A DISSERTATION ON

"A STUDY ON EARLY ENTERAL FEEDING IN ACUTEPANCREATITIS - NASOGASTRIC VS NASOJEJUNAL FEEDING"

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M.S. (General Surgery)Branch –I

INSTITUTE OF GENERAL SURGERY,

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CERTIFICATE

This is to certify that the dissertation titled "**A STUDY ON EARLY ENTERAL FEEDING IN ACUTE PANCREATITIS - NASOGASTRIC VS NASOJEJUNAL FEEDING**" is the bonafide work of **Dr.MISHALL PRASANNAN**(exam no 221711010) during his M.S. (General Surgery) programme between **2017 - 2020**, and was done under my supervision and is, herewith submitted in the partial fulfilment of**M.S. (BRANCH-I) - General Surgery,May 2020** examination of **The Tamil Nadu Dr.M.G.R. Medical University**.

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DECLARATION

I hereby, declare that this dissertation titled "A STUDY ON EARLY ENTERAL FEEDING IN ACUTE PANCREATITIS - NASOGASTRIC VS NASOJEJUNAL FEEDING" represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice , and are acknowledged.

I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award , degree or diploma to any other University board , either in India or abroad.

This is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery Degree Branch- I (General Surgery).

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“Research is to see what everybody else has seen, and to think what nobodyelse has thought.” - Albert Szent-Gyorgyi

I realize with a deep sense of humility and gratefulness that whatever little I have done now would not have been possible, but for certain mentors, who have enlightened my path to wisdom.

“Surgery is learnt by apprenticeship and not from textbooks,

not even from one profusely illustrated ” - Ian Aird.

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INTRODUCTION

Acute pancreatitis is a deadly and inflammatory condition of the pancreas that is painful and extremely distressing to both the patient and the treating medical personnel. Inspite of all the advances in intensive care medicine over the recent past, the mortality and morbidity of acute pancreatitis has always been unchanged at about 10%. On top of that, the organ being relatively inaccessible makes it very difficult to diagnose with clinical and surgical acumen.

The imaging studies available at our disposal seem inadequate to deal with proper diagnosis of the pancreatic conditions, making surgery and direct vision among the last line of investigations to deal with this organ. The chronic and hereditary variants of pancreatitis go on to devastate the patient for many more years to come. Sufferers have to endure so much pain and malnutrition, and are most likely left with a significantly increased risk of pancreatic malignancy in the future

Acute pancreatitis is managed initially by keeping the patients under complete bowel rest through nil per oral and supplementing the patient with adequate intravenous hydration and analgesics. The rationale behind this line of management is that enteral feeding causes stimulation of the exocrine activity of the pancreas and further triggers the inflammatory cascade of acute pancreatitis.

But, there have been many studies that suggest that enteral feeding can reduce the infection rates by making the protective barrier of the gut, thus limiting the translocation of bacteria to the blood stream and causing septicemia.

Studies so far which compared early versus delayed enteral feeding in acute pancreatitis have shown that patient with acute severe pancreatitis can be started safely with enteral feeding within 24hrs of onset of symptoms. This also reduces complications and allows early tolerance to oral diet and decreases hospital stay. It also reduces the need for parenteral nutrition, thereby reducing the financial burden on patients.

Enteral feeding can be given through either nasogastric (NG) or the nasojejunal (NJ) route.Nasojejunal route of feeding has been shown to be far more successful in early delivery of enteral nutrition to the patient, but feeding through nasogastric route is also not far behind.

NJ tube placement is a cumbersome procedure because, although both endoscopy andfluoroscopy are highly effective for placement of small bowel feeding tubes, it can take a medical personnel up to 30 minutes to achieve accurate post-pyloric placement of a feeding tube iin the jejunum. In contrast, Nasogastric tube placement is a very easy bedside procedure and can be administrated by a much wider spectrum of medical personnel. Therefore, NJ tube placement can become expensive and inconvenient compared with NG tube placement.

This study aims to compare patients with acute severe pancreatitis at Madras medical college, subjected to nasogastric and nasojejunal feeding and following them up close for their response and arriving at a conclusion for the same.

AIMS AND OBJECTIVES

1. To assess any difference between NG and NJ routes, in tolerance, acute phase response, and pain.

2. To study the clinical course of patients managed conservatively

REVIEW OF LITERATURE

The incidence of acute pancreatitis (AP) has significantly gone up during the past two decades. AP is responsible for upwards of three lakh hospitaladmissions annually in the United States of America. Most of these patients develop amild and self-limited course; but, about 10% to 20% of patients havea very rapidly progressing inflammatory cascade which is associated with prolongedlength of hospital stay and significantly increased mortality and morbidity.

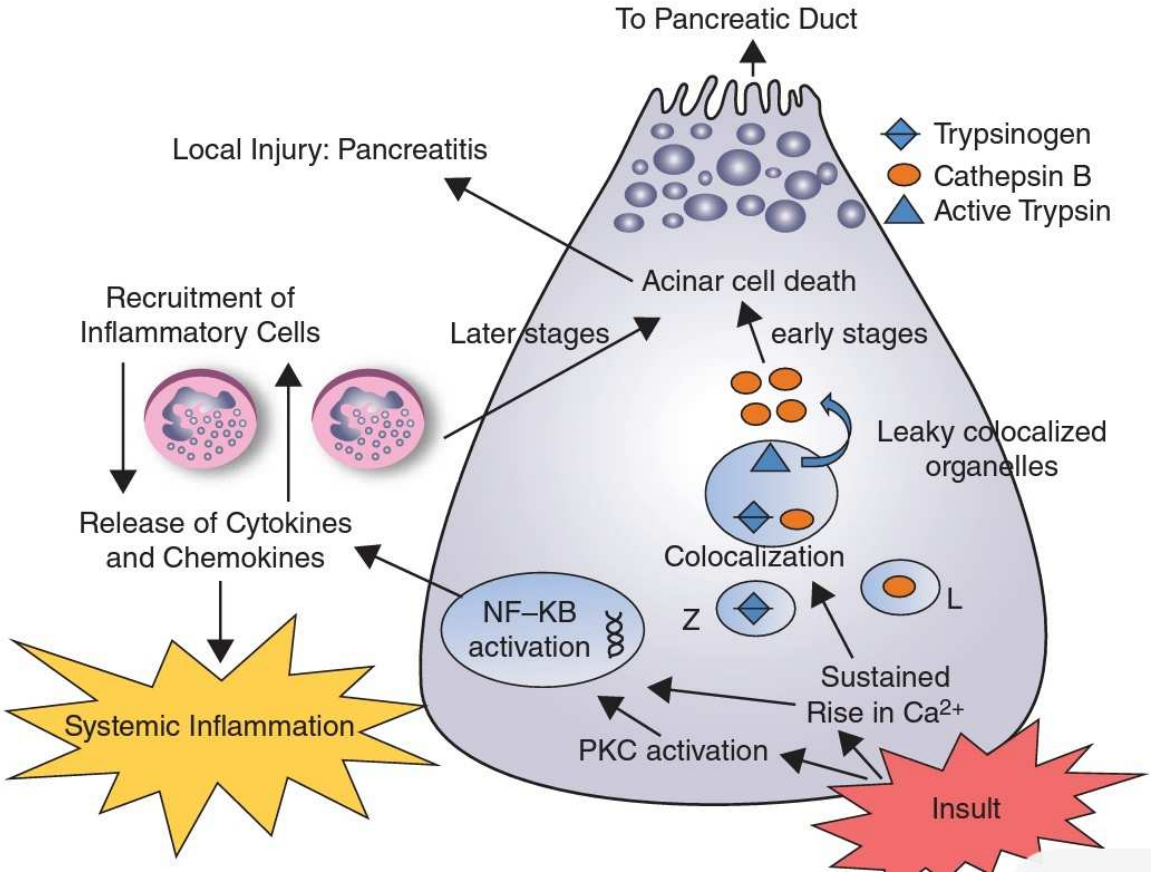
Patients with mild pancreatitis have a mortality rate of lessthan 1%, but in severe pancreatitis, this increases up to 10% to 30%. The mostly encountered cause of death in these group of patientsis found to bemultiorgan dysfunction syndrome. Death in these pancreatitis hasa bimodal distribution; in the initial 2 weeks, called as the earlyphase, the multiorgan dysfunction syndrome is actually the endevent ofan intense inflammatory cascade which is triggered initially by pancreatic parenchymal inflammation. Mortality after the initial 2 weeks,which is also called as the late period, is caused by septic complications.

Pathophysiology

The exactpathophysiology and events whereby the predisposing factors such as alcohol and cholilithiasis cause pancreatitis is not completelyknown. Most of the available research believe that AP is the end result ofabnormal activation of pancreatic enzyme inside the pancreatic acinii.

Immunolocalization studies have revealed that after about fifteen minutes of injury to the pancreatic parenchyma, both lysosomes and zymogen granules colocalize inside

pancreaticacinar cells. The important fact that zymogen and lysosomecolocalization actually occurs before elevation of serumamylase levels, edema of the pancreas, and other indicators of pancreatitis are evident shows thatcolocalization is one of the earliest steps in the pathophysiology and is not a consequence of acute pancreatitis.



A few studies also suggest thatthe lysosomalenzyme cathepsin B is the reason of activation of trypsin in these colocalizationorganelles. In vitro and in vivo studies have revealed an elaborate model of acinar cell death which isactullyinduced by premature activation of the enzyme trypsin.

In this model, once thecathepsin B in trypsinogen an lysozomesinside the zymogen granules come in contact by colocalization which is induced by the pancreatitis-inciting stimuli, the activated trypsin then causes leakage of thecolocalized organelles, releasing all the cathepsin B into the cytosol.

This is the cytosolic cathepsin B that is responsible for the induction necrosis or apoptosis, culminating to acinar cell death.This further shows that we can use cathepsin B inhibitors as a prophylactic measure to prevent acinar cell death and controlling of the inflammatory cascade.Genetic research has also shown that mice which had their cathepsin B knocked out have a drastically decreased severity of pancreatitis.

Intra-acinar activation of pancreatic enzyme causes digestionof normal pancreatic parenchyma. As a reply to this initial abuse, theacinar cells start to release proinflammatory cytokines, such astumor necrosis factor-α (TNF-α), interleukin (IL)- 1, IL-6, and IL-2, and other anti-inflammatory mediators, like IL-1 receptor antagonist and IL-10. These mediators cause propagation of local and systemic response, and as such do not cause any direct injury to the pancreas.

So , TNF-α, IL-7 and IL-1, macrophages and PMNs are actively recruited into the pancreatic parcenchyma and which will cause the releaseof more interleukins and TNF aplha, reactive oxygen species,prostaglandins, leucotrienes and platelet activating factor.

This inflammatory cascade further aggravates the attack on the pancreas because it results in an increase in the capillary permeability and tries to destroy the microcirculationof the pancreas.

In more severe cases, this inflammatory cascade eventually leads to pancreatic hemorrhage and pancreatic necrosis. On top of all these events, the chemicals released bymobilised neutrophils in the pancreas aggravate the pancreatic insult because they cause activation of the pancreatic enzymes.

In about 80 to 90 percent of acute pancreatitis , this inflammatory cascade is usually self-limiting. In the unfortunate remaining patients, a deadly cycle of repeated injury of the pancreaswithboth systemic and local inflammatoryreaction persists. In a very small subset of unfortunate patients, there will be a profound release of copious amount inflammatory mediators into the blood.

The activated neutrophils will cause acute lung injury and can by itself, induce theadult respiratory distress syndrome which is very frequently evident in people with severe pancreatitis. This persistent inflammatory response is responsible for the cause of death in the early phase ofpancreatitis.

Risk Factors :

Cholilithiasis and alcohol abuse account for 70% - 80% of cases of acute pancreatitis. In pediatric age group, Blunt trauma of the abdomen and afew systemicdiseases account for the conditions that lead to pancreatitis.Autoimmune and drug-induced pancreatitis should be kept in mind when treating patients with Sjogrens disease and systemic lupus erythematosus.

Biliary Pancreatitis :

Cholelithiasis emerges as the most common cause of acute pancreatitis in the developed world. It is usually seen in females with their age between their 50s and 70s. The exact mechanism which triggers the attack on the pancreas is not yet fully understood , but there are two theories to work with as of now.

These are 1. Obstructive theory, and 2.Reflux theory

In obstructive theory, excessive pressure inside the main and accessory pancreatic duct is the reason for pancreatic injury. The increase inintraductalpressure is the result of continuous secretion of pancreaticjuice in the presence of pancreatic duct obstruction.

The Reflux theoryimplies that gallstones which have been impacted in the ampulla of vater produces a common channel which will allow reflux of bile salts into the pancreatic duct. In vivo models have revealed that bile salts triggersnecrosis of the acinar cells directlyby increasing the cytoplasmic calcium concentration. Sadly, this remains to be proved in humans.

Alcoholic Pancreatitis

Worldwide, ethanol ingestion in excess accounts for the second most common cause of acute pancreatitis. It is more common in men in the age group of 35 to 45. Only about 5 to 10 percentage of patients who consume alcohol developAcute pancreatitis. The following factors contribute to ethanol-induced pancreatitis : 1. ethanol abuse (>100 g/day for more than 5 years), 2.Smoking,and 3.Genetic predisposition.

When compared with nonsmokers, the relativerisk of alcohol-induced pancreatitis among those who have the habit of smoking is 4.9.Consumption of ethanol leads to a wide variet of detrimental effects in the pancreas.

First, Ittriggers the production of IL-1 and TNF-αthroughproinflammatory pathways such as nuclear factor κB(NF-κB).Secondly, Italso increases the expression and activity of caspases which areproteases that mediate apoptosis.

On top of all these, alcohol also causes a decrease in the pancreatic perfusion, induces spasm of the sphincter of Oddi, and causes obstruction of the pancreatic ducts through precipitation of proteinsinside the pancreatic ducts.

Anatomic Obstruction

Abnormal flow of the pancreatic secretion into the second part of the duodenum can lead to pancreatic injury. These occur in pancreaticneoplasms , infestation of pancreas by parasites, and congenital anomalies.

Pancreas divisumhas a controversial assossciation with acute pancreatitis. Patients with this anatomical variation have arelative outflow obstruction through theminor papilla which leads to amild lifetime risk for development of pancreatitis. They can be managed with minor papillotomy and Endoscopic retrograde cholangiopancreatography(ERCP) with stenting.*Ascarislumbricoides* and annular pancreas have also caused cases of acute pancreatitis.It is very rare for a patient having a tumor in the pancreas to develop acute pancreatitis , but there have been case reports regarding the same.

ERCP Induced Pancreatitis

Commonest complication after the procedure of ERCP is acute pancreatitis, with an incidence of 5% of patients.

It is more common in therapeutic ERCP than diagnostic ones.

The incidence is increased in the patientswho have had repeated attempts of cannulation, dysfunction of the sphincter of Oddi,and those with abnormal accessory ducts visualised after injection of contrast material.

Drug-Induced Pancreatitis

The following drugs have been assossciated with the causation of acute pancreatits

1. Sulfonamides
2. Metronidazole
3. Erythromycin
4. Tetracyclines
5. Didanosine
6. Thiazides
7. Furosemide
8. 3-hydroxy- 3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins),
9. Azathioprine,
10. 6-mercaptopurine,
11. 5-aminosalicylic acid
12. Sulfasalazine
13. Valproic acid
14. Acetaminophen.
15. Antiretroviral agents

Drug assosciated pancreatitis accounts for about 2 percent of the total number of patients affected with acute pancreatitis.

