"A COMPARATIVE STUDY BETWEEN RANSON CRITERIA AND MODIFIED COMPUTED TOMOGRAPHY SEVERITY INDEX TO DETERMINE ACCURACY IN PREDICTING PROGNOSIS IN A PATIENT DIAGNOSED WITH ACUTE PANCREATITIS, A CROSS SECTIONAL STUDY"

BY

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Dr. SHRISHAIL C. METGUD, MS
Professor and Head,
Department of General Surgery,
J. N. Medical College, Belagavi
Date: 30/12/22
Place: Belagavi

Dr. N. S. MAHANTSHETTI, MD
Principal,
J. N. Medical College,
Belagavi
Date:
Place: Belagavi

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

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iii

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Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 14-12-2022.

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Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BH0120013,
Postgraduate Student,
2020-21 Batch,
Department of General Surgery,
J. N. Medical College, Belagavi.

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Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ ** **Date: 25/01/2021

To,

REG NO: BH0120013
PG student in Surgery,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

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(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

29

ABSTRACT

Background- An acute attack of pancreatitis is a condition characterised by inflammation of the organ itself. The most characteristic symptom is sudden stomach discomfort, and the diagnosis is confirmed by elevated levels of blood amylase and lipase. (1) Acute Pancreatitis can be classified as Mild and Severe disease (3). Inappropriate pancreatic enzyme release and activation cause acute pancreatitis by causing tissue destruction and an inflammatory response. The majority of individuals with necrotizing pancreatitis cannot be identified accurately by any of the specific clinical or laboratory indicators despite their widespread use in clinical practise. In an attempt to overcome these obstacles, many scoring systems have been devised, each of which incorporates clinical and laboratory criteria into its determination of whether a patient has severe pancreatitis. Acute pancreatitis severity is measured according to the prevalence of certain abnormalities that serve as prognostic signals, severe symptoms, risk factors, or objective indicators. (8)

AIMS AND OBJECTIVES

To-compare Ransons scoring system and Modified CT severity-index-scoring and assess which of the two scores is more accurate in predicting prognosis in a patient with acute pancreatitis.

METHODS

Cross-sectional study conducted for a time period of one year from January-december 2021 in patients who were admitted with complaints of severe pain-abdomen, nausea/vomiting at KLE Hospital, Belagavi.

A total of 40 patients were included in the study and were subjected to CT imaging and other investigations.

Duration of pain and vomiting, Duration of stay, Diet-changed on-day, Day-amylase lipase returns to normal, Day of discharge, Other complications were compared. All the above data were gathered and based on the parameters set, the two scores were assessed and compared in order to determine which score is more accurately predicting-prognosis.

RESULTS:

According to MCTSI score, out of the 40 patients: 15 (37.50%) had mild, 11 (27.50%) had moderate and 14 (35.00%) had severe grading. Out of the 14 severe cases, 10 (71.43%) had severe clinical course

According to Ranson's score, out of the 40 patients: 26 (65.00%) had non-severe and 14 (35.00%) had severe grading. Out of the 14 severe cases, 11 (78.57%) had severe clinical course.

Test Result Variable(s)	AUC	Std. Error	95% Confidence Interval		P-value
			Lower Bound	Upper Bound	
MCTSI Score	0.784	0.081	0.626	0.942	0.003
RANSONS Score	0.757	0.085	0.591	0.923	0.008

CONCLUSION:

The study shows that MCTSI score is more accurate than Ransons score in predicting prognosis in acute pancreatitis patients. From our observations; both the scoring systems were able to accurately predict prognosis. MCTSI score was able to predict the chance of occurrence of complications better than Ransons criteria. We thereby conclude that MCTSI score was more accurate than Ransons in predicting prognosis.

LIST OF ABBREVIATIONS

KLES	–	Karnataka Lingayat Education Society
MRC	–	Medical Research Centre
AP	_	Acute Pancreatitis
CP	_	Chronic Pancreatitis
CECT	_	Contrast Enhanced Computed Tomography
USG	_	Ultrasonography
DB	_	Dorsal Bud
VB	_	Ventral Bud
IUL	_	Intra uterine life
CBD	_	Common bile Duct
PD	-	Pancreatic duct
SMA	-	Superior mesenteric artery
PDA	-	Pancreatico-duodenal artery
GDA	-	Gastro duodenal artery
SMV	-	Superior mesenteric Vein
PV	-	Portal vein
VIP	-	Vasoactive intestinal peptide
ER	-	Endoplasmic Reticulum
ERCP	-	Endoscopic retrograde cholangiopancreatography
C.I.	-	Confidence interval
CRP	-	C-reactive Protein
SD	-	Standard deviation
DOHS	-	Days of hospital stay

AUC	-	Area under curve
APACHE	-	Acute physiological assessment and chronic health
CTSI	-	Computed tomography severity index
MCTSI	-	Modified computed tomography severity index
MODS	-	Multi organ dysfunction syndrome
CRP	-	C-Reactive Protein
IAP	-	Intra Abdominal Pressure
LDH	-	Lactate Dehydrogenase
SGOT	-	serum glutamic-oxaloacetic transaminase

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF CHARTS

OBJECTIVES

- To compare Ranson's and MCTSI prognostic score for pancreatitis and assess which is more accurate in predicting prognosis in patients diagnosed with acute pancreatitis.

INTRODUCTION

An acute attack of pancreatitis is a condition characterized by inflammation of the organ itself. The most characteristic symptom is sudden stomach discomfort, and the diagnosis is confirmed by elevated levels of blood amylase and lipase. Mild pancreatic injuries account for 80% of all cases, and these patients often make full recoveries. Patients who are still alive have a very serious condition that has both local and systemic consequences. In 80% of instances with acute pancreatitis, the condition is moderate and resolves without problems, but in up to 20% of cases, the condition is much more serious and results in major morbidity and even fatality.(1)

The death of Alexander the Great is only one of many cases in history that may have been caused by AP. Some 2200 years later, Reginald Huber Fitz presented the first thorough examination of the illness.

Fifty-three individuals were described in detail by Fitz, who distinguished between hemorrhagic, suppurative, and gangrenous presentations of the illness. AP, he theorized, develops when an inflammation of the gastroduodenal junction spreads through the pancreatic duct.

Chiari proposed in 1896 that pancreatic autodigestion—the pancreas "giving in to its inherent digestive properties"—was the fundamental pathophysiological mechanism of the condition. (2)

AP can be classified as 2

a) Mild - self-limiting disease (edematous)

b) Severe - necrosis (MODS and death) (3)

Table 1- Etiological factors of acute pancreatitis (4)(5)
1) biliary tract disease
2) Chronic alcoholism
3) Mumps
4) Hyperthyroidism
5) Carcinoma
6) Steroid usage
7) Hereditary (mutation of PRSS1, SPINK1, and CFTR)
8) Drugs
9) Infection
10) Traumatic

Pathogenesis- Inappropriate pancreatic enzyme release and activation cause AP by causing tissue destruction and inflammatory response.There are three major initiating events-

A) Pancreatic duct obstruction

B) Primary acinar cell injury

C) Defective intracellular transport of proenzymes within acinar cells.

There are three stages of illness progression that may be identified in AP. When the pancreatic acini are damaged or disrupted, pancreatic enzymes seep into the surrounding tissue, causing the first phase. Pancreatic oedema, vascular injury, bleeding, and necrosis are all brought on by the pancreatic enzyme trypsinogen leaking out of the organ and being activated into trypsin. Inflammation inside the pancreas and outside it define AP's second and third stages, respectively. (5)

When a patient presents with sudden-onset, back-radiating epigastric pain accompanied by nausea and vomiting, it is imperative to rule out a wide variety of potentially fatal conditions, including those involving the cardiovascular (myocardial infarction), gastrointestinal (peptic ulcer disease with perforation or bleeding), and hepatic (acute pancreatitis) systems. Clinical history and physical examination results are supported by appropriate investigations to narrow the differential diagnosis and direct the management and treatment of an illness and its potential consequences.(6)

Abdominal discomfort and, often, indications of peritonitis in the upper abdomen are clinical indicators of acute pancreatitis. Due to pancreatic fluid and bleeding into the retroperitoneum, bruising-like redness may form around the umbilicus (Cullen's sign) or in the flanks (Grey Turner's sign). Tetany as a consequence of hypocalcemia, is another uncommon symptom. Patients with acute pancreatitis often present with a variety of abnormal biochemical parameters, including hyperglycemia, hypoalbuminemia, high blood urea nitrogen, and raised creatinine levels, in addition to hemoconcentration. (7)

Prior to doing the laboratory tests, the diagnosis of acute pancreatitis is initially made based on the patient's medical history and physical examination findings. It used to be recognized that the presence of acute pancreatitis was indicated by a blood amylase level that was three or four times greater than the upper limit of normal. The biliary aetiology may be identified if the alanine aminotransferase level is more than 150 IU/L during an episode of acute pancreatitis.(6)

It has long been known that individuals with acute pancreatitis frequently have glaring changes in clinical indicators and some unexpected findings from standard laboratory tests. For instance, a low blood calcium level (7.5 mg/dL) during an acute pancreatitis episode is a concerning indicator that is often seen in patients who are

very unwell. Serum glucose levels over 250 mg/dL and post-rehydration serum creatinine levels above 2 mg/dL have also been linked to an increased mortality risk. Patients with a serious, possibly fatal type of disease can be identified by signs of multiple organ failure as well as some specific aberrant clinical and laboratory results.

Severe pancreatitis has long been known to cause significant changes in clinical indicators and certain aberrant findings in regular laboratory testing. For instance, a low blood calcium level (7.5 mg/dL) seen in the background in cases of acute pancreatitis is a concerning sign observed predominantly in individuals with severe disease. The risk of patient death has also been connected to blood glucose levels above 250 mg/dL and serum creatinine levels after rehydration exceeding 2 mg/dL. Patients with a severe, potentially fatal disease may be recognized by the presence of multiorgan failure symptoms as well as specific abnormal clinical and laboratory findings. The majority of individuals with necrotizing pancreatitis cannot be identified accurately by any of the specific clinical or laboratory indicators despite their widespread use in clinical practice. In an attempt to overcome these obstacles, many scoring systems have been devised, each of which incorporates clinical and laboratory criteria into its determination of whether a patient has severe pancreatitis. Acute pancreatitis severity is measured according to the prevalence of certain abnormalities that serve as prognostic signals, severe symptoms, risk factors, or objective indicators. (8)

Table 2- Some useful prognostic factors used in different scoring system
1) blood glucose
2) Glucagon
3) Serum amylase and lipase
4) Granulocyte elastase
5) Il6 , crp
6) Age
7) Serum tnf alpha
8) Raised IAP
9) Serum amyloid a
10) Procalcitonin
11) Comorbidities. (9)

Multi-factor scales have been used for Acute Pancreatitis since the 1970s; examples are the Ranson and APACHE II scores. In order to quantify the severity of CT scans, the Balthazar CTSI has been developed since 1990. Computed tomography (CT) with intravenous contrast medium injection is the technique of choice for assessing for pancreatic necrosis or determining the extent of acute fluid collections in the pancreas and extra pancreas. It has been determined that these two variables serve as prognostic markers of Acute Pancreatitis severity. In order to increase prognostic accuracy, a modified CTSI was created. It is based on a combined assessment of peripancreatic fluid collections, and the degree of pancreatic necrosis was developed to improve prognostic accuracy. (10)

Usage of ultrasonography in Acute Pancreatitis is limited by abdominal fat and ileus (increased intestinal gas). But USG is the research of choice investigation for pancreatitis induced by gallstones. The investigation of choice for Acute Pancreatitis is CECT of the abdomen and it is the gold standard.(11)

Acute pancreatitis is characterized by fluid collections, peripancreatic fascial thickening, peripancreatic fat stranding, and focal or widespread pancreatic hypertrophy. Necrosis of the pancreas may be localized or widespread, and it manifests as non-enhancing regions of pancreatic parenchyma. When it comes to identifying pancreatic necrosis, CT offers a 100% sensitivity rate and an 87% specificity rate(12)

Over the last two decades, many radiologic prognostic scoring systems have been developed. Balthazar et al. 1990.'s CT severity index (CTSI) has seen the greatest usage in both clinical and research contexts. The CTSI is a numerical scoring system that accounts for both the degree of extra pancreatic inflammation and the amount of pancreatic necrosis. A revised version of the CTSI (MCTSI) was developed in 2004 to address a variety of concerns about the original. The MCTSI is superior to the CTSI because it standardizes the evaluation of pancreatic parenchymal necrosis and peri-pancreatic inflammation and includes extra pancreatic effects. (3)

No comparison has been performed between both radiological, that is modified ct severity index and clinical prognostic scoring system, Ranson criteria. In order to better understand the relative efficacy of the MCTSI and the Ranson criteria in assessing clinical severity in a cohort of acute pancreatitis patients who were being routinely monitored, we conducted this research.

REVIEW OF LITERATURE

The exocrine and endocrine tissue that make up the pancreas may be distinguished based on their morphology, and the organ itself is derived from the endoderm.(13)

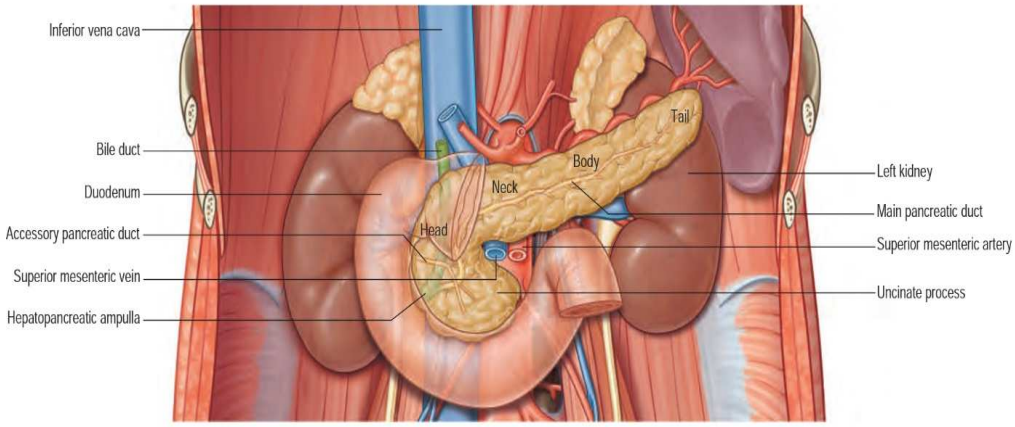


Fig-1 Parts of pancreas

Anatomy(14)

One of the biggest digestive glands is the pancreas. The gland's exocrine portion, which secretes enzymes for lipid, carbohydrate, and protein digestion, makes up the majority of the structure. Cell clusters located throughout the gland's substance produce hormones that regulate glucose homeostasis and the motility and function of the upper gastrointestinal tract.

Lobulated surfaces, a firm to soft texture, and a rosy cream colour characterise a healthy pancreas. The anatomical divisions used to characterise it are the head, neck, body, tail, and uncinate process.

