A STUDY ON EVALUATION OF INTRAABDOMINAL PRESSURE IN ACUTE PANCREATITIS

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulation for the award of the

Degree of M.S (general surgery)

BRANCH – I

DEPARTMENT OF GENERAL SURGERY

STANLEY MEDICAL COLLEGE AND HOSPITAL

TAMILNADU DR.M.G.R MEDICAL UNIVERSITY, CHENNAI

MAY- 2020

CERTIFICATE

This is to certify that dissertation "A STUDY ON EVALUATION OF INTRA-ABDOMINAL PRESSURE IN ACUTE PANCREATITIS" is a bonafide record of work done by **Dr.RAJKUMAR.S**, in the Department of General Surgery, Stanley Medical College, Chennai, during his Post Graduate Course from 2017-2020. This is submitted in partial fulfillment for the award of **M.S.DEGREE EXAMINATION - BRANCH I (GENERAL SURGERY)** to be held in May 2020 under the **Tamilnadu DR.M.G.R. Medical University, Chennai.**

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DECLARATION

I **Dr.RAJKUMAR.S** solemnly declare that this dissertation titled "A STUDY ON EVALUATION OF INTRAABDOMINAL PRESSURE IN ACUTE PANCREATITIS"**,** is a bonafide work done by me in the Department of General surgery, Govt. Stanley Medical College and Hospital, Chennai under the supervision of **Prof. Dr.T.SIVAKUAMR M.S.,** and **Prof. Dr.A.ANANDI M.S.** This dissertation is submitted to the Tamilnadu Dr MGR Medical University, Chennai in partial fulfillment of the university regulations for the award of M.S, degree (General Surgery), branch – 1 examination to be held in May 2020.

DATE:

PLACE:

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PLAGIARISM CERTIFICATE

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ETHICAL COMMITTEE CERTIFICATE

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INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK : EVALUATION OF INT RABDOMINAL PRESSURE IN ACUTE PANCREATITIS.

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DESIGNATION : PG IN MS GENERAL SURGERY
DEPARTMENT : DEPARTMENT OF GENERAL SURGERY, GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.12.2018 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

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2. You should not deviate from the area of the work for which you applied for ethical clearance.
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MEMBER SECRETARY, 7/12/18
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INTRODUCTION

Acute Pancreatitis is a multi-system disease with an unpredictable course that surgeons are faced with great difficulty to treat. Due to the significant advances in technology, we have learned much about the pathophysiology and natural course of this disease. Conservative treatment still remains the main modality of treatment, while a subset of patients may require surgical intervention.

The dreaded complication of acute pancreatitis is the development of Intraabdominal hypertension and Abdominal compartment syndrome

Mortality and morbidity in acute pancreatitis is due to the development of pulmonary, cardiovascular and renal insufficiency which may lead to multiple organ dysfunction which in turn is caused by intra-abdominal hypertension and abdominal compartment syndrome. ACS/IAH ultimately affect the blood flow to vital organs worsening the prognosis of the disease. It is a fact that IAH is rampantly seen in ICU patients, surgical emergencies like intestinal obstruction and acute pancreatitis.

Acute pancreatitis causes increased capillary permeability leading to third space losses leading to intraabdominal hypertension. Measurement of intraabdominal pressure can help in early intervention in the disease process of acute pancreatitis that can lead to reduction in its various complications, hence it has become a necessity for surgeons to have a sound knowledge of the disease process and its various clinical features to plan an intensive and individualized patient catered therapeutic approach

Radiological investigations such as CT scan cannot be used to identify intraabdominal hypertension or abdominal compartment syndrome. However, radiological investigations can help to assess the severity and potential complications of acute pancreatitis. Intra-abdominal pressure can be measured by a simple bedside test, the Intra vesical pressure measurement

The World Society on Abdominal Compartment Syndrome (WSACS) initiated in 2006 has become the authority on IAH and has formulated guidelines for the measurement of IAP. Several studies conducted in acute pancreatitis patients have shown an incidence of 40-70% to have IAH and 10-40% to have ACS.

Many prognostic indicators are being used in evaluation of disease progression in acute pancreatitis such as APACHE, Imrie, serum CRP, however, intra vesical pressure measurement, a low cost method which provides minimal discomfort to the patient would be ideal prognostic indicator.

AIMS AND

OBJECTIVES

Evaluation of intra-abdominal pressure by intra vesical method as a marker of severity in acute pancreatitis and its complications

METHODOLOGY

Design of study

Non-randomized prospective study

Sample size

50 cases admitted in general surgery ward

Duration

8 months

Inclusion criteria

Patients admitted to SMCH as cases of acute pancreatitis

Patients of both sexes of any age

Patient willing to participate in the study

Exclusion criteria

Patients not requiring bladder catheterization

Patients not willing to participate in the study

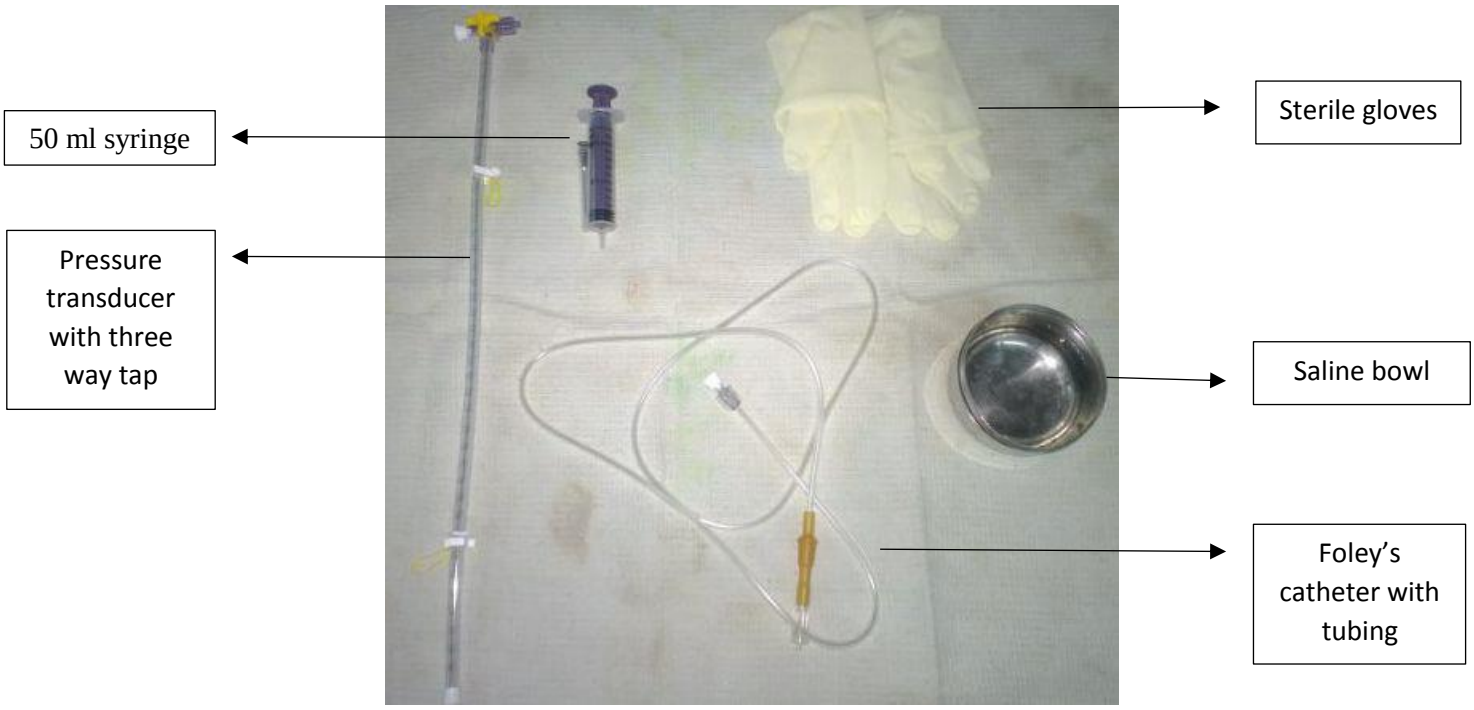
METHODOLOGY

All patients admitted in the Department of General surgery with a diagnosis of acute pancreatitis were included in the prospective study. The Diagnostic criteria for acute pancreatitis were clinical signs and symptoms along with a 3-fold increase in serum amylase levels. Patients were excluded if they declined to consent in the study or were deemed not to be catheterized as per clinical grounds.

Patient demographics (Age, sex, duration of symptoms, etiology) along with basic vital parameters (pulse rate, blood pressure, temperature, saturation, respiratory rate, urine output) were recorded. Laboratory investigations recorded included hemoglobin, hematocrit, serum creatinine, blood urea, serum electrolytes, leukocyte counts and arterial blood gas analysis using which APACHE score was calculated. Radiological investigation such as USG and contrast enhanced CT scan of the abdomen was done to evaluate the Balthazar score.

Intra – abdominal pressure monitoring was done by the intra vesical route, done by catheterization of the urinary bladder using Foley's attached to a three way stopcock in turn connected to a water manometer. The patient was put in a supine position. Before measuring, the bladder was completely drained and the point of measurement taken as zero was at the iliac crest in mid-axillary line. After clamping the urinary drainage using the three way tap, around 25ml of saline was instilled into the bladder using a syringe.

The intra-abdominal pressure was measured at the end of expiration after confirming the fluctuations with each heartbeat. Measurements were recorded at regular intervals, 8th hourly. The usage of vasoactive drugs and development of multi-organ dysfunction was evaluated in patients and recorded. Duration of stay in ICU and hospital stay and final outcome were also recorded. The results were documented and tabulated



EQUIPMENT USED

REVIEW OF LITERATURE

INTRODUCTION

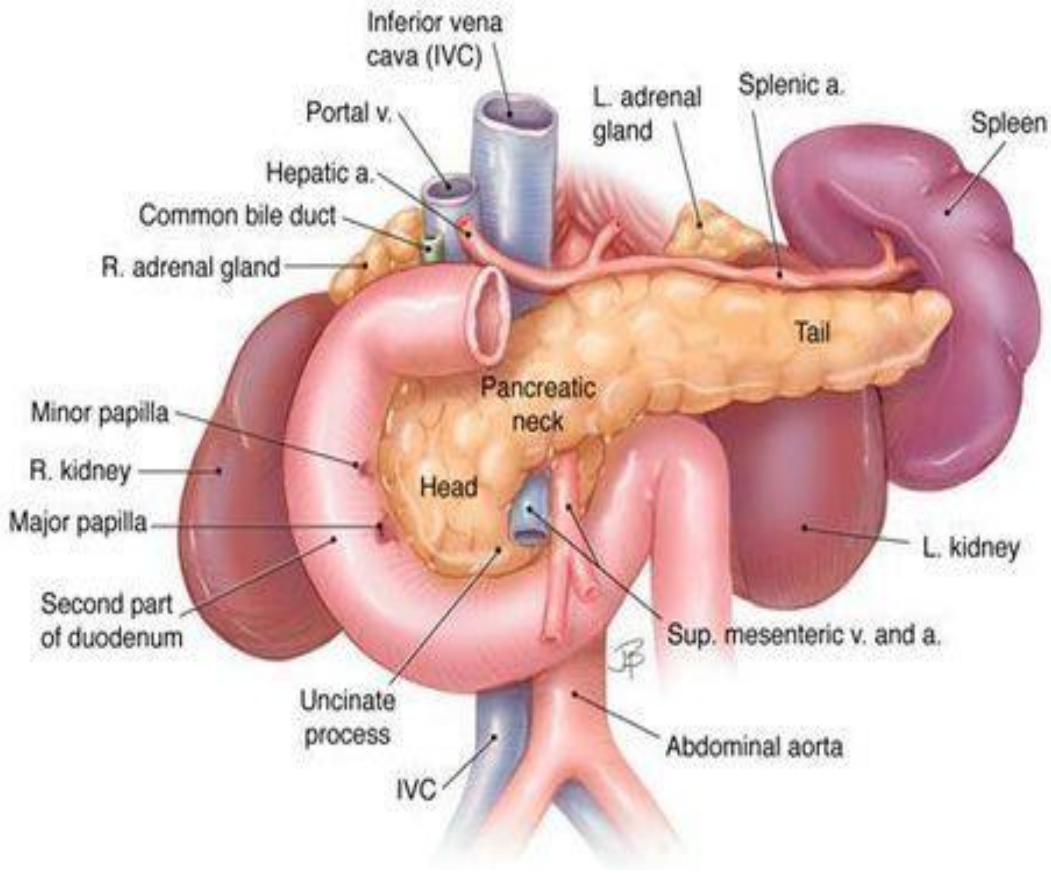
The Pancreas (pan= all, kreas = flesh) is a retroperitoneal gland that is partly exocrine and partly endocrine. The exocrine part secretes the digestive juice, and the endocrine part secretes hormones. It is a soft, lobulated and elongated organ. The pancreas lies transversely across the posterior abdominal wall, at the level of first and second lumbar vertebrae.

The Pancreas is divided into head, neck, body and tail. It is about 15-20 cm long, 2.5-3.8 cm broad and 1.2-1.8 cm thick and weighs about 80-90 grams. The head is enlarged and lies within the concavity of the duodenum. The tail reaches the hilum of the spleen. Lesser sac separates the pancreas and stomach from each other. Due to its position in retroperitoneum, pain originating from it radiates to the back.

Regions of pancreas

The Head of the pancreas lies in the C-loop of the duodenum, posterior to the transverse mesocolon. The vena cava, the right renal artery and both renal veins lie posterior to the head of the pancreas. The neck of the pancreas lies directly anterior to the portal vein. The superior mesenteric vein joins the splenic vein and then continues toward the porta hepatis as the portal vein at the neck of the pancreas. The inferior mesenteric vein joins the splenic vein at its junction with the portal vein.

The uncinate process and the head of the pancreas wrap around the right side of the portal vein and end posteriorly near the space between the superior mesenteric vein and superior mesenteric artery. Venous branches draining the pancreatic head and uncinated process enter along the right lateral and posterior sides of the portal vein.



PANCREAS AND ITS RELATIONS

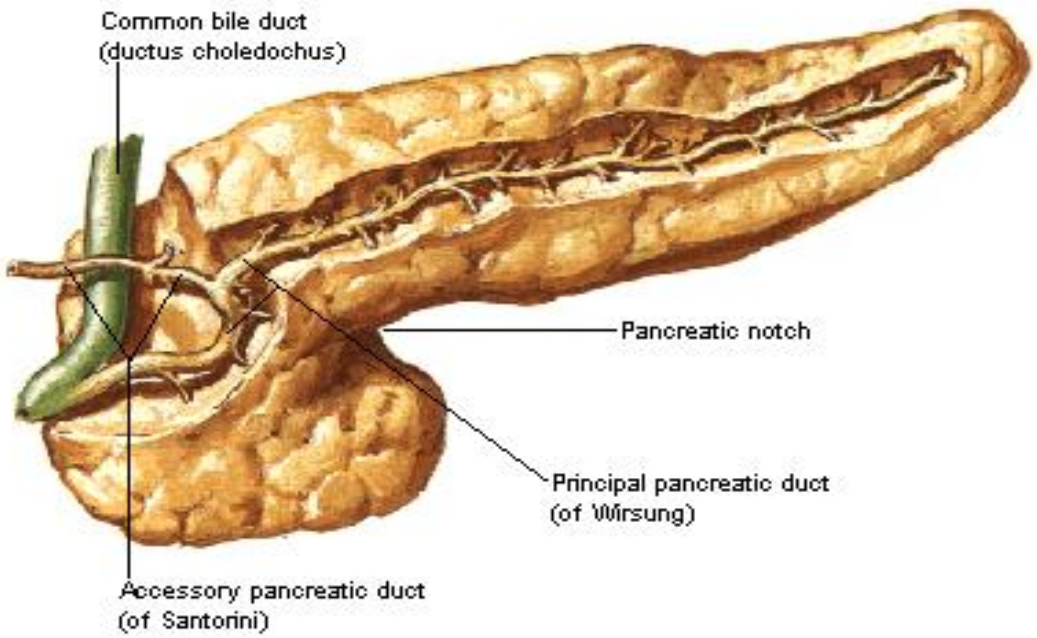
The common bile duct runs on the posterior aspect of the pancreatic head, through the pancreatic parenchyma to join the main pancreatic duct at the Ampulla of Vater. The splenic vein runs on the back of the pancreas. The splenic artery runs parallel and just superior to the vein along the posterior superior edge of the body and tail of the pancreas.

DUCT ANATOMY

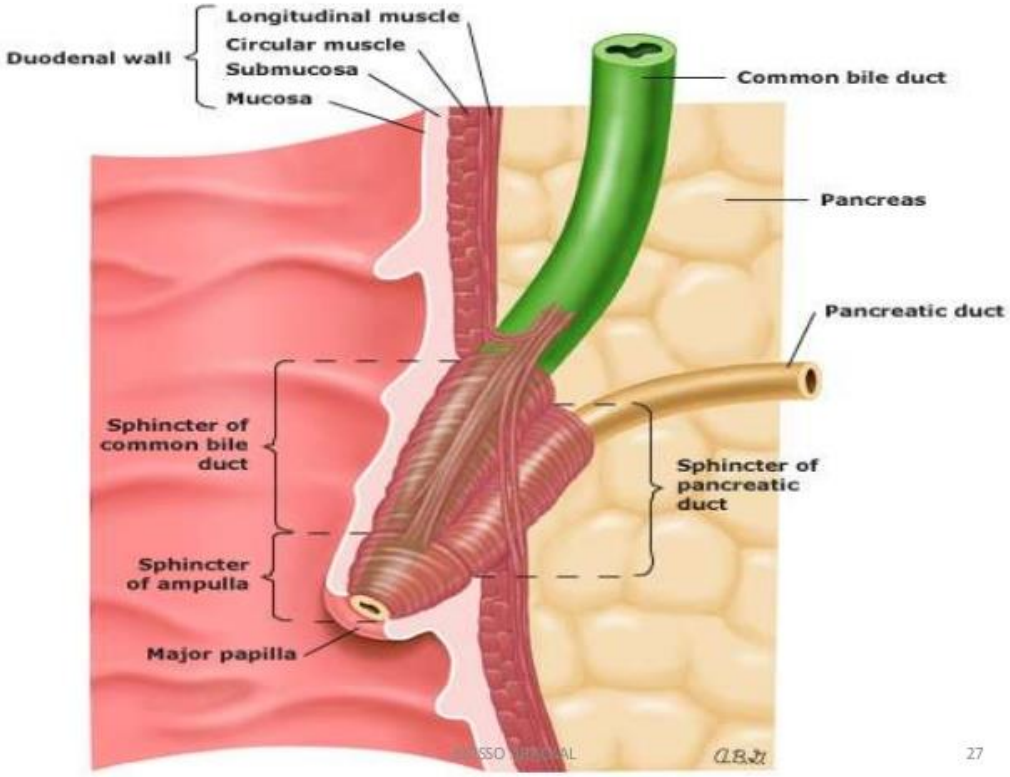
The pancreas is formed by the fusion of the smaller ventral bud arising from hepatic diverticulum and the dorsal bud arising from duodenum. The Duct of the ventral bud becomes the Duct of Wirsung, while the dorsal duct becomes Duct of Santorini.

Both the ducts fuse with each other in the head of pancreas so that Duct of Wirsung forms and fuses with common bile duct to form the main pancreatic drain draining into the second part of duodenum.

The main pancreatic duct is about 2 to 3 mm in diameter and travels between the superior and inferior borders of the pancreas, closer to the posterior than the anterior surface.



The Sphincter of Oddi is formed by the muscle fibers around the ampulla. It creates a high-pressure zone between the ductal system and the duodenum thereby regulating the flow of bile and pancreatic juice into the duodenum and prevention of regurgitation of intestinal contents into the ductal system.

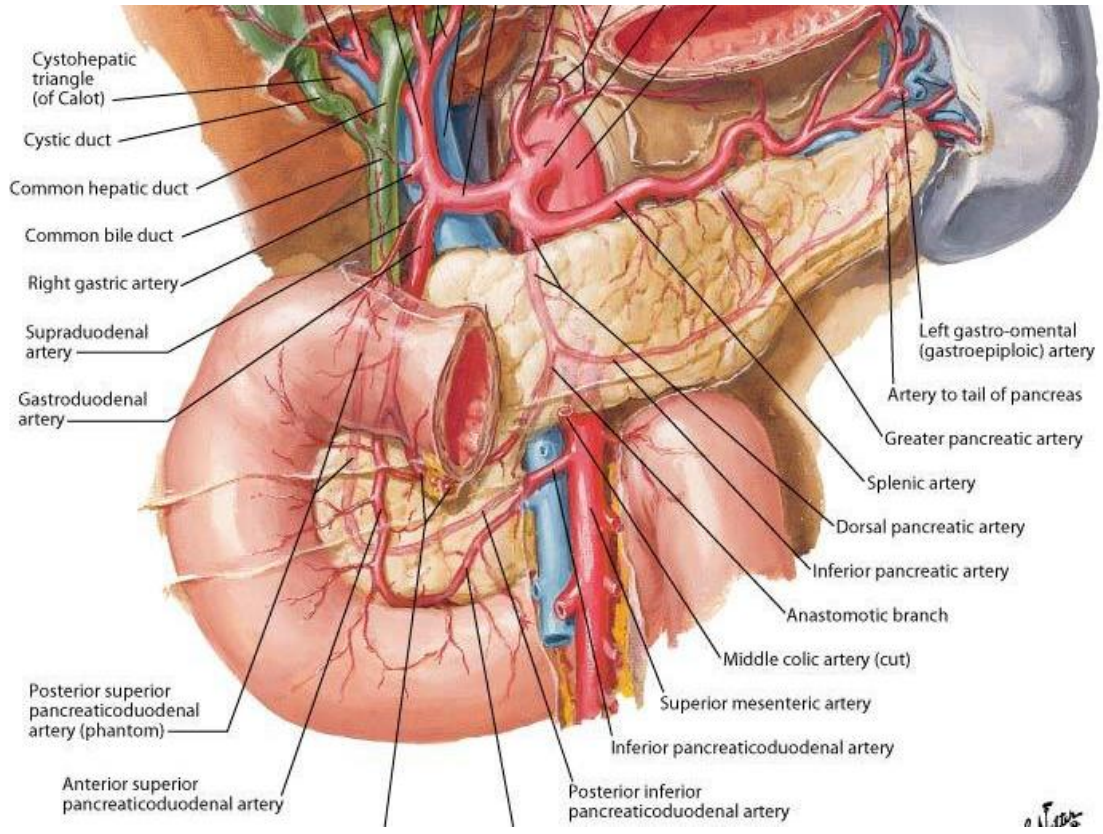


SPHINCTER OF ODDI

ARTERIAL SUPPLY OF PANCREAS

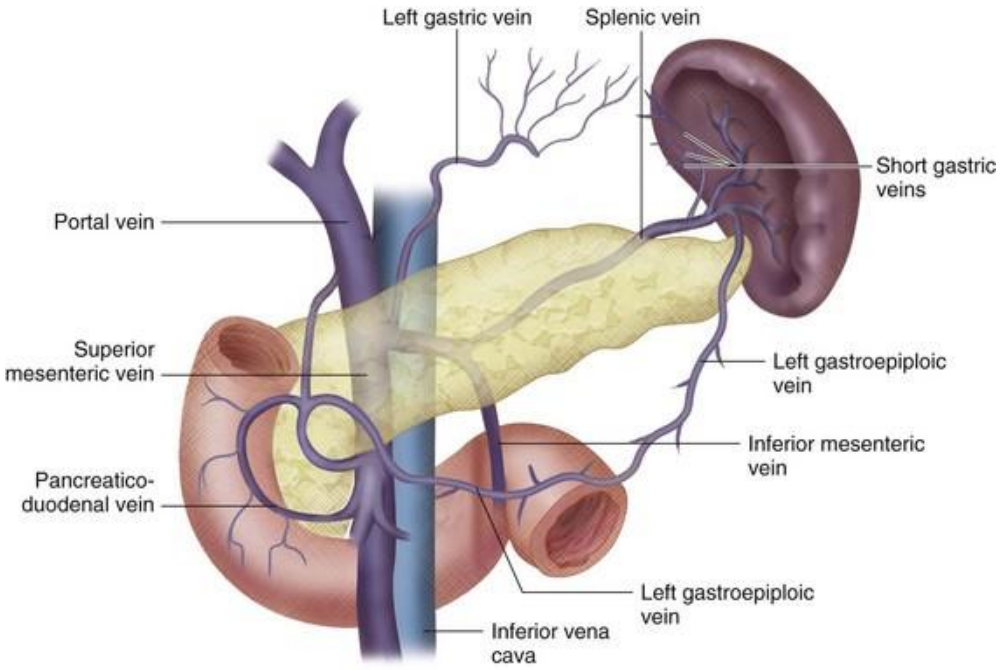
Arterial supply of pancreas is through the celiac trunk and superior mesenteric arteries, in turn through arterial arcades formed within the body and tail of the pancreas. The celiac trunk gives off splenic and common hepatic arteries. The dorsal and greater pancreatic arteries arise from the splenic artery, the gastroduodenal artery arises from the common hepatic artery, then dividing into anterior and posterior superior pancreaticoduodenal branches anastomosing with the anterior and posterior branches of the inferior pancreaticoduodenal artery, branches of the superior mesenteric artery.