Metabolic Factors

1.Hypertriglyceridemia:

Triglyceridemetabolites can cause direct pancreatic injury. Type I, II,
and type V hyperlipidemia are more commonly associated with causing pancreatitis. A serum triglyceride level greater than 2000 mg/dL can clinch the diagnosis.Thehypertriglyceridemia which is secondary to hypothyroidism, diabetes mellitus, or consumption of alcohol typically never induces acute pancreatitis.

2.Hypercalcemia:

This is claimed to be through the activation of trypsinogen to trypsin which in turn leads tointraductalprecipitation of calcium, resulting in ductal obstruction . This causes subsequent attacks of acute pancreatitis. A similar mechanism allows patients of hyperparathyroidism to suffer from acute pancreatitis.

Miscellaneous Conditions:

A few other causes of acute pancreatitis are as follows:

1. Blunt and penetrating abdominal trauma
2. Prolonged intraoperative hypotension
3. Excessive pancreatic manipulation during surgery
4. Splenic artery embolization.
5. Scorpion venom stings
6. Perforated duodenal ulcers.

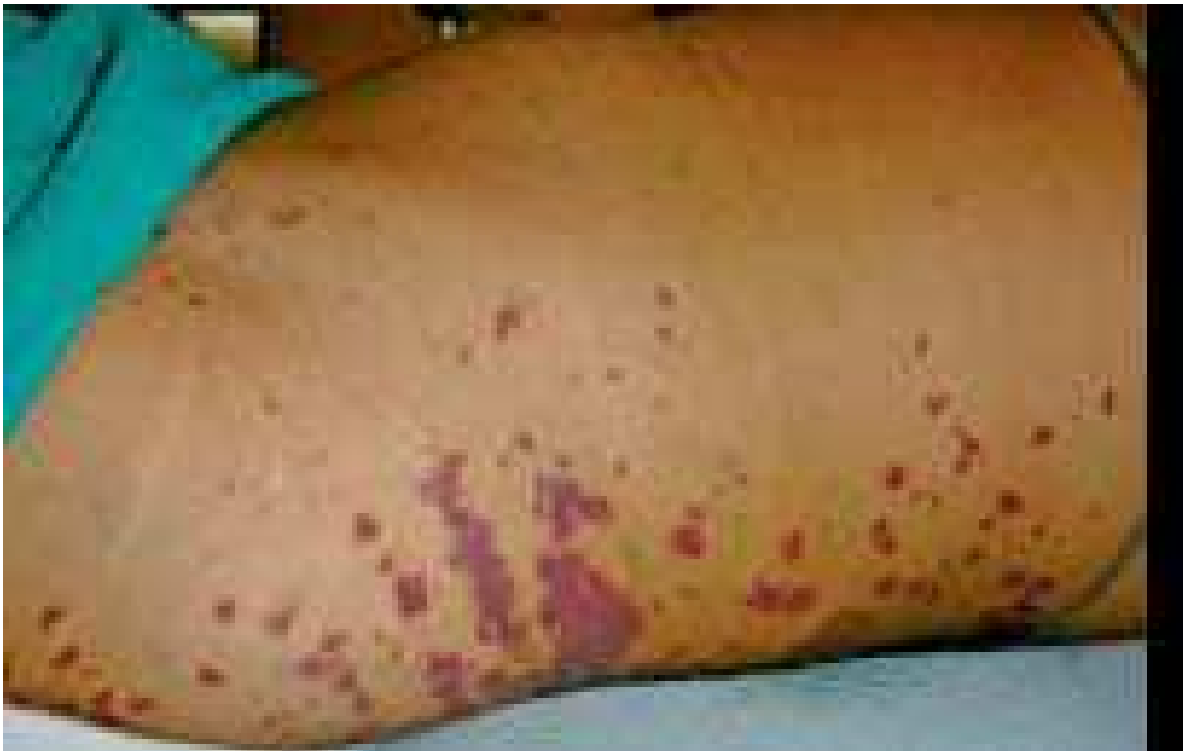
Clinical Manifestations

The most striking symptom of acute attack of pancreatitis is pain in the epigastric or periumbilical region with radiation of pain to the back. There is also nausea / vomiting that does not relieve the severe pain.The pain is very constant and if there is decrease or disappearance of the pain, there should some other diagnosis kept in mind.

Dehydration can follow,There is usually poor skin turgor in cases of pancreatitis with massive fluid loss in the third space, accompanied with tachycardia, hypotension, anddryness of the mucous membranes. Changes in the mentation quickly follows if the disease cascade is severe enough. The examination findings of the abdomen can vary as per the severity of the disease. In cases of mild pancreatitis, the abdominal examination may be normal or may show only slight tenderness in the epigastric region.

In severe pancreatitis there will be significant distention of the abdomen, along with generalized guarding, and rebound tenderness. The degree of pain described by the patient usually does not correlate with the abdominal examination findings or the extent of the disease process. Acute pancreatitis progressing to cause retroperitoneal bleeding will usually present with discolouration around the peri umbilical region and flanks of both sides.

Jaundice can occur when there is concominantcholedocholithiasis or edema of the head of the pancreas leading to obstruction of the infra pancreatic common bile duct. Left sided pleural effusion will cause decreased breath sounds , and dullness on percussion.



Discoloration of the flanks seen in acute pancreatitis

Diagnosis

Clinical findings along with a rise in the serum levels of pancreatic enzyme levels is usually enough to clinch the diagnosis of acute pancreatitis. Elevation of about threefold or higher of the levels of amylase and lipase level is usually required.

Serum amylase has a lesser half life in the serum when compared to the half life of lipase in the serum. In the cases which do not present to the hospital within the first 24 to 48hours of the onset of symptoms, the determination of serum lipase levels is regarded as a more sensitive indicator in order to make the diagnosis.

Serum levels of lipase is a more specific marker of AP. This is because elevated serum amylase levels is seen in a variety of other diseases like peptic ulcer disease, mesenteric ischemia, salpingitis,and macroamylasemia.

Pancreatitis patients are usually hyperglycemic; have leukocytosis and commonly present with abnormally elevated liver enzyme levels.

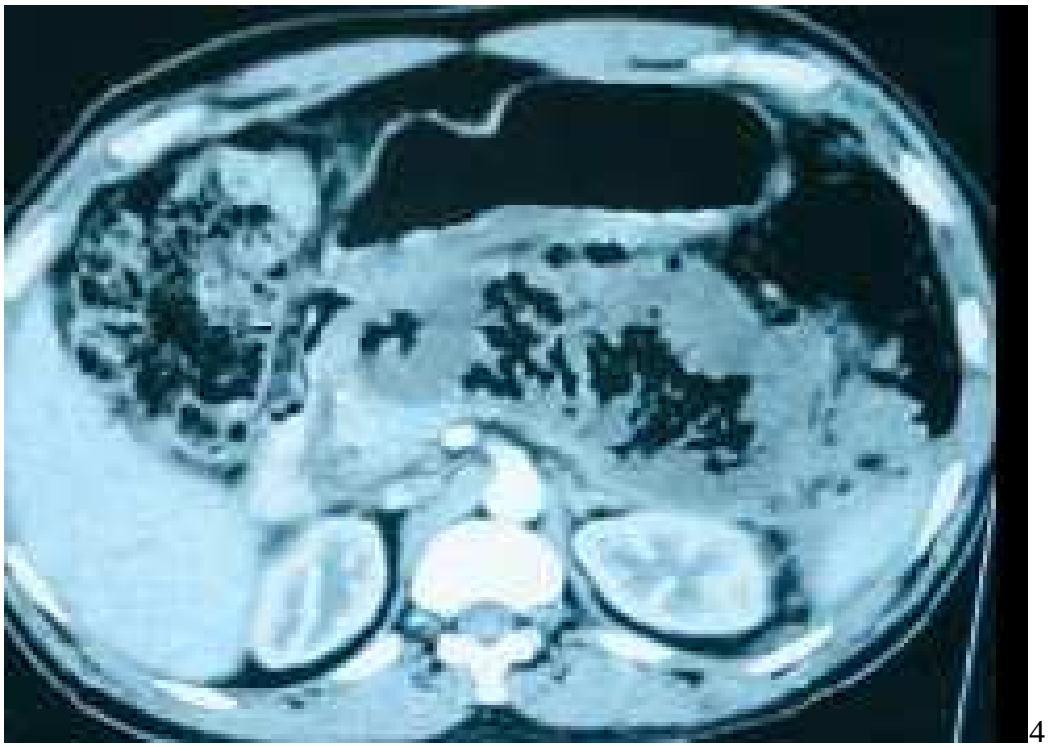
Elevated ALP along with an increased in serum levels of amylase and lipase has a 95 percent positive prediction value for biliary pancreatitis.

Imaging Studies

Simple abdominal radiographs are usually not much of a use to make a diagnosisof pancreatitis, but they can be very helpful in ruling out other conditions, like perforated ulcer disease.

A variety of nonspecific findings in AP include multiple air-fluid levels suggesting paralytic ileus, colonic spasm at the splenic flexure producing a colon cut off sign, and severe pancreatic head edema causing widening of the c loop of duodenum.

The use of ultrasound in making a diagnosis of pancreatitis is limited by the intra-abdominal fat and increased bowel gas as a esult of the ileus. Ultrasound has a very high sensitivity of picking up gall stones. A Combined elevationof liverenzymes andpancreatic enzyme levels along with the presence of gallstones on an ultrasound has a higher sensitivity (97%) and specificity(100%) for making a diagnosisof acute biliary pancreatitis.

 [4]

CT scan in a case of acute pancreatitis

The best modality for diagnosing acute pancreatitis is through Contrast-enhanced computed tomography (CT) especially when the study is done using a multidetector CT scanner. The portal venous phase is the most valuable contrast phase (65 to 70 seconds after the injection of contrast material).

This allows for the evaluation of the viability ofthe pancreatic parenchyma, the amount of peripancreatic inflammation,andthe presence or absence of intra-abdominal free air or fluid collections. In cases of acute renal failure, noncontrast CT scanning may also be of value by identifying fluid collections or extraluminal air. An abdominal magnetic resonance imaging (MRI) is usefulforthe evaluation the extent of necrosis,peripancreaticinflammation, and the presence offree fluid.

However, the limiting factors are its cost and availability. The fact thatpatients requiring imaging are usually critically ill and need to be in Medical intensive care units limit its

applicability in the acute phase.Although magnetic resonance cholangiopancreatography (MRCP)is not usually indicated in the acute setting of AP, its main role is in the evaluation of patients with unexplained or recurrent pancreatitisas it allows for complete visualization of the biliary andpancreatic ductal anatomy.

In addition, intravenous (IV) administrationof secretin will increasethe pancreatic duct secretion, which causes a transient distention of the pancreatic duct. Secretin MRCP is used in patients with AP with no evidence ofany predisposing condition to help in ruling out pancreas divisum, intraductalpapillary mucinous neoplasm (IPMN), or a small pancreatic ductal tumor.

In cases of gallstone pancreatitis, endoscopic ultrasound(EUS) is used in the evaluation of persistentcholedocholithiasis. RoutineERCP for a case of suspected gallstone pancreatitis usually reveals no evidence ofpersistent obstruction in most cases, and may actually increase symptoms and severity of pancreatitis because of manipulation of the gland.

EUS has been proved sensitive in identifying choledocholithiasis; as it allows for examination of the biliary tree and pancreas , along with no risk of worseningof the pancreatitis., ERCP can be used selectively as a therapeutic measure in patients in whom persistent choledocholithiasisis confirmed by EUS.

Assessment of Severity of Disease:

The Ranson scoring system was designed to evaluate the severity of AP. It predicts theseverity of the disease using 11 parameters obtained at the time of admission and 48 hours later.

The number of parameters poitive correlates directly with the morbidity and mortality of the patient. If three or more of the Ranson criteria are positive, Severe pancreatitis is diagnosed.

One of the main disadvantage of Ransons scoring is that it does notpredict the severity of disease at the time of the admission of the patient,as six of the parameters are assessed only after 48 hours of admission.

TheRanson score has a very low positive predictive value (50%) and very highnegative predictive value (90%). It is used only to rule out severe pancreatitis or to assess the sseverity of acute pancreatitis.

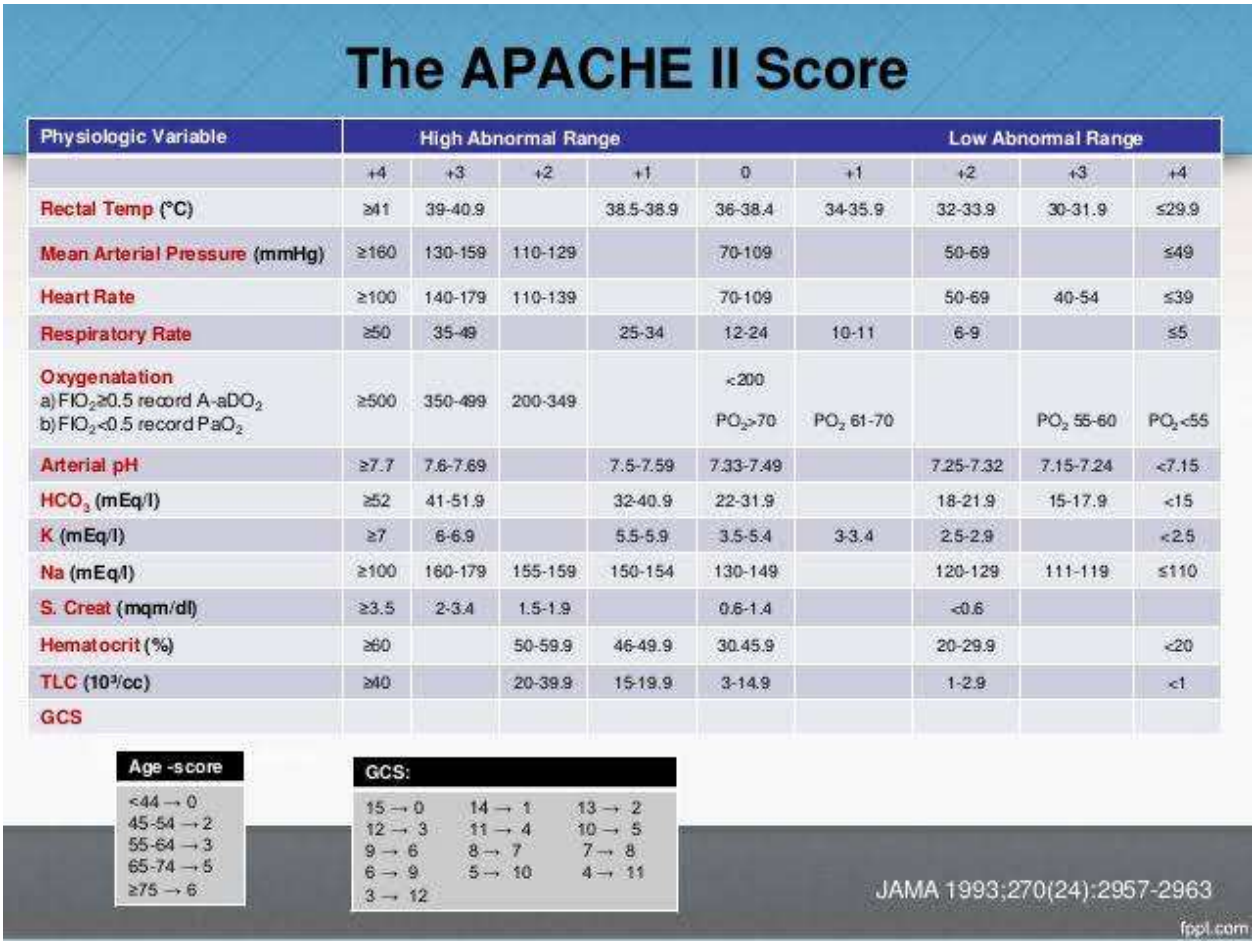
Table 68.3 Scoring systems to predict the severity of acute pancreatitis: in both systems, disease is classified as severe when three or more factors are present.