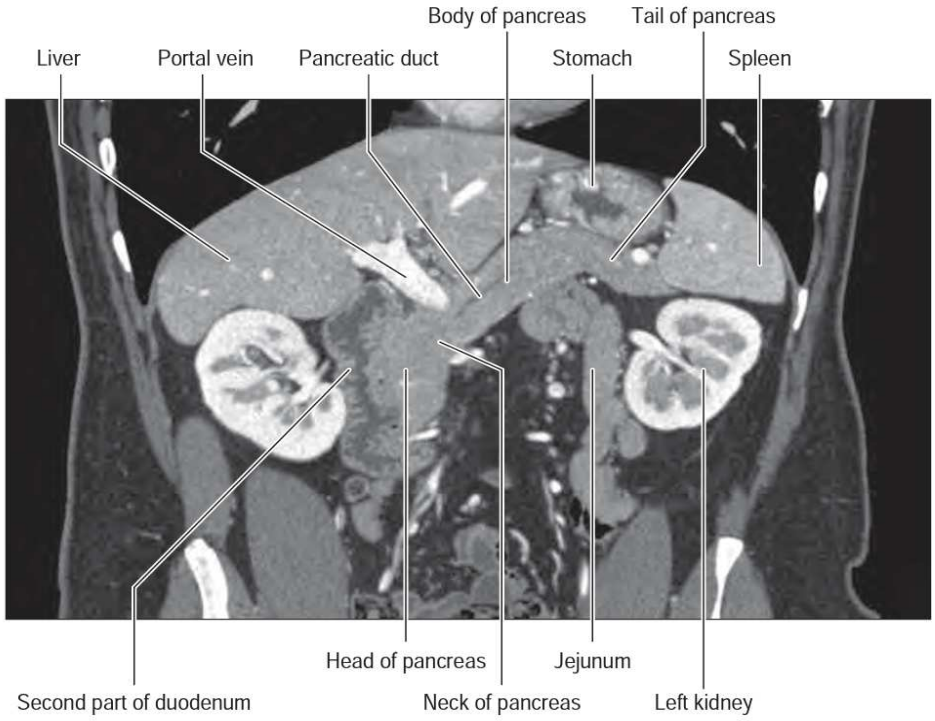


Figure 2- coronal-reversed computed tomogram, the pancreas can be seen in context with other organs in the upper abdomen.

The gland, which resembles a flattened "tongue" of tissue resting in the retroperitoneum, is 12–15 cm long in adults. It has a medial end (head) that is thicker and lateral ends that are thinner (tail). The remaining portion of the gland moves transversely and somewhat cranially the whole time as it goes posteriorly through the stomach, then into the retroperitoneum, and eventually into the hilum of the spleen.The gland's head may be found in the duodenum's 'C' loop. It rests most anteriorly on the neck and medial body, forming a characteristic shallow curve that "drapes" over the spinal column and other retroperitoneal structures.

Its volume in adults ranges from 40 to 170 cm3, but on average it is between 70 and 80 cm3. Males typically have a larger capacity than females. Age-related increases in pancreatic volume peak in the fourth decade. Atrophic changes occur in

the gland beyond age 60, when adipose connective tissue takes the role of exocrine tissue.

The origin of the transverse mesocolon passes through the parietal peritoneum, which is located on the ventral side of the pancreas. The pancreatic head is supplied by the fusion fascia of Treitz, while the pancreatic body and tail are supplied by the fusion fascia of Toldt.

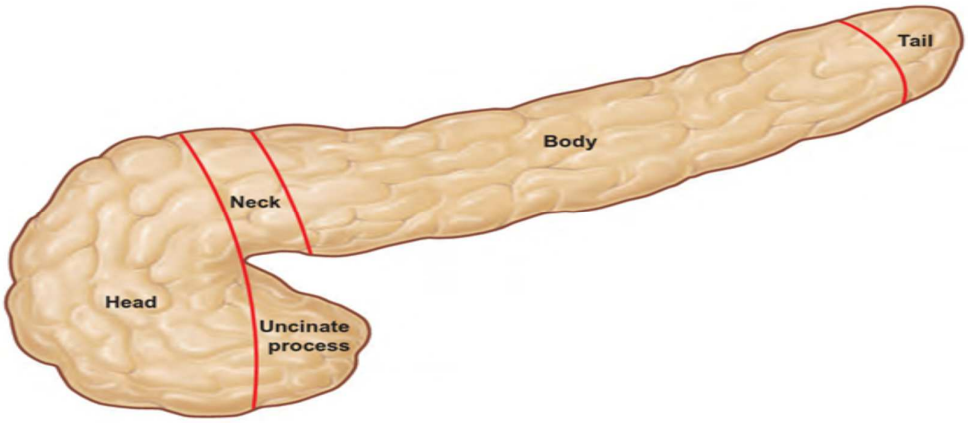


Figure-3 Divisions of pancreas

HEAD

Within the duodenal notch, the pancreatic head is situated anterior to the spinal column and to the right of the midline. Despite being the pancreas' biggest and thickest region, it is flattened in the anteroposterior direction.The initial portion of the duodenum extends superiorly from its location on a short mesentery at the pylorus. Anteriorly, this portion of the body is covered. The duodenal border of the head is flattened, somewhat concave, and adherent to the second half of the duodenum, particularly around the duodenal papillae. The superior and inferior pancreaticoduodenal arteries surround this area.

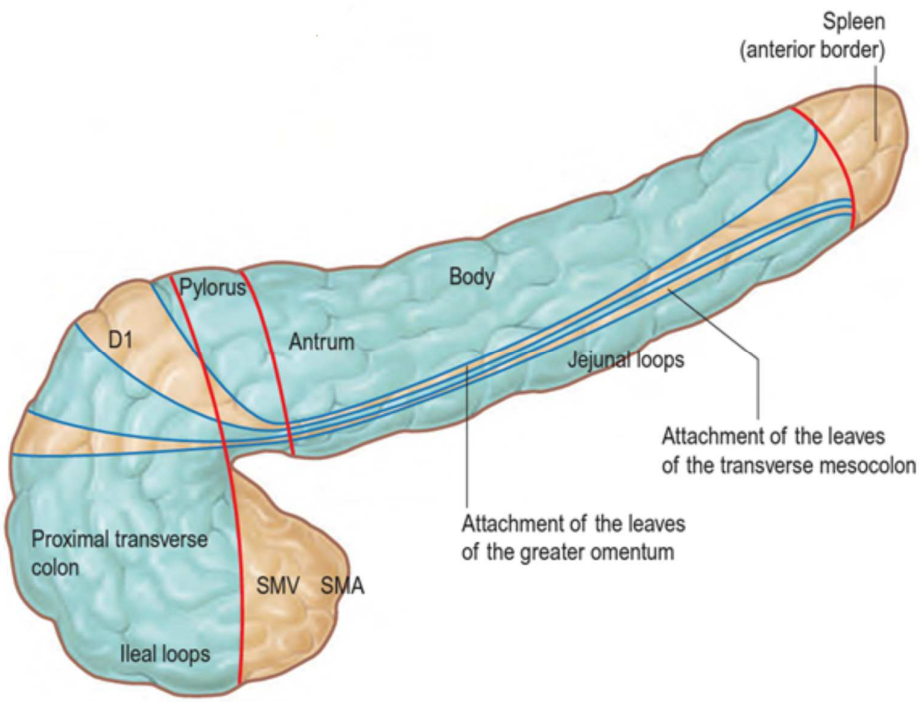


Figure 4- Relations of pancreas anteriorly

The uncinate process and the inferior border of the pancreatic head unite as one continuous structure above the third portion of the duodenum. The front of the skull, which is coated with periosteum, serves as the origin of the transverse mesocolon. The main duodenal papilla is situated close to the location where the common bile duct and pancreatic duct converge. The inferior vena cava, the right crus of the diaphragm, and the terminus of the right gonadal vein are all connected by a network of veins and arteries that runs from the posterior side of the pancreatic head. Abutting the midline, the pancreatic neck and head become one structure.

NECK

The pancreatic duct is about 2 cm wide at its neck, which connects the organ to the rest of the body. Before the portal vein developed, this is the pancreas' most anterior region (at the point where the superior mesenteric vein fuses with the splenic vein in the transpyloric plane). Since tumour development into these arteries may

make excision of pancreatic cancer difficult, this relationship is essential for evaluating the disease. The portal vein and superior mesenteric vein both pass through the back of the neck. The inferior mesenteric vein is located where the superior mesenteric vein and the splenic vein meet in about one-third of the population. The peritoneum covers the front of the pancreatic neck close to the pylorus. At the intersection of the head and neck, the gastroduodenal artery's anterior superior pancreaticoduodenal branch descends in front of the gland.

BODY

The pancreas' body, which extends from the gland's neck to its tail and gradually thins as it goes, is its longest portion. With front and posterior surfaces as well as superior and inferior borders, it has a somewhat triangular cross-section.

While the posterior layer and the transverse mesocolon are continuous with the anterior surface, the anterior surface of the peritoneum covers the larger omentum and is reflected anteriorly and inferiorly from the surface of the gland.There is a separation between the transverse mesocolon's two layers along this surface. This tiny sac separates the front aspect of the pancreas from the stomach just before where the transverse mesocolon connects. The left renal vein, the fourth segment of the duodenum, the duodenojejunal flexure, and the coils of the jejunum are all attached to it at its anterior end. It is positioned posteriorly in the infracolic compartment.

The peritoneum is absent from the rear of the pancreatic. The toldt fascia connects the left renal vein, the upper pole of the left kidney (which is encircled by perirenal fascia), the left suprarenal gland, and the left crus of the diaphragm. The splenic vein creates a series of varyingly deep indentations in the parenchyma as it passes from left to right along this surface of the gland.

Superior border- On the right, the upper pancreatic border is rounded, whereas on the left, it tapers and sharpens. Omental tuberosities often develop on the right side of the body, rising from the end of the superior border and crossing over the level of the lower curve of the stomach. The splenic artery and the common hepatic artery both pass through the top border of the liver, the latter to the left and the former to the right of the actual gland. The celiac artery has branches that may be seen towards the top border.

Inferior border- Superior mesenteric arteries protrude from behind the pancreas towards its neck at the medial end of the inferior border. The inferior mesenteric vein turns into the splenic vein at the intersection of the superior and inferior mesenteric veins, which is situated laterally. When performing left-sided colonic resections and using computed tomographic (CT) imaging, this location is helpful for locating the inferior mesenteric vein.

TAIL

The pancreas has a medial link to the rest of the organ and is thinnest and most lateral in the tail. The splenorenal ligament ranges in length from children to adults, measuring around 1.5 cm in children and 3.5 cm in adults. It may terminate at the base of the splenorenal ligament or extend up to the splenic hilum after ligation or stapling of the splenic arteries, leaving it vulnerable to damage during splenectomy. The splenic vein and its branches, as well as the splenic artery and its offspring, are located behind the tail.

UNCINATE PROCESS

The inferomedial portion of the gland's head is continued in a hook-like fashion by the uncinate process. In terms of embryology, it differs from the rest of the gland. Before moving forward into the small intestine's root, the superior mesenteric

vein and, in rare cases, the superior mesenteric artery descend on the mesentery's front surface. The third portion of the duodenum, which is medially anterior to the abdominal aorta and above the uncinate process, is vulnerable to compression by a pancreatic tumour. The left renal vein, uncinate process, and third half of the duodenum may all be visible on sagittal cross-sectional imaging, respectively, between the superior mesenteric artery and the abdominal aorta.

Pancreatic ducts

Drainage of the exocrine pancreatic tissue is received by a large number of small lobular ducts. The single main and auxiliary duct layout is the most common arrangement for the principal ducts that drain the pancreas.The dorsal and ventral pancreatic ducts' embryological development is depicted by this configuration.

Normally, the primary pancreatic duct (of Wirsung) travels from left to right within the gland's contents. It is situated halfway between the pancreas' superior and inferior borders, typically a little more toward the gland's posterior surface. The "herringbone pattern" is formed when the gland's lobular (secondary) ducts connect at its tail, and when additional lobular ducts are produced, they almost form a right angle to the gland's axis.

The duct in adults can frequently be seen on ultrasonography; it has a head diameter of about 3 mm, a body diameter of about 2 mm, and a tail diameter of about 1 mm. The quality of the duct improves beginning in the 50s. As it nears the gland's neck, it curves to the right, toward the bile duct. Upon joining, the two ducts widen into a common channel that pierces the duodenal wall at an oblique angle. Hepatopancreatic ampulla is a possible growth in this channel (of Vater). A few mucosal folds in the main pancreatic duct's terminal portion prevent pancreatic juice

reflux. The common pancreaticobiliary channel can range in length from 5-7 mm in healthy persons.

The anterior part of the pancreatic head drains through the auxiliary (dorsal) pancreatic duct (of Santorini). It is made up of several smaller lobular ducts located deep inside the brain, and it is much smaller than the main duct. It often connects to the main pancreatic duct around the neck of the gland or the first inferior branch. The minor duodenal papilla, found about 2 cm proximal to the main papilla, is usually the site where the auxiliary duct emerges. If the duodenal end of the accessory duct fails to form, the lobular ducts leak into the main duct through one or more tiny channels. Abnormalities in the formation and fusion of the dorsal and ventral pancreatic ducts might cause variations in the architecture of the main and auxiliary ducts.

Embryology (7,13)

Development begins at the 4th week of gestation.

Pancreas is endodermal in origin. Primitive endoderm gives rise to dorsal bud (DB) and ventral bud (VB). DB appears first.

1. DB blossoms into – Body, tail, neck, head of the pancreas.
2. VB is a part of hepatic diverticulum. Biliary tree formation and communication is maintained by the VB.

Around 4th to 8th week of the intrauterine life (IUL) VB rotates and fuses with the DB posteriorly in the clockwise fashion. At the completion of the 8th week of the intrauterine life both the dorsal and the ventral buds are fused together. The expansion of the DB duct as it leaves the duodenum creates the duct of Santorini, commonly referred to as the minor pancreatic duct (PD). The Duct of wirsung, also known as the CBD, is formed when the small venous PD that originates in the hepatic diverticulum joins the CBD.

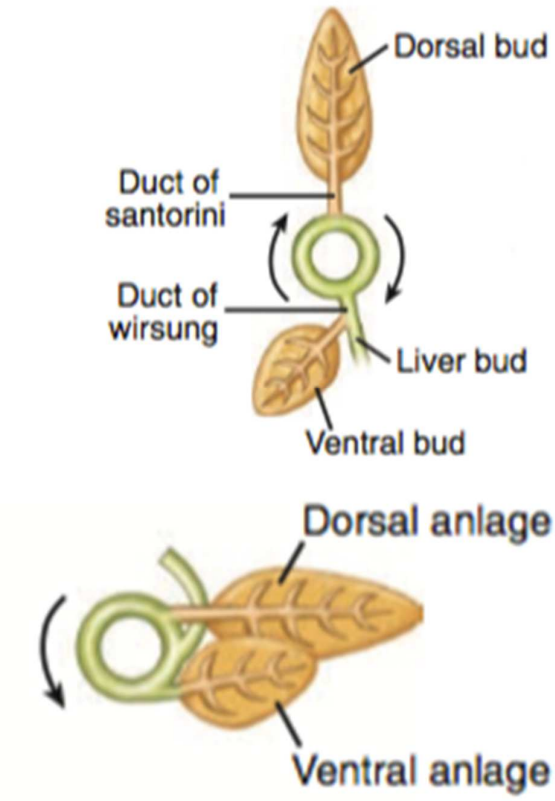


Figure 5- Embryology of Pancreas

VB + CBD - Major PD. The preponderance of the pancreas drains through the main PD. Drains the body and tail with accessory PD. Near the centre of the second part of the duodenum, the major papilla, also known as the ampulla of vater, receives both the CBD and the main pancreatic duct's discharge. The bile juice reaction is prevented by the pancreatic duct's twofold the pressure of the CBD. A collection of muscles called the sphincter of Oddi controls how much bile and pancreatic juice enters the duodenum.

Accessory PD channels entering the second section of the duodenum, two centimetres from the vater's ampulla.

Blood supply of pancreas

Arterial supply-

The superior mesenteric artery and its branches, as well as the celiac trunk, provide the pancreas with a substantial amount of circulatory supplies.

1. Anterosuperior PDA (pancreaticoduodenal artery) - bestows blood to superior surface of the head, a divaricate of gastroduodenal artery (GDA)
2. Anteroinferior PDA - a divaricate of SMA, supplies anteroinferior surface.
3. Posterosuperior PDA - bestows superior posterior surface, a divaricate of GDA
4. Posteroinferior PDA - supplies inferior posterior surface, a branch of SMA
5. Splenic artery - Supplies body and tail
6. Superior PDA + inferior PDA - joins to complete the arcade.

