2015	acute pancreatitis	1/9/2015	4	2	6	conservative	Chronic Pancreatitis
6	Mahantesh Golasangi	513 med 1st	29 M	1/5/2015	acute pancreatitis	1/9/2015	3	0	4	conservative	Improved
7	Manikanth Hadimani	937 med 4th	42 M	1/11/2015	acute pancreatitis	1/11/2015	7	3	6	conservative	ARDS
8	Davalmalik Mulla	2271 med 1st	35 M	3/15/2015	acute pancreatitis	3/15/2015	5	2	5	conservative	Improved
9	Allabee Hulajanti	11619 surg 1st	90 F	4/6/2015	acute pancreatitis	4/17/2015	5	3	Not done	conservative	Improved
10	Sphoorthi Patil	9848 pedia 3rd	3 F	4/10/2015	acute pancreatitis	4/18/2015	4	0	4	conservative	Improved
11	Prakash Dengi	12539 surg 1st	34 M	4/13/2015	acute pancreatitis	4/21/2015	6	1	6	conservative	Hypocalcemia
12	Basavaraj Hugar	11975 surg 5th	25 M	4/16/2015	acute pancreatitis	4/25/2015	3	0	4	conservative	Improved
13	Vishwanath Navi	11807 surg 5th	16 M	4/15/2015	Acute/Chronic Pancreatitis (Gallstones)	5/16/2015	0	0	6	Frey's Procedure on 05-05-2015	Improved
14	Prabhuling Kamble	12047 med 3rd	61 M	4/22/2015	acute pancreatitis	4/24/2015	6	3	Not done	conservative	Improved
15	Umashankar Joshi	10795 surg 6th	38 M	4/11/2015	acute pancreatitis	4/15/2015	3	0	Not done	conservative	Improved
16	Nivedita	10783	20 F	4/16/2015	acute pancreatitis	4/22/2015	4	0	4	conservative	Chronic Pancretitis
17	Bhilaji Pawar	11463 surg 3rd	50 M	5/13/2015	acute pancreatitis	5/18/2015	6	2	Not done	conservative	Improved
18	Ravi Kambale	15881 pedia 3rd	7 M	5/19/2015	acute pancreatitis(Pancreatic Necrosis Abscess)	6/21/2015	5	3	6	Necrosectomy+Peritoneal Lavage in GA onn 26-05-2015	Improved
19	Mallikarjun Patil	20223 med 3rd	35 M	6/2/2015	acute pancreatitis	6/9/2015	7	3	4	conservative	Improved
20	Mallikarjun Karjol	17686 surg 3rd	30 M	6/3/2015	acute pancraetitis	6/11/2015	7	3	5	conservative	Improved
21	Vishwanath Kumashi	18982 surg 2nd	25 M	6/12/2015	acute pancraetitis	6/17/2015	3	0	4	conservative	Improved
22	Jitani Bepari	19540 surg 3rd	35 M	6/16/2015	acute pancreatitis	6/24/2015	4	2	6	conservative	Improved

23	Gurubasayya Vastrad	21230 med 3rd	32 M	6/20/2015	acute pancreatitis	6/26/2015	3	0	6	conservative	Improved
24	Fayas Inamdar	21277 surg 6th	33 M	6/21/2015	acute pancreatitis	6/28/2015	6	4	6	conservative	Improved
25	Vittal Choori	22276 surg 5th	60 M	6/24/2015	acute pancraetitis	7/1/2015	3	0	4	conservative	Improved
26	Hulageppa Goudar	23433 Surgery 3rd	50 M	7/16/2016	acute pancreatitis	7/21/2016	3	0	4	conservative	Improved
27	Ningappa Ainapur	23917 surg 6th	55 M	7/25/2015	acute pancreatitis(gallstones)	8/6/2015	5	2	6	conservative	Chronic Pancreatitis
28	Honappa Kabade	23946 med 1st	40 M	7/30/2015	acute pancreatitis	8/7/2015	3	0	2	conservative	Improved
29	Nagesh Benal	24896 surg 6th	24 M	8/2/2015	acute pancreatitis	8/12/2015	7	3	6	conservative	Improved
30	Appashi Biradar	41685 surg 5th	26 M	12/27/2015	acute pancreatitis	1/2/2016	6	1	4	conservative	Hypocalcemia
31	Tayavva Durgamuragi	41446 med	50 F	12/30/2015	acute pancreatitis	1/13/2016	6	1	5	conservative	Improved
32	Siddappa Byakod	41591 med 4th	50 M	12/31/2015	acute pancreatitis	1/9/2016	4	0	6	conservative	Improved
33	Chotesaab Jahagirdar	953 surg 1st	55 M	1/9/2016	acute pancreatitis	1/12/2016	3	1	4	conservative	Improved
34	Sharanappa Sankanur	697 surg 4th	35 M	1/16/2016	acute pancreatitis	1/23/2016	4	2	4	conservative	Improved
35	Basappa Bannur	1699 surg 2nd	48 M	1/16/2016	acute pancreatitis	1/22/2016	6	1	5	conservative	Improved
36	Umesh Hatti	2672 surg 1st	33 M	1/22/2016	acute pancreatitis	2/1/2016	5	0	4	conservative	Improved
37	Bandachari Sangam	2839 surg 1st	83 M	1/23/2016	acute pancreatitis	2/1/2016	7	2	6	conservative	ARDS & ARF
38	Gurusiddappa Patil	211	56M	1/28/2016	acute pancreatitis	2/1/2016	9	3	8	conservative	EXPIRED
39	Siddamma Janawad	931	30F	2/1/2016	acute pancreatitis	2/19/2016	2	0	7	frey's Procedure on 05-02-2016	Improved
40	Bhimanna Humbalnur	5408 surg 2nd	40 M	2/3/2016	acute pancreatitis	2/19/2016	4	2	6	conservative	Pseudocyst of pancreas
41	Anil Bhendre	5418 surg 2nd	27 M	2/7/2016	acute pancreatitis	2/21/2016	5	3	5	conservative	Improved
42	Siddappa Somagar	1210 Med 5th	48 M	2/9/2016	acute pancreatitis	2/16/2016	3	0	6	conservative	Improved
43	Rajshekar Chanda	1538	30M	2/20/2016	acute pancreatitis	3/6/2016	5	3	7	conservative	Chronic Pancreatitis
44	Prakash Handge	7745 med 1st	40 M	4/3/2016	acute pancreatitis	4/20/2016	6	1	6	conservative	Improved
45	Basavaraj Ujarati	8057 med 3rd	34 M	4/5/2016	acute pancreatitis	4/10/2016	5	1	6	conservative	Improved
46	Shrishail Parashetty	11831 med 1st	30 M	4/7/2016	acute pancreatitis	4/14/2016	7	4	5	conservative	Improved
47	AfZal Rajesab	4170	41M	4/10/2016	acute pancreatitis	4/15/2016	5	0	4	conservative	Improved
48	Shashikant Ambure	12010 surg 1st	30 M	4/11/2016	acute pancreatitis	19--04-2016	6	1	5	conservative	Improved
49	Chidanand Kulekumatagi	13707 med 2nd	45 M	4/17/2016	acute pancreatitis	4/26/2016	4	2	6	conservative	Improved
50	Laxman Pundibij	20566 surg 2nd	27M	6/21/2016	acute pancreatitis	6/27/2016	5	1	6	conservative	ARDS