Ranson score	Glasgow scale
On admission	**On admission**
Age >55 years	Age >55 years
White blood cell count >16 x 10^9/L	White blood cell count >15 x 10^9/L
Blood glucose >10 mmol/L	Blood glucose >10 mmol/L (no history of diabetes)
LDH >700 units/L	Serum urea >16 mmol/L (no response to intravenous fluids)
AST >250 Sigma Frankel units per cent	Arterial oxygen saturation (PaO_2) <8 kPa (60 mmHg)
Within 48 hours	**Within 48 hours**
Blood urea nitrogen rise >5 mg per cent	Serum calcium <2.0 mmol/L
Arterial oxygen saturation (PaO_2) <8 kPa (60 mmHg)	Serum albumin <32 g/L
Serum calcium <2.0 mmol/L	LDH >600 units/L
Base deficit >4 mmol/L	AST/ALT >600 units/L
Fluid sequestration >6 litres	

ALT, alanine aminotransferase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; PaO_2, arterial oxygen tension.

Alternatively, AP severity can be addressed by the Acute Physiology andChronic Health Evaluation (APACHE II) score.It is based on the patient's age, the previous health status, and 12 other routine physiologicmeasurements.

APACHE II provides a general measure of theseverity of disease. A score of 8 or higher definessevere pancreatitis. The main advantage is that it can be used at the time of admission to the hospital and repeated at any time. However the limitation is that it is complex, and notspecific for AP, and is based on the patient's age, which can easily upgrade the AP severity score. APACHE II has a positive predictivevalue of 43% and a negative predictive value of 89%.



Using CT imaging characteristics, Balthazar and associateshaveestablished the CT severity index. This index, correlatesthe CT findingswith the patient's outcome. In 1992, the International Symposium on Acute Pancreatitis had defined severe pancreatitis as the presence of local pancreaticcomplications (necrosis, abscess, or pseudocyst) or any evidence of organ failure.

CT severity index		Modified CT severity index	
Prognostic indicator	Points	Prognostic indicator	Points
Pancreatic inflammation		Pancreatic inflammation	
Normal pancreas	0	Normal pancreas	0
Enlargement of pancreas	1	Pancreatic abnormalities with or without peripancreatic inflammation	2
Pancreatic abnormalities with peripancreatic inflammation	2	Pancreatic or peripancreatic fluid collection or fat necrosis	4
Single fluid collection	3		
Two or more collections or gas	4		
Pancreatic necrosis		Pancreatic necrosis	
None	0	None	0
<30%	2	<30%	2
30–50%	4	>30%	4
>50%	6		
		Extrapancreatic complications (pleural effusion, ascites, parenchymal complications, GI tract involvement)	2

Severe pancreatitis isusually diagnosed when there is anyevidence of organ failure or the presence of a local pancreatic complication.

Onset of pancreatitis and correlates closely with the severity of the disease. A serum CRP level of 150 mg/mL orhigher defines severe pancreatitis.

One of the major limitation of CRP is that itcannot be used on admission; and the sensitivity of the assay decreaseswhen CRP levels are measured within 48 hours after the onset of symptoms. Other than serum CRP, a variety of studies have shownother biochemical markers such as (e.g., serum levels of pro-calcitonin, IL-1, IL-6, elastase) that alsocorrelate with the

severity of the disease. However, their major limitation is their cost, and they are notavailable widely across all hospitals.

Treatment

The cornerstone in the management of AP is aggressive fluid resuscitation withisotonic crystalloid solution regardless of the cause or the severity of the disease. The infusion rate shouldbe individualized and should be adjusted on the basis of age, co-morbidities, vitals, mental status of the patient, skin turgor, and hourly urine output.

Those who do not respond to initial fluid resuscitation or those who have significantrenal, cardiac, or respiratory comorbidities usually requireinvasive monitoring with a central venous access and a Foleycatheter for continuous bladder drainage. Along with fluid resuscitation,these patients requirecontinuous pulse oximetry as one of the most commonsystemic complications of AP is hypoxemia due to acutelung injury associated with this disease.

Patients will benefit from supplementary oxygen to maintain their arterial saturation above 95%. Effective analgesia is also very important. Narcotics are usually used, especially morphine. After systemic administration of morphine, there is anincrease in tone in the sphincter of Oddi; but this does not create any negative impact on the patients suffering from AP.

There is also no proven benefit in treating AP with drugs likeantiproteases(e.g., gabexatemesilate, aprotinin), platelet-activating factorinhibitors (e.g., lexipafant), or pancreatic secretion inhibitors.

Nutrition in a case of Acute severe pancreatitis :

Nutritional support is crucial in the treatment of AP. Oral feeding is usually impossible because of persistent ileus, persistent pain, or intubation. A few patients with severe AP usually develop recurrentpain shortly after the oral route has been restarted. The mainoptions to provide nutritional support in such cases are through enteral feeding andtotal parenteral nutrition (TPN).

Even there is no major differencein the mortality rate between both types of nutrition, enteral feeding is associated with fewer infectious complications andalso reduces the need for pancreatic surgery. Eventhough TPN provides almost all nutritional requirements, it is associated with significant mucosal atrophy, reduced intestinal blood flow, increased risk of small bowel bacterialovergrowth , antegrade colonization with colonicbacteria, and an increase in bacterial translocation.

Patientswith TPN have also more central line infections and metabolic complications such as hyperglycemia and electrolyte imbalance.So wheneverpossible, enteral route of nutrition should be used rather than the TPN.Given the significant increase in mortality rates associated with septic complications of severe pancreatitis, a large number of physiciansadvocated the use of prophylactic antibiotics during the 1970s.

Recentmeta-analyses and systematic reviews that have evaluated a large number of randomized controlled trials have shown that prophylactic antibioticsdo not decrease the frequency of surgical intervention,infected necrosis, or mortality in patients suffering from severe pancreatitis.

In addition, they are usually associated with gram-positive cocci infection,such as by *Staphylococcus aureus,* and *Candida* infection, which isseen in about 5% to 15% of patients.

ROUTE OF NUTRITION

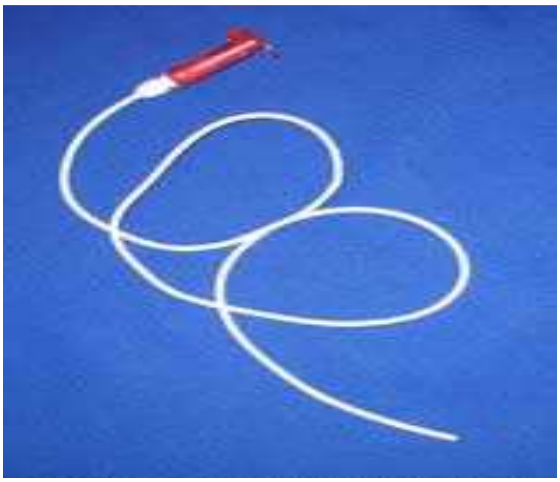
Many practitioners believe that the delivery of nutrients proximal to the duodenojejunalflexure will induce the release of cholecystokinin (CCK), and cause an exacerbation of the inflammatory process in the pancreas, as a result of stimulation of the exocrine part of pancreas. A lot of animal and human studies earlierhave shown an increase in the exocrine pancreatic secretion after starting enteral feeding, with an even greater response to intragastric feeding. However, none of these studies were actually carried out in acute pancreatitis (AP) where animal studies have shown that pancreatic exocrine secretion, in response to CCK stimulation, is suppressed. In addition, it isa known concept that the neural pathways affect the pancreatic secretion and the presence of nutrient particles in the jejunum causes a significant CCK release. The delivery of enteral feed distal to the doudenojejunal flexure does not prevent duodenal exposure to nutrients, as a degree of reflux is inevitable.

One study demonstrates that about 15% of tubes inserted for the purpose ofnasojejunal (NJ) feeding pass spontaneously through the pylorus; however, nasogastric (NG) feeding is found to be safe in the critically ill and ventilated patients. Adequate and a reliable placement of an NJ tube involves either sitting at the endoscopy suite or under radiographical screening, thereby exposing the critically ill patient to the risks of intrahospital transfer and delaying introduction of feeding. On top of all these, the risk of fiberopticduodenoscopy is also greater in a sick patient, and thus potentially

posinglogistical problems for the radiologist and/or endoscopist,asthese tubes require more frequent readjustment .

NASO GASTRIC TUBE

NASO JEJUNAL TUBE



Many studies have now shown that jejunal routeof feeding to be cheaper than total parenteral nutrition (TPN), and is associated with lesse septic complications and a possible modulation of the acute phase response.

A few studies have faced with the potential problems associated with insertion of NJ tubes and the delays which can occur in the introduction of feeding, which can occur because of the need to place the nasojejunaltubes under fluoroscopic or endoscopic guidance.

As a result of all of these ongoing research works, I have decided to address the following questions for my researchFirst, is NG route of feeding as safe and as effective as NJ feeding? Secondly, would the NG feeding result in exacerbation or reactivation of acute pancreatitis or a resurgence of pain?andthirdly, would NG feeding avoid some of the problems related to the insertion and use of the NJ tubes, namely the delay in insertion and the introduction of feeding and complications of the procedure undertaken to insert the tube? In an attempt to answer these questions we conducted a larger randomized study

MATERIALS AND METHODOLOGY

Study Centre	Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai
Duration of Study	Dec 2017 to Jan 2019
Study Design	Randomised control study
Sample Size	Approximately 6 0

Inclusion Criteria :

1.All patients with both clinical and biochemical presentation of Acute pancreatitis (abdominal pain + serum amylase at least 3 times the upper limit of the reference range),

2.An Acute Physiology and Chronic Health Evaluation (APACHE) II score of 6 or more.

Exclusion criteria :

Patients under 18 yr of age and pregnant females.

Methodology :

Patients were divided by simple randomisation into those who receive either nasogastric tube or a nasojejunal tube.

Patients who had nasogastric route of early enteral nutrition were labelled as Group A and the patients who revievednasojejunal mode of feeding were labelled Group B.

All Patients who fit the inclusion criteria will be observed and Monitored for the inflammatory response by daily measurement of APACHE II score, CRP levels, visual analogue score (VAS) for pain,

Feeds were commenced at full strength and a rate of 30 ml/h increasing to 100 ml/h over 24–48 h. The caloric target was 2,000 kcal per day. This was chosen over an individually calculated target in an attempt to simplify administration.

Each of these parameters was then observed on the day of commencement of feed and the following 4 days. Patients in both groups were followed throughout the period of hospitalization to detect any evidence of increase in the severity of pancreatitis as a result of the introduction of feeding.

RESULTS AND ANALYSIS

Analysis of the average scores of the Apache 2 scores, CRP and VAS scores of the patients who were subjected to the route of nasogastric enteral nutrition.

	Day 1	Day 2	Day 3	Day 4
Apache 2	29.3	28.6	27.8	26.9
CRP	81.3	126.6	133.23	137.33
VAS	4.5	4.7	4.4	3.93

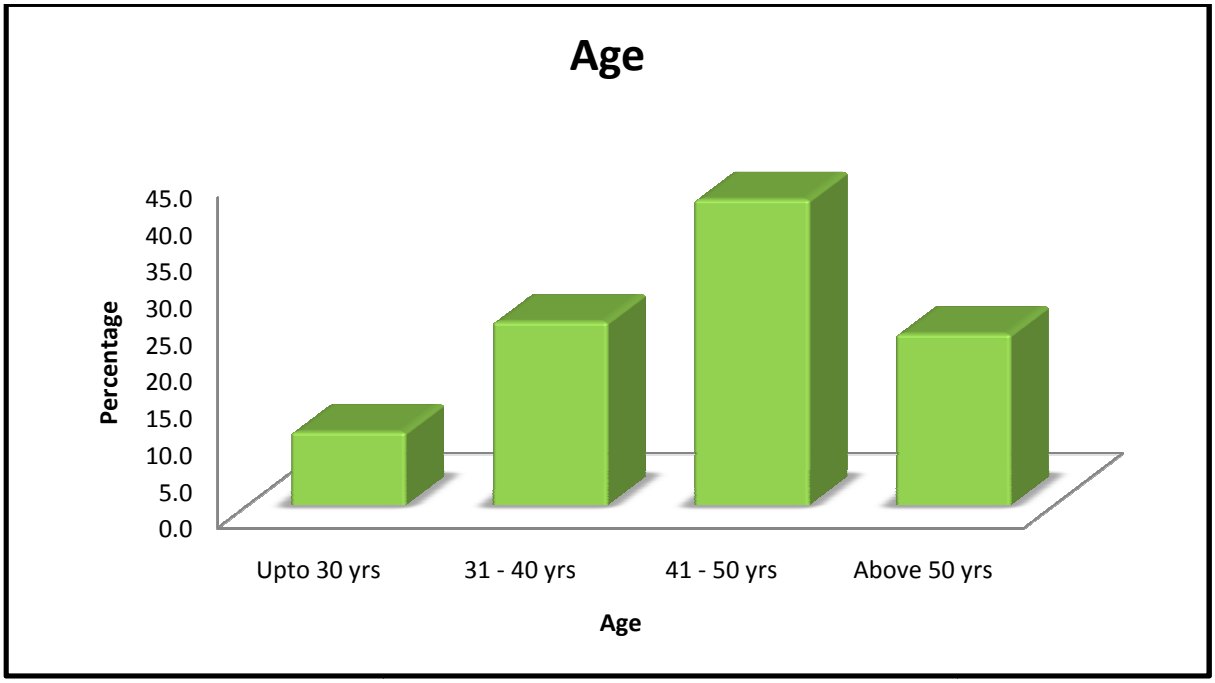
Analysis of the average scores of the Apache 2 scores, CRP and VAS scores of the patients who were subjected to the route of nasojejunal nutrition.

	Day 1	Day 2	Day 3	Day 4
Apache 2	23.4	24	24.5	24.9
CRP	74.6	97.2	114.6	132.2
VAS	4.4	5.3	5.6	5.7

1. The collected data were analysed with IBM.SPSS statistics software 23.0 Version.
2. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.
3. To find the significant difference between the bivariate samples in Independent groups the Unpaired sample t-test and the Mann-WHitney U test was used.
4. For the multivariate analysis for repeated measures the Repeated measures of ANOVA was used with Bonferroni correction to control the type I error on multiple comparison and the Friedman test was used.
5. To find the significance in categorical data Chi-Square test was used similarly if the expected cell frequency is less than 5 in 2×2 tables then the Fisher's Exact was used.
6. In all the following statistical tools the probability value .05 is considered as significant level.