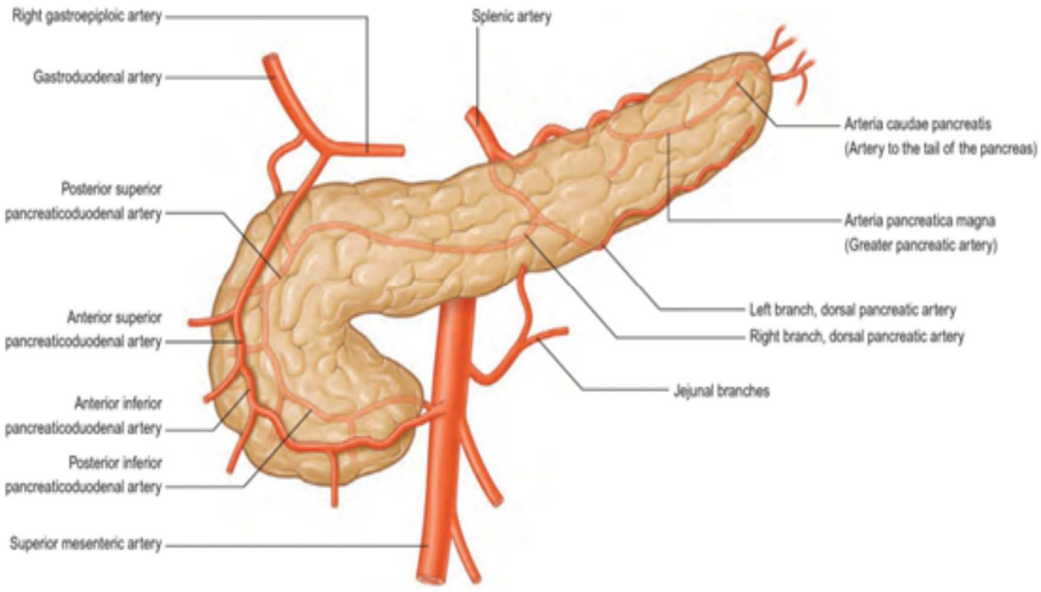


Figure 6- Blood Supply of Pancreas

Venous drainage

The splenic vein and inferior mesenteric vein, which exit from the pancreas' posterior surface, body, and tail, respectively, provide blood to the PV.

1. Antero-superior PD vein which further drains into PV
2. Antero-inferior PD vein + right gastroepiploic vein+ middle colic vein which further drains into the SMV.

The splenic vein and inferior mesenteric vein, which drain from the body, tail, and posterior surface of the pancreas, provide blood to the PV.

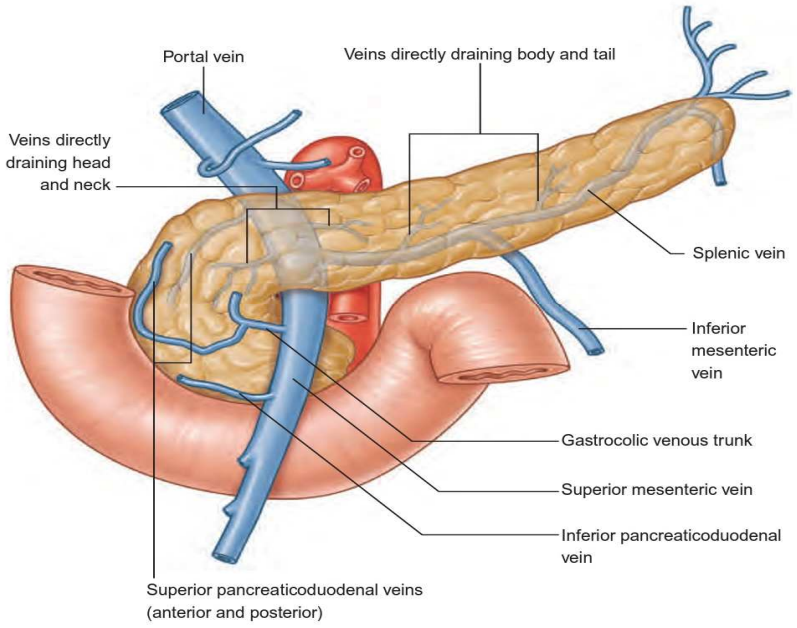


Figure 7- Venous drainage of pancreas

Lymphatic drainage(15)

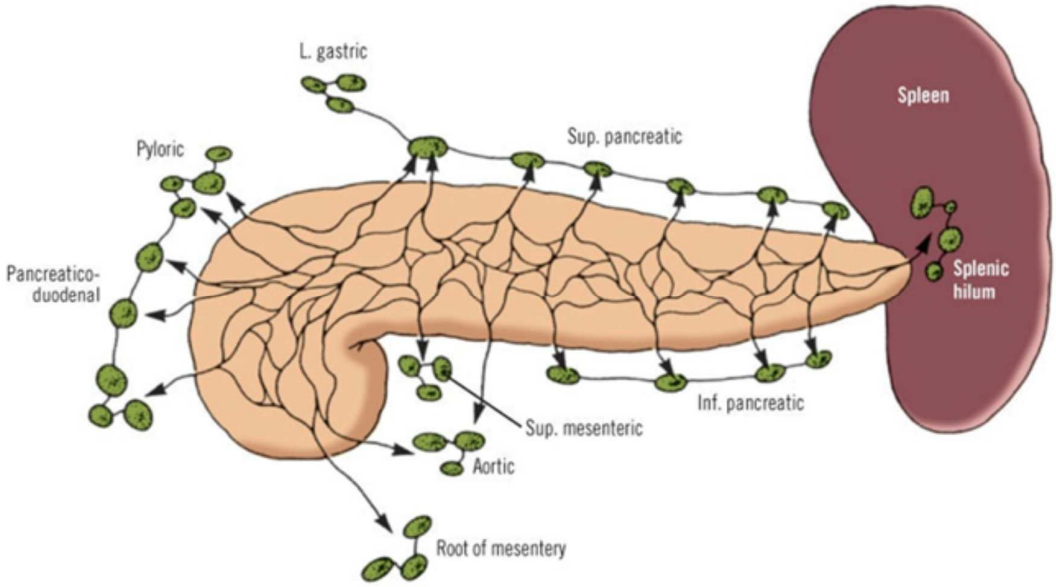


Figure 8- Lymphatic drainage

The nodes in the vicinity receive lymph in a centrifugal fashion.

The pancreas' lymphatic vessels emerge from a dense interanastomosing network around the pancreatic lobes.

These lymphatics drain into five groups of lymph nodes: superior nodes, inferior nodes, anterior nodes, posterior nodes, and splenic nodes.

Neuroanatomy (7)

- Sympathetic & parasympathetic nervous system.
- Sympathetic - inhibits the secretions
- Parasympathetic - stimulates the secretions.

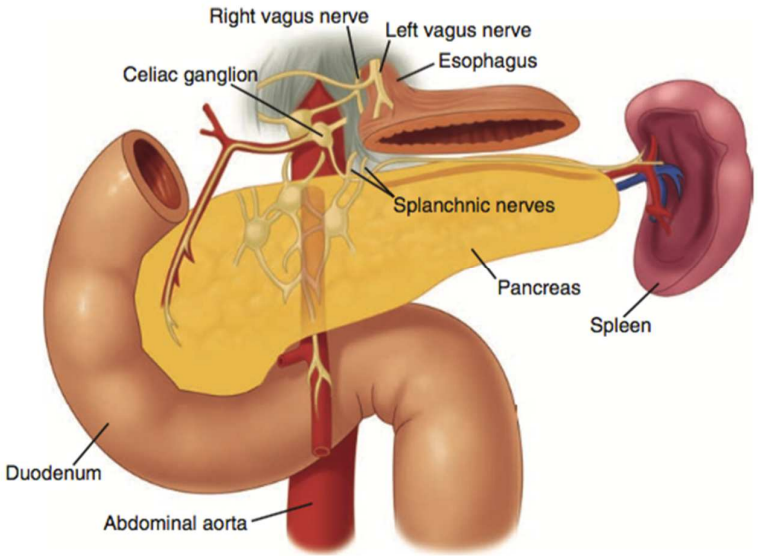


Figure 9- Pancreas neuroanatomy

Physiology of pancreas

Exocrine function (7,16)

- Colourless and odourless fluid secretion.
- 1500 to 2500 ml daily.
- Alkaline pH -8.0.
- Secreted by centriacinar and ductal cells.
- Pancreatic enzymes are influenced by parasympathetic supply.

Pancreatic secretion has two components (7,16)

1. Water and the electrolyte secretion –
    a. The main ion is the bicarbonate.
    b. Synchronised by secretin - hormone which is liberated from duodenal mucosa.

c. The stream of Pancreatic juice - stimulated by secretin.

d. Rich in bicarbonate

e. Vasoactive intestinal peptide (VIP) also synchronises water and bicarbonate secretion in the pancreas.

f. VIP controls cyclic AMP which in turn stimulates bicarbonate secretion.

g. Inhibited by - Glucagon, somatostatin and antidiuretic hormone.

2. Secretion of enzymes- Digestive enzymes synthesized in polysomes. Endoplasmic reticulum (ER) binds digestive enzymes. Ribosomes form the secretory proteins. These proteins are transferred to rough ER. These enzymes constitute the active and inactive forms. Inactive forms are chymotrypsinogen A and B, carboxypeptidase A and B, trypsin. Active forms are amylase, lipase, deoxyribonuclease and ribonuclease. These enzymes are activated in the duodenum. Trypsinogen is converted to trypsin by enterokinase. Trypsin in turn activates carboxypeptidase and chymotrypsinogen. Inside intestine proteins break down into peptides and amino acids by pancreatic proteolytic enzymes, fat is broken into glycerol and fatty acids by lipase, starch is degraded to disaccharides by amylase. (7,16)

Stimulants of pancreatic secretion.

a. Pancreozymin,

b. Cholecystokinin,

c. Gastrin

d. Vagus nerves

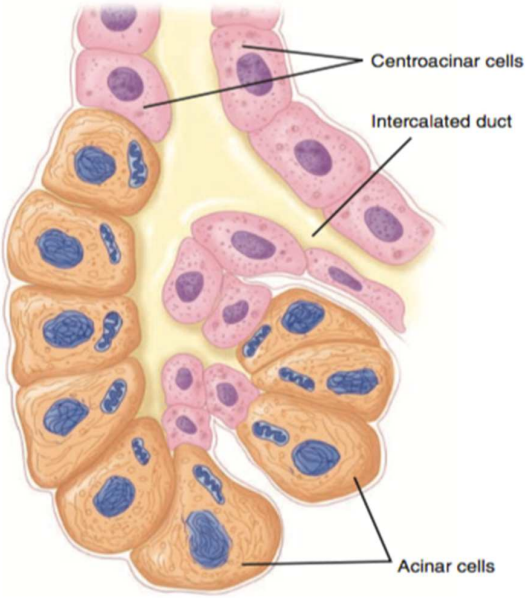


Fig 10. Histology of Pancreas

Exocrine secretion of the pancreas(7,16)

It has 4 phases

1. Cephalic phase - smell and sight of food initiates this stage by vagal stimulation resulting in secretion of enzyme rich juice in low volume.
2. Gastric phase - when meal reaches the stomach and causes gastric distension causes release of gastrin, which in turn stimulates the enzyme secretion by pancreas.
3. Intestinal phase - Synchronises secretin which controls pancreatic juice and bicarbonate secretion.
4. Postcibal phase - inhibition of pancreatic secretion.

Endocrine secretion of pancreas (7,16)

Endocrine secretions of pancreas are mainly derived from islet cells of langerhans. There are about 1 million islet cells approximately.

Islet cells consists of

1. Alpha cells (20%) - constitute the outermost layer and secretes glucagon.
2. Beta cells (75%) - constitute the central layer and are the source of insulin.
3. Delta cells (5%) - constitute the intermediate layer – produces somatostatin and gastrin.
4. Pancreatic Polypeptide cells
    - Small in number
    - Scattered all over the pancreas.
    - They are responsible for secretion of VIP, pancreatic and gastrointestinal inhibitor polypeptide

Acute Pancreatitis

Most cases of acute pancreatitis, a pancreatic inflammatory condition, are self-limiting and improve without serious consequences. However, it might result in issues and significant mortality in up to 20% of patients. The globe has been studying its causes and methods of spread for centuries.(17)

The diagnosis of acute pancreatitis requires two of the following factors, according to the 2012 revision of the Atlanta classification.

- Constant abdominal discomfort with acute pancreatitis (rapid onset of severe pain in the epigastric region that may spread to the back);
- serum lipase activity (or amylase activity) that is at least three times over the upper limit of normal; and

- Contrast-enhanced computed tomography (CECT) and, less often, magnetic resonance imaging (MRI) or transabdominal ultrasonography features typical of acute pancreatitis. (18)(19)

According to research by Claude Bernard et al., acute pancreatitis may be brought on by bile backing up into the pancreatic duct. Eugene Opie claimed that gallstone migration into the common bile duct is the main cause of acute pancreatitis in 1901[4], which sparked more study and conflicting ideas. Although there were formerly believed to be just one cause of pancreatitis, several have now been discovered. The exact aetiology of acute pancreatitis is yet unknown. The causes of acute pancreatitis have been the subject of several theories.(17)

Pathogenesis (20)

Inappropriate pancreatic enzyme release and activation cause acute pancreatitis by causing tissue destruction in the pancreas and an inflammatory response.(21) Trypsin, like the other pancreatic enzymes, is produced in the pancreas as a proenzyme. Prophospholipase and proelastase are two other proenzymes that may be released as a result of intrapancreatic trypsin activation and can subsequently break down fat cells and harm blood vessel elastic fibers, respectively. Inflammation and small-vessel thromboses, which may clog and burst already weakened arteries, harm acinar cells and increase the intrapancreatic activation of digesting enzymes. Misuse of pancreatic enzymes may result from three primary triggers.-

1. The most frequent causes of pancreatic duct obstruction are gallstones and biliary sludge, but other factors, such as periampullary neoplasms (such as pancreatic cancer), choledochoceles (a congenital cystic dilatation of the common bile duct), parasites (particularly Ascaris lumbricoides and Clonorchis sinensis organisms), and pancreas divisum, may increase intrapancreatic ductal pressure, which is

brought on by obstruction, leads to the accumulation of fluid rich in enzymes in the interstitium. In contrast to other pancreatic enzymes, which are secreted as inert zymogens, lipase is created in an active form and has the capacity to cause local fat necrosis. It is thought that when adipocytes die, "danger" signals are released, stimulating periacinar myofibroblasts and leukocytes to secrete pro-inflammatory cytokines and other inflammatory mediators. This, in turn, causes local inflammation and promotes the growth of interstitial edoema through a leaky microvasculature. Edema may result in vascular insufficiency and ischemia damage to acinar cells.

2. Damage to the primary acinar cells causes the release of digestive enzymes, inflammation, and the pancreas to begin digesting itself. Several different types of endogenous, exogenous, and iatrogenic insults may cause damage to acinar cells. Chemokines that attract mononuclear cells are produced when acinar cells undergo oxidative stress, which may create free radicals and cause membrane lipid oxidation and the activation of transcription factors including AP1 and NF-B. Abnormal activation of digestive enzymes seems to be triggered by an increase in calcium flow. The activation of trypsin is mostly controlled by calcium. Trypsin has a tendency to cleave and inactivate itself when calcium levels are low, but when calcium levels are high, autoinhibition is abolished, and activation of trypsinogen by trypsin is encouraged.
3. The inability of acinar cells to properly transport proenzymes intracellularly. Normal acinar cells have distinct mechanisms for transporting both digestive enzymes and lysosomal hydrolases. Pancreatic proenzymes are misdirected to the intracellular compartment harboring lysosomal hydrolases in animal models of

acinar damage. Afterward, the lysosomes are disturbed and the activated enzymes are released once the proenzymes have been activated.