ARTERIAL SUPPLY OF PANCREAS



VENOUS DRAINAGE OF PANCREAS

Venous drainage of the pancreas is similar to its arterial supply. The anterior and posterior pancreatico-duodenal veins drain the head of the pancreas. The posterior-superior pancreatico-duodenal vein enters the superior mesenteric vein, laterally at the superior border of neck of the pancreas. The anterior and poster inferior pancreatico-duodenal veins drains in the superior mesenteric vein, along the inferior border of the uncinate process. The splenic venous system drains the body and tail of pancreas.



VENOUS DRAINAGE OF PANCREAS

LYMPHATIC DRAINAGE OF PANCREAS

Pancreas has a diffuse lymphatic drainage. The network of lymphatics of pancreas are formed near the major vessels. The superior lymphatics, run closely with the splenic vessels and the inferior lymphatics along inferior pancreatic artery. The left side of the body and tail of pancreas, drain into lymph nodes in the hilum of spleen. The right side of the body and the neck of pancreas, drain into lymph nodes near the superior border of the head. They also receive tributaries from the anterior and posterior surfaces of the pancreas.

HISTOLOGY

The Pancreatic mass is composed of 85% of exocrine mass, 10% of the gland is extracellular matrix, 4% by blood vessels and the major ducts and 2% of the gland is endocrine tissue.

Exocrine pancreas

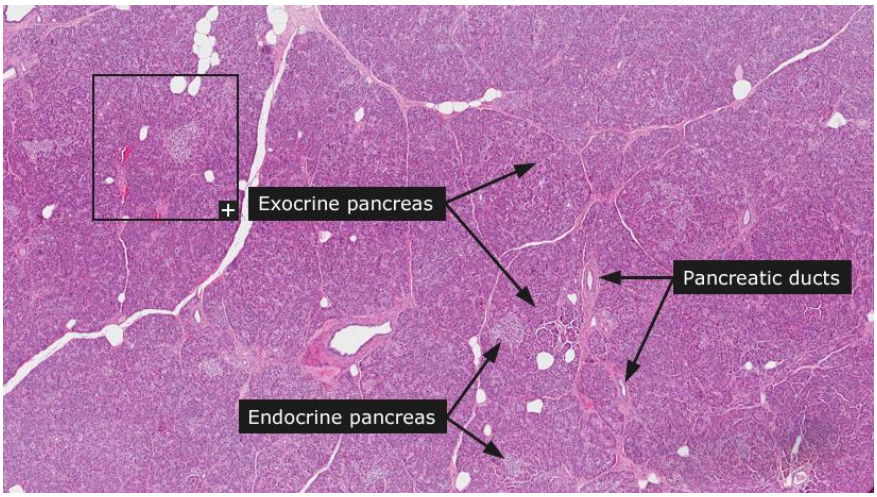
The Exocrine pancreas is highly branched and lobulated ductal system that terminates in secretory acini. The smallest ducts are termed as intercalated ducts. Intercalated ducts merge into intralobular ducts which merge into interlobular ducts.

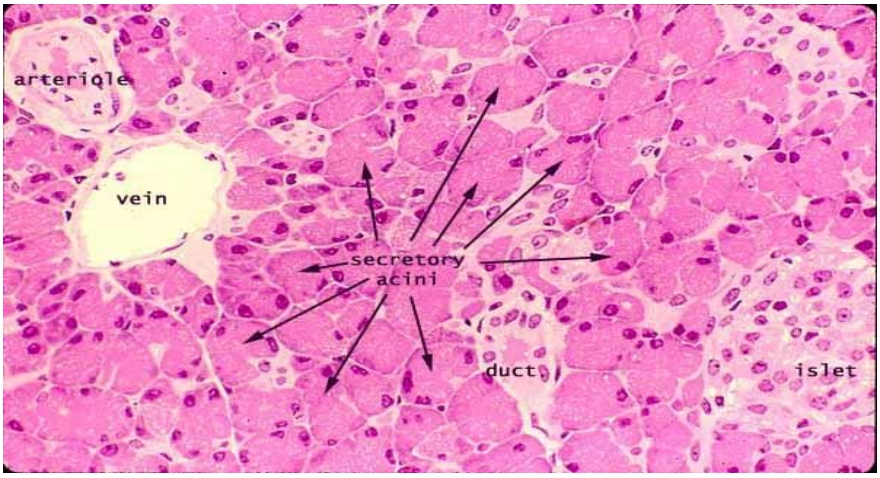
Interlobular ducts drain into the main pancreatic duct which releases the secretions of the exocrine pancreas into the duodenum.

Pancreatic acinar cells are pyramidal shaped that line the terminal pancreatic acini and primarily secrete pancreatic zymogens stored in cytosolic granules. Pancreatic ductal cells line the pancreatic ducts and primarily secrete an aqueous bicarbonate-rich fluid.

Endocrine Pancreas

The Endocrine pancreas (2%) is formed of three functionally distinct polygonal cell types, Alpha Cells (25%), Beta Cells (60%), and Delta Cells (5%), which can only be distinguished using special stains and secrete distinct peptide hormones. Alpha cells secrete glucagon, Beta cells secrete insulin, and Delta cells secrete somatostatin.





PHYSIOLOGY

The ***Exocrine mass*** secretes about 600-800ml of odourless and alkaline pancreatic juice. Pancreatic juice consists of amylase, protease and lipase which help in dissolution of carbohydrate, protein and fat from its complex form into absorbable forms. The alkaline pH is due to the presence of bicarbonate ions, and helps to neutralize the acid chyme from the stomach.

Pancreatic amylase is the only enzyme in active form. It hydrolyses starch and glycogen into glucose, maltose, maltotriose and dextrin. Hydrolysis of these ingredients into glucose is accomplished by the brush border enzymes of the enterocyte.

Trypsinogen is a proenzyme whose activation in the pancreas is prevented by inhibitors secreted by acinar cells called serine protease inhibitor Kazal type I. Failure of

inhibitor expression due to mutation in PRSS1 gene leads to familial pancreatitis. Trypsin activates multiple enzymes such as elastase, carboxypeptidase and phospholipases.

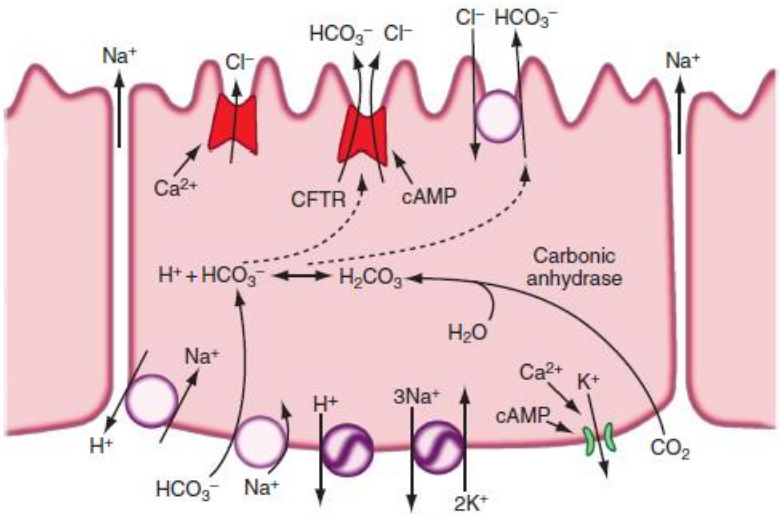
Pancreas secretes three lipases:

1. Lipase (or TG lipase)

2. Phospholipase A2,

3. Carboxylesterase

Triglyceride molecules are broken down by pancreatic lipase down into two fatty acid molecules. Colipase is believed to form a complex with lipase and bile salts.



BICARBONATE SECRETION BY PANCREAS

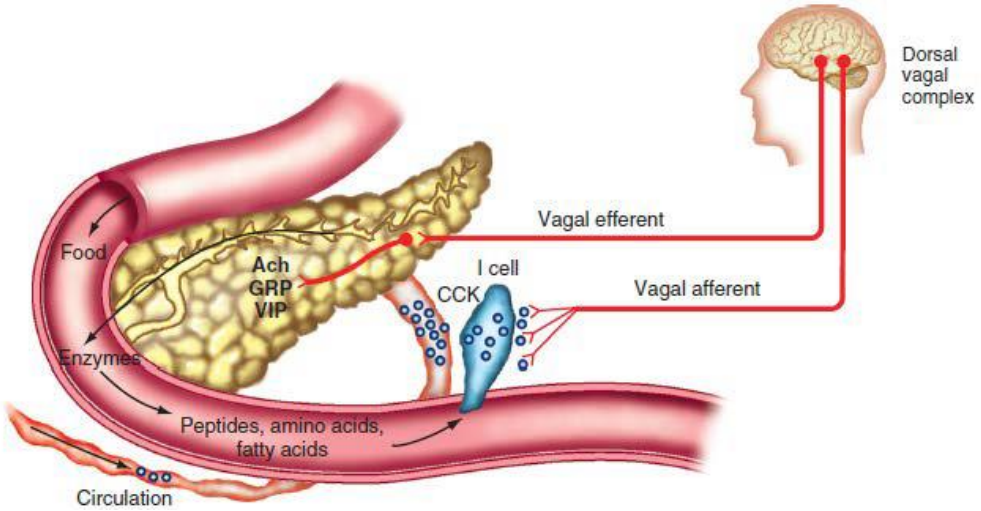
Phases of secretion

Cephalic phase – Visual and gustatory stimulation causes vagal mediated activation of acinar cells to release digestive enzymes. It is low in bicarbonate content.

Gastric phase – Due to gastric distension by ingested food causing vagal mediated release of digestive enzymes.

Intestinal phase – It is both hormonal and vagal mediated. The amount of bicarbonate secretion by ductal cells is high in response to secretin. The release of secretin is in turn dependent upon the pH of the delivered to the duodenum.

Cholecystokinin is an important mediator for secretion in intestinal phase



Endocrine pancreas is composed of Islets of Langerhans cells. They vary in size ranging from 40-900 nm and are located near major arterioles. The five major categories

of cell are alpha cells, beta cells, delta cells, epsilon cells and PP cells. These cells produce the vital hormones such as insulin, glucagon, somatostatin, ghrelin and PP. The functions of these hormones are

Glucagon - increased hepatic glycogenolysis and gluconeogenesis

Insulin- Decreased gluconeogenesis, glycogenolysis, fatty acid breakdown, and ketogenesis, increased glycogenesis, protein synthesis

Somatostatin- Inhibits GI secretion, secretion and action of all GI endocrine peptides, Inhibits cell growth

Ghrelin- Decreases insulin release and insulin action

Pancreatic Polypeptide- Inhibits pancreatic exocrine secretion and section of insulin, facilitates hepatic effect of insulin

ACUTE PANCREATITIS

Acute pancreatitis refers to an acute inflammatory process involving the exocrine pancreatic parenchyma. Pancreatitis occurs due to release of pancreatic enzymes within its own parenchyma resulting in auto digestion. Varied etiological factors cause injury to acinar cells thereby causing premature activation and release of enzymes into its own parenchyma. Patients may present from mild abdominal discomfort to multi-organ

failure. It ranges from a mild self-limiting inflammation to critical disease leading onto infected pancreatic necrosis, multiple organ failure and has a high risk of mortality. The clinical outcome has improved across time because of a more targeted approach to diagnosis, monitoring and management. The diagnosis of acute pancreatitis is to be considered when the patient presents with abdominal pain radiating to back, a threefold increase in serum amylase and lipase values from the baseline and radiological features suggestive of acute pancreatitis.

ETIOLOGY

Acute pancreatitis is caused by a wide spectrum of etiological factors, understanding them is vital to the treatment catered to the patient. Approximately 80% of patients suffering from acute pancreatitis is caused due to either alcohol or gallstone disease.

The average time of presentation is around third to fourth decade for alcoholics and drug induced pancreatitis while it presents later in fifth decade in gallstone. Incidence in males is higher due to more propensity to alcohol while females are more affected due to formation of gallstones.

ALCOHOL – Ethanol is among the leading factors that cause acute pancreatitis in young males. Smoking has been established as an addictive factor in the pathogenesis by a factor of 4.9 when compared to non-smokers The Amount and drinking pattern have found to be directly related to acute pancreatitis rather than the type of alcohol ingested. Ethanol is a metabolic toxin that causes a secretory burst in acinar cells combined with

spasm of the sphincter of Oddi inciting acute pancreatitis. Ethanol also increases ductal permeability, which allows prematurely activated enzymes to seep into the pancreatic parenchyma. It increases the protein content of pancreatic juice, decreases bicarbonate levels, and trypsin inhibitor concentration. Protein plugs formation due to calcium also contribute by causing an obstructive element to pancreatic outflow.

BILIARY TRACT DISEASE – Gallstones that are smaller in diameter (<5mm) are more likely to travel down and get lodged in the distal part of the Ampulla of Vater which might allow bile to reflux into the pancreatic duct. It might also be due to incompetence caused by stone passage through the sphincter allowing duodenal fluid and bile to reflux into the pancreatic duct. A third proposition is ductal hypertension due to a gallstone obstructing the pancreatic duct. The backpressure caused by ductal hypertension leads to minor ductal disruption causing extravasation of pancreatic juice and its subsequent activation.

HEREDITARY PANCREATITIS - An autosomal dominant disorder caused by mutation in cationic trypsinogen gene (PRSS1) leading to premature activation of trypsinogen and abnormal ductal secretory mechanisms. Mutations in SPINK1 protein blocks the binding site causing release of inhibition over trypsin leading to acute pancreatitis.

TUMORS – In patients over 40 years, tumors of pancreatic or periampullary region may present with acute pancreatitis. Intraductal papillary mucinous neoplasm (IPMN) is commonest to present with pancreatitis. In elderly patients with idiopathic pancreatitis, pancreatic tumors should be considered.

DRUGS – It is a rare etiology accounting for <1% acute pancreatitis patients. The mechanisms implicated being a hypersensitivity reaction, occurring 4-8 weeks after a drug initiation. Secondly, accumulation of metabolite of the drug which might present several months after drug intake and some drugs have been found to be intrinsically toxic to the pancreatic parenchyma.

Acetaminophen	Ifosfamide
5-Aminosalicylic acid compounds	Interferon-α
Sulfasalazine	Isoniazid
Azodisalicylate	Lamivudine
Mesalamine	Lisinopril
L-Asparaginase	Losartan
Azathioprine	Meglumine
Benazepril	Methimazole
Bezafibrate	Methyldopa
Cannabis	Metronidazole
Captopril	6-Mercaptopurine
Carbimazole	Nelfinavir
Cimetidine	Norethindrone/mestrol
Clozapine	Pentamidine
Codeine	Pravastatin
Cytosine arabinoside	Procainamide
Dapsone	Pyritinol
Didanosine	Simvastatin
Dexamethasone	Sulfamethazine
Enalapril	Sulfamethoxazole
Erythromycin	Stibogluconate
Estrogen	Sulindac
Fluvastatin	Tetracycline
Furosemide	Trimethoprim/ sulfamethoxazole
Hydrochlorothiazide	Valproic acid
Hydrocortisone	

HYPERTRIGLYCERIDEMIA **-** It is the third most common cause of pancreatitis, (2% to 5% of cases). Serum triglyceride levels more than 1000 mg/dL with obvious latescent (milky) serum due to increased concentrations of chylomicrons can result in attacks of acute pancreatitis. The mechanism is not clear, but damage to pancreatic acinar cells or endothelial cells is by the release of free fatty acids by lipase. The hydrolysis of TGs releases free fatty acids that induce free radical damage to cell membranes. Disorders of lipoprotein metabolism are conventionally divided into primary (genetic) and secondary causes, including diabetes mellitus, hypothyroidism and obesity/metabolic syndrome.

HYPERCALCEMIA**-** Hypercalcemia is implicated with acute pancreatitis. Proposed mechanisms include calcium salts deposition into pancreatic duct and calcium mediated premature activation of trypsinogen within the pancreatic parenchyma. Primary hyperparathyroidism causes acute pancreatitis (less than 0.5% of all cases). Hypercalcemia can also be caused by metastatic bone disease, TPN, sarcoidosis, vitamin D toxicity and high dose calcium infusion in cardiopulmonary bypass.

INFECTION - Viruses (*mumps, hepatitis A, B and C, coxsackievirus, herpesviruses, cytomegalovirus, herpes simplex, varicella-zoster and Epstein Barr virus*), MMR vaccine; bacteria (*Mycoplasma, Leptospira, Salmonella, TB, Legionella and brucellosis*); fungi (*Aspergillus, Candida*) and parasites (*Toxoplasma, Ascaris , Cryptosporidia, lumbricoides, Clonorchis sinensis*). Obstruction of the Duct of Wirsung by parasites such as *C.sinensis and A.lumbricoides* can cause pancreatitis.

VASCULITIS – It is a rare cause which may present from mild to life threatening forms of acute pancreatitis. SLE and polyarteritis nodosa can cause vasculitis of parenchymal vessels leading to acute attacks.

TRAUMA – Blunt injury to the pancreas is more common than penetrating injuries. Blunt injury causes compression of pancreas against the spine leading to ductal disruption and enzymatic release.

IATROGENIC – Acute pancreatitis can be caused in many surgical interventions such as ERCP, retroperitoneal lymph node dissection, removal of GIT (distal gastrectomy, colectomy) and nephrectomy. Approximately 5%-10% of post ERCP patients suffer from acute pancreatitis. Sphincter of Oddi dysfunction and high-pressure contrast increase the risk of an attack.

POST-SURGICAL – Surgeries involving the thoracic and abdomen cavity can cause pancreatitis in the post-surgical period. Cardio-pulmonary bypass has high chance of causing hyperamylasemia which can lead to necrotizing pancreatitis. Liver transplantation may rarely cause pancreatitis.

PANCREATIC DIVISUM – It is a congenital maldevelopment of pancreas where the minor and major papilla fail to fuse with each other. Minor papilla obstruction and accumulation of secretions within the duct has been proposed to cause pancreatitis.

MISCELLANEOUS – Other rare causes include Sphincter of Oddi dysfunction, Crohn's disease, Celiac disease, severe burns and autoimmune conditions. Smoking has been

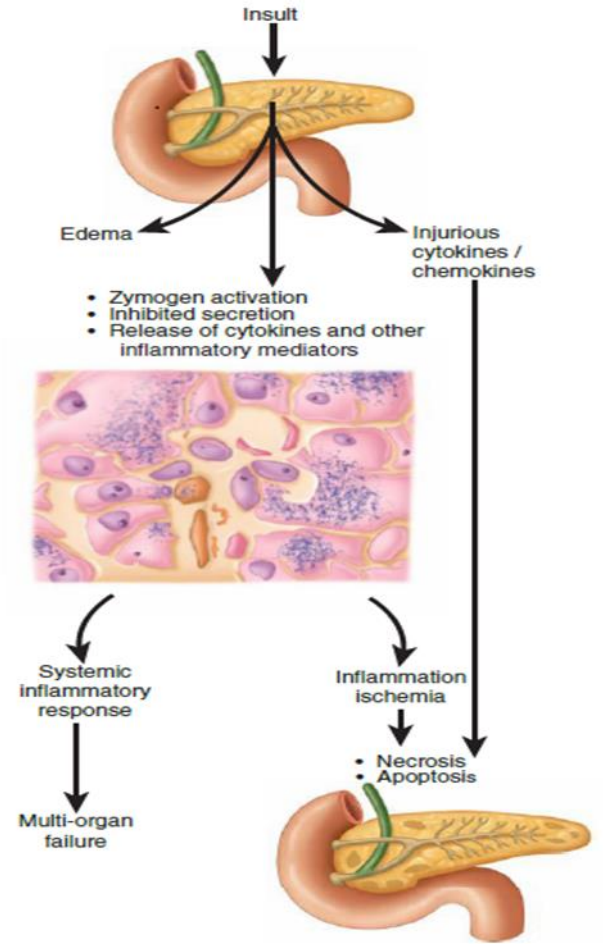
found to be an additive factor. Disruption of musical barrier has been thought to cause hyperamylasemia in these conditions.