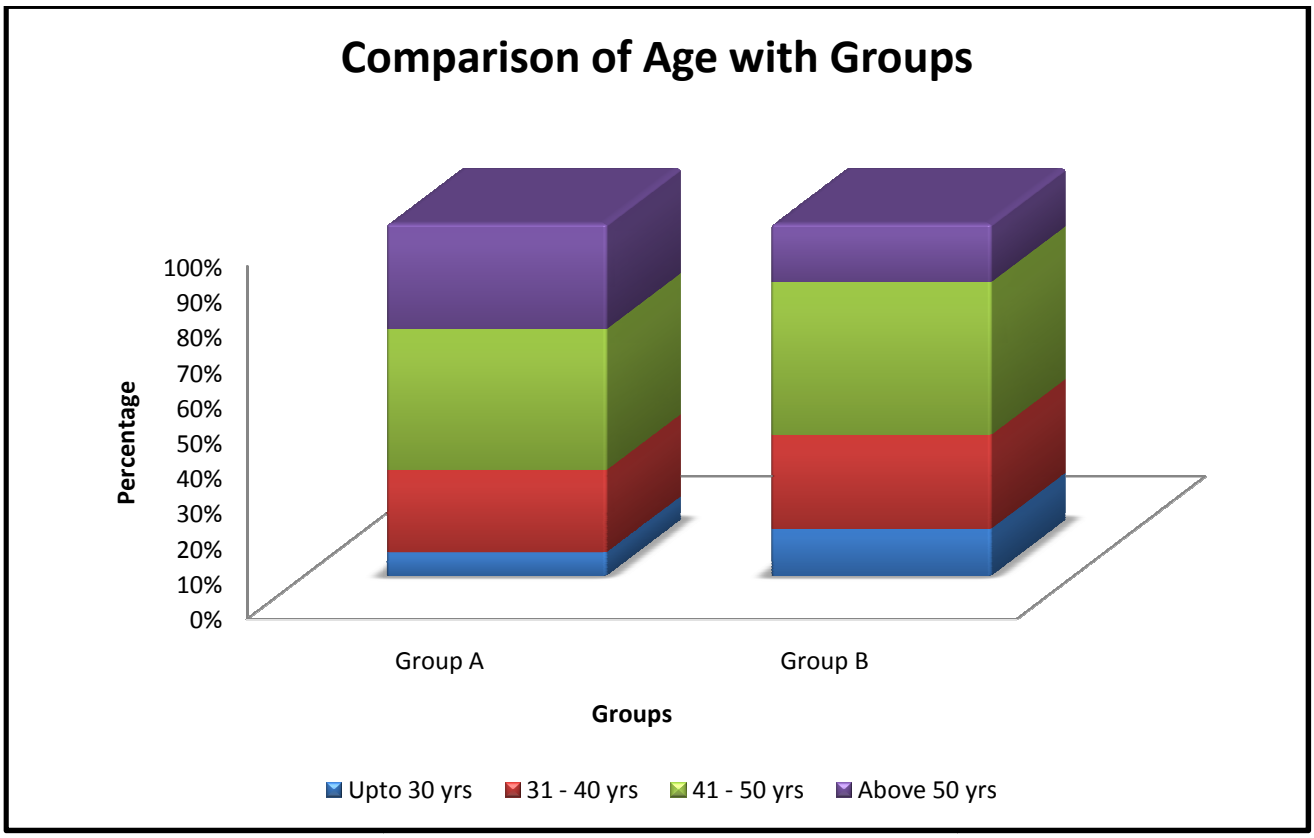
Age analysis among the entire sample size :

	Frequency	Percent
Upto 30 yrs	6	10.0
31 - 40 yrs	15	25.0
41 - 50 yrs	25	41.7
Above 50 yrs	14	23.3
Total	60	100.0



Age wise analysis of the entire subject group showed that a majority of the people suffering from acute severe pancreatitis were between the age of 41 to 50 years.

Comparison of Age with Groups							
			Groups		Total	□ 2 - value	P-value
			Group A	Group B			
AGE	Upto 30 yrs	Count	2	4	6	1.916	0.590 #
		%	6.7%	13.3%	10.0%		
	31 - 40 yrs	Count	7	8	15		
		%	23.3%	26.7%	25.0%		
	41 - 50 yrs	Count	12	13	25		
		%	40.0%	43.3%	41.7%		
	Above 50 yrs	Count	9	5	14		
		%	30.0%	16.7%	23.3%		
Total		Count	30	30	60		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at P>0.05 level							



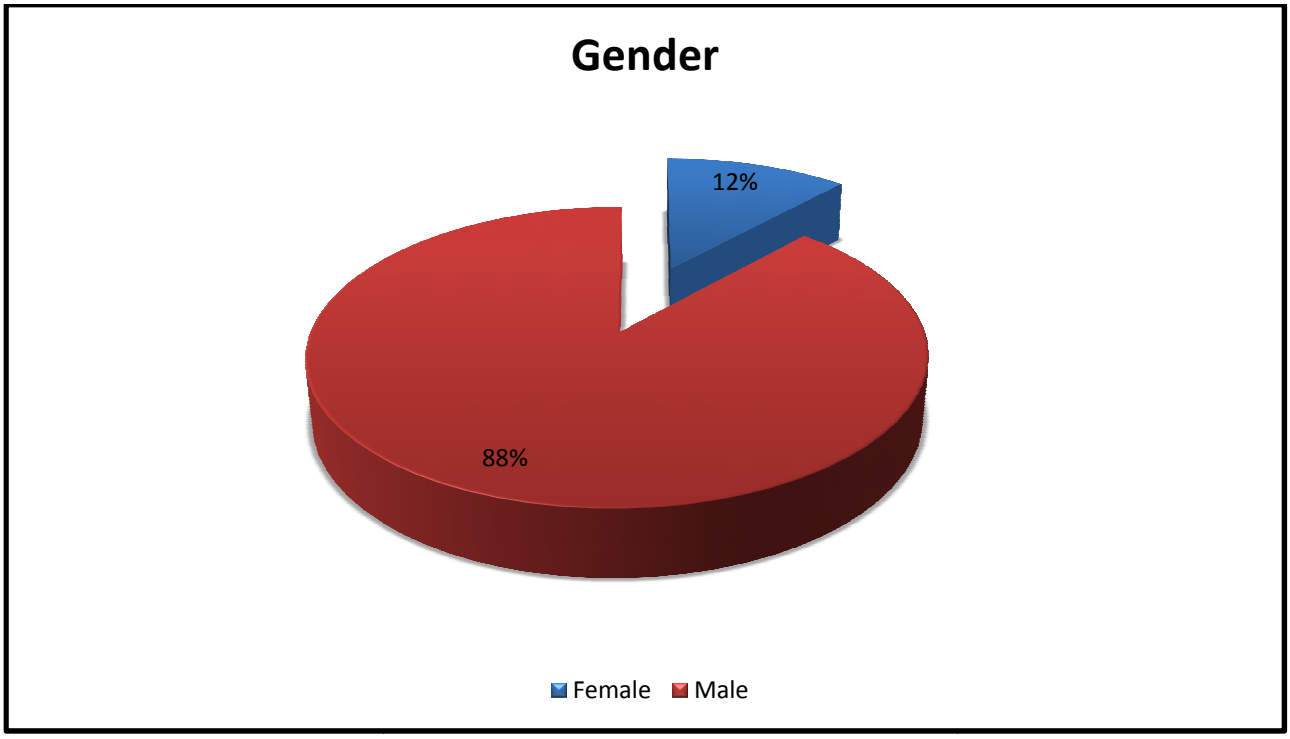
The subjects were randomised into two groups depending on their route of early enteral nutrition.

Group A recieved nasogastric nutrition whereas Group B recieved Nasojejunal mode of nutrition. Comparison of age among these two groups showed no statistical significance. P-value more than 0.05.

Gender analysis

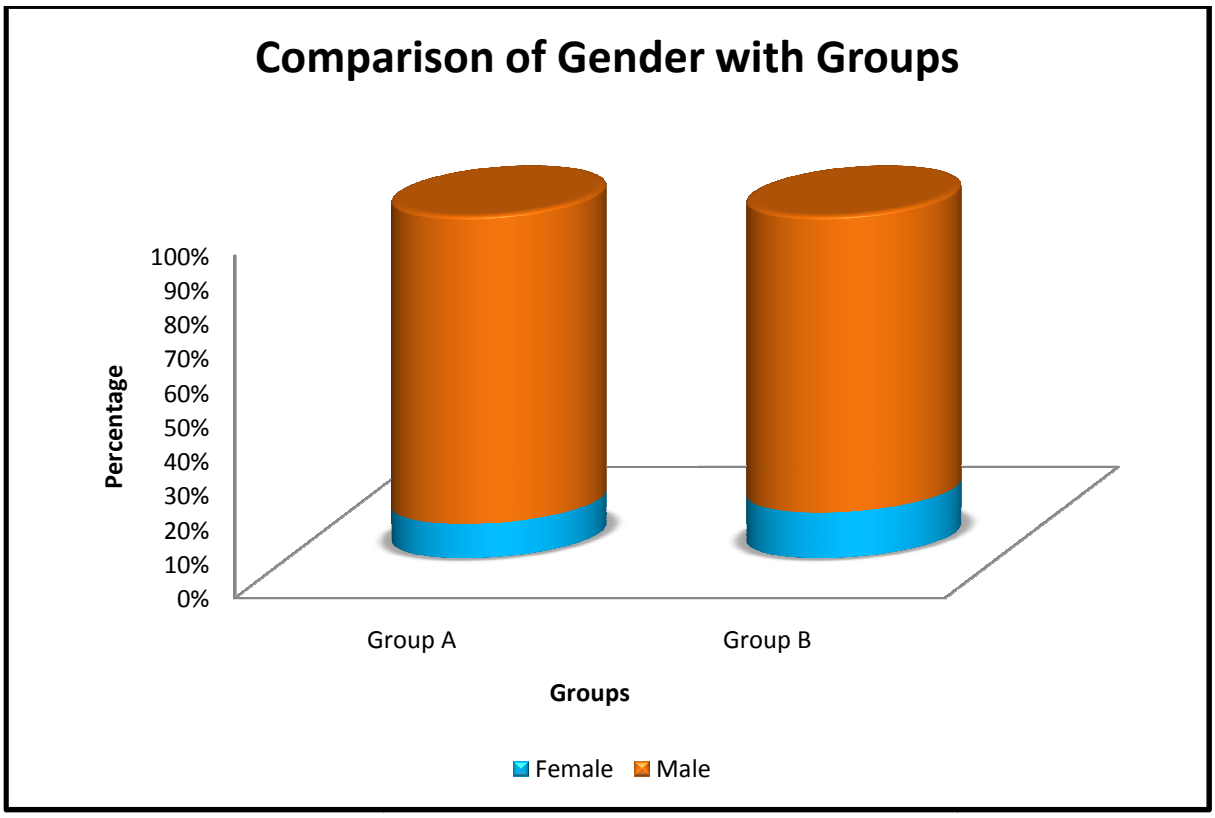
Gender analysis showed that as much as 88.3% of the sample size were males and 11.7% to be that of females.

	Frequency	Percent
Female	7	11.7
Male	53	88.3
Total	60	100.0



Comparison of Gender with Groups							
			Groups		Total	⍰2 - value	P-value
			Group A	Group B			
SEX	Female	Count	3	4	7	0.162	1.000 #
		%	10.0%	13.3%	11.7%		
	Male	Count	27	26	53		
		%	90.0%	86.7%	88.3%		
Total		Count	30	30	60		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at P>0.05 level							

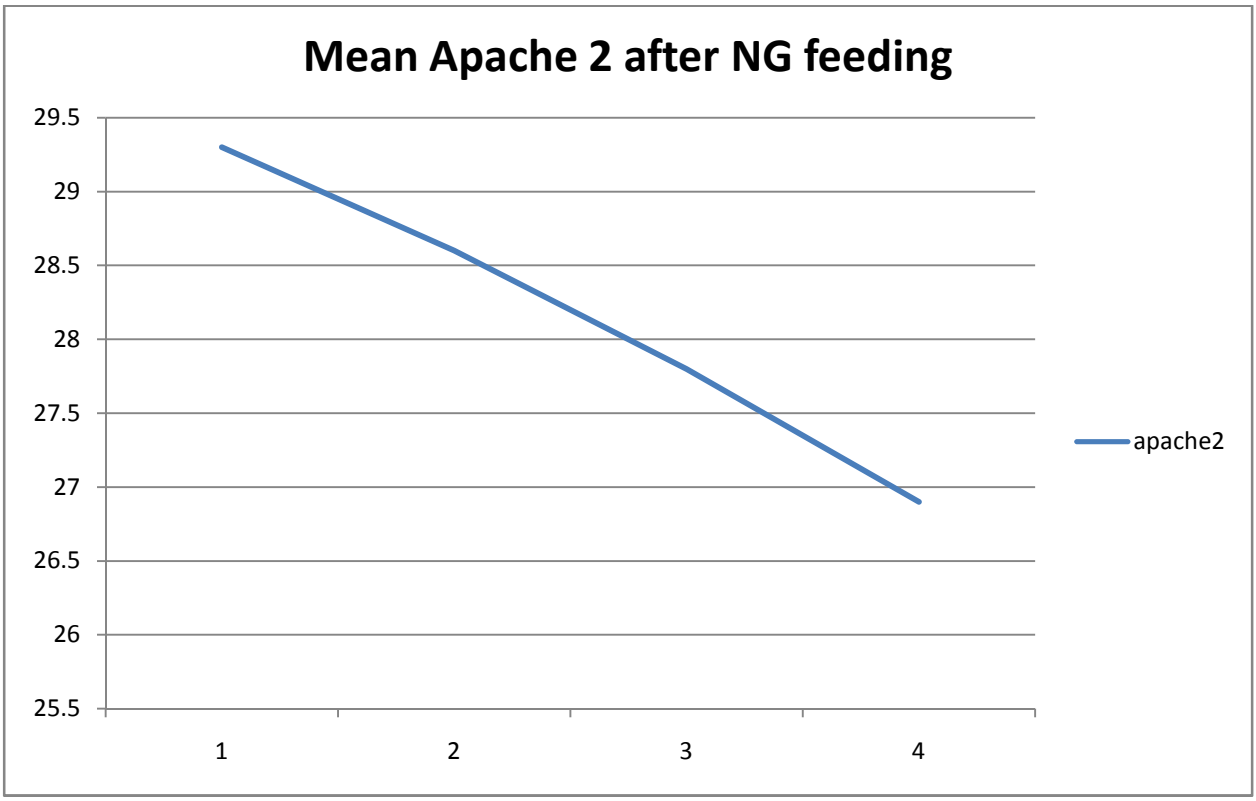
Among the two groups also, there was no statistical significance, the p-value being more than 0.05.



