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Comparison of Ranson, Bisap, CTSI Scores and CRP in Predicting Severity, Organ Failure and Mortality in Acute Pancreatitis

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I. INTRODUCTION

Acute pancreatitis is one of the important causes of acute abdomen. Patients of acute pancreatitis usually get admitted in the surgical emergency units. Generally chronic alcoholism is the important cause of acute pancreatitis in men &biliary tract pathology is the common etiology in women. Various epidemiological studies are concluded that most cases of acute pancreatitis are attributed to chronic alcoholism.

The significant variability in data indicates that the accurate measurement of the magnitude of problem is very difficult for the researchers. This is because of the following factors. 1)Lack of standardization in diagnosing acute pancreatitis with the laboratory and clinical background 2)variation in the inclusion and exclusion criteria's in different studies 3)regarding tools used for the consumption of alcohol 4)confusion between acute and chronic forms

The interesting fact is that the incidence of alcohol induced pancreatitis is increasing over the past decade. A study in United Kingdom shows that alcoholic pancreatitis incidence has increased from 14.5 per lakh population during 1989-1990 to 20.7 per lakh population in the year 1999-2002. Clinically acute pancreatitis occurs only in five percent of heavy alcoholics.

Most of the acute pancreatitis patients had only milder forms, which can be managed conservatively and they recovered completely. The severe forms occurred only in 15% of patients. In recent studies the mortality rate is greatly decreased (from 35-85%-10-20%). Severe acute pancreatitis has two stages of the disease. Initial inflammation and necrotic changes of the parenchyma is followed by signs of systemic inflammatory response syndrome leads to multi organ failure within 7-10 days. About half of patients with severe forms of pancreatitis died in the first week due to complications like ARDS/MODS. The mortality rate in patients with MODS stage varies from 30 % to maximum 100 %. These patients need early resuscitation, intensive care to prevent multi organ/respiratory failure. Some patients need surgical interventions like retroperitoneal drainage and necrosectomy in pancreatic necrosis and infected necrosis.

Scoring systems utilize various biochemical, clinical and radiological parameters to evaluate the severity of acute pancreatitis. The scoring systems aid in assessing the prognosis, plan treatment and to predict mortality and severity. This prospective study attempts to compare the various scoring systems to predict severity of pancreatitis.

KEY WORDS:Pancreatitis, Severity scoring systems, Ranson, CTSI, BISAP, CRP

AIMS AND OBJECTIVES

- The study is undertaken for the comparison of RANSON, BISAP, CTSI SCORES AND CRP in predicting severity, organ failure and mortality in acute pancreatitis
- The study aims to find the single most reliable score to predict the severity of pancreatitis.
- To study the usefulness of the scores in predicting the severity of pancreatitis and as guide for referral to higher center .

II. REVIEW OF LITERATURE

Acute pancreatitis has challenging clinical course in the prediction of severity and prognosis. According to most of the case reports the severe cases contributes to 15-25%. In the following ways we can assess the severity.We can assess the severity from history, symptoms and signs, laboratory findings, radiological imaging. In the initial periods the serum amylase is the only investigation used to diagnose acute pancreatitis. In

1970, John Ranson attempted to define the objective criterias to assess the severity of acute pancreatitis. During the early phase of 1980, the severity of pancreatitis is assessed only intra operatively after seeing the amount of necrosis and the presence of signs of infection in necrotic tissues. After the introduction of CT the severity assessment of acute pancreatitis becomes an uncomplicated one. In initial days the morphological criteria is used. Nowadays the presence of multi organ failure dominates the clinicalpicture.

