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RESEARCH ARTICLE

CT EVALUATION OF ACUTE PANCREATITIS AND ITS COMPLICATIONS USING MODIFIED CTSEVERITY INDEX

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Abstract

Objectives 1: To grade the CT findings of patients with acute pancreatitis according to MCTSI 2. To correlate the grading systems with patient outcome in terms of a. Organ failure b. Mortality c. Duration of hospital stay 3. To determine sensitivity, specificity and positive predictive value of MCTSI in predicting the above mentioned complications.

Materials and Methods: This study was conducted in the Department of Radio-diagnosis, Mysore medical college and research institute, Mysore from November 2016 to April 2018. The study comprised of a total of 50 patients. Of 50 patients diagnosed with acute pancreatitis who underwent contrast- enhanced MDCT within 3 days of the onset of symptoms during the study period were included in the study. The severity of the pancreatitis was scored using modified CT severity indexes. Patient clinical outcome was scored using parameters such as: mean duration of hospital stay, the need for surgical intervention, occurrences of infection, end organ failure and death. For the modified CT severity indexes, the correlation between the severity of pancreatitis and patient outcome was estimated using the percentage, frequency charts, and chi-square test.

Results: This was a prospective study of 50 cases of clinically diagnosed acute pancreatitis and confirmed by serum amylase and lipase levels. Modified CT severity index grading was done after contrast enhanced CT of abdomen and pelvis in all patients. Correlation of modified CT severity index grades was done with patient outcome taking local and systemic complications, duration of hospital stay. The age group of patients was 15 to 66 years with maximum patients (36%) between 26 and 30 years. 86% of patients were male. Male to female ratio was 8.6: 1.4 with male preponderance. Chronic alcohol abuse was the most common cause of pancreatitis (76%), second was gallstones (16%) and others (8%) All patients presented with pain abdomen. 92% had vomiting, 30% patients had fever and 10% of patients had jaundice at the time of presentation. Amylase was elevated in 86% patients. Lipase was elevated in 90% patients. 86% patients had features of pancreatitis on ultrasound and in 14% ultrasound was normal. Pancreatic inflammation was seen in 100% of patients. 30% patients had no evidence of pancreatic necrosis on CT scan. 54% of patients had less than 30% necrosis and only 16% had more than 30% necrosis. 48% patients had no evidence of extrapancreaticComplications. 52%

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patients had one or more extra pancreatic complications. According to Modified CT Severity Index, 6% patients had mild, 70% patients had moderate and 24% had severe pancreatitis. Duration of hospital stay ranged from 3 to 25 days with mean duration of 9.5 days. Mortality rate was 0%. 38% patients are considered to have end organ failure. Hepatic failure is the most common system failure seen in 22% patients. 36% patients had evidence of systemic infection. 10% patients required surgical interventions.

Conclusion: There was highly significant correlation between the MCTSI score and the prediction of end organ failure, systemic infection and duration of hospital stay than CTSI score. MCTSI is a very useful tool for the screening of patients with acute pancreatitis for the classification of severity accurately and to predict the clinical outcome when used before three days of symptom onset. Key words: Computed Tomography; Modified CT Severity Index; Acute pancreatitis; pancreatic necrosis; Patient outcome.

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Introduction:-

Acute pancreatitis is one of the most complex and clinically challenging conditions of all abdominal disorders. Early assessment of the cause and severity of acute pancreatitis is of utmost importance for prompt treatment and close monitoring of patient with a severe disease. Imaging plays an important role in the management of the patient with acute pancreatitis. CT, in particular, has revolutionized pancreatic imaging, and what was once considered a hidden organ may now be accurately and noninvasively imaged.[1,2]

Between 10% and 20% of cases of acute pancreatitis are considered severe[3].Treatment of patients with acute pancreatitis based on the initial assessment of disease severity. Severe pancreatitis is characterized by a protracted clinical course, multiorgan failure, and pancreatic necrosis2.Individual laboratory indexes (markers of pancreatic .injury, markers of inflammatory response), while promising, have not yet gained clinical acceptance. Numeric grading systems like RANSON and APACHE II are commonly used today as indicators of disease severity. While RANSON score cannot be used for the first 48 hrs, APACHE score is cumbersome to use.4,5

Contrast-enhanced computed tomography was later used to evaluate the prognosis of patients with acute pancreatitis. CT severity index was used initially which was popularly called Balthazar scoring system. This scoring system is based on pancreatic morphology, the number of peripancreatic fluid collections and pancreatic necrosis.

Now Modified Computed Tomography Severity Index (MCTSI) has been introduced which differs from the Computed Tomography Severity Index (CTSI) by including the presence of extrapancreatic complications and grading the peripancreatic fluid collection in terms of presence or absence instead of the number of fluid collections.[5,6]

Objectives:-

1. To determine the value of computed tomography evaluation in the early diagnosis of acutepancreatitis.
2. To evaluate the complications using modified computed tomography severity index.
3. To grade the CT findings of the patient with acute pancreatitis according to modified CT severity index.
4. To correlate the grading systems with patient outcome in terms of
   a. Organ failure
   b. Mortality
   c. Duration of hospital stay
5. To determine sensitivity, specificity and positive predictive value of modified CT severity index in predicting the above-mentioned complications.

Review of Literature:-

Historical background[7,8,9]:

The pancreas is truly a noble organ. Known to the ancient Greeks, its name in Greek "pan kreas" translates as "all flesh." Aristotle believed its function was to protect.The pancreas was apparently first discovered by Herophilus, a Greek anatomist and surgeon. It was in the 18th century that the main duct of Wirsung was described as well as its first cannulations to perform studies on pancreatic secretion.

Thedigestiveactionofpancreaticsecretionswasdiscoveredalmost200years afterthediscoveryofpancreas,laterEberlein1834,PurkinjeandPappenheimin1836 andValentinin1844observedthatthe emulsification offat,proteolyticactivity,anddigestion ofstarchrespectivelybypancreaticjuiceandextracts.ItwasBernard who subsequentlydemonstratedthedigestiveactionofpancreaticjuiceonsugar,fats,and proteins,usingsecretionsfrompancreaticfistulapreparations.Thehistologicstructureofthepancreas wasfirstdescribedby Langerhansin1869 thereafterbyHeidenhain.

Kuhneintroducedthetermenzymeandisolatedtrypsinin1876whichledtothe identificationofpancreaticamylaseandlipase.ItwasChepovalnikoffin1889,astudentof Pavlovwhodiscoveredenterokinaseintheduodenalmucosawhichisessentialfor activationoftheproteolyticenzymes.In 1895,Dolinskystimulatedpancreaticsecretion byinstillingacidintotheduodenum;thisledtothediscoveryofsecretinbyBaylissandStarling,whichprovedtobenotanenzym ebutthefirsthormonetobeidentified.

EmbryologyofPancreas[10,11]:

Embryologically, thepancreasisderivedfromtheendodermoftheembryonicforegut and arises from a dorsal and ventral pancreatic bud. The larger dorsal bud is the precursor of the anterior portion of the head as well as body and the tail, while the smaller ventral bud develops into the posterior head and uncinate process. The dorsal and ventral ducts fuse into onemajor duct, the duct of Wirsung, which empties into the duodenum along with the CBD at the ampulla of Vater.

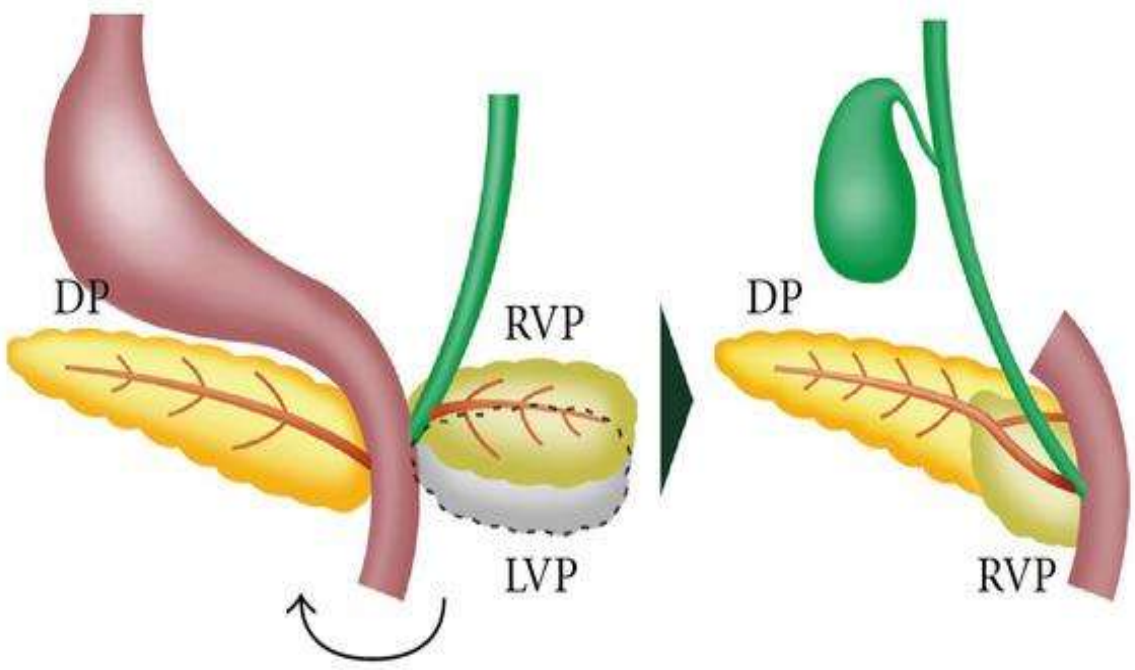


Figure 1:- Normal development of the pancreas. The ventral pancreatic anlage is initially paired, with the left lobe subsequently disappearing during development. The ventral pancreatic anlage fuses side by side with the dorsal anlage.(RVP, right ventral pancreatic anlage; LVP, left ventral pancreatic anlage; DP, dorsal pancreatic anlage

Variations in the pancreatic ductal branching pattern are common (Figure 2) and can predispose to pancreatitis.

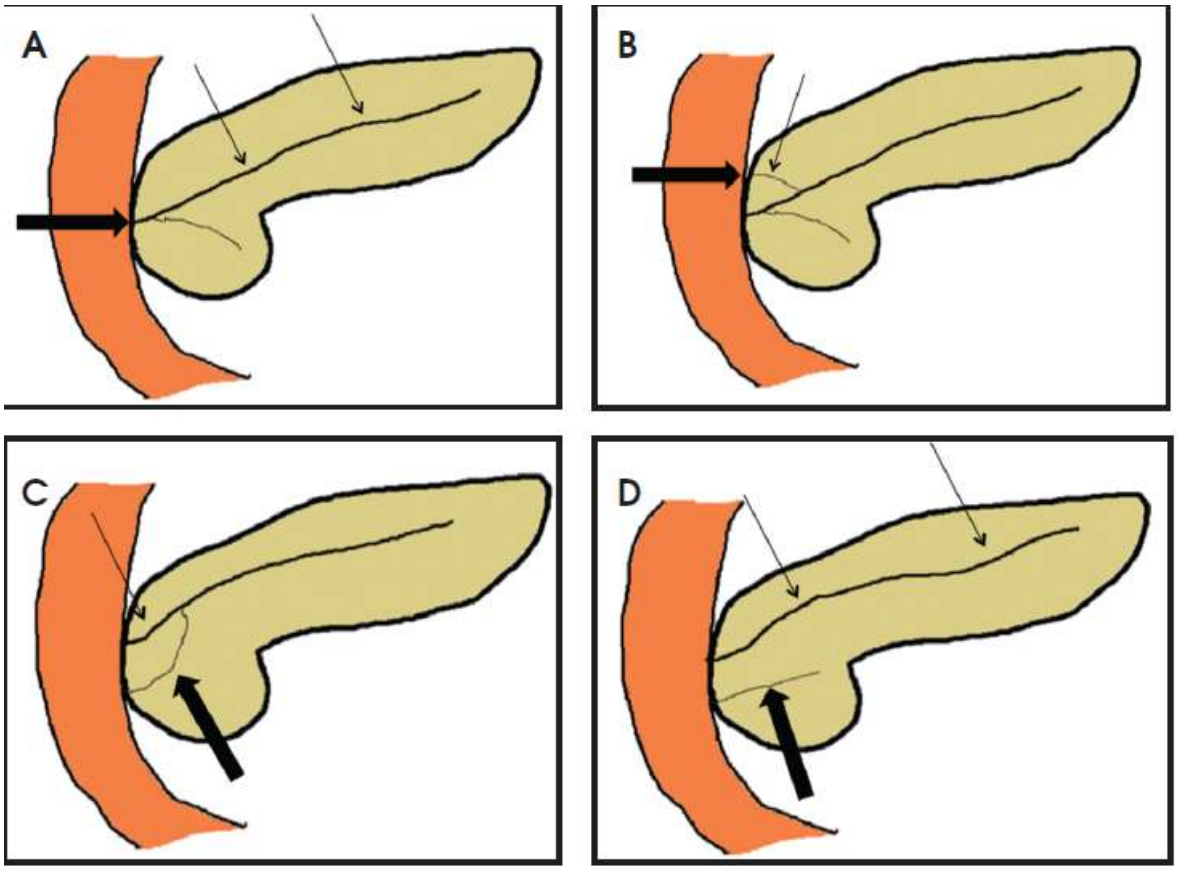


Figure 2:- Colored schematic diagram showing normal and variant pancreatic ductal anatomy. (A) Normal pancreatic ductal anatomy with the main pancreatic duct of Wirsung (thin arrows) emptying into the major duodenal papilla at the ampulla of Vater (thick arrow). (B) Normal variant where an accessory pancreatic duct (thin arrow) empties into the minor papilla (thick arrow). (C) Dorsal-dominant drainage where the main pancreatic duct (thin arrow) empties into the minor papilla and the accessory pancreatic duct (thick arrow) empties into the major papilla. (D) Pancreas divisum with complete separation of the major (thin arrows) and minor pancreatic ducts (thick arrow).

Anatomy Of Pancreas[12,13]:

The pancreas is a soft, elongated, flattened gland measures 12 to 20 cm in length. It is a retroperitoneal organ located in the anterior pararenal space posterior to the stomach and bounded by the c-loop of duodenum on the right side.Composedof the following parts:

1. The headofthepancreas lies within theconcavityoftheduodenum.
2. Theuncinateprocessemergesfromthelowerpartoftheheadandliesdeeptosuperiormesentericvessels.
3. Theneckofthepancreasistheconstrictedpartbetweentheheadandthebody.
4. Thebodyliesbehindthestomach.
5. Thetailistheleftendofthepancreas.Itliesincontactwiththespleenandrunsinthelienorenalligament.

The average normal measurements of the pancreas in CT is as follows: head – 23 +/- 3mm, neck – 19 +/- 2.5mm, body – 20 +/- 3mm and tail – 15 +/- 2.5mm. The AP dimension of pancreas must be smaller than the AP diameter of the adjacent vertebra on CT.

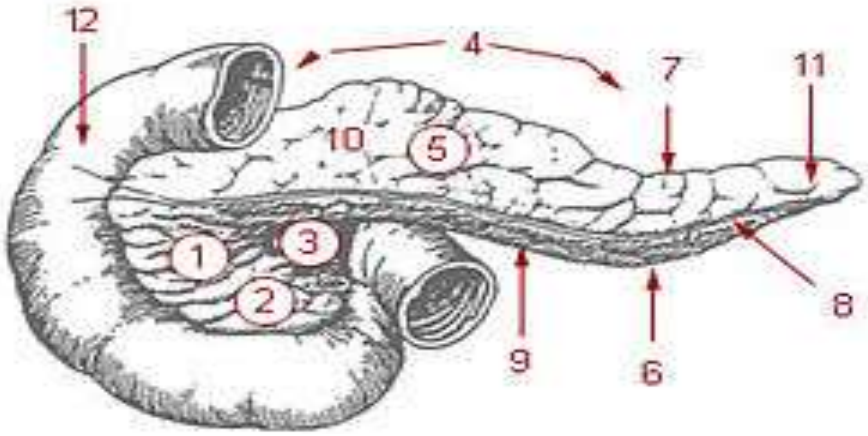


Figure 3:- Anatomy of pancreas.

Head of pancreas 2:
Uncinate process of pancreas 3: Pancreatic notch 4: Body of pancreas 5: Anterior surface of pancreas 6: Inferior surface of pancreas 7: Superior margin of pancreas 8: Anterior margin of pancreas 9: Inferior margin of pancreas 10: Omental tuber 11: Tail of pancreas 12: Duodenum

Bloodsupply of Pancreas:
Arterial supply:
1. Thesuperiorpancreaticoduodenalarteryabranchofthegastroduodenalartery
2. Theinferiorpancreaticoduodenalarteryfromsuperiormesentericartery
3. Thepancreaticbranchesofsplenicarterythelargestofthosebranchesiscalledthearteriapancreaticamagna.

Venous drainage:
Thebodyandneckofthepancreasdrainintosplenicvein;theheaddrainsintothesuperiormesentericandportalveins.

Lymphaticdrainage:
Lymphisdrainedviathesplenic,celiacandsuperiormesentericlymphnodes.

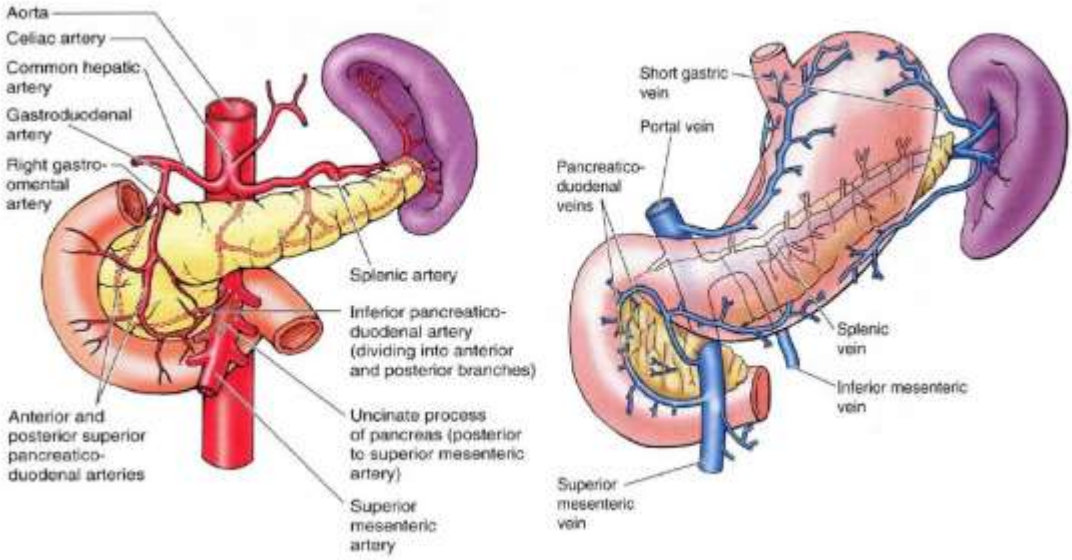


Figure 4:- Pancreaticbloodsupply.

Pancreaticduct:

Themainpancreaticduct ofWirsungbeginsnearthetailofthepancreas.Itisformedfromanastomosesofductulesdrainingthelobulesofthegland.Itpassesfromlefttorightadditionalductsjoinit.Inthetailandbody; theductliesmidwayandslightlyposteriorbetweenthesuperiorandinferiormargins.Atthelevelofthemajorpapilla,the ductturnshorizontallytojoinusuallywiththecommonbileduct.Thisshortcommonsegmentistheampullaofthebileduct,which terminatesintheduodenalpapillaandisguardedbythesphincterofOddi.(Fig-5,Fig-6)

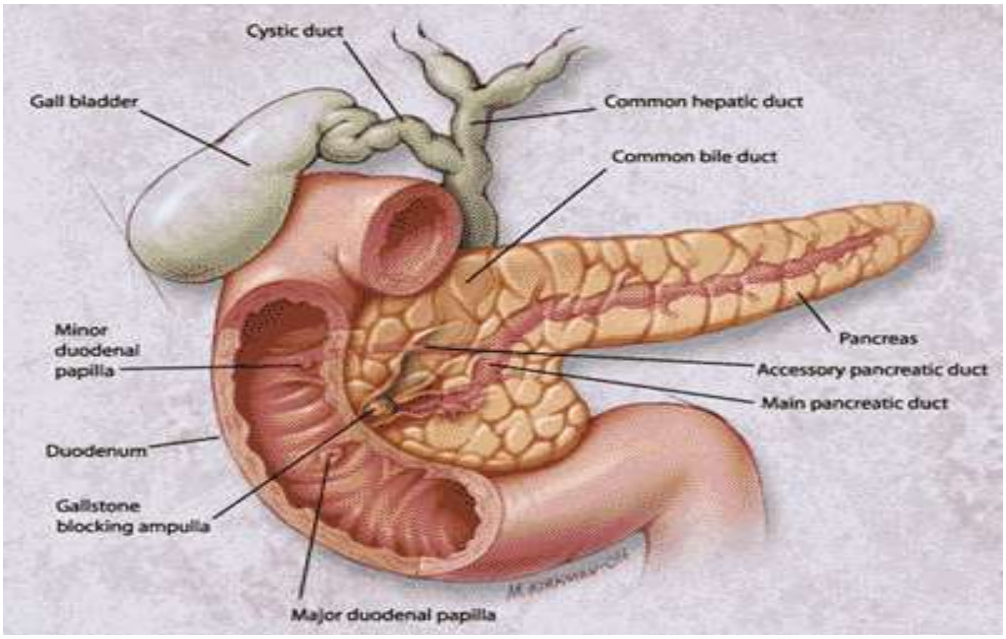


Figure 5:- The pancreaticduct system.

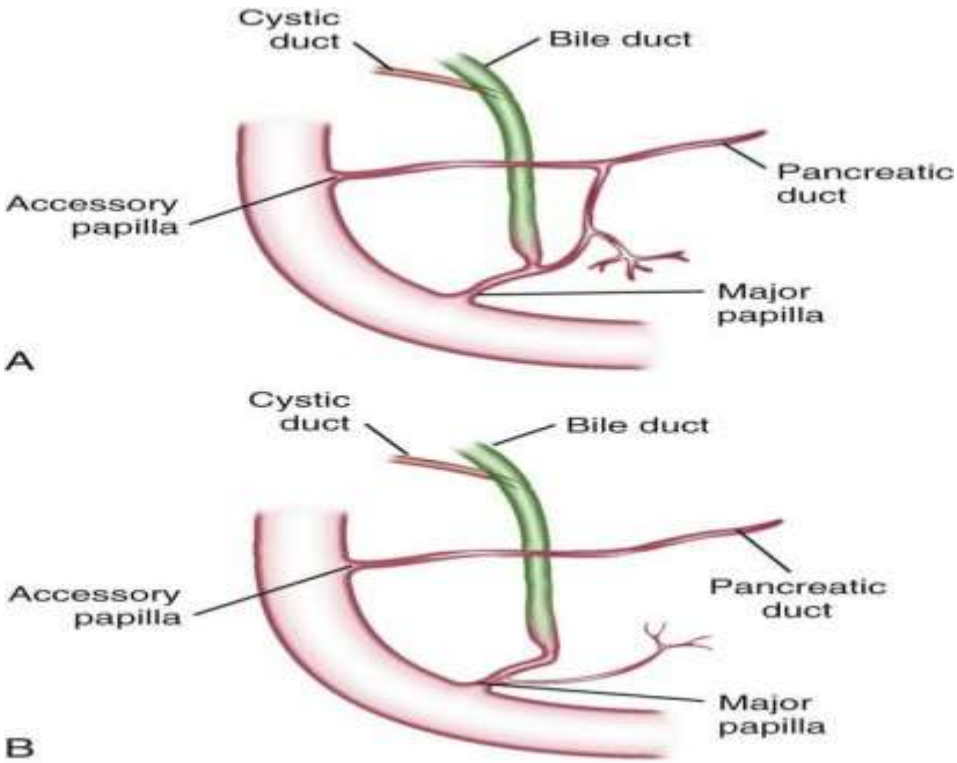


Figure6:- The pancreaticduct system.

Thefunctionsofpancreas[14]:
Thepancreasisadual-functiongland,havingfeaturesofbothendocrineandexocrineglands.Thefunctionalunitoftheexocrinepancreasiscomposedofanacinusandits drainingductule

Thepartofthepancreaswithendocrinefunctionismadeupofapproximatelyamillioncellclusterscalledislets of Langerhans.

Thereareseveraldifferenttypesofcellsthatcompriseeofsfol,lowinghcellse:
1. Alpha:Produceglucagon,whichraisesthelevelofbloodglucosebetweenmeals, byconvertingfatandproteinintointermediatemetabolites,whicheventuallyareconvertedtoglucose.
2. Beta:Produceinsulinandamylin,whichlowerthelevelofbloodglucosebyinhibitingthesecretionofglucagon;slowsthe emptyingofthestomach.
3. Delta:Producesomatostatin,whichinhibitsthereleaseofspecifichormonesandreduces therateofabsorptionoffoodfromthecontentsofthesmallintestine
4. Gamma:Produceapolypeptide,whichreducestheappetite
5. ➢Pancreatic Polypeptide:itis a36aminoacidwhichactsascholecystokinin antagonist. Itsuppressespancreaticsecretionandstimulatesgastricsecretion.
6. Thepancreasissusceptibletoavarietyofdiseaseswhichinclude[20,21]
7. Pancreatitis.Whichistheinflammationofthepancreas;Pancreatitiscanbeeitheracuteorchronic.
8. PancreaticCancerIs thefifthleadingcauseofcancerdeathsworldwideandhasahighmortalityrateofasatthetimeofdiagnosismostofthetimet hemalignancywouldbespreadaggressively.
9. TypeIDiabetes: Occurs duetodysfunctionoftheendocrinepartofthepancreas.

Acutepancreatitis:
Definition:
According to the 1992 Atlanta Symposium [15], acute pancreatitis was defined as an acute inflammatory process of the pancreas that may also involve peripancreatic tissues and/or remote organ systems.

Historicalaspectsof acutepancreatitis[16,17]:
1889-
Fitzclassifiedtheseverityofacutepancreatitisusingevidenceofpancreatichemorrhageanddisseminatedfatnecrosisasmorphologicalfeaturesofthe severe disease.
1974- Ransonlaidclinicalcriteriatoassesstheseverityofacutepancreatitis.
1983-
Theattenuationvaluesofpancreaticparenchymaduringanintravenousbolusstudyisusedasanindicatorofpancreaticnecrosisandasapredictorofdiseaseseverity.
1985- Balthazargradingsystem.
1990- IntroductionofCTSeverityIndex.
1992–InAtlanta,InternationalSymposiumonAcutePancreatitishasclassifiedthis entityintomildacutepancreatitisandsevereacutepancreatitis.
2004- IntroductionofModifiedCTSeverityIndex.

AcutePancreatitisisoneofthemostcommondiseasesaffectingtheexocrinepancreas.Theincidenceofacutepancreatitisisincreasingasaresultof the populationisbecomingincreasinglyoverweightwhichpredisposestocholecystolithiasis.[18]Theoverallmortalityratefrom acutepancreatitishasdeclinedonlygraduallytoapproximately5%to10%[19].

Acutepancreatitisisbestdefinedclinicallybyapatientpresentingwithtwoofthe followingcriteria[22]:
1. Symptoms,suchasepigastricpain,consistentwiththedisease;
2. ASerumamylaseorlipasegreaterthanthreetimestheupperlimitofnormal;
3. Radiologic imagingconsistentwiththediagnosis,usuallyusingultrasonography , computed tomography (CT) ormagneticresonanceimaging(MRI).

Pancreatitis isclassifiedasacuteunlessthereisCT, MRI, orendoscopicretrogradecholangiopancreatography(ERCP)findings ofchronicpancreatitiswhenpancreatitisisclassifiedaschronicpancreatitis,andanyepisodeofacutepancreatitisis consideredanexacerbationofinflammationsuperimposedonchronicpancreatitis.

Etiology of pancreatitis[14]:

Table 1:- Etiology of pancreatitis.

Common causes	Uncommon causes	Causes for recurrent pancreatitis
• Gallstones • Alcohol • Hypertriglyceridemia • ERCP • Trauma • Post-operative • Sphincter of Oddi dysfunction	• Vascular causes and vasculitis • Connective tissue disorders • Carcinoma pancreas • Hypercalcemia • Periampullary diverticulum • Pancreas divisum • Hereditary pancreatitis • Cystic fibrosis • Infections (mumps, CMV etc) • Autoimmune	• Alcohol • Gall stones • Drugs • Hypertriglyceridemia • Pancreatic divisum • Pancreatic carcinoma

Table 2:- Drugs causing pancreatitis[24]:

• Alpha-methyldopa • 5-Aminosalicylate • (mesalamine) • Azathioprine • Cimetidine • Cytosinearabinoside • Dexamethasone • Trimethoprim/sulfamethoxazole	• Ethinylestradiol/lynestrenol • Furosemide • Isoniazid • 6-Mercaptopurine • Metronidazole • Norethindrone/mestranol • Valproicacid • Selindac

Congenital anomalies causing pancreatitis[23,24]:

Pancreatic Divisum:

Pancreas divisum is the most common congenital anomaly of the pancreatic ductal system. This condition results from the failure of fusion of the ventral and dorsal pancreatic anlage and relative obstruction to the flow of pancreatic juice is generated because the majority of the gland empties through the dorsal duct (duct of Santorini) into the minor papilla, which is small. The ventral duct (duct of Wirsung), which opens into the major papilla, drains only the ventral pancreatic anlage, which forms the head and uncinate process. Symptoms of pancreatitis can sometimes develop even in patients with a small communication between the ventral and dorsal ducts, which drain separately into the duodenum, representing an anatomic variant (dorsal dominant duct syndrome).

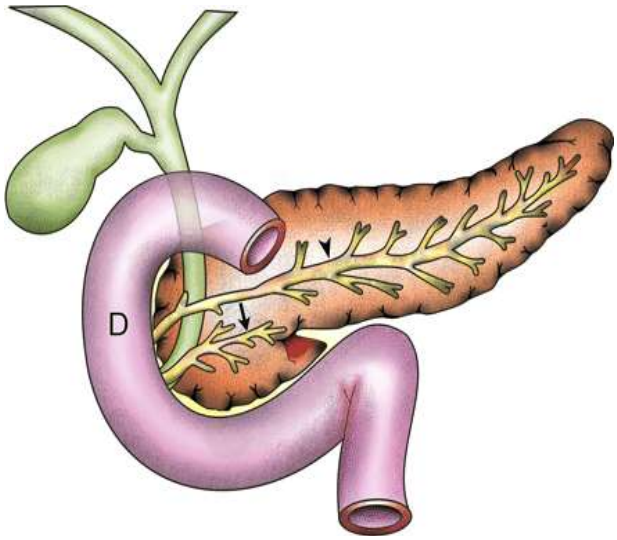


Figure 7:- Pancreas divisum.

Dorsal duct (arrowhead) draining the majority of the gland into the minor papilla. The smaller ventral duct (arrow) drains the head and uncinate process of the pancreas into the major papilla; the CBD also drains into the major papilla. D – duodenum.

Long pancreaticobiliary duct:
There can be an anomalous fusion of the pancreatic and biliary ducts when the ducts have a common channel of greater than 15mm or fuse outside the duodenum, this predisposes the reflux of pancreatic enzymes into the biliary tree and the reflux of bile into the pancreas leading to pancreatitis.

Annular Pancreas:
Annular pancreas is a rare congenital anomaly in which a ring of pancreatic tissue surrounds the second part of the duodenum. It occurs in about one in 20,000 persons. There is a bimodal distribution, with one-half of cases manifesting during childhood (commonly with duodenal obstruction) and the other half manifesting in adults in the 4th to 5th decades of life. In adults, annular pancreas may beincidentally detected, or the patient may present with symptoms of peptic ulcer disease (24.8% of cases) or pancreatitis (13.3%).

Pathophysiology And Course Of The Disease[25]:
The pathophysiology of acute pancreatitis is generally considered in three phases.
1. In the first phase, there is premature activation of trypsin within pancreatic acinar cells. Once trypsin is activated, it activates a variety of injurious pancreatic digestive enzymes.
2. In the second phase, there is intrapancreatic inflammation.
3. In the third phase, there is extrapancreatic inflammation.

Determinants of the natural course of acute pancreatitis are pancreatic parenchymal necrosis, extrapancreatic retroperitoneal fatty tissue necrosis, biologically active compounds in pancreatic ascites, and infection of necrosis.

Early in the course of acute pancreatitis multiple organ failure is the consequence of various inflammatory mediators that are released from the inflammatory process and from activated leukocytes attracted by pancreatic injury. During the late course, starting from the second week, local and systemic septic complications are dominant. Bacteriologic analysis of intraoperative smears and aspirates reveals predominantly gram-negative organisms derived from the intestine, most frequently Escherichia Coli[26].

In the majority of patients, acute pancreatitis is mild. In 10–20%, the various pathways that contribute to increasedintrapancreatic and extrapancreaticinflammation result in systemic inflammatory response syndrome (SIRS). In someinstances, SIRS predisposes to multiple organ dysfunction or pancreatic necrosis. Necrosis occurs early, within the first 24–48 hours, and with few exceptions remains stable during a given episode of acute pancreatitis.

Symptoms[14]:
Abdominal pain – mild to incapacitating, steady and boring located in the epigastrium and periumbilical region. The pain often radiates to the back, chest, flanks and lower abdomen. The pain is more intense when the patient is supine and is partially relieved by sitting with the trunk flexed and knees were drawn up.
Nausea and vomiting.
Abdominal distention.

Physical Examination[14]:
General examination:
Low-grade fever
Tachycardia
Hypotension
Shock
Jaundice rarely
Erythematous skin nodules
Cullen sign – faint blue discoloration around the umbilicus due to hemoperitoneum.
Grey Turner sign – blue/ purple discoloration of the flanks reflects tissue catabolism of hemoglobin.
The Cullen sign and Grey Turner sign are rare findings and their presence indicates severe necrotizing pancreatitis.

Respiratory system:
Basal crepitations
Pleural effusion (usually left sided)

Abdomen:
Tenderness in the epigastric region.
Muscle rigidity may be present but to a lesser extent compared to the pain.
Bowel sounds are diminished or absent.
Pancreatic pseudocyst may be palpable.

Complications[28]:
Local:
Necrosis – sterile/infected.
Pancreatic fluid collections – abscess/pseudocysts.
Involvement of contiguous organs.
Obstructive jaundice.

Systemic complications:
Pulmonary:
Pleural effusion
Atelectasis
Pneumonitis
ARDS

Cardiovascular:
Hypotension
Shock
Sudden death
Pericardial effusion

Hematologic:
DIC
Erosion into major blood vessels
Vessel thrombosis

Gastrointestinal:
Peptic ulcer disease

Erosive gastritis

Renal:
Azotemia
ATN

Metabolic:
Hyperglycemia
Hypertriglyceridemia
Hypocalcemia
Encephalopathy

CNS:
Psychosis
Fat emboli

Bone:
Necrosis

The presence of organ failure determines the outcome in cases that are difficult to manage. The respiratory failure (defined as Po2 < 60) is usually the predominant cause. The other important systemic complications are shock (systolic BP of < 90mm Hg and tachycardia > 130), renal failure (serum creatinine> 2mg/dl), abdominal bleeding (>500 ml / 24 hr), central nervous system failure (Glasgow Coma Scale score of less than 6 in the absence of sedation or by thesudden onset of confusion or psychosis), hepatic failure (serum bilirubin levels greater than 100 µmol/L or alkaline phosphatase levels greater than three times the upper limit of the normal range) and hematologic system failure (hematocrit level of less than 20%, WBC of less than 2,000/mm^3, or platelet count of less than 40,000/mm^3). Out of these, the presence of respiratory failure, shock, renal failure, and abdominal bleeding are the most important predictors of the outcome.

These patients need intense monitoring, correction of the metabolic abnormalities and supportive measures. It is a major cause of mortality in the first 2 weeks after an acute episode of pancreatitis.

The presence of local complications is an important cause of morbidity and may further necessitate interventions. It is a significant cause of mortality after 2 to 3 weeks of an acute episode.

Classification and definitions of four categories for the severity of acute pancreatitis[26]:
Table 3:- Classificationoftheseverityofacutepancreatitis.

Severity category	**Localcomplications**		**Systemiccomplications**
Mild	No(peri)pancreaticcomplication	and	Noorganfailure
Moderate	Sterile(peri)pancreaticcomplication	or	Transientorganfailure
Severe	Infectious (peri)pancreaticcomplication	or	Persistentorganfailure
Critical	Infectious (peri)pancreaticcomplication	and	Persistentorganfailure

Terminologies Associatedwithpancreatitis[30-33]:
Pancreaticnecrosis:
Itreferstofocalordiffusenonviablepancreaticparenchymaandusuallyperipancreaticfatnecrosis.Pancreaticnecrosiscanbe sterileorinfected.Peripancreaticnecrosisdescribesnecroticfattyandstromaltissuearoundthepancreas.

Acutefluidcollection:
Itreferstothefluidlocatedinornearthepancreasthatlacksadefinitewallandtypicallyoccursearlyinthecourseofacutepancrea

titis.Anacutefluidcollectionoccursin30%to50%ofcases ofacutepancreatitisandmostoftenitresolvesspontaneously.

Pancreaticpseudocyst:
Itreferstothefluidcollectionthatpersistsfor4to6weeksandbecomes encapsulatedbyawallo ffibrousorgranulationtissue.Whenapseudocystislocatedwithinthebodyofthepancreas,thecystma ycontainnecroticpancreaticdebrisevenwhenthepseudocystisfluid-appearingwithlowattenuationonCT.Thetermfora walled-offfluid-appearingpseudocyst-likestructureinvolvingthepancreasis walledoffpancreaticnecrosis(WOPN).WOPNispancreaticnecrosisthathasliquefiedafterfivetosixweeks.

Pancreaticphlegmon:
Itreferstothetermoftenusedbyradiologiststodescribeaninflammatorymass.

Diagnosis[15]:
There is a general acceptance that a diagnosis of acute pancreatitis requires two of the following three features:
1. Abdominal pain characteristic of acute pancreatitis,
2. Serum amylase and/or lipase ≥3 times the upper limit of normal,
3. Characteristic findings of acute pancreatitis on CT scan.

This definition allows for the possibility that an amylase and/or lipase might be <3 times the upper limit of normal in acute pancreatitis. In a patient with abdominal pain characteristic of acute pancreatitis and serum enzyme levels that are lower than 3 times the upper limit of normal, a CT scan must be performed to confirm a diagnosis of acute pancreatitis. In addition, this definition allows for the possibility that the presence of abdominal pain cannot be assessed in some patients with severely altered mental status due to acute or chronic illness.

DifferentialDiagnosisof AcutePancreatitis[14]:
1. Perforated viscous
2. Acute cholecystitis and biliary colic
3. Acute intestinal obstruction
4. Mesenteric vascular occlusion
5. Renal colic
6. Closed-loopintestinalobstruction
7. Inferiorwallmyocardialinfarction
8. Dissecting aortic aneurysm
9. Ruptured ectopic pregnancy.

SeveritypredictorsofPancreatitis:
Severe Pancreatitis: Clinical Evaluation
Recognition of severe pancreatic injury by means of clinical examination is unreliable. Clinical parameters such as tachycardia, orthostatic hypotension, shock, respiratory distress, and signs of peritonitis are consistent with a severe attack. They are however rarely seen and are not specific, and usually develop late which limits their clinical use.

Flank ecchymosis (Grey-Turner sign) or periumbilical ecchymosis (Cullen sign) are more specific indicators for severe acute pancreatitis and have been associated with a 37% mortality rate. These signs are rarely present, however, and they often appear 48–72 hours after the onset of symptoms [67].

Diagnosis on the basis of the clinical evaluation was missed in 30%–40% of patients with fatal necrotizing pancreatitis until the time of autopsy. Thus, individual clinical signs have only limited value for the assessment of the severity of acute pancreatitis [68].

Organfailure:
Thereisconsiderableinterestamongpancreatologistsinusingorganfailuretopredictseverity.TheAtlantacriteriadefined which organ systemsareofimportance: pulmonary,renal,andcardiovascularsystem.
Multisystem organfailureisdefinedastwoormoreorgansfailingonthesameday,ratherthanoneorganfailingononedayandanotherfailing onthesubsequentday.[46]

Patientswithmultisystemorganfailureorpersistentorganfailurehaveamuch higher mortality rateapproaching 50% as compared with patients with single and transientorganfailure.[47]

Persistentorganfailureisdefinedaslastinggreaterthan24hoursregardless of intervention.Survivalamongpatientswithorganfailureatadmissionhasalsobeenshowntocorrelatewiththedurationoforgan failure.When theorganfailurepersistsformorethan48 hours,mortalityis 36%.[48]

Peritoneallavage:
Percutaneousrecoveryofanyvolumeofperitonealfluidwithadarkcolororrecoveryofatleast20 mL of freeintraperitonealfluidofdarkcolorportendsasignificantmortality[49].Thesensitivityofperitoneallavageis36%to72%,and thespecificityis greaterthan80%to100%.

Laboratory Markers:
Hematocrit[49]:
A high hematocrit on admission or one that fails to decrease after 24 hours of rehydration is thought to be a sign of hemoconcentration due to retroperitoneal fluid loss and thus a marker of severe disease. An elevated hematocrit (>44%) is a predictor for the development of necrosis. The hematocrit should be observed at admission for prognostic purposes and followedprospectivelytoassistin guiding therateofintravenous hydration.

SerumAmylaseandLipaseLevels[50]:
Elevatedserumamylaseandlipaselevels,incombinationwithsevere

abdominal pain,oftentriggertheinitialdiagnosisofacutepancreatitis.Serumlipaserises4to8 hours fromtheonsetofsymptomsandnormalizes within7to14days aftertreatment.

Serumamylasemaybenormal(in10%ofcases) forcasesofacuteorchronic pancreatitis (depletedacinarcellmass)andhypertriglyceridemia.

Thecausesforfalsepositiveelevatedserumamylaseincludesalivaryglanddisease (elevated salivaryamylase),bowelobstruction, infarction, cholecystitis, and perforatedulcer.

C-reactiveprotein:

CRPisanacute-phasereactantproducedbytheliverandisusedextensivelyin Europeasamarkerofseverepancreatitis[51]

PolymorphonuclearLeukocyteElastase:
Polymorphonuclearleukocyteelastaserisesveryearly(beforeCRP) in acute pancreatitis.Highlevelshavebeenreportedtodifferentiateseverefrommilddisease.[52]

PhospholipaseA$_2$:
PLA$_2$ isinvolvedin thereleaseofprostaglandinsfromcellmembranesand degrades surfactant inthelung. It may play aroleinthepulmonarydysfunction
associatedwithacutepancreatitis.LevelsofcatalytictypeIIPLA$_2$havebeenreportedtodifferentiatebetweenmildandsevere diseasewithin24hoursofadmission.[53]

UrinaryTrypsinogenActivationPeptide:

The urinaryTAPmayserveasanearlypredictorofseverityinpatientswithacute pancreatitis.[37] ElevatedurinaryTAP(>30 nmol/L)correlateswithdiseaseseverity.Thetestcanbeappliedwithin12hoursof
admission.Thepositivepredictivevalueofan elevatedTAPis 80%andthenegativepredictivevalueapproaches 100%.

Procalcitonin:
This propeptideisaereactantthathas beenshowntodifferentiatemildfromsevereacutepancreatitiswithinthefirst24hoursaftersymptom onset. A serum strip test has been developed that has a sensitivity of 86% and a specificityof95%indetectingorganfailure.[54]

Interleukin-6:
IL-6isanacute-phase-reactantcytokinethatisproducedby avarietyofcellsandinduceshepaticsynthesisofCRP.Severalstudieshaveshownthatitis areasonablygoodmarkertodifferentiatemildfromseveredisease,butthetestisnotreadilyavailable.[55]

Scoring indices:
The Atlanta criteria[26] defines severity by the presence of organ failure or pancreatic necrosis on dynamic contrast-enhanced CT scan, other acceptable markers of severe pancreatitis include three or more of Ranson's criteria score[42]fornon-gallstonepancreatitis and an Acute Physiology and Chronic Health Evaluation (APACHE-II) score of greater than eight[43].

BISAP (Bedside Index for Severity in Acute Pancreatitis)[44]:
Itisasimplescoringsystemthatincludes5variablestodetermineseverityearly in thecourseofepancreatitis.The BISAPscore provides a single point for 5 parameters:
1. bloodureanitrogen(BUN)greater than25mg/dL,
2. impairedmentalstatus,
3. systemicinflammatoryresponsesyndrome,
4. agegreaterthan60,and/or
5. thepresenceofapleuraleffusion,

ABISAPscoregreaterthan3isassociatedwithaseven-toa twelve-foldincreasein developingorganfailure.The MarshallScoringSystem[45]for organ failure is commonlyusedbyintensivistsforpatientsadmittedtoanintensivecareunit.

RansonScore[44]
Ransoncriterionisaclinicalpredictionruleforpredictingtheseverityof acute pancreatitis whichwasintroducedin1974.(Table-4)

Table4: Ransoncriteriaforassessmentoftheseverityof AP
Ranson'scriteriaofseverityofacutepancreatitis:
Onadmission:
1. Age>55years
2. Whitebloodcount>16000/mm3
3. Glucose>11.0mmol/l
4. Lactatedehydrogenase>350 IU/l
5. Aspartate aminotransferase>250U/l

Duringinitial48hours:
1. Packedcellvolumedecrease>10%
2. Bloodureanitrogenincrease>1.8mmol/l
3. Calcium<2mmol/l
4. Partialpressureofoxygen<60mmHg
5. Basedeficit>4mmol/l
6. Fluidsequestration>6l

Ranson'sscoreinterpretation:
1. Score<3mildpancreatitis
2. Score5to6moderatepancreatitis
3. Score7to8more :severepancreatitis

Ranson'sscoreandmortalitycorrelation:
1. Score0to2:2%mortality
2. Score3to4:15% mortality
3. Score5to6:40% mortality
4. Score7to8:100% mortality

Ranson'sscoreof≥8indicatessubstantialpancreaticnecrosis(atleast30%pancreatic necrosis accordingtocontrast-enhancedCT).

Ransoncriteriadrawbacks:
1. Differentscoringforgallstonepancreatitisandnon-gallstonepancreatitis.
2. AnaccurateRanson'sscoretakes 48hourstocalculate.
3. Notalllaboratoriesmeasureallthe parameters(e.g., LDH) ofRanson'scriteria.
4. Theoverallsensitivityof theRanson'scorefordiagnosingseverediseaseisonly 40%to88%andspecificityis 43% to90%.
5. Thepositivepredictivevalueisapproximately50%andthenegativepredictive valueof around90%.

Therefore, thebestuseofRanson'scriteriaistoexcludeseveredisease.

Apache(Acutephysiologyscoreandchronichealthevaluation)[44]:
Thefirstmajorattemptatasystemtoquantifytheseverityofillnessin ICUpatients was theAPACHEsystemintroducedbyKnausetalin1981.

Apachei:
In the original form, APACHE contained 34 physiologicmeasurementsand includedmanycontinuousvariables.Shortlyafteritsintroduction, APACHE1system was notlikedbecauseofpracticalproblemslikethecollectionof a largenumberofvariables. Apacheii

ThisisthesecondversionoftheAPACHEsystemandcontainedrefinementsbased on experiencewiththeoriginalAPACHEsystem.Itcontains12continuous variablesfromtheoriginalAPACHEsystemandalsotakesintoaccounttheageofthe patient,premorbidconditionsandGlasgowcomascale.[43](Table-5)

Table-5:- APACHEscoreforassessmentoftheseverityofacutepancreatitis.

Physiologicvariable:
1. temperature
2. Meanarterialpressure(mmHg)
3. Heartrate
4. Respirations
5. ArterialpH
6. PaO2 (mmHg)
7. Serumsodium
8. Serumpotassium
9. Serumbicarbonate (mmol/L)Serumcreatinine(mg/dl)Hematocrit(%)
10. Whitebloodcellcount
11. GlasgowComaScore

APACHEIIscore=A+B+C.
APACHEIIscore>8points predicts 11% to18% mortality

Glasgowcriteria:
TheGlasgowcriterionisvalidforbothgallstoneandalcohol-inducedpancreatitis

(Table-6).

Table6:- Glasgowcriteriaforassessmentoftheseverityofacutepancreatitis.

Criteria	Value
Age(yr)	>55
WBCcount(x100/mm^2) Glucose(mg/dl) BUN(mg/dl) LDH (IU/1) Albumin(g/dl) PaO2	>15

(mmHg) Calcium(mg/dl)	>180 >45 >600 <3.3 <60 <8

Glasgowscoreinterpretation:
If apatientscores3ormoreitindicatesseverepancreatitisandthepatientshould betransferredtoITU.

Balthazar Scoring[6]:
Developed in the early 1990s by Emil J. Balthazar et al. the Computed Tomography Severity Index (CTSI) is a grading system used to determine the severity of acute pancreatitis. The numerical CTSI has a maximum of ten points and is the sum of the Balthazar grade points and necrosis score.

Table 7:- Balthazar Grade.

Balthazar Grade	Appearance on CT	CT Grade Points
Grade A	Normal CT	0 points
Grade B	Focal or diffuse enlargement of the pancreas	1 point
Grade C	Pancreatic gland abnormalities and peripancreatic inflammation	2 points
Grade D	Fluid collection in a single location	3 points
Grade E	Two or more fluid collections and/or gas bubbles in or adjacent to the pancreas	4 points

Table 8:- Necrosis Score.

Necrosis Percentage	Points
No necrosis	0 points
0 to 30% necrosis	2 points
30 to 50% necrosis	4 points
Over 50% necrosis	6 points

CTSI's staging of acute pancreatitis severity has been shown by a number of studies to provide a more accurate assessment than APACHE II, Ranson, and CRP level.[69,70.]

Modified CT severity index:
The modified CT severity index correlated more closely with patient outcome measures than the currently accepted CT severity index, with similar interobserver variability. [70]

However, the most recent study conducted showed no significant differences between the CTSI and the MCTSI in evaluating the severity of AP. Compared with APACHE II, both CT indices more accurately diagnose clinically severe disease and better correlate with the need for intervention and pancreatic infection. [72]

Table 9:- Modified CT Severity Index.

Prognostic Indicator	Points
Pancreatic inflammation	
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4

Pancreatic necrosis	
None	0
≤ 30%	2
> 30%	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement)	2

Severe Acute Pancreatitis As Defined By Atlanta Symposium:
Early Prognostic Signs:
1. Ranson signs ≥3
2. APACHE-II score ≥8
3. Organ Failure
Shock–systolic pressure <90 mmHg
PaO2 ≤60 mmHg
Creatinine>2.0 mg/L after rehydration
Gastrointestinal bleeding >500 cc/24 h
4. Local Complications
Necrosis
Abscess
Pseudocyst

Others including disseminated intravascular coagulation (platelets ≤100,000/mm^3, fibrinogen ≤100 mg/dL, fibrin split products >80 μg/mL), or a severe metabolic disturbance (serum calcium ≤7.5 mg/dL)

The diagnostic guideline I: look for risk factors of severity at admission
Risk factors for severe acute pancreatitis[64]:
Obesity – BMI > 30
Old age > 55yrs
Organ failure at admission and
Pleural effusion and/or infiltrates

The importance of established risk factors of severity of acute pancreatitis at admission is to transfer those patients who are most likely to have a severe episode to a step-down unit or an intensive care unit for closer supervisionGender and etiology have no prognostic significance.

Diagnostic Guideline II: Determination Of Severity By Laboratory Tests At Admission Or ≤48 H:
The two tests that are most helpful at admission in distinguishing mild from severe acute pancreatitis are APACHE-II score and serum hematocrit.

It is recommended that APACHE-II scores be generated during the first 3 days of hospitalization and thereafter as needed to help in this distinction.

It is also recommended that serum hematocrit is obtained at admission, 12 h after admission, and 24 h after admission to help gauge adequacy of fluid resuscitation.

In this report, hematocrit ≥44 at admission and failure of admission hematocrit to decrease at 24 h were the best predictors of necrotizing pancreatitis.[49]

Diagnostic Guideline III: Determination of Severity during Hospitalization:
Pancreatic necrosis and organ failure are the two most important markers of severity in acute pancreatitis. The distinction between interstitial and necrotizing pancreatitis can be reliably made after 2–3 days of hospitalization, by contrast, enhanced CT scan [45].

Many patients with acute pancreatitis do not require a CT scan at admission or at any time during the hospitalization. For example, a CT scan is usually not essential in patients with recurrent mild pancreatitis caused by

alcohol. A reasonable indication for a CT scan at admission is to distinguish acute pancreatitis from another serious intra-abdominal condition, such as a perforated ulcer. A reasonable indication for a contrast-enhanced CT scan a few days after admission is to distinguish interstitial from necrotizing pancreatitis when there is clinical evidence of increased severity. The distinction between interstitial and necrotizing pancreatitis can be made much more readily when a contrast-enhanced CT scan is obtained on the second or third day after admission rather than at the time of admission.

Additional contrast-enhanced CT scans may be required at intervals during the hospitalization to detect and monitor the course of intra-abdominal complications of acute pancreatitis, such as the development of organized necrosis, pseudocysts, and vascular complications including pseudoaneurysms.

Contrast-enhanced CT scan is the best available test to distinguish interstitial from necrotizing pancreatitis. Interstitial pancreatitis is characterized by an intact microcirculation and uniform enhancement of the gland. Necrotizing pancreatitis is characterized by disruption of the microcirculation such that devitalized areas do not enhance. Whereas small areas of non-enhancement could represent intrapancreatic fluid rather than necrosis, large areas of non-enhancement clearly indicate a disruption of microcirculation and pancreatic necrosis

Complications in acute pancreatitis that can be recognized on abdominal CT scan include pancreatic fluid collections, gastrointestinal and biliary complications (such as obstruction of duodenum or stomach, inflammation of the transverse colon, and biliary obstruction), solid organ involvement (such as splenic infarct), vascular complications (such as pseudoaneurysms, splenic vein thrombosis with varices, portal vein thrombosis), and pancreatic ascites.Prompt transfer to an intensive care unit should take place for sustained organ failure. Transfer to an intensive care unit (or possibly a step-down care unit) should be considered if there are signs that suggest that the pancreatitis is severe or is likely to be severe. Additional danger signals that warrant close supervision by physicians and nursing staff in a step down unit but not necessarily urgent transfer to an intensive care unit include obesity(BMI >30), oliguria with urine output <50 mL/h, tachycardia with pulse >120 beats/min, evidence of encephalopathy, and increasing need of narcotics.

Overview of acute pancreatitis [50, 51]:
Overall, 85% of patients have interstitial pancreatitis; 15% (range 4–47%) have necrotizing pancreatitis. Among patients with necrotizing pancreatitis, 33% (range 16–47%) have infected necrosis.

Table 10:- Mortality in Acute Pancreatitis.

	Median(%)	Range (%)
All cases	5	2-9
Interstitial pancreatitis	3	1-7
Necrotizing pancreatitis	17	8-39
Infected necrosis	30	14-62
Sterile necrosis	12	2-44

The mortality in the absence of organ failure is 0, with single organ failure is 3% (range 0–8%), with multisystem organ failure 47% (range 28–69%).

Deaths within the first 2 weeks are generally attributed to organ failure; deaths after this interval are generally caused by infected necrosis or complications of sterile necrosis.

Role of Radiology:
Conventional imaging [40]:
Radiographic signs of acute pancreatitis include the sentinel loop sign (dilated air-filled duodenum or jejunum), the colon cutoff sign (dilated large bowel to the level of the splenic flexure), loss of the left psoas shadow, ascites, or a gasless abdomen Pleural effusions, atelectasis, or an elevated hemidiaphragm are suggestive of severe acute pancreatitis [41].

Thickened rugal and duodenal folds, indentation of the stomach, and enlargement of the C-loop of the duodenum are signs of acute pancreatitis on barium meal and follow-through studies [40].

Ultrasound[40, 56]:
Sonography of patients with acute pancreatitis is often limited by difficulty in visualizing the pancreas because of ileus and overlying bowel gas. Abnormal ultrasound findings like ascites, pleural effusions are seen in 33–90% of patients with acute pancreatitis.

Interstitial edema in acute pancreatitis is depicted on ultrasound as an enlarged hypoechoic gland. Although ultrasound may be used to identify peripancreatic acute fluid collections, it is not useful for the detection of necrosis, and therefore its main role in the imaging of acute pancreatitis is limited to the detection of cholelithiasis and choledocholithiasis and identification of fluid collections in the peritoneum, retroperitoneum, and pleural spaces.

CT:
Contrast-enhanced CT is the imaging modality of choice for the diagnosis and staging of acute pancreatitis [57]

Protocol [58]:
A three-phase (control, pancreatic parenchymal phase (40 seconds), and portal venous phase) protocol can be used for the initial assessment of acute pancreatitis. Positive oral contrast may be administered after the control scan.

Control phase –The use of positive oral contrast material may mask hemorrhage or calculi. So the initial control study may be done with no / negative oral contrast so that hemorrhagic collections and calculi may be identified in this phase.

The pancreatic parenchymal phase is the optimal phase for assessment for necrosis because normal pancreatic tissue enhances the greatest during this phase.

Subsequent imaging with CT is generally performed using a single-phase technique in the portal venous phase.

The suggestion that IV administration of iodinated contrast material can increase the severity and duration of acute pancreatitis has led to conflicting opinions regarding IV contrast usage and at present, the benefits of IV contrast administration appear to outweigh the potential risks.[52]

CT features:
The pancreas enhances uniformly in mild acute pancreatitis and may be normal or enlarged with a variable amount of increased attenuation in the adjacent fat, termed “stranding”.

Local edema is a common finding and may extend along the mesentery, mesocolon, and hepato-duodenal ligament and into peritoneal spaces. Extension of edematous fluid into the anterior perirenal space may create a mass effect and a halo sign with sparing of the perinephric fat.

Peripancreatic fluid collections consist of exudate, peripancreatic fat tissue necrosis, or hemorrhage.

An organized “pseudocyst” may be formed. Edema is differentiated from fluid collections by the identification of fat islands of normal tissue within edematous fluid[13].
Non-enhancement of all or part of the gland is termed "necrosis". CT is 100% specific for necrosis if greater than 30% of the gland is non enhancing[40]. Necrosis develops between 24 and 48 hours after the onset of acute pancreatitis, and therefore CT within the first 12 hours may be falsely reassuring.

Pancreatic abscess formation is usually observed 4–6 weeks after the onset of acute pancreatitis as an area of low attenuation containing pus and a thick wall that may enhance after IV contrast administration. Air bubbles may be found within the collection.

Necrosis and abscess are considered among the most important imaging features of acute pancreatitis because they have prognostic relevance and may need intervention by either interventional radiologists or by the surgeons [40].

International Symposium, held in Atlanta, GA, in 1992, established a clinical based classification system for acute pancreatitis [2]. The goal was to establish international standards of definitions of acute pancreatitis and its

complications to make possible valid comparisons of the severity of illness and the results of therapy and also to establish a

ground for future trials. This was a group of 40 international authorities from six medical disciplines and 15 countries.

Interstitial pancreatitis was defined as focal or diffuse enlargement of the pancreas with enhancement of the parenchyma that is either homogeneous or slightly heterogeneous in response to IV contrast. There may be inflammatory changes in peripancreatic fatty tissue characterized by a hazy appearance.

Pancreatic necrosis was defined as diffuse or focal areas of nonviable pancreatic parenchyma that was typically associated with peripancreatic fat necrosis. The criteria for the CT diagnosis of necrosis included focal or diffuse well-marginated zones of nonenhanced pancreatic parenchyma greater than 3 cm in size or greater than 30% of the pancreas.

An extrapancreatic fluid collection was defined as pancreatic fluid that extravasates out of the pancreas during acute pancreatitis into the anterior pararenal spaces and other areas as well.

A pancreatic pseudocyst was defined as a collection of pancreatic juice enclosed by a non epithelialized wall that occurs as a result of acute pancreatitis, pancreatic trauma, or chronic pancreatitis. It is generally believed that a period of at least 4 weeks is required from the onset of acute pancreatitis to form a well-defined wall composed of granulation and fibrous tissue. [59]

Severe pancreatitis was defined as pancreatitis associated with organ failure and/or local complications (necrosis, pseudocyst).

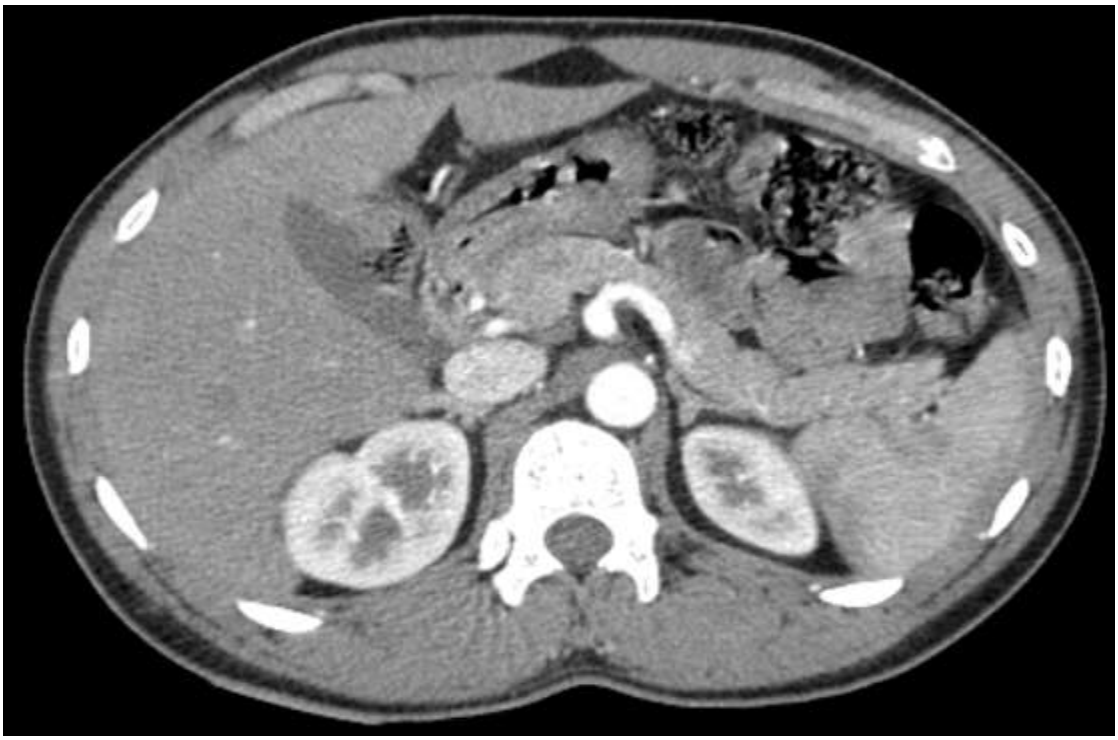


Figure 8:- AxialCECTsectionofthe normalpancreasin the arterialphase.

MRI[13]:

MRI may be performed using unenhanced and contrast-enhanced T1-weighted and fat-suppressed T2-weighted sequences. Heavily T2 weighted thick slab and thin sections are obtained for delineation of the ductal anatomy.

An enlarged edematous gland that is low signal on T1-weighted and high signal on T2-weighed MRI is observed [5]. Acute pancreatitis is sometimes associated with pancreatic ductal dilatation, which can be clearly identified and examined on T2-weighted images.

T2-weighted images are also useful for the detection of acute pancreatic collections and pseudocyst.

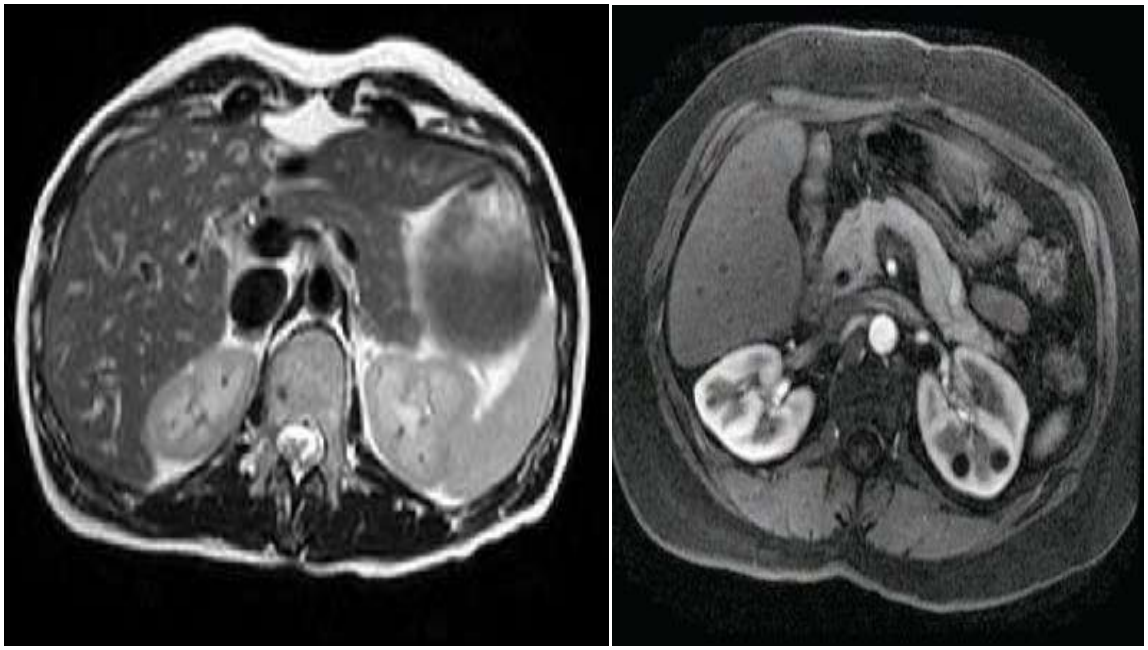
It has the advantage of demonstrating possible choledocholithiasis, the presence or absence of ductal distention, disruption or leakage of the pancreatic duct, and the size, location, and possible communication of a pseudocyst with the pancreatic duct. In addition, it better demonstrates local hemorrhage in or around the pancreas and helps assess the internal consistency and drainability of fluid collections which may influence the choice of treatment[60].

The pancreatic duct disconnection occurs when necrosis affects the ductal epithelium and an isolated segment of viable pancreatic tissue is disconnected from the duodenum. This creates persistent fistulation and inflammation with an increased incidence of infection. Diagnosis of disconnection of the main pancreatic duct requires visualization of a necrotic region of at least 2 cm in size, viable pancreatic tissue proximal to the necrosis [61].

Early MRCP may sometimes be of limited value for identifying the cause of acute pancreatitis because collections may compress the pancreatic and biliary ducts obscuring gallstones. MRCP may be of benefit when iodinated contrast administration is contraindicated or if disconnection of the main pancreatic duct is suspected.

MRI has not been widely used in the care of patients with acute pancreatitis. While CT scan remains the primary imaging technique to evaluate patients with acute pancreatitis, recent reports have indicated that MRI has some advantages: no concern regarding radiation exposure, the greater ability of MRI as compared to CT to distinguish necrosis from fluid, and the overall reliability of MRI as compared to CT scan in staging the severity of acute pancreatitis and its complications [62]. Accurate identification of retained bile duct stones and pancreatic duct leaks. Disadvantages of MRI include lack of availability when urgently needed, variation in quality among centers, and the difficulty of supervising a critically ill patient undergoing MRI.

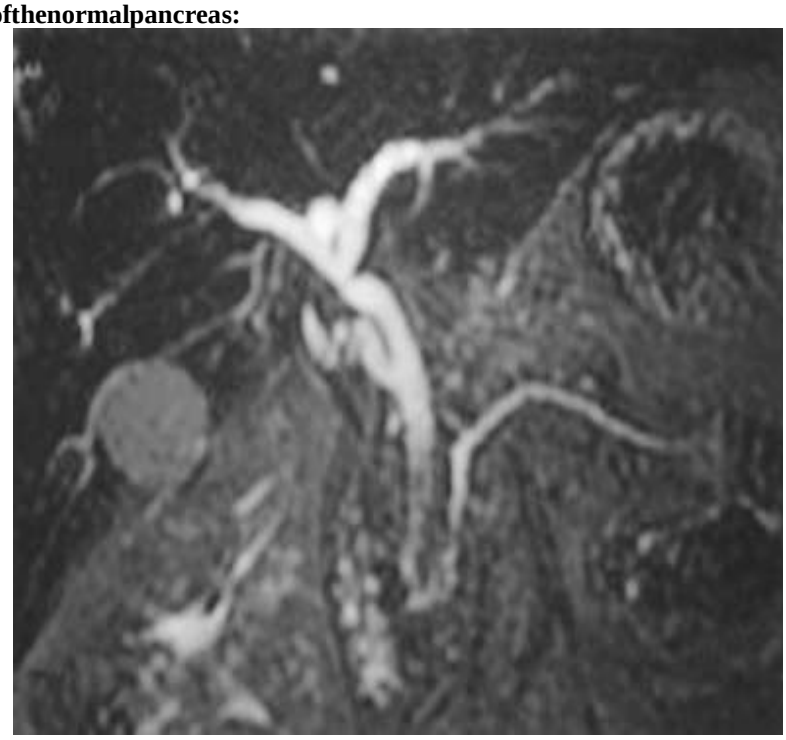
In the imaging of acute pancreatitis using MRI the same descriptive terminology as that used in CT is used.



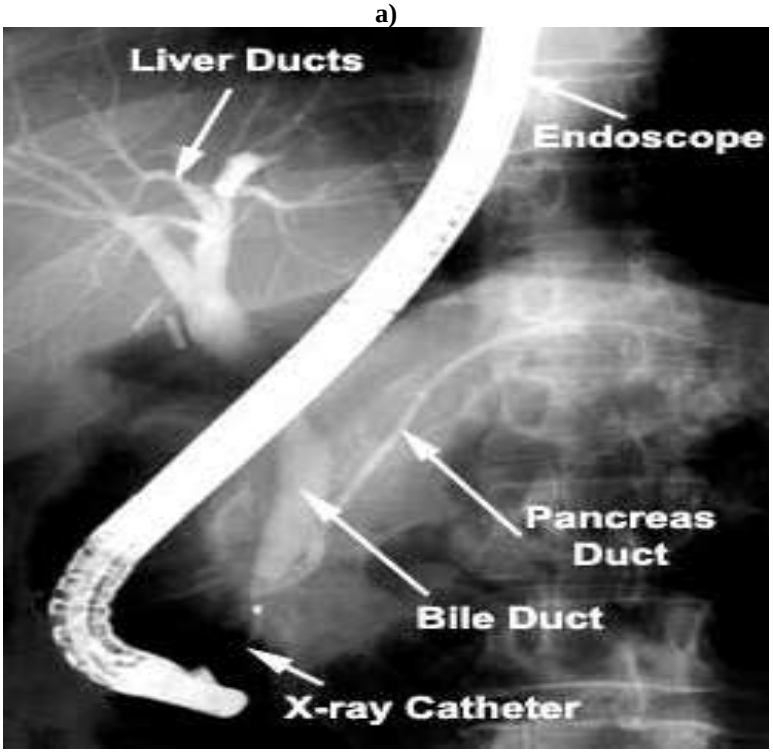
a **b**

Figure 9:- (a)AxialT2Weighted and(b)gadolinium-enhanced

T1Weightedimage ofthenormalpancreas:



a)



b)

Figure 10: - (a)MRCPand(b)ERCPdepictingthenormalanatomyofthe

Pancreatic duct,commonbileduct,commonhepaticduct, andintrahepaticducts:

Role of intervention radiology[63]:

CT is the modality of choice to guide the intervention procedures because the retroperitoneal location and the adjacent bowel loops can be better evaluated by CT.

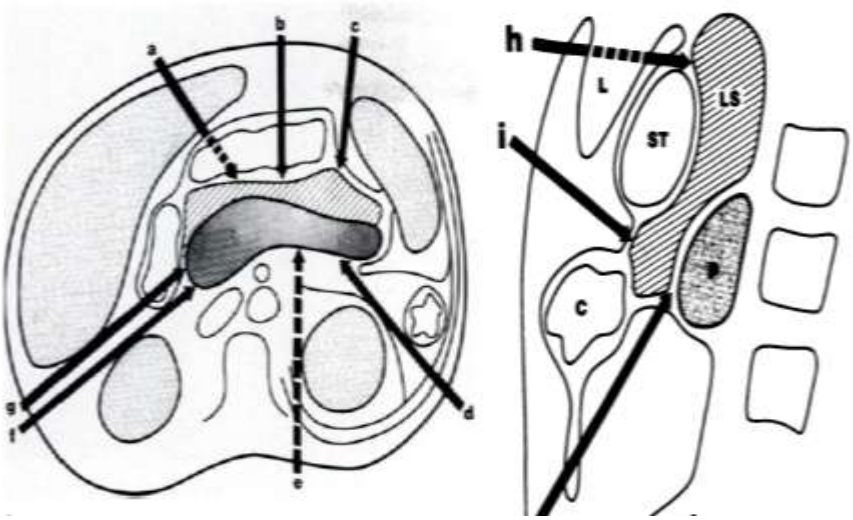


Figure 11:- Access routes to pancreatic intervention.

Transhepatictransgastric, b) transgastric, c) gastrosplenic, d) left anterior pararenal space to body and tail of the pancreas, e) paravertebral, f) right pararenal, g) pancreatic head through the duodenum, h) transhepatic, i) gastrocolic to head and body of pancreas j) transmesocolic.

L: liver, ST: stomach, LS : lesser sac, C: colon.

The most common approaches include left anterior para renal space approach for tail collections and gastrocolic for head and body collections.

Treatment:

Treatment Guideline I: Supportive Care [35]

Prevent hypoxemia and ensure adequacy of fluid resuscitation.

It is recommended that supplemental oxygen is administered until there is no further threat of hypoxemia. Aggressive IV fluid replacement is of critical importance to counteract hypovolemia caused by third space losses, vomiting, and diaphoresis.

The abdominal pain is relieved with a parenterally administered narcotic medication.

Treatment Guideline II: Transfer To An Intensive Care Unit[35]:

Prompt transfer to an intensive care unit should take place for sustained organ failure. In particular, sustained hypoxemia, hypotension refractory to a bolus of IV fluids, and possibly renal insufficiency that does not respond to a fluid bolus (such as a serum creatinine>2.0 mg/dL) warrant prompt transfer to an intensive care unit.

Treatment guideline III: Nutritional support[36]:

In general, oral intake is usually initiated when the abdominal pain has subsided, abdominal tenderness has markedly decreased, nausea and vomiting have ceased, bowel sounds are present.

Treatment Guideline IV: Use of Prophylactic Antibiotics in Necrotizing Pancreatitis[37]:

The use of prophylactic antibiotics to prevent pancreatic infection is not recommended at this time among patients with necrotizing pancreatitis.

Treatment Guideline V: Treatment of Infected Necrosis [38]:

CT-guided percutaneous aspiration with Gram's stain and culture is recommended when infected necrosis is suspected. Treatment of choice in infected necrosis is surgical debridement. It is impossible to distinguish these

conditions clinically unless CT scan shows evidence of air bubbles in the retroperitoneum. Another technique is percutaneous catheter drainage of infected necrosis.

Treatment Guideline VI: Treatment of Sterile Necrosis [38]:
Sterile necrosis is best managed medically during the first 2–3 wks. After this interval, if abdominal pain persists and prevents oral intake, debridement should be considered. This is usually accomplished surgically, but percutaneous or endoscopic debridement is a reasonable choice.

Treatment Guideline VII: Role of ERCP and Biliary Sphincterotomy in Gallstone Pancreatitis [39]:
ERCP is indicated for clearance of bile duct stones in patients with severe pancreatitis, in those with cholangitis, in those who are poor candidates for cholecystectomy, in those who are postcholecystectomy, and in those with strong evidence of persistent biliary obstruction.

Material and Methods:-

Cases of suspected acute pancreatitis referred to the department of Radio-diagnosis, Mysore Medical College and Research Hospital to the contrast-enhanced computed tomography on the clinical suspicion/diagnosis of acute pancreatitis, altered biochemical parameters (serum amylase, serum lipase) in favor of acute pancreatitis were included in this study. The study period wasbetween 1/11/2016 – 31/04/2018.

Study Design:
Hospital-based prospective study.

Study Area:
Mysore Medical College and Research Institute, Mysore.

Sample Size:
As per the biostatistician's opinion the minimal sample size requiredwas 32 cases for satisfactory statistical analysis. However a total number of50 cases are included under the advice of the biostatistician

Statistical Analysis:
Sensitivity, Specificity, and Positive Predictive value are analyzed.

MethodofCollectionof Data:
Cases of acute pancreatitis are graded according to the modified CT severity index. The patients are assessed on the 5^{th} day after the initial CT examination and on the day of discharge as follows:
Development of organ failure defined as :

1. Shock - systolic BP less than 90 mmHg
2. Respiratory failure - PO2 less than 60 mmHg
3. Renal failure - serum creatinine> 2 mg / dl
4. Gastrointestinal bleed > 500ml / 24 hr

Number of days of hospital stay.
Mortality.

Equipment:

Figure 12:- Dual slice Computed Tomography scaner(GE health care system).

Imaging Protocol:

Plain and post-contrast study of the abdomen and pelvis was done. It consists of the acquisition of contiguous axial sections, of thickness 7 mm of abdomen and pelvis and 5mm in the region of interest in cranio-caudal direction from the level of the xiphisternum to pubic-symphysis before and after administration of oral and intravenous iodinated contrast. Positive oral contrast was administered after the control scan. 100ml of the non-ionic contrast agent are given intravenously at the rate of 3 ml/sec. The study was done with the pancreatic parenchymal phase at 40 sec and venous phase at 90 sec. Reconstruction was done with a slice thickness of 3mm. All images were viewed in a range of soft tissue window settings.

Clinical details, laboratory and computed tomography findings of the caseswere recorded as per the proforma.

Theseverityofpancreatitiswasscoredusing modifiedCTseverityindexandclassifiedintothreecategories(mild,moderateandsevere).Themodifiedindexis a10 pointscoringsystemderivedbyassessingthedegreeofpancreaticinflammation(0to4 points)pancreaticnecrosis(0to4points)andextrapancreaticcomplications(0or2 points).Clinicaloutcomeparametersincludedthelengthofhospitalstay,theneedforsurgicalintervention and the occurrence of infection, organ failure and death.

Collected data were analyzed using descriptive statistics by using tabulations, graphs, charts and proportions, percentages.

Inclusion criteria:
1. All the patients who are suspected/diagnosed with acute pancreatitis based on clinical and laboratory findings (serum amylase & serum lipase).
2. Patients who diagnosed acute pancreatitis on ultrasound
3. Patients who present as acute on chronic pancreatitis.

Exclusion criteria:
1. Chronic pancreatitis.
2. Pancreatic trauma.

Ethical committee clearance:
Ethical clearance was obtained from the institution.

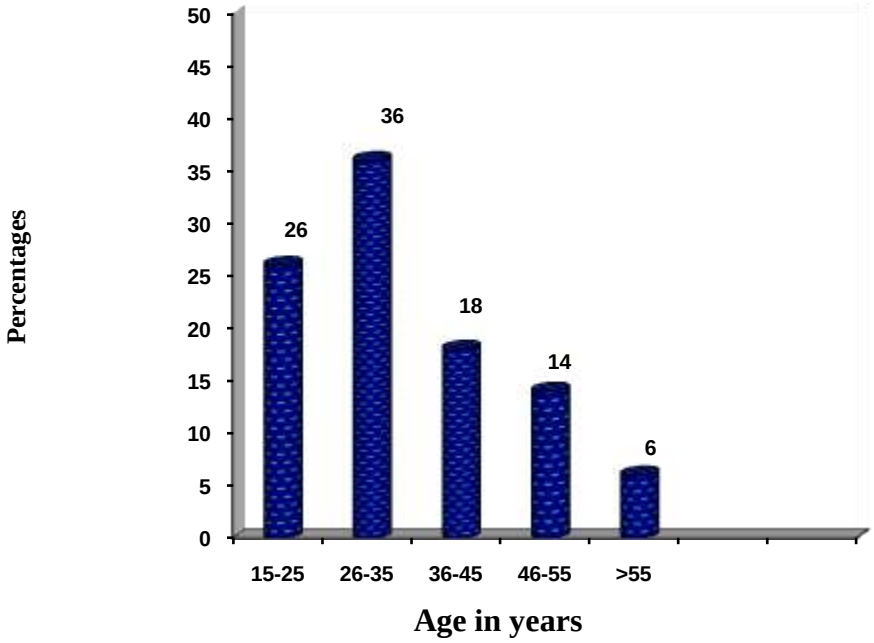
Results and Analysis:-

This study was conducted in the Department of Radio-diagnosis, Mysore medical college and research institute, Mysore from November 2016 to April 2018.Thestudy comprisedofatotalof50 patients.

Studygroup characteristics:
Agedistribution:
Table 11:- Age distributionofthestudygroup.

Age group (inyears)	Numberofpatients	Percentage
15-25	13	26
26-35	18	36
36-45	9	18
46-55	7	14
Above55	3	6
Total	**50**	**100**

The studyincludedpatientsbetweentheagegroupprangingfrom15yearsto66years withameanageof 34.32yrs.Themaximumnumberofpatientswasseenintheagegroupof26-35years of the agegroupwhichconsistedof18 (36%)patients.(Graph-1and Table-9)



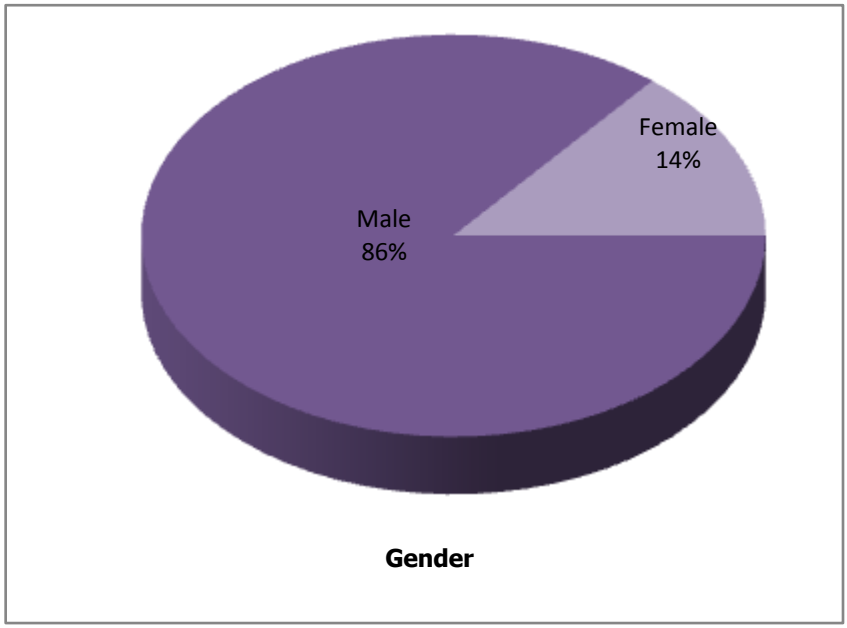
Graph1:- Age distribution of patients with acute pancreatitis.

Sexdistribution:

Table 12:- Sex distributionofthestudygroup.

Gender	Number of patients	%
Male	43	86
Female	7	14
Total	50	100

Inourstudy,outof 50cases,43(86%)weremaleand7(14%)were females withamaletofemaleratioof 8.6:1.4. (Graph-2,Table10)



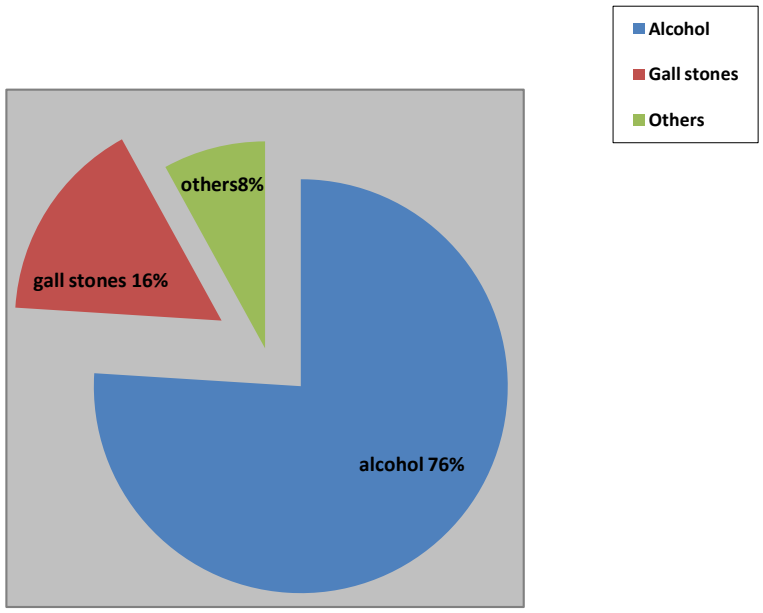
Graph 2: Gender distribution of patients with acute pancreatitis

Etiology of pancreatitis:

Table 13:- Etiology of pancreatitis.

ETIOLOGY	Number of patients (n=50)	%
Alcohol	38	76
Gallstones	8	16
Others	4	8
Total	50	

Inourstudy,3 8 of50patientswerealcoholiccomprisingof76%,8(16%)patients hadgallstonesandremainingpatientsweregroupedasothers whichconsistedof4patients.(Graph -3,Table-11)



Graph 3:- Etiology of pancreatitis.

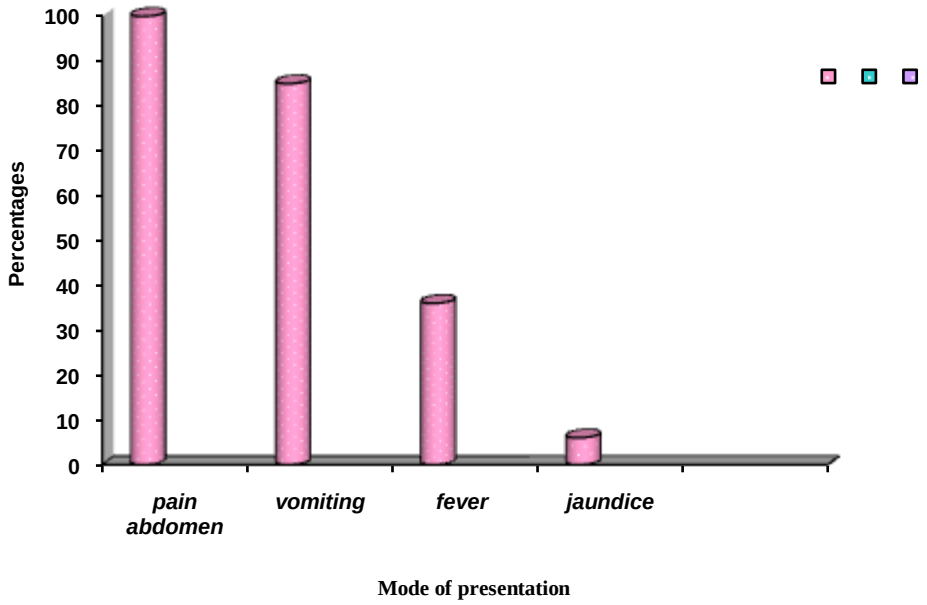
Diagnosis of pancreatitis:

Mode of presentation:

Table 14:- Mode of presentation.

Mode of presentation	Number of patients (n=50)	%
Pain abdomen	50	100
Vomiting	46	92
Fever	15	30
Jaundice	5	10

All50patients(100%)presented with painabdomen.46patients(92%)had vomiting,15(30%)patientshadfeverand5(10%)of patientshadjaundiceatthetimeofadmission(Graph -4 and Table-12).



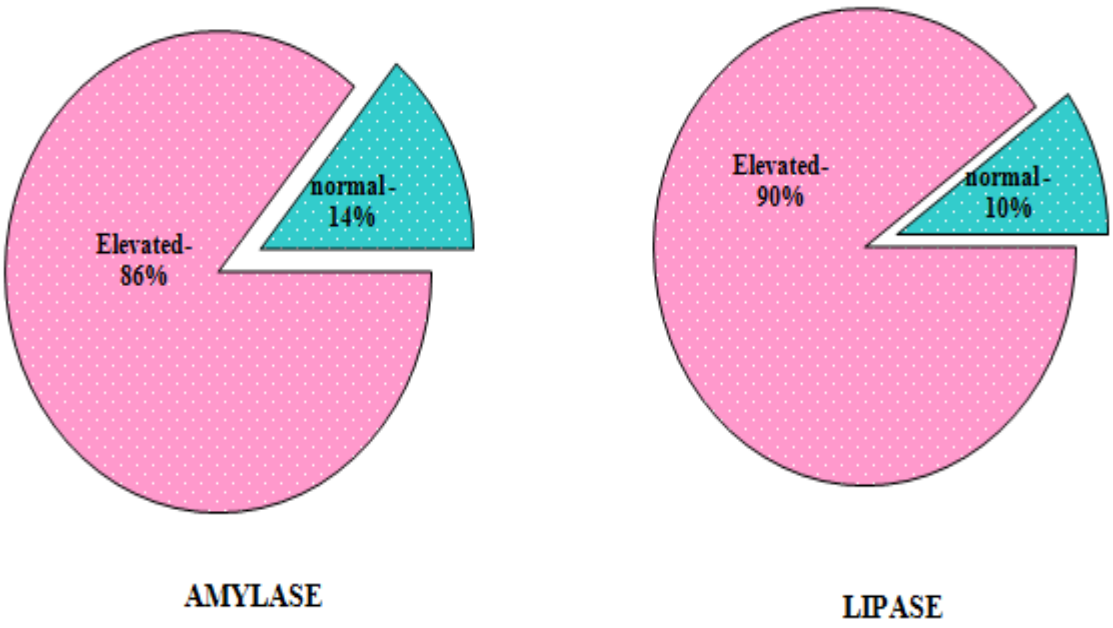
Graph 4:- Modeofclinicalpresentationofacutepancreatitis.

Laboratory diagnosis:

Table 15:- Patientswithelevatedamylaseandlipaselevels.

Lab parameters	Elevated (%)	Normal (%)
Amylase	86	14
Lipase	90	10

Amylasewaselevatedin43(86%)patientsatpresentation.Lipasewaselevated in45(90%)patients atpresentation(Graph-5 and Table-13).



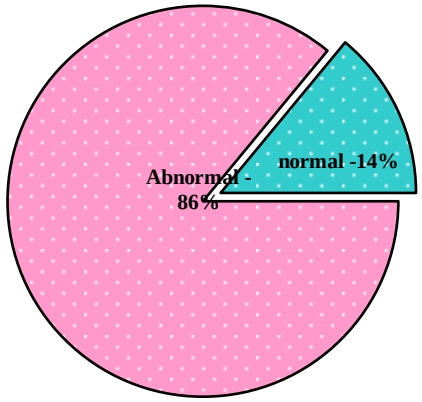
Graph 5:- Patientswithelevatedamylaseandlipaselevels.

Ultrasonography:

Table 16:- Patients withnormalorabnormalfindingsonultrasonography.

USG	Normal	Abnormal
Number of patients	7	43
Percentage	14	86

In7 (14%)patientsultrasoundwasnormal. 43 (86%)patientshadabnormalultrasoundfindingssuchasbulkypancreaswithalteredechogenicity,peripancreaticfatstrandin g,fluidcollection,ascitesorpleuraleffusion(graph-6,table-14)



Graph 6:- Patients withnormalorabnormalfindingsonultrasonography.

Computedtomographicevaluation:

After diagnosingAcutePancreatitis based on the clinicalpresentation, biochemicalparameters, andultrasonography,patientsweresubjectedtoCTscanofthe abdomenaccordingtothestandardprotocol.Theseverityofthepancreatitis was assessed byassigningpointsystembyusingModifiedCTSeverityIndexandCTSeverityIndex.

Modifiedctseverityindex:

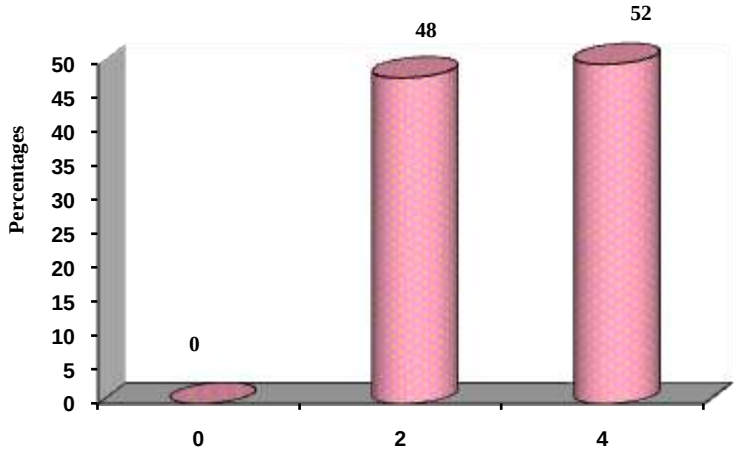
Themodifiedindexis a10pointscoringsystemderivedbyassigningpoints tothedegreeofpancreaticinflammation(0to4points)pancreaticnecrosis(0to4points)and extrapancreaticcomplications(0or2points).Allpatientsweregradedintomild(score0-3),moderate (score4-6) orsevere (score7-10).

Pancreaticinflammation:

Table 17:- Pancreatic inflammation.

Score	Number of patients	Percentage(%)
0	0	0
2	24	48
4	26	52

In ourstudy,24(48%)patientshadintrinsicpancreaticabnormalitieswithorwithoutinflammatorychangesinperipancreaticfat, towhom 2pointswereassigned.Remaining26(52%)patientshadpancreaticorperipancreaticfluidcollectionorperipancreaticfatnecr osis,to whom4pointswere assigned.(Graph-7, Table-15)



Graph:- Percentageofpatientswithscoringbasedonpancreatic.

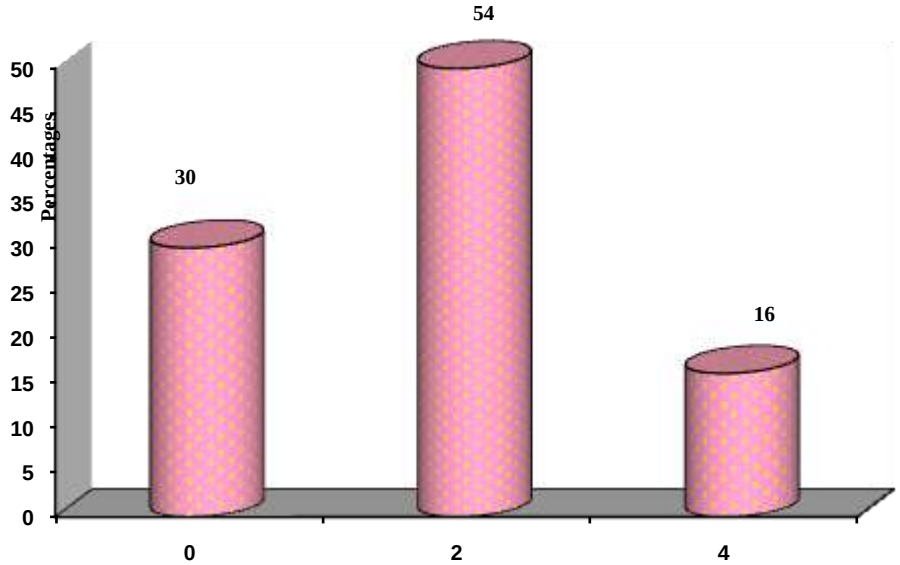
Inflammation inCT:
Pancreaticnecrosis:
Table 18:-Numberandpercentageofpatientswithscoringbasedonthepancreaticnecrosis.

Pancreatic necrosis	Number of patients	Percentage(%)
Nil (0)	15	30
<30% (2)	27	54
>30% (4)	8	16

Atotalof15(30%)patientshadnoevidenceofpancreaticnecrosisonCTscan.

27(54%)patientshadlessthan30%necrosistowhich2pointswereassigned.only8 (16%) patientshadmorethan30%necrosis, towhich4points wereassigned(table- 16, graph -8)

Pancreatic necrosis:



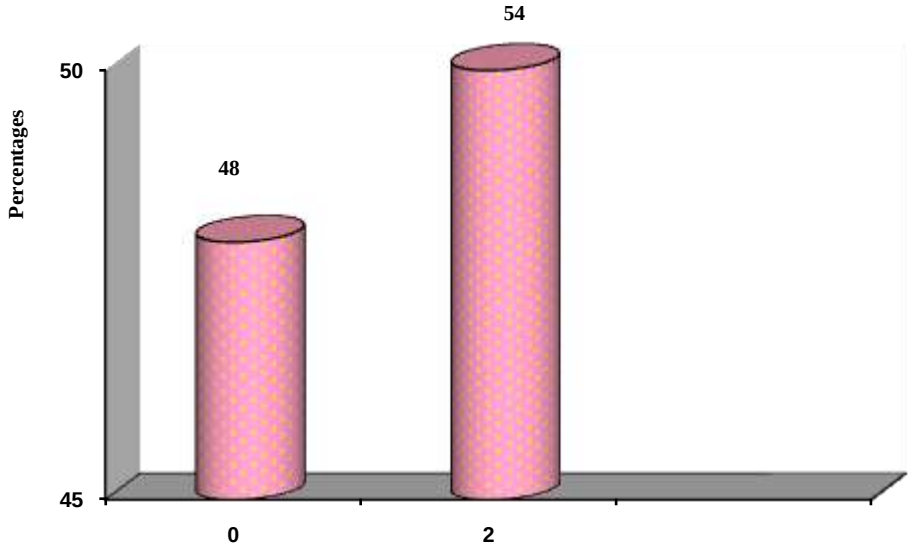
Graph 8: Percentageofpatientswithscoringbasedonthepancreaticnecrosis.

Extrapancreaticcomplications:

Table 19:- Number and percentage of patients with scoring based on theextrapancreaticcomplications.

Extra pancreatic complications	Number of patients	Percentage(%)
absent (0)	24	48
Present (2)	26	52

Inourstudy,13patients(26%)hadbothascitesandpleuraleffusion.9patients (18%)hadonlyisolatedascites.4 patientshadisolated left pleural effusion and 2 patients had isolated bilateral pleural effusion.1 patientisfoundtohavesplenicvein thrombosisand3foundtohaveportal vein thrombosis.



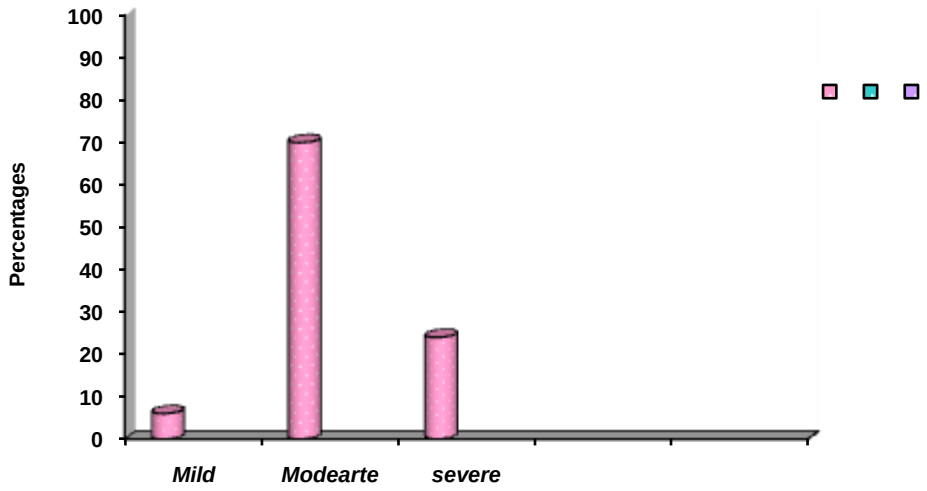
Graph 9:- Percentage of patients with extra pancreatic complicationsbased on MCTSI.

ModifiedCTSeverityindex:

Table 20:- NumberandpercentageofpatientswithgradesofseverityassessmentbasedonMCTSI.

Severity	Number of patients	Percentage(%)
Mild (0-2)	3	6
Moderate (4-6)	35	70
Severe (8-10)	12	24

ModifiedCTSeverityindexwascalculatedbyaddingpointsassignedtoeach parameter.The severity ofpancreatitisisclassifiedintothreecategories: mild (0-2 points),moderate (4-6 points)andsevere(8-10 points).According to the Modified CT Severity Index,thepatientsweregradedintomild(n=3),moderate(n=35)andsevere(n=12)i.e.6%patients hadmild,70%patients hadmoderateand24%hadsevere pancreatitis (Table-18 and Graph -10)

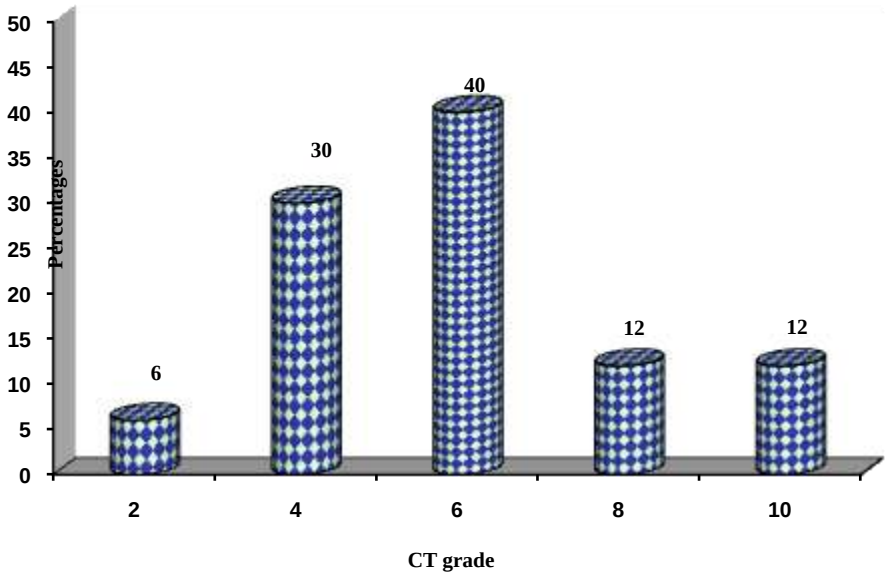


Graph 10:- Percentageofpatientswithgradesofseverityassessment.

Basedonmctsi:

Table 21:- Distribution of CT grade in patients with AP using MCTSI.

CT grade	Number of patients (n=50)	%
2	3	6
4	15	30
6	20	40
8	6	12
10	6	12



Graph 11:- Distribution of CT grade in patients with AP.

CTSeverityindex:

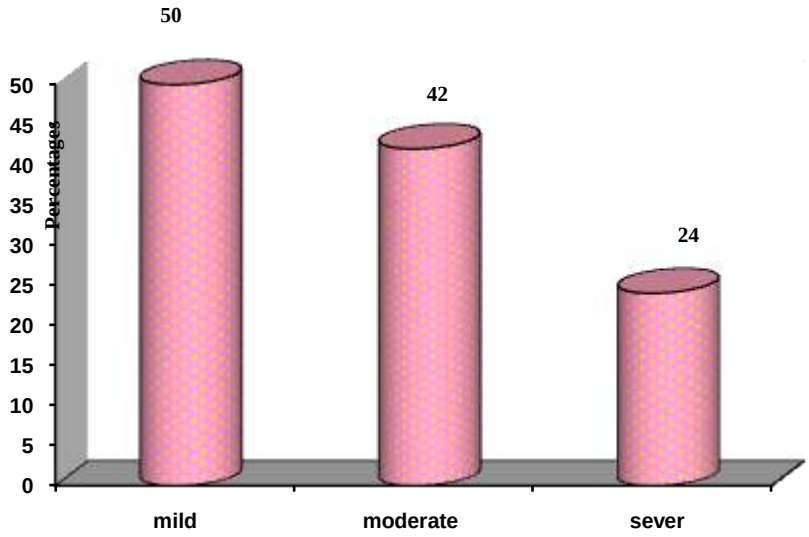
Theseverityofpancreatitisisscoredusing CTseverityindexandclassifiedintothreecategories(mild,moderateandsevere).T

heCTSIis a10pointscoringsystem derivedbyassigningpointstothedegreeofpancreaticinflammation(0to4points)and pancreaticnecrosis(0to6points)

Table 22:- NumberandpercentageofpatientswithgradesofsevarityassessmentbasedonCTSI.

Severity	Number of patients	Percentage(%)
Mild	25	50
Moderate	21	42
Severe	4	8

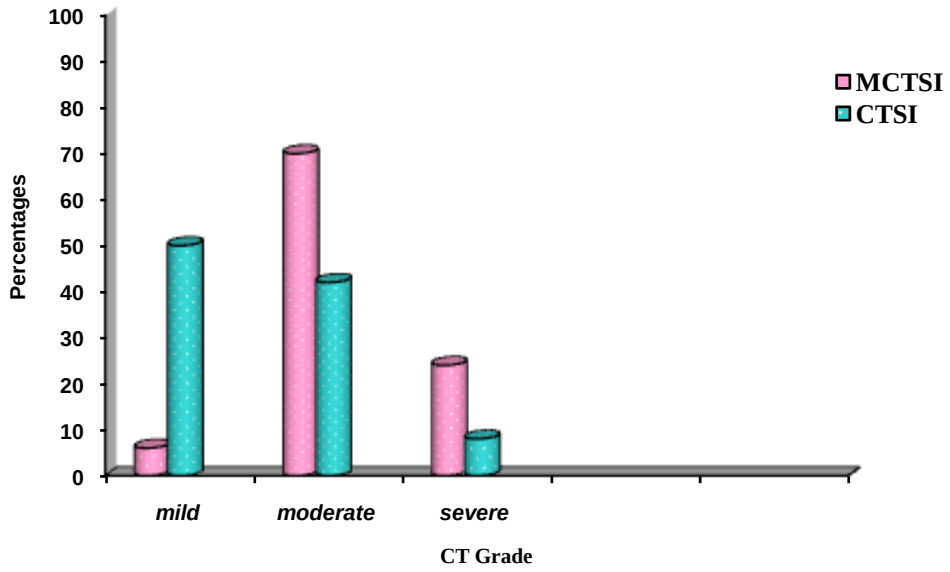
AccordingtotheCTSeverityIndex,thepatientsweregradedintomild(n=25), moderate(n=21)andsevere(n=4).50%of patientshadmild,42% of patientshadmoderateandonly 8% of patients hadseverepancreatitisas perCTSIscore(Graph-12,Table -20).



Graph12:- NumberandpercentageofpatientswithgradesofsevarityassessmentbasedonCTSI.

Comparison oftotalpatientsbelongedtoeachseverity indexes basedon MCTSI andCTSI:
Thegraph-13clearlydepictsthedistributionofpatientsineachcategory.In CTSIscoringsystem,25patients(50%)belongedtomildcategory,21(42%)patients hadmoderateandonly 4(8%) patientbelongedtoseverepancreatitis.ButinMCTSIscoringsystem 3(6%)patientsfoundtohavemildpancreatitisand35 (70%)patientsfoundtohavemoderateand12(24%)patients severepancreatitis.

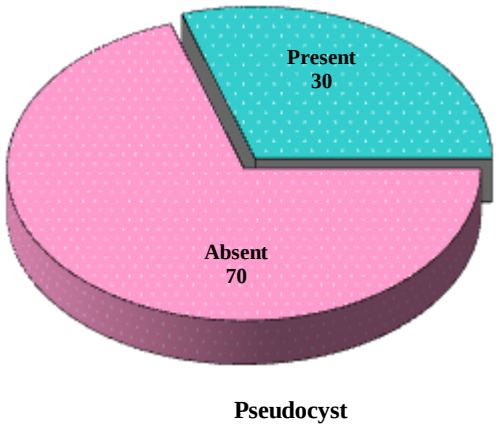
Thediscrepancyisattributedtotheinclusionofextrapancreaticcomplicationsin MCTSIscoringsystem.Hence,twoextrapoin tswereaddedtotheseverityindexinadditiontothepancreaticinflammationandnecrosis.



Graph13:- Percentage ofpatientseachseverity indices basedon MCTSI andCTSI.

Table 23:- Patients developing Pseudocyst as a consequence of AP:

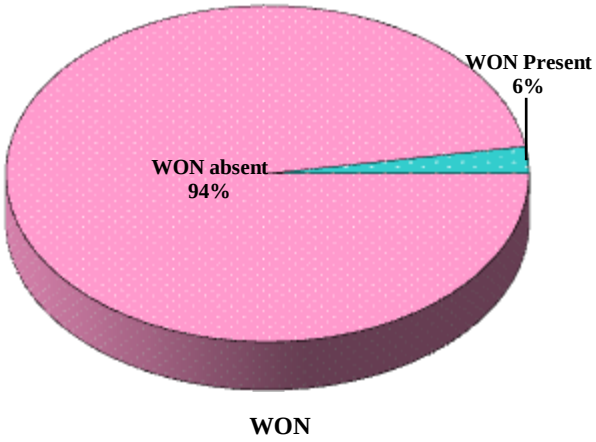
Pseudocyst	**Number of patients (n=50)**	**%**
Absent	35	70
Present	15	30



Graph 14:- Patients developing pseudocyst as a consequence of AP.

Table 24:- Patients developing walled off necrosis in AP.

Abscess	**Number of patients (n=50)**	**%**
WON absent	47	94
WON present	3	6



Graph 15:- Patients developing WON in AP.

Patientoutcomeparameters:
Clinicaloutcomeofpatientsinourstudywasassessedbythefollowingparameters: end-organfailure,evidenceofsystemicinfection,surgicalinterventionand the durationofhospitalstay.

Endorganfailure:
Patientsarefollowedupforthepresenceorabsenceofdysfunctioninsixseparateorgan systemsasdefined[60].End-organfailureisdefinedasthepresenceorabsenceofdysfunctioninanyofthesixseparateorgansystemsas defined.

Respiratoryfailure:PaO2oflessthan60mmHgorbytheneedforventilatorysupport.

Cardiovascularsystem:Systolicbloodpressureoflessthan90mmHgintheabsenceofhypovolemiawithsigns of peripheralhypoperfusionorbytheneedfora continuousinfusionofvasopressororinotropicagentstomaintain asystolicbloodpressureofmorethan90mmHg.

Centralnervoussystemfailure:GlasgowComaScalescorelesserthan6.

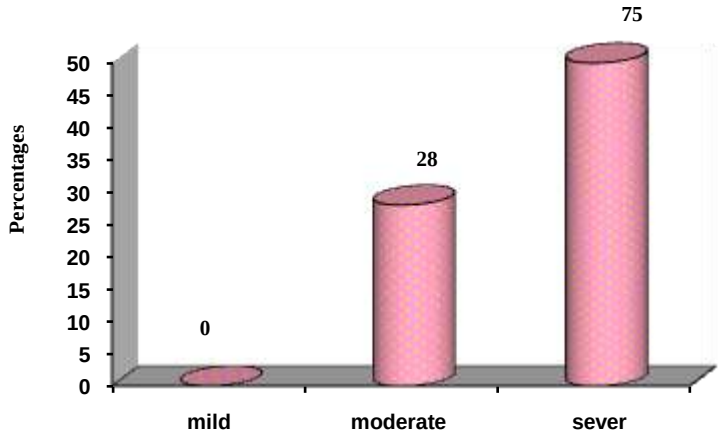
Renalfailure:Eitheraserumcreatininelevelthatexceeded300µmol/L(3mg/dL) orurineoutputofless than500mL/24hrorless than180mL/8hrorbytheneedforhemo-or peritonealdialysis.

Hepaticfailure:Serumbilirubinlevels greater than100µmol/L(3mg/dL)

Hematologicsystemfailure:Hematocritlevelofmorethan50%,WBCofless than2,000/mm3, or plateletcountofless than40,000/mm319of 50patients(38%)arefoundtohaveendorganfailure.Hepaticfailurewasseenin 11(22%)patientsisthemostcommonsystemfailureinpatientswithacute pancreatitisinourstudy. Cardiovascular systemfailureseenin4 (8%),Renalfailurein2(4%) patients.Respiratoryfailure in one patient (2%) andCNSfailureisseenin2(4%)patient.3 patientsdevelopedraiseinhematocritvalue.Of these19patientswhodevelopedend-organfailure,10 patientshadmoderateand9 patientshadseverepancreatitisaccordingtotheMCTSI,end-organ failureisseenin28%and75%of patientswhohadmoderateandseverepancreatitisrespectively(p=0.002) (Table-22, Graph-16).

Table 25: Numberandpercentageofpatientswho developed EOF basedonMCTSI.

Severity	Number of patients	Number of patients who developed EOF	Percentage(%)
Mild	3	0	0
Moderate	35	10	28
Severe	12	9	75



Graph 16:- Percentageofpatientswho developed EOF basedon MCTSI.

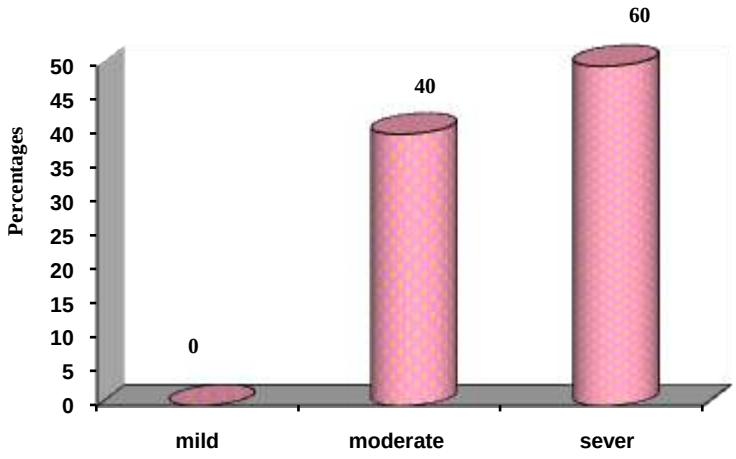
AccordingtoCTSI, of these19patients,5patientshadmild,11patientshadmoderate and3patients hadseverepancreatitis. 26% ofpatientswhohadmildpancreatitishad end-organfailure,whereasendorganfailureisseenin 57%and15%of patientswho had moderateandseverepancreatitisrespectively(p=0.012). Theabovestatisticsshowthathighlysignificantcorrelationexistsbetweenthepredictionofend-organfailurewiththeclassificationaccordingtotheMCTSI(p=0.002)thanCTSI(p=0.012).

Systemic infection:

Atotalof15(36%)patientswhohadfeverandleukocytosiswereconsideredtohave systemic infection. Of these 15patients, none of them hadmild, 9 patientshadmoderateandpatientshadseverepancreatitisaccordingtotheMCTSI.systemicinfectionisseenin 40% and 60% of patients who had moderate and severe pancreatitis respectively.(p=p=0.001)(Graph-17,Table-23)

Table 26: Numberandpercentageofpatientswho developed systemic infections basedon MCTSI.

Severity	Number of patients	Number of patients who developed systemic infections	Percentage(%)
Mild	3	0	0
Moderate	35	9	25.7
Severe	12	10	83.3



Graph 17:- Percentageofpatientswho developed systemic infections basedon MCTSI.

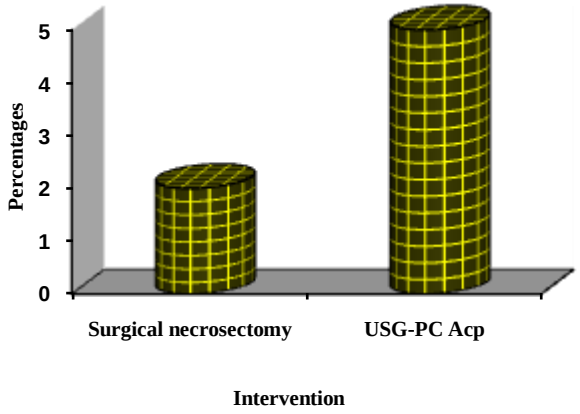
AccordingtoCTSI, of these15patients,6patientshadmild,7patientshadmoderate and2patients hadseverepancreatitis. 40% ofpatients who had mild pancreatitishadsystemicinfection,whereassystemicinfectionwasseenin46%and 13%ofpatientswhohadmoderateandseverepancreatitisrespectively(p=0.172)(Figure-21).

Withabovestatisticalvalues,itcanbeconcludedthatthereisahighly significant correlationbetweenthepredictionofsystemicinfectionwiththeclassificationaccording totheMCTSI(P=0.001),compared to the classification accordingtoCTSIwhichisnotstatisticallysignificant(p=0.172).

Surgicalintervention:
Surgical intervention was required in 5 (10%) patients. Four patients or USG guided aspiration had pseudocyst. One patient underwent surgical necrosectomy. The statistical p-value is not significant. (table- 24 and graph-18).

Table 27:- Numberandpercentageofpatientswho needed Intervention in AP:

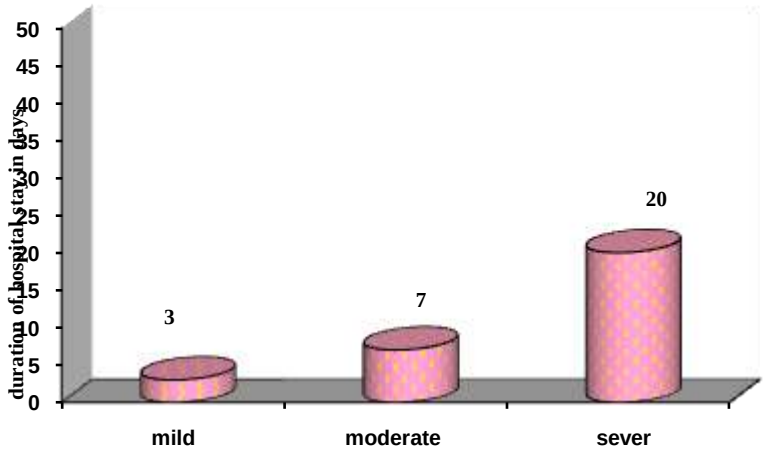
Intervention	**Number of patients (n=50)**	**%**
Surgical necrosectomy	1	2
USG - PC Aspiration	4	8



Graph 18:- Percentageofpatientswho needed Intervention in AP.

Durationofhospital stay:
Durationofhospitalstayinourstudywasrangingfrom 3to25dayswitha mean durationof 9.5days.Themeandurationofhospitalizationinmild,moderateandseverereclassesofAcutePancreatitisaccordingtoModifiedCTSeverityIndexwas3,7and20 days respectively. (Graph-19)Whereasitwas 6,12and17daysrespectivelyas pertheCTSeverityIndex.

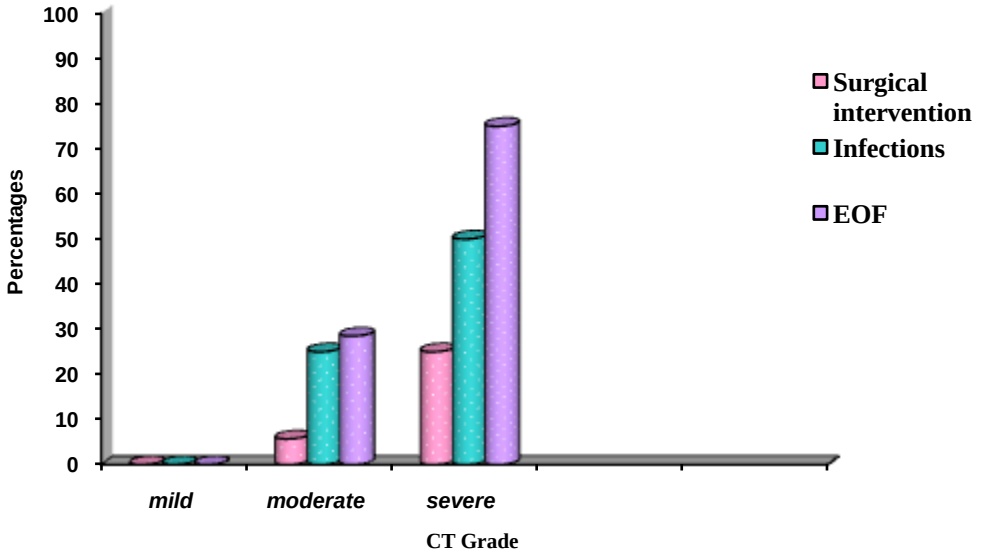
TheabovevaluesshowthatmeandurationofhospitalizationcorrelateswellwiththeseverityclassificationbasedontheMCTSI.



Graph 19:- Mean Durationofhospitalizationinseveritybasedon MCTSI.

Table28:- PatientoutcomesanddurationofhospitalizationinseveritybasedonModifiedCT Severity Index.

MCTSI	MILD	MODERATE	SEVERE
Totalnumberofpatients	3	35	12
Meandurationofhospitalization(indays)	3	7	20
Surgicalintervention	0 (0%)	2 (5.7%)	3(25%)
Infection	0 (0%)	9 (25%)	6(50%)
End-organfailure	0 (0%)	10 (28.5%)	9 (75%)



Graph 20: Patientoutcomesbasedon MCTSI

Mortalityrate:
The mortalityrateinourstudywas 0%.

Table 29:- Accuracy of CT grading in predicting systemic complications with EOF.

	Systemic complication absent	Systemic complication present	Total
CT grade(8-10)	3	9	12
CT grade < 8	28	10	38
Total	31	19	50

Sensitivity = True positive / True positive + False negative
Specificity = True negative / True negative + False positive
Positive predictive value = True positive / Total positives
Negative predictive value = True negative / Total negatives
Sensitivity = 100%
Specificity = 93.7%
Positive predictive value = 38.29%
Negative predictive value = 100%

Table 30:- Accuracy of CT grading in predicting local complications.

	Local complication absent	Local complication present	Total
CT grade moderate and severe	29	18	47
CT grade mild	3	0	3
Total	32	18	50

Sensitivity = 100
Specificity = 90.3%
Positive predictive value = 75%
Negative predictive value = 73.6%

Discussion:-

This was a prospective study conducted from November 2016 to April 2018 in Mysore medical college and research institute.50 cases diagnosed as acute pancreatitis based on history and serum amylase and lipase levels were included in this study. These patients underwent CECT of the abdomen and pelvis and were graded according to the modified CT severity index. The grades were correlated with patient outcome in terms of systemic complications leading to end-organ failure, local complications, duration of hospital stay.

Studygroup characteristics:
Ageincidence:
The mean age of patients in the study was 34.32years.Maximumnumberofpatientswas seenintheagegroupof26-35years ofthe agegroupwhichconsistedof18 (36%)patients. The age group affected reflects the etiologies of alcohol and gallstones which are common in the age group 26 to 35yrs.

Sexdistribution:
Most of the patients were male(86%)as compared to female (14%).As alcoholwasthemostcommonetiologicalfactorinourstudywhichiscommoninmales,ahigh M:F ratiowasobserved.

These observations were similar to that of a study conducted by Lankish et al [66] on 602 patients of acute pancreatitis which showed no correlationbetween age, gender with the severity of acute pancreatitis. The study also showed the maximum incidence of acute pancreatitis in the age group of 31 to 40 years similar to our study.

Table31:- Ageandsexdistributioncomparisonindifferentstudies.

Studyseries	Totalnoofpatients	Male	Female	M: F	Ageinyears
Presentstudy	50	43	7	8.6:1.4	15-66 (mean-34)
Morteleetal[71]	66	37	29	1.2:7	19-87 (mean-53)
Bollenetal[72]	179	107	89	1.2:1	21-91 (mean-45)
Freenyetal[75]	34	26	8	3.2:1	31-71 (mean-56)
Bollenetal[73]	150	84	66	1.2:1	21-91(mean-54)
Jaureguietal[74]	30	19	11	1.7:1	18-82 (mean-45)

Etiology:

Chronic alcohol abuseisthemostcommonetiologicalfactorinourstudyconstituting76%ofcases.SimilarresultswereobservedbyDugernier TL[74]andFreenyetal[73].Incontrary,studiesdonebyBollenTLetal[63]andJaureguietal[72]showedbiliarystones as thepredominantetiologicalagent.

Table32:- Etiologicalfactorscomparisonindifferentstudies.

Studyseries	Biliary stones	Alcoholabuse	Miscellaneous	
Presentstudy	16%	76%	8%	
Bollenetal[63]	34%	22%	44%	
DugernierTL[74]	22%	60%	-	
Freenyetal[73]	20%	35%	-	
Jaureguietal[72]	53%	27%	-	

Assessment of severityofacutepancreatitis:

The CT grades were classified into 2, 4, 6, 8 and 10 according to the MCTSI. We further classified the grades into mild (grade 2), moderate (grade 4 and 6) and severe (grade 8 & 10). The previous studies by Bollen et al [47] and Mortele et al [46] have classified grade 2 as mild,grade 4 and 6 as moderate and grade 8 and 10 as severe similar to our study.

The maximum patients were seen to fall in the grade 6 category (40%) and minimum patients (6%) were seen in grade 2 category. Similarly, most of the patients were of moderate CT severity (70%) and minimum patients had a mild grade (6%). Severe pancreatitis was present in 24 percent of patients. According to the study by Bollen et al [47], the morphologic severity of pancreatitis was graded as mild in 86 (44%), moderate in 75 (38%), and severe in 35 (18%) cases. In contrast to our study, their study had patients with severe pancreatitis as the minimum number of patients. Fewer patients in the mild grade in our study may possibly be explained by decreased use of CECT in mild cases of AP as CECT in not indicated in mild forms unless the diagnosis of AP is by itself doubtful or development of complications is suspected.

Most of the patients needed ward stay ranging from 2 to 25 days. 3 days was the mean stay in mild grade, 6 days was the mean duration of stay in moderate grade and 20 days was the mean stay in the severe grade of pancreatitis.

The most common segment of the total duration of hospital stay was from 8 to 14 days (41%). A strong correlation was seen between patient's CT grade and total duration of hospital stay. A study by Mortele et al [46] (published in 2004) showed a significant correlation between

MCTSI grade of pancreatitis and length of hospital stay (3 days for mild pancreatitis, 8 days for moderate and 12 days for severe grades). The differences between both the studies regarding the number of days of stay may be due to the differences in protocols regarding management in the individual hospital, the preferences of the treating doctors and the current standards and advances in the management of acute pancreatitis.

The local complications identified in the study were pseudocysts and abscess formation. Pseudocyst was seen in 15 patients (30%) in our study.Pseudocyst formation occurred in 50% of patients in a study conducted by Gonzalez et al [53].WOPN was detected in 3 patient (6%). The total percentage of patients developing local complications in the study was 36%.Presence of local complications was positively associated with CT grading. No local complications were seen in patients with mild pancreatitis. About 35 % of patients with moderate pancreatitis and 60 % of patients with severe pancreatitis had developed local complications.

In our study intervention was needed in form of surgical debridement in one patient with grade 10 of AP. Radiological intervention was needed in 4 patients(5%). Aspiration of pseudocyst was needed in 4 patient with the severe grade of pancreatitis. Thus patients who need an intervention have more severe CT grades. This is similar to the study by Bollen et al [47] which demonstrated that development of local complications and the need for intervention was significantly associated with grade of pancreatitis.

19of 50patients(38%)arefoundtohaveendorganfailure.Ofthese19patientswhodevelopedend-organfailure,10 patientshadmoderateand9 patientshadseverepancreatitisaccordingtotheMCTSI,end-organ failureisseenin28%and75%of patientswhohadmoderateandseverepancreatitisrespectively.A significant association was noted between the development of systemic complications and grading of AP by Bollen et al [47].

Atotalof15(36%)patientsdevelopedsystemicinfection. Of these 15patients, none of them hadmild, 9 patientshadmoderateand 6 patients hadseverepancreatitisaccordingtotheMCTSI.systemicinfectionisseenin 40% and 60% moderate andseverepancreatitisrespectively.

No mortality due to pancreatitis was observed in our study. In the study by Bollen et al mortality was seen in 6% of patients and in 1.5 % of patients in the study by Mortele et al.

The accuracy of MCTSI in predicting the systemic complications were as follows: Sensitivity = 100%, Specificity = 93.8%, Positive predictive value 38.2 %, Negative predictive value = 100%.

The accuracy of MCTSI in predicting the local complications were as follows: Sensitivity = 100%, Specificity = 90.32%, Positive predictive value = 75%, Negative predictive value = 73.6%.

Table 33:- Comparisontablebetweenthepresentstudyandothersstudies.

Studyseries		**Ourstudy**	**Morteleetal[60]**	**Bollenetal[63]**
Totalnoofpatients		50	66	196
MCTSI	**Mild**	3 (6%)	34 (52%)	86 (44%)
	Moderate	35 (70%)	22 (33%)	75 (38%)
	Severe	12 (24%)	10 (15%)	35 (18%)
	Mild	25 (50%)	42 (63%)	136 (69%)

CTSI	**Moderate**	21 (42%)	19 (28%)	41 (21%)
	Severe	4 (8%)	5 (9%)	19 (10%)
Duration of hospital stay indays		3-25(mean-9.5)	0-34(mean-7)	0-113 (mean-6)
Surgicalintervention		5 (10%)	10 (15%)	19 (10%)
Infection		15 (35%)	21 (32%)	7 (4%)
End-organfailure		19(38%)	9 (14%)	38 (19%)
Death		0 (0%)	2 (30%)	11 (6%)

The MCSTI accurately correlated with pancreatic inflammation and the need for intervention compared with APACHE II. CT is the modality of choice for detecting the local complications. Thus the MCTSI is as useful as APACHE II in predicting the severity of AP in terms of the number of days of hospital stay and organ failure and is better than APACHE II in detecting the local complications and confirming necrosis in AP. But MCTSI has a disadvantage that the study cannot be carried out within 48 hrs as this is the time taken to demonstrate necrosis. However, APACHE II score can be calculated even at the time of admission.

Recommendations on the basis of observations from our study:

1. We would propose that APACHE II can be used in early stages of AP and be supplemented by MCTSI which can be used after 48 hrs after admission to further manage the patients.
2. The grading of AP can be classified as mild (grade 2 and grade 4), moderate (grade 6) and severe (grade 8 and 10) contrary to other previous studies which classified it into mild (grade 2), moderate (grade 4 and grade 6) and severe (grade 8 and 10) as grade 2 and 4 patients had similar outcome.
3. Patients who have a severe grade of AP should be transferred to a center which has the facility of ICU if needed.
4. As patients with the moderate and severe grade of AP have a higher possibility of local complications a follow-up study with ultrasound / CT may be considered in these patients.

The limitations of the study are as follows:

1. The small sample size.
2. Nonrandomized study.
3. Patients with mild acute pancreatitis could not be included in the present study as a CT scan is not indicated in all cases of pancreatitis

Summary:

This was a prospective study of 50 cases of clinically diagnosed acute pancreatitis and confirmed by serum amylase and lipase levels. CT severity index and Modified CT severity index grading were done after contrast-enhanced CT of abdomen and pelvis in all patients. Correlation of CT severity index and modified CT severity index grades was done with patient outcome taking local and systemic complicationsand duration of hospital stay as parameters.

1. The age group of patients was 15 to 66 years with maximum patients (36%) between 26 and 30 years.
2. 86% of patients were male.Maletofemaleratiowas 8.6:1.4withmalepreponderance.
3. Chronicalcoholabusewasthemostcommoncauseofpancreatitis(76%),thesecondwas gallstones (16%) andothers(8%)
4. Allpatients presentedwithpainabdomen.92%hadvomiting,30% of patients hadfeverand 10% ofpatientshadjaundiceatthetimeofpresentation.
5. Amylasewaselevatedin86%patients.Lipasewaselevatedin90%patients.
6. 86%of patientshadfeaturesofpancreatitisonultrasoundandin14%ultrasoundwas normal.

7. Pancreaticinflammationwasseenin 100% ofpatients. 30% of patientshadnoevidenceofpancreaticnecrosisonCTscan. 54%ofpatients hadlessthan30%necrosis andonly16%hadmorethan30%necrosis.
8. 48%of patientshadnoevidenceofextrapancreaticcomplications.52%of patients hadoneormoreextrapancreaticcomplications.
9. Accordingtothe ModifiedCTSeverityIndex,6% of patients hadmild,70%of patients had moderateand24%hadseverepancreatitis.
10. Durationof hospital stayrangedfrom3to25days with a meandurationof9.5days.
11. Mortalityratewas 0%.
12. 38%of patientsareconsideredtohaveendorganfailure.Hepaticfailureisthemostcommonsystemfailureseenin22%patients.36 %of patients hadevidenceofsystemicinfection.10% of patients requiredsurgicalinterventions.

Conclusion:-

1. Grading by modified CT severity index has a significant correlation with the necessity of ICU admission, duration of ICU stay and total duration of hospital stay.
2. Modified CT grading correlates directly with the development of local and systemic complications.
3. Modified CT severity index can be used to predict the possibility of developing local and systemic complications and necessity of ICU admission.
4. Modified CT severity index can predict the need for interventions.
5. ExtrapancreaticcomplicationswhenincludedintheCTscoringsystem(MCTSI) weresignificantlycorrelatedwithend-organfailureandadverseclinicaloutcome. HenceMCTSImaybemoreusefulscoringsystemthanCTSI.
6. MCTSIisaveryusefultoolforthescreeningofpatientswithacutepancreatitisfor theclassificationofseverityaccuratelyandtopredicttheclinicaloutcomewhenusedwithinthreedaysofsymptomonset.

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