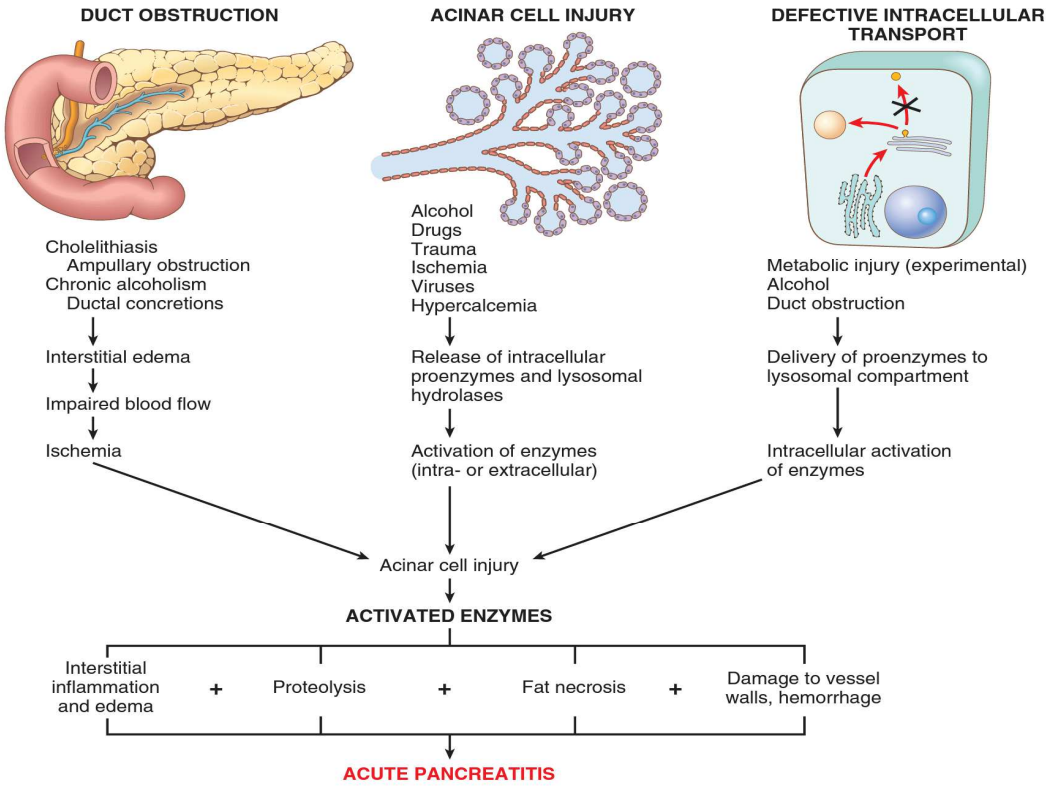


Figure 11- pathogenesis of pancreatitis

Table 3 -Causes of acute pancreatitis
ALCOHOL- Alcohol exposure--Pancreatic secretion inhibited, microtubular dysfunction and thereby oxidative stress leading to intracellular disturbance and necrosis (22)
Biliary pancreatitis (23)
Hyperlipidemia.(24)
Hereditary/genetic- Genes associated - CFTR,SPINK1,Cationic trypsinogen gene PRSS1,Celiac disease (25)
Hypercalcemia- high calcium – accelerated conversion of trypsinogen totrypsin- autodigestion, accumulation of calcium – ductal obstruction and calculi (26)
Traumatic- Traumatic abdominal injuries caused by blunt force or penetration, Manometry of the sphincter of oddi, post-ERCP pancreatitis(27), ERCP sphincterotomy, Iatrogenic complication during surgery
Pancreatic duct obstruction
Infectious- Viral hepatitis (A,B,C,D & E), Coxsackie virus and echovirus, Hemorrhagic fever virus, Cytomegalo virus, Legionella, Leptospira, Salmonella(28)
Venom
Drugs- Hydrocodone, Furosemide, Hydrochlorthiazide, Triamterene, Prednisone, Acetaminophen, Propoxyphene, Metformin, Estradiol, Methylprednisolone (29)
Idiopathic

Diagnosis

Patients presenting with epigastric discomfort, nausea, vomiting, and abdominal pain that radiates to the back should have acute pancreatitis on the differential diagnosis. Long-lasting discomfort, on the order of several days.

The results of a clinical examination are condition-specific in the case of acute pancreatitis. Abdominal discomfort on palpation and the lack of bowel noises may be signs of more severe illness in some people, whereas in others, even minimal disease may not be palpable.

Cullens and turners signs are associated mostly with hemorrhagic pancreatitis.

Patients with moderate pancreatitis may disclose nothing on a physical examination, whereas those with severe illness may be hypotensive and comatose. The patient usually performance levels of agitation, such as a fast heart rate and shallow breathing. Cyanosis may occur under severe circumstances. The belly may be considerably bloated, and the epigastrium may be noticeably enlarged. Upper abdominal tenderness is common, however it may be felt anywhere in the belly. Muscle stiffness is uncommon although intermediate spasms are common. In roughly 1 percent of people, there is a discoloration of the flank that is described as grey or green.

In most instances of nonfatal acute pancreatitis, a diagnosis is not made at all, therefore we have no idea how frequently this occurs. However, in 30% to 40% of individuals with fatal acute pancreatitis, the diagnosis is missed until postmortem. There are two situations in which a correct diagnosis may be difficult. When stomach pain and vomiting occur soon after an operation on the upper abdomen, it's easy to

chalk it up to the anesthesia and forget about the likelihood of pancreatitis. Another solution exists for those who aren't experiencing severe stomach distress. Cardiopulmonary failure, mental disorientation, and/or hypothermia are all possible symptoms. When sudden collapse occurs after surgery or in other situations, it is important to evaluate biochemical and imaging investigations for potential pancreatitis. (30)

Laboratory tests

Biochemical testing for acute pancreatitis involves looking for pancreatic enzymes like lipase and amylase that are secreted by inflamed pancreatic tissue.(31) According to the Atlanta criteria, pancreatitis may be diagnosed if the patient's serum amylase and/or lipase levels are three times the upper normal range.

Newer tests like CRP, procalcitonin and LDH are being studied for their accuracy in diagnosing acute pancreatitis. (30)

Multiple Prognostic Indexes

Patients suffering from acute pancreatitis have been shown to have a number of anomalies in their clinical parameters and normal laboratory testing. A low blood calcium level (7.5 mg/dL) detected in the background in cases of acute onset pancreatitis is an example of a concerning sign seen predominantly in patients with severe disease. Blood creatinine status after rehydration over 2 mg/dL and serum glucose levels above 250 mg/dL have both been connected to a higher risk of patient death. Patients with a severe, possibly fatal type of illness may be identified by the presence of indicators of multiorgan failure and certain aberrant clinical and laboratory results. Studies have shown that the death rate rises to 50% when distant

organ failure symptoms are present. The majority of patients with necrotizing pancreatitis cannot be reliably identified by any of the clinical or laboratory signs, despite the fact that they are all useful in clinical practice.

In order to better identify individuals with severe pancreatitis, many scoring systems have been developed that include both clinical and laboratory criteria. Acute pancreatitis is graded using a grading system that takes into account a variety of abnormalities (prognostic signals, grave indications, risk factors, and objective indicators). Not only do these modifications in physiology not correlate well with the degree and breadth of local illness, but they also lack diagnostic specificity since they may be detected in a wide range of other disorders as well.

The Ranson et al. system from 1974 is still often used when addressing numerical systems (henceforth, the Ranson system or Ranson signs). Eleven objective signals (five established at the outset and six after 48 hours) form the basis of this system. To a similar extent, death and morbidity rates rise in tandem with the prevalence of risk factors. There is no mortality in patients with fewer than three positive symptoms, while the mortality rate is above 50% in those with six or more indicators. When more than six severe symptoms are present, necrotizing pancreatitis is almost always the diagnosis. The system's value lies mostly at the extremes of the severity spectrum, when it serves as an objective indication of sickness. When there are two or less severe symptoms, the pancreatitis is moderate, and when there are more than six, the pancreatitis is severe. Patients with three to five grave symptoms, which is frequent, lack adequate association with disease severity or progression of necrosis. The approach also requires the gathering of 11 measurements, which totals 48 hours of observation time for a trustworthy evaluation.

Table 4- Ranson's criteria
At admission or diagnosis
1)age >55
2) White blood cell count > 16000/ml
3)Blood glucose level >200 mg/dl
4)Above-average levels of lactate dehydrogenase in the blood (>350 IU/L)
5) SGOT >250
During initial 48 hours
1) Significant blood loss (hematocrit decline >10%)
2) Elevated Urea Nitrogen Levels in the Blood (>5 mg/dl)
3) Calcium levels in the blood below 8 mg/dl
4) PaO2 in the arteria >60 mmHg
5) Lack of Bases, Detectable at >4 mEq/L
6) Forecasted Fluid Suppression more than 6000 ml (32)(33)

A score of 3 or less implies a mild disease whereas more than three implies a severe disease.

Since the development of the Ranson system, a number of other grading systems have been developed, each using a unique set of characteristics but sharing a comparable capacity to predict the future. The Glasgow original or modified method, the Simplified Acute Physiology (SAP) score, and simplified prognostic criteria have all been used.

IMAGING EVALUATION

Traditional abdominal radiography and barium tests may help with the diagnosis of pancreatitis and the identification of late complications (abscesses, strictures, and fistulas), but they are ineffective for determining the severity of the condition in the first stages. However, abnormal chest x-rays may be useful for predicting severity, either on their own or in combination with renal function tests (plasma creatinine level). 15 percent to 55 percent of those with acute pancreatitis have been shown to have pulmonary abnormalities (infiltrates, effusions), most often in those with severe disease. Bilateral or left-sided pleural effusions have a higher prognostic value.

Ultrasonographic Evaluation

Ultrasonography (USG) should be performed as soon as feasible on acute pancreatitis patients to check for gallbladder and/or common duct stones. But only in the early stages of the sickness is US helpful. Intestinal gas may sometimes conceal the pancreas, making it difficult to identify pancreatic necrosis. The discovery of intraparenchymal and retroperitoneal fluid collections corresponds poorly with pancreatic necrosis. Acute pancreatitis patients with abnormal US findings ranged from 33% to 90%. In those with severe disease, interstitial edema may result in a diffusely enlarged and hypoechoic gland as well as extrapancreatic fluid collections (in the smaller sac or anterior pararenal area).

CT Evaluation

The clinical care of acute pancreatitis has been revolutionized by the advent of CT for use in diagnosis and staging. The majority of the clinical and laboratory parameters discussed up to this point are used to evaluate the systemic effects of pancreatitis and only make a cursory effort to indirectly infer the presence and degree of pancreatic damage.

Contrast-enhanced CT scanning methods make it easy to assess the severity of pancreatic necrosis and peri-pancreatic collections. (8)

To increase the early prognostic usefulness of CT in instances of acute pancreatitis, the first CT severity index was developed.

CT Severity Index	
Prognostic indicator	Points
Pancreatic inflammation	
Normal pancreas	0
Focal or diffuse enlargement of pancreas	1
Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2
Single, ill defined fluid collection or phlegmon	3
Two or more poorly defined collections or presence of gas in or adjacent to the pancreas	4
Pancreatic necrosis	
None	0
<30%	2
30-50%	4
>50%	6

Figure 12- CT Severity Index

A patient receives three points for a CT grade D, for instance, and an additional six points for more than 50% necrosis, for a total index score of nine. A statistically significant correlation between patients' CT severity index groups and an increasing incidence of morbidity and mortality was found. Patients with a severity score of 0 or 1 had nil mortality and morbidity rates, whereas patients with a severity level of 2 had a mortality and morbidity rate of 4%. With a severity level of 7 to 10, there were 92 percent of complications and 17 percent mortality rates.(34)

Modified CT Severity Index	
Prognostic indicator	Points
Pancreatic inflammation	
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4
Pancreatic necrosis	
None	0
<30%	2
>30%	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement)	2

Figure 13- Modified CT severity Index

The CT severity score has been shown to be an effective method for estimating overall morbidity and mortality in patients with acute pancreatitis, but more recently, its drawbacks have come to light in the literature. One first finding is that there is no significant correlation between the score of this index and the presence of organ failure, extrapancreatic parenchymal difficulties, or peripancreatic vascular abnormalities. Second, using the CT severity score, there was no discernible difference between patients with 30 to 50 percent necrosis and those with more than 50 percent necrosis in terms of morbidity and mortality.(34)

A modified CT severity measure was developed to address these shortcomings by include elements that reflect organ failure and extrapancreatic consequences, both of which contribute to poor prognosis. A precise count of the collections is not essential since the modified CT severity index only distinguishes between the

presence and absence of acute fluid collections. Additionally, the distinction between those with 30–50% necrosis and those with more than 50% necrosis is no longer necessary since the new index essentially awards a score of 0 for the lack of pancreatic necrosis, 1 for moderate necrosis, and 2 for severe necrosis. The presence of extrapancreatic symptoms, such as pleural effusion, ascites, extrapancreatic parenchymal abnormalities, vascular issues, or involvement of the gastrointestinal system, may also be included into the analysis with a minor weighting (2 points or 20 percent).

The findings indicate that the modified CT severity index may correctly predict the prognosis of acute pancreatitis patients. ((34)(35)

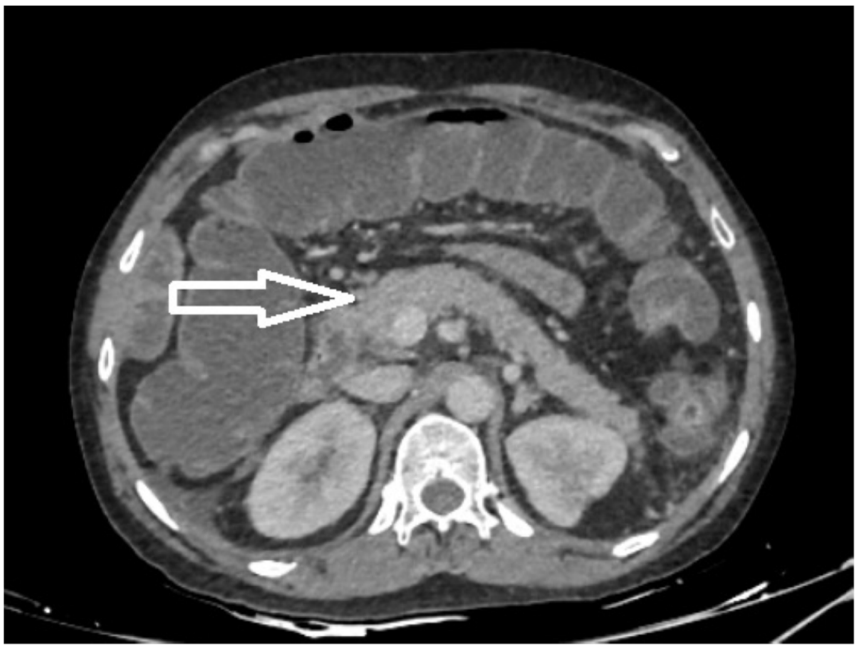


Figure 14- CT image of normal pancreas (arrow)

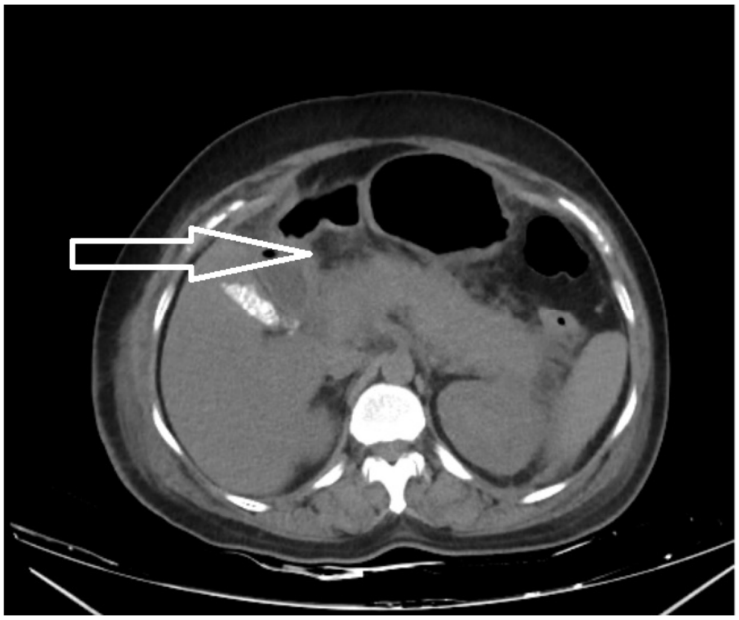


Figure 15- CT image shows a bulky pancreas with adjacent fat stranding (marked by the arrow).