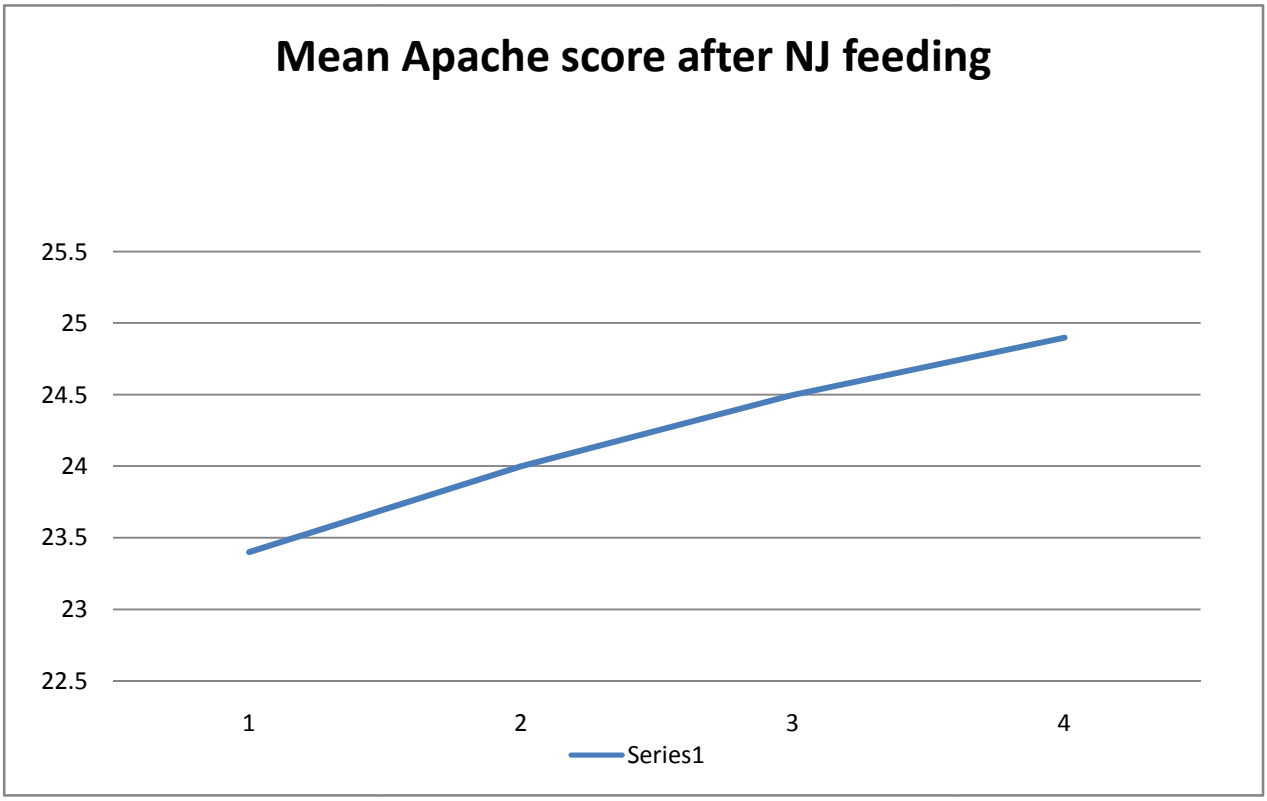
Male gender predominated both groups A and B.

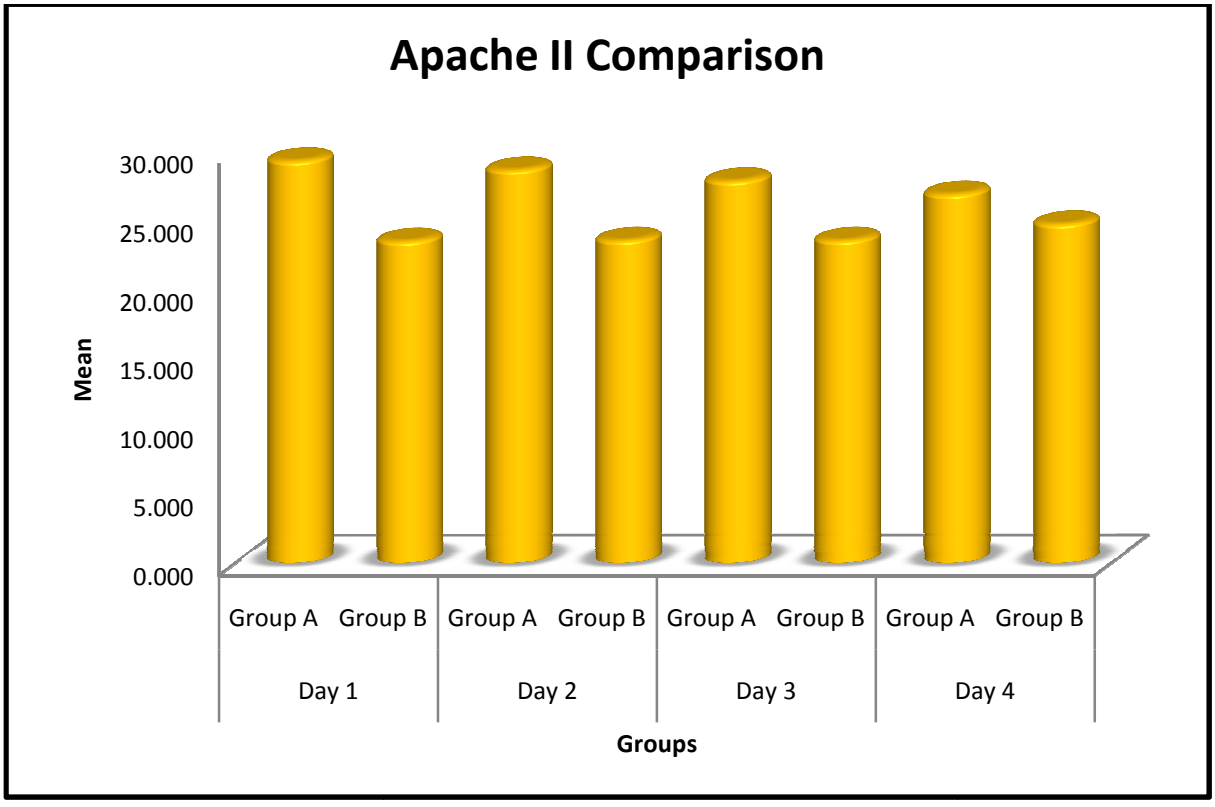
APACHE 2 scoring analysis

Serial monitoring of the ICU score using Apache 2 scoring system showed the following results.



The patients who were subjected to nasogastric mode of feeding showed a decreasing trend in the mean values of the Apache 2 scores drastically.


Mean Apache score after NJ feeding
25.5
25
24.5
24
23.5
23
22.5
1
2
3
4
Series1

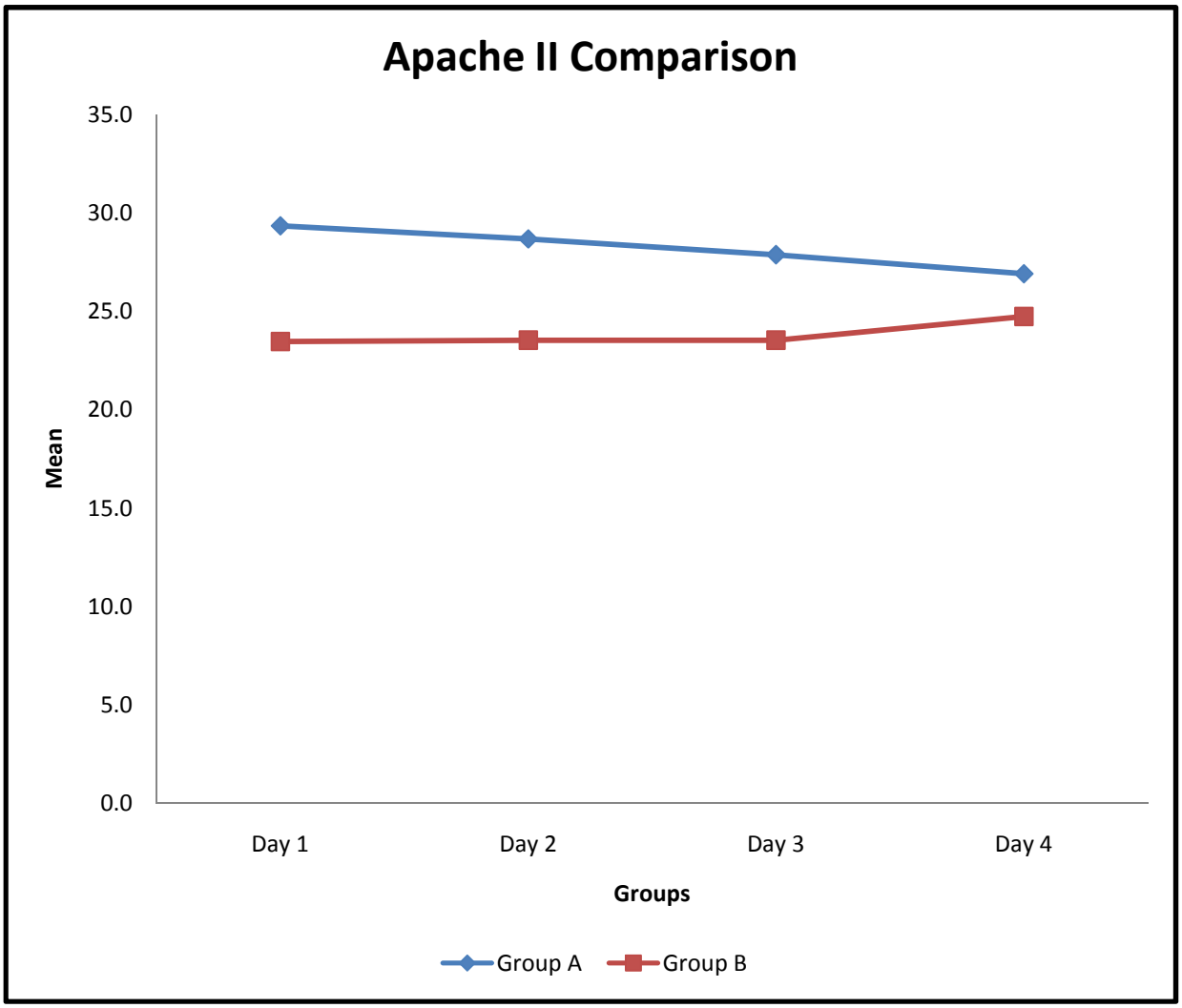

Apache II Comparison
30.000
25.000
20.000
15.000
10.000
5.000
0.000
Mean
Group A
Group B
Group A
Group B
Group A
Group B
Group A
Group B
Day 1
Day 2
Day 3
Day 4
Groups

APACHE II Comparison with Unpaired t-test						
Groups		N	Mean	S.D	t-value	P-value
Day 1	Group A	30	29.333	8.1678	2.487	0.015 *
	Group B	30	23.467	9.9437		
Day 2	Group A	30	28.667	6.9398	2.335	0.023 *
	Group B	30	23.533	9.8392		
Day 3	Group A	30	27.867	5.7460	2.017	0.050 *
	Group B	30	23.533	10.2713		
Day 4	Group A	30	26.900	5.8566	1.033	0.306 #
	Group B	30	24.733	9.8853		
* Sig at P < 0.05 and No Sig P > 0 .05 level						

When compared using the unpaired t-test, the Group A who had naso gastric feeds showed a gradual decrease in the mean Apache 2 scores from day 1 to day 4.

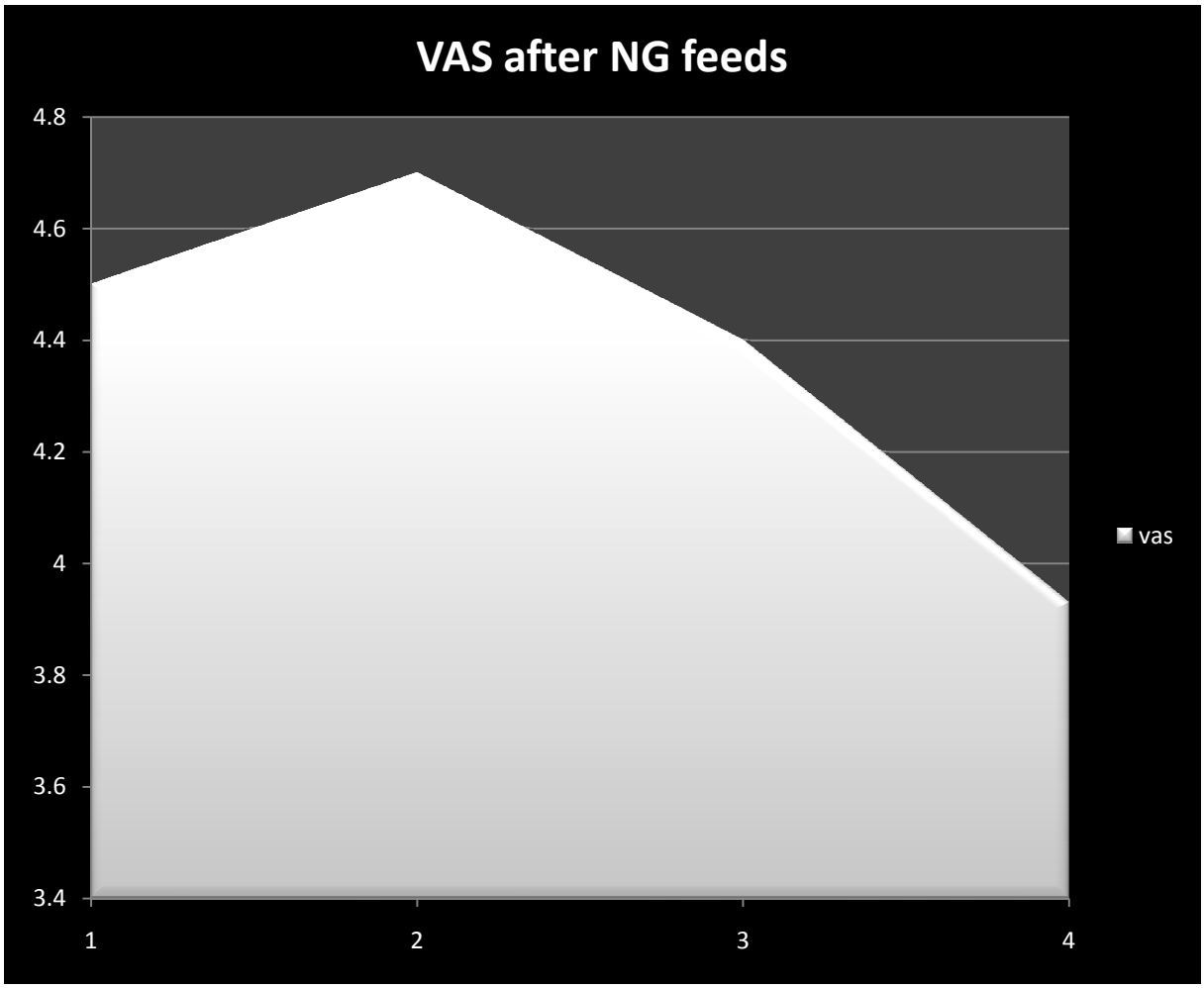
On the contrary, the patients who were in Group B showed a more or less same value of the mean Apache 2 scores with no big increase or decrease. The comparison has shown to be statistically significant P-values for days 1,2,3.