AGE AND COMORBID ILLNESS**:**
Advanced age & the presence of other comorbid conditions like diabetes and cardiac diseases associated with poorprognosis.
CLINICAL SIGNS:

Presence of fever, cardio vascular instability, respiratory distress, altered sensorium, mass abdomen, paralytic ileus increases the mortality to 25 % in operated patients and 70% in conservatively managed patients.
ATLANTA CLASSIFICATION:

This classification differentiates between mild and severe types of acute pancreatitis. It depends on the clinical and morphological findings.Mild forms otherwise called edematous pancreatitis will have minimal organ dysfunction that will lead onto good outcome. Morphology will reveal edema in interstitium and microscopic fat necrosis. The other form severe or necrotizing pancreatitis will have features of multi organ dysfunction, pancreatic necrosis, abscess, pseudocyst formation. Morphology reveals extensive fat necrosis andhemorrhagic areas in pancreas or peri pancreatic fat.
MULTI PARAMETER SCORING SYSTEM:

Ranson and Clement Imrie proposed the analysis of multiple clinical and biochemical parameters which contributes to complications and death. Their system paved the way for the development of multiple newer scoring systems.
RANSON SYSTEM:

This system analyzes the severity from the clinical and the biochemical parameters during and 48 hours after admission. A total of 11 points were taken. Five from the time of admission and the remaining from the next assessment after 48 hours from the abnormal values were taken. Since the accuracy and the positive predictive values are low in case of biliary disease induced pancreatitis this scoring system undergone some changes for biliary pancreatitis.
IMRIE AND GLASGOW SCORING SYSTEM:

This system is based on an interventional study in acute pancreatitis. It includes age of the patient at the time of admission and physiological and biochemicalparametersobtainedduringthefirsttwodaysofadmission.
There are some changes to improve the performance in biliary stone pancreatitis. Even after modification the sensitivity and the specificity remains only to a moderate level. The overall sensitivity is less than 70% and the positive predictive value is70%.

APACHE II system:

The Acute Physiology And Chronic Health Evaluation developed by intensive care research unit in United States. There are some changes from the initial system which reduces the variables from 35 to 11 which is called APACHE II. This system evaluates the patient in 24 hours and at 48 hours. The performance of APACHE scoring system is the same during hospitalization and at 24 hours. The results may vary depending upon the etiology of the disease and intensive care set up. The advantage of APACHE system is in its speed and easiness in application. There is also a possibility that recalculation is possible at any period throughout the disease for monitoring. The Atlanta classification proposed that score of 8 or more of APACHE II scoring system indicates severe attack.

ORGAN FAILURE SCORING SYSTEM:

This scoring system is applied in some studies to assess organ failure and prognosis. The MOF scoring system is mainly concentrating the predictionof survival based on organ failure. The cardio respiratory system, renal system, haematological, hepato biliary, neurological and alimentary system is monitored in this scoring system. The Marshall and SOFA are the newer system of organ failure scoring systems which describe the individual and multi organ failure. In both systems, six major systems are considered which include respiratory, cardiac, hepato-biliary, renal, neurological and haematological system. In SOFA scoring system specific parameters like ventilatory support and inotropic supports areadded.
MOF SCORING SYSTEM:

This system is based on the presence or absence of multi organ failure. This scoring system poorly assesses the complications like necrosis. The prediction of nonsurvival when the score is more than 3 is having high sensitivity and specificity.

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MARSHALL SCORING SYSTEM:

Marshall scoring system is a modified one because it excludes the liver function. Other systems are monitored. The parameters closely correspond to Atlanta classification. There are two scores assessed in first 3 days of admission. Marshall scoring system is having comparable results with APACHE II scoring system in prediction of mortality in acute pancreatitis.

BISAP SCORE

The bedside index for severity in acute pancreatitis (BISAP), a newer prognostic scoring system, has been proposed as a simple and clinically oriented severity scoring system for early identification of patients with acute pancreatitis

SOFA scoring system:

The SOFA scoring system gives the advantages like very easy calculations in therapeutic requirements and can compare acute pancreatitis with other diseases which are in need of critical care.

HEMATOCRIT:

More than 44% during admission is related to complications like necrosis or multi organ dysfunction.

BUN & SERUM CREATININE:

Renal failure (serum creatinine> 2 mgs%) is an important predictor of mortality.

BLOOD GLUCOSE:

At the time of admission if the blood sugar value is more than 250 mgs there is high chance of development of pancreatic necrosis, multi organ failure and mortality.

RANSON'S PROGNOSTIC SCORING SYSTEM

NON GALL STONEETIOLOGY

Duringadmission Within 48hours

Age>55 years hematocrit fall>10%

TLC>16000/mm^2 elevation of BUN >5mg%

Blood sugar>200mg% serum Ca^{++}< 8mg%

LDH>350 IU/L PaO_2< 55mmHg

AST >250U/dl base deficit >4 mEg/ L

Fluid sequestration >6

GALL STONE ETIOLOGY

Age >70 years hematocrit fall >10%

TLC>18000/mm^3 elevation of BUN > 2mg%

Blood sugar>220mg% serum Ca^{++}< 8mg%

LDH>400 IU/L base deficit >5 mEg/L

AST >250U/dl Fluid sequestration >4 litresTLC-Total leucocyte count , LDH-Lactatedehydrogenase AST-Aspartate transaminase., BUN-Blood urea nitrogen. RANSON'S SCORE 3 or Above- Defines severepancreatitis

TRYPSIN ACTIVATION PEPTIDE &CARBOXY PEPTIDE BACTIVATIONPEPTIDE:

According to atlanta scoring system the measurement of CPAP has both diagnostic and prognostic significance. It is also correlated with the severity of the disease. The limitation of this parameter is the requirement of RIA for its measurement.

CYTOKINES:

IL-6 & IL-8 measurements are having importance in assessing the severity of the disease. IL-6 level is a good indicator of severity. It is elevated enormously in complicated acute pancreatitis. The rise in CRP occurs 24-36 hours earlier than CRP. IL-8 is found to be the early marker in the prediction of severity in the first 24 hours of attack. It decreases rapidly over the next 72-96 hours. Its rise is in parallel line with IL-6. It is proved to be the best marker for monitoring the fatal complications.

PRO CALCITONIN:

This is one of the best biochemical parameter the level of which correlate well with the occurrence of infection and sepsis. Infection occurs in the setting of necrotic tissues which is having a major impact on survival and also alters the mode of treatment.

Radiological Assessment

Balthazar Classification And Severity Index:

Balthazar classifies the severity of acute pancreatitis into 3 different stages mild, moderate, severe.

Stage A (0): About 15-28 % of patients with acute pancreatitis are showing normal CT images. This indicates that the disease is very mild so that there is no fluid collection and no parenchymal changes. In this state the diagnosis is verychallenging.

Stage B: Alteration of parenchymal contour and heterogenous attenuation of the parenchyma are the imaging findings in this state.

Stage C: Along with the stage B changes there will be presence of streaky densities in the peri pancreatic region.

Stage D: It includes stage C changes and a single focus of fluid collection in the peri pancreatic region.

Stage E: There are multiple focus fluid collections in the peri pancreatic tissues along with the retro peritoneal air which indicates presence of infection.

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Mortele Modification Of Severity Index:

This is the modification of Balthazar severity index. It gives a score of 0 to the normal pancreas. Score of 2 indicates intra pancreatic abnormalities along with the presence or absence of peri pancreatic inflammatory process. Score of 4 denotes the presence of fluid collection in pancreas and/or the peri pancreatic region or the presence of pancreatic necrosis. Additional points are given depending upon the extent of pancreatic necrosis. Additional 2 points are also added if there are extra pancreatic complications like pleural effusion, free fluid in the abdomen, vascular involvement. Severity score of 0-3 indicates mild variant, 4-6 indicates moderate type, 7- 10 shows severepancreatitis.

CT SCORING : (BALTHAZAR)

GRADING	APPEARANCE	SCORE
A	Normal appearance	0
B	Focal or diffuse enlargement of Pancreas	1
C	Peripancreatic inflammation	2
D	Intra / extra pancreatic fluid Collection	3
E	2 or more fluid collection / air in pancreas or retroperitoneum.	4

CT NECROSIS INDEX:

This is based on necrosis seen in the contrast enhanced CT scan.

Percentage of necrosis	scoring
0% of necrosed pancreas	0
<33% of necrosed pancreas	2
33 – 50 % of necrosed pancreas	4
>50% of necrosed pancreas	6

CT SEVERITY INDEX**:**

CT severity index = CT Balthazar score + Necrosis index.

Score 0 – 3 ---------- mild acute pancreatitis.
Score4-6 moderate acutepancreatitis
Score 7orabove severe acute pancreatitis

BISAP SCORE

The bedside index for severity in acute pancreatitis (BISAP), a newer prognostic scoring system, has been proposed as a simple and clinically oriented severity scoring system for early identification of patients with acute pancreatitis

- BUN :>25mg/dl(1 point)
- Impaired mental status : GCS < 15 (1 point)
- SIRS : Evidence of SIRS(1 point)
- Age: Age >60 years (1 point)
- Pleural Effusion : Imaging reveals pleural effusion (1 point)

0-2 points: lower mortality
3-5 points: higher mortality.

C- Reactive protein

A C-reactive protein (CRP) value can be obtained 24-48 hours after presentation to provide some indication of prognosis. Higher levels have been shown to correlate with a propensity toward organ failure. A CRP value in double figures (ie, ≥ 10 mg/dL) strongly indicates severe pancreatitis

CORRELATION OF MODIFIED CTSI WITH OTHER PARAMETERS:

1. Length of hospitalization is increased in patients with severeforms.
2. The percentage of patients undergoing surgical procedure is also increased in severe forms ofpancreatitis.
3. Infections are more common in severeforms.
4. Organ dysfunction is common in severe forms when compared to the mild ones.

TREATMENT

Initial management:

The management principles include early diagnosis, to assess the severity, to identify the complications and manage the patient in a multi disciplinary approach. When the abdominal exploration is planned the diagnosis should be thought of and better to be confirmed or excluded because the incidence of morbidity and mortality is very high in postoperativepatients.

MANAGEMENT OF PAIN:

Meperidine analogues are the mainstay of analgesics used in the treatment of pancreatitis. Morphine is relatively contra indicated as it may increase the sphincter of oddi spasm.

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FLUID THERAPY

Intensive fluid therapy with electrolyte replacement is the mainstay of treatment in acute pancreatitis due to third space loss of fluid in retro peritoneum. This will also lead onto increased hematocrit and hypotension. Vomiting will lead onto metabolic alkalosis whereas hypotension will lead on to metabolic acidosis. Hypoalbuminemiaiscaused by both chronic alcoholism and the acute attack of pancreatitis. Hypocalcemiacan be treated by intra venous calcium administration.

Ryle's tube aspiration will not alter the course and the prognosis of the disease.

ROLE OF ANTIBIOTICS:

Prophylactic antibiotic may be of immense value in severe pancreatitis with infective complications.

FEEDING:

Early oral feeding is advisable if the patient is able to tolerate it because it will prevent bacterial translocation and infectious complications.

ENDOSCOPY :

Early endoscopic stone retraction in biliary stone pancreatitis reduces the incidence of further attacks and infective complications.

COMPLICATIONS:

1) PANCREATIC FLUIDCOLLECTION:This condition is observed in about half of patients with the initial stage of pancreatitis. There is absence of wall of fibrous tissue. Some collections are within the pancreas. Some are in the peri pancreatic tissues. More than 50% of the collections will resolve over time. Unresolved collections will turn into pseudocyst of pancreas. This condition is easily diagnosed by serial ultra sound or CT scan of the abdomen. The fluid may get infected which can be identified by the presence of air bubbles. This condition is managed by either surgical or percutaneousdrainage.
2) PESUDOCYST FORMATION: pseudocyst formed from unresolved pancreatic fluid collections in edematous pancreatitis. The contents are pancreatic secretions with large amount of enzymes which are covered with fibrous wall. It occurs not only within the pancreas but also in omental bursa. Approximately 50% pseudocyst of pancreas resolves spontaneously by rupture either into the pancreatic duct or other parts of alimentary canal. It may lead to onto further complications like obstruction to the pancreatic duct or intra pancreatic portion of bile duct and duodenum. It may also erode into adjacent organs. Bleeding into the cyst either from the artery or vein, infection of the cyst are also the other complications. Infected pseudocyst of pancreas can be drained either percutaneously or by surgical drainage
3) PANCREATIC FLUID COLLECTION:This condition is observed in about half of patients with the initial stage of pancreatitis. There is absence of wall of fibrous tissue. Some collections are within the pancreas. Some are in the peri pancreatic tissues. More than 50% of the collections will resolve over time. Unresolved collections will turn into pseudocyst of pancreas. This condition is easily diagnosed by serial ultra sound or CT scan of the abdomen. The fluid may get infected which can be identified by the presence of air bubbles. This condition is managed by either surgical or percutaneousdrainage
4) PSEUDO CYSTFORMATION: pseudocyst formed from unresolved pancreatic fluid collections in edematous pancreatitis. The contents are pancreatic secretions with large amount of enzymes which are covered with fibrous wall. It occurs not only within the pancreas but also in omental bursa. Approximately50% pseudocyst of pancreas resolves spontaneously by rupture either into the pancreatic duct or into the other parts of the alimentary tract. It may lead onto further complications like obstruction to the pancreatic duct or intra pancreatic portion of bile duct and duodenum. It may also erode into adjacent organs.
5) Post Necrotic Fluidcollection:Innecrotising pancreatitis, necrosis and disruption of pancreatic duct will lead onto leakage of secretions into the necrotic portion of the pancreas. It is often confussed with acute pancreatitis.
6) VASCULARINVOLVEMENT:Vessels involvement in acute pancreatitis is characterized by erosion of pancreatic vessels which will lead onto bleeding and subsequent pseudoaneurysm formation. Splenic vein thrombosis is also one more complication. CECT of the abdomen and MRI will be useful in diagnosing this condition. Bleeding will lead onto death of the patient if not controlled in the

right time. Bleeding from the pseudo aneurysm is suspected when there is a sudden drop in the hematocrit level during acute attack. It can be easily controlled with angiography followed by transcatheter embolization.

7) Biliary Tractinvolvement:Obstruction of bile duct due to edema of pancreatic head or compression by pseudocyst of pancreas. In necrotizing disease, the periductal inflammation also causes obstruction. Pseudocyst of pancreas can encroach directly into the duct and produces erosion of intra hepatic bileducts.

8) GASTRO INTESTINALINVOLVEMENT:Post necrotic fluid collections can traverse through the transverse meso colon, mesentry of jejunum and also into the retro peritoneal structures. Stomach and duodenum are also involved when the collections are present in the omental bursa or in the perinephric region.A rare condition called groove pancreatitis in which the inflammation occurs at the level of pancreatic head within the groove that separates the duodenum and thepancreas.

9) SPLENIC VEIN THROMBOSIS AND THE FORMATION OF GASTRICVARICES:This complication occurs in severe forms of pancreatitis. It may lead onto gastric varices formation. Since bleeding from these varices is rare, there is no requirement of prophylactic treatment. If bleeding occurs it can be controlled bysplenectomy.

10) PANCREATICASCITES:Secretions into the peritoneal cavity due to rupture of ducts or pseudocyst. It can be diagnosed by measurement of amylase level in free fluid. This condition is initially treated with conservative line of management like nil per oral, Ryle's tube aspiration and attempt to reduce the secretions by somatostatin analogues. 50% of patients will respond to this type of conservative management. It is usually getting resolved within 14-21 days. Recurrent ascites is treated by stenting of the pancreatic duct endoscopically. ERCP can be used to locate the site of disruption. If the site of disruption is in the distal part resection will be the ideal treatment. If it is in the proximal part, Roux –en-Y drainage can bedone.

11) PANCREATIC FISTULA**:** Pancreatico pleural fistula can occur when the disruption of duct occurs into retro peritoneal pancreatic secretions which will finally enter into the pleural space. It is a rarecondition

12) SYSTEMICCOMPLICATIONS:Organ dysfunction is common in the first week. Sometimes it can be transient& it will not be associated with fatal outcome. If it is persistent it may lead onto fataloutcome.

AIMS OF THE STUDY:

1. The study is undertaken for the comparison of RANSON, BISAP, CTSI SCORES AND CRP in predicting severity, organ failure and mortality in acute pancreatitis
2. The study aims to find the single most reliable score to predict the severity of pancreatitis
3. To study the usefulness of the scores in predicting the severity of pancreatitis and as guide for referral to higher center

III. MATERIALS AND METHODS

PATIENTS:

Total of fifty patients include both male and female of adult age group who got admitted in the emergency surgical ward with the symptoms of acute pancreatitis and evaluation confirmed the presence of this serious illness.

PLACE OF THE STUDY:

Government Rajaji Hospital, Madurai.

PERIOD OF THE STUDY:

From January 2019 to June 2020

TYPE OF THE STUDY.:

It is a prospective study.

INCLUSION CRITERIA:

Patients got admitted in the emergency surgical ward with the symptoms of acute pancreatitis in which the biochemical and radiological evaluationconfirmed the presence of acute pancreatitis.Patients who had recurrent attacks of acute pancreatitis also included in this study.

EXCLUSION CRITERIA:

1. Patients with chronic pancreatitis are excluded from this study.
2. Patients with known co morbid conditions like diabetes mellitus, systemic hypertension, coronary artery disease, chronic obstructive pulmonary disease, chronic kidney disease are excluded from thisstudy.
3. Paediatric age group not included in this study.

PARAMETERS USED:Clinicalmanifestations, Ranson's clinical scoring system, BISAP score, CRP levels ,Radiological: CT abdomen (CTSI)

DIAGNOSTIC CRITERIA:

1) Clinical signs and symptoms of acute

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pancreatitis as described in review ofliterature.

2) Elevated serum amylase

3) CT with contrast confirming the presence of acutepancreatitis

SEVERITY ASSESSMENT:

Based on Ranson's criteria, BISAP score, CRP levels and radiological (contrast enhanced CT abdomen-Balthazar scoring) findings

IV. OBSERVATIONS AND DISCUSSION

1. CLINICAL MANIFESTATIONS & THE SEVERITY OF THE DISEASE:

In this study, various clinical symptoms and signswereobserved. The following clinical manifestations were commonlyencountered.

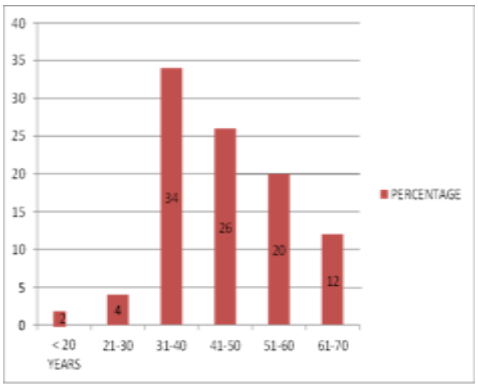
S.NO.	CLINICAL MANIFESTATIONS	PERCENTAGE
1.	Abdominal pain	100
2.	Abdominal distension	42
3.	Vomiting	72
4.	Fever	26
5.	Shock	6
6.	Guarding /rigidity	4
7.	Ascites	12

From this result, the presence of hemodynamic instability is an important factor associated with poor prognosis.

Thepresence of massive ascites isalso having pooroutcome. Those two patients who presented with significantguarding and rigidity had bad outcome in terms ofmortality.

2. ACUTE PANCREATITIS IN DIFFERENT AGEGROUPS:

AGE GROUP	NO OF PATIENTS	PERCENTAGE
<20 YEARS	1	2
21-30	2	4
31-40	17	34
41-50	13	26
51-60	10	20
61-70	7	14



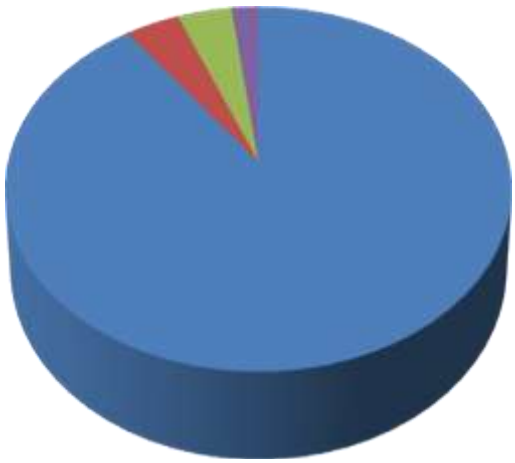
From this study most of the patients are in fourth decade ,followed by fifth and sixth decade. The disease is less common in older age group (>60yrs) and in younger age groups. So the high incidence of the disease occurs in middle age groups**.** .The incidence of disease in this age group is due to alcoholism.

3. SERUM AMYLASE AND THE SEVERITY OF THE DISEASE:

In this study, serum amylase levels were elevated enormously in recurrent attacks even though they were milder forms. In case of severe pancreatitis, the levels of amylase shows only mild to moderate elevation. Hence it is obvious that there is no correlation between the levels of serum amylase and the severity of the disease.

4. ETIOLOGY & THE SEVERITY OF THEDISEASE:

In this study of 50 patients, most of the patients were alcoholic. The various etiologies are listed below.



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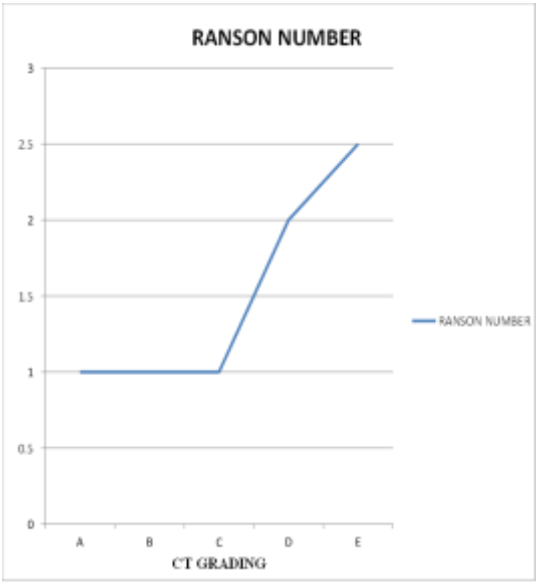
S.NO	ETIOLOGY	NO.OF PATIENTS	PERCENTAGE
1.	Alcoholism	45	90
2.	Biliary stones	2	4
3.	Post – operative	1	2
4.	Idiopathic	2	4

Almost all severe forms of pancreatitis were due to alcoholism. The two cases with biliary stone as the etiologic factor had milder form of acute pancreatitis. Of these fifty patients, only one patient developed pancreatits in the post – operative period

5. RANSON SCORE AND SEVERITY: Most of the patients had Ranson's score of 1 and 2. Out of 50 patients,5 patients expired. Four out of five hadRanson's score of 3 and one patient had Ranson's score of 2. From this we can conclude that the severity of the disease correlate well with the Ranson'sscore.

Ranson's score	Number of patients
0	5
1	32
2	9
3	4



6. BALTHAZAR SCORE AND THE SEVERITY OF THEDISEASE: The following table shows analysis of different CT scoring.

CT GRADING	NO OF MALES	NO OF FEMALES
A	0	1
B	35	2
C	4	0
D	3	1
E	4	0

From this study, we canconclude that most of the attacks of acute pancreatitis weremilderforms.
InonepatienttheCTabdomenshowednormal pancreas associated with elevated serum amylase and lipase levels. Grades A and B accounted for more than 75% of the patients.

7. SEX DIFFERENCE AND THE SEVERITY OFDISEASE:

From this study, it has been found acute pancreatitis is 11 times more prevalent in males. Most forms of severe pancreatitis occurred in male patients. The severity between the sex groups cannot be assessed correctly as the number of female patients in this study is insufficient.

8. CORRELATION BETWEEN RANSON'S SCORING AND CT SCORING IN SEVERITY OFDISEASE:

CT GRADING	AVERAGE RANSON'S NUMBER
A	1
B	1
C	1
D	2
E	2.5

Both CT grading and Ranson's number are correlating directly with the severity of the disease. From this study we can conclude that there is a positive correlation between the Ranson's scoring and CT scoring in assessing the severity of the disease as shown in the line diagram.

9. DURATION OF THE HOSPITAL STAY AND THE SEVERITY OF THEDISEASE:

CT GRADE	DURATION OF THE HOSPITAL STAY
A	10 days
B	7.7 days
C	10 days
D	6.5 days
E	11.5 days

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The duration of the hospital stay does not correlate with the CT or Ranson's grading as the patients with the higher grade had shorter hospital stay duetothe death of the patients which is because of the high mortality associated with the higher grades.

10. RANSON'S SCORE AND THE MORTALITYRATE:The five patients who died had Ranson's score of 2 or 3. Thus the Ranson's score is surely having an influence on the prognosis of the patient**.**

Ranson's score	No of deaths
0	0
1	0
2	1
3	4

Most of the patients having Ranson's score of 0 and1 have shown better signs of recovery than those with higher scores.

11. MORTALITY IN DIFFERENT CTGRADES:

CT grades	Number of deaths
A	0
B	0
C	0
D	2
E	3

From this study, CT gradings A, B & C show better prognosis when compared to grades D & E. CT grades D & E show bad outcome in terms of mortality. In grade D the mortality rate is 50 % (2 out of 4 patients expired). In grade E the mortality rate is 75 % (3 out of 4 expired).So CT grading system definitely correlates with severity of acute pancreatitis

FOLLOW – UP:

In this study, out of the 50 patients, 5 patients were died. In the remaining 45 patients, only20patientscameforthefollow–up.
Ofthese20patients,6 malepatients had recurrent pancreatitis and one female patient hadrecurrent attack. She developed pseudocyst of the tail of the pancreas for which she underwent distal pancreatectomy. CT abdomen of one male patient who had a recurrent attack showed pseudocyst of pancreas along with areas of necrosis. He underwent cystogastrostomy with necrosectomy through the cystogastrostomy site. Patient improved symptomatically after surgical intervention.

V. CONCLUSION

1. Clinical manifestations like hemodynamic instability, massive ascites and presence of guarding &ridgidity are associated with pooroutcome.
2. Serum amylase has role only in the diagnosis of acute pancreatitis. So the rise of serum amylase levels cannot be used as a parameter to assess the severity of acutepancreatitis.
3. Ranson's scoring has an important role in assessing the severity of acute pancreatitis.
4. Ranson score has better sensitivity and specificity in predicting sever acute pancreatitis.
5. CRP level is the single most reliable index that correlates with severity of acute pancreatitis.
6. CRP levels in double and triple digits almost always predict severe acute pancreatitis.
7. CRP levels can be used as a guide to refer patients with acute pancreatitis to higher center so as to manage severe acute pancreatitis that the patient might develop.

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