PATHOPHYSIOLOGY

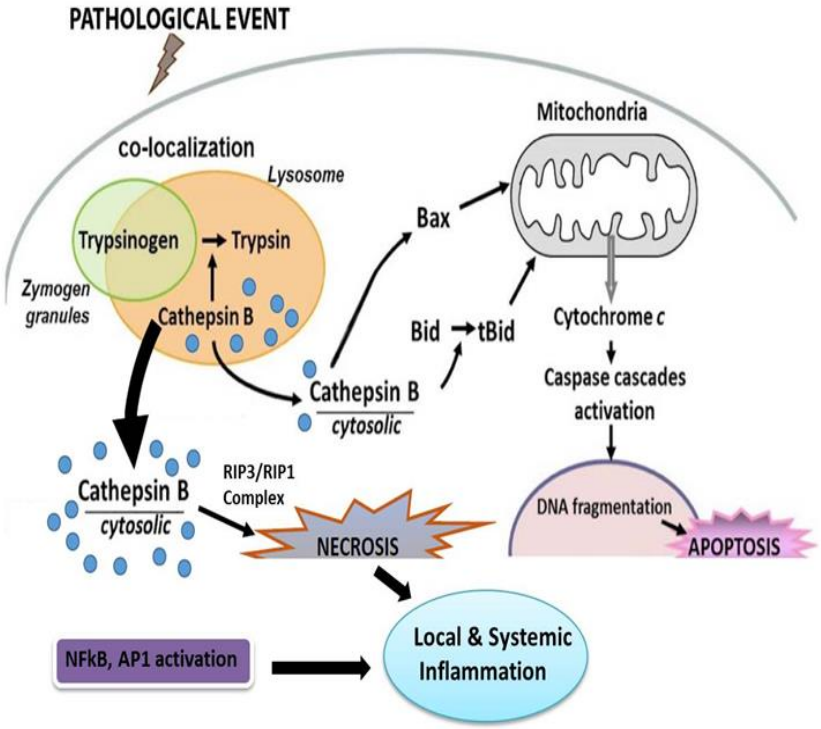
The Exocrine part of pancreas is responsible for secretion of major enzyme, Trypsin. The enzyme trypsin is an activator of phospholipase A2 (PLA2), carboxypeptidase and elastase. The pancreas protects itself from the destructive action of trypsin in a three-layered defense. Trypsin is formed and stored in its precursor form, trypsinogen. It is converted into its active form by the action of endopeptidase found in the brush border cells of duodenum. Pancreas protects itself by separating the area of enzyme production and site of action. The digestive enzymes created are stored in membrane bound organelles are called zymogen granules. The release of enzymes from zymogen granules is initiated by combination with lysosome containing cathepsin B. Studies has implicated that minor trauma can cause activation of cathepsin without secretion of enzymes, thus causing self-activation of enzymes within the cells of parenchyma. Pancreas provides a third barrier of protection by production of trypsin inhibitors.

Disruption of these protective barriers by various etiological factors causes activation of enzymes within the normal pancreatic parenchyma leading to

auto-digestion. Insult to the acinar cells causes activation of trypsin by cathepsin B within the co-localization vacuoles. Cathepsin and trypsin enter the cytoplasm and cause increased permeability of mitochondria and dissolution of cellular components releasing cytochrome C resulting in apoptotic cell death. Inflammatory process initiated by the acinar cell destruction attracts macrophages to be accumulated. Pro-inflammatory mediators like TNF alpha and interleukins 1 and 6 are produced by the macrophages. The cascade of events lead to ductal obstruction and acini barrier disruption which facilitates flow of pancreatic secretion into the interstitium resulting in a viscous cycle.



Vascular permeability increases in inflammation leading to the development of interstitial edema. Micro circulatory failure further exacerbates the injury to pancreas. Release of free radicals, C5a and activation of complement pathway results in reperfusion injury to pancreas. Inflammation of pancreas causes release of cytokines into the portal circulation, upon reaching the liver, Kupffer cells amplify the production of cytokines into the systemic circulation. Acute phase protein synthesis (C-reactive protein [CRP], IL-6) is increased causing SIRS and organ damage leading on to MODS and death.



Lecithin, an important constituent of pulmonary surfactant is cleaved by activation of phospholipase-A culminating in ARDS. Acute renal failure may be the result of hypotension and decreased intra vascular volume. Depression of myocardium and shock may be due to release of vasoactive peptides and a myocardial-depressant factor. Metabolic complications will include hyperlipidemia, hypocalcemia, hyperglycemia with or without ketosis and hypoglycemia.

CLINICAL FEATURES

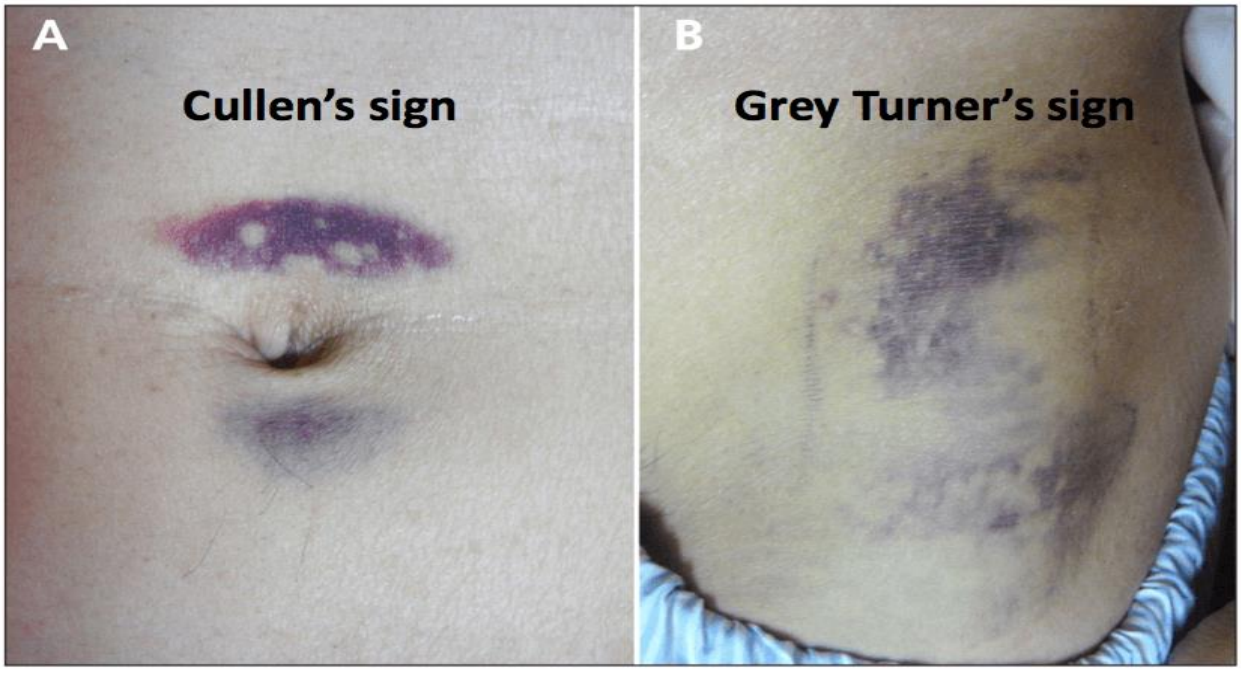
Abdominal pain is the cardinal symptom of presentation. Patients usually present with pain over the epigastric or right hypochondriac region. Sometimes the pain is diffusely present over the abdomen. Pain may mimic myocardial infarction when localized to lower chest or left hypochondrium. Onset of pain is rapid following ingestion of alcohol or a meal. Intensity of pain keeps increasing over time.

Pain is usually described to be knifing type or boring to the back type. Painless presentation may be fatal. Pain is relieved by leaning or stooping forwards. It is due to shifting of abdominal contents and reducing pressure over the inflamed pancreas. Pain is usually accompanied by nausea, retching and repeated vomiting.

Examination of the patient shows a variable consciousness level (oriented, confused, agitated, comatose) based on the severity of disease. Elevation in temperature occurs due to ongoing inflammatory process. Due to third space losses, the hydration

status is often reduced. Tachycardia and tachypnea occur as disease progresses. Hypotension occurs due to circulatory volume reduction and depression of myocardium. ARDS causes shallow type of breathing which occurs due to abdominal exudates or pleural effusion. Tenderness and guarding may be felt over the epigastric region. Bowel sounds may be decreased or absent because of ileus.

Discoloration of abdomen over flanks (Grey Turner sign) or around the umbilicus (Cullen's sign) occurs due to hemorrhagic complications. Patients may also present with panniculitis or subcutaneous nodular fat necrosis accompanied by polyarthritis. Fetor hepaticus, Spider Angioma, Dupuyten's contracture, ascites point towards ethanol induced pancreatitis. Tendon xanthoma and lipemia-retinalis are seen in hyperlipidemia induced pancreatitis. Band keratopathy may also be seen.



DIAGNOSIS

SERUM AMYLASE

Pancreatic injury causes increase in amylase level. Evaluation of serum amylase is cheap and a threefold increase over the baseline value helps in suggesting pancreatitis. It raises within 6 hours of onset of disease and is cleared through renal system. It has a half-life of 10 hours. Amylase level increases from onset of disease and persists usually for 3-5 days. The sensitivity and specificity of serum amylase levels in correlation to pancreatitis is low. In chronic pancreatitis, due to repeated destruction of acinar cells, an acute episode may not present with increase in serum amylase levels. Around half of patients with elevated serum amylase levels may be asymptomatic.

SERUM LIPASE

Serum lipase and serum amylase share a similar sensitivity level for the diagnosis of acute pancreatitis. However, serum lipase is not affected by other causes of hyperamylasemia, hence tends to have a higher specificity in diagnosing acute pancreatitis. Serum lipase level raises from the first day of disease, and it remains increased for a longer interval, thus providing a higher sensitivity.

ROUTINE BLOOD INVESTIGATIONS

The Total leukocyte count is markedly elevated as disease progresses and further increases in the presence of infection. The blood glucose also may be high or low in response to varying levels of serum glucagon and insulin. Gallstones induced pancreatitis

presents with elevated liver enzymes (AST, ALT and ALP) and bilirubin. Hypocalcemia seen in patients with acute pancreatitis is mainly related to the decreased serum albumin. MCV shows some variation in ethanol and non-ethanol related causes of acute pancreatitis. Alcoholic patients tend to have higher MCV due to the toxic effects of alcohol on the marrow. Hematocrit values are raised based on severity of disease and third space losses

TRYPSINOGEN AND TRYPSINOGEN ACTIVATED PEPTIDE

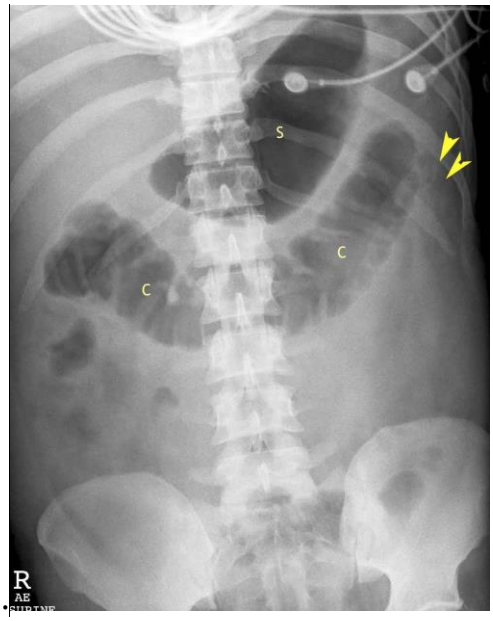
Measurement of urinary concentrations of trypsinogen activated peptide by a manual enzyme immunoassay method shows a good correlation with severity of acute pancreatitis on admission. The limited stability of the TAP assay and unavailability of a test kit restricts its usage as an emergency room test.

IMAGING

Plain X-ray

Based on the severity of disease and exudate level in abdomen findings may vary from no specific finding to focal ileus to colon cut off sign in severe disease. Spread and location of exudates from the pancreas determines the appearance of hollow viscus. Exudate in the lesser sac causes forward displacement of the stomach separating its contour with the transverse colon. Inflammation near the small bowel mesentery presents with small bowel abnormalities including ileus of one or more loops of jejunum (the sentinel loop), the distal ileum or cecum or the duodenum. Generalized ileus occurs as

disease progresses to a severe nature. Spread of the exudate over the colon produces spasm of that area of the colon with no air distal to the spasm (the colon cut-off sign) and dilated colon proximal to the spasm



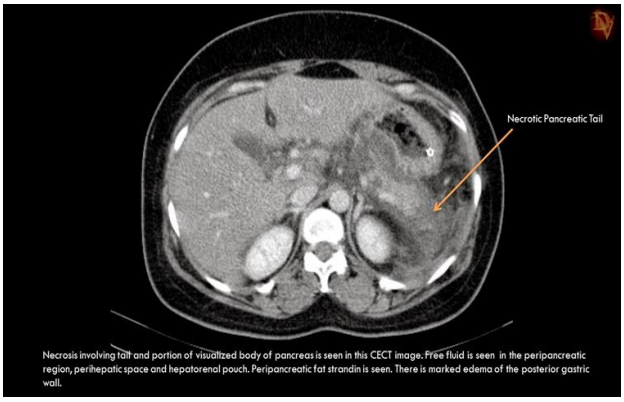
COLON CUT OFF SIGN

Ultrasound Abdomen

Pancreas being retroperitoneal in position is obscured by bowel gas and is not usually visualized, unless grossly enlarged or edematous. Abdominal ultrasound is useful to identify gallstones, choledocholithiasis, CBD diameter and ascites. Ascites is commonly seen in patients with moderate to severe pancreatitis, as protein rich fluid extravasates from the intravascular compartment to peritoneal cavity. Size of pseudocyst can be serially monitored by ultrasound.

CECT Abdomen

Diagnosis of acute pancreatitis, its severity and various intraabdominal complications can be assessed by CECT abdomen. Depending on the perfusion of the pancreas, CECT can diagnose normal pancreas, interstitial and necrotizing variants of pancreatitis. CECT scanning usually fails to identify pancreatic necrosis in the early stages of disease.

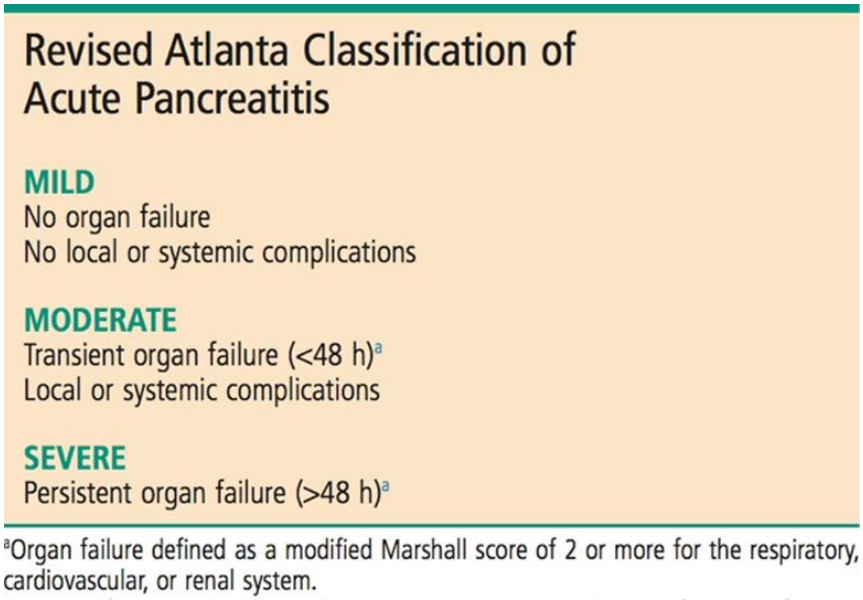


Inflammatory process – Balthazar's morphological index for acute pancreatitis		
Grade	Tomographic finding	Scoring
A	Normal pancreas.	0
B	Focal or diffuse pancreatic enlargement.	1
C	Pancreatic alterations associated with peripancreatic inflammation.	2
D	Single fluid collection.	3
E	Two or more fluid collections and/or presence of gas within the pancreas or within peripancreatic inflammation.	4
Pancreatic necrosis		
Tomographic finding		Scoring
Absence of necrosis.		0
< 30% necrosis.		2
30% to 50% necrosis.		4
> 50% necrosis.		6

CLASSIFICATION OF SEVERITY

Over the decades many classifications such as Marseille, Cambridge, Revised Marseille were devised to assess the severity of pancreatitis. In the last decade the assessment of severity was followed by guidelines based on Atlanta classification of 1992. Due to scientific advancement, the better understanding of pathophysiological process of pancreatitis lead to the revision of Atlanta classification in 2012, and is popularly used nowadays.

Atlanta classification 1992 categorized pancreatitis into mild and severe. Severe disease is defined by the presence of organ failure, local pancreatic complications on imaging (acute fluid collection, pancreatic necrosis, pseudocyst and pancreatic abscess), and/or poor prognostic scores (Ranson's ≥3 and/or APACHE II ≥8).The Revised Atlanta classification (2012) divides severity of acute pancreatitis into three groups: mild, moderate, and severe



SCORING SYSTEMS

RANSON'S SCORE

Severity of the pancreatic disease is calculated based on 11 parameters, obtained at the time of admission and/or 48 hours later. The mortality rate of acute pancreatitis, directly correlates with the number of parameters positive. If three or more criteria are fulfilled diagnosis of severe pancreatitis is made.

Ranson (alcoholic or other)	Ranson (biliary)
At admission	**At admission**
Age >55 y	Age >70 y
GB > 16 000/mm^3	GB > 18 000/mm^3
LDH > 350 U/l	LDH > 250 U/l
AST > 250 U/l	AST > 250 U/l
Glycemia >200 mg/dl	Glycemia >220 mg/dl
In 48 h	**In 48 h**
Drop in hematocrit > 10%	Drop in hematocrit > 10%
BUN increase >5 mg/dl	BUN increase >2 mg/dl
Calcium <8 mg/dl	Calcium <8 mg/dl
PO^2 <60 mmHg	PO^2 <60 mmHg
Bases deficit >4 mEq/l	Bases deficit >5 mEq/l
Fluid loss >6L	Fluid loss >4L
Each item worth 1 point (0 a 11 points)	

IMRIE'S PROGNOSTIC CRITERIA:

It is calculated during initial 48 hours, using the following parameters scored with one point each. A score of 3 or more was termed sever pancreatitis and values above 8 had a mortality of 50%

WBC count > 15000/mm3

Blood sugar > 10 mmol/L

Serum urea > 16 mmol/L (no response to IV fluids)

Po2 level < 60 mm Hg

Serum ca2+ level < 2 mmol/L

Lactic dehydrogenase> 600 IU/L

AST / ALT>200 IU/l

Serum albumin level < 32 g/L

MODIFIED GLASGOW CRITERIA:

This one was useful in both alcoholic and biliary pancreatitis.

The score ≥ 3 means severe disease requires ICU care.

PaO2 <8kPa or < 60 mm Hg

Age more than 55 years old

Neutrophilia with WBC count>15x109/L

Ca2+<2mmol/L or < 8 mg/dL

Urea >16mmol/L or > 45 mg/dL

Serum LDH >600 IU/L; AST>200 IU/L

Albumin <3.2g/dL

Sugar: >10mmol/L or >180 mg/dL

APACHE II SCORING

Acute Physiology and Chronic Health Evaluation (APACHE II) score is the most widely used scoring system in acute pancreatitis especially patients requiring ICU care. It has good negative predictive value and positive predictive value, in predicting severity of acute pancreatitis and can be performed daily. Decreasing values during the first 48 hours will suggest a mild attack, whereas increasing values suggest a severe attack. Studies suggest that mortality is less than 4% with a score < 8 and upto 18% with a score > 8.

APACHE II provides a general measure of the severity of disease, based on the patient's age, previous health status, and 12 routine physiologic measurements. An APACHE II score of 8 or more, defines severe pancreatitis. It has the advantage of be

used on a daily basis and has similar positive and negative predictive values as the Ranson score at 48 hours after admission.

The major advantage of the APACHE II scoring system, when compared to the other systems, is that, it can be used in monitoring patient's response to therapy.

The APACHE-II system assigns points for 12 physiologic variables, for age, and for chronic health status, in generating a total point score.

The 12 variables are

1. Temperature

2. Heart rate

3. Respiratory rate

4. Mean arterial blood pressure

5. Oxygenation

6. Arterial pH

7. Serum potassium

8. Serum sodium

9. Serum creatinine

10.Hematocrit

11.WBC count

12.Glasgow Coma Scale

The laboratory tests which are required are simple, routine and readily available.

APACHE-II scores on admission and within 48 hours help distinguish mild from severe pancreatitis and to predict death. Most patients with APACHE-II scores of 9 or less during the first 48 hours have a low mortality. Patients with APACHE-II scores of 13 or more have a high mortality ratio. At admission, sensitivity is 34% to 70%, and specificity is 76% to 98%. At 48 hours, sensitivity remains less than 50%, but specificity is close to 90% to a Score of ≥ 2 indicates presence of organ failure.

BISAP SCORE

The BISAP created recently includes:

1. Blood urea nitrogen (BUN) >25 mg / dl.

2. Impaired mental status (GCS < 15).

3. SIRS (Systemic Inflammatory Response Syndrome)

4. Age >60 years.

5. Pleural effusion

SIRS was defined by presence of two or more of the following criteria:

1. Pulse rate > 90/min.

2. Respiratory rate > 20/min or PaCO 2 < 32 mm Hg.

3. Temperature >100.4 F or < 96.8 F / < 36 or > 38 ° C.

4. WBC count >12,000 or < 4,000 cells/mm3, or presence of more than 10% immature blasts.

MANAGEMENT OF ACUTE PANCREATITIS

Patients diagnosed with acute pancreatitis require early and aggressive intravenous fluid replacement to maintain hemodynamic stability and to maintain perfusion of kidneys and pancreas.

Elimination or significant reduction in pain by providing adequate analgesia is of prime importance. Abdominal pain can be treated with opiate analgesics in a controlled-anesthesia pump. The patient is usually kept nil per oral until any nausea and vomiting have subsided. Opiate dosage is monitored carefully and adjusted on accordance to patient's condition. Studies show that morphine increases the tone of sphincter of Oddi, and serum amylase levels, its use in treating the pain in acute pancreatitis has not been shown to affect outcome adversely.

Nasogastric intubation is not routinely used. It is only used to treat gastric ileus or intractable nausea and vomiting. Use of proton pump inhibitors or H2 receptor blockers have been shown to be minimally beneficial. The patient should be monitored carefully for signs of early organ failure like hypotension, pulmonary failure, or renal failure by

closely monitoring vital signs and urinary output. Tachypnea should not be assumed to be due to abdominal pain. Oxygen saturation should be monitored and, if needed, arterial blood gas measurement is performed, and also oxygen supplementation is mandatory in case of hypoxemia. Patients suspected with early organ dysfunction should be transferred immediately to an ICU, as clinical deterioration can be rapid and fatal.

FLUID RESUSCITATION

Various retrospective and prospective clinical trials and laboratory experiments recommend aggressive volume replacement in patients with acute pancreatitis. Early in the course of acute pancreatitis inflammatory process causes extravasation of protein rich intravascular fluid into the general peritoneal cavity as well as retroperitoneum (third space loss), resulting in hemoconcentration and reduced renal perfusion with associated elevation of blood urea nitrogen. The third space losses lead to reduced perfusion pressure in the pancreas results in microcirculatory changes which cause pancreatic necrosis. Hematocrit more than 44% at admission and a failure of initial hematocrit to decrease at 24 hours, have been shown to be predictors of necrotizing pancreatitis. An elevated or rising BUN value is associated with high mortality. Early and vigorous hydration by IV fluids help to restore intravascular volume. The goal is to provide adequate intravascular volume to reduce the hematocrit and blood urea nitrogen, thus increasing pancreatic perfusion.

Ringer lactate is the preferred solution for initial hydration. Due to its isotonicity, bicarbonate content and stable pH it helps prevent the development of metabolic acidosis. It should be recognized that aggressive early volume replacement requires caution in certain groups of patients (such as elderly patients or those with a history of cardiac and/or renal disease) to avoid complications, like volume overload, pulmonary edema, and abdominal compartment syndrome.

RESPIRATORY CARE

Hypoxemia (oxygen saturation <90%) requires oxygen supplementation, either by nasal prongs or by face mask. Endotracheal intubation and assisted ventilation maybe required in patients whose hypoxia fails to correct with nasal oxygen or if there is respiratory fatigue and borderline respiratory reserve. A Swan-Ganz catheter is used to differentiate hypoxemia due to congestive heart failure or due to primary pulmonary damage. Acute respiratory distress syndrome (ARDS), is the most serious respiratory complication of acute pancreatitis. ARDS is associated with severe dyspnea, worsening hypoxia, and ultimately resulting in death. It usually occurs between the second and seventh day of onset of disease (but can be present at admission) and consists of increased pulmonary alveolar capillary permeability resulting in interstitial edema. Treatment for ARDS includes endotracheal intubation with positive end-expiratory pressure ventilation, with low tidal volumes to protect the lungs from barotrauma.

NUTRITION AND ANTIBIOTICS

Antibiotics are not commonly used in cases of mild pancreatitis. Antibiotics such as Meropenem and Ciprofloxacin have been studied and found to be useful in patients suffering from pancreatic abscess or infected necrosis.

Artificial nutritional support is the mode of nutrition in patients having severe acute pancreatitis, especially with pancreatic necrosis. A period of 4 to 6 weeks of artificial nutritional support may be necessary. TPN is the standard method of refeeding patients with severe acute pancreatitis. Recent studies have shown that enteral nutrition decreases small bowel bacterial overgrowth, and helps to improve intestinal mucosal barrier function, thereby reducing bacterial translocation and resultant fulminant infectious complications. The optimal route for the administration of enteral feeding is either through a nasojejunal or a nasogastric tube. Enteral feeding is cheaper, safer and is the preferred method of nutritional support.

THERAPEUTIC ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY (ERCP)

Various studies have proved that early ERCP (within 24 or 48 hours of admission) reduces complications, but not mortality in patients with severe gallstone-associated acute pancreatitis. The risks of the procedure include an increase in the severity of

pancreatitis, cholangitis and duodenal perforation. ERCP examination of the bile duct is not encouraged in cases of biliary pancreatitis due to low possibility of finding residual stones. Patients suspected of harboring an impacted stone in the distal common bile duct or ampulla should have a confirmation by radiologic imaging (CT, magnetic resonance cholangiopancreatography, or endoscopic ultrasonography) before intervention is planned.

SURGICAL THERAPY

It is recommended that patients with gallstone pancreatitis (mild or severe) should undergo cholecystectomy as soon as the patient has recovered and the acute inflammatory process has subsided. It is mandatory that the patient is subjected to cholecystectomy before his discharge.

Other surgeries in pancreatitis include debridement of pancreatic necrosis (necrosectomy) or drain a pancreatic abscess. Pancreatic necrosis can either be sterile or infected necrosis. Sterile necrosis can be managed non-operatively because the mortality of this condition without surgery is less than 5%. However, surgical therapy of infected pancreatic necrosis carries a substantial mortality of 15%

The types of necrosectomy operations that are in practice include necrosectomy with closed continuous irrigation via indwelling catheters, necrosectomy with closed drainage without irrigation, or necrosectomy and open packing.

COMPLICATIONS

Complications are divided into local and systemic

LOCAL - Fluid collections, pancreatic ascites/pleural effusion, pancreatic pseudocyst, pancreatic necrosis, infected pancreatic abscess, hemorrhage/pseudo aneurysm

SYSTEMIC

A. PULMONARY - Pneumonitis, basal atelectasis, ARDS, Pleural effusion

B. CARDIOVASCULAR – Hypotension, hypovolemia, sudden arrest & death nonspecific ECG (ST-T wave) changes, pericardial effusion

C. HEMATOLOGIC – Hemoconcentration, Disseminated intravascular coagulopathy

D. GI hemorrhage - Acid peptic disease, Gastric erosion, Portal/splenic vein thrombosis

E. RENAL – Oliguria, Azotemia, Renal vessel thrombosis

F. METABOLIC - Hyperglycemic state, Hypocalcemic state, Hyperlipidemia, Metabolic encephalopathy, sudden loss of vision (Purtscher's retinopathy)

G. CENTRAL NERVOUS SYSTEM - Acute psychosis, Fat embolism occlusion, Alcohol withdrawal syndrome

H. FAT NECROSIS - Intra-abdominal saponification, subcutaneous tissue necrosis

DEFINITIONS, DESCRIPTION OF ACS AND MEASUREMENT TECHNIQUES APPROVED BY WSACS

Compartment syndrome is defined as the state of increased pressure within a closed anatomical space causing decreased tissue perfusion and thereby threatening the viability of concerned tissues. IAH affects multiple systems and vital organs, especially renal, cardiac, respiratory and nervous systems. Decreased tissue perfusion is associated with increased afterload, decreased preload and extrinsic compression leading to hypo perfusion and decreased end organ oxygen delivery. The cascade of events resulting due to pressure-volume deregulation in the abdomen is termed as Abdominal Compartment Syndrome. Thus ACS is not a disease but a syndrome with a group of specific sign and symptoms.

1.INTRA-ABDOMINAL PRESSURE

The Abdomen is a closed cavity with fixed rigid and flexible walls. The fixed walls are the pelvis, spine and the costal arch; the flexible walls being diaphragm and the abdominal wall. The pressure within the abdomen at any given time is determined by the elasticity of the flexible wall and the character of the abdominal contents. The abdominal contents are primarily fluid in character and relatively non-compressible. In accordance to Pascal's law, the pressure measured at any point in the abdominal cavity represents the IAP throughout the abdomen. Hence intra-abdominal pressure is considered as a steady state pressure present within the abdominal cavity. IAP varies with each respiratory cycle

it decreases with diaphragmatic relaxation during expiration and increases with diaphragmatic contraction at inspiration. Also, it is directly affected by the volume of the hollow viscera (which may be filled with fecal matter, liquid or air) and the solid organs, the presence of blood, ascitic fluid or other space –occupying lesions (gravid uterus or tumor) and conditions such as third space edema and burns which limit the expansion of the abdominal wall.

The Normal IAP ranges up to 7 mmHg. Few physiological conditions such as pregnancy and morbid obesity are associated with chronically elevated IAP. Change in body position, mechanical ventilation, sepsis, organ failure and recent abdominal surgery are associated with increased IAP.

2. ABDOMINAL PERFUSION PRESSURE

Mean arterial pressure (MAP) minus IAP is Abdominal Perfusion Pressure (APP). Arterial inflow and the resistance to venous outflow are the main factors influencing MAP and IAP. APP serves as a reliable predictor of visceral perfusion and a possible endpoint for resuscitation. Therefore, APP is a superior parameter in predicting patient survival in cases of IAH and ACS. An APP more than 60 mmHg has been associated with better patient prognosis.

3. FILTRATION GRADIENT

Renal filtration gradient (FG) is the mechanical force promoting filtration across the glomerulus. FG is calculated by the difference between the glomerular filtration

pressure (GFP) and proximal tubular pressure (PTP). PTP is assumed to be equal to IAP in cases of IAH.

GFP = MAP -2 x IAP

Hence changes in IAP will affect renal filtration and urine production rather than MAP. Oliguria is one of the first sign of increased IAH.

4. INTRA-ABDOMINAL HYPERTENSION (IAH)

In a healthy person, the normal IAP is <7 mmHg. The Accepted physiological upper limit by the WSACS is 12mmHg occurring in conditions such as morbid obesity, chronic obstructive pulmonary disease and pregnancy. This shows that exert external pressure to the diaphragm or the abdominal wall can cause elevated normal pressure.

IAH is the sustained or repeated pathological increase of the intra-abdominal pressure above 12 mmHg.

Grades of IAH according to the level of IAP

Grade I : 12 – 15mmHg

Grade II : 16 – 20mmHg

Grade III : 21 - 25mmHg

Grade IV : >25mmHg

Subclassification of IAH according to duration

Hyper acute: Elevation of IAP for a few seconds to minutes as in straining, sneezing, defecation, laughing, coughing

Acute: Elevated IAP present over a few hours, seen in surgical cases (e.g. intra-abdominal hemorrhage or trauma)

Subacute: Elevated IAP present over days and found in medical cases

Chronic: IAP elevation develops over months (E.g. pregnancy) or years

(E.g. Intra-abdominal tumour, chronic ascites, morbid obesity, cirrhosis, peritoneal dialysis). These patients may develop either acute or subacute IAH when severely ill

5. ABDOMINAL COMPARTMENT SYNDROME (ACS)

Critical IAP in majority of patients seems to be between 10 and 15mmHg. There is microcirculatory reduction in the blood flow at this pressure and it indicates the initiation of ACS. IAH progresses to ACS as end organ dysfunction develops.

ACS consists of the following triad

a. Pathological acute increase in IAP >20-25mmHg

b. Adverse effects on end-organ function

c. Beneficial effects as a result of abdominal decompression

ABDOMINAL COMPARTMENT SYNDROME - CLASSIFICATION

According to the cause and duration, ACS may be classified as Primary, secondary or recurrent

1. *Primary ACS* (abdominal or surgical ACS) – Presence of acute or subacute IAH which results from intra-abdominal cause.

2. *Secondary ACS* (extra-abdominal or medical) Presence of subacute or chronic IAH from conditions that require massive fluid resuscitation, e.g. major burns or septic shock

3. *Tertiary or recurrent ACS* – Presents with resurgence of ACS following resolution of a prior episode

TECHNIQUES TO MEASURE INTRA-ABDOMINAL PRESSURE

Several techniques were proposed for the measurement of IAP and thereby the diagnosis of ACS. Clinical examination is highly unreliable and an undependable diagnostic tool with a positive predictive value and sensitivity of 40 to 60%. Abdominal girth measurements are equally inaccurate. The use of radiological investigations such as abdominal ultrasound, X-Ray abdomen, X-ray chest or computerized tomography (CT) are also ineffective in the quantification of IAP and the diagnosis of IAH. However, these

investigations are used in the identification of the cause of IAH. Identification and diagnosis of IAH/ACS is made by frequent and accurate measurement of IAP. Measurement of IAP is an accurate, safe and cost-effective method for determining the presence of IAH and deciding on the treatment protocol.

Given the exceedingly favorable risk benefit ratio of "measurement and monitoring IAP vs the associated morbidity and mortality of ACS/IAH", certain recommendations have been laid down.

(1) A baseline IAP measurement should be obtained if two or more risk factors for IAH/ACS are present.

(2) If the patient is identified to have IAH, serial measurements of IAP should be made throughout the course of the patient's critical illness.

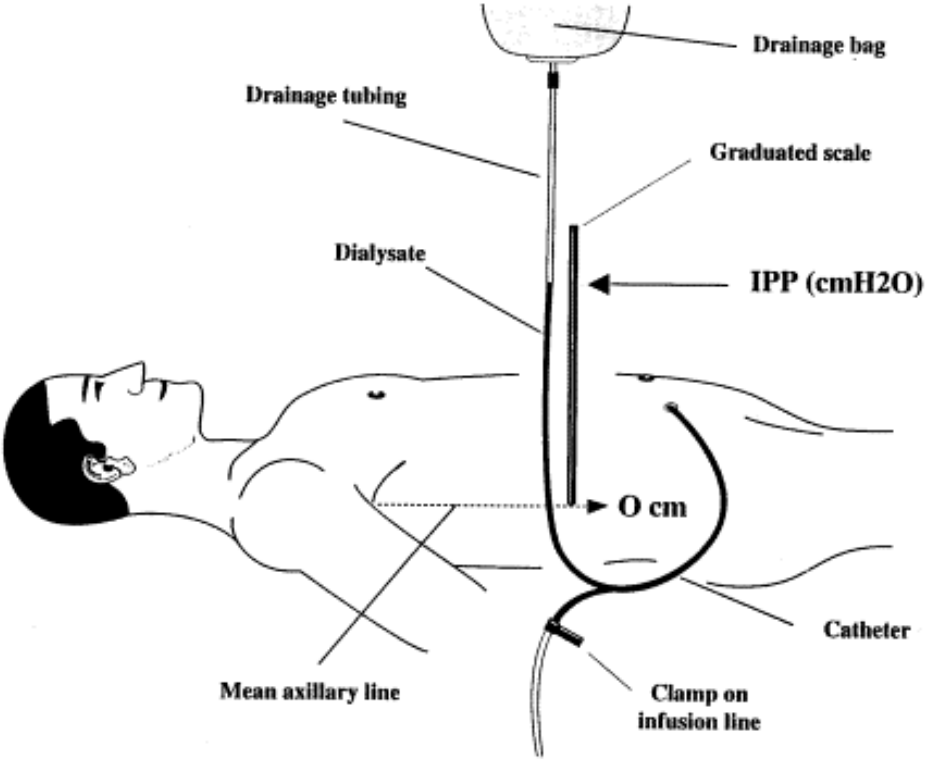
IAP can be measured intermittently or continuously, either directly or indirectly. An intra-peritoneal catheter installed during peritoneal dialysis, ascites drainage or during laparoscopic surgery can be used for obtaining the IAP directly.

Several indirect methods for obtaining IAP used are intravesical, rectal, gastric, uterine, inferior vena cava and airway pressure measurements. The intravesical route is considered the gold standard for IAP measurement and monitoring because of its simplicity and low cost. This technique is based on the fact that the wall of the bladder is very compliant. When a small amount of saline is infused, it functions as a passive reservoir and a transducer of the intra-abdominal pressure. Changes in the intravesicular

pressure reflect changes in the IAP sufficiently accurately for practical purposes. It is important to measure the IAP with the patient in the supine position as posture affects the IAP.

In patients with neurogenic bladder, bladder trauma, tense pelvic hematomas and outflow obstruction, the measurement of bladder pressure is not feasible. Hence an alternate method of measurement is used to monitor the IAP via the nasogastric route.

The intravesical route of measuring the IAP is performed by connecting the Foley's catheter to a three way tap which is then connected to a pressure transducer. The patient is placed in the supine position, he is catheterized using a Foley's catheter and the residual urine is drained. Later the Foleys catheter is clamped at a point distal to the point of pressure measurement. For every 20-degree head-up tilt, the IAP increases by 2mm. The catheter is connected to a pressure transducer and the point of mid-axillary line at the iliac crest is taken as the reference point where the intra-abdominal pressure value is zeroed. Around 25ml (if weight <20kgs, 1ml per kg) of saline is instilled into the bladder, 30 to 60secs later the reading is taken, providing time for detrusor muscle relaxation. Moreover, the measurement should be taken in the absence of active abdominal muscle contraction and at the end of expiration. Measurements are taken at regular intervals. Depending on the IAP the treatment modality is adjusted. As the Foleys catheter has to be clamped before each measurement, continuous monitoring of IAP using the intravesical route is challenging.



PATHOPHYSIOLOGY OF INTRA-ABDOMINAL HYPERTENSION/ ABDOMINAL COMPARTMENT SYNDROME

The increase in IAP critically affects the organs in the abdominal cavity as well other systems outside the abdomen. IAH and ACS mainly affect the regional blood flow. Progression from IAH to ACS is a graded response and not a binary effect.

According to WSACS, the recognition of IAH as an independent prognostic factor for critically ill patients will be gradually embedded in the "goal-directed" approach used in the ICU and will alter the decision-making process.

CARDIOVASCULAR SYSTEM

An increase in IAP causes upward displacement of the diaphragm. This decreases the intra-thoracic volume and thereby increases the intra-thoracic pressure (ITP). This is termed abdomino-thoracic transmission, and was seen in 20 to 80% of patients. ITP is generally assumed to be half of IAP. The increase in ITP compresses the heart directly, simultaneously reducing the ventricular contractility and compliance and significantly reducing the venous return resulting in deceased cardiac output. Compression of the aorta, pulmonary and systemic vasculature due to increase in IAP results in an increase in the afterload / systemic vascular resistance and concurrent activation of the renin-angiotensin-aldosterone pathway. This causes shunting of blood away from the abdominal cavity and leads to a temporary rise of MAP which later normalizes or even decreases. These effects occur with an IAP of 10mmHg in a normo-volemic patient; and at a lower IAP in a hypovolemic patient. Volume correction increases the preload temporarily, thereby improving the hemodynamics. It is also found that the application of positive end expiratory pressure (PEEP) aggravates the effects seen in the cardiovascular system.

The traditional intra-cardiac filling pressures such as Pulmonary Artery Occlusion Pressure (PAOP) and central venous pressure (CVP) are erroneously increased in IAH

due to abdomino-thoracic transmission of pressure. Hence these parameters cannot be used for monitoring the cardiac status of the patient. Both these cardiac parameters are the sum of ITP and intravascular pressure and not reflective of the true intra-vascular volume. Thus, it becomes more accurate to use volumetric indices such as global diastolic volume and right ventricular end diastolic volume index. Intravenous fluid resuscitation of the volume load and the preload responsiveness is assessed by dynamic parameter's such as stroke volume and pulse pressure. If dynamic or volume parameters are not available, hemodynamic monitoring is done using traditional filling pressures.

Transmural pressure is calculated by deleting ITP which is IAP/2.

Transmural CVP = CVP – IAP/2[23]

Transmural PAOP = PAOP – IAP/2[23]

IAH causes a rise in the inferior vena caval pressure due to compression and reduced emptying, leading to a parallel rise in femoral venous pressure. Correction of IAP restores the normal blood flow in femoral vessels. But several cases of pulmonary embolism have been reported following this normalization. This resembles the findings in ischemia-reperfusion models.

PULMONARY SYSTEM

The increase in the IAP causes an upward displacement of the diaphragm and increases the intra-abdominal volume. This diaphragmatic displacement causes an extrinsic compression of the pulmonary parenchyma leading to atelectasis. This leads to

reduced diffusion of oxygen and ventilation perfusion imbalance. Hypercarbia and arterial hypoxemia may occur owing to reduced capillary blood flow, increased alveolar dead space and decreased carbon dioxide excretion. Both mean airway pressure and inspiratory pressures are significantly increased, while pulmonary compliance and tidal volume are reduced. Changes in the ventilatory settings required to treat this secondary acute respiratory distress syndrome include

(1) Maintenance of transmural plateau pressure under 35cm of water

(2) PEEP adjusted to counteract IAP

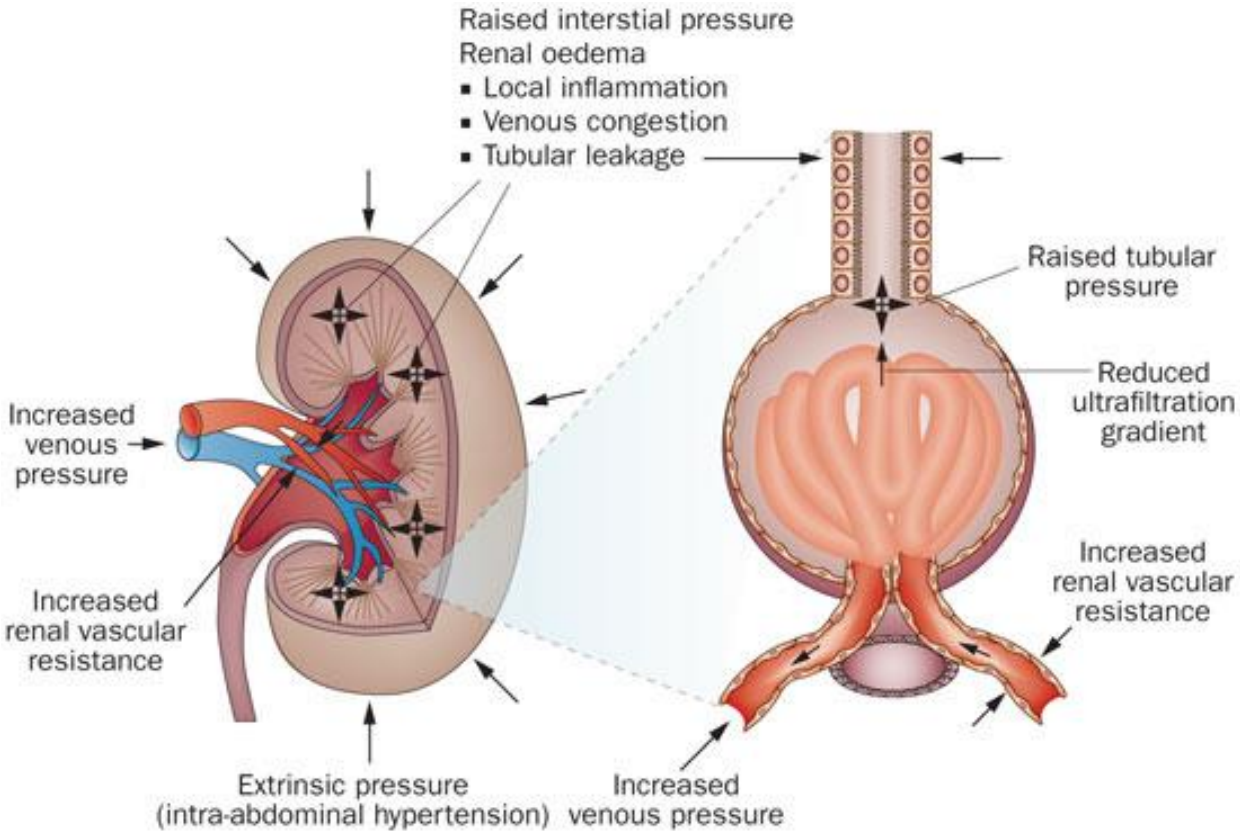
(3) Extravascular lung water index to be measured due to the risk of lung edema.

URINARY SYSTEM

In patients who originally had normovolemia and normal renal functions, IAH induced renal dysfunction becomes evident as oliguria at an IAP of 15mmHg and as anuria at an IAP of 30mmHg. Compression of renal vein and parenchyma and reduced renal perfusion, lead to microcirculatory failure of the functioning glomeruli and cortex. This results in glomerular and tubular dysfunction and significant reduction in urine output. Plasma antidiuretic hormone, renin and aldosterone levels are significantly elevated. The difference between Glomerular Filtration Pressure (GFP) and Proximal Tubular Pressure (PTP) is the mechanical force across the glomerulus, the Filtration Gradient (FG). Renal perfusion pressure is equal to GFP and is calculated by deducting IAP from MAP. PTP is equal to IAP. Hence,

FG = GFP – PTP = (MAP-IAP) – IAP

Therefore FG = MAP – 2 x IAP



Thus, the IAH induced renal dysfunction and prerenal azotemia will neither be responsive to fluid resuscitation nor vasopressors such as dopamine nor loop diuretics. It improves dramatically by appropriately and promptly reducing the elevated IAP.

Urinary bladder is also affected by increased IAP. Experimentally elevated IAP was found to induce structural (damage to epithelium, lamina propria and serosa), biochemical (malondialdehyde levels are increased) and contractility (bladder contraction potentiated by acetylcholine) changes in the urinary bladder.

GASTROINTESTINAL SYSTEM

The gastrointestinal system seems to be affected by even minimal change in the IAP.

a. The mucosal barrier function (affecting both bacterial translocation and influencing intermucosal nutritional flow)

b. Gastrointestinal motility are the two main functions altered.

It has been observed that the gut mucosa is very sensitive to increase in IAP. It causes

1. Compression of the mesenteric veins. This subsequently causes interstitial edema and ischemia

2. Reduction of the mesenteric blood flow, even at IAP of only 10 mmHg

3. Except adrenals (due to catecholamine release), diminished blood flow to all abdominal organs

4. Bacterial translocation, sepsis leading to multi-organ failure

5. Decreased intramucosal pH and perfusion, increased mucosal permeability and loss of intestinal mucosal barrier function.

After repeated episodes of such insults, IAH induced ischemia-reperfusion insults, the second hit in the multiorgan failure two-hit model takes place. These effects are called as acute intestinal distress syndrome and acute bowel injury. The parameter to be monitored and maintained is to keep APP above 60 mmHg.

Regarding the gastrointestinal motility, a decrease in the electrical and the mechanical motor activity of the small bowel has been attributed to the increased IAP. The contractile response is also inhibited by the elevated IAP.

HEPATOBILIARY SYSTEM

Even a small elevation of the IAP of around 10mmHg is associated with a reduction in the hepatic artery, vein and the portal circulation. This results in compensatory gastroesophageal collateral blood flow to the azygos vein. The liver is found to be highly susceptible to injury during IAH. Elevated IAP leads to enhanced hepatocyte proliferation, increased hepatocyte apoptosis, suggesting a liver repair response. Additionally, altered glucose metabolism and mitochondrial function and deceased lactate clearance are the physiologic effects of IAH. Certain conditions such as decompensated chronic liver disease, liver failure and liver transplantation are further complicated by the increase in the IAP.

NERVOUS SYSTEM

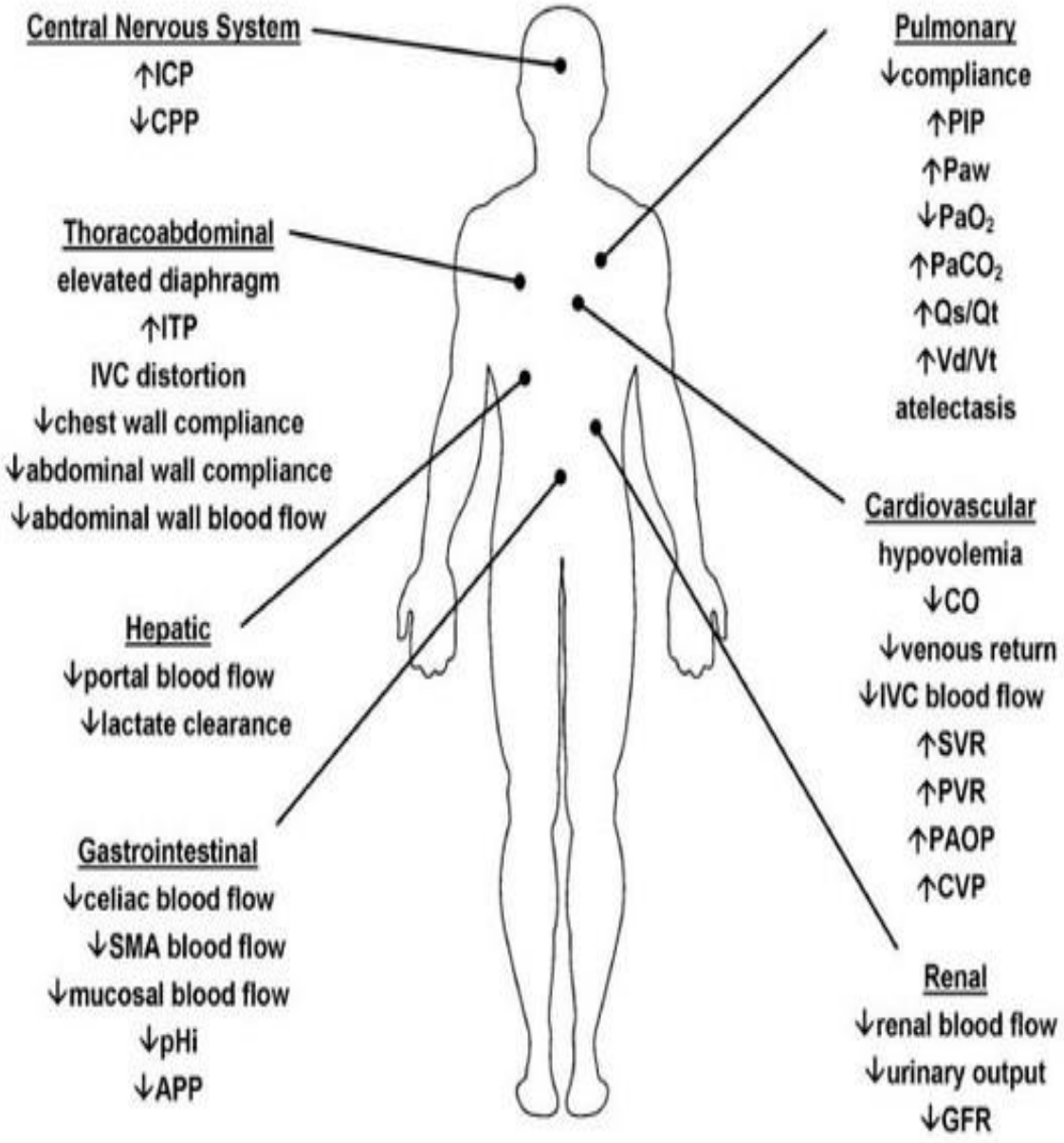
There have been several studies showing the concomitant increase in intracranial pressure (ICP) following increase in IAP as a part of poly-compartment syndrome.

Mechanism suggested:

1. Increased IAP causes increase in ITP which in turn increases the jugular venous pressure. This causes functional obstruction, impeding the cerebral venous outflow, increasing the cerebral blood volume causing elevation of ICP.

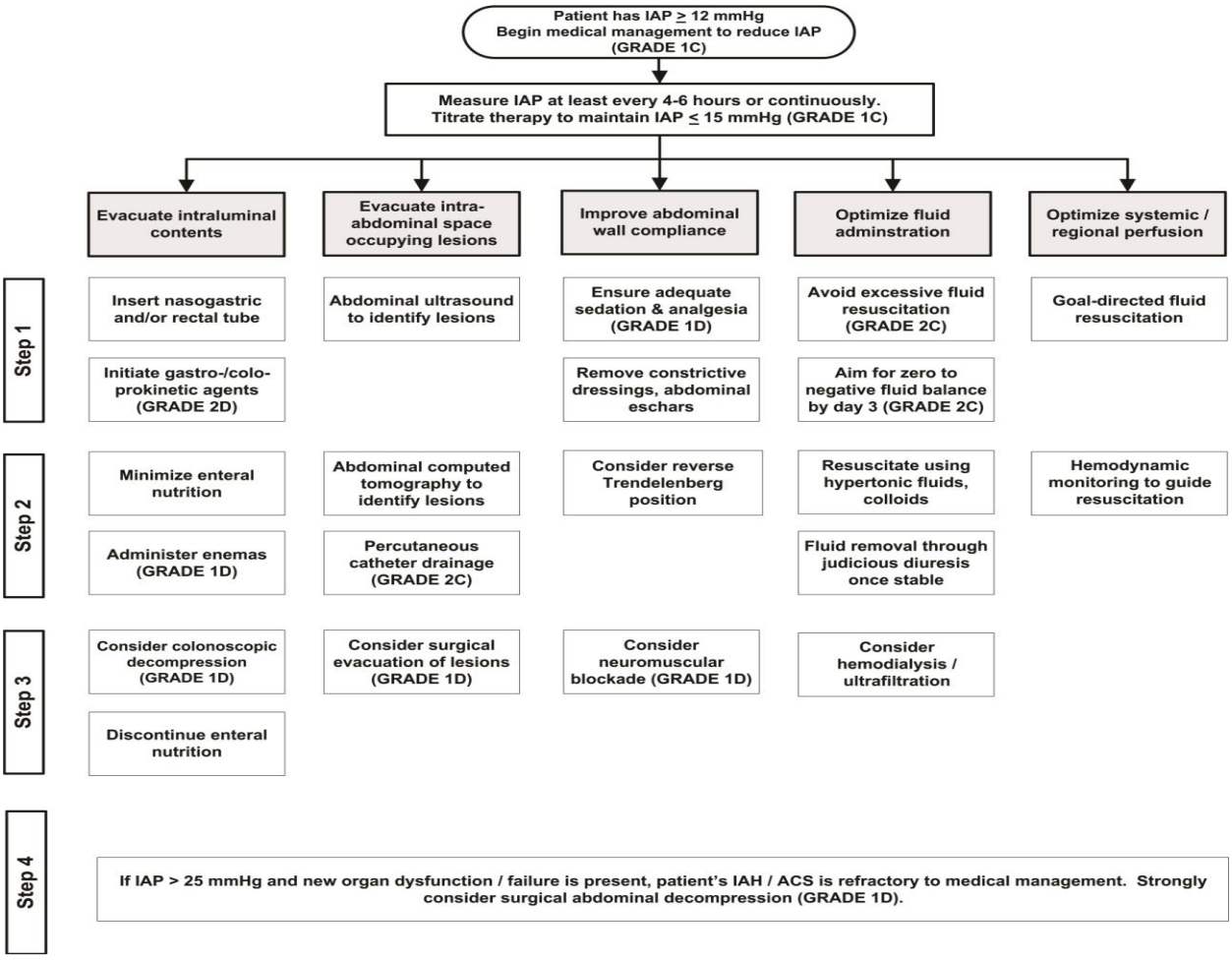
2. Functional obstruction causing decreased lumbar venous plexus blood flow due to increase inferior vena caval pressure. This causes decreased cerebrospinal fluid (CSF) absorption which takes place in the lumbar cisterns. This increase in the CSF pressure is thereby transmitted causing increase in the ICP.

SUMMARY OF EVENTS IN VARIOUS SYSTEMS DUE TO INCREASE IN IAP



IAH / ACS MEDICAL MANAGEMENT ALGORITHM

- *The choice (and success) of the medical management strategies listed below is strongly related to both the etiology of the patient's IAH / ACS and the patient's clinical situation. The appropriateness of each intervention should always be considered prior to implementing these interventions in any individual patient.*
- *The interventions should be applied in a stepwise fashion until the patient's intra-abdominal pressure (IAP) decreases.*
- *If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm.*



THE MANAGEMENT PROTOCOL FOR IAH/ACS RECOMMENDED BY WORLD SOCIETY OF THE ABDOMINAL COMPARTMENT SYNDROME

1. Patients on ICU admission should be screened for independent risk of IAH/ACS

2. In the presence of two or more risk factors, baseline IAP is measured. If IAH is present, serial IAP monitoring is done which helps in guiding patient's resuscitation

3. The main aim is that APP should be maintained above 60mmHg

4. A trial of sedation and neuromuscular blockade in cases of mild IAH, helps in muscle relaxation and decreasing IAP. Neuromuscular blockade useful in cases of third-space fluid and tight abdominal closure.

5. Positioning of the patient – supine. Head end of bed elevation causes increase in IAP

6. Nasogastric tube/enemas/rectal tubes/prokinetic agents/endoscopic decompression are useful as both air and fluid within the hollow viscera increase IAP

7. Fluid resuscitation should be optimal

8. Percutaneous catheter decompression in cases of intraperitoneal abscess, blood or fluid in symptomatic ACS under ultrasound guidance.

9. Open abdominal decompression in selected patients not responding to medical management and those unfit for percutaneous drainage

RESULTS

AGE DISTRIBUTION

The mean age distribution of the patients is 38.34 years with a standard deviation of 8.4 years ranging from 22 to 56 years and a median of 38 years.

AGE	**YEARS**
Mean	38.34
Median	38.00
Mode	30
Std. Deviation	8.426
Minimum	22
Maximum	56

Table 1: Age Distribution

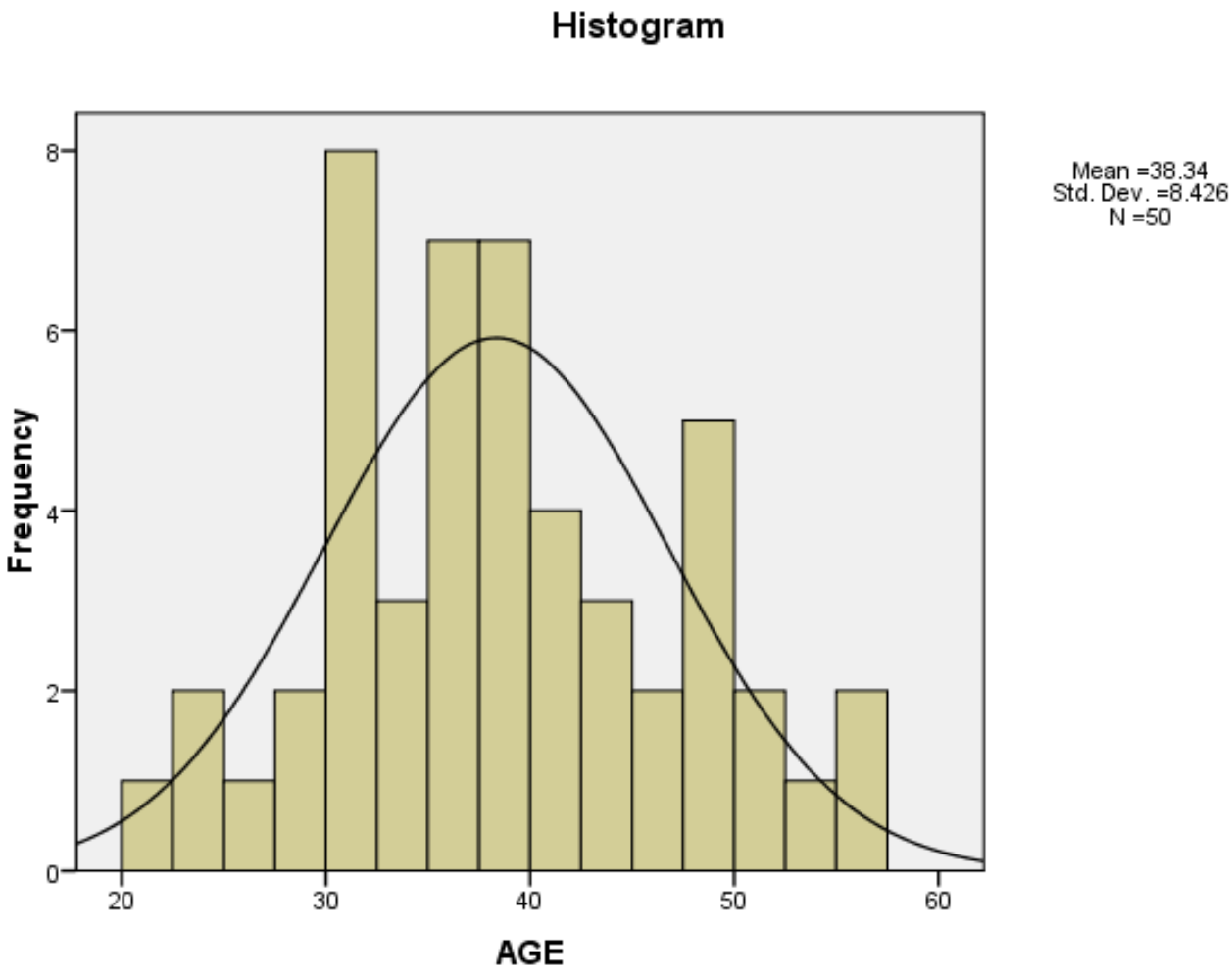


Figure 1: Age Distribution

Gender Distribution

Out of fifty patients. 84% (n=42) of them were males while the rest (n=8,16%) were females.

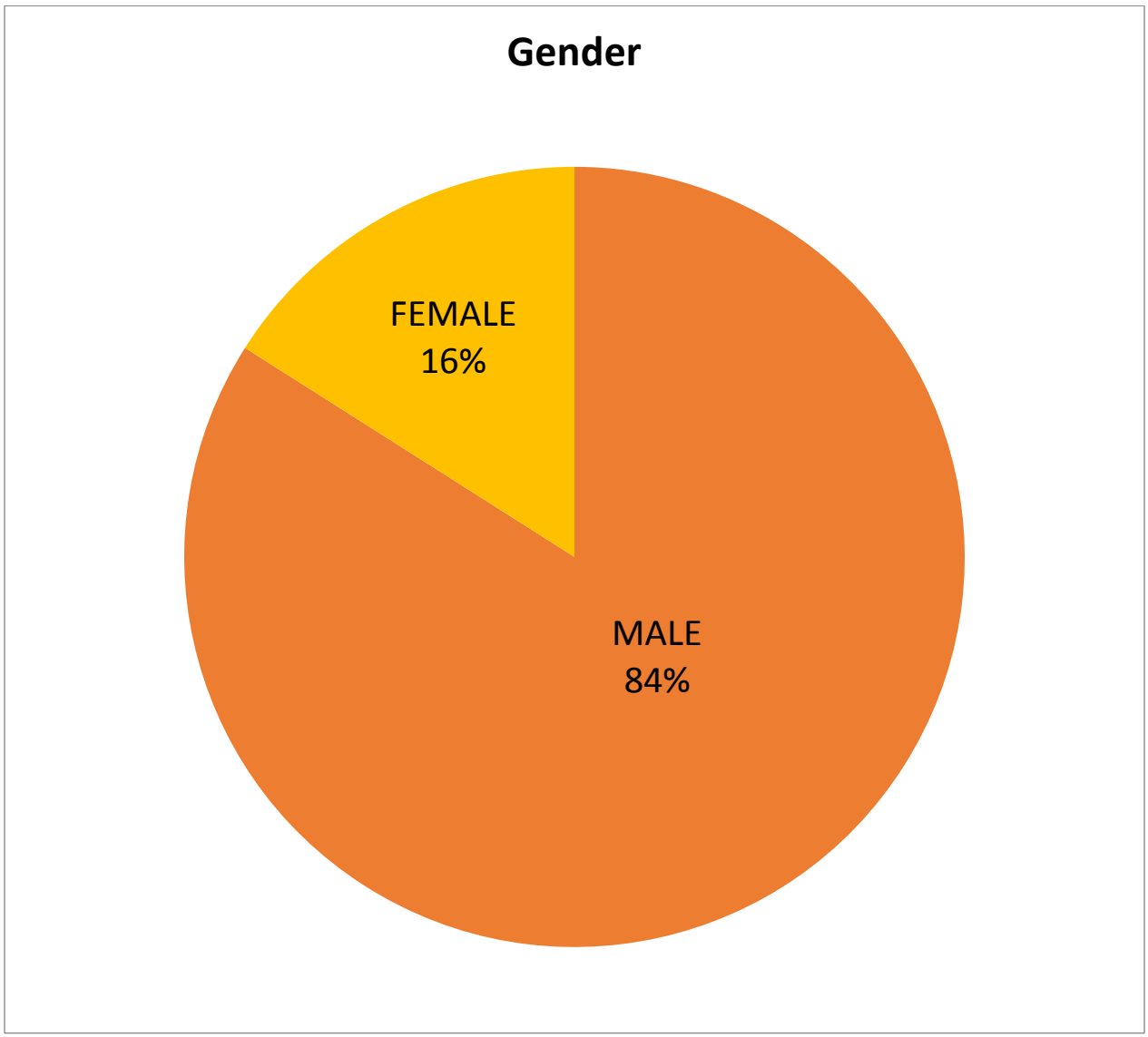


Figure 2: **Gender Distribution**

AETIOLOGY

Out of 50 patients, 74% of them (n=37) were alcoholics while 12% of them had gallstone and idiopathic aetiology each and only one of them had drug as an aetiology.

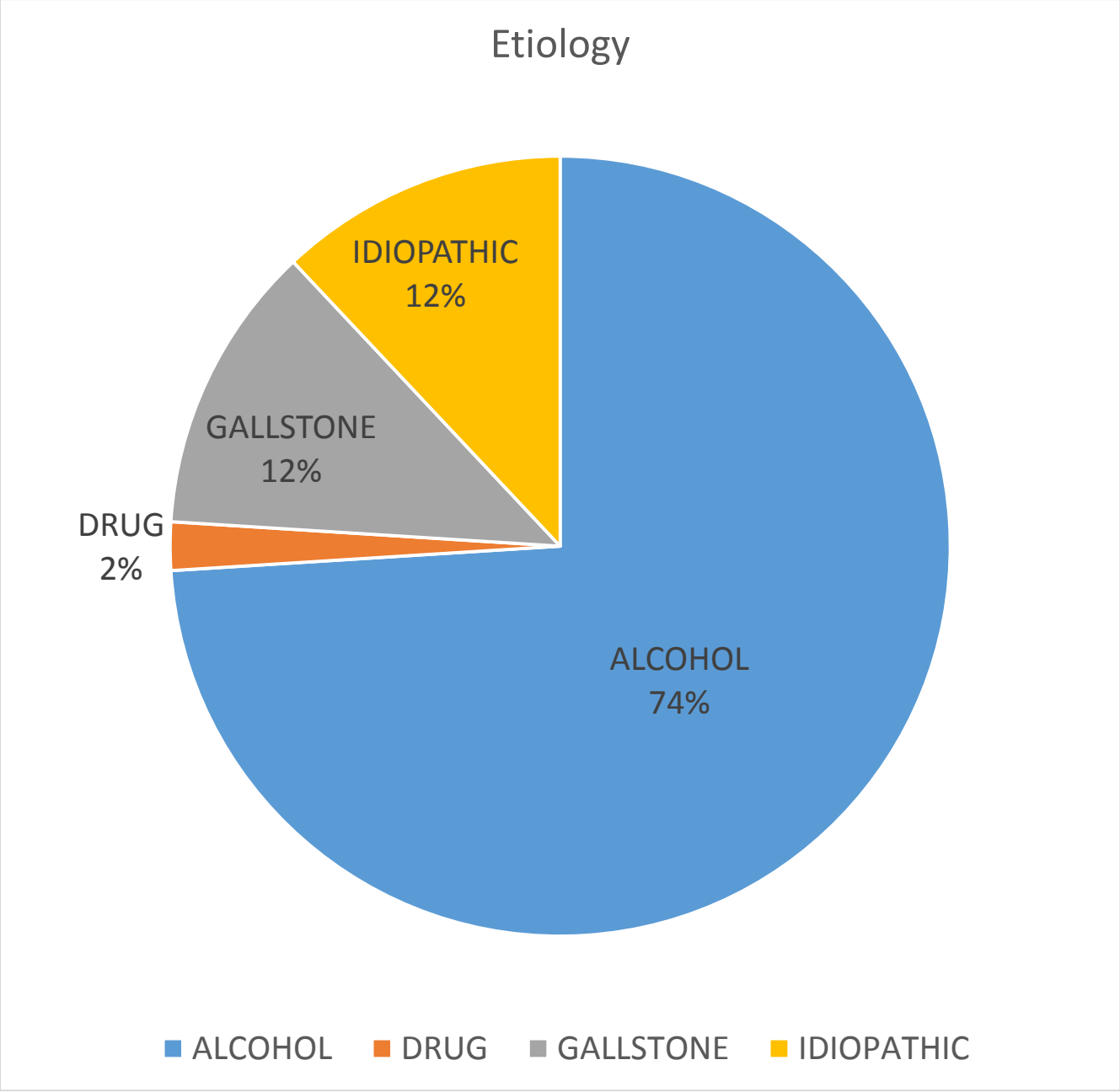


Figure 3: Aetiology

BALTHAZAR CT Score

The Balthazar CT score ranged between 2 and 8. The following table and figure shows the distribution of the scores among the patients.

BALTHAZAR	Frequency	Percent
2	1	2.0
3	3	6.0
4	5	10.0
5	6	12.0
6	12	24.0
7	15	30.0
8	8	16.0
Total	50	100.0

Table 2: Balthazar CT score

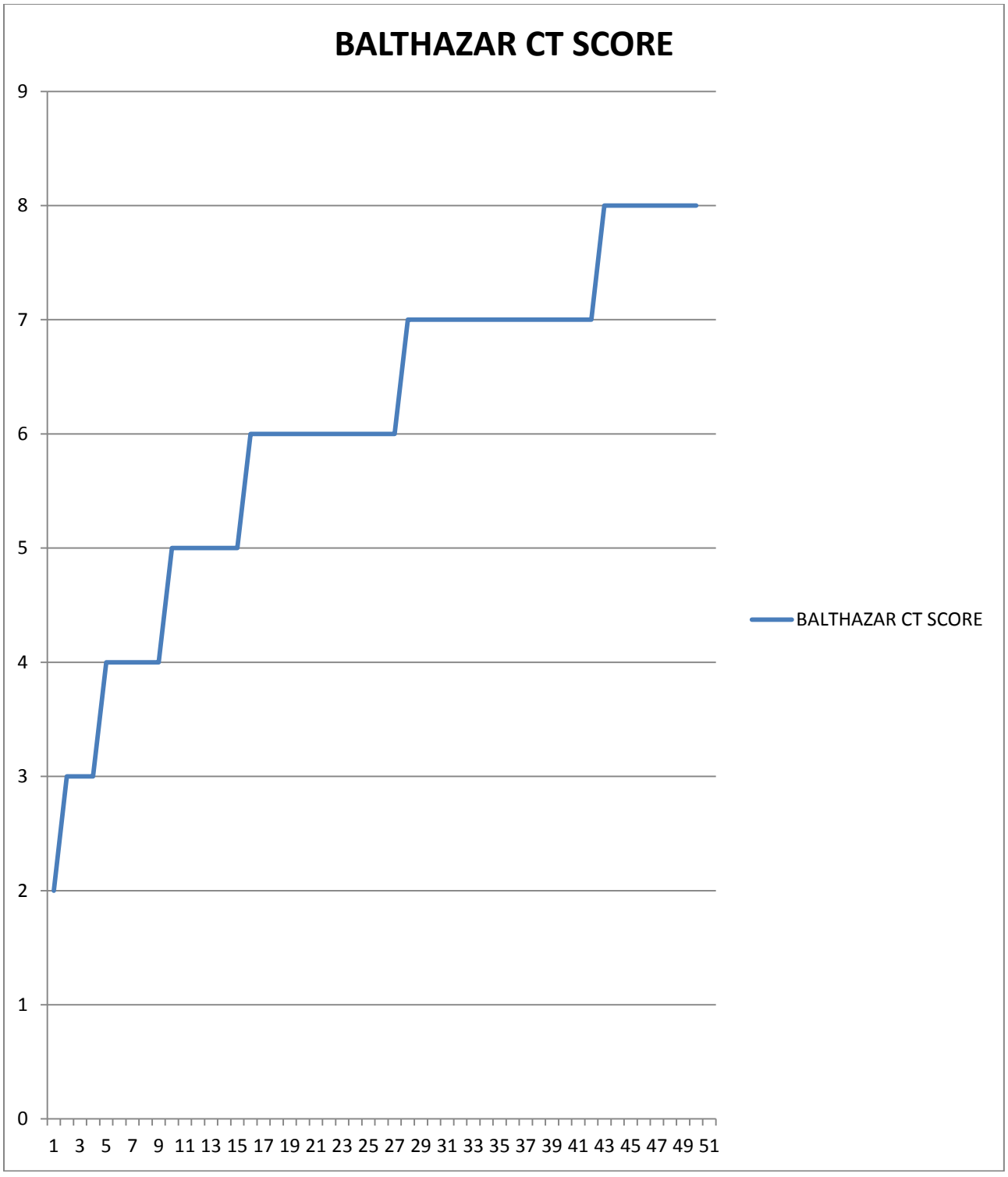


Figure 4: Balthazar Score

APACHE SCORE

The APACHE score ranged between 4 and 33. The following table and figure shows the distribution of APACHE scores in the study population.

APACHE	Frequency	Percent
4	8	16.0
5	4	8.0
6	5	10.0
7	1	2.0
8	17	34.0
9	2	4.0
10	4	8.0
14	2	4.0
15	3	6.0
16	1	2.0
17	1	2.0
24	1	2.0
33	1	2.0
Total	50	100.0

Table 3: APACHE

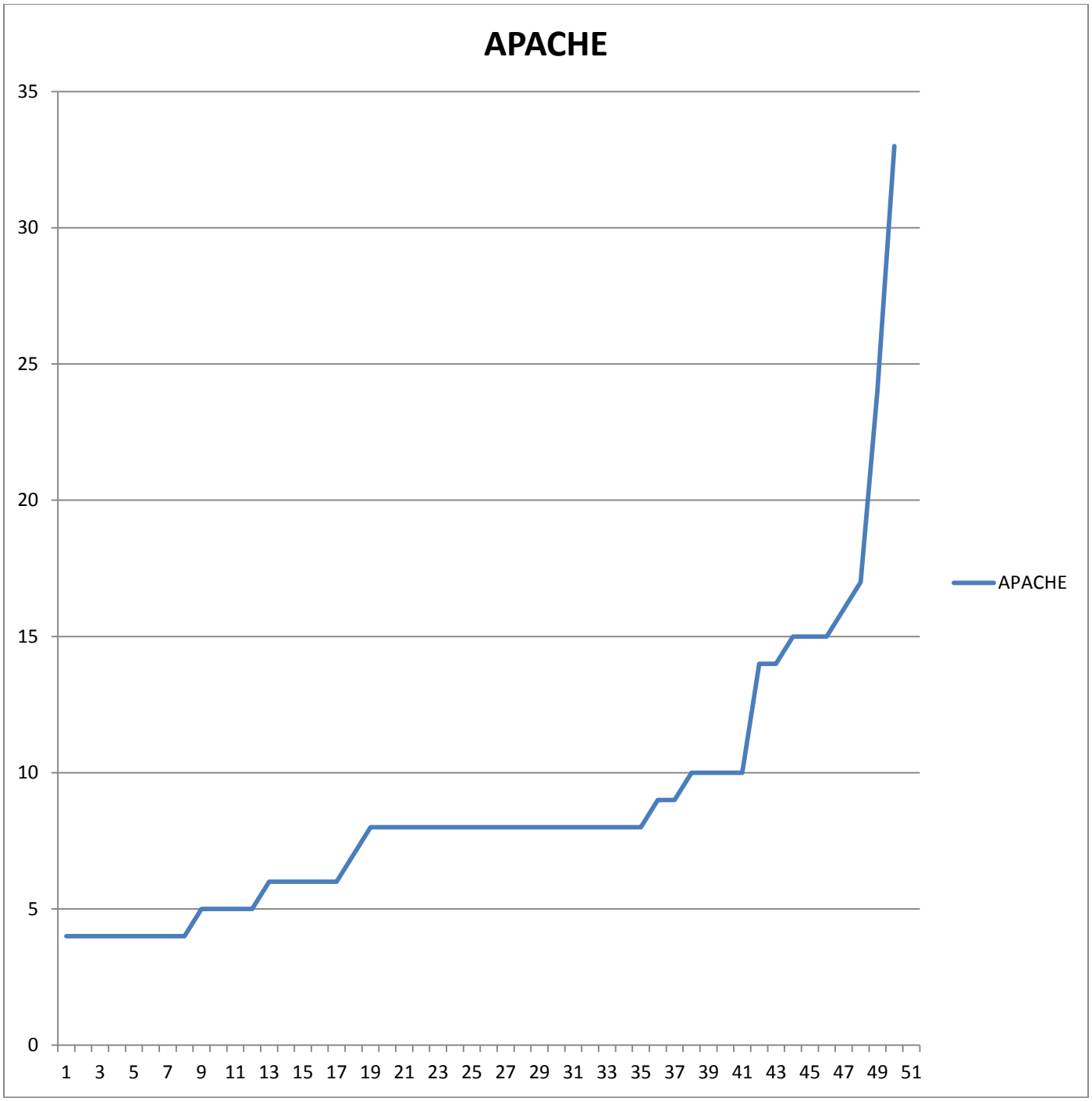


Figure 5: APACHE

VASOACTIVE DRUG

Out of 50 patients, 11 (22%) of them had been given vasoactive drugs.

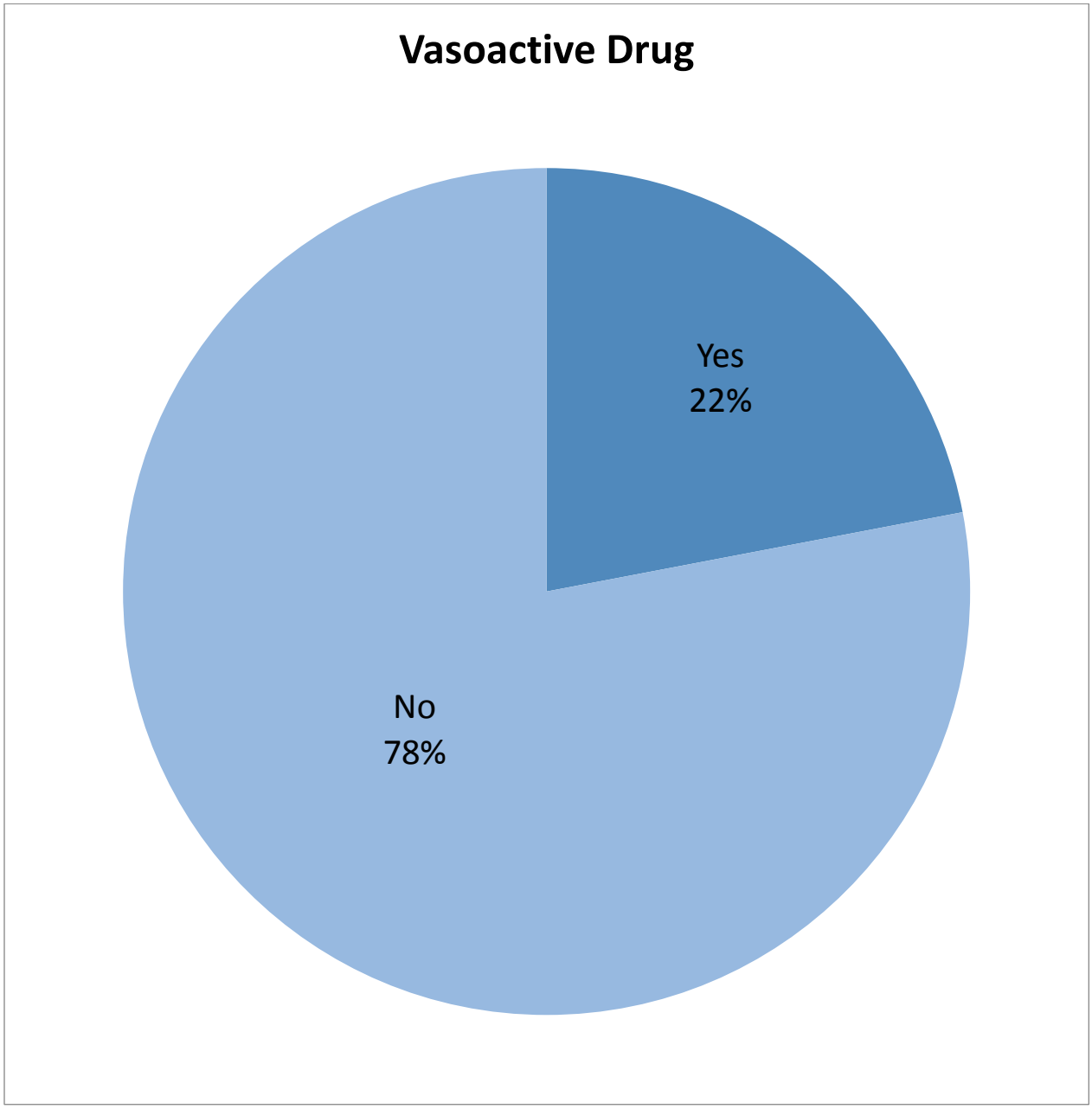


Figure 6: Vasoactive Drug

MODS

Out of 50 patients, 14 (28%) of them had MODS.

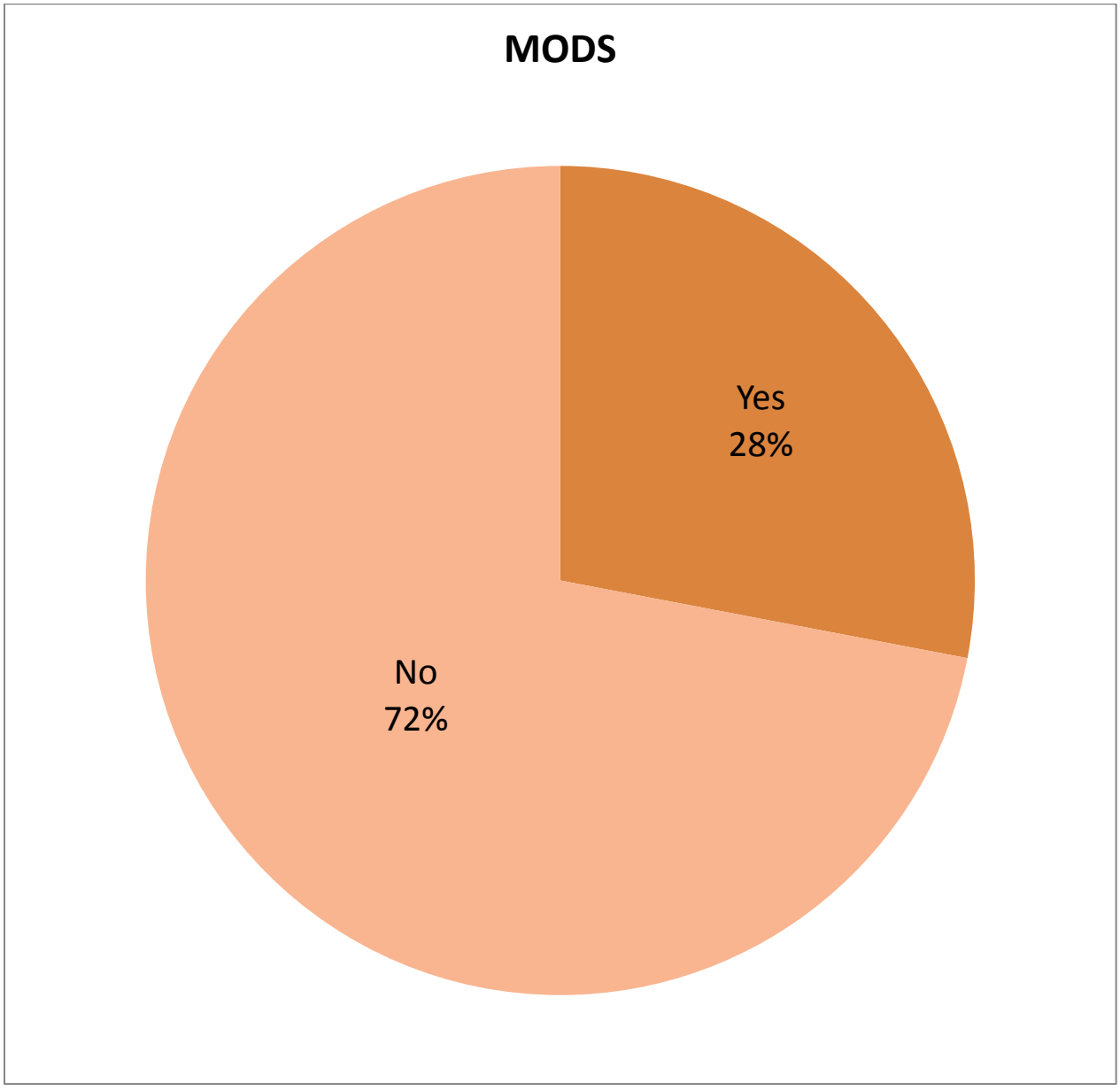


Figure 7: MODS

The Duration of symptoms, Duration of Hospital Stay and Duration of ICU stay

The mean duration of symptoms is 2.42 days (range:1-5 days), the mean duration of ICU admission is 1.92 days (range: 0-6 days) and mean duration of hospital stay is 6.66 days (range:1-13 days).

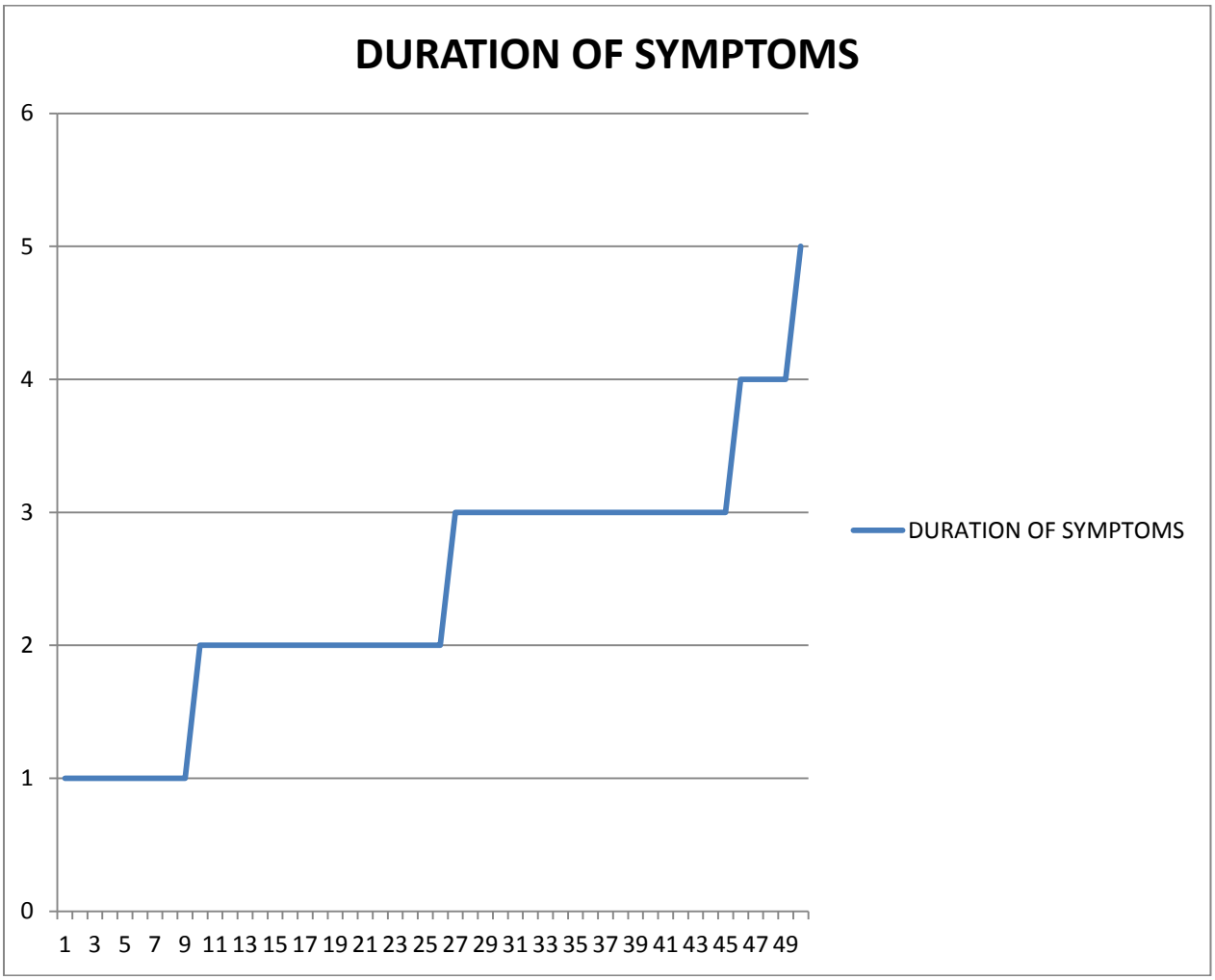


Figure 8: Duration of symptoms

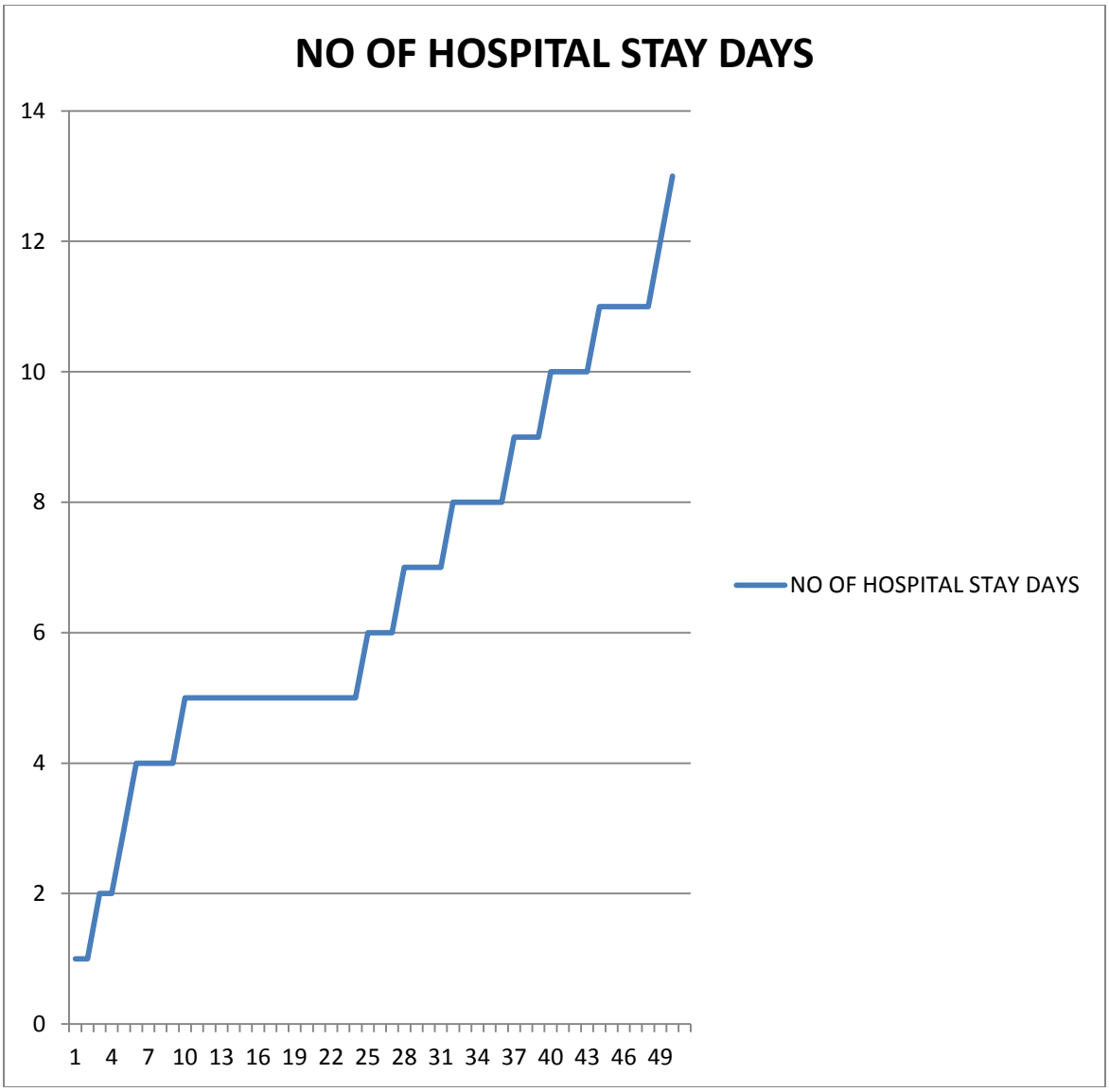


Figure 9: Number of hospital stay days

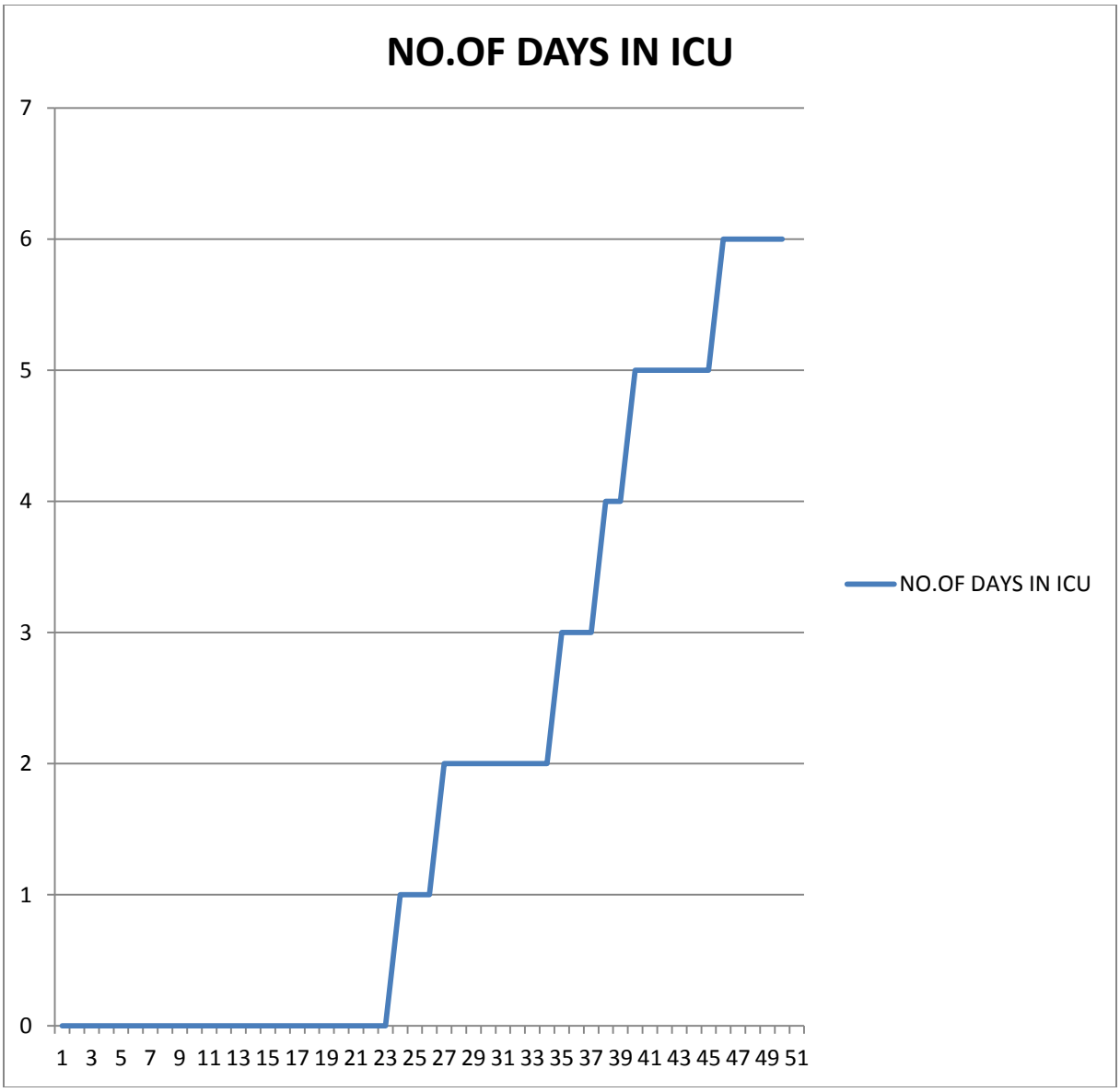


Figure 10: Number of days in ICU

OUTCOME OF THE ILLNESS

Out of 50 patients, 12% (n=6) expired while the rest 88% (n=44) were discharged.

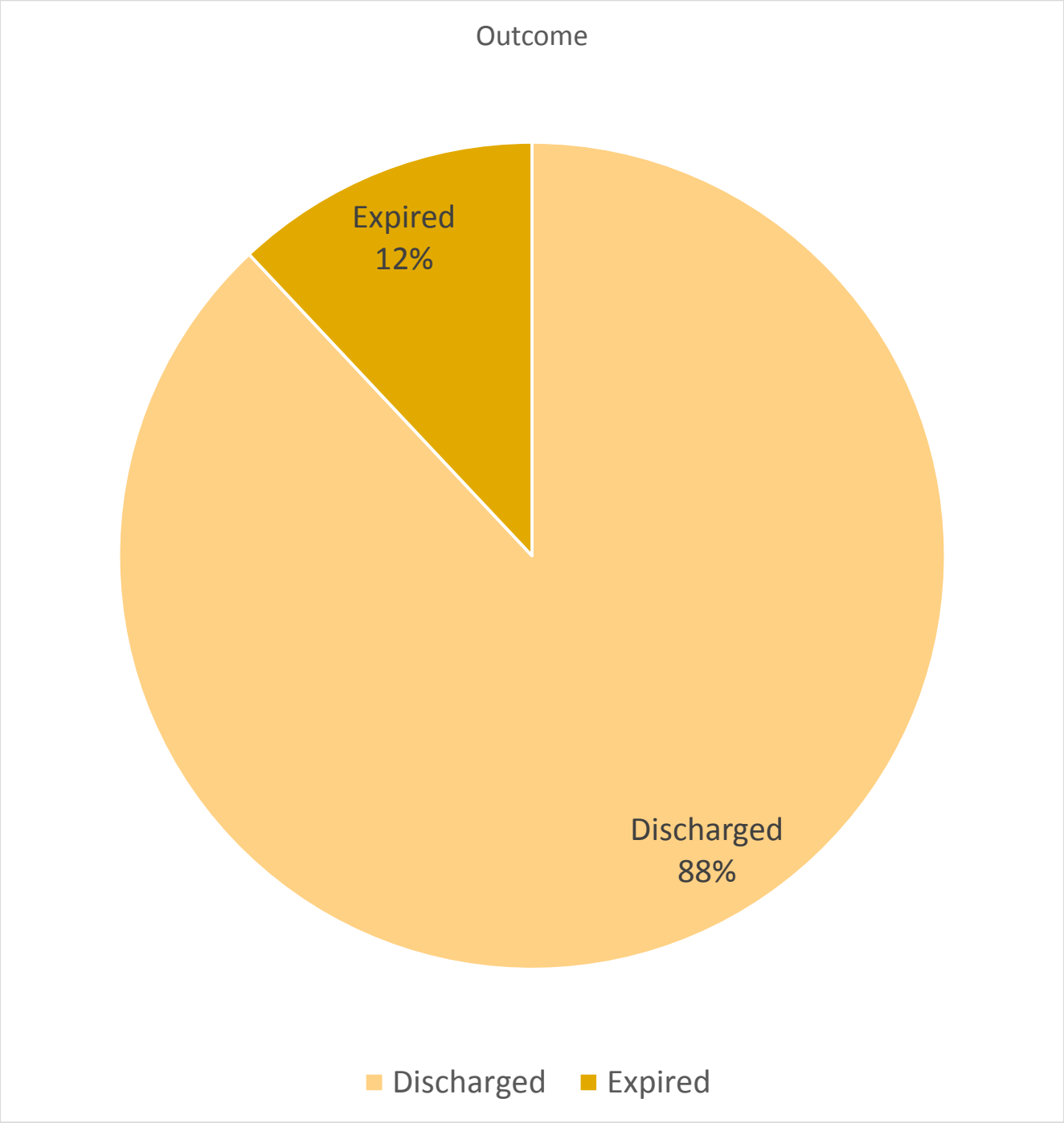


Figure 11: Outcome of the illness

INTRABDOMINAL PRESSURE

The intraabdominal pressure was measured serially every 8th hourly and the findings were noted and correlated with other parameters. All of them had intraabdominal hypertension with a mean of 9.55mmhg (ranging between 4.13 and 20 mmhg). The following figure shows the distribution of serial IAP between patients.

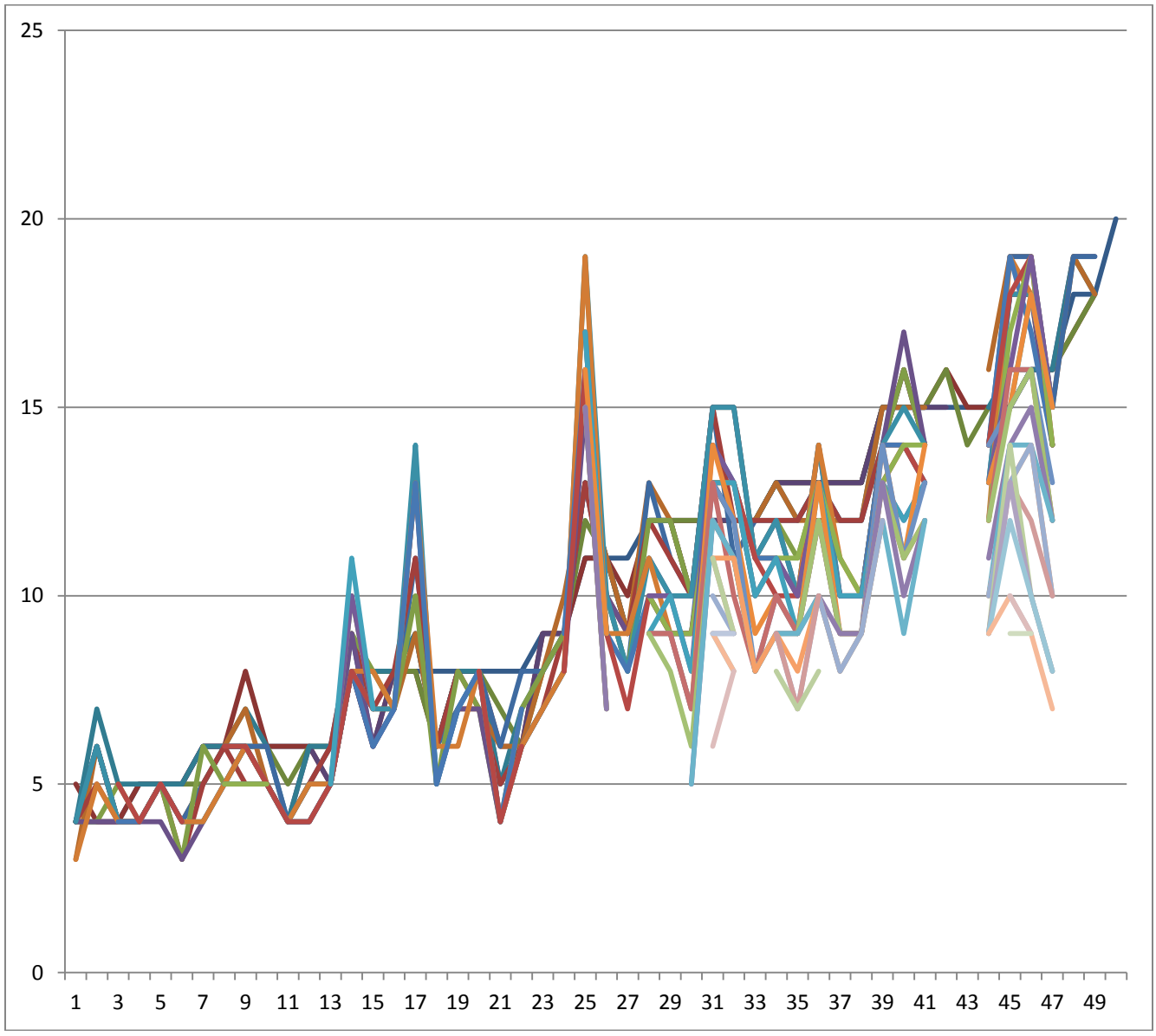


Figure 12: The mean intraabdominal pressure distribution

Comparison of IAP with the outcome of the illness

The mean IAP varies significantly between discharged and expired patients. The mean IAP in discharged group is 8.65 mm Hg (range: 4.13-15.18 mm Hg) and the mean IAP in expired group is 16.14 mm Hg (range: 9.9-20 mm Hg). Mann-Whitney U test was performed and it yielded significant results with $p<0.05$ ($p=0.0328$).

Category	**Discharged**	**Expired**	**Mann-Whitney U test p-value**
Total # of patients	44 (88%)	6 (12%)	23.16 P=0.0328 P<0.05 Significant
Mean IAP (mm Hg)	8.65	16.14	
Range	4.13-15.18	9.9-20	

Table 4: Comparison of IAP with the outcome of the illness

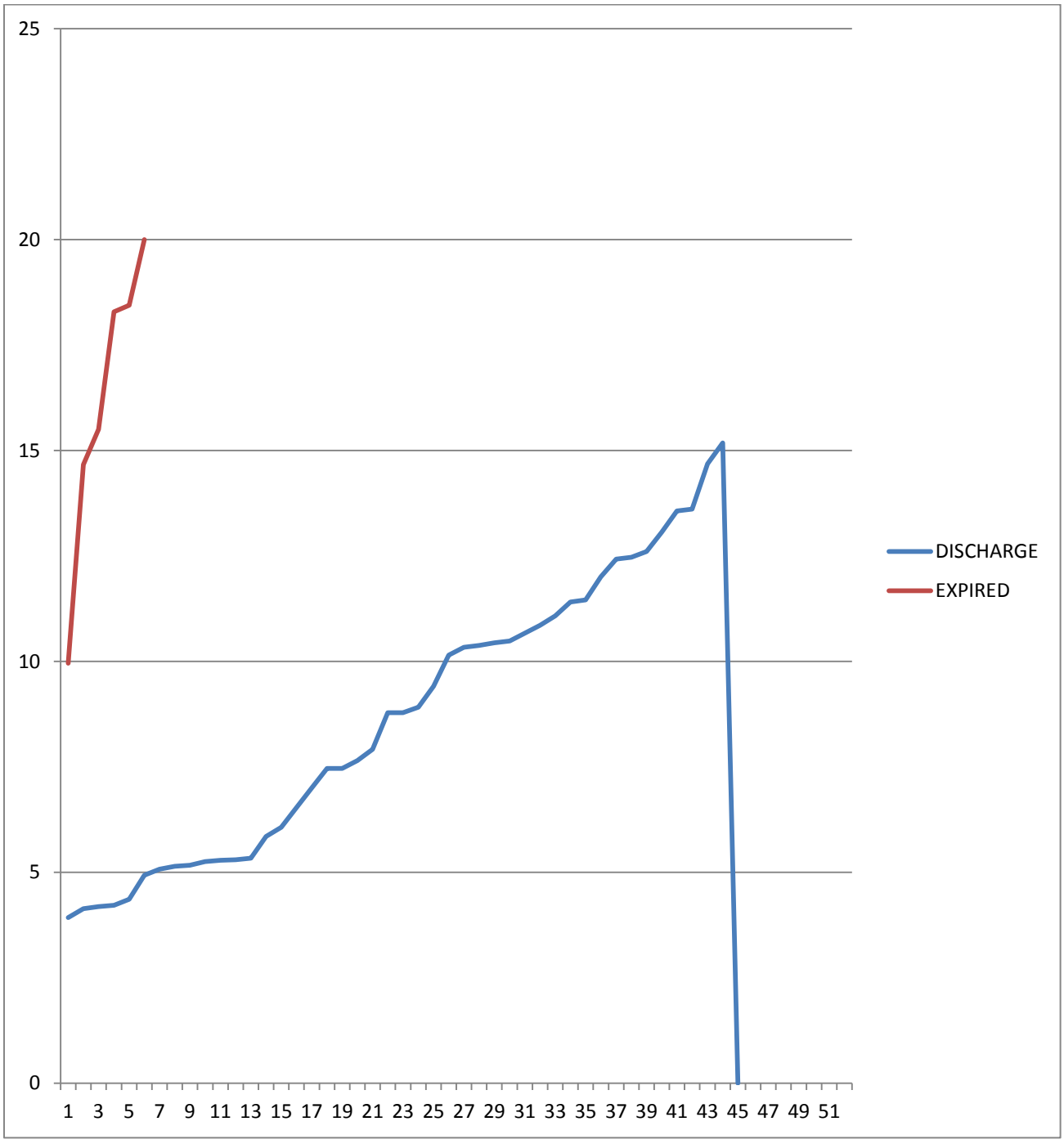


Figure 13: Comparison of IAP with the outcome of the illness

Correlation of severity of the disease with IAP

Correlation tests between IAP with APACHE score, hospital stay and ICU admission shows that higher the intraabdominal pressure (correlation r =0.91, p=0.00071), greater is the severity of the illness with increased admission to ICU (correlation r =0.95, p=0.00059) and longer hospital stay (correlation r =0.97, p=0.000091). All the tests were highly significant since $p<0.005$.

Variables	R-value	p-value	Significance
IAP and Clinical Severity	0.91	0.00071	Highly Significant
IAP and Hospital Stay	0.95	0.00059	Highly significant
IAP and ICU admission	0.97	0.000091	Highly Significant

Table 5: Correlation of severity of the disease with IAP

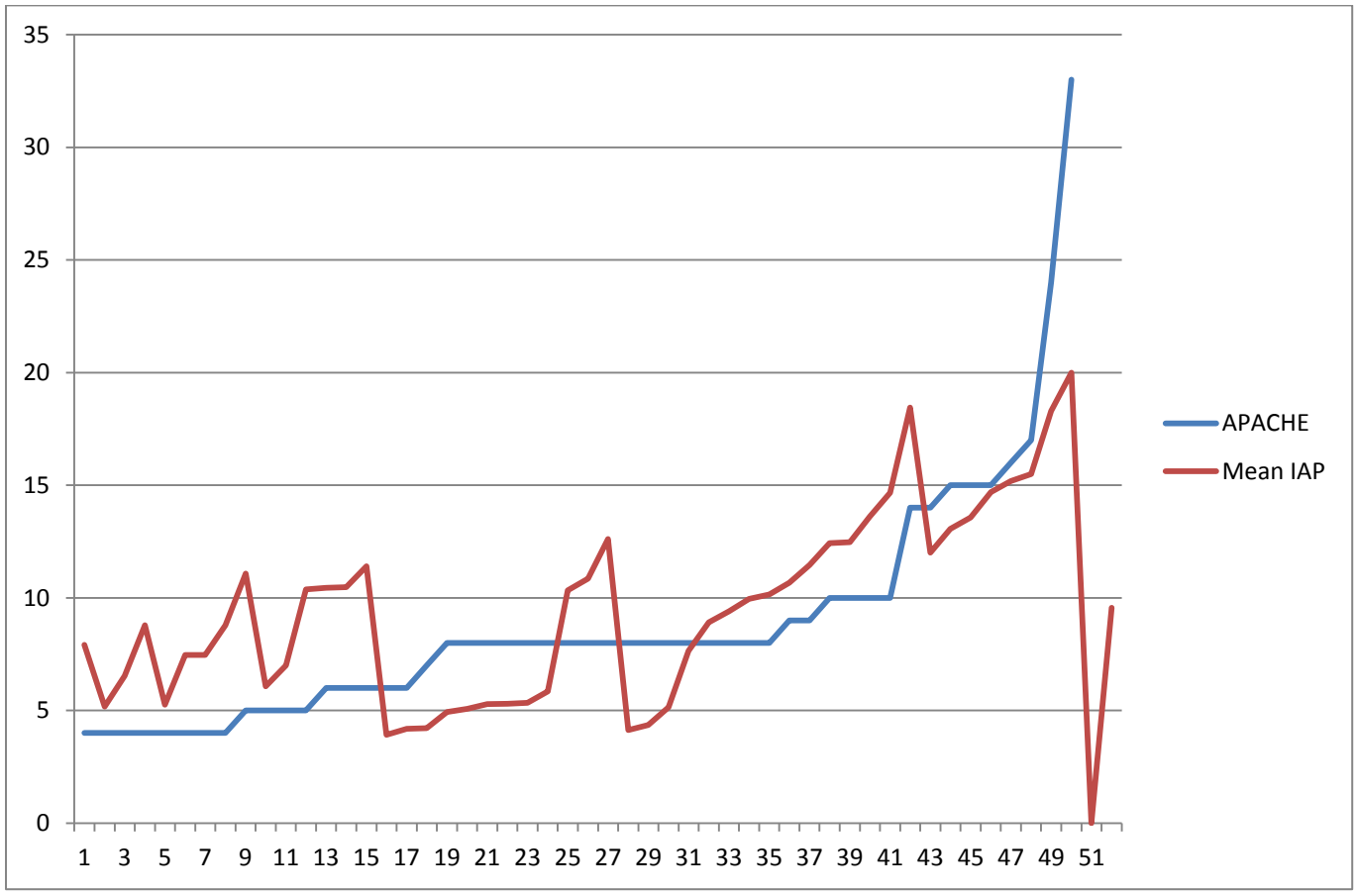


Figure 14: Correlation between clinical severity and IAP

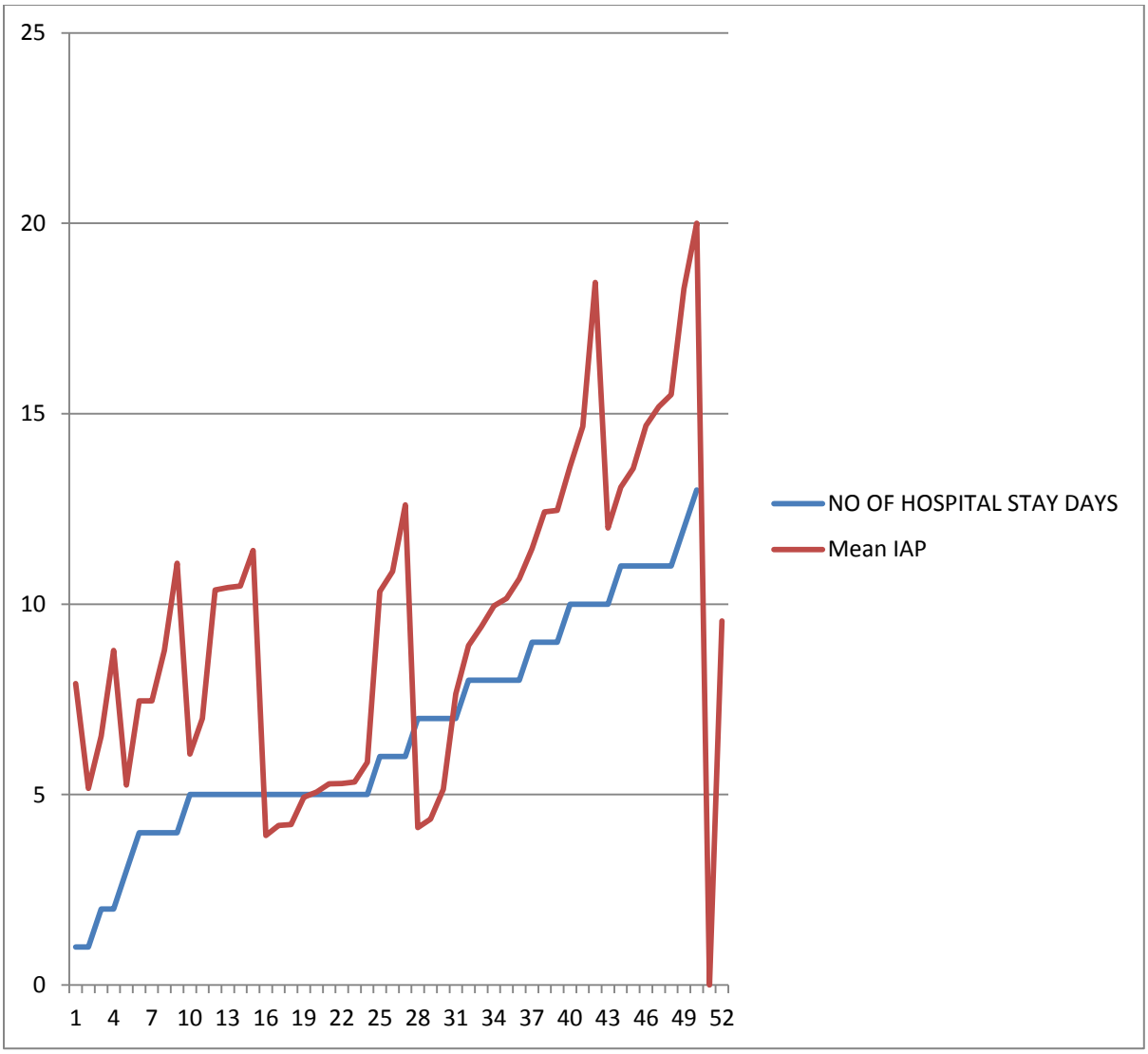


Figure 15: Correlation between hospital stay and IAP

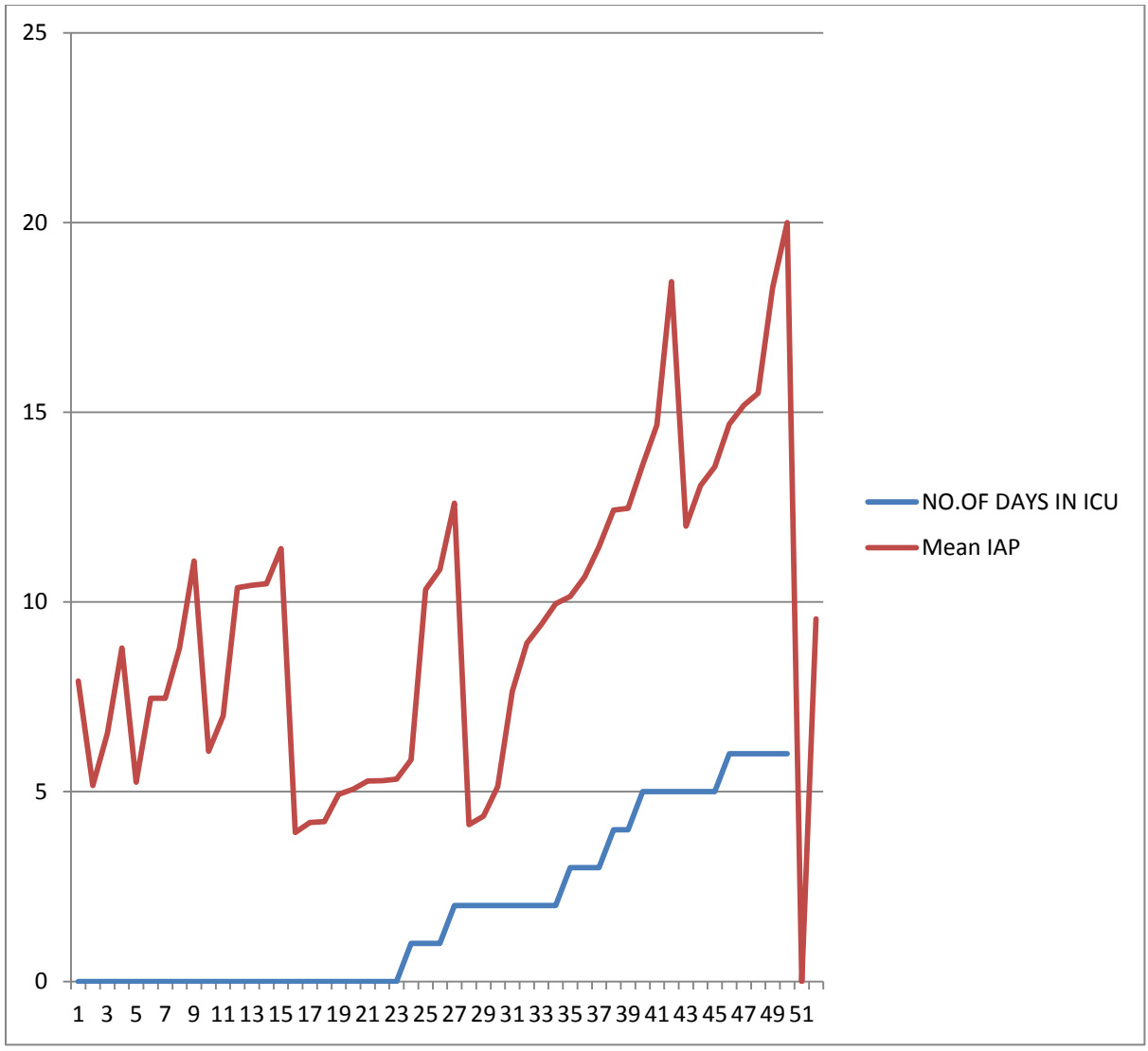


Figure 16: Correlation between ICU admission and IAP

DISCUSSION

A total of 50 patients admitted with acute pancreatitis were recruited in this study. Mean patient age was 38.34 years of age (range, 22 to 56 years). The median admission IAP was 9.5 mm Hg; 33 patients (66%) had severe pancreatitis as defined by the Atlanta criteria. 13 patients developed complications of their pancreatitis.

Patients with intra-abdominal hypertension on admission had a 19% mortality ratio compared to 3% mortality in patients with no intraabdominal hypertension on admission. Twenty-six patients had intra-abdominal hypertension on admission and one patient developed intraabdominal hypertension during their stay in hospital stay. In this study, male preponderance (84%) compared to female (16%) was observed. aetiology of acute pancreatitis was attribute to primarily to alcohol (74%). Gallstone disease (12%), idiopathic (12%) and drug (2%) were other factors for causation of acute pancreatitis.

Acute pancreatitis defined by Balthazar score revealed 23 patients as severe pancreatitis. Out of the 23 patients diagnosed with increased Balthazar score, 18 patients (78%) had intraabdominal hypertension on admission and 12 patients (52%) developed complication of disease. APACHE score more than 8 was used in classifying the patients to have severe pancreatitis and the mean score was 12.9. Out of 50 patients, 74% were termed severe pancreatitis as measured by APACHE score.

The mean duration of symptoms was 2.4 days (1 to 5 days). Mean duration of ICU stay was 1.92 days and mean duration of hospital stay was 6.6 days. Mean IAP measurement of the survivors was 8.65 mmHg (range from 4.13 to 15.18mm Hg). Non – survivors had a higher IAP mean value of 16.14 mm Hg (range 9.9 to 20mm Hg). Mann

Whitney U test showed that IAP had a significant value of 23.16 ($p<0.05$). Correlation studies between IAP and ICU stay was highly significant ($p<0.05$).

Correlation studies between IAP with clinical severity of the patients showed that higher the IAP, greater is the severity of illness (r value – 0.91). The p value was 0.00071(highly significant). The mean daily IAP was found to be in an increasing trend during the initial week of illness in the non-survivors. IAP was found to increase in line with development of multi-organ failure. Patients with intraabdominal hypertension were treated with more intravenous fluids to maintain a positive fluid balance.

CONCLUSION

IAP is a good predictor of complications and mortality across the entire course of illness in acute pancreatitis and it compares favorably well as predictive marker with other validated prognostic scoring systems. This study showed that mortality rates were higher within the first week of illness which is supported by a Scottish study in which the authors conclude that 40-60 % of all acute pancreatitis deaths occurred with the first week. Isenmann et al concluded that patients with acute severe pancreatitis with development of necrosis have a mortality of 40% on day 1. This study corroborates with similar findings that significantly greater day I IAP was seen in non-survivors compared to survivors. It should be noted that IAP readings were only recorded from patients who needed catheterization based on clinical grounds, stating that there is a small undefined subset of patients with very mild acute pancreatitis who were not included in this study. In this study patients were not subjected to decompressive laparotomy even in the presence of intraabdominal hypertension, which is the current practice with poor operative outcome.

Intraabdominal hypertension and abdominal compartment syndrome often develop in acute pancreatitis. An increase in IAP leads to reduced splanchnic flow. Diebel et al , demonstrated that mesenteric artery flow reduction of 37% with an IAP of 20 mm Hg and even higher reduction of flow in intestinal mucosa 39% with only 10 mm Hg. This decrease of mesenteric blood flow causes loss of integrity of mucosal barrier by ischemia and reperfusion injury. This derangement causes bacterial translocation and subsequent entry into the bloodstream, thereby causing superinfection.

The results of this study demonstrate that IAP is a good predictor of mortality and organ failure in acute pancreatitis and correlates well with other validated prognostic scores Measurement of IAP is an easy, effective and inexpensive method. In conclusion IAP serves as a good prognostic marker in acute pancreatitis.

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ANNEXURES

ABBREVIATIONS & ACRONYMS

ACS: Abdominal compartment syndrome

IAH: Intra-abdominal hypertension

CT: Computerized tomography

ICU: Intensive care unit

USG: Ultrasound

WSACS: World Society Abdominal Compartment Syndrome

APP: Abdominal perfusion pressure

MAP: Mean arterial pressure

IAP: Intra-abdominal pressure

FG: Filtration gradient

GFP: Glomerular filtration pressure

PTP: Proximal tubular pressure

ITP: Intra-thoracic pressure

PAOP: Pulmonary artery occlusion pressure

ICP: Intra-cranial pressure

CSF: Cerebrospinal fluid

CPP: Cerebral perfusion pressure

ARDS: Acute respiratory distress syndrome

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

DISSERTATION TOPIC:

EVALUATION OF INTRAABDOMINAL PRESSURE IN ACUTE PANCREATITIS

PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI

NAME AND ADDRESS OF PATIENT:

I, _____ have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.

I understand that I will not get any payment for taking part in this study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Volunteer:

Signature/Thumb impression of the Volunteer

Date:

Witnesses:

(Signature, Name & Address)

Date:

Name and signature of investigator

PROFORMA

- NAME
- AGE/SEX
- IP NO
- DURATION OF SYMTOMS
- CO-MORBIDITIES
- PAST HISTORY
- ETIOLOGY
- APACHE SCORE
- BALTHAZAR SCORE
- ADMISSION IAP
- VASOACTIVE DRUGS – YES / NO
- MODS – YES / NO

DAYS	1	2	3	4	5	6	7	8	9	10	11	12	13	14
IAP AT 8HRS														
IAP AT 16 HRS														
IAP AT 24 HRS														

OUTCOME – DEATH / DISCHARGE

MASTER CHART

NO	NAME	AGE	SEX	DURATION OF SYMPTOMS	IP NO	ETIOLOGY
1	SENTHIL KUMAR	38	M	3	1826547	ALCOHOL
2	AGLIN ANGEL	32	F	3	1955465	ALCOHOL
3	AHMED	35	M	3	1834858	ALCOHOL
4	ARUL MURUGAN	35	M	2	1835647	ALCOHOL
5	ARUMUGAM	42	M	3	1836428	ALCOHOL
6	RAMESH	40	M	3	1840919	ALCOHOL
7	RANGANATHAN	33	M	2	1842596	ALCOHOL
8	RADHIKA	22	F	3	1844477	GALLSTONE
9	SENTHIL	34	M	3	1845267	IDIOPATHIC
10	SULAIMAN	54	M	1	1845685	GALLSTONE
11	RAMU	37	M	3	1846789	ALCOHOL
12	ABDUL RAHIM	49	M	2	1849738	ALCOHOL
13	KRISHNAN	39	M	1	1851178	IDIOPATHIC
14	ASHOK	24	M	2	1851308	ALCOHOL
15	MUJUPUR RAHMAN	48	M	2	1852756	ALCOHOL
16	BOOPATHY	41	M	2	1853265	ALCOHOL
17	LAKSHMI	55	F	3	1854742	ALCOHOL
18	KUMAR	30	M	2	1854752	IDIOPATHIC
19	SARAVANAN	31	M	2	1854864	IDIOPATHIC
20	SESHAREDDY	45	M	3	1856974	ALCOHOL
21	PARTHIBAN	30	M	1	1856987	ALCOHOL
22	RAJA	37	M	2	1856999	ALCOHOL
23	RAJMOHAN	30	M	1	1862167	ALCOHOL
24	RAMESH	39	M	3	1865475	ALCOHOL
25	SATHYANARAYANAN	48	M	2	1873195	ALCOHOL
26	PARTHIBAN	23	M	3	1873329	DRUG
27	SIVA	38	M	3	1874524	ALCOHOL
28	PRAKASH	27	M	3	1874596	ALCOHOL
29	VIJAYKANTH	32	M	3	1878323	ALCOHOL
30	MUKUNTH	28	M	2	1878391	ALCOHOL
31	RANIKALA	43	F	1	1878817	GALLSTONE
32	NAGARAJ	36	M	2	1880245	ALCOHOL
33	MURUGAN	49	M	3	1880702	ALCOHOL
34	ARULMOZHI	37	F	1	1884524	IDIOPATHIC
35	CHITHRA	36	F	4	1885698	GALLSTONE
36	KALAKADHI	56	M	2	1896541	ALCOHOL
37	VINOTH	29	M	2	1896574	ALCOHOL

38	PRASAD	43	M	3	1898978	ALCOHOL
39	MOHAN	42	M	4	1899654	GALLSTONE
40	NISAMUDEEN	45	M	3	1900785	ALCOHOL
41	GEETHA	38	F	4	1901845	IDIOPATHIC
42	GOPI	30	M	2	1902658	ALCOHOL
43	RAVIRAM	39	M	1	1903657	ALCOHOL
44	PAUL	43	M	2	1913989	ALCOHOL
45	VASU	32	M	1	1914586	ALCOHOL
46	SIVALINGAM	48	M	4	1918628	ALCOHOL
47	SENTHIL KUMAR	39	M	2	1921288	ALCOHOL
48	KARUPPAIAH	52	M	3	1923647	ALCOHOL
49	PACHAIYAMMAL	33	F	5	1924157	GALLSTONE
50	THANDAPANI	51	M	1	1928417	ALCOHOL

BALTHAZAR CT SCORE	APACHE	VASOACTIVE DRUG	MODS	OUTCOME	IAP	IAP	IAP
3	4	NO	NO	DISCHARGE	6	6	5
6	8	NO	NO	DISCHARGE	15	15	15
7	8	YES	YES	EXPIRED	8	8	8
7	10	NO	NO	DISCHARGE	15	15	15
6	8	NO	NO	DISCHARGE	12	12	12
7	8	NO	NO	DISCHARGE	13	13	13
8	33	YES	YES	EXPIRED	20		
8	10	NO	YES	DISCHARGE	11	11	12
6	4	NO	NO	DISCHARGE	4	5	4
7	8	NO	NO	DISCHARGE	15	15	15
6	9	NO	NO	DISCHARGE	12	12	12
5	8	NO	NO	DISCHARGE	8	6	8
7	9	NO	NO	DISCHARGE	13	13	13
2	4	NO	NO	DISCHARGE	9	9	8
4	5	NO	NO	DISCHARGE	8	8	8
7	8	NO	NO	DISCHARGE	12	12	12
6	6	NO	NO	DISCHARGE	5	5	4
8	15	YES	YES	EXPIRED	15	16	16
7	24	YES	YES	EXPIRED	15	15	14
4	6	NO	NO	DISCHARGE	4	4	6
6	5	NO	NO	DISCHARGE	6	6	6
4	10	NO	YES	DISCHARGE	8	8	8

7	8	YES	YES	DISCHARGE	13	13	13
8	8	NO	NO	DISCHARGE	12	12	12
8	17	YES	YES	EXPIRED	18	17	17
6	4	NO	NO	DISCHARGE	4	4	4
7	8	NO	NO	DISCHARGE	11	11	11
5	4	NO	NO	DISCHARGE	6	8	6
6	4	NO	NO	DISCHARGE	8	6	6
7	14	YES	YES	DISCHARGE	15	15	15
4	6	NO	NO	DISCHARGE	8	8	8
5	8	NO	NO	DISCHARGE	13	13	13
3	8	NO	NO	DISCHARGE	9	9	9
7	7	NO	NO	DISCHARGE	8	8	8
5	10	NO	YES	DISCHARGE	12	12	12
6	5	NO	NO	DISCHARGE	6	6	6
8	14	YES	YES	DISCHARGE	16	16	16
7	4	NO	NO	DISCHARGE	6	6	5
7	8	NO	NO	DISCHARGE	8	6	7
6	5	NO	NO	DISCHARGE	6	6	6
8	15	YES	YES	DISCHARGE	16	16	16
4	8	NO	NO	DISCHARGE	11	10	9
5	8	NO	NO	DISCHARGE	12	12	12
6	4	NO	NO	DISCHARGE	5	5	5
3	8	NO	NO	DISCHARGE	8	6	6
7	16	YES	YES	EXPIRED	18	18	18
8	15	YES	YES	DISCHARGE	15	15	15
5	8	NO	NO	DISCHARGE	13	13	13
7	6	NO	NO	DISCHARGE	5	5	5
6	6	NO	NO	DISCHARGE	6	5	6

IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP
6	6	5	5	5	6	4	4	4	
15	15	15	14	14	14	14	14	14	14
8	8	8	9	9	9	9	8	8	8
15	15	15	16	16	16	17	15	14	14
12	12	13	13	12	12	11	11	11	10
13	13	13	12	12	12	12	12	11	11
13	13	13	13	13	15	15	19	19	16
4	4	3	4	4	4	4	4	3	4

15	15	15	14	14	14	14	14	13	13
12	12	12	11	11	12	10	10	9	9
6	8	7	6	7	8	6	7	8	6
13	12	12	12	12	11	10	10	10	10
9	8	8	8	7	8	7	7	7	
8	8	7	7	8	7	7	7	7	7
10	10	10	10	10	10	10	10	9	9
4	5	4	4	4	4	4	4	4	4
15									
6	7	6	6	5	4	4	6	5	
6	6	6	6	6	5	5	5	5	5
9	9	9	11	11	10	13	14	13	13
13	13	12	13	13	13	14	14	14	13
12	13	13	15	15	15	15	15	13	14
19	19	19	19						
4	5	4	4	4	5	4	4	4	4
11	11	11	10	10	10	10	10	9	9
6	7	7	6	5	6	6	6	6	5
6	6	6	6	6	5	5	5	6	5
15	15	16	14	14	14	13	13	12	14
8	8	7	7	8	8	7	7	6	7
13	12	12	12	12	10	10	10	11	10
9	9	10	9	9	9	8	8	8	9
8	8	7	8	7	7	7	8	8	8
12	12	12	11	12	15	15	15	13	13
5	6	5	6	5	5	5	5	5	5
16	16	19	19	18	18	18	18	18	17
4	4	4	4	4	4	4	4	4	4
5	5	6	6	5	4	4	4	4	4
6	6	5	5	5	4	4	5	5	4
16	16	15	15	15	14	14	15	15	14
9	9	9	8	9	8	9	8	9	8
12	12	12	12	12	11	11	11	11	11
5	5	5	5	5	5	4	5	5	5
6	7	6	8	6	7	6	6	6	7
19	18	18	19	19	19				
15	16	19	19	18	18	18	18	19	19
13	12	12	12	12	10	10	10	10	10
5	5	4	4	3	3	3	4	4	4
5	6	5	6	6	5	5	5	5	5

IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP
13	13	13	13	14	14	13	13	13	12
8	10	10	11	12	12	16	16	16	
14	14	12	12	11	11	11	11	10	9
10	10	10	9	9	9	9	9		
10	11	11	11	10	10	10	9	9	9
16	17	17	17	16	15	15	15	15	13
13	14	13	13	14	13	12	12	12	12
9	9	10	10	9	9	9	8		
7	7	7	7						
10	11	10	9	9	9	9	9	9	9
9	9	8	8	7	7	7	6	5	5
4									
6	5	5	5	5	4	4	4		
15	15	15	15	12	12	12	11	11	10
13	13	13	13	13	12	12	12	10	10
13	13	14	13	14	13	13	12	12	12
5	4	4							
9	9	9	9	9	7	7	7	7	
6	5								
14	12	13	13	13	14	12	12	11	10
10	11	10	10	9	9	9	9	9	8
8									
8	7	8	7						
13	13	13	13	12	12	10	11	11	11
5	5								
19	19	19	18	18	16	16	16	15	14
4									
4									
4									
14	14	15	15	15	13	12	12	12	12
7									

11	10	10	10	9	8	8	8	8	8
5									
6	7								
18	17	16	15	15	15	16	15	14	14
10	10	10	10	9	9	9	9	9	9
4	4								
5	5	5	5						

IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP	IAP
12	12	11	10	10	10	8	8	8	8
9	9	9	8	9	8	8			
13	10	10	10	9	8	8			
12	12	10	10	10	10	8	8	8	8
8	7	7	7						
8	8	8							
10	10	10	8	8	8	8	8	8	8
11	10	11	11	9	9	9	9	6	6
10	10	9	9	9	9	9			

8	8								
11	9	9	9	9	8	8	9	8	
14	14	12	10	10	10	9	9	9	9
10	10	10	8	8	8	7			
8									
13	13	13	14	13	12	10	10	10	9
9	9								