APACHE II comparison by Repeated measures of ANOVA					
Groups	Days	Mean	S.D	F-value	P-value
Group A	Day 1	29.3	8.2	4.007	0.035 *
	Day 2	28.7	6.9		
	Day 3	27.9	5.7		
	Day 4	26.9	5.9		
Group B	Day 1	23.5	9.9	1.300	0.280 #
	Day 2	23.5	9.8		
	Day 3	23.5	10.3		
	Day 4	24.7	9.9		
* Sig at P < 0.05 and No Sig P > 0 .05 level					

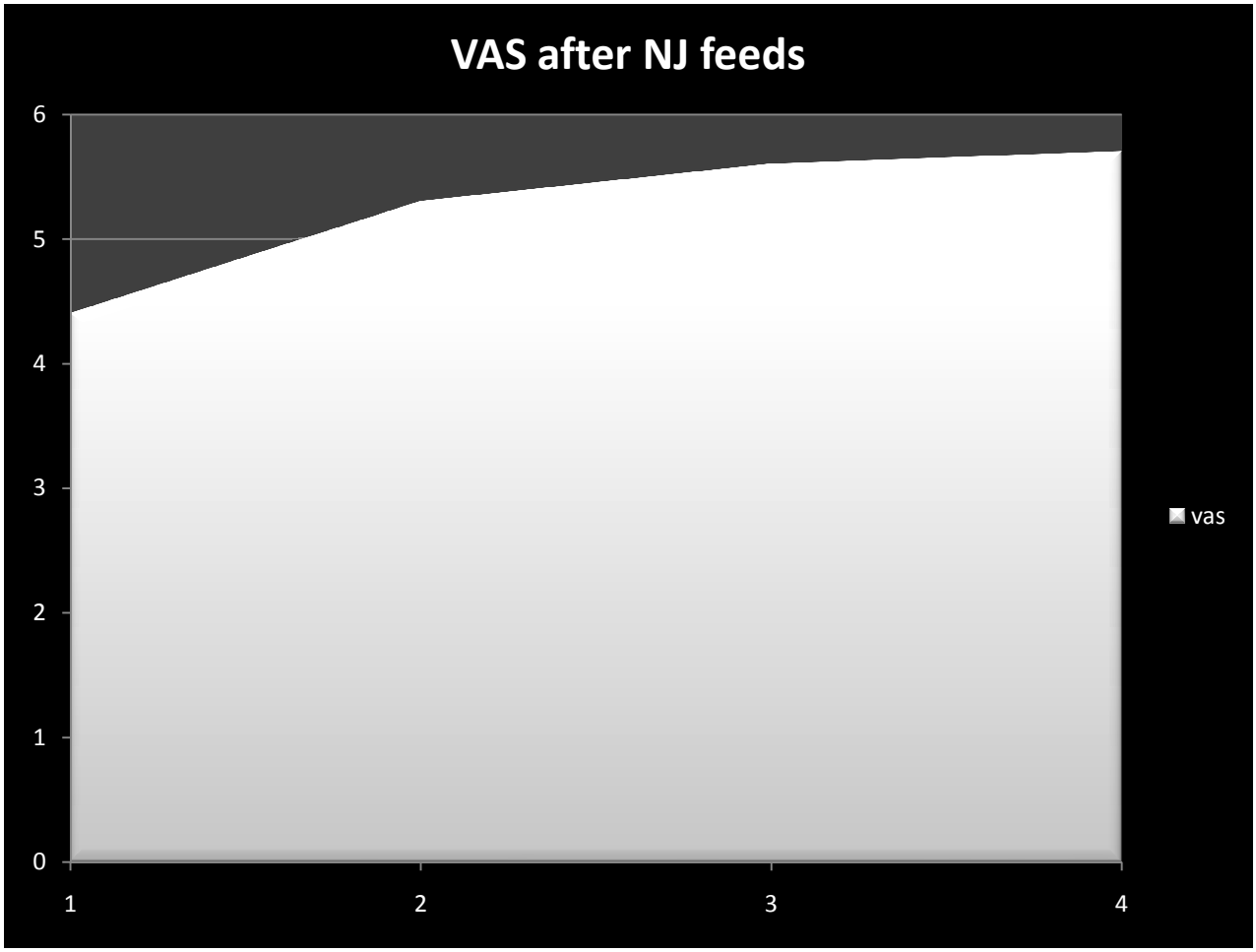


The comparison of mean Apache 2 scores using ANOVA also showed a statistically significant advantage for the patients in Group A.

VAS analysis :



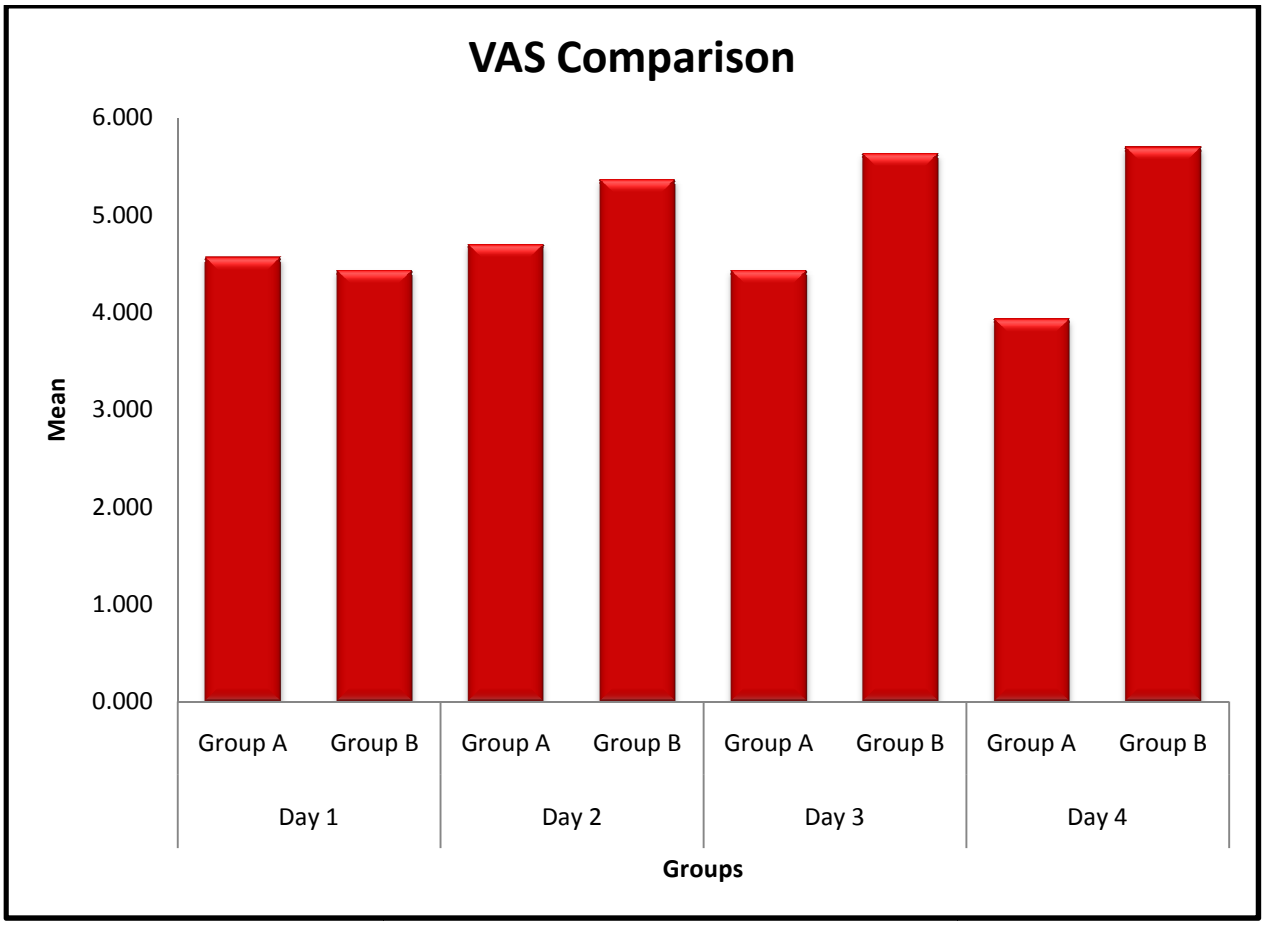
The patients who underwent enteral feeding through the naso gastric route showed a marked decrease in the visual analogue scoring.The visual analogue scores showed an overall value that remained unchanged throughout the course of nasojejunal feeding in acute pancreatitis.



VAS Comparison with Mann-Whitney U test						
Groups		N	Mean	S.D	Z-value	P-value
Day 1	Group A	30	4.567	1.6750	0.472	0.637 #
	Group B	30	4.433	1.8696		
Day 2	Group A	30	4.700	1.7050	1.086	0.277 #
	Group B	30	5.367	2.3413		
Day 3	Group A	30	4.433	1.6333	2.239	0.025 *
	Group B	30	5.633	2.0254		
Day 4	Group A	30	3.933	1.6595	3.447	0.001 **
	Group B	30	5.700	1.9146		
** Highly Sig P < 0.01 ,* Sig at P < 0.05 and No Sig P > 0 .05 level						

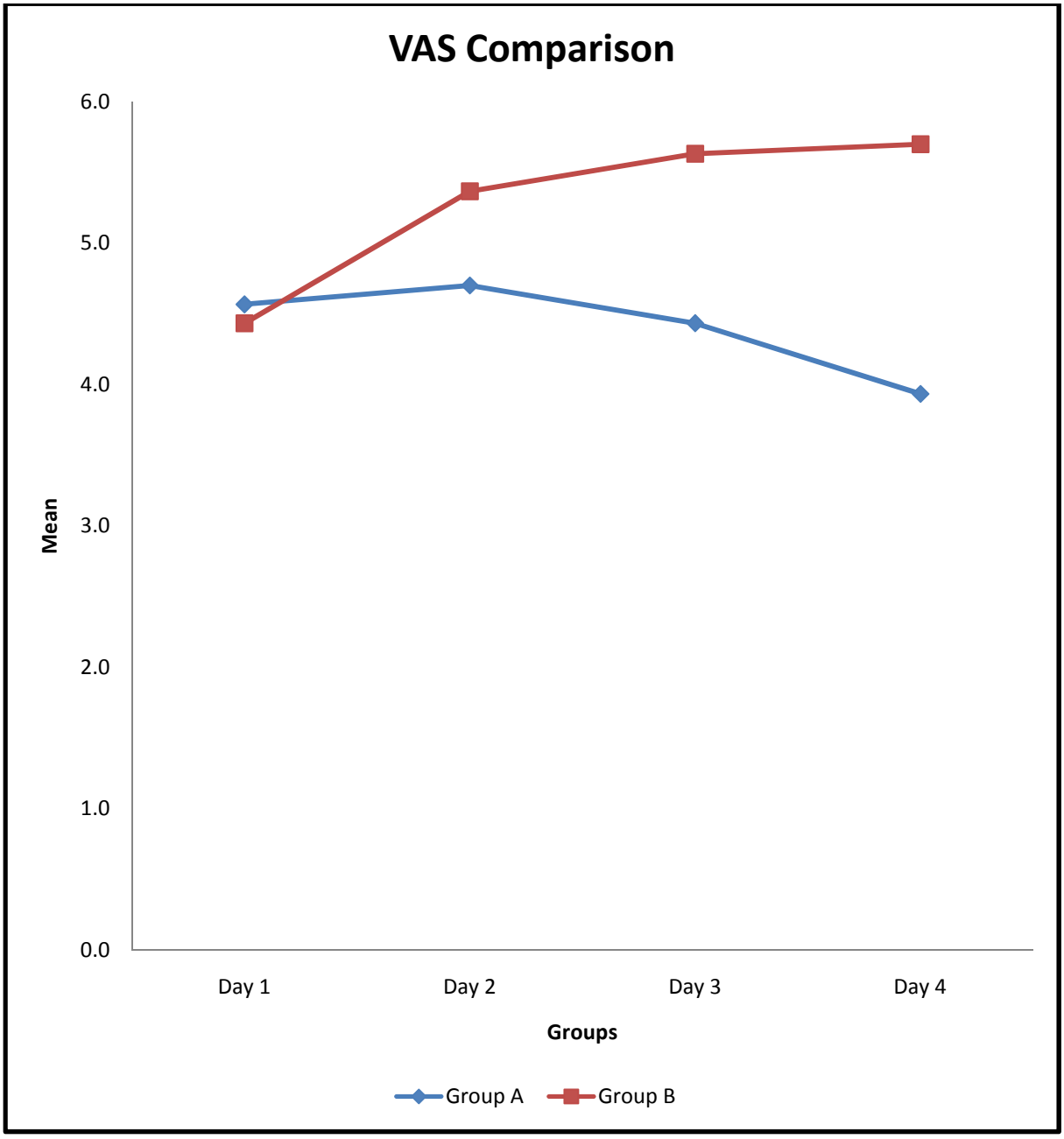
The Pain score of VAS on serial monitoring after starting early enteral feeds for both groups of patients showed that there is a gradual increase in the overall pain score among the patients who received nasogastric route of feeding and this has been found to be statistically significant.

Meanwhile, the subjects who have been received a nasojejunal route of enteral feeding had a little higher mean VAS scores when compared with Mann-Whitney U test.