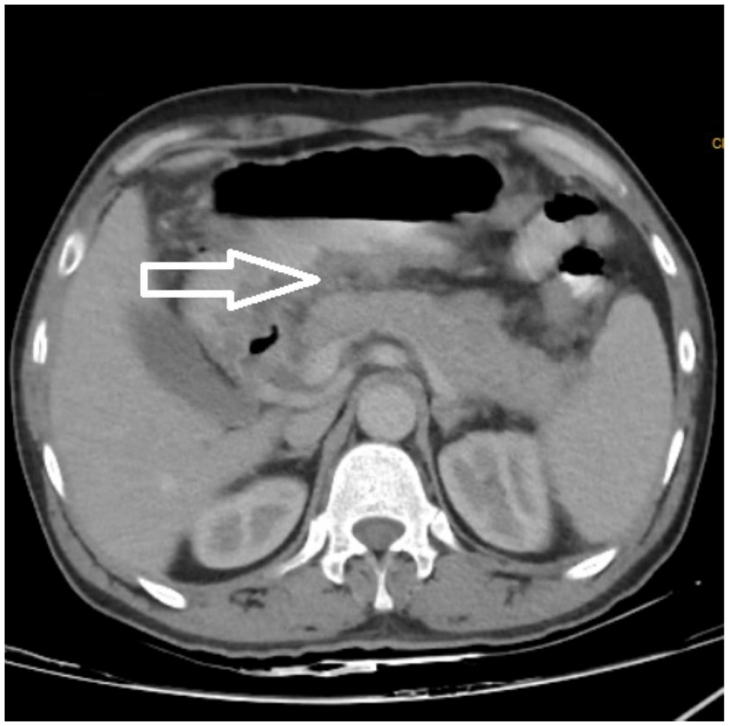


Figure 16- peripancreatic fat stranding with fluid

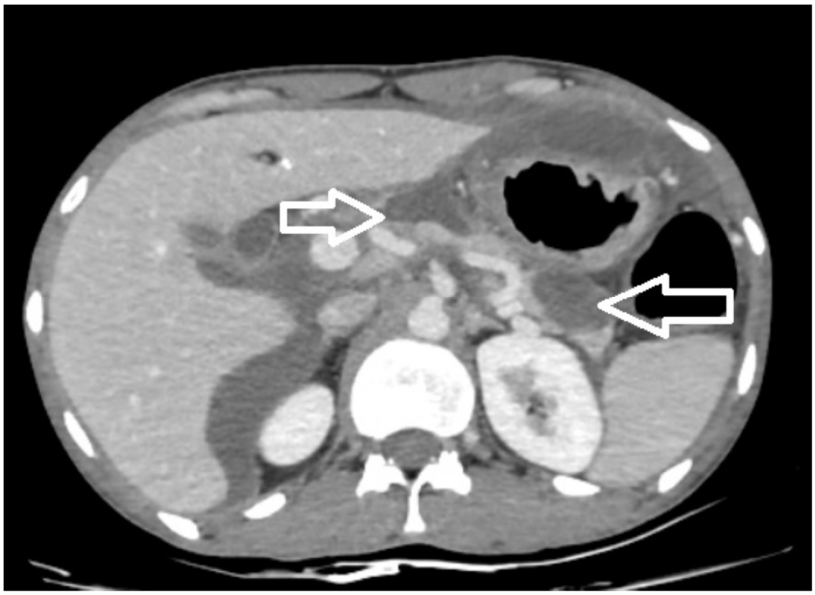


Figure 17- Intrapancreatic collections

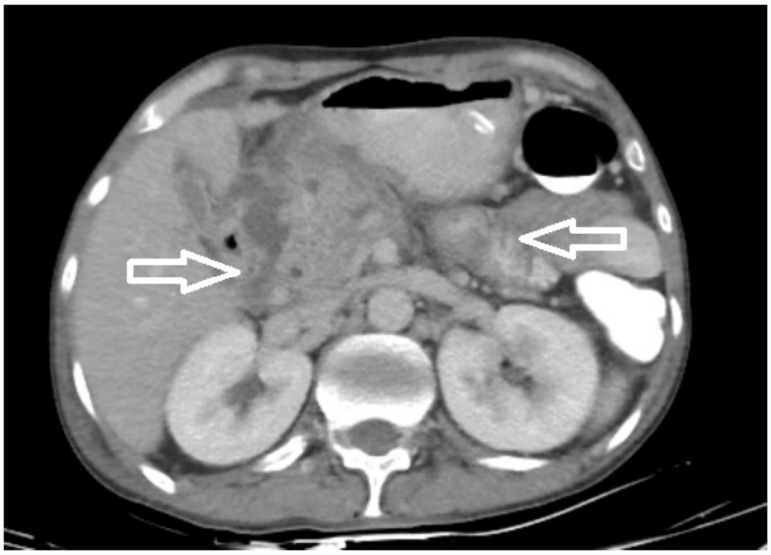


Figure 18- pancreatic necrosis with peripancreatic necrotic collections

Comparison

The Ranson analytic criteria for the diagnosis of severe pancreatitis were recently shown to be weaker prognostic indicators than CT assessment results owing to greater sensitivity and specificity.

Magnetic Resonance Imaging

The popularity of magnetic resonance imaging (MR imaging) as a helpful noninvasive choice for patient assessment and acute pancreatitis staging has been boosted by high-field MR imaging, rapid gradient-echo breath-hold procedures, and fat-suppression treatments. Patients who have severe allergies to iodinated contrast material or who have renal insufficiency might benefit greatly from MR imaging. On gadolinium-enhanced T1-weighted gradient-echo MR imaging, pancreatic necrosis may appear as nonenhanced parenchyma. Images produced with fat suppression help much with detecting subtle parenchymal abnormalities, whether they are systemic or localized. Images with a T2 weighting may show regions of bleeding, pseudocysts, and fluid collections with great detail. When compared to CT pictures made with intravenous contrast material and collimation smaller than 5 mm, the results of MR imaging should be similar. To further characterize ambiguous CT abnormalities or to stage acute pancreatitis, MR imaging may be used as a valid and additional modality.(36)

MATERIALS AND METHODS

Study Design: A cross sectional study

Study period: January 2021 to December 2021

STUDY POPULATION: Patients hospitalised at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi with discomfort abdomen with or without nausea vomiting and radiation of pain to the back.

SELECTION CRITERIA:

INCLUSION CRITERIA:

1) All cases admitted with severe pain abdomen and all other symptoms and signs pointing towards a clinical diagnosis of pancreatitis
2) Age more than 18 years
3) A first-time case of pancreatitis

EXCLUSION CRITERIA:

1) Younger than 18-year-old and older than 75 years
2) Any patient presenting beyond 3 days of pain.
3) Hemodynamically unstable patients
4) Patients with raised creatinine levels unable to undergo Contrast Enhanced CT scan
5) Patients allergic to contrast materials used in CECT
6) Patients with CKI not able to tolerate fluids

Sample size: Increased to 40.

Sample size formula:

The formula for determining the minimal sample size using two proportions is

$$n = \frac{(z_\alpha + z_\beta)^2 \bar{p}(1-\bar{p})}{d^2}$$

where p_1 and p_2 are the proportions of the two groups.

$$p = \frac{p_1 + p_2}{2} \text{ and } d = p_1 - p_2$$

The degree of significance is associated with zα, while the strength of the test is associated with zβ. In order to achieve a level of significance of 5%, zα = 1.96, and in order to achieve 80% power, zβ = 0.84.

By taking proportion of success, p_1 = 38% and p_2 = 71% the sample size obtained is 36.

To get the confirmative results the sample size will be raised to 40.

METHODS:

PROCEDURE:

All the patients presenting with symptoms and signs pointing towards the diagnosis of acute pancreatitis will undergo clinical examination and will be assigned a ranson score and also undergo a CT scan and were scored as per Modified CTSI within 48 hours of admission. Certain parameters like 1) Pain 2) Duration of stay in hospital 3) Death at which day 4) Pseudocyst resolution 5) Diet is changed on which day 6) Other complications 7) Day of discharge 8) Day at which amylase and lipase return to normal 9) Calcium level, Glucose, Haemoglobin will be taken into consideration and will be compared for every patient having a particular score on both

ranson and MCTSI scoring. The comparison will be charted based on all the parameters and studied to asses which is a more accurate prognostic scoring for acute pancreatitis.

STATISTICAL ANALYSIS

Since this is an observational research, we shall do our analysis as follows.

The average and standard deviation for continuous variables will be calculated. Any continuous variables will be compared using suitable statistical procedures, such as the student's unpaired t test, if the data is split into two groups based on some qualitative attribute.

To evaluate the performance of a potential new diagnostic method, its sensitivity, specificity, positive predictive value, and negative predictive value will be computed.

Discrete variables will be represented by median.

Rates, ratios, and percentages will be used to describe the category information. Chi-square, test of proportion, or Fisher's exact tests will be used to examine the correlation between the outcome, clinical, and demographic variables.

A nonparametric test will be applied for the discrete data.

An appropriate graph will be utilised to show the contrast.

The significance level for all tests will be set at a p-value of less than 5 percent (0.05).

RESULTS

STATISTICAL METHODS:

Descriptive analysis:

Quantitative data were analysed using measures such as mean and standard deviation, whereas qualitative data were analysed using frequency and percentages. The median and interquartile range were used to summarise non-normally distributed quantitative data (IQR). Data was also shown using more conventional methods, such as bar charts, pie charts, and box plots.

In order to guarantee that all quantitative variables were normally distributed within their respective explanatory variable categories, we visually assessed their distributions using histograms and normality Q-Q plots. The Shapiro-Wilk test was also performed to ensure normality. For this study, we assumed the data followed a normal distribution if the probability of the Shapiro-Wilk test was larger than 0.05.

Categorical outcome / Crosstab:

Chi square/Exact Fisher's test was used to evaluate categorical outcomes between study groups. (Fisher's exact test was employed if the total number of subjects was less than 20, or if the anticipated value in any one of the cells was less than five.)

Normal 2 group (Independent sample t-test):

Using an independent sample t-test for quantitative measures with a normal distribution, mean values were compared across groups (2 groups)

It was declared significant when the probability level was less than 0.05. In order to analyse the data, IBM SPSS version 22 was used. (1)

Normal more than 2 group (ANOVA):

ANOVA (>2 groups) was used to compare the means of quantitative parameters with a normal distribution across the different research groups. If an ANOVA revealed a statistically significant difference, the appropriate post hoc test (LSD/Bonferroni) was employed to determine whether or not the differences between the groups were statistically significant.

A statistically significant difference was defined as having a probability of less than 0.05. Statistics were analysed using IBM SPSS version 22. (1)

1. IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.

Table 5: Descriptive analysis of age in study population (N=40)

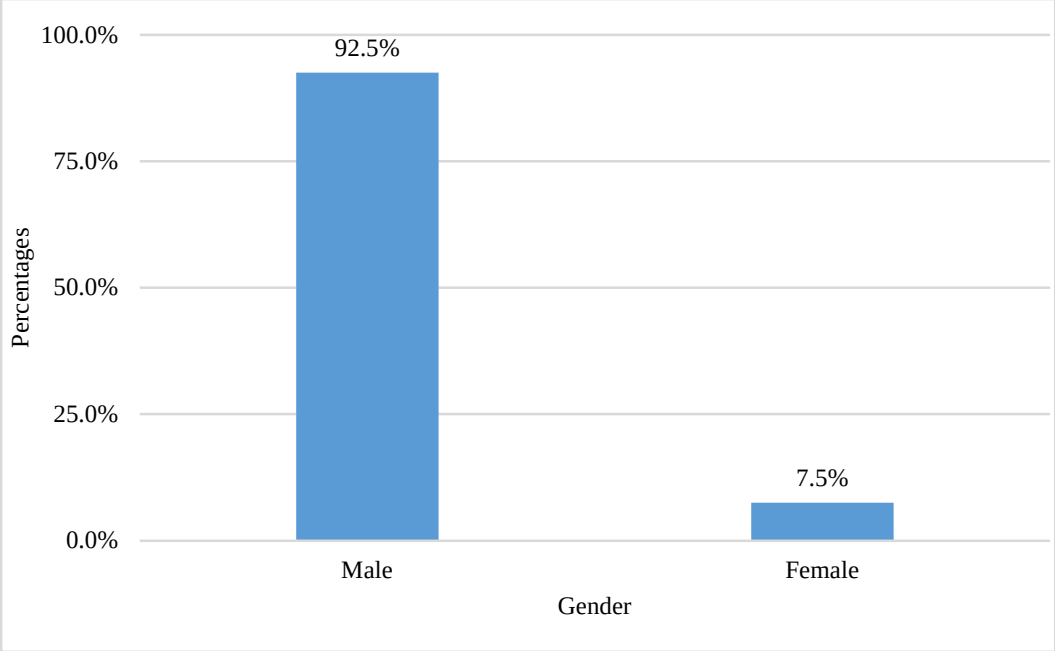
Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Age	40.6 ± 12.31	38.50	18.00	73.00	36.66	44.54

The average age of the study population is 40.6 years, ranging from 18 to 73.

Table 6: Descriptive analysis of gender in the study population (N=40)

Gender	Frequency	Percentages
Male	37	92.50%
Female	3	7.50%

Chart 1: Bar chart of gender in the study population (N=40)



Majority of the patients were male and only 7.5% of the study population were females. The male: female ratio is 12:1.

Table 7: Descriptive analysis of DOHS in study population (N=40)

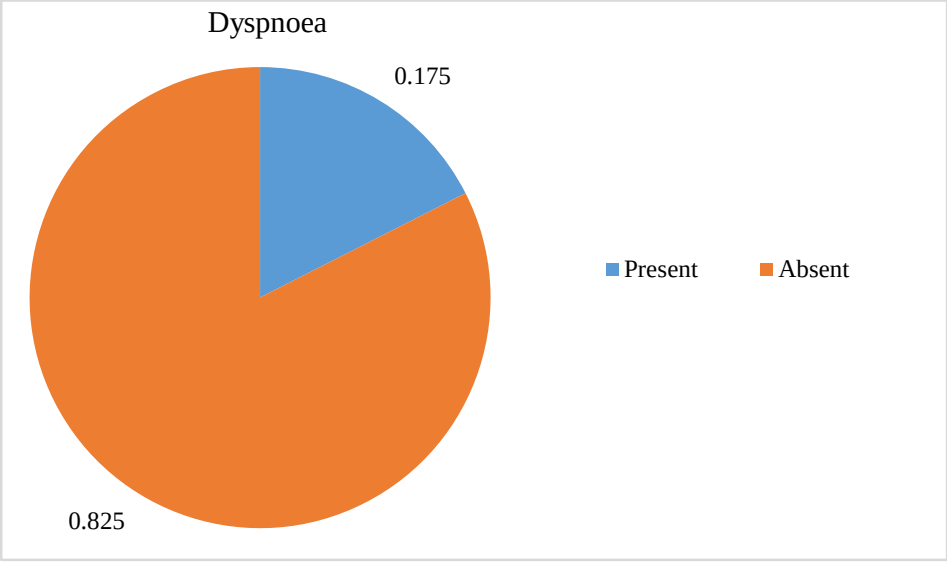
Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
DOHS	9.88 ± 4.39	9.00	5.00	28.00	8.47	11.28

The average hospital stay was 9.88 days and ranged between 5 to 28 days.

Table 8: Descriptive analysis of Parameter a in the study population (N=40)

Parameter	Frequency	Percentages
Dyspnoea		
Present	7	17.50%
Absent	33	82.50%

Chart 2: Pie chart of Dyspnoea in the study population (N=40)



About 82 percent of the patient had dyspnea.

Table 9: descriptive analysis of etiology'

ETIOLOGY	Number	%
Alcohol	24	60
Cholelithiasis	6	15
Choledocholithiasis	2	5
Unknown	8	20

In our study, alcohol intake is the most common cause for AP with 60% followed by unknown etiology as seen in 20%. Cholelithiasis was seen in 15% patients and choledocholithiasis in 2 patients.

Table 10: Descriptive analysis of Parameter in study population (N=40)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Respiratory Rate	22.93 ± 4.94	22.0	16.0	39.0	21.4	24.5
WBC Count	12093.75 ± 5186.57	11500.0	4200.0	24400.0	10435.0	13752.5
LDH	307.25 ± 175.04	236.0	116.0	986.0	251.3	363.2
Amylase	570.43 ± 658.78	382.5	19.0	3192.0	359.7	781.1
Lipase	675.38 ± 600.91	482.5	35.0	2544.0	483.2	867.6
Blood Glucose	158.25 ± 69.96	140.0	75.0	347.0	135.9	180.6
AST	89.25 ± 92.91	48.5	9.0	324.0	59.5	119.0
Fall In HCT >10% After 48h	10.13 ± 5.89	11.0	0.0	25.0	8.3	12.0

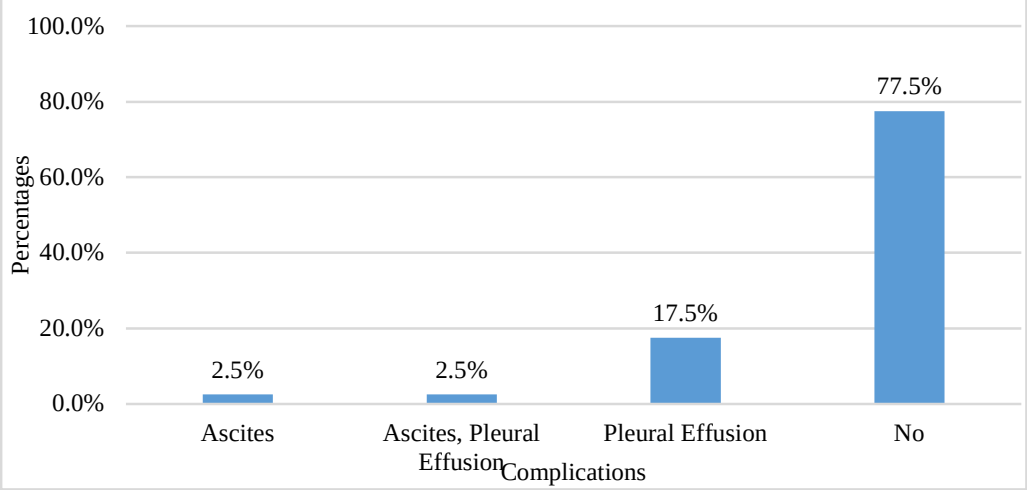
Increase In Bun After 48 H	4.22 ± 5.1	2.0	0.0	16.5	2.6	5.9
Calcium	8.05 ± 1.04	7.9	5.0	9.8	7.7	8.4
Pao2	87.43 ± 11.83	92.0	51.0	96.0	83.6	91.2
Base Deficit	3.05 ± 2.04	3.5	0.0	8.0	2.4	3.7
Fluid Sequestration	1.55 ± 1.28	2.0	0.0	4.0	1.1	2.0
Diet Changed Which Day	7.05 ± 3.27	6.0	3.0	18.0	6.0	8.1
Return Of Enzymes to Normal	8.08 ± 3.73	7.0	0.0	20.0	6.9	9.3

In our study serum amylase and lipase were raised in all study population consistent with acute pancreatitis

Table 11: Descriptive analysis of complications in the study population (N=40)

Complications	Frequency	Percentages
Ascites	1	2.50%
Ascites, Pleural Effusion	1	2.50%
Pleural Effusion	7	17.50%
No	31	77.50%

Chart 3: Bar chart of complications in the study population (N=40)



In our study two complications were noted being ascites and pleural effusion. Pleural effusion was seen in 7 patients, ascites in 1 and both of them in 1 patient.

Table 12: Descriptive analysis of MCTSI score/grade, Ransons score in study population (N=40)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
MCTSI Score/Grade	4.1 ± 2.02	4.0	2.0	10.0	3.5	4.8
Ranson's Score	2.9 ± 1.75	2.5	0.0	7.0	2.3	3.5

Table 13: Descriptive analysis of ransons score in the study population (N=40)

Ransons Score	Frequency	Percentages
Mild	20	50.00%
Severe	20	50.00%

In our study 20 patients had a mild prognostic score as per Ransons scoring and the rest 50% of the patients had severe prognostic score. Ransons score has a total score of 11. A score of 3 or less is considered to have a good prognosis with a milder disease course and more than 3 being severe disease prognosis.

Chart 4: Bar chart of mctsi score in the study population (N=40)

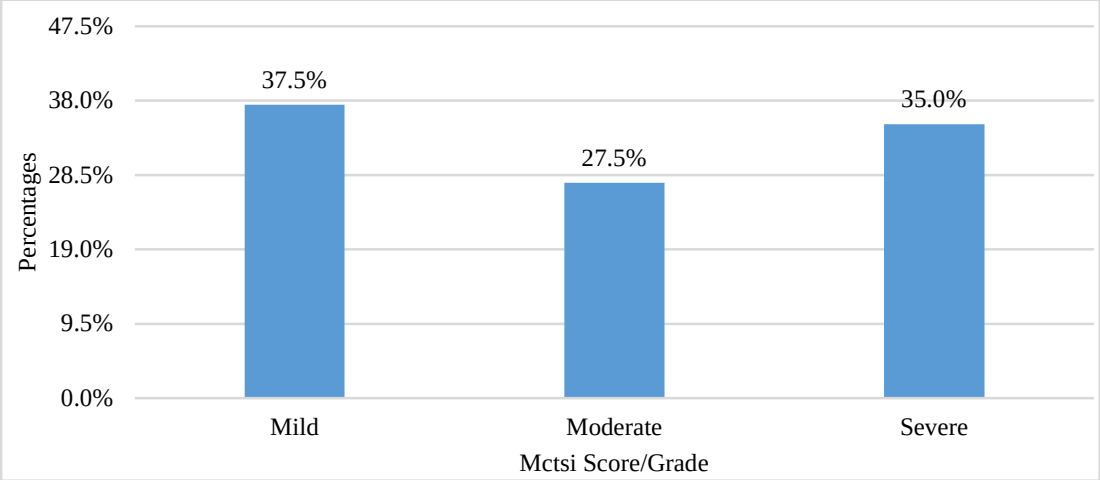
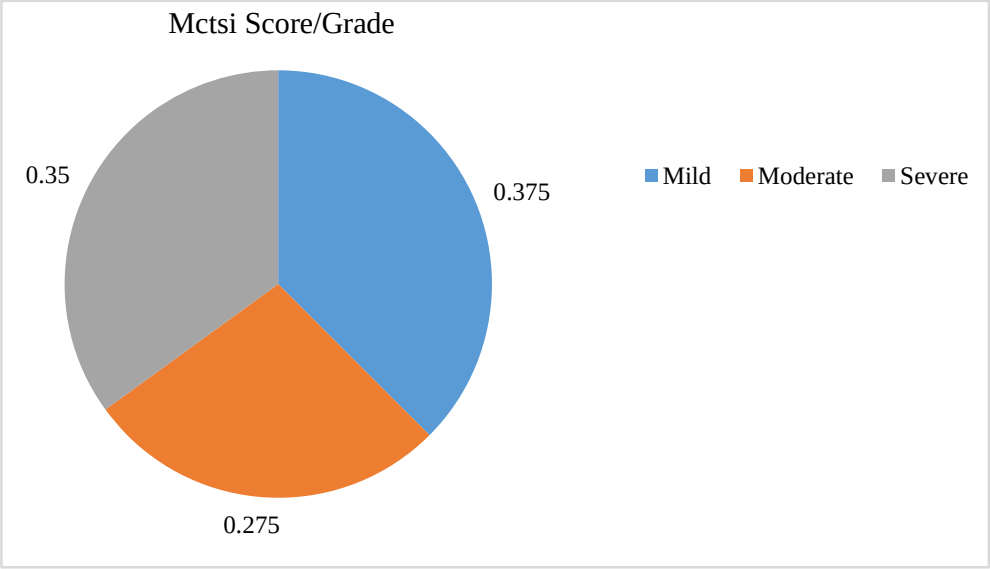


Table 14: Descriptive analysis of MCTSI Score/Grade in the study population (N=40)

MCTSI Score/Grade	Frequency	Percentages
Mild	15	37.50%
Moderate	11	27.50%
Severe	14	35.00%

Chart 5: Pie chart of MCTSI SCORE/grade in the study population (N=40)



In our study 15 patients showed a mctsi score of 0-2 that falls in the mild category, followed by moderate that is a score of 2-4 showed in 11 study patients. However 14 of the study population showed score of 6 or more that falls in the severe category.

Table 15: Comparison of mean of DOHS between ransons score (N=40)

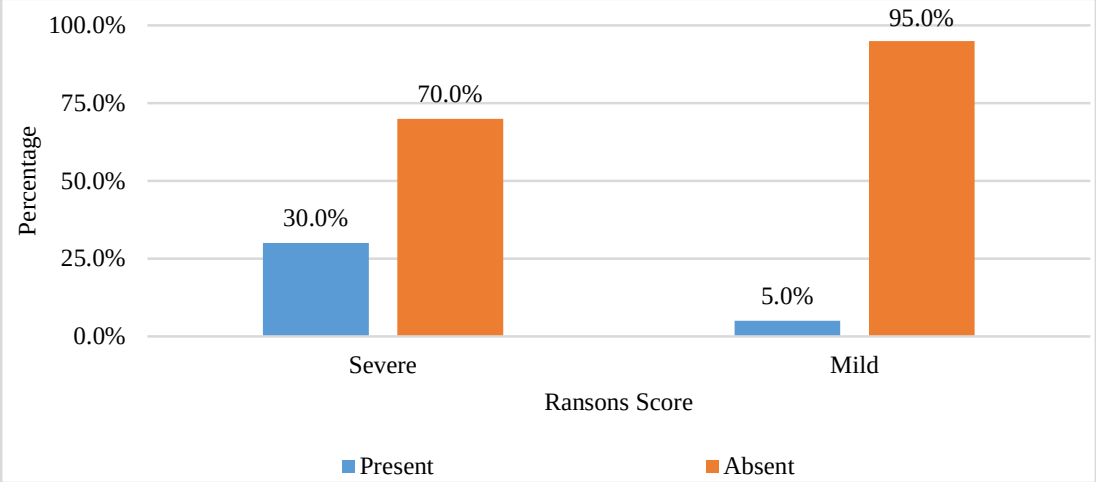
Parameter	Ransons Score (Mean± SD)		P value
	Mild (N=20)	Severe (N=20)	
DOHS	11.1 ± 5.36	8.65 ± 2.78	0.077
Diet Changed Which Day	8.3 ± 3.89	5.8 ± 1.85	0.013
Return Of Enzymes to Normal	9.5 ± 4.2	6.65 ± 2.58	0.014

The above table states that although the ransons score was mild the patient showed a protracted course when it came to the DOHS, the day diet was changed and the day enzymes came back to normal.

Table 16: Comparison of Dyspnoea between ransons score (N=40)

Parameter	Ransons Score		Chi square	Fisher exact P value
	Severe (N=20)	Mild (N=20)		
Dyspnoea				
Present	6 (30%)	1 (5%)	4.329	0.091
Absent	14 (70%)	19 (95%)		
Hypotension				
Absent	20 (100%)	20 (100%)	*	*

Chart 6: Cluster bar chart of comparison of Dyspnoea between ransons score (N=40)

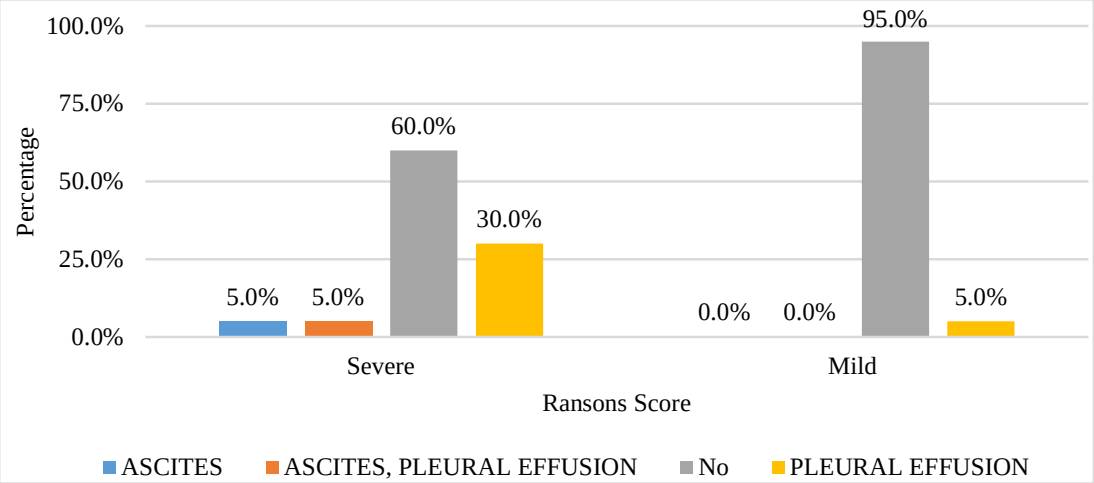


The above table and chart shows that a total of 7 patients showed dyspnea out of which 6 had a severe ransons score and one had not severe scoring. Hypotension was not seen in any of the study population.

Table 17: Comparison of complications between ransons score (N=40)

Complications	Ransons Score		Chi square	P value
	Severe (N=20)	Mild (N=20)		
Ascites	1 (5%)	0 (0%)	7.15	0.067
Ascites, Pleural Effusion	1 (5%)	0 (0%)		
No	12 (60%)	19 (95%)		
Pleural Effusion	6 (30%)	1 (5%)		

Chart 7: Cluster bar chart of comparison of complications between ransons score (N=40)



The above table and chart shows that complications were mostly seen in patients having severe prognostic ranson scoring.

Table 18: Comparison of complications between MCTSI score (N=40)

Parameter	MCTSI Score/Grade			F value	P Value
	Mild	Moderate	Severe		
DOHS	8.87 ± 2.53	8.82 ± 3.49	11.79 ± 5.96	2.16	0.130
Diet Changed Which Day	6.47 ± 2.29	6.36 ± 3.2	8.21 ± 4.02	1.40	0.259
Return of Enzymes to Normal	7 ± 2.42	7.18 ± 3.6	9.93 ± 4.45	2.39	0.066

The above table states that the course of the disease, defined by parameters such as DOHS, diet changed on which day and the day at which enzymes return to normal, were congruent with the predicted course defined by MCTSI scoring system.

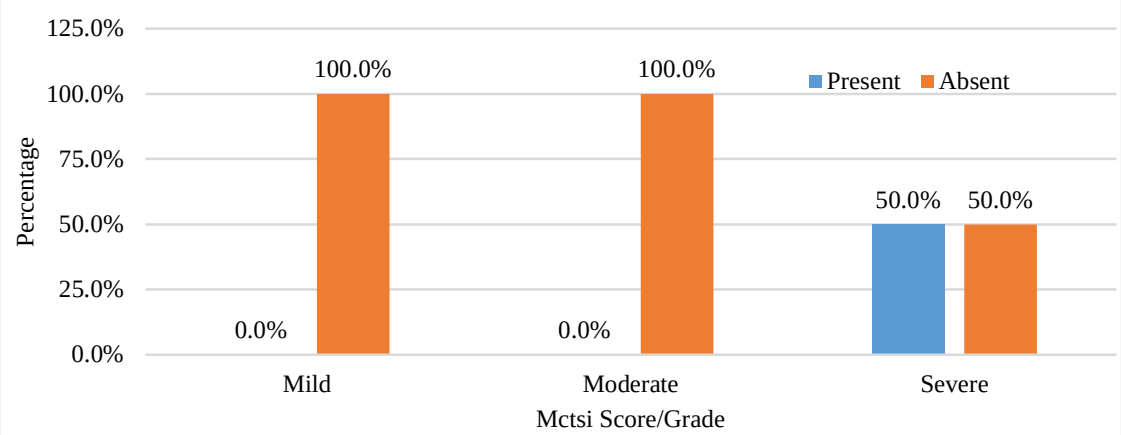
Table 19: Pairwise comparison table

Parameter	Mean Difference	P-value
DOHS		
Mild Vs Moderate	0.048	0.977
Mild Vs Severe	-2.919	0.074
Moderate Vs Severe	-2.968	0.093
Diet Changed Which Day		
Mild Vs Moderate	0.103	0.936
Mild Vs Severe	-1.748	0.154
Moderate Vs Severe	-1.851	0.164
Return of Enzymes to Normal		
Mild Vs Moderate	-0.182	0.898
Mild Vs Severe	-2.929	0.033
Moderate Vs Severe	-2.747	0.063

Table 20: Comparison of dyspnoea across MCTSI score/grade (N=40)

Parameter	MCTSI Score/Grade			Chi square	P value
	Mild (N=15)	Moderate (N=11)	Severe (N=14)		
Dyspnoea					
Present	0 (0%)	0 (0%)	7 (50%)	15.76	<0.001
Absent	15 (100%)	11 (100%)	7 (50%)		
Hypotension					
Absent	15 (50%)	11 (50%)	14 (50%)	*	*

Chart 8: Cluster bar chart of comparison of dyspnoea across MCTSI score/grade (N=40)

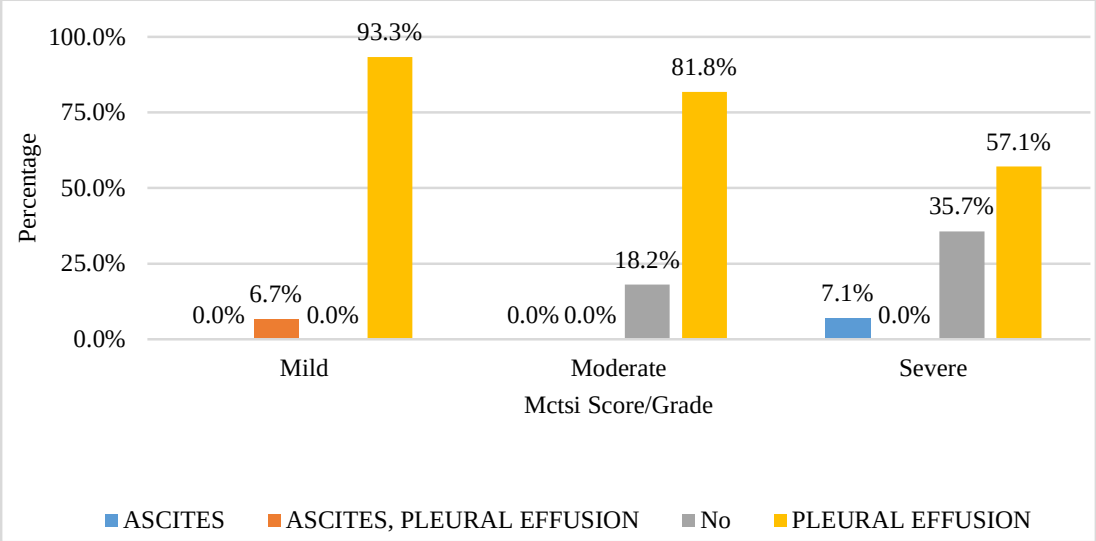


The above table and chart proves that only the study population having severe prognostic scoring showed compications such as dyspnea.

Table 21: Comparison of complications across MCTSI score/grade (N=40)

Complications	MCTSI score/grade			Chi square	P value
	Mild (N=15)	Moderate (N=11)	Severe (N=14)		
Ascites	0 (0%)	0 (0%)	1 (7.14%)	10.07	0.122
Ascites, Pleural Effusion	1 (6.67%)	0 (0%)	0 (0%)		
Pleural Effusion	0 (0%)	2 (18.18%)	5 (35.71%)		
No	14 (93.33%)	9 (81.82%)	8 (57.14%)		

Chart 9: Cluster bar chart of comparison of complications across MCTSI score/grade (N=40)

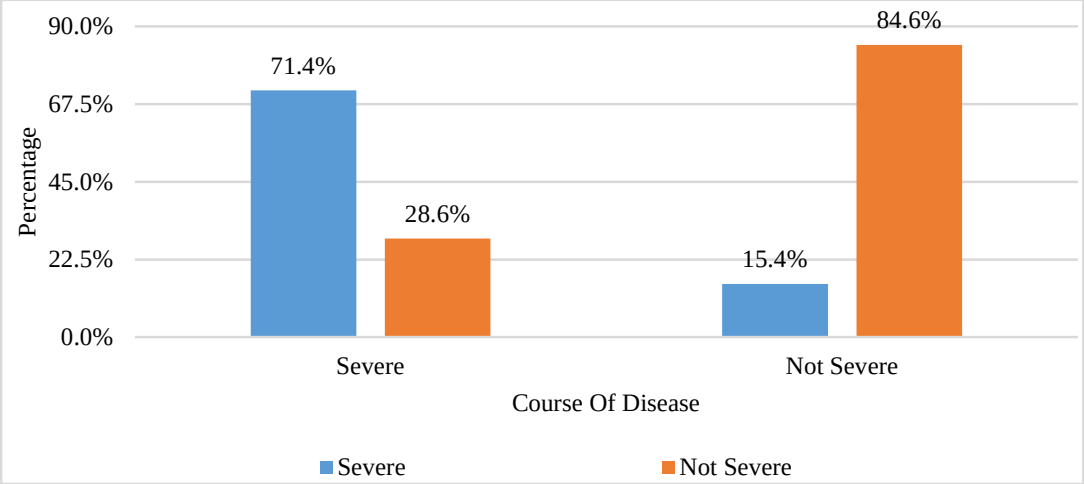


The above table and proves that only the study population having severe prognostic scoring showed compications such as ascites and pleural effusion.

Table 22: Comparison of Course of Disease with MCTSI SCORE (N=40)

MCTSI Score	Course Of Disease		Chi square	P value
	Severe (N=14)	Not Severe (N=26)		
Severe	10 (71.43%)	4 (15.38%)	12.564	**<0.001**
Not Severe	4 (28.57%)	22 (84.62%)		

Chart 10: Cluster bar chart of comparison of MCTSI score between course of disease (N=40



The above comparison shows that MCTSI score has a P value of less than 0.001 and is statistically significant in accurately predicting the course of acute pancreatitis.

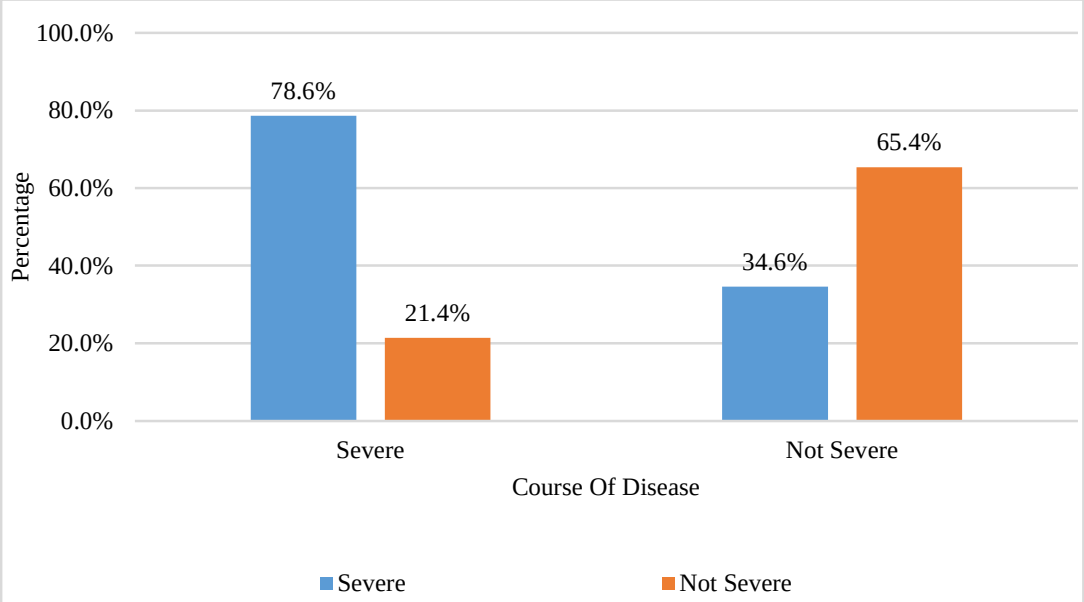
Table 23: Predictive validity of MCTSI SCORE in predicting Course of Disease (N=40)

Parameter	Value	95% CI	
		Lower	Upper
Sensitivity	71.43%	41.90%	91.61%
Specificity	84.62%	65.13%	95.64%
False positive rate	15.38%	4.36%	34.87%
False negative rate	28.57%	8.39%	58.10%
Positive predictive value	71.43%	41.90%	91.61%
Negative predictive value	84.62%	65.13%	95.64%
Diagnostic accuracy	80.00%	64.35%	90.95%

Table 24: Comparison of Course of Disease with RANSONS SCORE (N=40)

RANSONS SCORE	Course of Disease		Chi square	P value
	Severe (N=14)	Not Severe (N=26)		
Severe	11 (78.57%)	9 (34.62%)	7.033	**0.008**
Not Severe	3 (21.43%)	17 (65.38%)		

Chart 11: Cluster bar chart of comparison of RANSONS SCORE between course of disease (N=40)

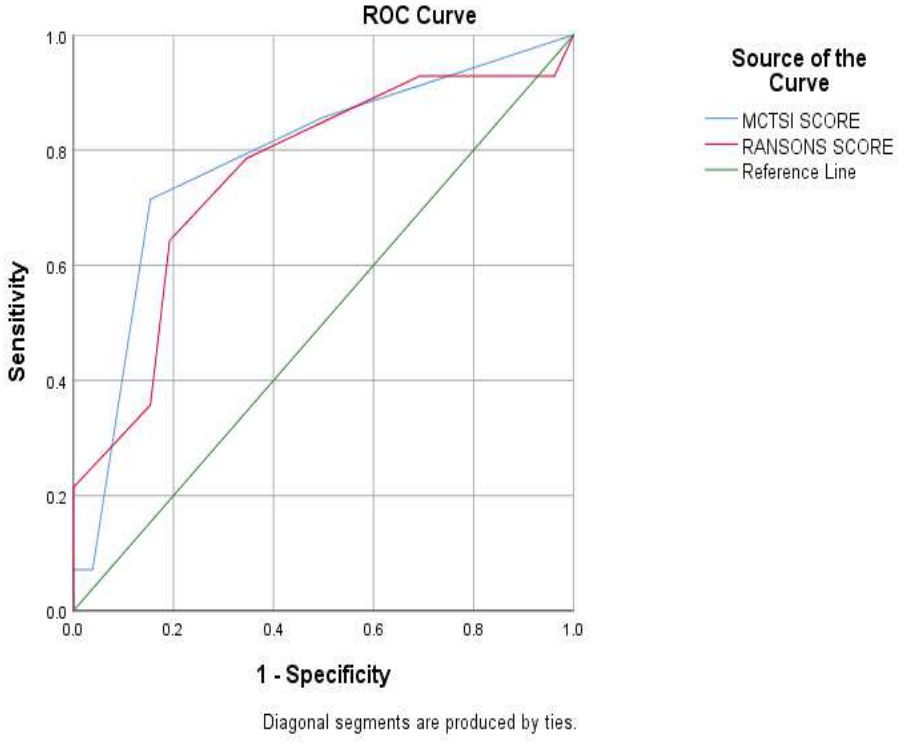


The above table and chart proves that ransons score is statistically significant in predicting prognosis.

Table 25: Predictive validity of RANSONS SCORE in predicting Course of Disease (N=40)

Parameter	Value	95% CI	
		Lower	Upper
Sensitivity	78.57%	49.20%	95.34%
Specificity	65.38%	44.33%	82.79%
False positive rate	34.62%	17.21%	55.67%
False negative rate	21.43%	4.66%	50.80%
Positive predictive value	55.00%	31.53%	76.94%
Negative predictive value	85.00%	62.11%	96.79%
Diagnostic accuracy	70.00%	53.47%	83.44%

Chart 12: Predictive validity of MCTSI & Ranson's score in predicting Course of Disease (N=40)



The area under the curve for both ransons and MCTSI scoring system.

Table 26: Area under the curve for predictive validity of MCTSI & Ranson's score in predicting Course of Disease (N=40)

Test Result Variable(s)	AUC	Std. Error	95% Confidence Interval		P-value
			Lower Bound	Upper Bound	
MCTSI Score	0.784	0.081	0.626	0.942	0.003
RANSONS Score	0.757	0.085	0.591	0.923	0.008

The table thereby comparing all the statistical data proves that MCTSI score is more accurate in predicting prognosis than ransons as MCTSI score has a P value of 0.003 and ransons having 0.008.

DISCUSSION

In this research, the prognostic value of the MCTSI was compared to that of the Ranson's score in determining the outcome of patients with a diagnosis of acute pancreatitis.

In Belagavi, India, at the KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, the study lasted for a full year in the General Surgery Department.

The disease progression of acute pancreatitis varies and differs in different patients. It ranges from a mild form, where the patient only complains of pain, vomiting, and diarrhoea, to a severe form, where the patient may suffer from sepsis, Multi-organ dysfunction syndrome, and even death.

When it comes to problems with the gastrointestinal organs, acute pancreatitis has been called the worst possible outcome. It's the most terrifying disaster because of how quickly it strikes, how much pain it causes, and how quickly it kills you. (2)

Introduced in 2004, the CTSI has proven to be a valuable tool for evaluating patients with acute pancreatitis and has since gained widespread acceptance around the world. It depends on whether or not pancreatic inflammation and necrosis are present, and how severe they are. Moreover, it facilitates the quantification of patients' prognoses. However, it has certain restrictions since it doesn't account for organ failure, extrapancreatic parenchymal lesions, or peripancreatic vascular issues. Mortele et al incorporated the above shortcomings and made it into modified CTSI to make the scoring more accurate.(34)

Acute pancreatitis is difficult to diagnose since its symptoms overlap with those of other conditions that may produce sudden abdominal pain. For clinical decision making, it is helpful to classify acute pancreatitis into mild, moderate and severe. Both Modified computed tomography severity index and CTSI are proven to

have good correlation with predicting prognosis of Acute Pancreatitis patients but MCTSI showed to be a more sensitive parameter. (35) Hence we chose MCTSI as our radiological score system as one component of comparison.

Compared to other scoring systems including APACHE, Glassgow, and BISAP, the Ransons scoring system has been shown to have more accuracy in predicting pancreatic problems, as demonstrated by the research conducted by Yajie Li et al. (37)

The absence of significant results from comparative studies between different scoring systems led us to conduct a study to compare between a radiological that is MCTSI and a clinical prognostic scoring that is Ranson's scoring.(37) (38–40)

Both the MCTSI and the Ransons score played important roles in predicting the prognosis of acute pancreatitis in a research involving 30 patients that was conducted by Prem Chand, Rommel Singh, et al., however the difference between the two was not statistically significant. (40) However, we identified statistically significant differences between the two rating systems after analysing data from 40 individuals with acute pancreatitis.

Prem Chand et al. have recorded complications such as ascites, pleural effusion, pseudocyst, paralytic ileus, Acute respiratory distress syndrome, Renal failure, Multiorgan failure and pancreatic fistula and found ascites to be the most common complication. In our study, we have recorded ascites and pleural effusion, with pleural effusion being the most common complication.(40)

According to research conducted by Biswanath Sahu et al., prolonged alcohol consumption is the leading cause of pancreatitis. (41) Stephanie L et al., in their research found that gallstone disease was the leading cause of pancreatitis bypassing

alcohol intake.(39) In our study population, alcoholism was revealed to be the leading cause of acute pancreatitis.

In their study, Stephanie L et al found the length of hospital stay difference between non-severe and severe ranson scoring to be 1 day.(39) In our study, we found the difference to be 2.45 days and the difference between mild vs. severe MCTSI to be almost three days.

In their study, Ting Kai Leung et al. used parameters such as complications, duration of hospitalization, and mortality rate as their comparing factors for different scoring systems.(10) In our research, we accounted for factors like how long patients stayed in the hospital, whether or not they experienced complications, whether or not their enzyme levels returned to normal, and when patients made adjustments to their diet.

The findings of the above mentioned studies are comparable with our study. The Ranson score and the Modified CTSI were used to predict the outcomes of forty patients admitted with symptoms of abdominal discomfort with or without radiation to the back and nausea and vomiting. On analysis, 20 patients were found to have mild and 20 of them, severe score in ransons scoring system. However, 15 individuals had a mild MCTSI score, 11 had a moderate score, and 14 had a severe score. Patients spent an average of 9.88 +/-4.39 days in the hospital, with stays ranging from 5 to 28 days. Throughout the course of the illness, problems manifested themselves in 9 individuals. Several metrics were used to evaluate the two rating scales. The disease's progress was separated into two groups, one with a large number of parameters and the other with a small number of parameters, and the two anticipated progressions were compared.

Different from previous research, we were able to establish a statistically significant difference between the two scoring systems by using our own, original criteria for comparison.

No intervention was observed in our study.

Both Ransons and MCTSI scoring systems are excellent scoring systems when it comes to prognosticating patients with Acute Pancreatitis. In our study we found MCTSI to be more accurate in predicting prognosis as compared to Ransons score.

CONCLUSION

In this research of 40 patients, it was shown that the MCTSI score was more accurate in predicting the prognosis of the patient than the Ransons scoring system was in patients diagnosed with Acute Pancreatitis. We conclude that the MCTSI score was a better scoring system as compared to ransons scoring system.

SUMMARY

Inflammation of the pancreas characterises acute pancreatitis. Different stages of the illness manifest in different ways, thus it's crucial to make an accurate prognosis.

Predicting a patient's prognosis accurately is crucial for delivering effective treatment and reducing the risk of patient disability and death.

This study is aimed to compare two scoring systems in order to find a more reliable parameter to prognosticate the disease.

A cross-sectional study was conducted in the Division of General Surgery at KLES Dr. PK and MRC, Belgaum, between January 2021 and December 2021.

A total of 40 patients with pain abdomen diagnosed with acute pancreatitis were chosen and prognostic scoring was calculated using both Ransons and MCTSI and then compared with the chosen parameters to assess which was more accurate.

Both Ransons criteria and MCTSI score predicted prognosis properly but when compared, MCTSI was found to be more effective at predicting prognosis.

LIMITATIONS

Although in our research we were able to reach a conclusion the study has limitations.

1) It has a small study population.
2) Single Center Study
3) No new parameter was found which could help in prognosticate pancreatitis in a more accurate way.

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ANNEXURE I- CONSENT FORM

CONSENT TO PARTICIPATE IN RESEARCH STUDY:

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form or it has been read to me, and has been explained to me in my vernacular language and had all my questions answered. I will be given a copy of this consent form.

1. I understand that I am participating in the comparative study between Ranson scoring system and MCTSI in a patient of acute pancreatitis.
2. I confirm that I have read and understood the information in the patient information sheet. Procedure is explained to me in detail along with information about the advantages and disadvantages of taking part in the study. I have been given the opportunity to discuss all aspects of the trial, to ask questions and hereby consent to participation in the trial outlined above.
3. I understand that the decision to take part in this study is completely voluntary and I am aware that I can choose to withdraw from the study at any point of time.
4. I consent to the photographing or recording of the procedure to be performed including appropriate portions of my body, for medical, scientific or educational purposes provided my identity is not revealed in the pictures or by the descriptive texts accompanying them.
5. I understand that there is no significant risk involved in the test that would be done in this study.

6. No guarantee or assurance has given by anyone as to the results that may be obtained.
7. My signature on this form signifies that I have willingly decided to participate after understanding the above information.

Participant's Name/ legally authorized _____

Representative

Signature _____

Name and signature of witness _____

Name and signature of interviewer _____

Date:

Place:

(If a patient has limited ability to read and write, an impartial witness should be present during the entire informed consent discussion and patient's legally acceptable representative should sign on the patient's behalf.) In these instances the patient his/her thumb impression taken in place of signature.

I, as the patient's legally acceptable representative was present during the consenting procedure and understand the preceding information describing this study. All of the questions regarding the study and the patient's participation in it have been answered to my satisfaction. I state that all aspects of the study were clearly presented during the consent procedure. The patient is willing to participate in this study and I sign below on his/her behalf testifying to this effect.

Name of the patient:

Name of representative:

Relationship to the patient:

Signature of representative:

Date:

Impartial witness declaration:

By signing the consent form I attest that the information was accurately explained to and apparently understood by the patient and the representative (if applicable) and that the informed consent was freely given by the patient.

Name of impartial witness:

Signature: Date –

ANNEXURE II: PROFORMA

PROFORMA

NAME:

AGE :

SEX :

IP NO:

CECT NUMBER:

DATE OF ADMISSION:

DATE OF DISCHARGE:

HOSPITAL STAY (DAYS):

ADDRESS:

OCCUPATION:

HISTORY OF ALCOHOL INTAKE:

HISTORY OF DRUG INTAKE

PAST HISTORY :

SYMPTOMS:

PAIN

FEVER

VOMITING

DIORRHEA

DYSPNEA

ALTERED SENSORIUM

GENERAL EXAMINATION

PALOR:

PULSE:

BP:

GUARDING/RIGIDITY:

TENDERNESS:

BOWEL SOUNDS:

ASCITES:

BMI:

RESPIRATORY RATE:

INVESTIGATIONS:

CBC :

TC:

RANDOM BLOOD SUGAR:

BLOOD UREA NITROGEN:

SERUM CREATININE:

SERUM AMYLASE :

SERUM LIPASE :

TOTAL BILIRUBIN

DIRECT:

INDIRECT :

LDH:

SGOT :

SGPT :

Ranson score
On admission
Age >55 years
White blood cell count >16 × 10^9/L
Blood glucose >1.1 mmol/L (>200 mg/dL)
LDH >350 units/L
AST >250 units/L
Within 48 hours
Haematocrit fall of 10% or greater
Blood urea nitrogen rise >5 mg/dL (1.8 mmol/L) despite fluids
Arterial oxygen saturation (PaO_2) <8 kPa (60 mmHg)
Serum calcium <2.0 mmol/L
Base deficit >4 mmol/L
Fluid sequestration >6 litres

SERUM CALCIUM:

PaO2 :

FALL IN HAEMATOCRIT (IN %) AFTER 48HR:

BASE DEFICIT:

FLUID SEQUESTRATION:

PROBABLE CAUSE:

CECT Abdomen MCTSI score:

Duration of pain and vomiting

Duration of stay in hospital

Diet is changed on which day

Day at which amylase lipase returns to normal

Other complications

Table 1

IN PATIENT NO/CECT NO	AGE/SEX	NO OF DAYS OF HOSPITAL STAY	Etiology	HYPOTENSION	RESPIRATORY RATE	WBC COUNT	LDH	AMYLASE	LIPASE	BLOOD GLUCOSE	AST	FALL IN HCT >10% AFTER 48H	INCREASE IN BUN AFTER 48 H	CALCIUM	PaO2	BASE DEFICIT	FLUID SEQUESTRATION	DIET CHANGED WHICH DAY	RETURN OF ENZYMES TO NORMAL	COMPLICATIONS	MCTSI SCORE/GRADE
1037070/C49069	47/M	8	Alcohol	-	22	9900	218	109	408	107	307	10	10.6	5	96	8	3	6	8		2
1037384/C39323	54/M	9	Cholelithiasis	-	30	5000	200	2375	2301	347	37	10	1.8	9.3	51	6	3	5	7	PLEURAL EFFUSION	6
1040890/C36915	47/M	28	Unknown	-	18	22000	411	501	469	200	57	0	2	7.2	94	6	3	18	20	PLEURAL EFFUSION	6
1048524/C32606	30/M	9	Alcohol	-	18	7000	176	63	76	307	16	2	4.2	9.6	94	3	0	6	8		2
1048787/C32742	35/M	6	Alcohol	-	26	13300	254	137	480	220	37	19	0	8.4	88	2	2	5	6		2
1052050/C48228	34/M	10	Alcohol	-	16	11200	200	300	160	87	26	0	3.5	8.6	94	2	2	6	8		2
1052147/C47117	53/M	8	Choledocholithiasis	-	22	13000	284	529	1310	247	25	5	4.5	8	94	5	0	5	6		4
1054189/C36566	57/M	9	Alcohol	-	18	4400	116	416	958	180	52	0	4.5	7.7	93	2	4	6	6		2
1055341/C41570	55/M	7	Cholelithiasis	-	22	18800	180	331	437	120	64	9	2	8.2	89	4	1	5	6		4
1056738/C37701	28/M	11	Unknown	-	28	24400	284	972	1366	154	61	12	16.5	6.3	80	4	0	7	9		6
1066537/C11817007	52/M	14	Alcohol	-	20	9200	362	841	959	83	25	12	3.67	7.6	92	2	3	12	13		4
1068429/C41442	37/M	8	Cholelithiasis	-	24	12100	240	474	910	108	324	11	10.5	7.1	92	6	1	5	7		6
1070318/C45137	21/F	5	Unknown	-	20	18500	160	303	233	100	13	8	2	9.4	92	2	0	3	3		4
1072697/C47079	73/M	19	Alcohol	-	24	18900	362	112	111	265	270	14	0	8.2	96	3	2	15	18		6
1074759/C52562	27/M	6	Cholelithiasis	-	36	14600	383	2258	1927	190	270	9	10.8	8.6	54	4	2	5	6	ASCITES	8
1074963/C30386	41/M	5	Alcohol	-	24	12200	542	309	428	294	87	9	0	8.8	88	4	0	4	5		4
1074983/C43045	28/M	6	Unknown	-	34	8600	205	3192	620	142	82	14	2	8.9	87	4	2	4	6	PLEURAL EFFUSION	6
1075641/C23054	39/M	5	Alcohol	-	20	4200	182	34	68	291	116	12	2	7.8	90	2	2	4	0		2
1077266/C43415	33/M	7	Alcohol	-	24	6200	229	482	386	92	21	11	4.3	9.4	94	5	2	6	5		4
1077974/C52625	40/M	10	Cholelithiasis	-	30	21700	789	657	1034	226	76	12	4	7.2	76	4	2	7	8	PLEURAL EFFUSION	6
1078769/C47625	35/M	7	Alcohol	-	20	6000	363	1124	1150	121	66	11	2	7.1	88	4	3	5	6		6
1080304/C43165	34/M	10	Alcohol	-	20	6250	170	305	485	162	262	6	16.5	9.2	88	4	0	6	8	PLEURAL EFFUSION	4
1080448/C44198	36/M	13	Unknown	-	22	10700	370	738	924	182	260	14	5	7.6	90	4	2	10	13		6
1080451/C44199	47/M	11	Alcohol	-	20	9600	182	150	168	107	74	21	1	7.6	94	2	2	9	9		2
1081714/C44458	51/M	8	Alcohol	-	20	18900	389	479	533	181	112	11	0	7.6	92	5	2	6	6		4
1082566/C44691	51/M	14	Alcohol	-	18	14000	200	1095	1020	200	180	2	16.5	9.2	92	0	0	8	10		6
1116162/C11671231	62/M	8	Alcohol	-	39	8900	374	465	1279	146	189	3	6	7.2	58	4	0	8	7	PLEURAL EFFUSION	10
1118389/C27927	56/M	9	Choledocholithiasis	-	22	9200	182	154	469	106	45	6	2	7.6	88	5	1	7	8		2
1119657/C11995436	18/F	10	Unknown	-	24	14400	187	335	199	84	23	16	0	8.2	96	0	2	7	9	-	2
1122296/C11827170	26/M	7	Alcohol	-	20	14900	232	349	490	105	23	17.2	0	7.6	94	0	0	5	6		2
1122449/C235436	45/M	5	Alcohol	-	24	11800	400	117	60	125	34	7	0	9	90	0	0	3	5	-	2
1123013/C33872	25/M	9	Alcohol	-	22	8500	187	325	61	87	17	11	0	8.3	96	1	0	7	6		2
1123239/C12123787	53/M	16	Alcohol	-	24	17600	537	19	35	138	24	12	0	7.6	92	1	4	13	15	PLEURAL EFFUSION	4
1123352/C12139222	25/M	11	Unknown	-	20	18000	383	609	889	91	27	12	15.42	7.6	55	5	4	8	10		6
1124386/C42969	50/F	10	Alcohol	-	20	8300	200	158	304	155	9	0	0	7.8	92	0	0	7	9		2
1081470/C47033	38/M	10	Cholelithiasis	-	22	7600	200	180	156	75	19	10	0	9.8	94	3	2	6	7		2
1121300/C12060566	38/M	15	Unknown	-	20	13600	986	148	227	146	42	19.3	0	5.9	94	4	2	13	10	ASCITES, PLEURAL EFFUSION	2
1122704/C122966	31/M	15	Alcohol	-	22	7600	176	596	2544	124	25	8	3.27	9.6	90	0	2	10	12		6
1123248/C46188	39/M	10	Alcohol	-	22	9100	290	499	895	116	146	25	10.28	8.6	92	2	2	4	6		4
1124273/c37576	33/M	7	Alcohol	-	20	13600	505	577	486	119	27	14.7	1.87	7.5	88	0	0	6	6		4

RANSONS SCORE
5
3
5
1
2
0
2
2
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4
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1
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1
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1
1
4
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