"PROSPECTIVE OBSERVATIONAL STUDY ON SERUM CALCIUM AND SERUM LACTATE RATIO IN ACUTE PANCREATITIS FOR PREDICTING SEVERITY, CLINICAL OUTCOME AND HOSPITAL STAY DURATION AT KLES PRABHAKAR KORE HOSPITAL"

BY

REG NO: BH0121007

Dissertation

Submitted to
KAHER, Belagavi, Karnataka,
In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY (M.S.)

in

GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY
JAWAHARLAL NEHRU MEDICAL COLLEGE,
KAHER, BELAGAVI – 590010
KARNATAKA.

DECEMBER-2024 / JANUARY -2025

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,

BELAGAVI, KARNATAKA.

Endorsement

This is to certify that the dissertation entitled entitled **"PROSPECTIVE OBSERVATIONAL STUDY ON SERUM CALCIUM AND SERUM LACTATE RATIO IN ACUTE PANCREATITIS FOR PREDICTING SEVERITY, CLINICAL OUTCOME AND HOSPITAL STAY DURATION AT KLES PRABHAKAR KORE HOSPITAL"** is a bonafide research work done by **REG No. BH0121007.**

Dr. S.C. METGUD$_{M.S}$
Professor and Head
Department of General Surgery,
KAHER's J.N.Medical College,
Nehru Nagar, Belagavi -590010

Professor & HOD
Dept. of General Surgery
J.N. Medical College, BELAGAVI

Date:
Place: Belagavi

Dr. (Mrs)N. S. MAHANTASHETTI$_{M.D(Peads)}$
Principal
KAHER's J.N.Medical College,
Nehru Nagar, Belagavi -590010

PRINCIPAL
JAWAHARLAL NEHRU MEDICAL COLLEGE
BELAGAVI

Date:
Place: Belagavi

KAHER
BELAGAVI
J.N. MEDICAL COLLEGE

UNDERTAKING

I, REG No. BH0121007 here by declare that the information and the data mentioned in my dissertation entitled entitled **"PROSPECTIVE OBSERVATIONAL STUDY ON SERUM CALCIUM AND SERUM LACTATE RATIO IN ACUTE PANCREATITIS FOR PREDICTING SEVERITY, CLINICAL OUTCOME AND HOSPITAL STAY DURATION AT KLES PRABHAKAR KORE HOSPITAL"** belongs to me and is original. I am aware of the definition of Plagiarism as detailed below:

- An act or instance of using are closely imitating the language and thoughts of another author without authorization and the representation of that authors work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorised use or imitation.
- The deliberate or reckless representation of another's words, thoughts, or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original-one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then, I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the university.

Date: 10/07/24

Place: Belagavi

REG NO: BH0121007

PLAGIARISM CLEARANCE

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MoE (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

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

Ref No: MDC/PG/ Date: 26-06-2024

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: **"PROSPECTIVE OBSERVATIONAL STUDY ON SERUM CALCIUM AND SERUM LACTATE RATIO IN ACUTE PANCREATITIS FOR PREDICTING SEVERITY, CLINICAL OUTCOME AND HOSPITAL STAY DURATION AT KLES PRABHAKAR KORE HOSPITAL"** has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **07%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J. N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BH0121007
Postgraduate Student,
2021-22 Batch,
Department of General Surgery
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
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 No.MDC/JNMCIEC/ 10 **Date: 27/09/2022**

To.
REG NO: BH0121007
PG Student in General Surgery,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PROSPECTIVE OBSERVATIONAL STUDY ON SERUM CALCIUM AND SERUM LACTATE RATIO IN ACUTE PANCREATITIS FOR PREDICTING SEVERITY, CLINICAL OUTCOME AND HOSPITAL STAY DURATION AT KLES PRABHAKAR KORE HOSPITAL.",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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

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

LIST OF ABBREVIATIONS

ACG	:	American College of Gastroenterology
AP	:	Acute Pancreatitis
APACHE-II	:	Acute Physiology and Chronic Health Evaluation II
BISAP	:	Bedside Index for Severity in Acute Pancreatitis
ABG	:	Arterial Blood Gas
BUN	:	Blood Urea Nitrogen
CRP	:	C-Reactive Protein
CT	:	Computed Tomography
ERCP	:	Endoscopic Retrograde Cholangiopancreatography
FeCa	:	Fractional Excretion of Calcium
HbA1c	:	Hemoglobin A1c
L1, L2	:	First and Second Lumbar Vertebrae
MRCP	:	Magnetic Resonance Cholangiopancreaticography
MPD	:	Main Pancreatic Duct
MRI	:	Magnetic Resonance Imaging
PTH	:	Parathyroid Hormone
SIRS	:	Systemic Inflammatory Response Syndrome
SMA	:	Superior Mesenteric Artery

SMV	:	Superior Mesenteric Vein
UCa: UCr	:	Urine Calcium to Creatinine Ratio
iCa	:	ionized Calcium
LAMA	:	Left Against Medical Advice
POF	:	Persistent Organ Failure

ABSTRACT

Background: Acute pancreatitis (AP) is a significant cause of morbidity and mortality worldwide, often progressing to severe disease characterized by organ dysfunction and extended hospital stays. Traditional prognostic indices like Ranson, BISAP, APACHE-II, and Balthazar are used to predict disease severity, with total serum calcium being included in some scales. Sustained global increases in cytosolic calcium (Ca^{2+}) due to stimuli like bile and ethanol can lead to premature trypsinogen activation, vacuolization, and pancreatic acinar cell death in the pancreas, contributing to hypocalcemia. Elevated serum lactate, a marker of tissue hypoxia, has also been associated with poor outcomes in AP, suggesting that the serum calcium to serum lactate ratio may be a valuable predictor of disease severity than compared to individual marker Serum Calcium or Serum Lactate. This study aims to evaluate the efficacy of serum calcium and serum lactate levels, and their ratio, in predicting the severity, clinical outcomes, and hospital stay duration in patients with acute pancreatitis.

Material and Method: A prospective observational study was conducted from January 1, 2023, to December 31, 2023, involving 49 patients diagnosed with acute pancreatitis at KAHER's Dr.Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi. Participants aged 21-60 years with acute pancreatitis admitted within 72 hours of symptom onset were included. Exclusions were made for those under 21, pregnant or lactating women, and patients with sepsis unrelated to AP. After performing Allens Test, ABG done within 3 days of symptom onset or upon admission upto 24 hours. Serum calcium and lactate levels were measured in mmol/L and the calcium to lactate ratio was analyzed for correlation with disease severity and

outcomes. Statistical analysis utilized R software and SPSS, with significance set at $p \leq 0.05$.

Results: The study included 49 participants, with a mean age of 39 years, predominantly male (85.7%).Comorbidities were present in 42.9% of the patients, with diabetes and hypertension being most common. The mean serum amylase was 869.0±74.1 U/L and lipase was 1170.1±960.9 U/L. Severe pancreatitis was observed in 30.6% of patients, while 34.7% had mild and 34.7 % had moderate disease. Significant differences in serum calcium and lactate levels were noted with disease severity. Patients with severe pancreatitis had lower serum calcium, higher serum lactate, and lower calcium-lactate ratios, which correlated with prolonged hospital stays and increased mortality. A lower calcium-lactate ratio was linked to worse outcomes and extended hospitalizations.

Conclusion: Serum calcium and lactate levels, and their ratio, may serve as effective biomarkers for predicting acute pancreatitis severity. Lower calcium-lactate ratios and higher lactate levels are indicative of severe disease, correlating with poorer clinical outcomes and longer hospital stays. These findings support the use of these markers in routine clinical practice to identify high-risk patients early and optimize management strategies.

Keywords: Acute Pancreatitis, Serum Calcium, Serum Lactate, Prognostic Markers, Disease Severity, Hospital Stay.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Acute pancreatitis is a leading global contributor to morbidity and death, and it is becoming more common in both men and women. About one-third of patients may experience acute severe pancreatitis, which can lead to progressive organ dysfunction usually brought on by an inflammatory response. One of the mechanisms of hypocalcemia is thought to be prolonged continuous rise in cytosolic Ca++ (due to stimulus like bile and ethanol) is responsible for early activation of trypsinogen, vacuolization and pancreatic acinar cell death of pancreas[1]

This condition is linked with a significant risk of mortality and morbidity as well as a prolonged hospital stay, so it's critical to diagnose and treat acute pancreatitis as soon as possible.[2]

Prognostic indices, such as Ranson, APACHE-II, BISAP, and the Balthazar computed tomography severity index, are frequently used to forecast the severity of the illness process and to direct care. A position for total serum calcium exists in several AP prognostic severity ratings. [3]

Anaerobic glucose metabolism produces lactate, which is often regarded as a sign of tissue hypoxia. Furthermore, prior research has discovered that elevated arterial lactate levels indicate critical tissue hypoperfusion, which is highly correlated with a higher risk of morbidity and death in patients in critical condition. According to a recent study, high blood lactate is a novel biomarker that may be useful in predicting worse patient outcomes upon admission, particularly in terms of mortality prediction. Therefore, we believe that arterial lactate might be a useful diagnostic for AP patients' risk stratification.[4]

Hence, serum calcium and serum lactate (measured in mmol/L) can be useful to predict severity of pancreatitis. Moreover, much literature on Serum Calcium**:** Serum Lactate Ratio levels and its outcomes predicting severity in Acute Pancreatitis have not been found.

Total Serum Calcium and Serum Lactate are simplified markers that can be readily measured, which might be helpful to identify severity of cases early in disease onset as hypocalcaemia is found to be a poor prognostic marker and elevated lactate level is a marker of sepsis, tissue hypoxia and necrosis. This study is done to help in early detection of severity of acute pancreatitis, correlating the Serum calcium and Serum Lactate Ratio value to the severity of pancreatitis and clinical outcome.

AIMS AND OBJECTIVES

Objectives

- To calculate Ratio of Serum Calcium and Serum Lactate in confirmed cases of acute pancreatitis for predicting severity of the disease, clinical outcome and duration of hospital stay.

REVIEW OF LITERATURE

Anatomy of Pancreas

The pancreas is a digestive gland located behind the abdomen between the 1st Lumbar and 2nd Lumbar vertebrae. It is located in the upper belly horizontally, with spleen on left and duodenum on right surrounding it. Its four structural components are head, neck, body & tail. The head is enclosed by the duodenum's C loop and is in close proximity to the renal vein and inferior vena cava, whereas the tail reaches towards the splenic hilum. The pancreas secretes both endocrine and exocrine substances, such as insulin and glucagon, which are released into the circulation by the pancreatic islets of Langerhans and transported to the duodenum by the main and auxiliary pancreatic ducts, respectively.[5]

Structure and division

The pancreas is segmented into four sections: head, neck, body & tail. The pancreatic head, which is the larger portion of the gland, is surrounded by C-shaped curve of the duodenum. As it descends to the duodenum's lower portion, the bile duct either travels via a groove on the top posterior side of the head or is lodged in its tissue. The pancreatic body extends over the aorta and the 2nd Lumbar vertebra after emerging from the neck. The peritoneal layer covers the front aspect of the pancreatic body but is absent from the posterior. It connects with superior mesenteric artery (SMA), aorta, left kidney, left suprarenal gland, and renal vessels.[5]

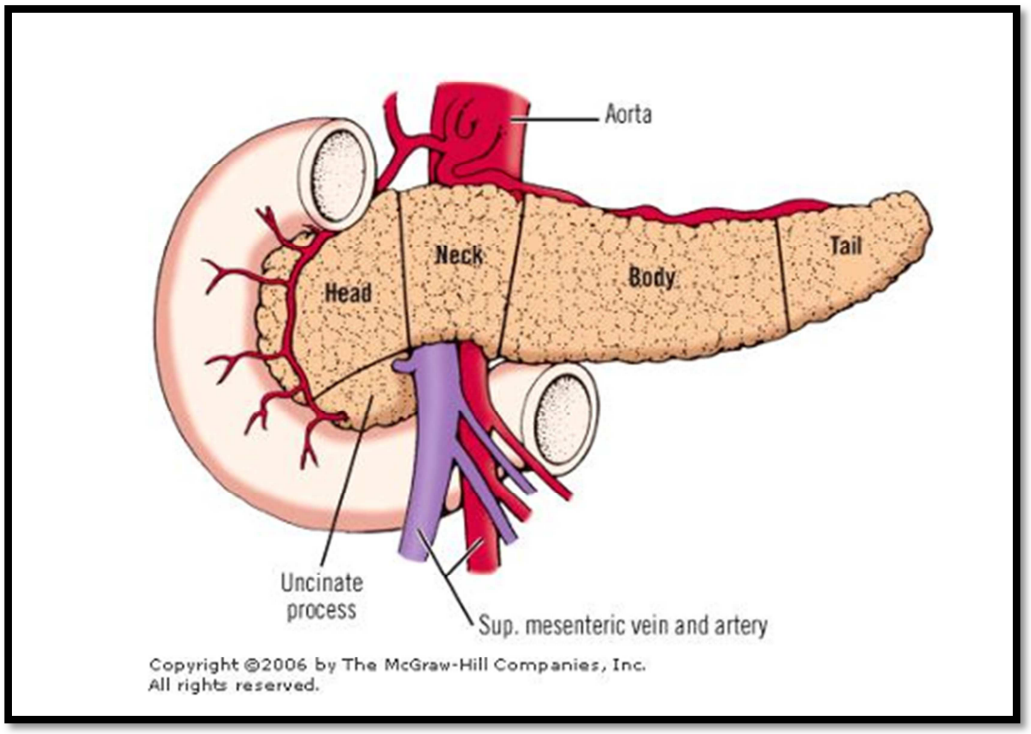

Figure 1: Parts of pancreas

The pancreas has a short neck, and its tail connects to left colic flexure and the splenic hilum in front of left kidney. The bile duct and the main pancreatic duct combine to form hepatopancreatic ampulla, which opens into the duodenum's descending section. The sphincter of Oddi, which is composed of smooth muscle, regulates the flow of bile and pancreatic juice into the ampulla while preventing the backward flow of duodenal contents.[5]

Exocrine pancreatic tissue, mostly consisting of pancreatic acini, makes up around 80% of the pancreas. These acini have pyramid-shaped cells with tip facing the lumen. The nucleus and endoplasmic reticulum are located in the basal area, enabling the generation of enzymes, and there are many zymogen granules in the apical region. Within Golgi complexes, digestive enzymes are kept in secretory vesicles. On the basolateral membrane of acinar cells, neurotransmitter receptors such

as acetylcholine, cholecystokinin, and secretin control the release of digestive enzymes. Endocrine cells found in the Langerhans islet of the pancreas release hormones into the circulation through a sophisticated capillary network that is part of the pancreatic blood flow. Endocrine hormones are released into the bloodstream directly, in contrast to exocrine enzymes, which are released via the process of exocytosis. There are four different types of endocrine cells in the Langerhans islet: glucagon-producing A cells, insulin-producing B cells, somatostatin-producing D cells, and pancreatic polypeptide-producing F cells.[5]

Stellate cells, epithelial structures originating within the pancreas, contribute to inflammation and fibrosis in conditions like chronic pancreatitis.

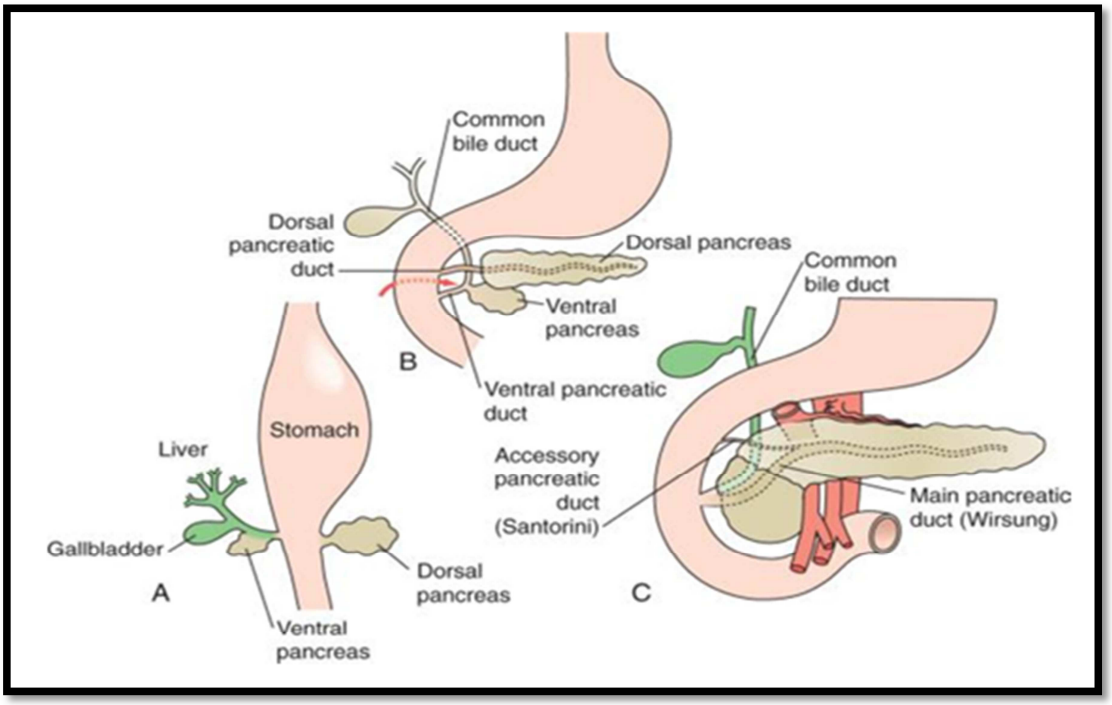

Figure 2: Development of pancreas

Embryology

The posterior foregut endoderm gives rise to the pancreas. Dorsal and ventral buds are first produced by this endoderm, and they begin to elongate about the fourth week of pregnancy. By the sixth week, the ventral pancreatic bud rotates around emerging duodenum and eventually fuses with dorsal bud, leading to formation of pancreas by seventeenth week of pregnancy. Consequently, the ventral bud contributes to the bottom portion of the pancreas and the uncinate process, whereas the dorsal bud develops the top portion of the pancreatic head, body, and tail.

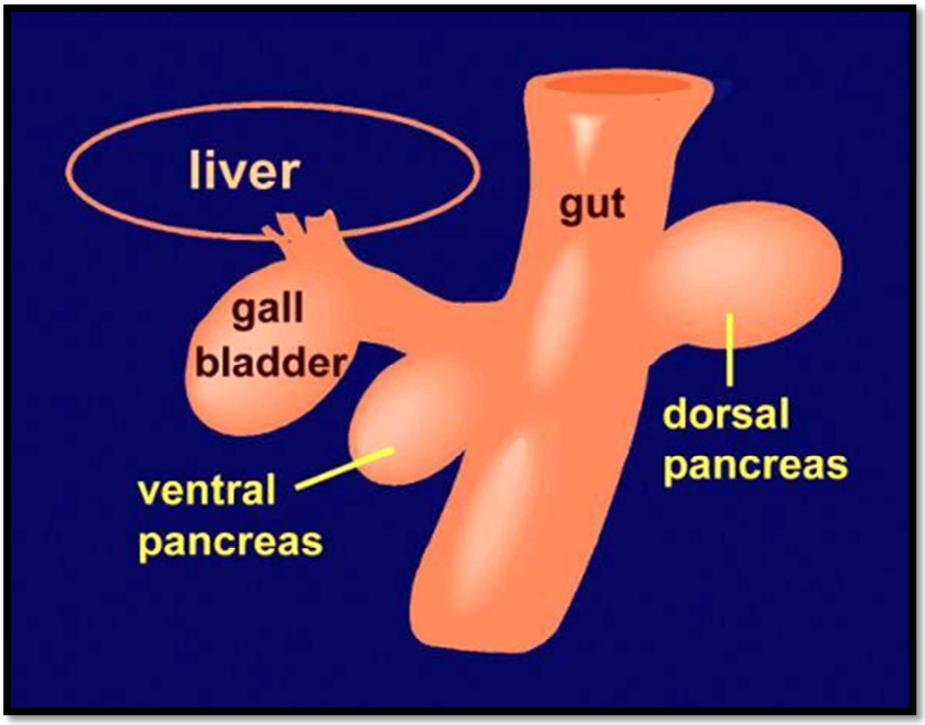

Figure 3: Early development of pancreas

Ductal system of pancreas

Each pancreatic lobule typically contains between 20 and 200 acinar cells that discharge into smaller intercalated ducts. These intercalated ducts join to form intralobular ducts, which then combine to create secondary pancreatic ducts that eventually empty into the main pancreatic duct (MPD). Over time, the diameter of the MPD increases, measuring approximately 1-2mm in the tail, 2-3mm in the body, and 3-4mm in the head of the pancreas. The uncinate process has its own duct, which also connects to the MPD.[5]

Pancreatic enzymes are secreted through two ducts: the Wirsung duct (main pancreatic duct) and the Santorini duct (accessory pancreatic duct). The ampulla of Vater allows the major pancreatic duct, which is joined by the ventral duct, to open into the duodenum.The dorsal duct contributes to both main pancreatic duct and minor or accessory ducts of Santorini. Although the minor ducts usually empty into the ampulla of Vater, in about 5% of people, they can drain independently.

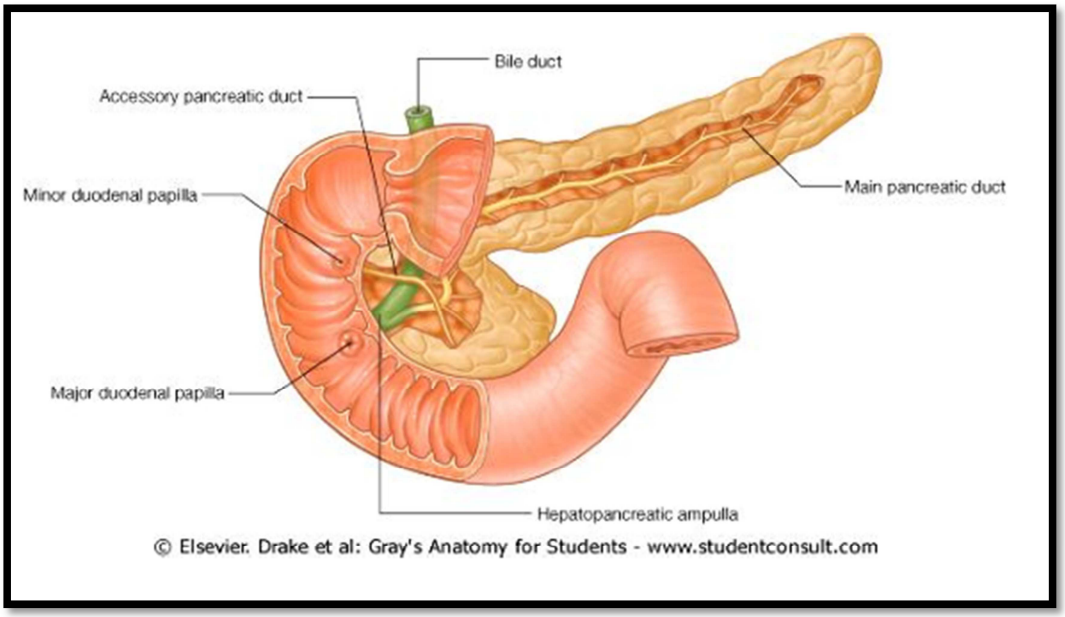

Figure 4: Ductal system of pancreas

Dimensions: The pancreas generally has a length ranging from 12 to 15 cm (4.7 to 5.9 inches).

Width: The width of the pancreas varies across its length. The widest section, the head, typically measures between 5 and 8 cm (2 to 3 inches). In contrast, the body and tail are slimmer, with widths of about 2 to 3 cm (0.8 to 1.2 inches).

Thickness: The thickness of the pancreas also differs, with the head being thicker than the body and tail. The head's thickness is approximately 2 to 3 cm (0.8 to 1.2 inches), while the body and tail are thinner, usually measuring around 1 to 2 cm (0.4 to 0.8 inches).

Weight: In adults, the pancreas weighs on average between 70 and 100 grams (2.5 to 3.5 ounces).

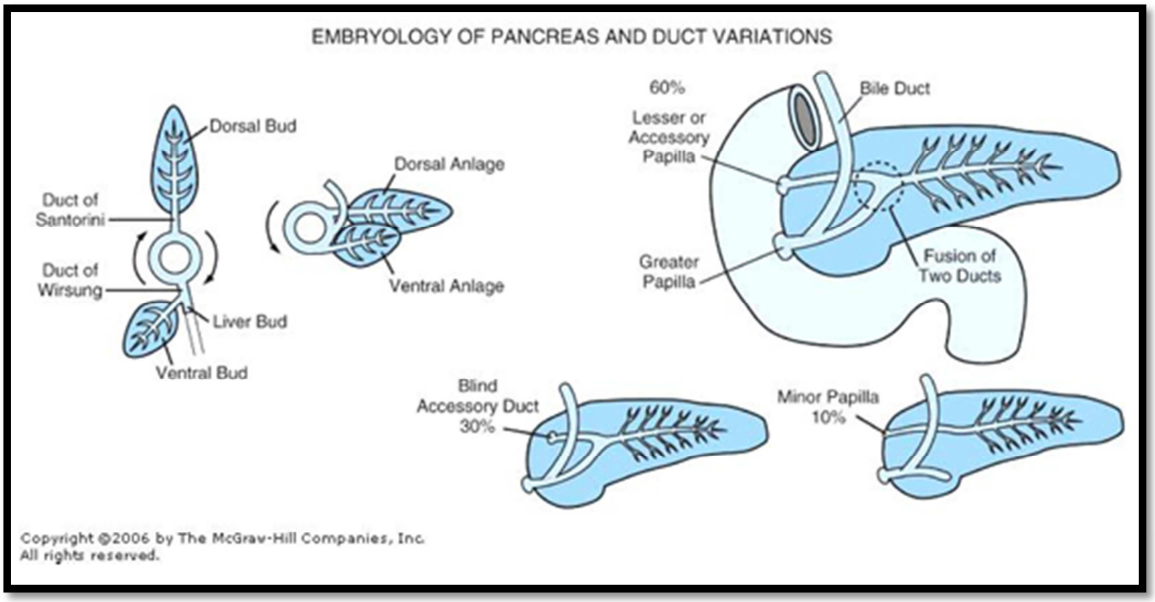

Figure 5: Ductal variation

Blood and lymphatics supply

Arteries of pancreas

The pancreas receives its blood supply from the common hepatic artery, superior mesentric artery (SMA), and splenic artery, which is a branch of the celiac trunk. [6,7]

Body and tail: splenic artery & branches

Pancreatic head: gastroduodenal artery - a branch of common hepatic artery, inferior pancreaticoduodenal artery - a branch from superior mesenteric artery.

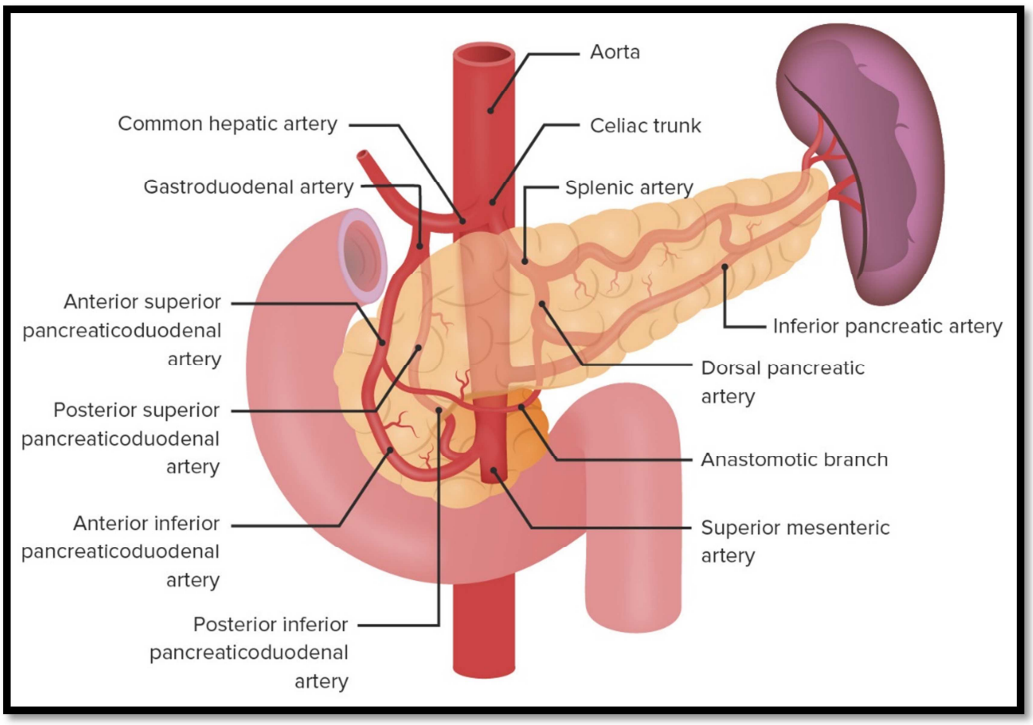

Figure 6: Arteries of pancreas

Venous supply:

Body and neck: by splenic vein drainage

Pancreatic head: into superior mesenteric vein (S M V)

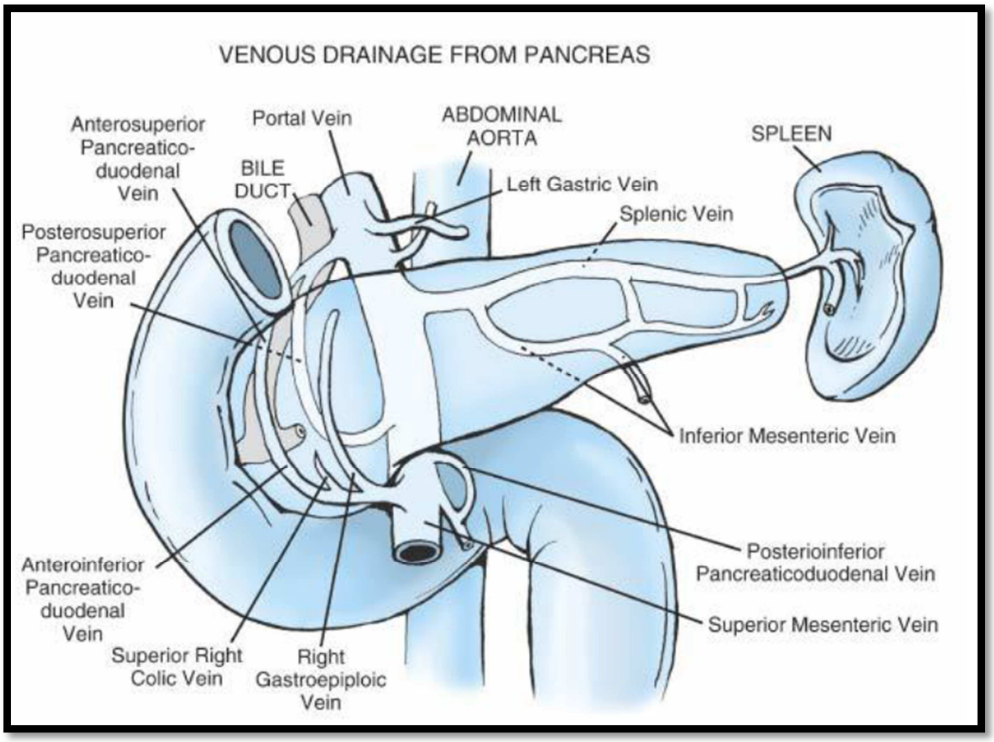

Figure 7: Venous drainage of pancreas

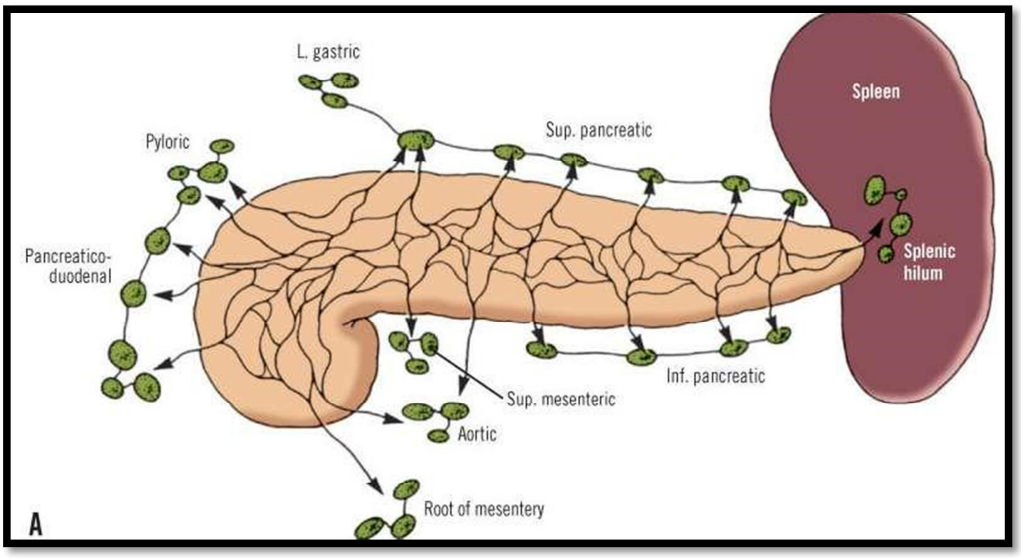

Figure 8: Lymphatic drain of pancreas

Nerve:

Sensory, parasympathetic, and sympathetic nerves form an intricate network that innervates the pancreas.[8] Moreover, the pancreas houses an intrinsic nerve network. Pancreatic acinar cells receive both sympathetic and parasympathetic innervation. Parasympathetic fibers, originating from the posterior vagal trunk, mainly control secretion. Hormones generated in the duodenum and proximal intestinal mucosa, such as cholecystokinin and secretin, as well as stomach acid, are the main factors controlling pancreatic secretions. The celiac plexus and the T6–T10 thoracic splanchnic nerves innervate the sympathetic nervous system.

Physiological variants

- Partial or dorsal agenesis
- Ectopic pancreatic tissue (3-5%)
- Pancreas divisum
- Pancreatic dysfunction or dysgenesis

PANCREATITIS

Acute pancreatitis is a hazardous response to pancreatic injury, but chronic pancreatitis can cause long-term harm to the organ's structure and function, impacting its endocrine and exocrine functions. In the United States, acute pancreatitis leads to approximately 200,000 hospitalizations annually, and this number is increasing.[11]

Etiology

In the US, the leading causes of acute pancreatitis are gallstones (which account for 35–40% of cases) and alcohol usage (30–40% of cases). Nevertheless, the etiology is complex and includes things like hypertriglyceridemia, pancreatic duct

injuries, genetic factors (e.g., PRSS1 gain-of-function mutations, CFTR, and SPINK1 gene mutations), post-endoscopic retrograde cholangiopancreatography (ERCP), and autoimmune pancreatitis. In chronic pancreatitis, alcohol consumption is the major cause, while smoking is also a significant risk factor.

Epidemiology

Every year, acute pancreatitis causes around 275,000 hospital admissions. Eighty percent of people with hospitalized pancreatitis have just moderate symptoms and can go home in a few days. Two percent of cases of acute pancreatitis result in death overall.[11] The recurrence rate of acute pancreatitis varies between 0.6percent and 5.6percent, depending on the underlying cause. Alcohol-induced pancreatitis has the highest rate of recurrence.[12]

The annual incidence of chronic pancreatitis is between 5 and 12 cases per 100,000 individuals. It impacts 50 out of every 1Lakh people. The most common age group affected is 30 to 40 years, and males are more frequently affected than females.[13]

Pathophysiology

It can result from two primary mechanisms: injury to the pancreatic duct and acinar cells. In this situation, a lack of adequate release of pancreatic enzymes by pancreas causes self-digestion and inflammation of the pancreatic tissue. Alcohol can trigger acute pancreatitis through both direct toxic effects and immune system responses.[14] Gallstones can cause a temporary blockage of the pancreatic duct, which is thought to be the mechanism behind pancreatitis induced by ERCP. After a series

of acute episodes, this can progress to chronic pancreatitis, marked by inflammation and scarring in the pancreas, leading to pancreatic insufficiency over time.

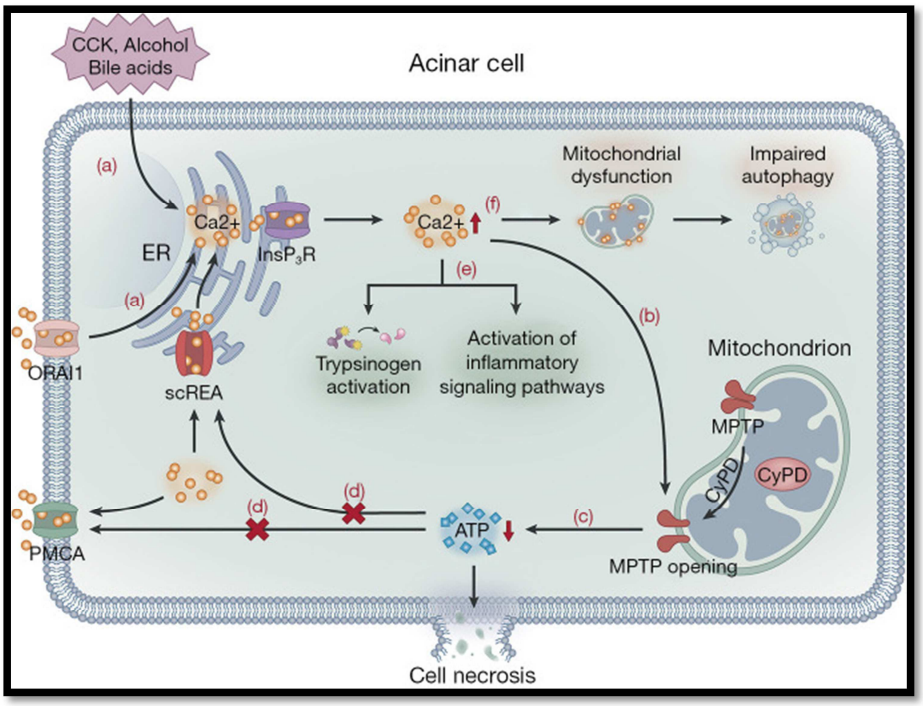

Figure 9: Pathomechanism of acute pancreatitis[15]

Histopathology

The degree of the disease, in particular whether or not necrosis is present, dictates the histologic points in acute instances. The condition can be categorized as:

- Interstitial or edematous pancreatitis
- Hemorrhagic necrotizing pancreatitis

In AP, the pancreas may show signs of edema, necrosis, and fat stranding. Fat necrosis is minimal in mild cases but more extensive in severe cases, often accompanied by tissue damage and localized bleeding. Mononuclear infiltrates, fibrosis, and the possibility of pancreatic calcifications are the hallmarks of chronic pancreatitis.

Symptoms

Acute pancreatitis usually manifests as intense abdominal pain starting in the epigastric area and spreading to the back, often described as sharp and accompanied by nausea and vomiting.

A comprehensive social history, including alcohol consumption patterns, should be obtained. Details of recent medical procedures like ERCP and the current medication regimen should also be recorded. Additionally, patients should be questioned about any family history of pancreatic conditions.

The initial assessment should include checking vital signs (pulse, BP, RR, and temperature) to evaluate hydration levels. Jaundice might indicate biliary obstruction, while an ileus calls for an abdominal examination to assess abdominal tenderness, guarding, and reduced bowel sounds. Ecchymosis in the flanks may suggest Grey-Turner sign, and bruising around the umbilicus could indicate Cullen's sign, both potentially indicating pancreatic necrosis resulting in intra-abdominal bleeding.[16] A patient suffering from severe pancreatitis may also experience alterations in their mental state. While acute pancreatitis commonly leads to nausea, abdominal pain, and vomiting, it can sometimes be pain-free, with patients possibly showing symptoms like steatorrhea (fat in the stool) and weight loss.

Evaluation

According to the latest guidelines from the American College of Gastroenterology (ACG), diagnosis of acute cases require the presence of at least two of the following criteria: pain abdomen consistent like acute pancreatitis, a serum lipase level greater than three times the typical upper limit, and imaging results

indicating acute pancreatitis. Additionally, the guidelines recommend against routine imaging, such as computed tomography (CT) scans/ magnetic resonance imaging (MRI), for all patients at the initial diagnosis. Imaging should be reserved for cases where symptoms persist or when the diagnosis remains unclear after two to three days of hospitalization.[17]

Upon admitting a patient with acute pancreatitis, it's crucial to assess the initial severity of the condition, paying close attention to signs of organ failure, such as respiratory, cardiovascular, or renal dysfunction. Patients can be classified as having mild, moderately severe, or severe acute pancreatitis based on the updated Atlanta classification.[18]

It is advised to request testing for serum lipase, lactate, triglycerides, complete blood counts, comprehensive metabolic panels, and C-reactive protein (CRP) upon admission. Monitoring for rising hematocrit or blood urea nitrogen (BUN) levels can help determine how acute pancreatitis is progressing. Moreover, the patient's clinical status can be evaluated using the systemic inflammatory response syndrome criteria (SIRS).[19]

In patients with chronic pancreatitis, amylase and lipase levels may be elevated or fall within normal range. Additional tests, such as fecal fat analysis, stool elastase, and alpha-1-antitrypsin levels, can offer further diagnostic information. Assessing HbA1c levels can help identify potential endocrine dysfunction related to chronic pancreatitis.

Diagnostic testing: Amylase has traditionally been a key marker for diagnosing acute pancreatitis and can be measured through blood or urine tests, including clean catch or 24-hour collection methods. Normal values can vary between

laboratories, making it important to differentiate pancreatic amylase from other isoforms. If amylase levels are elevated while lipase levels remain normal, it suggests a non-pancreatic origin of the condition. The lipase-to-amylase ratio can help distinguish between gallstone-induced and alcoholic pancreatitis, as gallstones typically cause a greater increase in amylase levels, while alcohol consumption leads to a higher rise in lipase levels. Ratios above 2 or 5 indicate alcoholic pancreatitis with high specificity. Elevated alanine transaminase levels are particularly specific for diagnosing gallstone-induced pancreatitis. Although combining amylase and lipase tests can improve specificity, it does not significantly increase sensitivity.[20,21]

Differential diagnosis

The differential diagnosis of acute pancreatitis includes,

- Peptic ulcer disease
- Perforated viscus
- Cholecystitis
- Intestinal obstruction
- Acute mesenteric ischemia
- Choledocholithiasis

Differential diagnosis for chronic pancreatitis includes

- Chronic mesenteric ischemia
- Pancreatic malignancy
- Acute recurrent pancreatitis

Complication of pancreatitis include

Local complications

- Walled off necrosis (>4wks)
- Peri-pancreatic fluid collection
- Pancreatic pseudocyst (>4wks)
- Acute necrotic collection (<4wks)

Systemic complication includes;

- Compartment syndrome
- Acute kidney injury (AKI)
- Acute respiratory distress syndrome
- Disseminated intravascular coagulation

Calcium metabolism

Calcium plays a crucial role in the body, participating in numerous biochemical processes. It's vital for heart health, maintaining bone structure, muscle function, and serves as a signaling molecule in various biochemical pathways. The levels of calcium in the blood are carefully regulated by hormones like parathyroid hormone (PTH), calcitonin, and calcitriol to ensure stability.

The body needs to obtain calcium through dietary sources, and its absorption in the digestive tract is affected by hormones like PTH and calcitriol. [22]

Blood calcium levels can be assessed through a venous blood sample, with normal ranges being “8.8 mg/dL to 10.4 mg/dL for total calcium and 4.7 mg/dL to 5.2 mg/dL for ionized calcium’. Total level of calcium should be adjusted for albumin

levels, which can influence the results due to its role as carrier molecule. Calcium levels can also be determined from urine, using measures like calcium concentration, the calcium to creatinine ratio (UCa: UCr), or fractional excretion of calcium (FeCa). Imbalances in calcium levels can arise from various diseases or treatments that impact hormone production, receptor sensitivity, absorption in the gut, and kidney function. It's important to note that inaccuracies in reported calcium values can happen because of laboratory errors, highlighting the need for careful specimen handling, sampling procedures.[24,25]

Etiology

Calcium ranks as the fifth most abundant element in the human body and is the most prevalent cation. A typical person weighing 70 kg carries around 1 kg of calcium, roughly equivalent to 25 mol. The vast majority, about 99percent, is found in the skeleton, mostly as extracellular crystals with a composition similar to hydroxyapatite. Soft tissues and extracellular fluid hold the remaining 1percent of the body's calcium. In the bloodstream, nearly all calcium is found in the plasma.[24,26]

In the circulation, roughly 50% of calcium exists in its free form, known as ionized calcium. Another 40percent is bound to proteins, predominantly to albumin (80percent) and to a lesser extent, globulins (20percent). The remaining 10 percent is made up of other tiny, diffusible organic and inorganic anions, such as citrate, lactate, and bicarbonate. There is biological activity in the free calcium fraction. The pH scale affects how calcium binds to proteins. In contrast to acidosis, which decreases binding and increases free calcium, alkalosis increases the negative charge on proteins, improving binding and decreasing free calcium. In lab conditions, serum-free calcium concentration varies by around 0.2 mg/dL (0.05 mmol/L) for every 0.1 unit variation in pH. Changes in protein and anion concentrations, pH, or free and total calcium

levels can lead to the redistribution of calcium among the three plasma pools, either acutely or over time.[27–30]

Pathophysiology

Calcium is crucial for various cellular functions and is tightly regulated within the body to maintain homeostasis. Key hormones like parathyroid hormone (PTH), calcitonin, and calcitriol play roles in managing calcium levels.[29]

PTH increases calcium absorption in the gut by stimulating calcitriol production, promotes calcium release from bones by activating osteoclasts, and boosts calcium reabsorption in the kidneys. Calcium-sensing receptors in the parathyroid gland monitor serum calcium levels, ensuring balanced PTH secretion. Mutations in these receptors can lead to calcium level imbalances.[31–33]

Calcitonin, produced by the thyroid gland, lowers serum calcium by inhibiting osteoclast activity and reducing calcium absorption in the intestines and kidneys.

In the renal system, PTH enhances calcium reabsorption and phosphate excretion. In chronic kidney disease, PTH's effectiveness can decrease, leading to imbalances.[34]

In the gastrointestinal system, calcium absorption depends on calcitriol levels. Factors like age, gender, and dietary compounds can influence calcium absorption rates.[31]

In the musculoskeletal system, calcium stored in bones as hydroxyapatite is released based on serum calcium levels. PTH and calcitonin influence bone remodeling, affecting bone strength and density.[34,35] Muscle contraction relies on

calcium's role in activating proteins like troponin, facilitating actin-myosin interaction.

In the cardiovascular system, calcium stabilizes heart muscle cell membranes and influences myocardial contractility. Dysregulation can lead to arrhythmias, and calcium supplements' cardiovascular risks remain inconclusive, warranting further research.[36–38]

Additionally, calcium acts as a cofactor in blood coagulation. It's essential for platelet adhesion and various steps in the coagulation cascade. Low calcium levels can contribute to coagulopathy and increased bleeding risk. Overall, calcium's regulatory mechanisms and its multifaceted roles highlight its significance in maintaining optimal health.[39]

Acute pancreatitis (AP) is a common gastrointestinal disorder characterized by inflammation of the pancreas, with varying degrees of severity ranging from mild to severe. The early prediction of disease severity and clinical outcomes is essential for optimal management and resource allocation in clinical settings. The ratio of serum calcium to serum lactate can serve as a potential biomarker for predicting the severity and clinical outcome of acute pancreatitis.

Serum Calcium and Acute Pancreatitis

Calcium plays a crucial role in various physiological processes, including cell signaling, muscle contraction, and enzyme regulation. In acute pancreatitis, alterations in calcium homeostasis have been observed, with hypocalcemia being a common finding, particularly in severe cases.[40] Hypocalcemia in acute pancreatitis can result

from calcium sequestration in damaged pancreatic tissue, impaired parathyroid hormone secretion, or alterations in vitamin D metabolism.[41]

Serum Lactate and Acute Pancreatitis

Lactate is a by-product of anaerobic metabolism and serves as a marker for tissue hypoperfusion and hypoxia. Elevated serum lactate levels have been associated with tissue ischemia and organ dysfunction in various critical illnesses, including acute pancreatitis.[42] In AP, elevated lactate levels may reflect the severity of pancreatic necrosis, systemic inflammatory response, and multiorgan failure.[43]

Serum Calcium and Serum Lactate Ratio in Acute Pancreatitis

The ratio of serum calcium to serum lactate has been proposed as a potential prognostic marker in acute pancreatitis. A lower calcium-to-lactate ratio may indicate a more severe disease course, with increased inflammation, tissue damage, and organ dysfunction.[44] This ratio can be used in predicting the severity, clinical outcome, and hospital stay duration in patients with acute pancreatitis rather than only a single parameter Serum Calcium or Serum Lactate. This ratio includes 2 parameters and no measurement units makes it easier and reliable.

Clinical Implications

Early identification of patients at risk of developing severe acute pancreatitis can guide treatment decisions, including intensive care unit (ICU) admission, fluid resuscitation, and nutritional support.[17] The serum calcium-to-lactate ratio offers a simple, cost-effective, and readily available biomarker that can aid clinicians in risk stratification and prognostication.[46]

The serum calcium-to-lactate ratio in acute pancreatitis holds promise as a valuable tool for predicting disease severity, clinical outcomes, and hospital stay duration. Further research is needed to validate its utility in diverse patient populations and healthcare settings. Nonetheless, its potential to enhance risk stratification and inform clinical decision-making underscores its importance in the management of acute pancreatitis.

Various articles discussing the serum calcium and serum lactate ratio in acute pancreatitis

Overview of Serum Calcium:

The range of normal serum calcium is 2.1–2.6 mmol/L (8.5–10.5 mg/dL). Serum calcium exists in two forms: ionized (iCa) and nonionized (ionized when attached to albumin or anions such as citrate, bicarbonate, and phosphate). Ionized calcium is physiologically active. Direct iCa measurement is more accurate and is now favored over "corrected calcium" estimations based on total calcium. Studies on animals have shown that hypocalcemia is a poor prognostic sign and is frequently observed in people in critical care, especially in those with severe acute pancreatitis.

In a study conducted by Ahmed A et al., (2016) to assess the presence of hypocalcemia in acute pancreatitis. Hypocalcemia is commonly observed in cases of acute pancreatitis. Severe hypocalcemia can lead to both neurological and cardiovascular symptoms. The treatment of hypocalcemia through intravenous calcium infusion is a topic of debate, given that excessive intracellular calcium is a key factor in acinar cell damage during pancreatitis. This article discusses the methods and considerations for correcting calcium levels in patients with pancreatitis.[1]

In a study conducted by Peng T et al., (2017) to assess the serum calcium as indicator of persistent organ failure in acute pancreatitis. Of the 128 consecutive patients with acute pancreatitis (AP) included in the research, 29 experienced persistent organ failure (POF). Upon admission, the blood calcium level of patients with POF was substantially lower than that of patients without POF. With a hazard ratio of 0.21, serum calcium was found to be an independent risk factor for POF following multivariate logistic analysis. With a sensitivity of 89.7%, specificity of 74.8%, and area under the curve (AUC) of 0.888, POF was predicted by a blood calcium level of 1.97 mmol/L. The results imply that blood calcium levels at admission are associated with POF in AP patients independently and may be used as a predictor of prognosis. [47]

In a prospective study conducted by Pokharel A et al., (2017) to assess the prediction of severity of acute pancreatitis using total calcium and albumin corrected calcium. The study involved 80 patients with acute pancreatitis (AP). Concluded that the total calcium (TC) and adjusted calcium concentration (ACC) measured within the initial 24 hours serve as valuable indicators for predicting the severity of acute pancreatitis. [2]

In a study conducted by Edakkepuram U et al., (2017) to assess the total serum calcium and corrected calcium in severity of acute pancreatitis. Acute pancreatitis typically affects individuals aged 30-50. It is more prevalent in men, accounting for 80% of cases, compared to 20% in women. Alcohol consumption is the leading cause, contributing to 58% of acute pancreatitis cases, while gallstones account for 28%. Hypocalcemia and low adjusted calcium concentration (ACC) can help predict the severity of acute pancreatitis similarly to the BISAP score. However, these indicators do not outperform the BISAP score in predicting severity. [3]

In a study conducted by Thakur A et al., (2020) to assess the role of total and corrected calcium in predicting severity of acute pancreatitis with CT findings. Analysis of the data revealed a statistically significant negative connection between serum calcium and the CT severity index (CTSI). But there was no meaningful association found between amylase and CTSI. Correlation between adjusted calcium and CTSI was found to be significantly negative and significant. Additionally, there was a strong association between CTSI and serum albumin. On the other hand, lipase did not show any significant connection ($p \geq 0.05$). It was shown that total calcium and adjusted calcium could provide more accurate substitute indicators for determining the severity of a disease. Total calcium and adjusted calcium both showed a highly significant negative predictive value for ruling out the severity of acute pancreatitis and a good positive predictive value for suggesting disease progression.[48]

In a study conducted by Shu W et al., (2020) to assess the elevated lactate as independent predictor of poor outcome in acute pancreatitis. Patients with increased arterial lactate had considerably greater chances of multiple chronic organ failure, death, septic shock, pancreatic infection, abdominal compartment syndrome, pancreatic necrosis, and needing ventilator support when compared to those with normal levels. An elevated hazard ratio of 10 and the highest area under the curve, 0.78, were shown to be highly correlated with death when arterial lactate levels were ≥4 mmol/L. According to these results, patients with severe acute pancreatitis may benefit from early identification of high-risk individuals due to the independent correlation between increased arterial lactate levels at the outset and unfavorable outcomes, including death. [4]

In a study conducted by Doganay F et al., (2022) to assess the predictive performance of lactate as mortality predictor in acute pancreatitis. A total of 147

patients participated in the study, with a median age of 65 years (range 50-76). Among them, 91 were female (61.9%) and 56 were male (38.1%). The predictive value of lactate for 30-day mortality was assessed using ROC analysis. The analysis revealed a sensitivity of 82.61%, specificity of 79.84%, positive predictive value (PPV) of 43.2%, negative predictive value (NPV) of 96.1%, an area under the curve (AUC) of 0.821, and a Youden's J Index (YJI) value of 0.6245. Early identification of acute pancreatitis (AP) patients and prompt initiation of appropriate treatment can significantly reduce morbidity and mortality. Based on the findings, elevated lactate levels measured upon admission can serve as a valuable, quick, and straightforward method for predicting mortality in AP patients. [49]

In a study conducted by Zeng Z et al., (2024) to assess the serum lactate as indicator for short term and long term mortality in patients presenting with acute pancreatitis. A study included 895 acute pancreatitis (AP) patients admitted to the ICU. The serum lactate cut-off value predicting mortality at 30 days, 180 days, and 1 year in AP was determined to be 2.4 mmol/L. Thus, serum lactate levels can serve as indicators for both short-term and long-term mortality in AP patients admitted to the ICU. [50]

MATERIALS AND METHODS

Source of Data: The source of data were patients with acute pancreatitis admitted at KAHER'S Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Nehru Nagar, Belagavi and KLES Dr. Prabhakar Kore Hospital and Medical Research Centre.

Study Design: Prospective Observational Study

Study Period: The study was conducted from 1st January 2023 to 31st December 2023

Sample Size: Formula used for sample size calculation is,

$$n = \frac{Se\hat{} \, (100 - Se\hat{}) \, Z_{\propto/2}{}^2}{d^2 \, P}$$

where n is the sample size required, P is the occurrence of a state or condition (prevalence), $Se\hat{}$ is sensitivity, d is the maximum error required, $Z_{\propto/2}$ is the value corresponding to level of confidence required. TC of 8.20 mg/dl was computed as cutoff for severity of acute pancreatitis with sensitivity of 96%. Considering this result, at 95% confidence level, 5% maximum error and assuming the prevalence to be 30%, the sample size is given by,

$$n = \frac{0.96 \text{ x } (1 - 0.96) \text{ x } 1.96^2}{0.05^2 \text{x } 0.3}$$

$$n = 49.17248 \approx 49.$$

Hence, minimum sample size required in the study is 49. As sample size increases, accuracy of result increases.

Reference: Pokharel, A., Sigdel, P. R., Phuyal, S., Kansakar, P. B. S., & Vaidya, P. (2017). Prediction of Severity of Acute Pancreatitis Using Total Serum Calcium and Albumin-Corrected Calcium: A Prospective Study in Tertiary Center Hospital in Nepal. Surgery Research and Practice, 2017, 1–5.

Sampling technique: Convenient sampling

Inclusion Criteria: Patients willing to give written and informed consent, of either sex, of age group 21- 60 years and diagnosed as acute pancreatitis. All patients with acute pancreatitis who are admitted with in 72 hours of symptoms.

The diagnosis of acute pancreatitis should two of the following 3 features:

1. Upper abdominal pain of acute onset often radiating to the back.
2. Serum amylase or lipase activity greater than 3 times normal value, and
3. Findings on Abdominal imaging consistent with Acute Pancreatitis

Exclusion Criteria: Age< 21 years,

Pregnant and lactating females,

Patients in sepsis due to any other cause other than acute pancreatitis ruled out by detailed history, clinical examination, investigations.

Study protocol: After obtaining institutional ethical committee clearance and written informed consent, confirming the diagnosis of acute pancreatitis using thorough history and examination findings and necessary investigations, serum amylase, serum lipase, USG Abdomen/CECT abdomen for confirmation, Routine Blood investigations- Complete Blood counts, Renal Function Tests and Liver Function Tests, Classifying the subjects using Revised Atlanta Classification, then taking Ratio

of serum calcium and lactate from ABG (measured in mmol/L), then analyzing ratios of all the subjects at the time of admission, following up the course of hospital stay and at the time of discharge, correlating the ratio with severity of disease and duration of hospital stay.

Data collection procedure: After obtaining institutional ethical committee clearance and written informed consent, thorough history and examination findings are obtained. Following clinical and demographic data were collected name, sex, age, clinical history, alcohol consumption history, serum amylase, serum lipase, USG Abdomen/CECT abdomen for confirmation, other necessary investigations, Routine Blood investigations- Complete Blood counts, Renal Function Tests and Liver Function Tests, then measuring of serum calcium and lactate in mmol/L from ABG (Arterial Blood Analysis) at the time of admission or upto 24 hours of admission.

Does the study require any investigations or interventions to be conducted on patients or other humans or animals? If so, please describe briefly:

Yes. Investigations to be done on patients- Serum Amylase, Serum Lipase, ABG(Arterial Blood Gas) for Serum Calcium and Serum Lactate (in mmol/L), Abdominal Imaging studies, Routine Blood investigations- Complete Blood counts, Renal Function Tests and Liver Function Tests.

STATISTICAL ANALYSIS

Data were “analyzed using statistical software R version 4.2.0 and Microsoft Excel. Categorical variables were represented by frequencies and percentages. Continuous variables were represented by Mean ± SD / Median (Min, Max) form. Chi-Square test was used to check the association between categorical variables. Normality of variable is checked by Shapiro Wilk test and QQ plot. Two sample t test/Mann Whitney U test was used to compare means/distributions of variables between the groups. Applicability of serum calcium serum lactate ratio to severity of AP is checked by Logistic regression and Receiver Operating Characteristic (ROC) curves. Cut off values are obtained by simultaneously maximizing sensitivity and specificity.” P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

In present study total of 49 patients fulfilling inclusion criteria are included with mean age of 39yrs.

Table 1: Showing mean age of the patients

	Mean	**SD**
Age in years	39.0	11.8

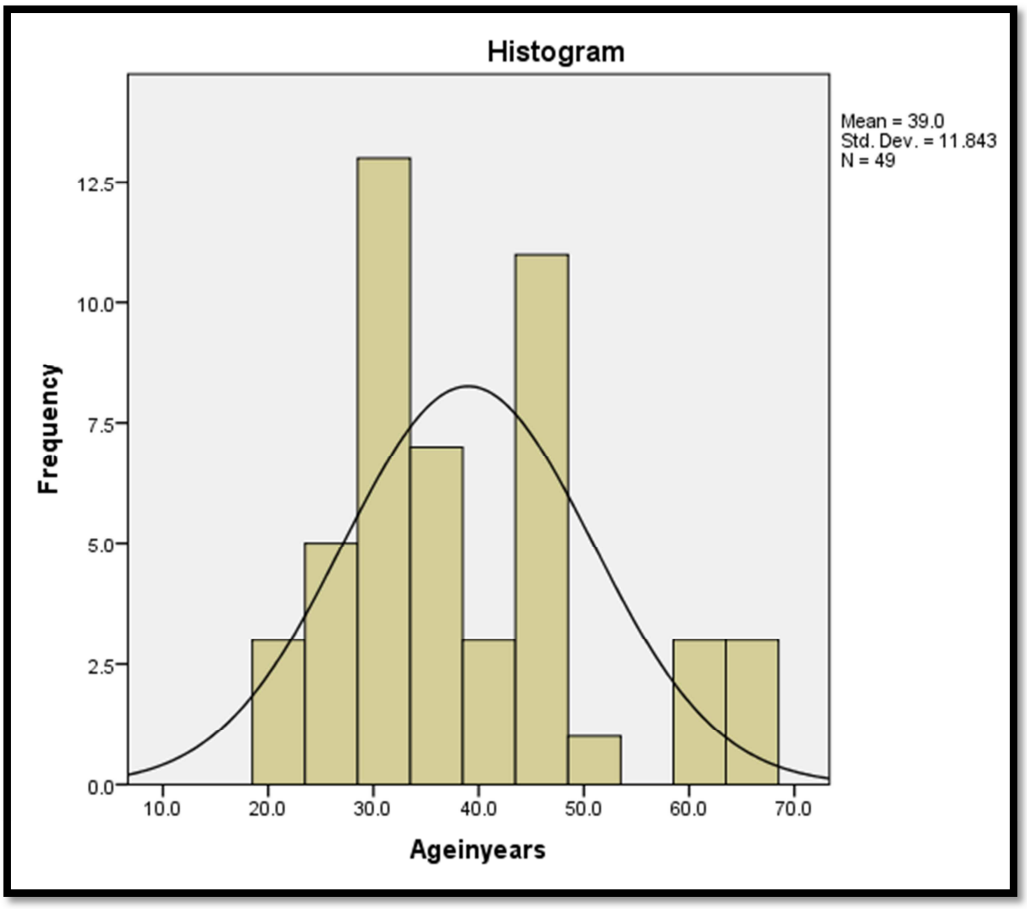

Figure 10: Showing mean age of the patients

Table 2: Showing distribution of gender of patients

		Count	N %
Gender	Female	7	14.3%
	Male	42	85.7%

Among included patients, 85.7% were male and 14.3% were female patients, with male preponderance.

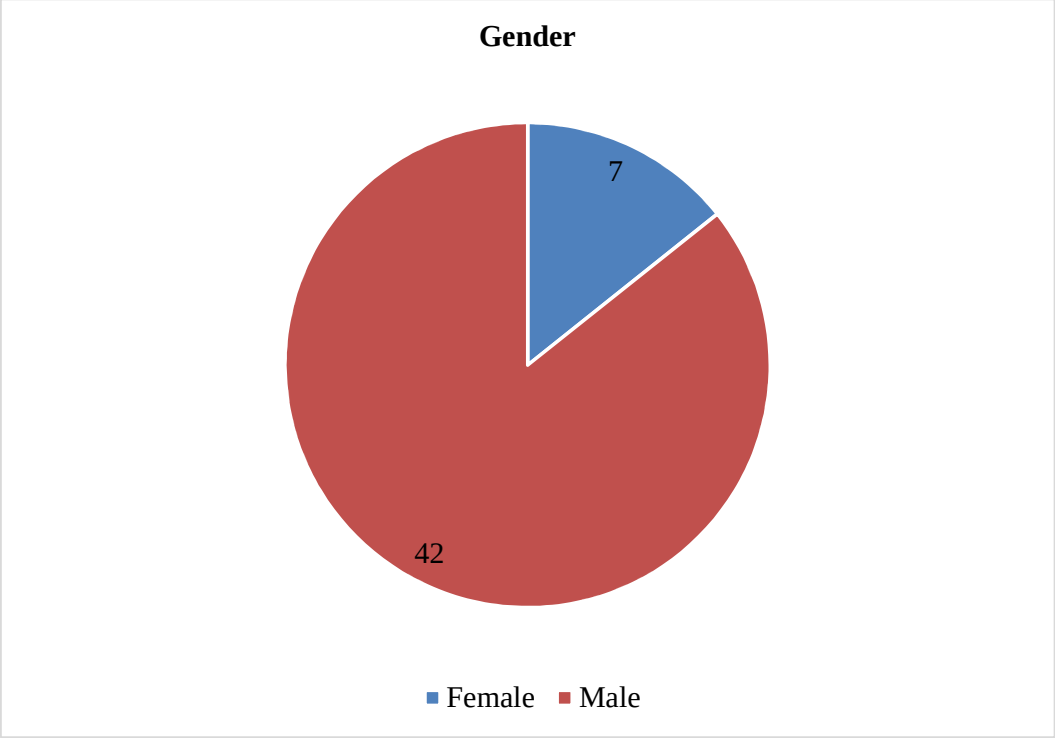

Figure 11: Showing distribution of gender of patients

Table 3: Showing presence of comorbidities

		Count	N %
Comorbidities	Diabetes Mellitus	9	18.3%
	Myocarditis	1	2 %
	Hypertension	8	16.32%
	DVT	1	2 %
	Dorsal Agenesis	1	2 %
	Pancreatic divisum	1	2 %
	Nil comorbidities	28	57.1%

Among the patients, 42.9% of the patients had comorbidities, with diabetes and hypertension being the most common.

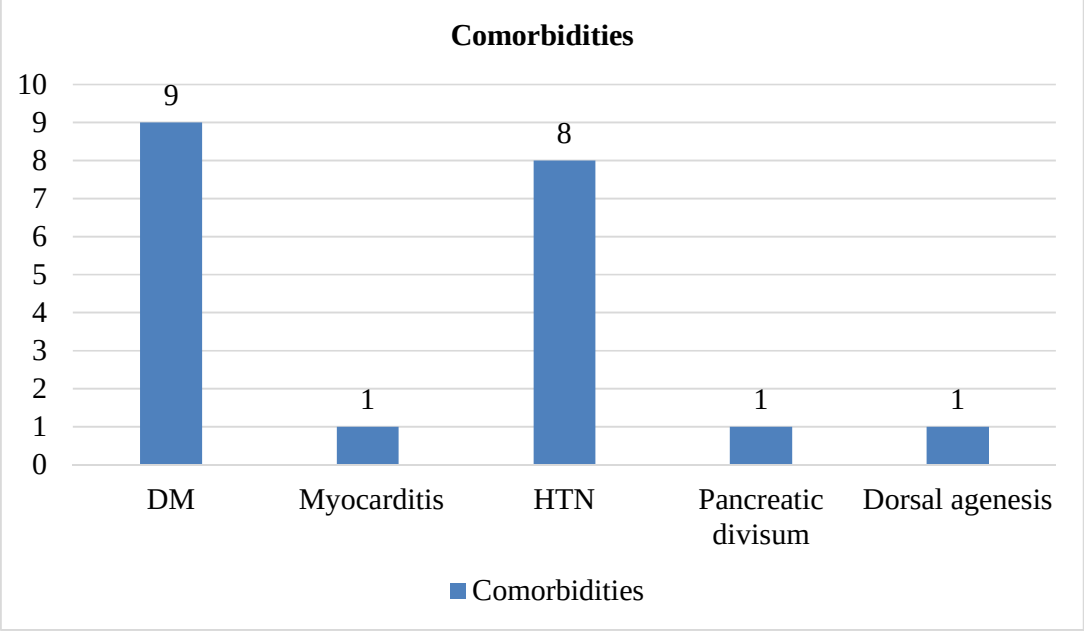

Figure 12: Number of patients with comorbidities

Table 4: Showing mean years of alcohol consumption

Alcohol Intake	Frequency(N=49)	Percent
<2yrs	8	**19.5 %**
2-5yrs	7	**17.1%**
6-15yrs	22	**53.6%**
>15yrs	4	**9.8%**
Alcohol Intake Mean = 8.8 years		**SD = 6.9**
Non Alcoholic	**8 patients**	**16.32 %**

Out of 49 subjects, 8(16.32 %) were Non-alcoholic and 41(83.67 %) were Alcoholic. The mean alcohol intake was found to be 8.8 years among the Alcoholics.

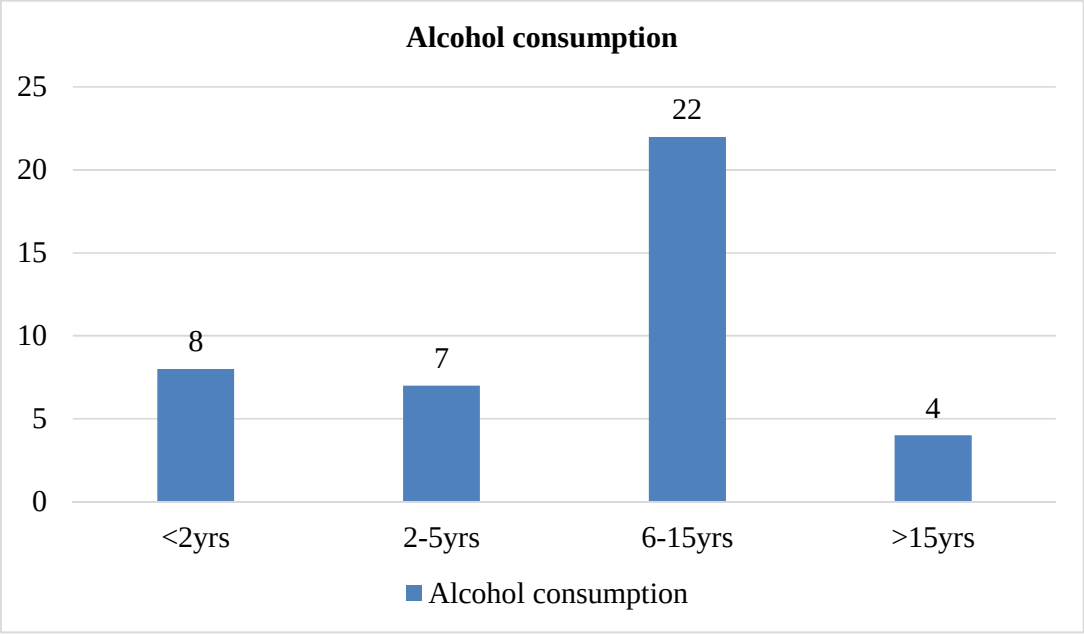

Figure 13: Showing mean years of alcohol consumption

Table 5: Showing mean level of amylase and lipase among patients

Test	**Mean** (U/L)	**SD**
Serum Amylase (U/L)	869.0	741.4
Serum Lipase(U/L)	1170.1	960.9

The mean serum amylase (in U/L) was found to be 869.0±74.1 and lipase (in U/L) was 1170.1±960.9.

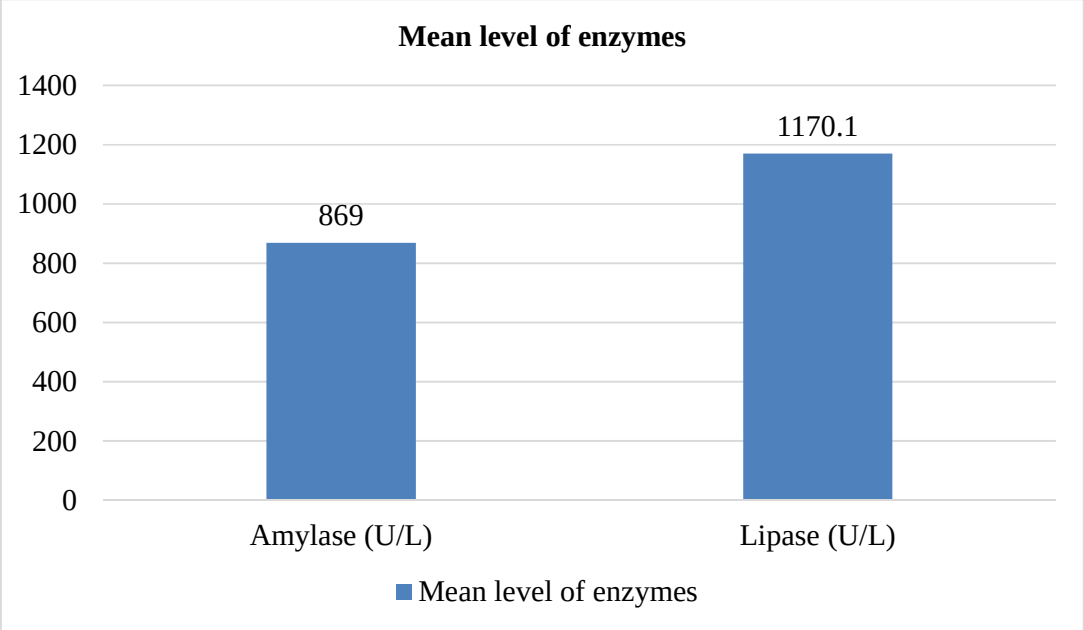

Figure 14: Showing mean level of amylase (in U/L) and lipase(in U/L) among all patients

Table 6: Showing mean level of serum calcium, lactate and serum calcium lactate ratio among patients

	Mean	SD
Serum Calcium(mmol/L)	0.794	0.332
Serum Lactate(mmol/L)	2.040	1.253
Ratio	0.530	0.363

The table provides the mean levels and standard deviations (SD) of serum calcium, serum lactate, and the serum calcium-to-lactate ratio among the patients studied.The average Serum calcium is 0.794 mmol/L, and the average serum lactate level is 2.040 mmol/L.

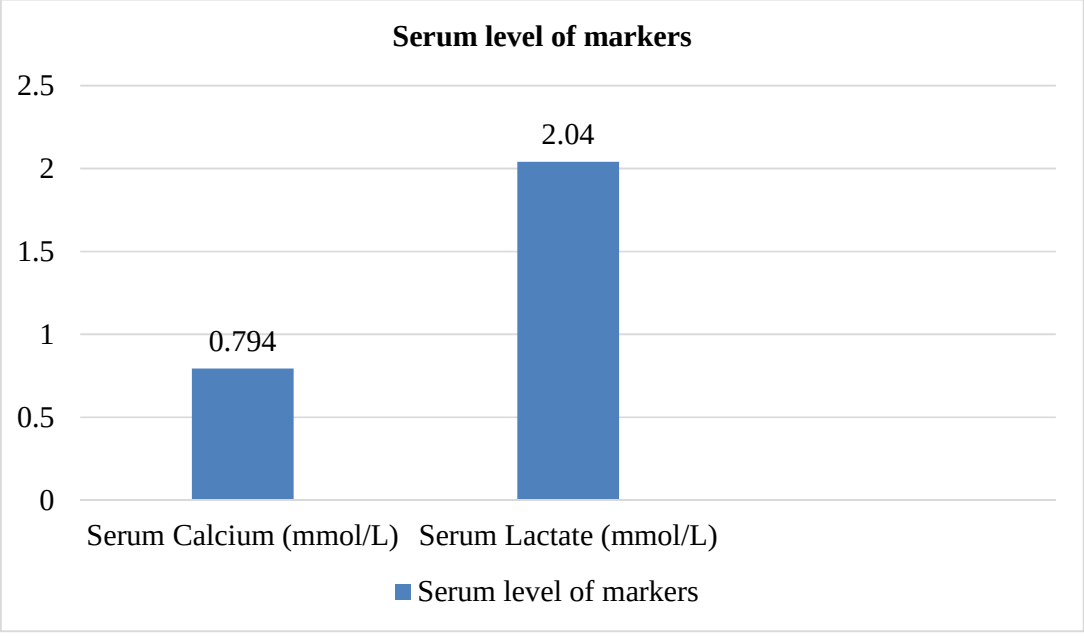

Figure 15: Showing mean level of serum calcium(mmol/L), lactate(mmol/L) and serum calcium lactate ratio among study patients

Table 7: Showing distribution of severity of pancreatitis

		Count	N %
Severity of Pancreatitis	Mild Pancreatitis	17	34.7%
	Moderate Pancreatitis	17	34.7%
	Severe Pancreatitis	15	30.6%

Among the patients, 34.7% of the patient presented with mild and moderate pancreatitis and 30.6% presented with severe pancreatitis,

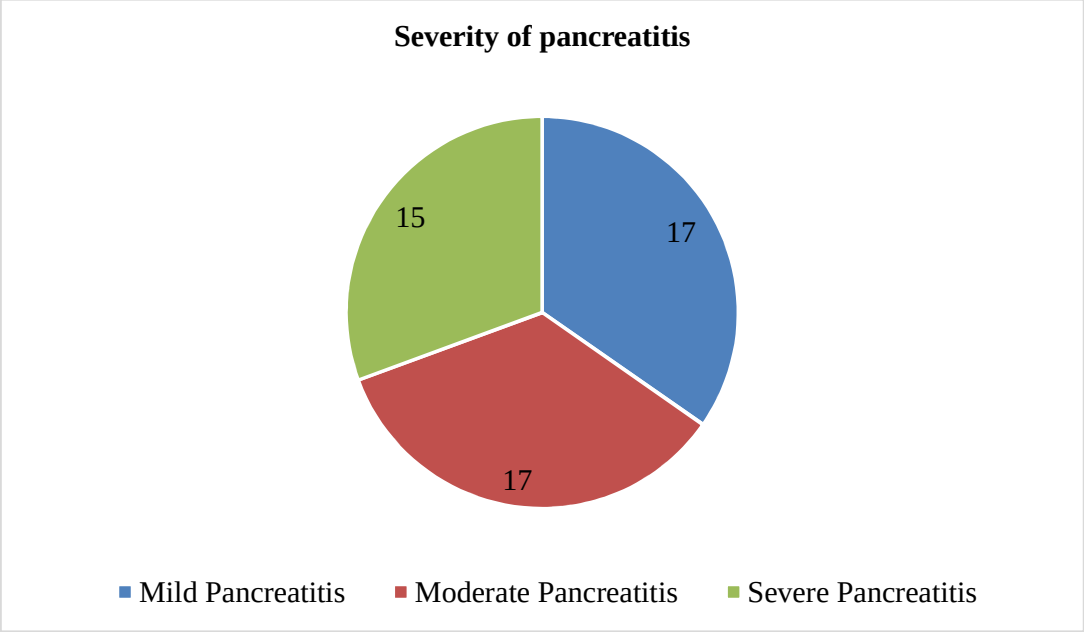

Figure 16: Showing distribution of severity of pancreatitis

Table 8: Distribution of intervention or surgery among patients

		Count	N %
Intervention / surgery	Dialysis	8	16.3%
	Pancreatic Necrosectomy	2	4.08%
	Ventilator support	7	14.28%
	ICD (Intercostal drain) inserted	1	2.04%
	Nil	31	63.2%

Among the intervention, majority underwent the dialysis (16.3%) followed by ventilator support (14.28%).

Among 5 cases of Gallstones Pancreatitis, only 1 underwent Cholecystectomy in the same admission, 1 left against medical advice. No Pancreatitis post-ERCP cases in the study sample.

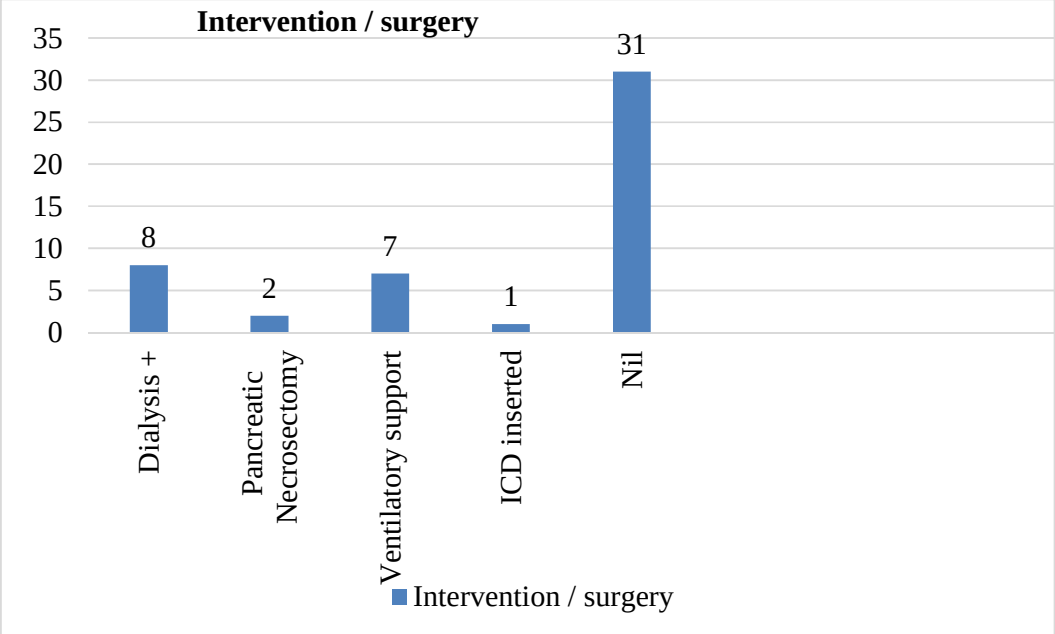

Figure 17: Distribution of intervention or surgery among patients

Table 9: Showing the clinical condition at discharge of patients

		Count	N %
Clinical condition at discharge	Expired	6	12.2%
	Improved	42	85.7%
	LAMA (Left Against Medical Advice)	1	2.0%

Among the outcome of the patients, 85.7% improved at discharge, 12.2% expired and 2% left against medical advice.

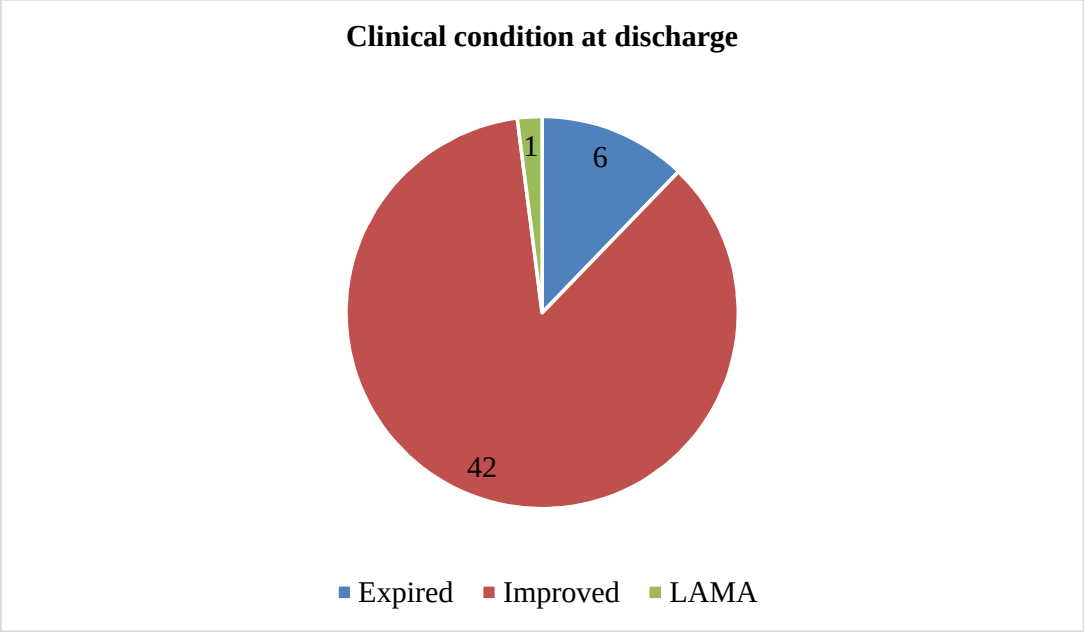

Figure 18: Showing the clinical condition at discharge of patients

Table 10: Showing mean duration of hospital stay

	Mean (days)	SD
Duration of Hospital Stay (in days)	12.1	7.6
ICU stay (in days)	5.5	6.1
Ward (in days)	6.6	6.0

The mean duration of hospital stay is 12.1 days

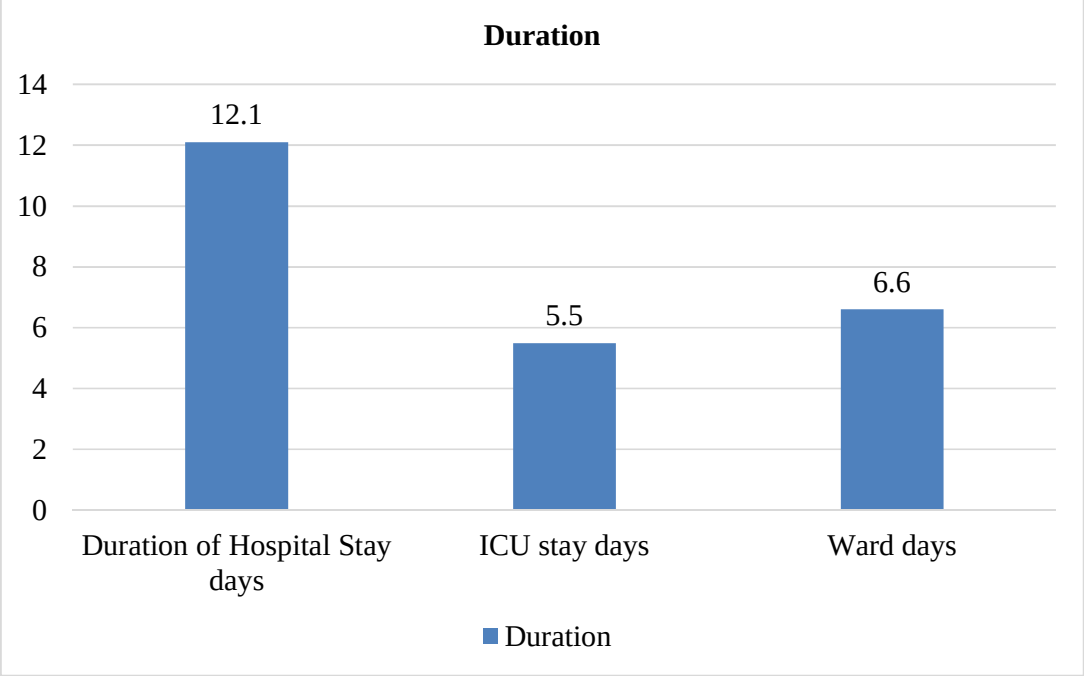

Figure 19: Showing mean duration of hospital stay(in days)

Table 11: Comparison of the individual serum calcium and serum lactate level with severity of pancreatitis

	Mild Pancreatitis		Moderate Pancreatitis		Severe Pancreatitis		ANOVA (p-value)
	Mean	SD	Mean	SD	Mean	SD	
Serum Calcium (mmol/L)	1.014	0.195	0.755	0.388	0.588	0.239	0.01
Serum Lactate (mmol/L)	1.335	0.510	1.893	0.945	3.005	1.561	0.01

On there is significant difference in mean level of the serum lactate and calcium with severity of the pancreatitis. The mean level of the serum calcium was significantly lower in severe pancreatitis compared to moderate and mild pancreatitis.($p<0.05$) whereas the serum lactate level was found to be significantly higher in severe pancreatitis compared to patients with moderate and mild pancreatitis.($p<0.05$)

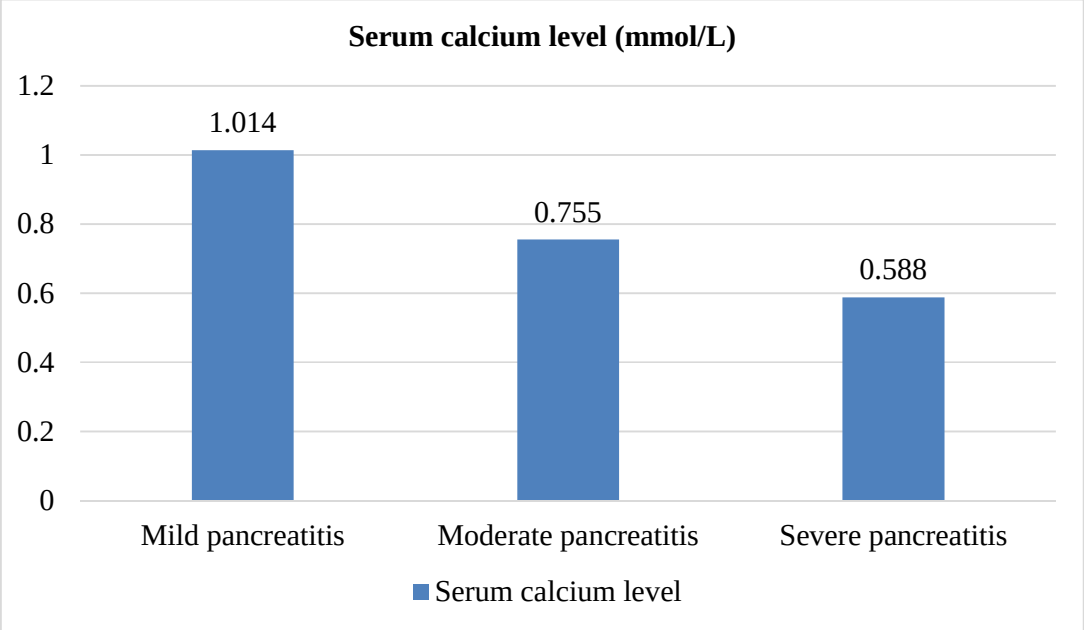

Figure 20: Comparison of the serum calcium level with severity of pancreatitis

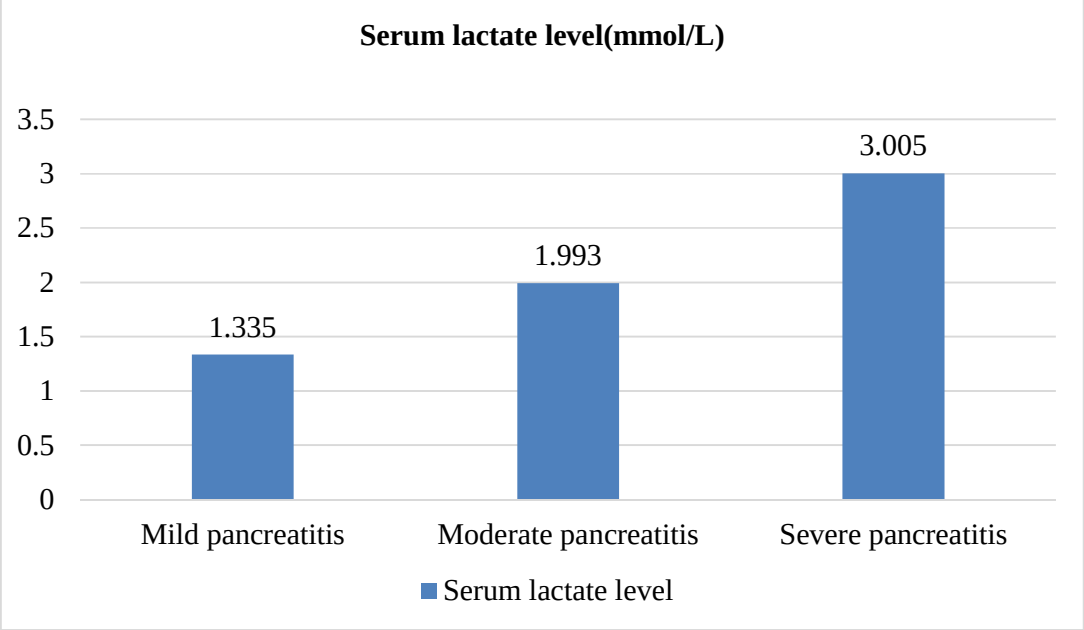

Figure 21: Comparison of the serum lactate level with severity of pancreatitis

Table 12: Comparison of the serum calcium - lactate ratio level with severity of pancreatitis

	Mild Pancreatitis		Moderate Pancreatitis		Severe Pancreatitis		ANOVA (p-value)
	Mean	SD	Mean	SD	Mean	SD	
Ratio of Calcium-Lactate	0.874	0.352	0.451	0.205	0.231	0.120	0.01

There is significant difference in mean level of calcium lactate ratio with severity of pancreatitis. The mean level of calcium-lactate was significantly lower in severe pancreatitis compared to patients in moderate and mild pancreatitis. ($p<0.05$)

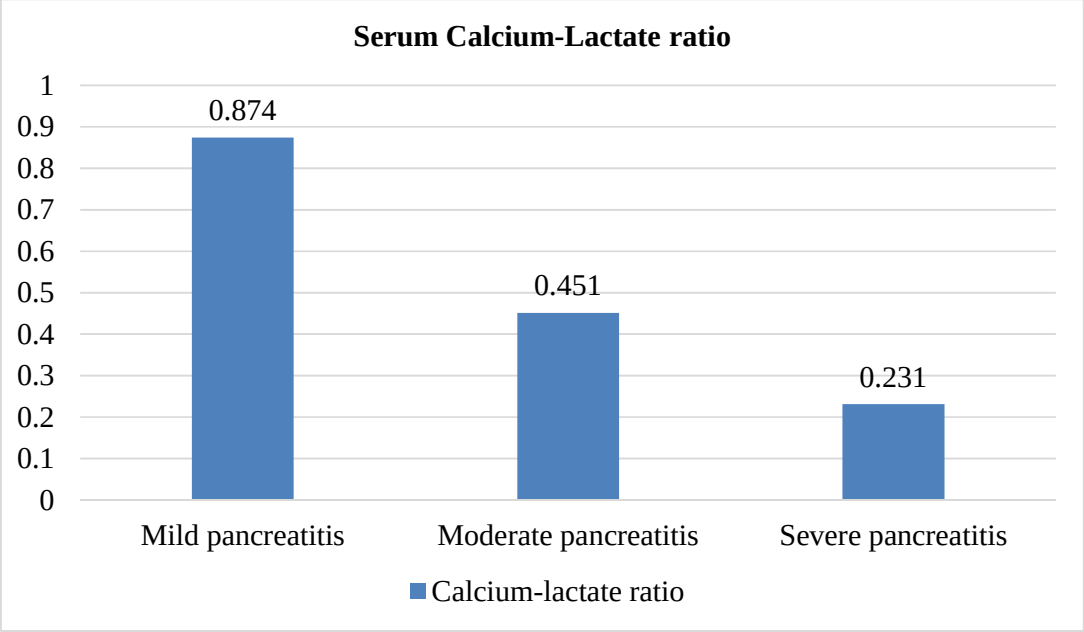

Figure 22: Comparison of the serum calcium - lactate ratio level with severity of pancreatitis

Table 13: Correlation of calcium to lactate ratio with hospital stay

Correlations		Duration of Hospital Stay days
Calcium-lactate Ratio	r	-0.573
	P value	0.000

There is significant negative correlation of the calcium lactate ratio with duration of hospital stay, showing the significant lower mean ratio is associated with longer hospital stay. r = correlation value, shows strength of correlation and p value 0.000.

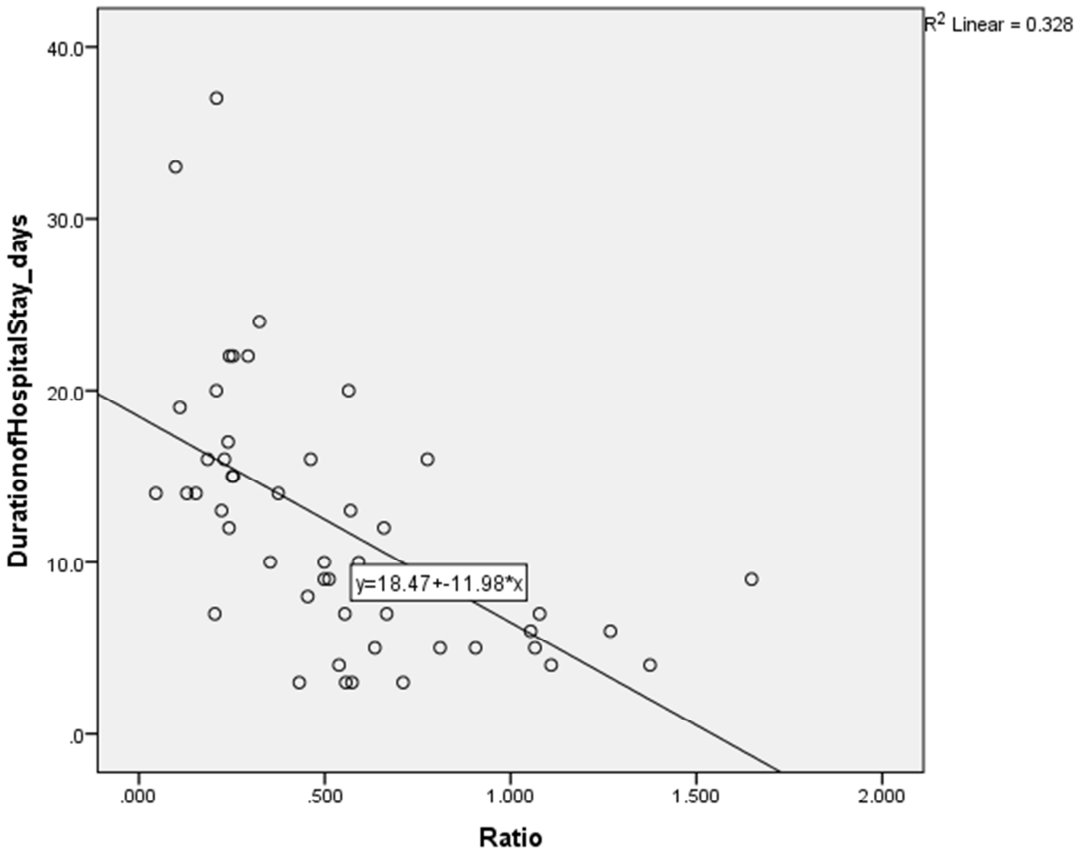

Figure 23: Correlation of calcium to lactate ratio with hospital stay

Table 1414: Comparison of hospital stay duration Mild, Moderate, Severe pancreatitis

	Mild Pancreatitis (n=17 cases)	Moderate Pancreatitis (n=17 cases)	Severe Pancreatitis (n=14 cases)
Mean days of hospital stay	6.1 days	13 days	16.85 days

The table compares the average hospital stay duration among the patients with varying severities of Pancreatitis. For those with mild Pancreatitis, the average hospital stay duration was 6.1 days, while patients with moderate Pancreatitis stayed an average of 13 days. In contrast, individuals with severe Pancreatitis had the longest hospital stays, averaging 16.85 days. This progression reflects that number of days of hospital stay depends on severity Pancreatitis, associated major acute illness and it is noted that surgical intervention alters the course and duration in the hospital.

Here in a case of Moderate Pancreatitis who underwent Pancreatic Necrosectomy, hospital stay was 37 days, whereas average stay duration for Moderate Pancreatitis case being 13 days. And other cases of Mild Pancreatitis with 20 days hospital stay duration due to associated Alcoholic Liver disease, whereas average stay duration for Mild Pancreatitis is 6.1 days

Table 15: Comparison of the mean level of calcium to lactate ratio with clinical outcome of patients

		Ratio		p-value
		Mean	SD	
Clinical condition at discharge	Expired	0.239	0.075	0.01
	Improved	0.579	0.369	

There is significant lower mean ratio of Calcium and Lactate level in patients expired compared to the patients improved at discharge in the study.($p<0.05$)

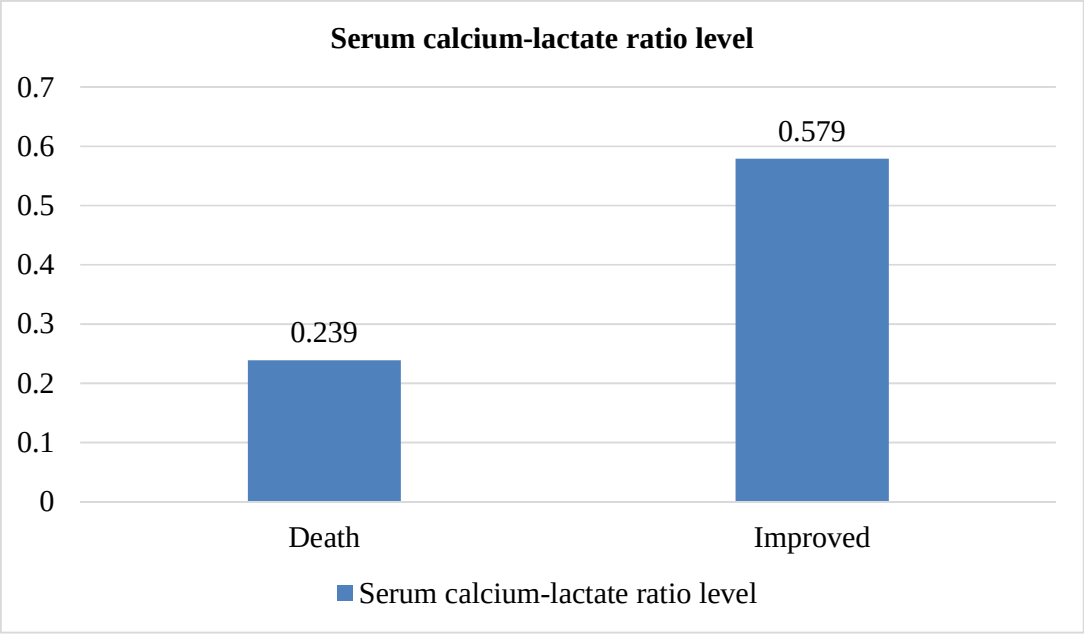

Figure 24: Comparison of the mean level of calcium to lactate ratio with clinical outcome of patients

DISCUSSION

Acute pancreatitis (AP) is a significant and growing global health concern, characterized by an inflammatory response to pancreatic injury. Its incidence and associated morbidity and mortality are increasing, with severe cases often leading to organ dysfunction and prolonged hospital stays.[51–53] Accurate and early prediction of AP severity is crucial for effective management and timely intervention. Traditional prognostic indices such as Ranson, BISAP, and APACHE-II have been instrumental in guiding clinical decisions but come with limitations in predictive precision.

Emerging evidence suggests that simple biochemical markers like serum calcium and serum lactate could offer valuable insights into AP severity and outcomes. Hypocalcemia, a known poor prognostic marker in AP, and elevated lactate, a marker of tissue hypoxia and sepsis, have shown potential as indicators of disease severity. However, the ratio of serum calcium to serum lactate has not been extensively studied in this context. Serum Lactate level is reliable in predicting the mortality in any cases and Lactate level can also predict hospitalization.[54,55]

This discussion explores the role of the serum calcium to serum lactate ratio in acute pancreatitis. We aim to evaluate its effectiveness in predicting the severity of the condition, clinical outcomes, and the duration of hospital stay, providing a potentially novel and practical tool for clinicians in the management of AP.

In present study total of 49 patients fulfilling inclusion criteria are included with mean age of 39yrs. Among included patients, 85.7% were male and 14.3% were female patients, with male preponderance. Among the patients the comorbidities were present in 42.9% of of the patients, among which diabetes and hypertension were most common among patients.

In concordance to present study Edakkepuram et al., documented with Acute pancreatitis typically affects individuals aged 30-50. It is more prevalent in men, accounting for 80% of cases, compared to 20% in women. Alcohol consumption is the leading cause, contributing to 58% of acute pancreatitis cases, while gallstones account for 28%.[3] However in study by Doganay F et al., documented patients in age group of 65yrs and among them 61.9% were female and 38.1% were male patients. [49]

Among the patients, 34.7% of the patient presented with mild and 34.7% of the patient presented with moderate pancreatitis and 30.6% presented with severe pancreatitis, There is significant difference in mean level of the serum lactate and calcium with severity of the pancreatitis. The mean level of the serum calcium was significantly lower in severe pancreatitis compared to moderate and mild pancreatitis ($p<0.05$) whereas the serum lactate level was found to be significantly higher in severe pancreatitis compared to patients with moderate and mild pancreatitis ($p<0.05$) There is significant difference in mean level of calcium-lactate ratio with severity of pancreatitis. The mean level of calcium-lactate ratio was significantly lower in severe pancreatitis compared to patients in moderate and mild pancreatitis. ($p<0.05$)

In concordance Ahmed A et al., documented with Hypocalcemia as most commonly observed finding in cases of acute pancreatitis. Severe hypocalcemia can lead to both neurological and cardiovascular symptoms. [1] According to Ammori et al., hypocalcemia was more common in severe pancreatitis attacks than in mild ones (86percent vs. 39percent, $P < 0.001$). [23] Another study by Peng T et al., documented with serum calcium levels upon admission are independently linked to POF in AP patients and could potentially serve as a prognostic indicator. [47] In similar to present study Pokharel A et al., documented 14% of the patients with severe pancreatitis. Also, the Total calcium (TC) and adjusted calcium concentration (ACC) measured

within the initial 24 hours serve as valuable indicators for predicting the severity of acute pancreatitis. [2]

An independent predictor of death in patients receiving intensive care unit (ICU) treatment was iCa <0.8 mmol/L, according to an Australian study including 7024 patients. [10]In line another study by Edakkepuram U et al., documented that the presence of hypocalcemia and low adjusted calcium concentration (ACC) can help predict the severity of acute pancreatitis similarly to the BISAP score. However, these indicators do not outperform the BISAP score in predicting severity. [3] In study by Thakur A et al., both corrected calcium and total calcium demonstrated a good positive predictive value for indicating disease progression and a highly significant negative predictive value for ruling out the severity of acute pancreatitis. [48] Severe Pancreatitis is complicated by sepsis, which becomes an important contributor to hypocalcemia. According to Whitted et al., circulating catecholamines that are elevated in sepsis may induce circulating calcium to move into the intracellular compartment, resulting in relative hypocalcemia. Due to a negative feedback loop, this raises PTH production and increases intracellular calcium excess, oxidative stress, and cell death. [9]

Among the intervention, majority underwent the dialysis (16.3%) followed by ventilator support (14.28%) Among the outcome of the patients, 85.7% improved at discharge, 12.2% expired and 2% left against medical advice. There is significant negative correlation of the calcium-lactate ratio with duration of hospital stay, showing the significant lower mean ratio is associated with longer hospital stay. There is significant lower mean ratio level in patients expired compared to the patients improved at discharge in the study.($p<0.05$)

In study by Shu W et al., documented that the higher level of the lactate was related with higher incidence of mortality among the patients with pancreatitis.[4] With above supporting evidence of Hypocalcemia and increased lactate in cases of severe Acute Pancreatitis, a combination of these two markers done with the same blood sample on Arterial Blood Gas analysis could be more fitting and reliable than single marker. Early identification of acute pancreatitis (AP) patients and prompt initiation of appropriate treatment can significantly reduce morbidity and mortality. Based on the findings, elevated lactate levels measured upon admission can serve as a valuable, quick, and straightforward method for predicting mortality in AP patients. [49]

SUMMARY

- In present study total of 49 patients fulfilling inclusion criteria are included with mean age of 39yrs.
- Among included patients, 85.7% were male and 14.3% were female patients, with male preponderance.
- Among the patients the comorbidities were present in 42.9% of the patients, among which diabetes and hypertension were most common among patients.
- The mean alcohol intake years among the study patients was found to be 8.8 years. 41 out of 49 study population was alcoholic.
- The mean serum amylase (U/L) was found to be 869.0±74.1 and lipase (U/L) was 1170.1±960.9.
- Among the patients, 34.7% of the patient presented with mild and 34.7% of the patient presented with moderate pancreatitis and 30.6% presented with severe pancreatitis.
- The mean level of Ratio of Serum calcium-lactate in
    1. Mild Pancreatitis=0.874 (Range:0.522-1.226),
    2. Moderate Pancreatitis=0.451 (Range 0.246-0.656),
    3. Severe Pancreatitis =0.231 (Range: 0.111-0.351).
- Among the intervention, majority underwent the dialysis (16.3%) followed by ventilator support (14.28%)
- Among the outcome of the patients, 85.7% improved at discharge, 12.2% expired and 2% left against medical advice.

- The mean duration of hospital stay in cases of Mild Pancreatitis=6.1 days, Moderate Pancreatitis= 13 days, Severe Pancreatitis=16.85 days. Associated acute illness and surgical intervention altered the hospital duration stay.
- There is significant difference in mean level of the serum lactate and calcium with severity of the pancreatitis. The mean level of the serum calcium was significantly lower in severe pancreatitis compared to moderate and mild pancreatitis.($p<0.05$) whereas the mean serum lactate level was found to be significantly higher in severe pancreatitis compared to patients with moderate and mild pancreatitis.($p<0.05$)
- There is significant difference in mean level of calcium lactate ratio with severity of pancreatitis. The mean level of calcium-lactate ratio was significantly lower in severe pancreatitis compared to patients in moderate and mild pancreatitis. ($p<0.05$).
- There is significant negative correlation of the calcium lactate ratio with duration of hospital stay, showing the significant lower mean ratio is associated with longer hospital stay.
- There is significant lower mean ratio level in patients expired compared to the patients improved at discharge in the study.($p<0.05$)

LIMITATIONS OF THE STUDY

- Single Centre Observational Study
- Less Sample size
- Need more detailed studies, to evaluate various other factors affecting the calcium and lactate level.
- Lack of sequential assessment at periodic interval of Serum Calcium-Lactate Ratio to assess course in hospital, effect of ongoing treatment/intervention/surgery and its use in predicting prognosis.

STRENGTHS OF THE STUDY

- Novel biomarker and both measured in single blood sample.
- Easily available and Economical.
- Easy to interpret values and quick to assess the Severity.
- Single value at time of admission can help assess the severity and stratify the risk, which will help in early intervention.

CONCLUSION

The study underscores the utility of serum calcium and lactate levels, and particularly their ratio, as biomarkers for predicting the severity of acute pancreatitis. Patients with severe pancreatitis exhibit significantly lower serum calcium, higher serum lactate levels and low calcium-lactate ratios. Ratio of Serum Calcium and lactate of 0.231 (Range: 0.111-0.351). And lesser indicates Severe Pancreatitis. These markers not only correlate with disease severity but also predict clinical outcomes and duration of hospitalization. A lower calcium-lactate ratio is indicative of a more severe disease course, longer hospital stays, and higher mortality risk. Hence, routine measurement of these parameters could enhance clinical decision-making, facilitating early identification of patients at greater risk and implementing in various management strategies in acute pancreatitis.

BIBLIOGRAPHY

1. Ahmed A, Azim A, Gurjar M, Baronia AK. Hypocalcemia in acute pancreatitis revisited. Indian J Crit care Med peer-reviewed, Off Publ Indian Soc Crit Care Med. 2016;20(3):173–6.

2. Pokharel A, Sigdel PR, Phuyal S, Kansakar PBS, Vaidya P. Prediction of severity of acute pancreatitis using total serum calcium and albumin-corrected calcium: a prospective study in tertiary center hospital in Nepal. Surg Res Pract. 2017;2017.

3. Edakkepuram U, NK MN, Parambil SM, Matad S. Total serum calcium and corrected calcium as severity predictors in acute pancreatitis: a prospective study in a tertiary hospital in South India. Int Surg J. 2017;4(2):560–3.

4. Shu W, Wan J, Chen J, He W, Zhu Y, Zeng H, et al. Initially elevated arterial lactate as an independent predictor of poor outcomes in severe acute pancreatitis. BMC Gastroenterol. 2020;20:1–9.

5. Talathi SS, Zimmerman R, Young M. Anatomy, abdomen and pelvis, pancreas. In: StatPearls [Internet]. StatPearls Publishing; 2023. p. 1–6.

6. Lewis MP, Reber HA, Ashley SW. Pancreatic blood flow and its role in the pathophysiology of pancreatitis. J Surg Res. 1998 Feb;75(1):81–9.

7. Kulenović A, Sarac-Hadzihalilović A. Blood vessels distribution in body and tail of pancreas- a comparative study of age related variation. Bosn J basic Med Sci. 2010 May;10(2):89–93.

8. Love JA, Yi E, Smith TG. Autonomic pathways regulating pancreatic exocrine secretion. Auton Neurosci. 2007 Apr;133(1):19–34.

9. Whitted AD, Stanifer JW, Dube P, Borkowski BJ, Yusuf J, Komolafe BO, et al. A dyshomeostasis of electrolytes and trace elements in acute stressor states: Impact on the heart. *Am J Med Sci.* 2010;340:48–53.

10. Egi M, Kim I, Nichol A, Stachowski E, French CJ, Hart GK, et al. Ionized calcium concentration and outcome in critical illness. *Crit Care Med*. 2011;39:314–21.

11. Fagenholz PJ, Castillo CF, Harris NS, Pelletier AJ, Camargo CAJ. Increasing United States hospital admissions for acute pancreatitis, 1988-2003. Ann Epidemiol. 2007 Jul;17(7):491–7.

12. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. Gastroenterology. 2013;144(6):1252–61.

13. Lankisch PG, Breuer N, Bruns A, Weber-Dany B, Lowenfels AB, Maisonneuve P. Natural history of acute pancreatitis: a long-term population-based study. Am J Gastroenterol. 2009 Nov;104(11):2797–805

14. Apte M V, Pirola RC, Wilson JS. Mechanisms of alcoholic pancreatitis. J Gastroenterol Hepatol. 2010 Dec;25(12):1816–26.

15. Zheng Z, Ding Y-X, Qu Y-X, Cao F, Li F. A narrative review of acute pancreatitis and its diagnosis, pathogenetic mechanism, and management. Ann Transl Med. 2021 Jan;9(1):69.

16. Mookadam F, Cikes M. Images in clinical medicine. Cullen's and Turner's signs. N Engl J Med. 2005 Sep;353(13):1386.

17. Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol. 2013;108(9):1400–15; 1416.

18. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62(1):102–11.

19. Yang CJ, Chen J, Phillips ARJ, Windsor JA, Petrov MS. Predictors of severe and critical acute pancreatitis: a systematic review. Dig Liver Dis. 2014;46(5): 446–51.

20. King LG, Seelig CB, Ranney JE. The lipase to amylase ratio in acute pancreatitis. Am J Gastroenterol. 1995;90(1):67–9.

21. Lankisch PG, Petersen M. Lipase/amylase ratio: not helpful in the early etiological differentiation of acute pancreatitis. Z Gastroenterol. 1994;32(1):8–11.

22. Gennari C. Calcium and vitamin D nutrition and bone disease of the elderly. Public Health Nutr. 2001 Apr;4(2B):547–59.

23. Ammori BJ, Barclay GR, Larvin M, McMahon MJ. Hypocalcemia in patients with acute pancreatitis: A putative role for systemic endotoxin exposure. *Pancreas.* 2003;26:213–7.

24. Reid IR, Bolland MJ. Controversies in medicine: the role of calcium and vitamin D supplements in adults. Med J Aust. 2019 Nov;211(10):468–73.

25. Baird GS. Ionized calcium. Clin Chim Acta. 2011 Apr;412(9–10):696–701.

26. Schöfl C. [Update - Calcium Metabolism]. Dtsch Med Wochenschr. 2019 Aug;144(16):1125–32.

27. Wilson RF, Binkley LE, Sabo FMJ, Wilson JA, Munkarah MM, Dulchavsky SA, et al. Electrolyte and acid-base changes with massive blood transfusions. Am Surg. 1992 Sep;58(9):535.

28. Maier JD, Levine SN. Hypercalcemia in the Intensive Care Unit: A Review of Pathophysiology, Diagnosis, and Modern Therapy. J Intensive Care Med. 2015 Jul;30(5):235–52.

29. Zheng J, Zeng X, Wang S. Calcium ion as cellular messenger. Vol. 58, Science China. Life sciences. China; 2015. p. 1–5.

30. Nordin BE. Calcium and osteoporosis. Nutrition. 1997;13(7–8):664–86.

31. Sorensen MD. Calcium intake and urinary stone disease. Transl Androl Urol. 2014 Sep;3(3):235–40.

32. Greger R, Lang F, Oberleithner H. Distal site of calcium reabsorption in the rat nephron. Pflugers Arch. 1978 May;374(2):153–7.

33. Talmage R V, Mobley HT. Calcium homeostasis: reassessment of the actions of parathyroid hormone. Gen Comp Endocrinol. 2008 Mar;156(1):1–8.

34. Duque EJ, Elias RM, Moysés RMA. Parathyroid Hormone: A Uremic Toxin. Toxins (Basel). 2020 Mar;12(3).

35. Fong J, Khan A. Hypocalcemia: updates in diagnosis and management for primary care. Can Fam Physician. 2012 Feb;58(2):158–62.

36. Wang L, Manson JE, Song Y, Sesso HD. Systematic review: Vitamin D and calcium supplementation in prevention of cardiovascular events. Ann Intern Med. 2010 Mar;152(5):315–23.

37. Morotti A, Charidimou A, Phuah C-L, Jessel MJ, Schwab K, Ayres AM, et al. Association Between Serum Calcium Level and Extent of Bleeding in Patients With Intracerebral Hemorrhage. JAMA Neurol. 2016 Nov;73(11):1285–90.

38. Masilamani K, van der Voort J. The management of acute hyperkalaemia in neonates and children. Arch Dis Child. 2012 Apr;97(4):376–80.

39. Palta S, Saroa R, Palta A. Overview of the coagulation system. Indian J Anaesth. 2014;58(5):515–22.

40. Jones MR, Hall OM. Calcium and phosphate metabolism in chronic pancreatitis. World J Gastroenterol. 2019;25(22):2690–700.

41. Garg PK, Tandon RK. Survey on calcium and vitamin D deficiency in acute pancreatitis: A retrospective analysis. Indian J Gastroenterol. 2019;38(1):41–7.

42. Wu BU, Bakker OJ, Papachristou GI, Besselink MG, Repas K, Van Santvoort HC, et al. Blood urea nitrogen in the early assessment of acute pancreatitis: An international validation study. Arch surgery,. 2014;147(9):882–8.

43. Mofidi R, Duff MD, Wigmore SJ, Madhavan KK, Garden OJ, Parks RW. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. J Br Surg. 2006;93(6):738–44.

44. Gomatos IP, Xiaodong X, Ghaneh P, Halloran C, Raraty M, Lane B, et al. Prognostic markers in acute pancreatitis. Expert Rev Mol Diagn. 2014;14(3):333–46.

45. Bollen TL, Singh VK, Maurer R, Repas K, Van Es HW, Banks PA, et al. A comparative evaluation of radiologic and clinical scoring systems in the early prediction of severity in acute pancreatitis. Off J Am Coll Gastroenterol ACG. 2012;107(4):612–9.

46. Wu BU, Johannes RS, Sun X, Tabak Y, Conwell DL, Banks PA. The early prediction of mortality in acute pancreatitis: a large population-based study. Gut. 2008;57(12):1698–703.

47. Peng T, Peng X, Huang M, Cui J, Zhang Y, Wu H, et al. Serum calcium as an indicator of persistent organ failure in acute pancreatitis. Am J Emerg Med. 2017;35(7):978–82.

48. Thakur A, Goyal G, Aggarwal S, Singh S. Role of total calcium and corrected calcium in comparisons with computed tomography to predict severity of acute pancreatitis. Int Surg J. 2020;7(2):399–403.

49. Doganay F, Ak R, Yilmaz E. Predictive performance of lactate as a mortality predictor in patients with acute pancreatitis. J Coll Physicians Surg Pakistan. 2022;61:49–73.

50. Zeng Z, Huang R, Lin H, Peng H, Luo J, Ding N. Serum Lactate Is an Indicator for Short-Term and Long-Term Mortality in Patients with Acute Pancreatitis. Dig Dis Sci. 2024;1–12.

51. Iannuzzi J, King J, Leong J, Quan J, Windsor J, Tanyingoh D, et al. Global Incidence of Acute Pancreatitis Is Increasing Over Time: A Systematic Review and Meta-Analysis. Gastroenterology. 2021;162:1–6.

52. Tonsi AF, Bacchion M, Crippa S, Malleo G, Bassi C. Acute pancreatitis at the beginning of the 21st century: the state of the art. World J Gastroenterol. 2009 Jun;15(24):2945–59.

53. Szatmary P, Grammatikopoulos T, Cai W, Huang W, Mukherjee R, Halloran C, et al. Acute Pancreatitis: Diagnosis and Treatment. Drugs. 2022 Aug;82(12):1251–76.

54. Seker YC, Bozan O, Sam E, Topacoglu H, Kalkan A. The role of the serum lactate level at the first admission to the emergency department in predicting mortality. Am J Emerg Med. 2021 Jul;45:495-500.

55. Alshiakh SM. Role of serum lactate as prognostic marker of mortality among emergency department patients with multiple conditions: A systematic review. SAGE open Med. 2023;11:11–9.

56. Isogai M, Yamaguchi A, Hori A, Kaneoka Y. LDH to AST ratio in biliary pancreatitis—a possible indicator of pancreatic necrosis: preliminary results. Am J Gastroenterol 1998;93:363–367.

57. B Gloor, C A Müller, M Worni, M E Martignoni, W Uhl, M W Büchler, Late mortality in patients with severe acute pancreatitis, British Journal of Surgery, Volume 88, Issue 7, July 2001, Pages 975–979

58. Shinzeki, M., Ueda, T., Takeyama, Y. et al. Prediction of early death in severe acute pancreatitis. J Gastroenterol 43, 152–158 (2008)

59. Connor S, Alexakis N, Raraty MG, et al. Early and late complications after pancreatic necrosectomy. Surgery 2005;137(5):499–505.

60. Current surgical management of acute pancreatitis. Acta Chir Belg 2006;106(2):165–171.

61. Raraty MG, Halloran CM, Dodd S, et al. Minimal access retroperitoneal pancreatic necrosectomy: improvement in morbidity and mortality with a less invasive approach. Ann Surg 2010;251(5):787–793

62. Park DH, Lee SS, Moon SH, et al. Endoscopic ultrasound-guided versus conventional transmural drainage for pancreatic pseudocysts: a prospective randomized trial. Endoscopy 2009;41(10):842–848.

63. Bakker OJ, van Santvoort HC, van Brunschot S, et al. Endoscopic transgastric vs surgical necrosectomy for infected necrotizing pancreatitis: a randomized trial. JAMA 2012;307(10):1053–1061.

64. Loveday BP, Petrov MS, Connor S, et al. A comprehensive classification of invasive procedures for treating the local complications of acute pancreatitis based on visualization, route, and purpose. Pancreatology 2011;11(4):406–413

65. Thoeni RF. The revised Atlanta classification of acute pancreatitis: its importance for the radiologist and its effect on treatment. Radiology 2012;262(3): 751–764.

66. Anand N, Park JH, Wu BU. Modern management of acute pancreatitis. Gastroenterol Clin North Am 2012;41(1):1–8.

ANEXURE I – INFORMED CONSENT FORM

KAHERs JNMC, BELAGAVI

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **"OBSERVATIONAL STUDY ON SERUM CALCIUM AND SERUM LACTATE RATIO IN ACUTE PANCREATITIS FOR PREDICTING SEVERITY, CLINICAL OUTCOME AND HOSPITAL STAY DURATION AT KLES PRABHAKAR KORE HOSPITAL".** My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANEXURE II – PROFORMA / QUESTIONNAIRE USED FOR DATA COLLECTION

Name:

IP no.

Sex:

Age:

Date of admission:

Date of discharge:

HISTORY OF PRESENTING COMPLAINTS: pain abdomen since__,

radiating to back ____,

Breathlessness _____,

Urinary symptoms- decreased frequency of urination ____,

fever __

similar complaints in the past ___,

H/O ICU admission for the same ___

H/o trauma

PAST HISTORY:

H/O surgery ____

Co-morbidities ___

PERSONAL HISTORY:

Alcoholic:____, quantity and duration _____, last drink _____

Other addictions: _____

GENERAL PHYSICAL EXAMINATION:

Pallor

Icterus

Cyanosis

Clubbing

Oedema

Lymphadenopathy

Vital Signs:

PR:____ /min;

BP: _____mm Hg;

SpO_2-

Temperature:

Urine frequency/ output-

SYSTEMIC EXAMINATION:

Abdomen:

skin discolouration -

Cardio Vascular System:

Respiratory System:

INVESTIGATIONS:

Serum Amylase:

Serum Lipase:

Hb:

TLC:

Creatinine:

Urea:

Total bilirubin:

IMAGING:

On ABG,

Serum Calcium:

Serum Lactate:

Ratio:

No. of days of hospital stay:

No. of days in ICU:

No. of days in ward:

Severity (Using modified Atlanta):

Mild Pancreatitis

Moderate Pancreatitis

Severe Pancreatitis

Clinical Condition at the time of discharge: ____

Improved /Worsened/ Death/Left against Medical Advice

ANEXURE III – MASTERCHART

S.No	Age (in years)	Sex	IP Number	Co-morbidities	Alcohol Intake	Other addictions	Serum Amylase	Serum Lipase	DIAGNOSIS	Serum Calcium	Serum Lactate	Ratio	Severity of Pancreatitis	INTERVENTION/SURGERY	Duration of Hospital Stay_days	ICU stay_days	Ward_days	Clinical condition at discharge
1	32	Male	1186028	Nil	6	Smoker since 6 yrs	1102	712	Acute Necrotising Pancreatitis with B/l Pleural Effusion	0.424	1.2	0.35	Moderate Pancreatis	nil	10	0	10	Improved
2	37	Male	1164705	Nil	10	Nil	507	674	Recurrent Acute Pancreatitis	1.22	1.1	1.11	Mild Pancreatis	nil	4	0	4	Improved
3	40	Male	1176037	DM	15	smoker since 15 yrs	483	1557	Acute Pancreatitis with AKI with B/L Pleural effusion	0.674	3.3	0.20	Severe Pancreatis	nil	7	3	4	Improved
4	48	Male	1189348	HTN	10	Nil	1092	1649	Acute Pancreatitis with AKI with left Pleural effusion	0.319	7.01	0.05	Severe Pancreatis	nil	14	10	4	Improved
5	31	Male	119963	Nil	8	tobacco	391	1787	Acute	0.602	2.89	0.21	Severe	Dialysis +, ventilatory	20	15	5	Expire

			9			chewer since 12 yrs			Necrotising Pancreatitis with B/l Pleural Effusion with Alcohol withdrawal				Pancreatitis	support				d
6	32	Male	1179161	Nil	6	Nil	869	899	Acute Pancreatitis with AKI	1.94	3.89	0.50	Moderate Pancreatitis	Nil	9	3	6	Improved
7	30	Male	1204748	Nil	1	Nil	533	417	Acute Necrotising Pancreatitis with AKI with B/L Pleural Effusion	0.457	1.8	0.25	Severe Pancreatitis	Dialysis +, ventilatory support	15	14	1	Improved
8	36	Male	1204033	Nil	10	Nil	3207	4442	Acute Pancreatitis with AKI	0.871	1.53	0.57	Severe Pancreatitis	Dialysis +	13	12	1	Improved
9	30	Male	1197938	Nil	6	tobacco chewer since 5 yrs	1500	1800	Acute Necrotising Pancreatitis with Severe Pulmonary Embolism	0.402	1.24	0.32	Severe Pancreatitis	Dialysis +, Pancreatic Necrosectomy, USG guided drainage	24	15	9	Expired
10	45	Male	1002602 7	HTN, DM	11	tobacco chewer since 8 yrs	2550	2388	Acute Pancreatitis with AKI with MODS	0.649	2.21	0.29	Severe Pancreatitis	Dialysis +, ventilatory support	22	8	14	Expired

									with Alcohol Withdrawal									
11	65	Female	10001437	HTN, DVT		Nil	1800	1130	Acute Biliary Pancreatitis with AKI with B/L pleural Effusion (Cholelithiasis)	0.8	3.6	0.22	Severe Pancreatitis	Dialysis+, Ventilatory Support	13	13	0	Left Against Medical Advise
12	21	Male	10004536	Nil	0.5	Nil	2187	2020	Acute Necrotizing Pancreatitis	0.354	1.46	0.24	Moderate Pancreatitis	Nil	12	3	9	Improved
13	33	Male	10029352	HTN	2	Tobacco Chewer since 2 yrs	1244	1605	Recurrent Acute Pancreatitis with AKI with B/L Pleural Effusion with Delirium Tremens	0.206	1.87	0.11	Severe Pancreatitis	Dialysis +	19	19	0	Expired
14	52	Female	10006717	Nil		Nil	1622	3729	Acute Necrotizing Pancreatitis	0.885	1.14	0.78	Moderate Pancreatitis	Ventilatory support for 1 day	16	8	8	Improved
15	30	Male	1186509	Nil	2	Tobacco	485	438	Recurrent Necrotizin	0.501	0.76	0.66	Moderate Pancreati	Nil	12	5	7	Improved

						chewer since 6 months			g Pancreatitis				tis					
16	26	Male	1184554	Nil	2	Nil	191	857	Recurrent Acute Pancreatitis	0.978	1.08	0.91	Mild Pancreatitis	nil	5	0	5	Improved
17	38	Male	1182048	Nil	5	Tobacco chewer since 25 years	544	688	Recurrent Acute Necrotizing Pancreatitis with AKI	0.65	2.67	0.24	Severe Pancreatitis	Dialysis+, Ventilatory Support, Malecot catheter Drainage of Necrotic Collection	22	22	0	Expired
18	60	Female	10029906	HTN		Tobacco Chewer since 30 yrs	665	778	Acute Biliary Interstitial Edematous Pancreatitis	0.878	2.035	0.43	Mild Pancreatitis	Nil	3	1	2	Improved
19	45	Male	10031353	Nil	30	Smoker since 2 years	1674	2125	Acute Necrotising Pancreatitis	0.844	1.07	0.79	Moderate Pancreatitis	Nil	9	6	3	Improved
20	48	Male	10029357	DM	5	Nil	610	1724	Acute Pancreatitis with B/L Pleural effusion wit Uncontrolled Diabetes	1.072	2.32	0.46	Moderate Pancreatitis	Nil	16	12	4	Improved
21	66	Male	10016932	HTN, DM	20	Nil	1192	1822	Acute Interstitial Edematous	1.012	2.03	0.50	Mild Pancreatitis	Nil	10	7	3	Improved

									Pancreatitis with Right Pleural Effusion								
22	25	Male	10020447	Nil	2	Nil	282	545	Walled off Pancreatic Necrosis with collection with B/L Pleural Effusion	0.372	1.55	0.24	Moderate Pancreatitis	Nil	17	8	6	Improved
23	33	Male	1182361	DM+, Myocarditis	8	Nil	723	1371	Acute Necrotizing Pancreatitis with ARDS with Myocarditis	0.52	2.07	0.25	Severe Pancreatitis	Ventilatory support +	15	10	5	Improved
24	59	Female	10007717	Nil		Nil	2650	3411	Acute Biliary Pancreatitis with Necrotic Collection (Cholelithiasis)	0.325	2.12	0.15	Severe Pancreatitis	nil	14	12	2	Improved
25	60	Male	1182356	DM	30	tobacco chewer +	956	1120	Acute Interstitial Edematous Pancreatitis with B/L	1.114	4.84	0.23	Severe Pancreatitis	NIL	16	13	3	Improved

									Pleural Effusion									
26	41	Male	10022620	Nil	10	tobacco chewer +	400	655	Acute Pancreatitis with WPW syndrome	1.1	0.8	1.38	Mild Pancreatitis	Nil	4	0	4	Improved
27	32	Male	10009989	Nil	5	Nil	233	365	Acute Pancreatitis	0.98	0.93	1.05	Mild Pancreatitis	nil	6	0	6	Improved
28	45	Male	10009705	NIL	15	tobacco chewer since 10 yrs	374	402	acute on chronic pancreatititis with collection	0.927	0.86	1.08	Mild Pancreatitis	nil	7	0	7	Improved
29	22	Male	10009011	pancreatic divisum		nil	1155	1673	acute on chronic pancreatitis with type 1 pancreatic divisum	1.2	1.8	0.67	Mild Pancreatitis	nil	7	2	5	Improved
30	65	Female	10010165	hypertension and hypothyroidism		nil	107	171	Acute Biliary Pancreatits	1.18	1.66	0.71	Mild Pancreatitis	nil	3	0	3	Improved
31	23	Female	10011805	Nil		nil	341	540	acute biliary pancreatitis	0.94	1.16	0.81	Mild Pancreatitis	underwent cholecystectomy and lateral pancreaticojejunostomy on 6/10/23	5	1	4	Improved
32	33	Male	100101	Nil	5	tobacco	107	456	acute	0.536	2.9	0.18	Moderate	ICD inserted	16	13	3	Improv

			66			chewer since 4 years			pancreatitis with pseudocyst with bilateral massive pleural effusion				Pancreatitis					ed
33	27	Male	10015212	Nil	2	Tobacco Chewer since 2 yrs	489	520	acute necrotizing Pancreatitis	0.72	1.3	0.55	Moderate Pancreatitis	nil	7	0	7	Improved
34	48	Male	10014708	Nil	3	nil	257	50	Acute on Chronic Calcific Pancreatitis	0.68	1.33	0.51	Moderate Pancreatitis	nil	9	0	9	Improved
35	37	Male	10015332	Nil	15	nil	113	44	acute on chronic pancreatitis	1.023	1.9	0.54	Mild Pancreatitis	nil	4	0	4	Improved
36	30	Male	10015693	Nil	4	tobacco chewer	434	412	Acute Pancreatitis (Groove Pancreatitis)	1.205	0.95	1.27	Mild Pancreatitis	nil	6	4	2	Improved
37	39	Male	10015968	Nil	10	Nil	567	1044	Acute Pancreatitis with Alcoholic Liver disease with B/L Pleural	0.632	1.12	0.56	Mild Pancreatitis	nil	20	8	12	Improved

									Effusion									
38	33	Male	100171 61	Nil	6	tobacco chewer +	448	548	Acute Pancreatitis with Dorsal Agenesis	1.3	1.22	1.07	Mild Pancreatitis	Nil	5	0	5	Improved
39	24	Male	100176 49	Nil	1	Nil	1328	2576	Acute Pancreatitis (Alcohol induced)	0.577	0.35	1.65	Mild Pancreatitis	nil	9	0	9	Improved
40	37	Male	100185 77	DM	8	Nil	500	1199	Recurrent Acute Pancreatitis (Alcohol induced)	1.002	1.8	0.56	Mild Pancreatitis	Nil	3	0	3	Improved
41	48	Male	100191 42	Nil	15	Nil	1412	1670	Acute on Chronic Pancreatitis with Walled off necrosis	0.45	1.2	0.38	Moderate Pancreatitis	Underwent Endovascular Coil embolisation	14	0	14	Improved
42	45	Male	100121 607	DM	12	tobacco chewer	516	668	Acute Necrotizing Pancreatitis with peripancreatic necrotic collection	0.942	2.074	0.45	Moderate Pancreatitis	nil	8	0	8	Improved
43	34	Male	100210 67	HTN	8	Nil	27	43	Acute Necrotiizin	0.714	3.42	0.21	Moderate Pancreati	Underwent Pancraetic Necrosectomy on	37	11	26	Improved

									g Pancreatitis with Large Peripancreatic collection				tis	24/11/23				
44	35	Female	10027548	Nil		Nil	390	442	Acute Biliary Pancreatitis with Obstructive Jaundice	0.432	3.35	0.13	Moderate Pancreatitis	nil	14	0	14	Improved
45	32	Male	10017294	Nil	10	Nil	406	504	Acute on Chronic Pancreatitis with Pseudocyst with B/L Pleural Effusion	0.489	4.963	0.10	Severe Pancreatitis	Underwent Whipples Pancreatico-duodenectomy with FJ	33	1	32	Improved
46	45	Male	10024141	Nil	18	smoking +	860	892	Acute Necrotozing Pncreatitis	0.966	1.634	0.59	Moderate Pancreatitis	nil	10	0	10	Improved
47	24	Male	10029936	Nil	5	tobacco chewer since 6 years	277	263	Acute Necrotizing Pancreatitis	1.003	1.58	0.63	Moderate Pancreatitis	nil	5	0	5	Improved
48	45	Male	10026027	Nil	7	nil	2335	1989	Acute Pancreatitis with AKI with MODS	0.749	2.96	0.25	Severe Pancreatitis	Nil	22	10	12	Expired

									with Alcohol Withdrawal									
49	45	Male	1005256	DM	12	tobacco chewer	448	520	Recurrent Acute Pancreatitis	1.089	1.9	0.57	Mild Pancreatitis	Nil	3	0	3	Improved