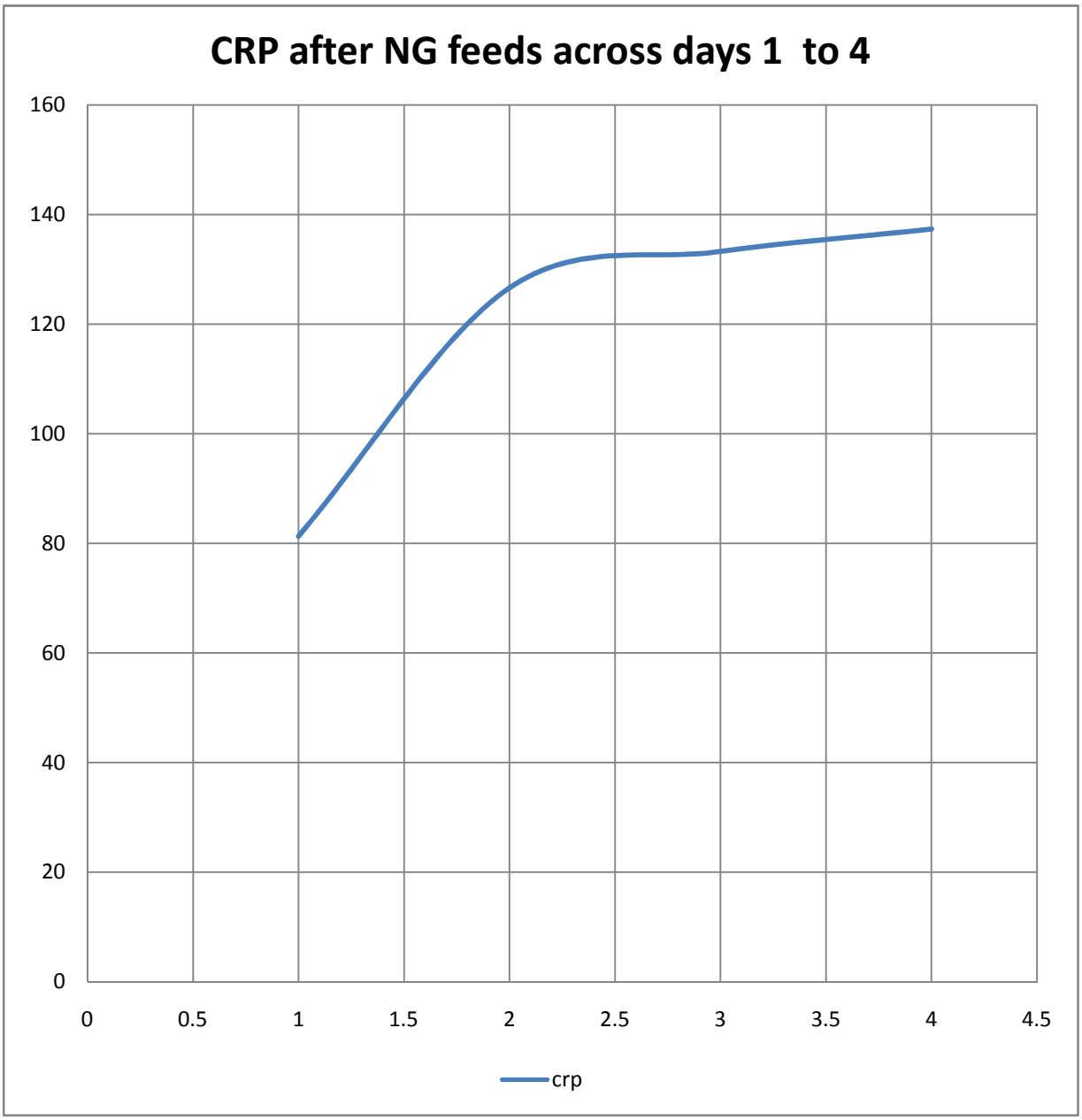


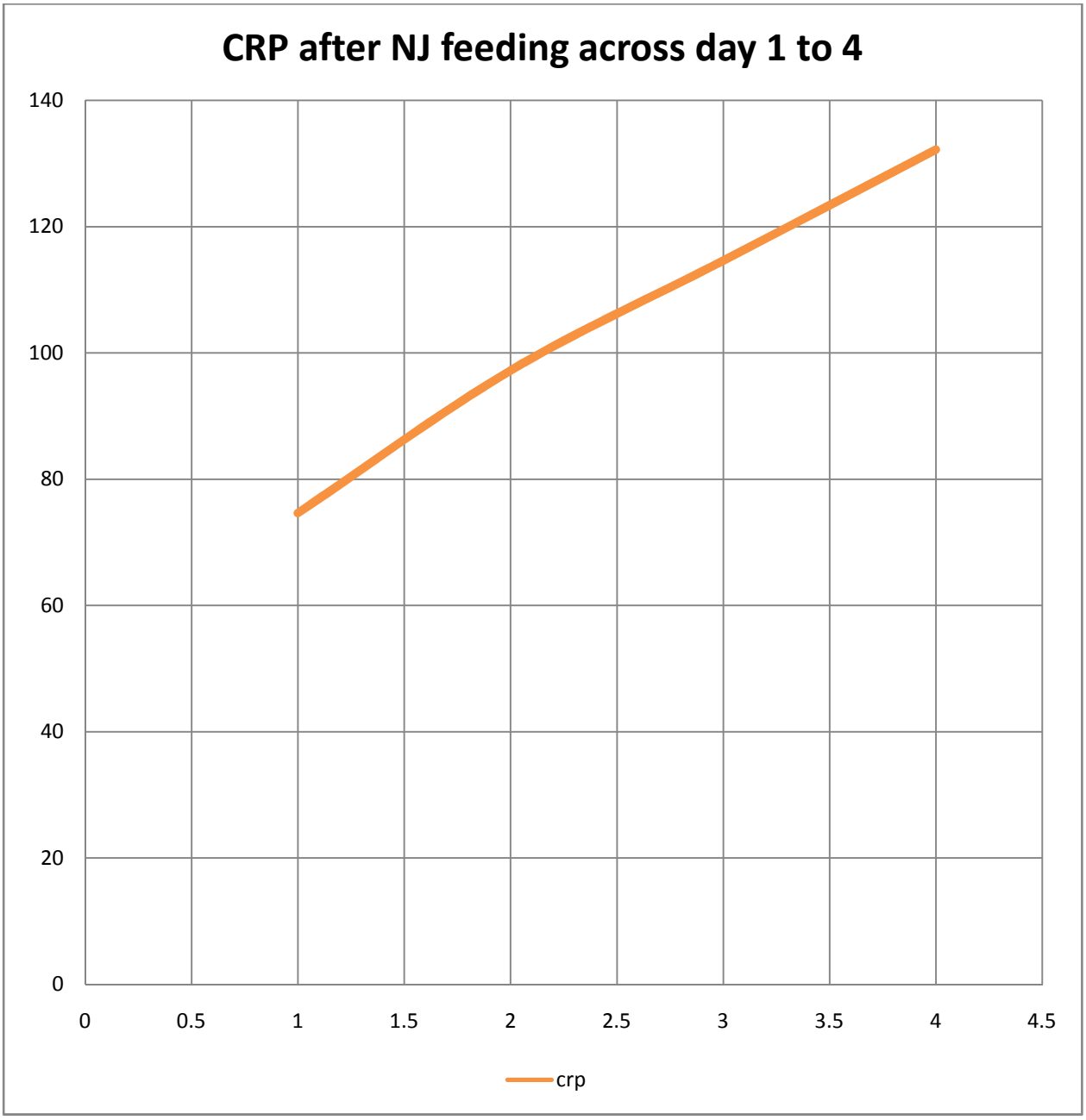
VAS comparison by Friedman test					
Groups	Days	Mean	S.D	Chi-value	P-value
Group A	Day 1	4.6	1.7	16.674	0.001 **
	Day 2	4.7	1.7		
	Day 3	4.4	1.6		
	Day 4	3.9	1.7		
Group B	Day 1	4.4	1.9	34.038	0.0005 **
	Day 2	5.4	2.3		
	Day 3	5.6	2.0		
	Day 4	5.7	1.9		
** Highly Statistical Significance P < 0 .01 level					

VAS comparison across day 1 to day 4 using Friedman test showed a highly significant chi value on favour of nasogastric method of early enteral feeding.


VAS Comparison
6.0
5.0
4.0
3.0
2.0
1.0
0.0
Mean
Day 1
Day 2
Day 3
Day 4
Groups
Group A
Group B

CRP analysis:

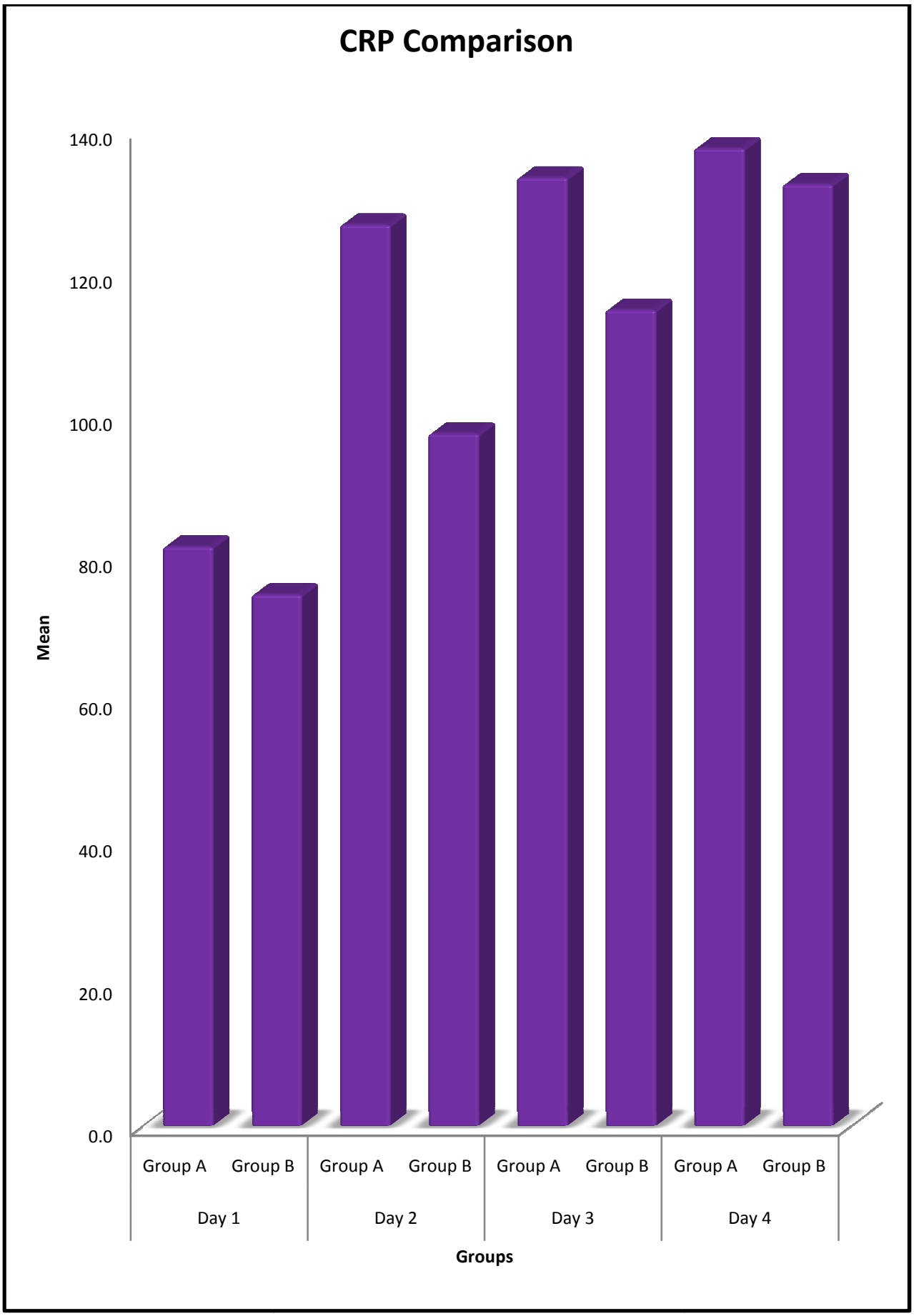




CRP levels showed not much of a difference among both the nasogastric and nasojejunal routes.

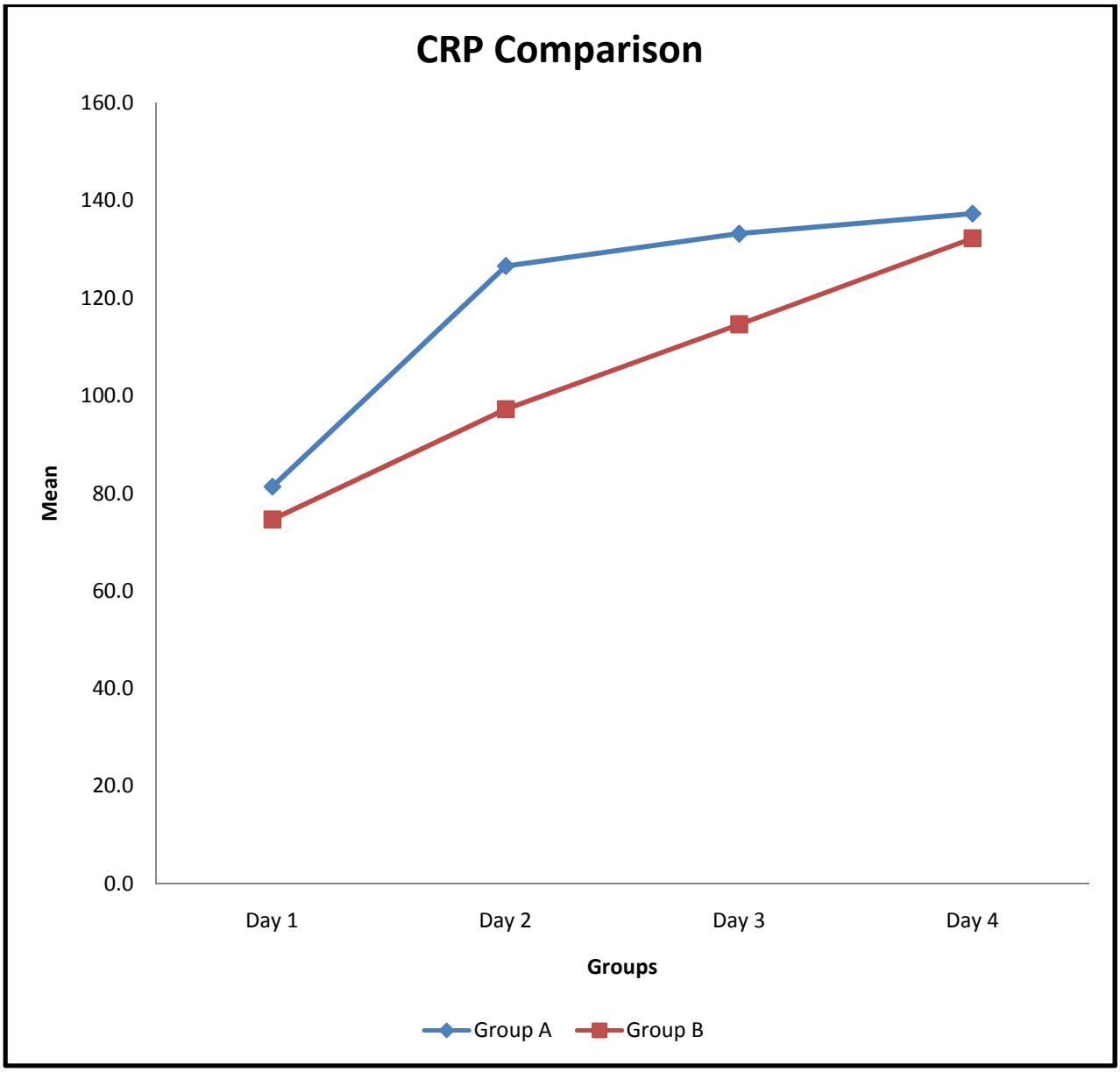
CRP Comparison with Unpaired t-test						
Groups		N	Mean	S.D	t-value	P-value
Day 1	Group A	30	81.4	50.5	0.471	0.639 #
	Group B	30	74.6	59.8		
Day 2	Group A	30	126.6	85.1	1.298	0.200 #
	Group B	30	97.2	90.1		
Day 3	Group A	30	133.2	73.6	0.888	0.378 #
	Group B	30	114.6	88.1		
Day 4	Group A	30	137.3	84.8	0.221	0.826 #
	Group B	30	132.3	92.8		
# No Statistical Significance at P>0.05 level						

There has been no statistical significance in the comparison of CRP levels in the serum on serial measurement after starting nasogastric and nasojejunal feeding as shown by all P-values being greater than 0.05 using the Unpaired t-test.


CRP Comparison
140.0
120.0
100.0
80.0
60.0
40.0
20.0
0.0
Mean
Group A
Group B
Group A
Group B
Group A
Group B
Group A
Group B
Day 1
Day 2
Day 3
Day 4
Groups

CRP comparison by Repeated measures of ANOVA					
Groups	Days	Mean	S.D	F-value	P-value
Group A	Day 1	81.4	50.5	14.622	0.0005 **
	Day 2	126.6	85.1		
	Day 3	133.2	73.6		
	Day 4	137.3	84.8		
Group B	Day 1	74.6	59.8	24.773	0.0005 **
	Day 2	97.2	90.1		
	Day 3	114.6	88.1		
	Day 4	132.3	92.8		
** Highly Statistical Significance P < 0 .01 level					

The mean CRP levels in the serum of both groups showed a gradual increase in the levels with a highly statistical significance when calculated using ANOVA.


CRP Comparison
160.0
140.0
120.0
100.0
80.0
60.0
40.0
20.0
0.0
Mean
Day 1
Day 2
Day 3
Day 4
Groups
Group A
Group B

DISCUSSION

The patients who were subjected to nasogastric mode of feeding showed a decreasing trend in the mean values of the Apache 2 scores drastically. The patients who underwent enteral feeding through the naso gastric route showed a marked decrease in the visual analogue scoring. The visual analogue scores showed an overall value that remained unchanged throughout the course of nasojejunal feeding in acute pancreatitis. CRP levels showed not much of a difference among both the nasogastric and nasojejunal routes.

Overall, both the subgroups of patients improved after being subjected to early enteral nutrition. The comparison between the nasogastric and nasojejunal routes of feeding show that, there is marked improvement in the pain levels among the subset treated with nasogastric route of feeding.

The decline in Apache 2 scores among the nasogastric feeders show that, in a critically ill patient, this mode of nutrition can actively reduce the sepsis scores when compared to all other modes of nutrition.

The levels of CRP , on the contrary, show no such change among both the groups of patients.

This concludes that the nutritive support given to the patient suffering from acute pancreatitis does not really have an impact on the severity of inflammation of the pancreas.

CONCLUSION

It has been clearly established that there is a role for early enteral feeding in patients of acute pancreatitis. In a critically ill patient who cannot be shifted out of the intensive care cubicle, there is a higher risk of increased disease progression when the patient is subjected to fluoroscopy or endoscopic intervention for adequate placement of a nasojejunal tube. And it is in such a scenario, the simple yet effective nasogastric tube comes to the rescue, bringing down the markers of sepsis and giving relief from agony for the patient. To conclude, we can advocate early enteral feeding by the use of a nasogastric tube without further deterioration of the disease condition of the patient.

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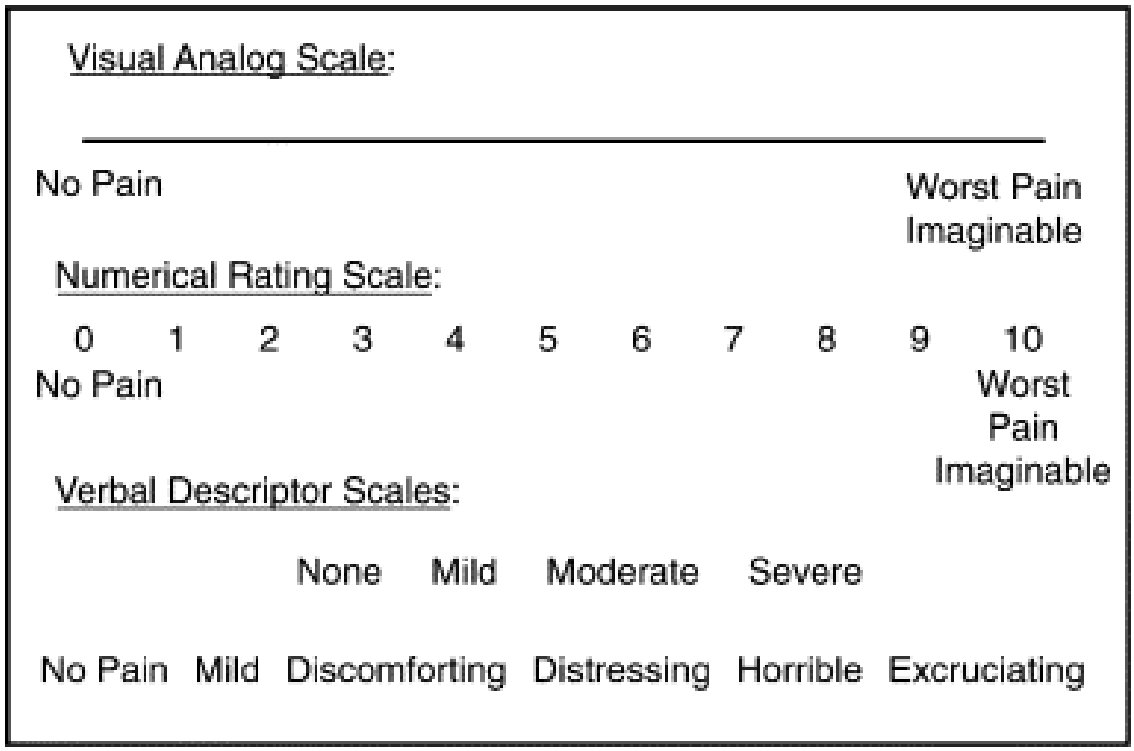
LIST OF ABBREVIATIONS

- AP Acute pancreatitis
- NJ Nasojejunal
- NG Nasogastric
- USG Ultrasonography
- CECT Contrast enhanced computerised tomography
- CRP C-Reactive Protein
- VAS Visual Analogue Score
- APACHE Acute Physiology and Chronic Health

ANNEXURE 1

VISUAL ANALOGUE SCALE FOR RECORDING PAIN SCALES



ANNEXURE II

INFORMATION SHEET

TITLE : "A STUDY ON EARLY ENTERAL FEEDING IN ACUTE PANCREATITIS - NASOGASTRIC VS NASOJEJUNAL FEEDING"

Name of Investigator : Name of Participant :

Purpose of Research :

1.to assess any difference between NG and NJ routes, in tolerance, acute phase response, and pain.

2. To study the clinical course of patients managed conservatively

Study Design : Randomised control Study

Study Procedures : Patient will be subjected to routine investigations, and application of either nasogastric or nasojejunal tube

Possible Risks : No risks to the patient

Possible benefits

To patient : A better understanding of their problem so has to devise a plan of management which suits their needs.

To doctor & to other people : If this study gives positive results, it can help determine the early identification, most effective diagnostic and treatment protocol for patients with acute pancreatitis. This will help in providing better and complete treatment to other patients in future.

Confidentiality of the information obtained from you : The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared

Can you decide to stop participating in the study : Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time

How will your decision to not participate in the study affect you : Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

Date :

Place :

ANNEXURE III

PATIENT CONSENT FORM

Study Detail : **"A STUDY ON EARLY ENTERAL FEEDING IN ACUTE PANCREATITIS - NASOGASTRIC VS NASOJEJUNAL FEEDING"**

Patient may check (☑) these circles

- I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.
- I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.
- I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

- I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.

- I hereby consent to participate in this study

- I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment

Signature/thumb impression	Signature of Investigator
Patient's Name and Address:	Study Investigator's Name:

QUESTIONAIRE

PATIENT DETAILS:

Name: Age: Sex:

IP No. :

ON ADMISSION:

MAIN COMPLAINTS:

ASSOCIATED COMPLAINTS :

CLINICAL EXAMINATION:

Pulse : BP :

RR : Temp :

Pallor : Icterus :

CVS : RS :

P/A : CNS:

INVESTIGATIONS :

CBC/RFT				
TC				
DC				
Hb %				
PCV				
RBC				
Platelets				
Glucose				
Amylase				
Lipase				
Na^+/K^+				

LFT				
Total Bili				
Dir. Bili				
SGOT				
SGPT				
Total Protein				
Sr. Albumin				

CXR :

Abdomen Xray :

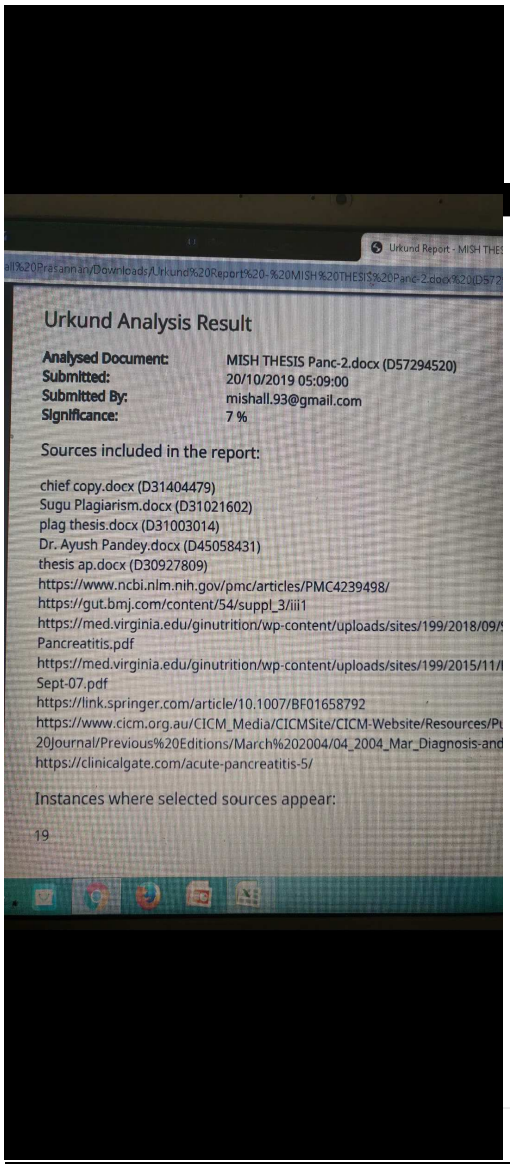
USG Abdomen :

TREATMENT

CONSERVATIVE MANAGEMENT WITH DAILY FOLLOW UP

FOLLOW UP :

PLAGIARISM ANALYSIS



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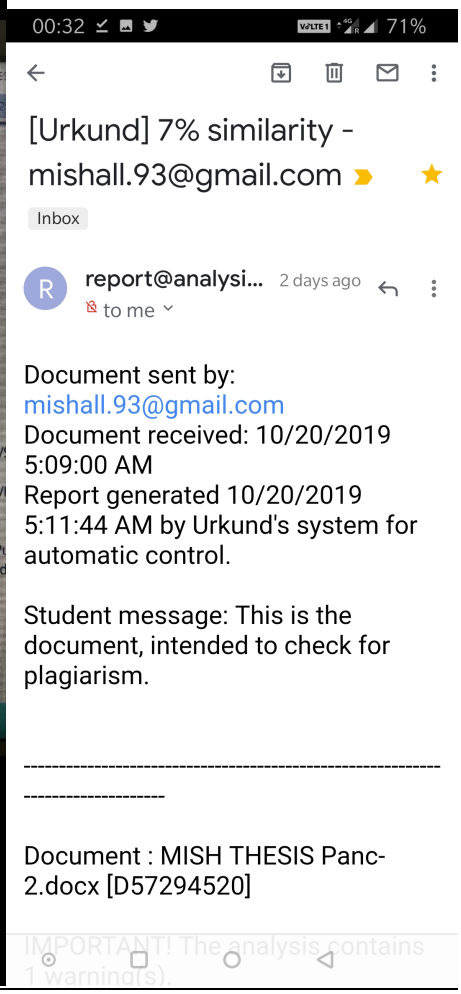

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CERTIFICATE

This is to certify that this dissertation work titled " **A STUDY ON EARLY ENTERAL FEEDING IN ACUTE PANCREATITIS - NASOGASTRIC VS NASOJEJUNAL FEEDING**" of the candidate **Dr.MISHALL PRASANNAN** with registration nnumber **221711010** for the award of M.S. degree in the branch of General Surgery. I personally verified the urukund.com website for the purpose of plagiarism check. I found that the uploaded thesis contains from introduction to conclusion pages and result shows **7% of plagiarism** in the dissertation.

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MASTER CHART

NAME	AGE	SEX	DAY-1			DAY-2			DAY-3			DAY-4		
			APACHE 2	**CRP**	**VAS**	**APACHE-2**	**CRP**	**VAS**	**APACHE-2**	**CRP**	**VAS**	**APACHE-2**	**CRP**	**VAS**
1	19	M	21	50	6	32	200	6	32	230	6	35	180	4
2	44	M	23	20	5	23	29	4	26	43	4	27	48	4
3	23	M	23	80	4	25	125	3	26	140	2	26	138	3
4	54	M	25	68	4	28	97	3	29	145	4	29	120	4
5	32	M	34	146	7	35	298	8	37	184	6	32	180	6
6	43	M	23	36	3	24	87	3	25	62	3	23	61	2
7	54	M	37	72	6	32	86	7	30	129	5	29	147	5
8	43	M	34	140	5	34	187	5	32	150	4	29	110	4
9	54	M	25	98	5	27	112	6	27	132	4	28	156	3
10	44	M	28	66	6	27	62	6	25	68	5	23	50	5
11	51	M	45	204	7	43	345	8	38	220	8	37	210	6
12	36	F	44	116	6	43	188	5	37	245	4	35	310	3
13	45	M	31	28	3	28	24	2	26	16	2	22	22	1
14	42	M	25	45	4	24	68	3	22	92	3	22	97	2
15	55	M	27	67	3	24	54	3	25	69	3	25	87	2
16	39	M	34	87	5	36	140	4	35	165	4	32	154	4
17	49	F	42	109	6	37	222	6	34	269	7	32	374	5
18	61	M	46	172	7	41	268	6	37	287	7	33	313	4
19	37	M	22	84	5	26	204	5	26	189	4	25	195	4
20	43	M	16	31	2	15	63	2	16	78	1	17	98	1
21	32	M	23	56	4	25	68	5	26	123	5	26	149	4
22	50	M	19	52	2	17	116	4	16	152	4	15	143	2
23	35	M	29	89	5	26	86	6	23	82	5	24	43	4
24	54	M	26	47	3	24	81	3	24	84	4	25	80	4
25	28	M	25	74	5	22	54	3	25	44	3	26	13	2

26	43	M	34	186	7	32	220	7	31	245	7	31	210	8
27	46	F	41	140	6	32	175	6	28	142	6	29	113	4
28	51	M	22	15	2	22	23	3	21	64	3	11	110	6
29	31	M	24	32	2	25	63	5	26	59	5	28	82	6
30	46	M	32	31	2	31	53	4	31	89	5	31	127	6
31	50	M	32	46	3	31	48	4	28	84	6	26	56	5
32	35	M	43	208	8	44	229	8	44	275	8	44	306	9
33	46	M	12	5	2	11	4	13	13	2	3	11	2	3
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35	35	M	33	48	3	32	65	5	28	87	5	28	177	6
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38	44	M	35	96	5	44	210	7	65	204	8	71	268	8
39	58	F	23	38	4	25	38	4	24	61	5	26	79	5
40	29	M	23	63	6	24	64	6	25	122	7	26	149	7
41	32	M	34	110	6	33	120	7	36	187	7	36	160	7
42	55	M	27	93	6	25	136	7	26	154	8	26	89	7
43	43	M	11	4	2	11	6	3	10	5	2	8	4	2
44	41	M	33	158	7	32	198	8	32	210	8	31	203	8
45	29	F	17	39	3	18	34	2	22	58	3	22	68	4
46	46	M	21	64	4	23	87	5	24	94	6	24	135	6
47	47	M	19	49	4	15	46	7	15	68	8	16	93	7
48	38	M	42	245	8	41	378	7	44	302	8	42	311	8
49	41	M	11	36	5	16	65	6	17	89	8	28	93	7
50	44	M	42	174	8	45	222	7	46	284	7	47	296	8
51	52	F	33	165	7	37	287	8	35	298	8	36	320	8
52	39	M	8	9	2	6	23	3	6	65	4	4	98	3
53	40	M	22	64	3	24	64	4	25	63	5	26	87	5
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57	27	M	13	42	3	15	32	3	15	24	3	15	10	3
58	38	F	22	34	3	25	65	2	26	84	3	26	89	5
59	55	M	20	69	5	19	89	3	19	115	4	19	165	5
60	54	M	17	64	3	16	64	4	18	98	5	18	87	5

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Mishall Prasannan
I Year Post Graduate in MS General Surgery
Institute of General Surgery
MMC/Chennai

Dear Dr.Mishall Prasannan,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON EARLY ENTERAL FEEDING IN ACUTE PANCREATITIS – NASOGASTRIC VS NASOJEJUNAL FEEDING " - NO.40122017**

The following members of Ethics Committee were present in the meeting hold on **12.12.2017** conducted at Madras Medical College, Chennai 3

1. Prof.P.V.Jayashankar :Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., Dean,MMC,Ch-3 : Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3 : Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch : Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3 : Member
6. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC : Member
7. Prof.Shanthy Gunasingh, Director, Inst.of Social Obstetrics,KGH : Member
8. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
9. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3 : Member
10. Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3 : Member
11. Prof.Bharathi Vidya Jayanthi,Director, Inst. of Pathology,MMC,Ch-3 : Member
12. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
13. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
14. Thiru K.Ranjith, Ch-91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee