"ROLE OF MULTI DETECTOR COMPUTED TOMOGRAPHY IN EVALUATION OF PANCREATIC PATHOLOGIES"

By

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Dissertation submitted to the

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In partial fulfillment of the requirements for the degree of

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In

RADIO-DIAGNOSIS

Under the guidance of

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ABSTRACT

BACKGROUND & OBJECTIVES

Most commonpancreatic pathologiesincludes pancreatitis and over past few years pancreatic neoplasms are increasing at alarming rates. Due to its retroperitoneallocation & anatomical relationship with bowel and major blood vessels, the pancreas pose a challenge to Radiologists in its imaging. Ultrasound is used as a screening modality but has its limitations ofbeing observer dependent, limited evaluation in obese & gas filled bowel loop conditions.With recent advances in imaging, currentlyMulti detector Computed tomography(MDCT) is beingused as initial imaging modality of choice for evaluation of pancreatic pathology.

Phase contrast studies using specific protocols helpedin improving the diagnostic accuracy for detection and characterizing of the pancreatic lesions. MDCT with its ability to acquire images at rapidrate and improved spatial resolution,have increased the accuracy of lesion detection as small as 2cm in a short scan duration. MDCT with triple phase contrast study using reconstruction techniques is a noninvasive technique that helps in excellent visualization, better characterization of pancreatic pathologies through phase wise study and assessment of lymph nodes, vascular involvement, distant metastasis and the resectability of a pancreatic tumour.

This study is undertaken to study various inflammatory, neoplastic & traumatic pathologies of pancreas along with extra pancreatic complications and difference in attenuation values of different pathologies using MDCT triple phase protocol.

AIMS & OBJECTIVES OF THE STUDY:

To assess role of MDCTwithtriple phase contrast study in:

1. The evaluation and characterisation of various inflammatory, neoplastic and traumatic pancreatic lesions.
2. To measure and assess the attenuation values (Hounsfield Unit) of various pathologies.
3. To classify pancreatic tumours based on imaging findings and correlate them pathologically where ever possible.
4. To evaluate pancreatic trauma cases and grade them as per AAST classification.

SOURCE OF DATA:

Data for the study is collected from the patients attending/referred to the Radiology department ofB.L.D.E.(DEEMED TO BE UNIVERSITY) Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur who fulfill the inclusion criteria.

METHOD OF COLLECTION OF DATA:

The study was done on patients, who visited the Department of Radio Diagnosis during the period fromNOVEMBER 2016 to AUGUST2018 with prior consent.

RESULT: In our study series of 78 cases, we got 36 cases of acute pancreatitis (17 – interstitial oedematous& 19 – necrotizing pancreatitis), 16 cases of acute on chronic pancreatitis, 10 cases of chronic pancreatitis, 14 cases of neoplasms and 2 cases of pancreatic trauma. Majority of the pancreatic pathologies included pancreatitis (79.5%) with preponderance in males. Among neoplasms, malignant were more common and adenocarcinoma in particular with more preponderance in females.

INTERPRETATION:MDCT with triple phase imaging protocol of pancreas helps in better evaluation of various pancreatic pathologies with phase wise characterization, detection of lesions as small as 1.5cm in size and assessment of resectability of a neoplastic lesion. Thus aiding in better, accurate diagnosis of pathologies and in further treatment planning.

.TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Pancreatic pathologies are now an increasingly common occurrence and a significant cause of morbidity and mortality. They may present with an array of symptoms while some are asymptomatic for long periods and others often encountered as incidental findings on evaluation for other pathologies.

Pancreatitis is one of the most complex and clinically challenging of all abdominal disorders.[1] Common aetiology includes alcohol consumption, gallstones, smoking, post ERCP, family history etc.

Globally as quoted by WHO in Global Burden of Disease Study 2013, about 17 million cases of pancreatitis occurred.[2] This resulted in 123,000 deaths, up from 83,000 deaths in 1990.[3]

Pancreatic neoplasm, another important group of pancreatic pathologies, which is the fifth most common cause of cancer deaths and accounts for approximately 3% of all cancers, has shown marked increase in its incidence worldwide[4].

Due to the increasing incidence and the myriad ways it has become necessary to evaluate imaging modalities that can help in early detection and in the proper evaluation and characterization of each of these lesions.

Modalities for imaging pancreas range from plain x-ray to Ultrasonography (USG), Endoscopic ultrasound (EUS), Endoscopic retrograde cholangio-pancreaticography (ERCP), Computed tomography (CT), Magnetic resonance imaging (MRI) and Positron emission tomography - computed tomography (PET-CT).

With the introduction of Multidetector Computed Tomography (MDCT), evaluation of pancreatic lesions allows data to be acquired during optimal pancreatic enhancement. The advent of triple phase contrast study aids in early detection of small and early pancreatic lesions. This technology permits thinner slices to be acquired during multiphasic scanning, with improved spatial resolution.[5] The use of multi planar reformatted images and 3-dimensional representations of the vascular structures as well as the ability to provide preoperative vascular mapping, helps in accurate staging of pancreatic tumors and aids in successful surgical resection.[6]

Since MDCT technologic advances facilitate early detection of small pancreatic lesions, they are likely to have an impact on the treatment of pancreatic diseases. Hence this study aims to assess the role of MDCT (triple phase study) in evaluation of pancreatic diseases.

AIMS AND OBJECTIVES

To assess role of Multi detector Computed Tomography - triple phase contrast study in:

1. The evaluation and characterisation of various inflammatory, neoplastic and traumatic pancreatic lesions.
2. To measure and assess the attenuation values (Hounsfield Unit) of various pathologies.
3. To classify pancreatic tumours based on imaging findings and correlate them pathologically where ever possible.
4. To evaluate pancreatic trauma cases and grade them as per AAST classification.

METHODOLOGY

This study evaluating the Role of MDCT- triple phase in the pancreatic pathologies was done on 78 cases. This study was conducted during the period from NOVEMBER 2016 to AUGUST 2018 in Radiology department B.L.D.E.U's Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

Source of Data:

The source of data for this study are patients from B.L.D.E.U's Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

Sample size:

A minimum sample size of 50 subjects will allow the study to determine the incidence of pancreatic lesions with a confidence interval of +/- 10% with finite population correction.

$$n = \frac{Z^2\, p(1- p)}{d^2}$$

Z = statistic at 5% level of significance

d is margin of error

p is expected prevalence rate

Statistical analysis:

Statistical analysis was done with the help of software package used for statistical analysis (SPSS 13). All the studies with $p<0.05$ are considered statistically significant and those with $p>0.05$ are considered statistically insignificant using Chi square test, ANOVA test and Kruskal Wallis test.

METHOD OF COLLECTION OF DATA:

Male and female patients of all ages who are referred to Department of Radiodiagnosis , Shri B.M. Patil Medical College Hospital and Research Center with the clinically suspected / diagnosed cases of pancreatic pathologies are selected based on the inclusion and exclusion criteria as study subjects. Total 78 subjects were recruited for the study.

Following are the inclusion criteria:

1. Patients presenting with suspicious clinical symptoms of pancreatic pathology and with associated biochemical parameters such as elevated amylase ,lipase levels.
2. Patients with suspicious or definite findings on ultrasound of pancreatic pathology.

Following are the exclusion criteria:

1. Patients with non pancreatic causes of upper abdominal pain.
2. Pregnant patients.
3. Patient suspected of congenital anomalies of pancreas.
4. Patients with renal insufficiency or elevated urea, creatinine levels that can be exacerbated by contrast and contrast allergy used for enhanced CT.

CONSENT:

Informed consent will be taken from all patients who will be selected on the basis of

1. Clinical symptoms and biochemical tests suggestive of pancreatic disease
2. Findings of pancreatic pathology on other imaging modalities.

PREPARATION OF PATIENT:

- All patients are ideally required with at least 6 hours of fasting before scan.
- However in acute cases ryle’s tube is used to empty the stomach.

CONTRAST:

- All patients received 100-120 ml of IV non iodine contrast with a monophasic injection technique by means of a power injector.
- The contrast material is administrated at a rate of 4 ml/s through antecubital vein.

TECHNIQUE:

- MDCT (triple phase study) will be performed on MDCT scanner (Siemen's 32 slice CT unit).
- The patient will be placed on gantry table in supine position with both arms above the head.
- All scans will be acquired in a cephalocaudal direction. A digitized AP scanogram will be obtained in suspended respiration. Non enhanced sections will be obtained throughout the abdomen.
- The AP scanogram is utilized to determine the superior extent of scan i.e. the dome of diaphragm and the inferior extent i.e. pubic symphysis.
- Before contrast injection the patient is asked to hyperventilate so that blood oxygen level would be high and hence they would be comfortable in holding their breath.
- Continuous 1.5mm thick slices were obtained in axial plane with a scan time of 6seconds at a 130KV tube voltage and 170 mA.

TRIPLE PHASE STUDY:

- Contrast scan is obtained in three phases after obtaining unenhanced MDCT followed by arterial phase (AP), pancreatic parenchymal phase (PPP), and portal venous phase (PVP).
- Arterial phase acquisition is initiated using "Smart Prep" bolus tracking with the ROI at descending aorta above the dome of diaphragm, once the vessel threshold

crosses 100HU the image acquisition is initiated with the delay of 8-10sec. The images were obtained from dome of diaphragm to abdominal aortic bifurcation.

- Pancreatic parenchymal phase acquisition is initiated following the arterial phase with a total delay of 18-20sec.
- Portal venous phase acquisition is initiated following the pancreatic parenchymal phase with a total delay of 40-50sec.
- The patient is instructed to hold and release the breath in between the phases.
- After the MDCT examinations, coronal and sagittal MPR images can also be created, using the data from the axial images, for reconstructed MPR images using a 1.0mm thickness interval.

REVIEW OF LITERATURE

BRIEF HISTORICAL BACKGROUND[7]

The pancreas has its first mention between 200 BC and 200 AD, and it was Talmund who labelled it as the “finger of the liver” in his literature. Galen (Claudius Galenus) gave the name pancreas, who thought its main function was for the support and protection of blood vessels. Vesalius considered pancreas as a stomach cushion.

In 1962 the pancreatic ducts of humans was first demonstrated by Wirsung. Almost 200 years after the discovery of pancreas the digestive capability of its secretions was discovered, later the emulsification of fat by Eberle in 1834, proteolytic activity by Purkinje and Pappenheim in 1836, digestion of starch by Valentin in 1844 were observed from pancreatic juice and its extracts. Bernard using the secretions from pancreatic fistula preparations demonstrated the digestive action of pancreatic juice on sugar, fats, and proteins.

In 1876, the term enzyme and isolated trypsin was introduced Kuhne which led to the identification of pancreatic amylase and lipase. An essential component for activation of the proteolytic enzymes was discovered in 1889 by Chepovalnikoff, a student of Pavlov in the duodenal mucosa and named it enterokinase. Stimulation of pancreatic secretion by instilling acid into the duodenum was done by Dolinsky in 1895. Later it was Bayliss and Starling that discovered secretin, which was the first hormone to be identified but not an enzyme.

In 1869, it was Langerhans who first described the histologic structure of the pancreas and then by Heidenhain. gave the pancreas its name. In Greek pancreas when translated meant whole flesh and was labelled so by Rufus of Ephesus, another Greek anatomist, possibly due to the fleshy consistency.

EMBRYOLOGY OF PANCREAS[8]

The pancreas is derived from the endoderm of the embryonic foregut, with the formation of a ventral and dorsal bud the pancreatic development begins. The ventral and dorsal bud communicates with the foregut through a duct. The ventral pancreatic bud becomes the head and uncinate process, and forms the hepatic diverticulum. The definitive pancreas is formed by the differential rotation and fusion of the ventral and dorsal pancreatic buds (Fig-1).

The pancreas is a soft, elongated, flattened gland 12 to 20 cm in length lying in the epigastrium and left hypochondrium areas of the abdomen composed of the following parts.[9]

- The head of the pancreas lies within the concavity of the duodenum.
- The uncinate process emerges from the lower part of head, and lies deep to superior mesenteric vessels.
- The neck of the pancreas is the constricted part between the head and the body.
- The body lies behind the stomach.
- The tail is the left end of the pancreas. It lies in contact with the spleen and runs in the lienorenal ligament.

The duct system of the pancreas is established as follows:

- The main pancreatic duct is formed by the duct of dorsal bud distally and in its proximal part by the duct of ventral bud.
- The accessory pancreatic duct is formed from the narrowed proximal part of the duct of the dorsal bud.

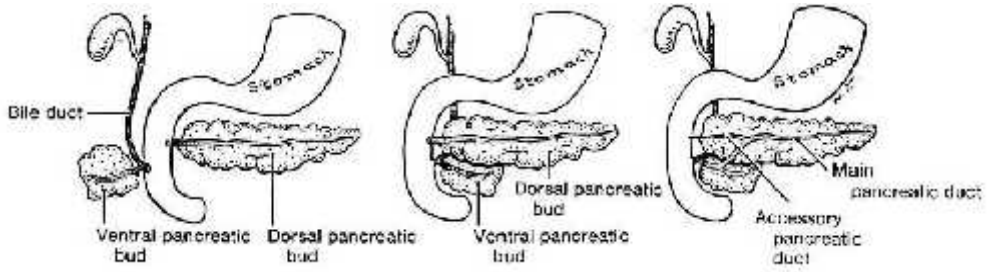


Figure 1 – Development of Pancreas

CT ANATOMY [10,11]

The pancreas is an exocrine and endocrine organ situated retroperitoneal in the epigastrium and left hypochondrium measuring approximately 15cm in length and weighing 60 - 100gms. It is descriptively divided into four parts viz the head, neck, body and tail.

LOCATION

It is situated in the anterior pararenal space (the most ventral of the three retroperitoneal compartments), which is defined ventrally by the posterior parietal peritoneum and dorsally by the anterior renal (Gerota's) fascia.

THE HEAD OF THE PANCREAS

It is the broad right end of the gland lying within the curve of the duodenum to the right of the superior mesenteric vein (SMV). It lies anterior to the inferior vena cavaseparated from it by a thin distinct plane of fat. The uncinate process is a curved beaklike inferior and medial extension of the head that originates lateral to the SMV and curves posteriorly behind it, approximately at the level of the left renal vein.

THE NECK OF THE PANCREAS

It is the narrowest portion and lies anterior to the superior mesenteric artery (SMA) and varies in thickness.

THE BODY AND TAIL

They lie in an oblique orientation, extending from the hilum of the spleen towards the midline of the body, passing anterior to the confluence of the superior mesenteric vein and splenic vein to form the portal vein. The body arches anteriorly over the superior mesenteric artery close to its origin, and is separated from it by a distinct fat plane. The body lies behind the lesser sac (omental bursa) and stomach and its dorsal surface is indented by the splenic vein.

PERITONEAL CONNECTIONS

The omental bursa is the potential space between the stomach and the pancreas. It is only seen when filled with fluid. The transverse mesocolon is formed by the fusion of the parietal peritoneal leaves. It extends anteriorly from the ventral surface of the pancreas along its entire length. These peritoneal communications serve as pathways for flow of inflammatory exudates in acute pancreatitis.

The pancreatic tail is usually at the same level or cephalic to the body and follows the splenic vessels up to the splenic hilum lie most distal part of the gland lies within the spleno-renal ligament where it becomes an intraperitioneal structure. Near the hilum, the tail lies anterior to the left adrenal gland, upper pole of the left kidney and medial portion of the spleen.

DUCTS

The main pancreatic duct (Wirsung's duct) runs the length of the pancreas and joins the common duct at the Vater's ampulla. In the head region it is seen running parallel and medial to the common bile duct ranging in diameter from 1 to 3 mm.

The accessory pancreatic duct -Santorini's duct is in the upper portion of the pancreas and is more horizontal than the Wirsung's duct.

The common bile duct (3-6 mm in diameter) is seen within the pancreatic head, close to its lateral and posterior surface, as a round or oval near water density structure.

SHAPE

Pancreas usually has a lobular morphology. The configuration of the pancreas is quite variable. Normally, there is a gentle decrease in the size of the gland as one move from the head to the tail. A common variation is a dumb - bell shaped pancreas in which the size of both the ends is equal.

SIZE

The normal antero-posterior measurements of the pancreas as best studied by Kneel and Sandvi are- Head 23 mm (±3mm), Neck 19 mm (±2.5mm), Body 20 mm (±3mm), Tail 15 mm(±2.5mm).

The size of the gland decreases with age but the ratio of the head to the body remains almost constant. The cranio - caudal measurements of the gland are in female's 4.2-7.8cms and male's 4.8-7.6cms. This measurement is important for the diagnosis of pancreas divisum in which the cranio-caudal diameter of the pancreas is increased. The pancreas lacks a true fibrous capsule. Its surface contour is smooth or lobular. Fatty replacement of the pancreatic parenchyma is seen in elderly individuals, obese patients and pathological conditions.

POSITION

The pancreatic position is very variable. The pancreatic head is not -fixed in position, though it invariably maintains a fixed relationship medial to the second part of the duodenum and lateral to the root of the superior mesenteric vessels, even if these structures are shifted to the left of the midline.

ATTENUATION

The attenuation of the pancreas is normally the same as soft tissue (30 to 50 HU). The normal pancreas increases in density after IV contrast administration. The more rapid the administration, the denser the enhancement. Normal enhancement with bolus injection can be visualized during arterial, capillary and venous phase.

BLOOD SUPPLY OF PANCREAS

The pancreas derives its blood supply from

- The superior pancreaticoduodenal artery a branch of the gastroduodenal artery
- The inferior pancreaticoduodenal artery from superior mesenteric artery
- The pancreatic branches of splenic artery the largest of those branches is called the arteria pancreatica magna.

The body and neck of the pancreas drain into splenic vein; the head drains into the superior mesenteric and portal veins.

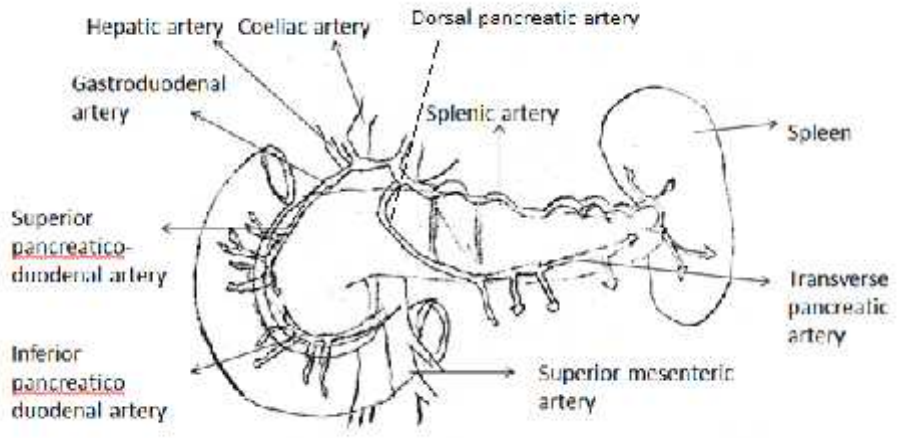


Figure 2 – Blood supply of Pancreas

LYMPHATIC DRAINAGE OF PANCREAS

Lymph is drained via the splenic, celiac and superior mesenteric lymph nodes.

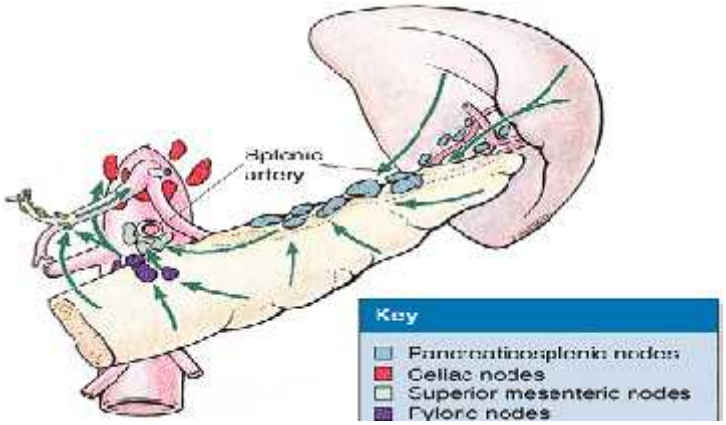


Figure 3 – Lymphatic drainage of Pancreas

PANCREATIC DUCT[9]

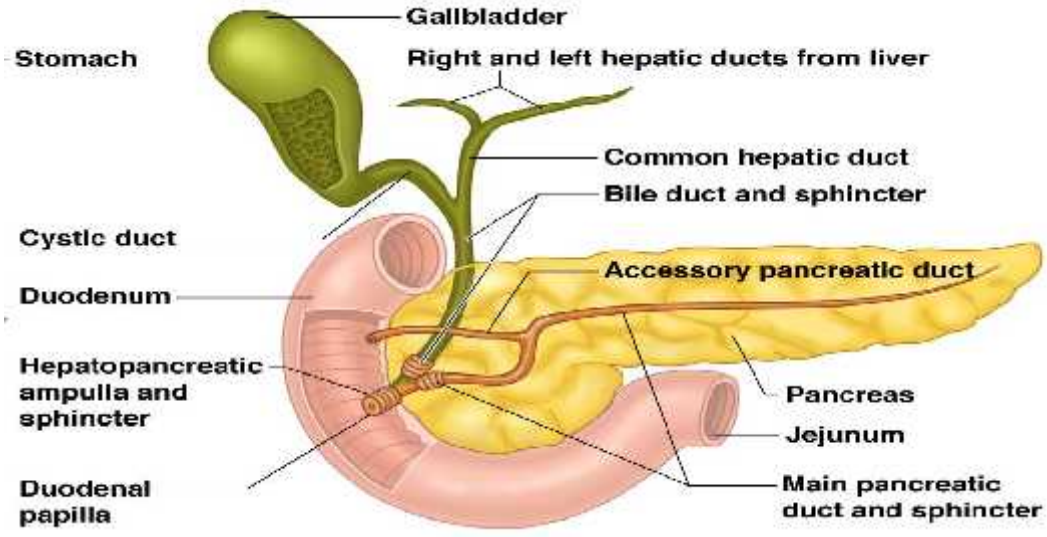


Figure 4 - Pancreatic duct system

The main pancreatic duct of Wirsung begins near the tail of the pancreas. It is formed from anastomoses of ductules draining the lobules of the gland. It passes from left to right, and joins the accessory duct i.e duct of Santorini in the head region. In the tail and body the duct lies midway and slightly posterior between the superior and inferior margins. At the level of the major papilla, the duct turns horizontally to join usually with the common bile duct. This short common segment is the ampulla of the bile duct, which terminates in the duodenal papilla and is guarded by the sphincter of Oddi. (Fig- 4). The accessory duct opens into duodenum at minor papilla.

THE PHYSIOLOGICAL FUNCTIONS OF PANCREAS [12]

The pancreas is a dual-function gland, having features of both endocrine and exocrine glands. The functional unit of the exocrine pancreas is composed of an acinus and its draining ductule. The part of the pancreas with endocrine function is made up of approximately a million cell clusters called islets of Langerhans.

There are several different types of cells (Fig-5) that comprise these Islets[10], these are:

Alpha: Produce glucagon, which raises the level of blood glucose between meals, by converting fat and protein into intermediate metabolites, which eventually are converted to glucose.

Beta: Produce insulin and amylin, which lower the level of blood glucose by inhibiting the secretion of glucagon; slows the emptying of the stomach.

Delta: Produce somatostatin, which inhibits the release of specific hormones and reduces the rate of absorption of food from the contents of the small intestine

Gamma: Produce a polypeptide, which reduces the appetite

Pancreatic Polypeptide: It is a 36 amino acid which acts as cholecystokinin antagonist. It suppresses pancreatic secretion and stimulates gastric secretion.

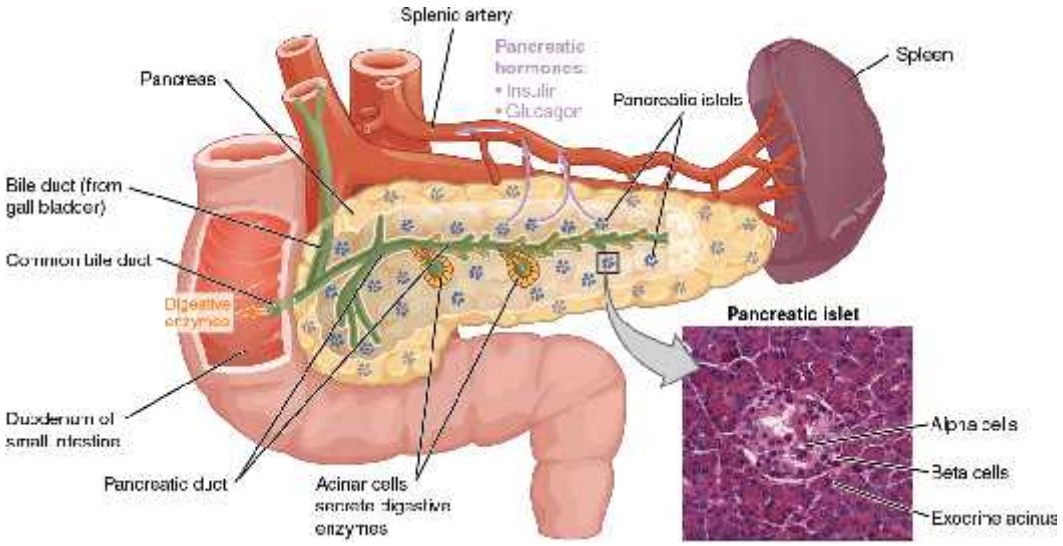


Figure 5 - Different cells of pancreas

SIGNS AND SYMPTOMS OF PANCREATIC PATHOLOGY[13,14]:

Clinical symptoms play a major role in detecting and diagnosing any pancreatic pathology. The common signs and symptoms are:

SYMPTOMS:

- Pain Abdomen
- Nausea
- Vomiting
- Weight loss
- Yellowish skin discoloration
- Anorexia
- Diarrhoea

SIGNS:

- Jaundice
- Palpable liver
- Palpable gall bladder (Courvoisier's sign)
- Cullen’s sign
- Gray Turner’s sign
- Abdominal tenderness
- Ascites
- Thrombophlebitis

LABORATORY WORK UP[13,14]

PANCREATITIS:

- Serum amylase & lipase
- Serum bilirubin
- Autoantibodies levels
- IgG &IgM levels.

PANCREATIC CARCINOMA

- A comprehensive metabolic panel that includes group of tests used for evaluation of liver and kidney function and to find the cause of jaundice.
- CA 19-9 (Cancer antigen 19-9): a tumor marker not specific for pancreatic cancer but may be used to distinguish pancreatic cancer from other cancers and to monitor recurrence. Few non-cancerous conditions can also cause elevated CA 19-9 levels. Recent research, however, suggests that it may be useful for early detection of pancreatic cancer when combined with a promising new microRNA-detecting test that is still under investigation.

- CEA (Carcinoembryonic antigen): a tumor marker used as a monitoring tool.
- Serum Amylase & lipase levels: may be elevated
- Other tests, such as fecal fat, stool trypsin & serum trypsinogen can be done to evaluate the functioning efficacy of pancreas and to assess the necessity of enzyme supplementation.

IMAGING MODALITIES:

In current scenario following imaging modalities can be used for evaluation of various pancreatic pathologies

- Ultrasound
- Multi detector Computed tomography (MDCT) with triple phase study.
- Magnetic resonance imaging (MRI)
- Magnetic resonance Cholangiopancreatography (MRCP)
- Endoscopic retrograde Cholangiopancreatography (ERCP)
- Positron emission tomography (PET)
- PET/CT - PET/MRI

VARIOUS PANCREATIC PATHOLOGIES

The pancreatic pathologies are broadly classified into five groups[11]

I. Developmental anomalies and variants
II. Inflammatory conditions
III. Tumours
IV. Infiltrative, metabolic and other disorders
V. Trauma

PANCREATIC PATHOLOGIES

Developmental anomalies and variants	**Inflammatory conditions**
A. Pancreas divisum B. Annular pancreas C. Agenesis of dorsal pancreas D. Pancreatic head lobulations E. Ectopic pancreas F. Uneven pancreatic lipomatosis	A. Acute pancreatitis B. Chronic pancreatitis C. Hereditary pancreatitis D. Groove pancreatitis E. Pancreatic tuberculosis.
Tumors (Solid/Cystic)	**Infiltrative, metabolic and other disorders**
A. Exocrine B. Endocrine. C. Mesenchymal D. Metastasis	A. Hemochromatosis B. Cystic fibrosis C. Fatty pancreas D. Heterotopic pancreas
Pancreatic trauma	

Table – 01 Various pancreatic pathologies

Above is the table (1) showing various pancreatic pathologies. Here in this study we will be dealing with following pancreatic pathologies:

1. Inflammatory pathologies
2. Pancreatic Neoplasm
3. Pancreatic trauma

INFLAMMATORY CONDITIONS

A. Acute pancreatitis

B. Chronic pancreatitis

C. Hereditary pancreatitis

D. Groove pancreatitis

E. Pancreatic tuberculosis.

A. ACUTE PANCREATITIS

Acute pancreatitis is an acute inflammatory process that is followed by complete restoration of structural and functional normalcy after the attack subsides, provided that no part of the pancreas has been destroyed by necrosis.

Causes of acute pancreatitis

- Choledocholithiasis and ethanol abuse — most common.
- Trauma
- Metabolic disorders (hyperlipidemia, hypercalcemia),
- ERCP-induced pancreatitis, medications (azathioprine, sulfonamides),
- Tumours,
- Congenital anomalies such as pancreas divisum.

Clinical features: Nausea, vomiting, abdominal pain, and rebound tenderness. Fever, tachycardia, and leukocytosis are often present.

Classification[15,16]

The 1992 International Symposium on Acute Pancreatitis classified acute pancreatitis into:

1. Mild acute (edematous or interstitial) type,
2. Severe acute (necrotizing) type.

This is not a perfect system of classification as intermediate forms of the disease do occur.

1. **Mild acute (edematous or interstitial) pancreatitis:**

Mild acute pancreatitis, also called edematous or interstitial pancreatitis, is a self-limiting disease with no or minimal organ dysfunction, no complications, a rapid response to appropriate conservative medical therapy, and prompt resolution of clinical manifestations and laboratory abnormalities, with an uneventful recovery.[18/11]

CT reveals [17,18, 19]

- Normal or minimally enlarged gland with low or heterogeneous glandular attenuation due to interstitial edema and a shaggy contour.
- The peripancreatic fat may be normal or hazy due to inflammation.
- Diffuse involvement of the gland is common; however, segmental forms, involving only the head of the pancreas, are occasionally seen.
- After contrast administration there is uniform enhancement of the gland.
- Heterogeneous low-attenuation areas in the peripancreatic space at the onset of an acute episode of pancreatitis represent a combination of fat necrosis, extravasated pancreatic fluid, nonspecific inflammation, and hemorrhage.

2. **Severe acute (necrotizing) pancreatitis:**

Severe pancreatitis occurs in 20% to 30% of all patients with acute pancreatitis. It is characterized by a protracted clinical course, multiorgan failure, and pancreatic necrosis.[20]

CT reveals [11,21,22]

- Focal or diffuse pancreatic enlargement
- Inflammation – pancreas and/or peripancreatic fat.
- Peripancreatic fluid collection.
- Necrotic areas in parenchyma
- Extrapancreatic complications like ascites, pleural effusion etc.

CT has an overall accuracy of 87%, with a sensitivity and specificity of 100% for the detection of extended pancreatic necrosis and a sensitivity of 50% if only minor necrotic areas are present.[11]

The modified CT severity index is found to have a stronger prognostic correlation than the accepted CT severity index and could also predict the length of hospital stay and development of organ failure.[22]

Clinical and CT Presentation of acute pancreatitis

1. Acute Fluid Collections.

Acute collections of enzyme-rich pancreatic juice occur in about 40% of patients early in the course of acute pancreatitis.[25]

These fluid collections may be around the gland or intrapancreatic. They lack a capsule and are confined only by the anatomic space within which they arise, most commonly the anterior pararenal space or lesser sac. They can dissect into other locations, including the mediastinum and the posterior pararenal space, and can involve solid organs (liver, spleen, kidneys) or the wall of an adjacent bowel loop.

Acute fluid collections appear hypo dense on CT; they are poorly defined with norecognizable capsule or wall, which distinguishes them from pseudocysts.[26]

2. Pseudocyst.

Pseudocysts are encapsulated, unilocular collections of pancreatic fluid and necrotic and proteinaceous material. They may occur in patients with chronic pancreatitis sometime during the course of their disease.[27,28]

It takes about 4 weeks or longer for a pseudocyst to evolve from acute pancreatic fluid collections. Pseudocysts most often are peripancreatic but can be found throughout the abdomen, as well as within the mediastinum and pelvis.

On CT, a pseudocyst appears as a round or oval fluid collection with a thin or

a thick wall that shows contrast enhancement. Gas bubbles within the pseudocyst may be due to infection, fistula formation, or internal cystotomy, and percutaneous fine-needle aspiration.[29,30]

Other complications, such as biliary and gastrointestinal tract obstruction, invasion into the spleen or liver, rupture into the peritoneum, and perforation into the gastrointestinal tract, can also be identified with CT.[17, 30]

3. Pancreatic Abscess.

Pancreatic abscesses are circumscribed intra-abdominal collections of pus located near the pancreas. They usually develop 4 weeks or more after the onset of acute pancreatitis and probably develop as a complication of limited necrosis with subsequent liquefaction and secondary infection.

The source of infection can be hematogenous or lymphatic, due to gastrointestinal fistula or perforation, or iatrogenic.[31]

The CT diagnosis of pancreatic abscess is based on the presence of a focal, low- attenuation collection with a relatively thick wall that often contains gas bubbles but gas bubbles are not specific for infection.

4. Infected Necrosis.

Necrotic pancreatic or peripancreatic tissue can become infected. It is often recognized on CT scans as bubbles of gas or air pockets within areas of pancreatic or peripancreatic -necrosis (emphysematous pancreatitis). CT is sensitive for detecting even the smallest amount of gas.[11]

It is important to distinguish abscess from infected necrosis, because the mortality rate for the latter is nearly double that of the former, and the specific therapy for each condition is different.[11]

On CT, an abscess is diagnosed when a normally enhancing pancreas is seen

with an adjacent fluid collection composed of liquid pus. Infected necrosis is diagnosed when a zone of nonenhancing heterogeneous pancreas is seen.

Needle aspiration is crucial, because the CT appearance of a low-attenuation zone of infected necrosis may be similar to that of an abscess.

5. Hemorrhage.

Hemorrhage in acute pancreatitis usually occurs as a late consequence due to either diffuse leakage from the inflamed granulation tissue or vascular injuries produced by the activated and extravasated pancreatic enzymes. The splenic artery and its branches or the pancreaticoduodenal arcade arteries are commonly affected.

CT usually shows high-attenuation fluid (blood) within the peritoneal cavity or retroperitoneum or within a preexisting fluid collection or pseudocyst.

REVISED ATLANTA CLASSIFICATION OF ACUTE PANCREATITIS[32]:

The revised Atlanta classification is designed to precisely describe patients with acute pancreatitis, standardize terminology across specialties, and help in treatment planning. It defines acute pancreatitis as IEP or necrotizing pancreatitis and distinguishes between an early phase (1st week) and a late phase (after the 1st week). The first phase is defined by clinical parameters, and the second phase is defined morphologically on the basis of contrast-enhanced CT findings combined with clinical staging.The most important change in the Atlanta classification is the categorization of the various pancreatic collections. In acute IEP, collections that do not have an enhancing capsule are called APFCs; after development of a capsule, they are referred to as pseudocysts (usually after the first 4 weeks). In necrotizing pancreatitis, a collection without an enhancing capsule is called an ANC (usually in the first 4 weeks) and thereafter a WON, which has an enhancing capsule. All four types of collection can be sterile or infected. The most important distinction between

collections in necrotizing pancreatitis and those associated with acute IEP is the presence of non liquefied material in collections due to necrotizing pancreatitis. In the early phase of pancreatitis, distinction between APFC and ANC by CT may be impossible and, if clinically needed for treatment planning, MR imaging or US may be used to determine the presence of non liquefied material. Depending on the time from onset of acute pancreatitis, any collection within the pancreatic parenchyma should be considered an ANC and not an APFC if less than 4 weeks have passed since the onset of symptoms or a WON and not a pseudocyst if a well-defined capsule has developed. Determination of superinfection is based on clinical presentation and on presence of air observed in collections by CT and if air is absent on CT, by percutaneous needle aspiration. Treatment planning is based on severity of pancreatitis and presence or absence of infection combined with clinical signs. The Revised Atlanta classification system with CT helps guide management and monitor the success of treatment and is shown in the table (2) below :

Type of collection	**Time (wk)**	**Necrosis**	**Location**	**Appearance**	**Infection**	**Drainage/ Surgery**
Interstitial Edematous Pancreatitis						
APFC	**</= 4**	**No**	Adjacent to pancreas, extra pancreatic only	Homogenous fluid attenuation, no liquefaction(debris),not encapsulated	Extremely rare	None
Pseudocyst	**>4**	**No**	Adjacent or distant to pancreas	Homogeneous, fluid attenuation, no liquefaction (debris), encapsulated rare rarely (for infection	Rare	Rarely (for infection or symptoms

				or symptoms		
Necrotizing Pancreatitis						
Sterile ANC	**</= 4**	**Yes**	In parenchyma and/or extrapancreatic	Heterogeneous, Nonliquefied material, variably loculated, not encapsulated	No	Based on clinical, percutaneous drainage at times, surgery rarely
Infected ANC					**Yes**	Percutaneous drainage, surgery later if needed
Sterile WON	**>4**	**Yes**	In parenchyma and/or extrapancreatic	Heterogeneous, nonliquefied material, variably loculated, encapsulated	No	Percutaneous drainage, based on clinical, surgery to follow if needed
Infected WON					**Yes**	Percutaneous drainage/ surgery to follow if needed

Table – 02 The Revised Atlanta Classification System

B. CHRONIC PANCREATITIS :

Chronic pancreatitis a disease of prolonged pancreatic inflammation and fibrosis, is characterized by irreversible morphologic and or functional abnormalities Clinically patients present with various clinical features like chronic upper abdominal pain, steatorrhea, diabetes or recurrent inflammation.

Three principal forms of chronic pancreatitis are currently recognized.[11]

1. Calcifying chronic pancreatitis - It presents with recurrent bouts of abdominal pain and the eventual development of intraductal calculi in a large proportion of cases. Causative factors include alcohol and tobacco use.

There are hereditary, tropical, idiopathic, and senile forms; the senile form is often painless.

2. Obstructive chronic pancreatitis - In this form, persistent obstruction of the pancreatic duct due to tumor or post inflammatory ductal stricture leads to atrophy of the upstream pancreas. Though often painless, it occasionally presents with clinically acute pancreatitis. Intraductal calculi are generally not seen.

3. Autoimmune pancreatitis. This is a chronic systemic lymphoplasmacytic inflammatory process involving the pancreas and other organs.

Typical CT manifestations of chronic pancreatitis —

- Irregular ductal dilation and strictures - dilated pancreatic duct is associated with irregularity owing to dilated side branches
- Parenchymal atrophy.
- Pancreatic calcifications - is the most specific CT manifestation of chronic pancreatitis.
- Focal or diffuse pancreatic atrophy, a secondary manifestation
- Biliary ductal dilation at the level of the pancreatic head is a nonspecific finding and in chronic pancreatitis, common bile duct stenosis tends to be longer and more gradually tapered.
- Mature pseudocysts when present with chronic pancreatitis seen with well-defined enhancing walls.
- The extensive lobular and periductal inflammation and fibrosis may result in the formation of benign inflammatory pancreatic masses.

AUTOIMMUNE PANCREATITIS:

Autoimmune pancreatitis (AIP) was first described by Sarles and colleagues in 1961 as "primary inflammatory sclerosis" of the pancreas, is a variant of chronic pancreatitis that involves an autoimmune process.

Pathologically it is characterized by marked fibrosis and lymphoplasmacytic infiltration of the pancreas and increased serum immunoglobulin G (IgG), especially the IgG4 subtype, or positive auto antibodies.

Its association with other autoimmune disorders such as Sjogren's syndrome, primary sclerosing cholangitis, primary biliary cirrhosis, ulcerative colitis, and systemic lupus erythematous is well known.

The clinical manifestation of AIP is varied and ranges from mild, nonspecific complaints such as upper abdominal pain and fatigability to obstructive jaundice and severe pain mimicking pancreatic malignancy. Occasionally, patients present with symptoms related to extrapancreatic organ involvement.[33] There is a preponderance of AIP in older men.

The classic CT appearance of the pancreas in ALP[11]

- Diffuse sausage-shaped enlargement of the pancreas
- Homogeneous attenuation,
- Moderate enhancement,
- A peripheral rim of a hypo attenuation referred as a "halo."
- Loss of lobularity is common; peripancreatic fat stranding is usually minimal.
- As the disease progresses, involution or retraction of the pancreatic tail is evident with diffuse or irregular attenuation of the pancreatic duct and compression or narrowing of the distal common bile duct due to pancreatic swelling are seen.

- Mild enlargement of the regional lymph nodes is also common.
- Extrapancreatic manifestations include focal lesions in the lungs, kidneys, liver, or tissue around the aorta, described as inflammatory pseudotumors.

C. HEREDITARY PANCREATITIS[34]

Hereditary pancreatitis (HP) is a rare cause of chronic pancreatitis (CP), first described by Comfort and Steinberg in 1952. It is an autosomal dominant relapsing pancreatitis with an estimated 80% incomplete penetrance. It generally manifests during childhood, with a long delay between the first manifestation and the diagnosis. However, a second peak may be attributable to the introduction of alcohol in the diet. . The type of mutation has no influence. The major risk and the main cause of mortality is pancreatic adenocarcinoma.

D. GROOVE PANCREATITIS[11]

This is a segmental form of pancreatitis with inflammation in the groove between the duodenum and the head of the pancreas; the rest of the pancreas enhances normally, and there is normal ductal morphology.

Contrast-enhanced CT demonstrates a sheet like lesion in the pancreaticoduodenal groove, effacing the groove and showing delayed enhancement. Cyst formation in the duodenal wall or pancreaticoduodenal groove has also been described.

E. PANCREATIC TUBERCULOSIS[11]

Tuberculosis of the pancreas is uncommon and usually occurs as a complication of miliary tuberculosis and immunodeficiency. In addition to the constitutional symptoms, pancreatic tuberculosis may present as acute or chronic pancreatitis, portal vein obstruction, and pancreatic mass mimicking abscess, carcinoma, obstructive jaundice, gastrointestinal bleeding, and peripancreatic abscess.

Focal involvement of the pancreas most frequently occurs in the pancreatic head, followed by the body and tail: diffuse pancreatic involvement is exceedingly rare. CT reveals a focal hypo dense lesion, often displaying internal densities. On contrast- enhanced CT the well-defined mass may show irregular margins with peripheral enhancement. Areas of central enhancement may give a multiloculated appearance.

PANCREATIC NEOPLASMS

Pancreatic carcinoma is the fourth most common cause of cancer deaths and accounts for approximately 3% of all cancers. Despite the advent of CT, US and MRI the prognosis remains grim, although improvement in imaging and percutaneous biopsy and biliary drainage techniques has expedited diagnosis and palliative management.

As most patients of pancreatic neoplasms present with inoperable tumours widespread use of non-operative methods for relieving obstructive jaundice in patients with non resectable tumours in the pancreatic head and periampullar regions has put greater demands on CT, in the pretreatment evaluation in these patients to determine resectable from unresectable tumours.[35]

CLASSIFICATION OF PANCREATIC NEOPLASMS[36]

- ❖ EPITHELIAL TUMOURS

I. Duct Cell tumors
    - Ductal cell adenocarcinoma

II. Variant carcinomas of duct cell origin
    - Pleomorphic giant cell carcinoma
    - Adeno squamous carcinoma
    - Mucinous cystic tumour

- Serous cystadenoma
- Intraductal papilloma
- Mucinous adenocarcinoma

❖ EXOCRINE TUMOURS OF ACINAR CELL ORIGIN

- Acinar cell carcinoma
- Acinar cyst adenocarcinoma
- Pancreaticoblastoma

❖ ENDOCRINE CELL TUMOURS

- Gastrinomas
- Insulinomas
- Glucagonomas
- VIP omas
- Somatostatinomas
- Non functional islet cell tumours

❖ TUMOURS OF UNCERTAIN HISTOGENESIS

- Small cell carcinomas
- Solid cysts (papillary cystic) tumour

❖ NON EPITHELIAL TUMOURS

- Sarcomas
- Dermoid cyst
- Lymphangioma
- Leiomyosarcomas
- Haemagiopericytomas
- Malignant fibrous histiocytoma

- Lymphoepithelial cyst

❖ LYMPHOMA

❖ METASTASIS

PANCREATIC ADENOCARCINOMAS

Adenocarcinoma accounts for more than 90% of the malignant tumors of the pancreas and is the fifth leading cause of cancer death in the West. It has been called the "silent killer" because of its silent course, late clinical symptoms and rapid growth pattern.

Location:

About two thirds of pancreatic adenocarcinomas occur in the head of the pancreas, the remainder are found in the body or tail or diffusely infiltrate the organ.

Pancreatic adenocarcinoma causes intense desmoplastic reaction and obstructs the pancreatic duct, with subsequent upstream duct dilation and parenchymal atrophy. If it arises in the head it causes obstruction of CBD.

Pancreatic adenocarcinoma can extensively infiltrate the retroperitoneum and invade the surrounding anatomic structures including the duodenum, stomach, mesenteric vessels, portal vein, neurovascular spaces and lymphatic channels.

It tends to metastasize to regional lymph nodes, liver, and peritoneum.

The clinical presentation

1. Varies according to the cancer's site of origin and its stage.

2. Long-standing abdominal pain, asthenia, reduced appetite and weight loss.

3. New-onset diabetes mellitus is present in 10% of patients.

4. Painless jaundice is present in 75% of patients.

5. Tumors in the body and tail tend to present with back pain.

Pancreatic adenocarcinoma is considered unresectable in the case of extrapancreatic invasion of major vessels (defined as tumour-to-vessel contiguity

>50%) such as the celiac artery, hepatic artery, portal vein, SMA or SMV; massive venous invasion with thrombosis; or distant metastasis to the liver, regional lymph nodes or peritoneum.

A tumor is classified as resectable when there is limited invasion into the SMV.

Role of MDCT

In Triple Phase, routine acquisition of images in the arterial phase is unnecessary for detection of pancreatic adenocarcinoma. Images of the pancreas obtained in the portal phase with multi– detector row CT most accurately display vascular invasion.

A dual-phase (PP and PVP) pancreatic protocol for MDCT is a sensitive technique for detecting and staging pancreatic adenocarcinoma and for detecting metastases to the liver and peritoneum.

The PP allows optimal tumor detection and mapping of the regional vascular structures; in this phase, the pancreatic adenocarcinoma appears as a low-density lesion compared with the normal pancreatic parenchyma. In the PVP, the tumor conspicuity against the normal pancreas may be reduced owing to contrast diffusion into the interstitium of the tumour, but the detection of liver and peritoneal metastasis and the visualization of portal venous structures are improved.

In about 10% of cases the mass is iso attenuating with contrast enhancement and cannot be directly observed. In these cases, useful indirect signs

- Stenosis of the distal common bile duct, stenosis of the pancreatic duct with upstream ductal dilation.
- Parenchymal atrophy,
- Double duct sign - stenosis of both the common bile duct and the pancreatic duct, with subsequent upstream dilation.

- Loss of lobulations of the pancreatic parenchyma, and deformity of the pancreatic contours.

MDCT is the modality with the highest global accuracy in the assessment of vascular invasion[14]. The likelihood of vascular infiltration by adenocarcinoma of the pancreas increases with greater tumour—to—vessel circumference contact.

- The likelihood of vessel infiltration is less than 3% when the tumour – to - vessel circumference contact is less than 90 degrees;
- It is between 29% and 57% for contact between 90 and 180 degrees.
- It is more than 80% for contact greater than 180 degrees.

Other useful criteria that indicate vascular invasion are the -teardrop- sign which refers to the shape of the portal vein or the SMV; the lack of preservation of a fat plane around the vessels; and dilation of the pancreaticoduodenal veins.

ENDOCRINE TUMOURS[11]

Tumours arising from the islet cells of the pancreas account for about 2% of pancreatic neoplasms. These tumours have an incidence of 1 in 100,000 and are usually divided into functional and nonfunctional tumours on the basis of hormone overproduction and the associated clinical syndromes.

Functional tumours are named by the predominant hormone they produce: insulinomas, gastrinomas, VIPomas, glucagonomas, and somatostatinomas. They can be associated with syndromes such - as multiple endocrine neoplasia type I (MEN-I), von Hippel-Lindau disease, and neurofibromatosis.

CLASSIFICATION[11,22]

A. Functional Tumours.

1. Insulinomas - they constitute about 50% of all endocrine tumours of the pancreas. They can cause hypoglycemia and tend to be small at diagnosis. Typically they present in patients between 30 and 60 years of age and are equally distributed between the sexes. Insulinomas are usually solitary, measure less than 2 cm in 90% of cases, and tend to be highly vascularized.

2. Gastrinomas - they account for about 20% of pancreatic endocrine tumours, have a predilection for males, and usually occur in the fifth decade. They arise predominantly in the so-called gastrinoma triangle, which is delimited by the junction of the neck and body of the pancreas medially, the junction of the second and third portions of the duodenum inferiorly, and the junction of the cystic duct and the common hepatic duct cranially. About 50% are located in the duodenum, 14% in the pancreas, and 13% in the lymph nodes; the remaining is found in various locations. They are usually smaller than 1cm; less vascularized than insulinomas, and can be multiple. Gastrinomas can induce Zollinger-Ellison syndrome. About 30% are associated with MEN-1.

3. VIPomas rank third in frequency among pancreatic endocrine tumors. They have a female predilection (3:1). VIPomas may be responsible for the WDHA syndrome (watery diarrhea, hypokalemia, and achlorhydria-hypochlorhydria).

4. Glucagonomas account for 1% of all endocrine tumours of the pancreas; they usually occur in the body or tail. Because of the difficulty in recognizing their main clinical manifestation, which is necrolytic migratory erythema. They are discovered when large (4 to 10 cm). Mild hyperglycemia may also occur.

5. Somatostatinomas occur more often in women with a mean age at presentation of

51 years. They are originally pancreatic in 50% of patients and duodenal in the remaining 50%; they are associated with neurofibromatosis type 1. These tumours may cause diabetes, gallbladder disease, and steatorrhea.

6. Parathyroid hormone- and adrenocorticotropic hormone- secreting pancreatic tumours are extremely rare.

B. Nonfunctional Tumors.

Nonfunctional pancreatic tumours manifest in the fourth or fifth decade, usually arise in the head of the pancreas and, because of the lack of symptoms from hormone overproduction, are large when discovered. They tend to manifest clinically when their size or infiltration causes abdominal pain, anorexia, and weight loss. They may range in size from 3 to 24cm; with 30% exceeding 10 cm. 90% of nonfunctioning pancreatic tumours are malignant.

CT appearance of endocrine tumours [11, 22, 36]

• When they are small, functional endocrine tumours are non-contour deforming, appear Isodense on non-enhanced CT, and enhance strongly during the arterial and portal phases of contrast administration.

• Sometimes they appear cystic and hypo enhancing and may contain discrete calcifications, especially if malignant.

• Some insulinomas may be hyper dense before contrast administration.

• Larger functional endocrine tumors may appear heterogeneous following contrast administration. Areas of central necrosis, as well as calcifications and retroperitoneal invasion, may be found in the case of malignancy.

• Nonfunctioning endocrine tumours usually present as large, well-defined, enhancing masses, with the degree of heterogeneity proportional to their size. When small. they may present as strongly and homogeneously enhancing masses. Areas of central

necrosis and calcifications may be found in larger lesions, usually malignant ones.

LYMPHOMA[11,37]

Primary pancreatic lymphoma is rare, constituting 0.5% of all pancreatic neoplasms.

Presenting clinical features are nonspecific and present with symptoms of carcinoma of head like abdominal pain weight loss, and jaundice. Sometimes CA 19-9 is elevated.

Lymphomas tend to occur in those aged 35 to 75 years (mean age, 55 years) and predominate in men (7:1). Lymphomas usually occur in the head of the pancreas (80% of cases).

At imaging, lymphomas may have one of two appearances -

- Localized mass, which frequently extends to extrapancreatic regions,
- Diffuse enlargement and replacement of the pancreas.

Both forms of pancreatic lymphoma tend to show a diffuse, invasive growth pattern that does not respect anatomic boundaries, infiltrating the retroperitoneal structures and gastrointestinal tract.

Useful features to differentiate lymphoma from pancreatic adenocarcinoma are the combination of a pancreatic head mass with a normal, nonobstructed pancreatic duct; lymph node enlargement below the renal veins; and large size.

Absence of calcifications and necrosis are other useful criteria to differentiate lymphomas from endocrine tumours. Lymph node enlargement limited to the peripancreatic region; and a normal leukocyte count favor primary pancreatic lymphoma rather than secondary involvement.

ACINAR CELL CARCINOMA

Acinar cell carcinoma (ACC) is a rare pancreatic tumour, accounting for about 1% of exocrine pancreatic neoplasms. It is characterized by the production of pancreatic enzyme by tumour cells. More often in women than in men, with a peak incidence in the 7th decade. ACC tends to arise as a single mass in the uncinate process and head of the pancreas (60% of cases). In about 80% of cases it exhibits exophytic growth. The mean tumour size at presentation is 7cm. A well-defined capsule circumscribes the tumour, which may present focal areas of discontinuity and infiltration into the surrounding organs.

The clinical presentation is heterogeneous, ranging from lack of symptoms-to jaundice. abdominal pain, vomiting, and weight loss. Some patients experience polyarthritis subcutaneous fat necrosis induced by hyperamylasemia.

Acinar cell cancers metastasize locally and disseminate to lymph nodes and liver.

CT reveals —

- Calcifications in the form of central punctate or stellate calcifications or peripheral punctuations or plaques seen in approximately 50% of tumours.
- In about 60% of cases, the tumour appears circumscribed by a well-defined and enhancing capsule, although areas .of capsular discontinuity and infiltration can be detected.
- Central necrosis is a common finding, present in about 80% of cases.
- The tumour enhances mildly in arterial and portal phases of dynamic contrast imaging.[38/39]

INTRAPANCREATIC METASTASES

Intrapancreatic metastases are rare, accounting for 2% of pancreatic tumours. Renal, lung, thyroid, breast, and colorectal cancers and melanoma can spread to the

pancreas, usually in the setting of diffuse disease. They may be single or multiple. At imaging, metastases tend to closely reflect primary tumour. Metastases from a hypervascular primary tumour such as renal cancer show intense enhancement during the arterial phase of dynamic contrast imaging.

- If small, they appear homogeneous.
- If large, internal heterogeneity with necrotic areas is found. Metastases from the colon mimic pancreatic adenocarcinomas.

CYSTIC LESIONS

Many benign as well as malignant conditions like simple pseudocyst, cystic neoplasm, ductal adenocarcinoma, and metastasis can mimic a cystic neoplasm on imaging. Differentiating cystic lesions is important, because the management and prognosis varies.

Types of Lesions[40]

Pathologic Classification of Cystic Pancreatic Tumours

- Pseudocyst
- Common cystic pancreatic neoplasms
    - Serous cystadenoma
    - Mucinous cystic neoplasm
    - Intraductal papillary mucinous neoplasm
- Rare cystic pancreatic neoplasms
    - Solid pseudopapillary neoplasm
    - Acinar cell cystadenocarcinoma
    - Lymphangioma
    - Hemangioma
    - Paraganglioma

- Solid pancreatic lesions with cystic degeneration
    - Cystic islet cell tumours (insulinoma, glucagonoma, gastrinoma)
    - Pancreatic adenocarcinomas
    - Metastasis
    - Cystic teratoma
    - Sarcoma
- True epithelial cysts

1. PSEUDOCYST.

Pseudocysts constitute the majority (30-90%) of cystic lesions of the pancreas. These are the most frequently encountered unilocular cystic lesions in patients with a clinical history of pancreatitis.

2. SEROUS CYSTADENOMA (SCA).

SCAs account for 30% of pancreatic cystic neoplasms, the majority of which are found in female with a median age of 65 years. 70% of these benign lesions demonstrate a polycystic or microcystic (honeycomb) pattern consisting of a collection of cysts (usually more than six) that range from a few millimeters up to 2 cm in size.

CT features[41]

- These lesions may appear solid owing to the compact arrangement of cysts.
- Fine, external lobulations are a common feature, and enhancement of septa and the cyst wall may be seen.
- A fibrous central scar with or without a characteristic stellate calcification is seen in 30% of cases and is considered virtually pathognomonic for serous cystadenoma.

3. MUCINOUS CYSTIC NEOPLASMS (MCNs)

MCNs comprise 44% to 49% of pancreatic cystic lesions. They occur predominantly in women, with a mean age of 47 years, but also occur in men.[40,42] The majority are solitary and multilocular with a few large compartments (between 2 and 6 cm) and microcystic, but they can be unilocular with a single compartment.

The majority have a smooth contour, although a lobulated contour may be seen. The mean size is greater than 5 cm (range, 3 to 20 cm), and they have a thick fibrotic wall that can calcify.

Small mural nodules may be undetected by MDCT is extremely sensitive. These cysts occasionally contain debris or hemorrhage. Peripheral eggshell or septal calcifications on CT are specific for mucinous cystic lesions and are predictive of malignancy.

4. INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS (IPMN).

IPMN comprises 21% to 33% of cystic pancreatic neoplasms and affects both sexes, with a slight male predominance, in the sixth to seventh decades.

Intermittent ductal obstruction occurs, and recurrent abdominal pain is the most frequent symptom. Back pain, jaundice, and weight loss are less common symptoms. Chronic obstruction may cause exocrine and endocrine insufficiency, leading to steatorrhea and diabetes mellitus.

IPMN is a mucin-producing tumour of the pancreas: it develops from the epithelial lining of the main pancreatic duct or its side branches, with variable ductal dilation.

Main-duct IPMN is a morphologically distinct entity, and although it does not present as a cystic lesion. This type of IPMN occurs predominantly in the head of the pancreas and only occasionally in the tail. Owing to mucin production, there is partial

or diffuse dilation of the main duct, which is filled with mucin; the ductal dilation is disproportionate to the degree of parenchymal atrophy.Excessive mucin secretion may result in bulging of major papillas into the duodenal lumen; this is considered a pathognomonic sign on cross-sectional imaging and is seen more often with malignant tumors.The inner surface of the dilated duct frequently contains mural nodules. CT reveals diffuse or segmental dilation of the main pancreatic duct, with or without polyploidy lesions

Side-branch or mixed IPMNs in which a side-branch tumour extends into the main duct. It present as unilocular or multilocular cystic lesions that communicate with the main pancreatic duct. Lack of visualization of communication on imaging does not exclude a side-branch IPMN. One or more branches of the pancreatic duct may be affected which consequently show cystic dilation.

In mixed IPMNs, in addition to the presence of a side-branch 1PMN, the main pancreatic duct contains papillary growth of columnar epithelium showing varying degrees of dysplasia. Mixed-type I PVIN is an advanced form of the side-branch type in which the IPMN has spread to the main pancreatic duct, or it is an ultimate form of the main-duct type in which the I PMN involves the branch ducts as well.

5. SOLID PSEUDOPAPILLARY TUMOURS (SPT)

Solid pseudopapillary tumor is an uncommon benign exocrine pancreatic tumour commonly found in Asian or African American women between the second and third decades of life. These comprise about 9% of pancreatic cystic tumours.

SPTs may be asymptomatic or they may present as a gradually enlarging, nontender abdominal mass or with vague abdominal pain, discomfort, or obstructive symptoms.[43] The classic CT features are[44]

- A well-encapsulated lesion with varying solid and cystic components owing to

hemorrhagic degeneration.

- Following contrast administration, enhancing solid areas are typically noted peripherally, whereas cystic spaces are usually more centrally located.

SPTs leading to malignant degeneration, invasion of adjacent structures, and metastases designated as solid pseudopapillary carcinomas. These malignant tumours are often older at presentation and have a male predilection. The most common site for metastasis is the liver; metastases exhibit complex features similar to those of the primary tumour. Rarely, lymph node metastasis, peritoneal spread, and multiplicity can occur.

6. LYMPHANGIOMA

Pancreatic lymphangiomas constitute less than 1% of all lymphangiomas. These slow-growing tumors are generally considered to be of pancreatic origin if they are within the parenchyma, adjacent to the pancreas, or connected to the organ by a pedicle. There is a female preponderance, and all regions of the pancreas are equally affected.
Clinical features :

- Most patients are asymptomatic, and the lesion is discovered incidentally on imaging studies.
- Others may present with vague abdominal pain, nausea, vomiting, palpable mass, or symptoms related to compression of neighboring organs.
- Occasionally, acute presentations may be related to torsion of the pedicle, rupture, or hemorrhage into the lymphangioma.

CT reveals a septated fluid-density lesion and can characterize content of the cysts.

7. HEMANGIOMA

Pancreatic hemangioma may present as a palpable abdominal mass, gastrointestinal bleeding, or a mass compressing adjacent structures such as the biliary tract or

duodenum.

CT reveals cystic lobulated mass with marked enhancement after intravenous contrast administration. Enhancement may be delayed owing to sclerosis within the tumor.

8. TRUE EPITHELIAL CYSTS[45]

True cysts of the pancreas differ from pseudocysts and retention cysts in terms of origin, histologic appearance, and clinical significance. True cysts are believed to develop anomalously from remnants of the embryologic ductal systems. Histologically, they have a lining of epithelial cells that may atrophy owing to pressure.They may be detected incidentally and are commonly associated with von Hippel-Lindau disease. CT reveals a well-defined unilocular cystic lesion with indistinct walls.

PANCREATIC TRAUMA[46]:

Blunt pancreatic trauma is rare, accounting to less than 2% of all abdominal injuries. These injuries often occur during traffic accidents as a result of the direct impact on the upper abdomen of the steering wheel or the handlebars. Isolated injuries are rare (<30%) owing to its anatomical location, however coexisting injuries are common (50%–98%).

Identification of a blunt injury of the pancreas may be difficult because imaging findings are often subtle. Delays in diagnosis, incorrect classification of the injury, or delays in treatment can increase the morbidity and mortality considerably. The morbidity and mortality associated with trauma to pancreas is remarkably high. The variability in morbidity and mortality is caused by several factors: the presence of coexisting injuries, the mechanism of injury, the time to diagnosis, the presence or absence of major ductal injury, which are considered to be predictors of outcome.

The probability of complications after pancreatic trauma in many cases is the

result of missed findings or diagnostic delays or both. Delayed diagnoses and therapeutic interventions often result in a difficult clinical course with a dubious outcome. However, within the first 48 hours after pancreatic injury, most patients succumb to hemorrhage from splenic, hepatic, or vascular injuries. Organ injuries most commonly associated with pancreatic trauma are hepatic (46.8% of cases), gastric (42.3%), major vascular (41.3%), splenic (28.0%), renal (23.4%), and duodenal (19.3%).

Approximately one-third of the patients who survive the first 48 hours develop complications related to their pancreatic injury. Common complications of pancreatic injuries include pancreatitis, pseudocysts, fistulas, intraabdominal abscesses, pneumonia, and anastomotic breakdown, and these are related to the development of multiorgan failure and septicemia. About 37% of late deaths are primarily attributable to the injury itself and usually occur within 1–3 weeks of the injury or later.

The time between the injury and the diagnosis and definitive treatment is an important factor in the development of complications and their resulting mortality. When a definitive diagnosis is delayed for more than 24 hours, up to 40% of patients are at risk of death, as opposed to 11% of those patients operated on within 24 hours.

Computed tomography (MDCT) provides the safest and most comprehensive means of diagnosis and grading of pancreatic injury in hemodynamically stable patients. Below is the table (3) representing **American Association for the Surgery of Trauma (**AAST) grading of pancreatic injury[46]:

Grade	Injury	Description
I	Hematoma	Minor contusion without duct injury
	Laceration	Superficial laceration without duct injury
II	Hematoma	Major contusion without duct injury
	Laceration	Major laceration without duct injury or tissue loss
III	Laceration	Distal transection or parenchymal injury with duct injury
IV	Laceration	Proximal transection or parenchymal injury involving the ampulla or bile duct
V	Disruption	Massive disruption of the pancreatic head

Table-03 AAST Grading of pancreatic injury

LITERATURE OF PANCREATIC PATHOLOGIES

CONVENTIONAL RADIOGRAPHY:

Abdominal x-ray has a limited role in evaluation of pancreatic pathologies but few times it may show certain important findings helpful in evaluation of pancreas. They are

Acute pancreatitis:

- Colon cut off sign : with air in transverse colon till splenic flexure and no bowel gas distally in Descending colon due to spread of inflammation along the mesocolon.
- Ileus
    - Sentinel loop: a single dilated jejunal loop in the upper abdomen
    - Diffuse ileus (smalll bowel dilatation) is most common

Chronic pancreatitis:

- Calcification in the pancreas
- Mass from a pseudocyst with wall calcifications.

Complications:

- Pleural effusion.
- Ascites.

Philip A. Sorabella et al[47](1975) in their prospective study of 429 patients, "undertaken to determine if the axial pancreatic view would improve detection of pancreatic body-tail enlargement. This view is produced by directing a roentgen-ray beam along the axis of the pancreatic body-tail cylinder. On this view and on the supine translateral view, a pancreatic body-tail space can be identified as a subdivision of the retrogastric space. When the pancreatic body-tail enlarges, this space selectively enlarges. The percentage change of this space is particularly impressive on the axial pancreatic view, where 10 of 12 pancreatic body-tail neoplasms were detected with only a 1.8 per cent false positive rate".

Owen J.O'Connor et al[48](2011) in their study stated that "conventional radiography and upper gastrointestinal series no longer play an important role in the diagnosis of acute pancreatitis. 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. 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".

Po-Cheng Liang et al[49](2017) presented a case of chronic pancreatitis with calcified pesudocyst and concluded when chronic pancreatitis is suspected, clinicians should consider arranging a plain abdominal X-ray because it can yield additional information for further treatment strategies.

ULTRASOUND (USG) :

Ultrasound is the first modality of choice for imaging of pancreatic pathology after clinical suspicion. It helps in studying about the structural, echogencity and ductal abnormalities of pancreas, etiology of pancreatitis in few cases and complications of various pathologies and even in traumatic evaluation of pancreas.

However, necrosis, better characterization of masses and complications of pancreatitis is not possible through USG in all cases and USG is of less role in case of obese patients or when excessive gas filled bowel loops compromise the pancreatic evaluation.

Acute pancreatitis:

- Edematous pancreas.
- Gall stones.
- Peripancreatic collection.
- Necrosis

Chronic pancreatitis:

- Atrophy of pancreas.
- Calcification in the pancreas.
- Mass from a pseudocyst with wall calcifications.
- Main pancreatic duct dilatation.

Tumor:

- Mass in pancreas.
- Change in contour
- Ductal dilatation.
- Calcific foci.

Complications:

- Thrombosis.
- Hemorrhage
- Pleural effusion.
- Ascites.

B Sigel et al[50] (1984) in their study using real-time ultrasound imaging at 122 operations for the complications of pancreatitis, adenocarcinoma, and islet cell tumors stated that ultrasound was useful in 69% cases of pancreatitis and 66% of tumors. They stated that USG helped in better definition of pathology & assessment of pancreas and surrounding structures, than x-ray studies, and yielded unique information about the etiology of abnormalities, its use during pancreatic operations can significantly aid the surgeon and recommended its wider application in surgical practice.

Radu Badea[51] (2005) in his study of Acute Pancreatitis. concluded that USG can be used as first imaging technique in pancreastic pathology evaluation.The diagnosis is based on increase in size of pancreas with altered echogenicity correlating to edema and necrosis. Complications can be diagnosed by identification of peripancreatic collections, necrosis, thrombosis and pseudo aneursyms in acute phase and pesudocyst and portal hypertension syndrome in late phase.

Masaru Koizu Mi et al[52] (2006) in their study concluded that "Ultrasonography (US) is capable of identifying pancreatic enlargement and inflammatory changes near the pancreas, and it may be useful in diagnosing acute pancreatitis. Although, in severe cases, visualization of the pancreas and peripancreatic tissue may be impaired by gas in the intestinal tract. Ultrasonography may also visualize abnormal findings associated with the etiology and morbidity of acute pancreatitis, such as ascites, gallstones, and cholangiectasis. It is particularly important to check for the presence of cholecholithiasis and cholangiectasis when judging whether endoscopic sphincterotomy for gallstone pancreatitis is required. US is also useful for screening for complications such as aneurysms".

Owen J.O'Connor et al[48](2011) in their study stated that "sonography of patients with acute pancreatitis is often negatively impacted by difficulty visualizing the pancreas because of ileus and overlying bowel gas. Abnormal ultrasound findings 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".

COMPUTED TOMOGRAPHY (CT):

LITERATURE ON ACUTE PANCREATITIS :

A. Margulis, et a1(1978)[53] and the other by **R. Levitt, et al**[54] highlighted the complementary use of US and CT for imaging of pancreas in disease. Both agreed that CT was useful when US failed in the diagnosis of acute pancreatitis due to

localized duodenal ileus.

William Silverstein et al (1981)[55] using CT and sonography, in a prospective study of 102 patients with acute pancreatitis, found CT to be of significantly greater value than US due to the high percentage (38%) of non-diagnostic studies with USG.

The authors categorized the patients into 6 groups based on CT findings.

1. Normal glands.

2. Disease limited in pancreas.

3. Involvement limited to contiguous peripancreatic fact.

4. Involvement of peripancreatic compartment.

5. Involvement of two or more intra-abdominal and retroperitoneal compartments.

6. Catergory 4 along with extension through lateroconal or Gerota's fascia or into the pelvis.

Michael P. Federle et al (1981)[56] used CT as the primary diagnostic tool in 10 cases of pancreatic abscess and 7 cases of infected pseudocyst. They found pancreatic gas collection as the only definitive feature of infection as seen in 5 cases (25%). They found that it was not possible to distinguish infected from non-infected pseudocysts or peripancreatic fluid collections on the basis of high attenuation of the cyst fluid, irregular cyst walls which enhance with intravenous contrast media.

R. Brooke, leffery et al (1982)[57] scanned 36 patients using both oral and intravenous contrast media at 1 cm intervals. They concluded that CT in acute pancreatitis is of considerable value in defining the presence and extent of peripancreatic inflammation. They found the following features of acute pancreatitis —

1. Normal pancreas seen in 3 cases.
2. 31 patients showed diffuse pancreatic enlargement associated with obliteration of peripancreatic fat planes.

3. Only 2 patients revealed focal enlargement of the head of the pancreas
4. In 18 cases showed pancreatic abscess or infected fluid collection.
5. Noninfected pancreatic or extrapancreatic fluid collections seen in 10 cases.
6. Haemorrhagic pancreatitis noted 3 patients.
7. Extensive phlegmonous inflammation of the retroperitoneal space and intra-abdominal abscess seen 2 patients each.

Michael C. Hill et al (1982)[58] in a study of 91 patients correlated the CT findings with the clinical type of acute pancreatitis viz, acute edematous pancreatitis, acute necrotizing (haemorrhagic suppurative) pancreatitis and acute exacerbation of chronic pancreatitis. The authors found that on follow up CT the features did not disappear with resolution of clinical symptoms, which was true of phlegmonous pancreatitis where CT finding persisted for months.

Balthazar EJ, et al (1985)[19] 83 patients of acute pancreatitis were assessed on the basis of clinical and laboratory findings (Ranson's Criteria; Prognostic signs). They classified the degree of severity on CT into five grades as follows,

These CT findings were co-related with the clinical follow-up, prognostic sign (Ranson's Criteria) complications and death.

Grade A and B patients did not have abscesses and none died regardless of the number of prognostic signs.

They found that abscesses occurred in 21.6% of the entire group, compared with 60% of Grade E patients. Pleural effusions were also more common in grade E pancreatitis. Furthermore, they found that the initial CT findings correlated with the clinical course of the patients. All patients with Grade A and B pancreatitis had a mild clinical course and were discharged in less than 2 weeks. Grade D and E pancreatitis had severe disease and developed abscesses and died.

They concluded that use of prognostic signs with initial CT finding resulted in improved prognostic accuracy and that early CT was a useful prognostic indicator of morbidity and mortality.

Ivan Vujie (1989)[59] reported pancreatic and peripancreatic arterial bleeding due to the proteolytic effect of pancreatic enzymes is one of the most life threatening complication of pancreatitis. The splenic artery, the pancreaticoduodenal and gastroduodenal arteries are most commonly involved. Erosion of a vessel can lead to free hemorrhage or formation of a pseudoaneursysm. Bolus dynamic CT is the most useful modality for diagnosis of vascular involvement with pancreatitis. CT could -

1. Establish the diagnosis of pancreatitis and identify the fluid collections in patients with hemorrhage.
2. Determine the extent of inflammatory process and its proximity to important vascular structures.
3. Diagnose the presence of hemorrhage on CT in fluid collection by displaying increased attenuation of contents (130 HU).
4. Identify pseudoaneurysm formation by displaying transient vascular enhancement in a cystic pancreatic mass.

Thrombosis of the peripancreatic tributaries of the portal vein, splenic vein and development of vascular collaterals is a known complication of pancreatitis.

Balthazar E J et al (1990)[24] after reviewing dynamic sequential CT in 88 patients of acute pancreatitis reported value of CT in predicting prognosis and found that pancreatic necrosis carried the highest mortality and morbidity rates.

The presence and degree of pancreatic necrosis was evaluated by using bolus injection of contrast material 50m1 of 60% iodinated at rate of 3 ml/sec, followed by 100 ml of contrast at rate of 1 ml/sec.

Patients with necrosis had 23% mortality and an 82% complication rate, while patients without necrosis had 0% mortality and 6% morbidity. Further, serious complications developed in patients who had >30% necrosis. They developed the CT severity index (Table 4) in which Grade A-E pancreatitis where assigned 0-4 points, plus 2 points for <30% necrosis, 4 points for 30-50% necrosis and 6 points for >50 % necrosis. Three categories were formed (0-3, 3-6, 7-10) which more accurately reflected the prognostic value of CT.

They reported that high complications (92%) and mortality (17%) exhibited in patients with severity index of 7 — 10 while no mortality and morbidity seen severity index of 0 to 1 patients.

Grade	**(Points)**	**Percentage Necrosis**	**(Points)**	**Severity Index = CT grade + percentage necrosis (points)**
A. Normal pancreas.	0	0	0	0
B. Focal or diffuse pancreatic enlargement.	1	0	0	1
C. Inflammation – pancreas and/or peripancreatic fat.	2	<30%	2	4
D. Single ill-defined peripancreatic fluid collection.	3	30-50%	4	7
E : Two or more ill-defined peripancreatic fluid collections.	4	>50%	6	10

Table – 4 showing CT Severity Index Grading

Emil. J. Balthazar (2002)[18] analyzed and discussed "advantages and limitations of the clinical, laboratory, and imaging prognostic indexes in his review article on Acute Pancreatitis: Assessment of Severity with Clinical and CT Evaluation. According to him contrast enhanced CT' is imaging modality of choice to help stage the severity of inflammatory processes, detect pancreatic necrosis and depict local complications. CT shown early overall detection rate of 90% and nearly 100% sensitivity after 4 days of pancreatic gland necrosis. CT severity index is excellent tool in correlating development of complications and death in this population".

Koenraad J. Mortele et al (2004)[23] assessed 266 patients of acute pancreatitis over a period of one year & modified CT severity index (Table 5)is used to show degree of severity.

They classified the degree of severity on CT into

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, extraparenchymal complications, or gastrointestinal tract involvement)	2

Table 5 showing Modified CT Severity Index

They concluded the modified CT severity index correlates more closely with patient outcome measures than the currently accepted CT severity index, with similar interobserver variability.

Gomez D et al[60](2012) in their study of 151 patients of pancreatitis with 117 cases of acute pancreatitis and 34 chronic pancreatitis, wanted "to assess the role of serum amylase and lipase in the diagnosis of pancreatitis and stated that the overall sensitivity and specificity of serum lipase levels in diagnosing pancreatitis was 96.6% and 99.4%, respectively. In comparison with serum amylase levels, the overall sensitivity and specificity in diagnosing acute pancreatitis were 78.6% and 99.1%, respectively".

Shalabh Jain et al[61] (2014) in their study of 150 patients of acute pancreatitis (M-99, F-51) compared the grading of severity between Balthazar CT Severity index and Modified CT severity index using MDCT and stated that "using Balthazar CTSI with the patient outcome, statistically significant correlation was found between the grades and the length of hospital stay ($p = 0.011$), development of infection ($p = 0.018$), occurrence of organ failure ($p = 0.027$), and mortality ($p = 0.019$). No correlation, however, was obtained between the score and the need for an interventional procedure ($p = 0.126$). In contrast, the correlation between the grades under the Modified CT Severity Index and outcome was much stronger ($p = 0.000$ for length of hospital stay, $p = 0.004$ for development of infection, $p = 0.024$ for occurrence of organ failure and $p = 0.013$ for mortality). It could also accurately predict the need for interventions ($p = 0.030$) and extra pancreatic complications. They concluded modified CTSI correlates more closely with patient outcome than the CTSI".

HS Batra et al (2015)[62] conducted a study on 50 patients of proven acute pancreatitis "to compare serum amylase and lipase in Acute pancreatitis. All 50 patients included

in the study had raised serum lipase, 42 patients had both amylase and lipase raised, 8 patients had amylase normal but lipase raised. In smaller hospitals where limited lab and radiological facilities are available, estimation of serum lipase will be a better choice over serum amylase in diagnosis of acute pancreatitis".

Bharat et al (2015)[63] in conducted a study on Role of Multidetector Computed Tomography in Pancreatitis with 42 patients of which 30 were diagnosed as acute pancreatitis. Features like oedematous parenchyma, necrosis, peri pancreatic inflammation and acute fluid collections were signs noted in acute pancreatitis on MDCT. They concluded MDCT is the imaging modality of choice in acute pancreatitis. The pancreatic parenchymal phase is the optimal phase for assessment for necrosis.

Avanesov M et al (2016)[64] conducted a study of 102 patients "to evaluate the additional value of dual-phase multidetector computed tomography (MDCT) protocols over a single-phase protocol on initial MDCT in patients with acute pancreatitis using three CT-based pancreatitis severity scores with regard to radiation dose. They assessed cases of interstitial oedematous and necrotizing pancreatitis separately, compared results under demographic, CT findings, complications under each category and concluded that an initial dual-phase abdominal CT after ≥72 h after onset of symptoms of acute pancreatitis was not superior to a single-phase protocolfor evaluation ofthe severity of pancreatic and extrapancreatic changes. However, the effective radiation dose may be reduced by 36% using a single-phase protocol".

LITERATURE ON CHRONIC PANCREATITIS :

Joseph T. Ferrucci Jr. et al (1979)[65] retrospectively studied the CT of 50 patients an EMI prototype whole body scanner (CT 5000) diagnosed as chronic pancreatitis proved by laparotomy or retrograde ductography and found that CT permitted a

positive diagnosis in 56% of patients.

They described the following signs.

1. 36% showed enlargement or mass, which was presumably due to inflammatory edema or fibrotic induration. Diffuse enlargement was accompanied by smooth well marginated contours with preservation of the peripancreatic fat planes. Nodular or irregular gland surface correlated with greater chronicity of symptoms. However focal mass lesions could not be differentiated from cancers.

2. Calcification was observed in 36%. It was noted in association with chronic signs. Intraductal calcification was seen as linearly arranged deposits conforming to the course of the duct. CT was better than plain radiography in picking up pancreatic calcification and helped differentiate it from vascular calcification.

3. Parenchymal atrophy was seen in 14% and with calcification in 83% patients. Atrophy was defined as head measuring < 1.5 cm, body < 1 cm, or pancreatic vertebral ratio < 0.5.

4. Duct dilatation was observed in 4%. They also described the pseudoduct sign produced by the normal splenic vein coursing along the dorsal aspect of the pancreatic body and tail.

5. Pseudocyst and abscess were described in 30% of patients. They were invariably a complication of well-established pancreatic inflammatory disease.

However in 16% of patients, CT showed a normal gland in the presence of chronic pancreatitis.

Allan Fishman, et al (1979)[66] studied 10 patients on the General Electric Model 7800 scanner with a scan time of 4.8 sec. They observed that an equal occurrence of pancreatic duct dilatation was found with pancreatic neoplasms and chronic pancreatitis. However dilatation of the pancreatic duct is frequently seen in chronic

pancreatitis on ERCP. It confirmed that a dilated duct, although specific for pancreatic disease has no specificity as to the etiology. Three patients in the present study demonstrated the double duct sign. Dilatation of both the common and pancreatic duct is caused by a lesion that simultaneously obstructs both ducts. Although originally described as a sign suggestive of neoplasm, one patient with chronic pancreatitis also demonstrated this sign.

Lincoln L. Berland et al (1981)[67] CT with thin slice thickness (5 mm instead of 10 mm) and small pixel size (1.1 instead of 1.3 mm) in 87 patients and made correlation with pancreatic ductography. They found that CT measurements of the pancreatic duct correlated well with measurements of pancreatic ductogram. They also noted that presence of calculi in the duct or duct radicals was a strong evidence of chronic pancreatitis.

They found that mildly dilated pancreatic ducts without calculi, which were irregular and tortuous, were a sign of chronic pancreatitis whereas enlarged but regular ducts were seen both in carcinoma and chronic pancreatitis.

They also described the simultaneous enlargement of the pancreatic and common bile ducts (the double duct sign) as being insensitive for differentiating chronic pancreatitis from pancreatic carcinoma.

E. J. Balthazar et al (1984)[68] did a retrospective analysis of abdominal CT examinations of 13 consecutive cases of gastric varices diagnosed over a period of 2 years. They found pathophysiology behind development of gastric varices in two patient was chronic pancreatitis with splenic vein thrombosis.

Patrick Luetmer et al (1989)[69] in a retrospective analysis of dynamic incremental contrast enhanced CT scanning of 56 cases of documented chronic pancreatitis found the following CT features :-

1. Dilatation of the main pancreatic duct 68 %.

2. Parenchymal calcification 56 %.

3. Parenchymal atrophy 54 %.

4. Fluid collection and focal pancreatic enlargement 30 % each.

5. Biliary ductal dilatation 29 %.

6. Alteration in the peripancreatic fat or fascia 16 %.

7. Normal gland 7 %.

They found that pancreatic ductal dilation and parenchymal atrophy were notably more prevalent than reported previously, which they attributed to improved pancreatic ductal visualization achieved with use of IV contrast enhancement improved resolution and faster scanning times.

John Haga (1994)[11] in his textbook noted that pancreatic calcification could be either parenchymal, ductal or both. It was important to differentiate between the two. Because surgical relief of pancreatic duct obstruction is beneficial in some patients.

Bharat et al[63] **(2015)** in conducted a study on role of MDCT in Pancreatitis with 42 patients of which 09 patients (all adults) as chronic pancreatitis and 03 patients had acute on chronic pancreatitis. Pancreatic parenchymal calcification, MPD dilatation and calculi, parenchymal atrophy, pseudocysts etc were features noted in chronic pancreatitis. They concluded that MDCT allows better detection of calcification, ductal dilatation and gland atrophy in chronic pancreatitis.

Bhatt A et al (2017)[70] did a study involving "50 patients of pancreatitis using laboratory tests, USG & CT evaluation of acute and chronic pancreatitis and stated that serum amylase is raised in more cases than lipase in pancreatitis, Ultrasound can detect presence of inflammation and characterize the size, shape and echo texture of the gland CT scan of abdomen with axial and coronal reconstruction is pre-requisite

for detailed evaluation of pancreas. CECT scan show better delineation and margins and extent of the gland than USG. CT scan is better than USG in determining the size, parenchyma, necrosis, calcification and complications associated with pancreatitis".

Oh HC et al (2017)[71] conducted a study of 211 cases of chronic patients to "assess low serum amylase and lipase values as simple and useful predictors to diagnose chronic pancreatitis and concluded that Serum amylase and/or lipase levels below the normal serum range are highly specific for chronic pancreatitis patients. Clinicians should not ignore low serum pancreatic enzyme values".

LITERATURE ON AUTOIMMUNE PANCREATITIS :

Graziani R et al[72] (2014) in their work stated that "MDCT and MR imaging are currently the most frequently performed imaging modalities for the study of pancreatic disease. In cases of suspected autoimmune pancreatitis (AIP), a dynamic quadriphasic study is recommended in both techniques. In the diffuse form of autoimmune pancreatitis (DAIP), the pancreatic parenchyma shows diffuse enlargement and appears, during the MDCT contrast-enhanced pancreatic phase, diffusely hypodense compared to the spleen because of lymphoplasmacytic infiltration and pancreatic fibrosis. During the venous phase of MDCT, the parenchyma appears hyperdense in comparison to the pancreatic phase. In the delayed phase it shows retention of contrast media. A "capsule-like rim" may be recognised as a peripancreatic MDCT hyperdense compared to the parenchyma. DAIP must be differentiated from non-necrotizing acute pancreatitis (NNAP) and lymphoma since both diseases show diffuse enlargement of the pancreatic parenchyma.

In the focal form of autoimmune pancreatitis (FAIP), the parenchyma shows segmental enlargement involving the head, the body-tail or the tail, with the same contrast pattern as the diffuse form. FAIP needs to be differentiated from pancreatic

adenocarcinoma to avoid unnecessary surgical procedures, since both diseases have similar clinical and imaging presentation.

The differential diagnosis is clinically difficult, and dynamic contrast-enhanced MDCT and MR imaging both have an important role. Furthermore, MDCT and MR imaging can identify the extrapancreatic manifestations of AIP, most commonly biliary, renal and retroperitoneal. Finally, in all cases of uncertain diagnosis, MDCT and/or MR follow-up after short-term treatment (2-3 weeks) with high-dose steroids can identify a significant reduction in size of the pancreatic parenchyma and, in FAIP, normalisation of the calibre of the upstream main pancreatic duct".

Lee-Felker SA et al[73] (2015) conducted a study on Contrast enhanced MDCT of 91 patients, which included 39 with autoimmune pancreatitis, 25 with pancreatic ductal adenocarcinoma, 27 with acute interstitial pancreatitis. They stated that autoimmune pancreatitis can be accurately differentiated from pancreatic ductal adenocarcinoma and acute interstitial pancreatitis on the basis of characteristic MDCT features as follows :

1. Sausage shape, low-attenuation halo, and absence of a pancreatic duct or biliary dilatation differentiated autoimmune pancreatitis from pancreatic ductal adenocarcinoma.
2. Sausage shape and absence of peripancreatic stranding differentiated autoimmune pancreatitis from acute interstitial pancreatitis.

LITERATURE ON HEREDITARY PANCREATITIS :

Lesniak RJ, Hohenwalter MD, Taylor AJ (2002) [74] in there study stated that CT showed parenchymal and intraductal calcifications occur in approximately 50% of hereditary pancreatitis patients. Intraductal calculi occur early in the course of the

disease; they tend to be large and rounded and are arranged in a linear pattern in the dilated main pancreatic duct. Pseudocysts may be seen.

A K P Shanbhogue et al[75] (2009) in their article stated that hereditary pancreatitis is seen in young age at onset, at least two acute attacks of pancreatitis with no underlying cause, family history of pancreatitis in a first- or second-degree relative. Imaging findings are nonspecific in acute conditions and in chronic there may be significant pancreatic atrophy, pancreatic calcifications, and calculi.

Ricardo Restrepo et al[76] (2016) in their article "to provide updates on acute pancreatitis in children regarding the imaging findings, causes, and complications stated that the incidence of Acute pancreatitis is increasing in children, imaging plays an important role in the diagnosis of acute pancreatitis and hereditary pancreatitis is an autosomal-dominant disease is nearly indistinguishable clinically or by imaging from other causes aside from early age of onset, with recurrent events occurring during the first decade of life and most often with a family history".

LITERATURE ON GROOVE PANCREATITIS :

Ankur Arora et al[77] (2015) in their study involving imaging of 33 patients with paraduodenal pancreatitis(PP) concluded that PP is a unique variant of chronic pancreatitis seen in men in their fourth or fifth decade with a history of chronic alcohol abuse and/or smoking, who present with recurrent episodes of upper abdominal pain often accompanied by obstructive GI symptoms. Improved diagnostic techniques such as MDCT, MRI/MRCP, and EUS can superiorly delineate the duodenal and juxtaduodenal abnormalities encountered in PP, which include medial duodenal wall thickening exhibiting increased enhancement, intramural and/or paraduodenal cysts, with or without a plate-like scar tissue in the groove region, which may at times contiguously involve the pancreatic head. As opposed to groove

pancreatic carcinoma, the peripancreatic arteries are neither infiltrated nor attenuated/encased; rather they get medially displaced.

Abd El-Aziz Mohamed El-Nekidya et al[78](2016) in there study involving contrast enhanced MDCT of 16 patients of groove pancreatitis had features of hypodense sheet in the pancreaticoduodenal (PD) groove in 12 patients with mild enhancement in the delayed phase seen in 6 of the them. Duodenal wall thickening was seen in 10 patients while associated cysts within the duodenal wall or in PD groove were seen in 6 patients and pancreatic head enlargement in 8 patients.

Reham M.Khalil et al[79] (2017) in their study involving histopathologically confirmed 15 groove pancreatitis (GP) cases to highlight the MDCT features , pure & segmental forms were seen in 6 & 9 patients. The most frequent findings noted were :

- Medial duodenal wall thickening & cysts,
- Duodenal luminal narrowing,
- Regional lymphadenopathies,
- Pancreatic involvement,
- Isolated groove affection,
- Pancreatic calcifications,
- Distal CBD narrowing & pancreatic duct abnormalities,
- Retro-peritoneal stranding.

LITERATURE ON TUBERCULAR PANCREATITIS :

Falkowski AL et al[80] (2013) in their case report on isolated tubercular pancreatitis stated that incidence is rare per se and radiological findings may include :

- Mostly solitary lesions with multiple cystic components, located in the pancreatic body or head and/or in peripancreatic lymph nodes.

- The cystic components of the lesion itself are typically hypoechoic (sometimes hypo-isoechoic) on ultrasound, hypodense on CT, hypointense on T1-weighted MRI and hyperintense on T2-weighted MRI.
- Contrast enhancement occurs in septations and also rim enhancement in the peripancreatic lymph nodes.
- Pancreatic duct is typically not dilated.
- The appearance of the pancreas may be heterogeneous, typically without calcifications.
- Associated findings might be ascites, mural thickening of the ileocecal region, peritoneal, mesenteric masses and splenic and/or hepatic lesions.

Chhaya J Bhatt, Kavita Vaishnav[81] (2014) in their study of MDCT in 20 patients of pancreatic masses which included 2 patients with pancreatic tuberculosis showed hypo dense mass on MDCT with no significant enhancement or peripheral enhancement or multiloculated appearance, and no surrounding invasion.

LITERATURE ON PANCREATIC NEOPLASMS

Majority are of epithelial origin of which 90% are duct cell adenocarcinoma and its variants. Endocrine cell tumors are the other major epithelial neoplasms arising from the Islets of Langerhans. The rest of the neoplasms are rare.

- **PANCREATIC ADENOCARCINOMA :**

Patric Freeny et al (1988)[82] In a study of 174 patients diagnosis of having pancreatic ductal adenocarcinoma, using dynamic CT (contrast 180-200m1) a correct diagnosis was made in 91% with 8% false positive and 1% false negative diagnosis.

They found the following CT features:

Pancreatic mass was founded in 96% patients with the mass being focal in

95% and diffuse in 5%. Of the focal masses 62% were in the head, 26% body & 12% tail. 83% of the focal masses showed diminished central enhancement with average attenuation value of 36 HU, while normal pancreatic parenchyma had average attenuation value of 87 HU. Upstream dilatation of the main pancreatic duct was noted in 68% patients and was smooth and parallel and was associated with parenchymal atrophy in 82%. Intrahepatic and 7 or extrahepatic biliary duct dilatation was seen in 58%. Isolated biductal dilatation without evidence of a focal mass as a sign of pancreatic carcinoma was noted in 4% patients.

They concluded that staging with CT was more accurate than with angiography and that CT staging criteria were reliable as no unresectable tumour based on CT was found to be resectable and only 3 of the 9 resectable tumours based on CT were found to be unresectable.

Scott J. Schulte et al (1991)[83] found that streaky infiltration of the fat surrounding the root of the superior mesenteric artery, periarterial lymph nodes visualization, focal mass within 1 cm of the root, mass obliterating the periarterial fat and circumferential encasement of the SMA root, were not specific for pancreatic neoplasm, but were also noted in acute pancreatitis, chronic pancreatitis and pancreatic abscesses.

Alec Megihow et al (1992)[84] concluded that thin slice thickness (5mm) CT was the primary diagnostic technique in the evaluation of pancreatic adenocarcinoma: A dynamic incremental bolus technique using intravenous contrast material, (150m1. of 60% ionic contrast material) allowed better evaluation of small hypo attenuating lesions, assessment of vascular encasement, maximum conspicuity of detectable hepatic metastasis than 10mm thick non-contiguous slices CT with contrast enhancement by drip infusion. Pancreatic adenocarcinomas appeared as a mass distorting the contour of the gland, which is hypo attenuating due to its schirrous

nature. Contrast enhancement allowed more accurate assessment of the tumour spread within the pancreas.

David S K Lu et al (1997)[85] studied 25 patients "with pancreatic adenocarcinoma by preoperative pancreatic-phase thin-section helical CT (40- to 70-sec delay. 2.5- to 3-mm collimation). Tumour involvement of the portal and superior mesenteric veins and the celiac, hepatic, and superior mesenteric arteries was prospectively graded on a 0-4 scale based on circumferential contiguity of tumour to vessel. Subsequent surgical results were then correlated with the CT grades who underwent local dissection during curative or palliative surgery also and they found involvement of vessel to tumour that exceeds one- half circumference of the vessel on CT is highly specific for unresectable tumour".

Oswald Graf et al (1997)[86] studied "40 patients of pancreatic adenocarcinomas with dual-phase helical CT (3-mm collimation: 1-mm overlapping reconstructions: 160 ml contrast medium injected at 4 ml/sec: scan delay: 18 sec for arterial phase. 60 sec for portal venous phase). Tissue enhancement and differences in tumour-to-pancreas contrast were compared. They reported that arterial phase helical CT in adenocarcinoma is of limited benefit. Lesion conspicuity is suboptimal and depiction of venous anatomy r is inferior to the depiction possible with venous phase helical CT".

Tatsuya Tabuchi et al (1999)[87] studied CT of 25 pancreatic adenocarcinomas in both early- and late-phase and compared it with surgical-pathologic findings. They reported that early phase CT was better than late phase CT in identifying and characterizing lesion. They noted that tumor detectability was 96 % on early phase CT and 64% on late phase CT. Sensitivity of anterior serosal wall invasion and vascular invasion is good with early CT.

Giles W. Boland et al (1999)[88] had done dual-phase thin-section dynamic helical CT using a pancreatic-phase and portal vein-phase protocol in 41 patients with pathologically proven pancreatic adenocarcinoma. The scan delay after initiation of the contrast bolus was 40 sec for the pancreatic phase and 70 sec for the portal vein phase. They observed that mean differences of enhancement between tumor and normal pancreas were significantly greater in the pancreatic phase (57 HU) than the portal vein phase (35 HU.) (p =.0001). Enhancement values of all the critical vascular structures were also significantly greater in the pancreatic phase than the portal vein phase.

Carlos Valls et al (2002)[89] had done CT of 76 patients with suspected pancreatic cancer for preoperative evaluation and staging with dual phase helical CT (3-mm collimation for pancreatic phase, 5-mm collimation for portal phase). Iodinated contrast material was injected IV (170 mL at a rate of 4 ml/sec); acquisition began at 40 sec during the pancreatic phase and at 70 sec during the portal phase. They observed that dual phase helical CT is useful for preoperative staging, but the major limitation with it is it may not detect small hepatic metastases.

Joel G. Fletcher et al (2003)[90] conducted a study involving 39 patients "suspected of having resectable pancreatic adenocarcinoma underwent triple-phase multi–detector row CT. They found that

-Mean tumor-to-gland attenuation difference was greatest on images obtained in the pancreatic phase .

-For tumor detection, sensitivity of the images obtained in pancreatic (0.97% [29 of 30]) and hepatic (0.93% [28 of 30]) phases was superior to that of those obtained in arterial phase (0.63% [19 of 30]).

-For vascular invasion detection, sensitivity of images obtained in the hepatic phase

(0.83) was better than that of those obtained in the pancreatic (0.58) and arterial (0.25) phases.

-Images obtained in the pancreatic phase demonstrated more flow artifacts and decreased attenuation in the superior mesenteric vein, compared with the artifacts revealed on images obtained in the hepatic phase

They concluded that routine acquisition of images in the arterial phase is unnecessary for detection of pancreatic adenocarcinoma. Images of the pancreas obtained in the hepatic phase with multi– detector row CT most accurately display vascular invasion".

Clare J. Roche et al (2003)[91] reported after prospective studying 62 patients with dual phase contrast CT and suggested that CT is not accurate overall for prediction and nodal involvement in resectable pancreatic ductal adenocarcinoma.

Rafael Vargas et al (2004)[92] conducted a study "on imaging findings related to vascular invasion and overall tumor resectability in 25 patients who underwent contrast-enhanced biphasic MDCT evaluation.

On MDCT 23 (92%) of 25 patients were deemed to have resectable pancreatic adenocarcinoma. The tumors in the remaining two (8%) were considered not resectable because of the presence of vascular invasion (which was confirmed in only one patient at surgery). Of those 23 patients deemed to be candidates for curative resection on the basis of MDCT results, 20 were found to have resectable adenocarcinoma at time of surgery, yielding a negative predictive value for MDCT of 87% (20/23 patients) for overall resectability. In the other three patients, adenocarcinoma was deemed to be unresectable because of small metastases to the liver (two patients) or to the peritoneum (one patient) discovered at surgery.For detection of vascular invasion, MDCT yielded a negative predictive value of 100%

with no false-negative findings and an accuracy of 99%.

The study concluded MDCT has excellent negative predictive value for vascular invasion and good negative predictive value for overall tumor resectability in patients with pancreatic adenocarcinoma, suggesting an improvement over previous results reported using single-detector".

Raptopoulous et al (2005)[93] arterial phase scanning is more optimal for the detection of small pancreatic adenocarcinomas due to the delay of tumor enhancement compared to pancreatic parenchymal enhancement. This was more often documented in smaller tumours than in larger ones.

CT.Imbriaco et al (2005)[94] concluded that "thin section single phase (portal phase) MDCT is an accurate technique for diagnosis and assessment of resectability with pancreatic neoplasm, after studying 71 patients with suspected pancreatic neoplasm. The technique provides optimal tumour-to-pancreas contrast and maximal pancreatic parenchymal and peripancreatic vascular enhancement"

Tomoaki Ichikawa et al (2006)[95] conducted a study "to evaluate the individual contributions of arterial, pancreatic parenchymal, and portal venous phase (PVP) images and the utility of coronal and sagittal multiplanar reformatted (MPR) images in the assessment of pancreatic adenocarcinoma using triple-phase MDCT in 31 patients with and 35 patients without pancreatic adenocarcinoma.

The image set composed of coronal and sagittal MPR images and of axial images obtained in all phases had a significantly higher sensitivity than the other image sets(93.5%). The sensitivity of the arterial phase image set (80.6%) was significantly lower than that of all other image sets.

They concluded that combination of pancreatic parenchymal phase and PVP imaging is necessary and efficient for the assessment of pancreatic adenocarcinoma.

The addition of coronal and sagittal MPR images increased the performance of MDCT, especially in the evaluation of local extension".

H Li et at (2006)[96] mentioned various signs of vascular invasion by pancreatic adenocarcinoma. These include

• Invaded veins showed more stenosis or obliteration compared to invaded arteries.

• Irregularity in vein wall also noted.

Swati D. Deshmukh et al (2010)[97] mentioned that pathways of extrapancreatic perineural invasion by pancreatic adenocarcinoma with 3D volume-rendered MDCT.

Takeshita K et al[98] (2010) conducted a study "to assess the enhancement pattern of early pancreatic cancer using contrast enhanced MDCT which involved 8 patients. The MDCT evaluation covered diameter, stenosis or obstruction of main pancreatic duct (MPD) ,loss of normal lobar texture and associated pancreatitis. Attenuation difference between normal pancreatic parenchyma and the tumor (AD-PT) were measured. Focal stenosis or obstruction of MPD with dilatation of distal MPD was demonstrated in all patients. Associated pancreatitis occurred in 6 patients with tumors measuring 12 mm or greater. Loss of normal lobar texture was recognized in four cases with the tumor measuring 14mm or greater. Statistically, low attenuated lesions and high attenuating lesions differed with respect to tumor size ($p<0.01$) and a positive relationship was demonstrated between tumor size and AD_PT ($r=0.84$). In seven cases, AD-PT is higher during the arterial phase than the pancreatic phase. They concluded early pancreatic cancer appears as low attenuating on early phase, high to iso attenuation during pancreatic and delayed phases in respect to the tumor size. Focal stenosis or obstruction with distal dilatation of MPD seem important in diagnosis of early pancreatic cancer".

Mahmoud A D et al (2014)[99] conducted a study involving 20 patients with pancreatic

masses , who underwent non contrast & contrast enhanced MDCT. 8 patients were found to have adenocarcinoma ,cystic adenocarcinoma in 1 patient ,infiltrative adenocarcinoma in 2 patient , intraductal papillary mucinous tumor in 2 patients, mucinous cyst adenocarcinoma in 1 patient,mucinous cystadenoma in 4 patients and pesudocysts in 2 patients. According to MDCT criteria 6 patients were considered suitable for tumor resection and 14 inoperable patients, 1 of 6 was unresectable during operation due to invasion of SMV with infiltration of mesenteric root.

The study concluded that multiphasic MDCT is the choice of imaging for diagnosis and predicting pancreatic masses and resectability.

Anuraj A et al[100](2016) conducted a study "involving 72 consecutive patients with pancreatic cancer who underwent preoperative contrast enhanced triple phase MDCT-pancreatic protocol. Out of this 31 patients deemed resectable and underwent surgery. The operative resectability of tumour in terms of vascular invasion, local spread and abdominal metastasis was assessed. Of 31 tumours 25 were completely resected and six were found to be unresectable at surgery, yielding a positive predictive value of 80.6% with six false-negative results for overall resectability. The study concluded MDCT is an effective pre-operative tool for assessing resectability with a good positive predictive value for overall resectability in pancreatic adenocarcinoma".

Tadros MY et al[101](2017) conducted "a study which included 30 adult patients who underwent triple-phase multi-detector row CT using a 16-slice machine. 15 had pancreatic malignancies (14 adenocarcinoma of which 6 were resectable and 8 were irresectable, and 11 patients showed hypo dense lesions, while 3 cases showed heterogeneous focal lesions. 1 distant metastasis) proven at biopsy and/or surgery with overall accuracy of tumor staging was 84%. The most common reported associated extra pancreatic finding was dilated CBD and intra hepatic biliary radical,

which was seen in 6 patients (42%). Pancreatic parenchymal phase and the portal venous phase are more useful for studying malignancies.11 patients had pancreatitis (acute and chronic), study confirmed that the currently accepted CT severity index is indeed a powerful tool with which we can predict morbidity in patients with acute pancreatitis. Main pancreatic duct dilatation, intra ductal calcification and variable degrees of pancreatic atrophy were detected in all cases of chronic pancreatitis. Pancreatic pseudocyst was found in 33% of cases. 03 patients had cystic benign tumors (2 mucinous cystadenoma, 1 serous cystadenoma) and 01 patient had neuroendocrine tumor (insulinoma) insulinoma appeared as well defined small lesion which enhanced more intensely than the normal pancreatic parenchyma in all phases. They concluded contrast enhanced multiphase pancreatic imaging by MDCT with its post processing techniques represents the imaging modality of choice for diagnosis of different adult acquired pancreatic disease".

- **ENDOCRINE TUMOURS[11]**

E. A. Eelkema et at (1984)[102] the CT characteristics of 27 patients with non-functional islet cell tumours and reported the following:

A mass was identified in 25 patients (96%) of average size of 3-24 cm with 8 tumours (31%) being larger than 10 cm. Six tumours (22%) contained calcification. On contrast administration the tumours partially and diffusely became hyper dense relative to the nearby normal pancreatic tissue. Hepatic metastasis was identified in 15 patients (36%), regional lymphadenopathy in 10 patients (37%), atrophy of the gland proximal to the tumour in six (22%), dilation of the biliary ducts in five (19%) and dilation of the pancreatic duct in four (15%). They found that although at times it was difficult to distinguish pancreatic adenocarcinoma from nonfunctional islet cell tumours, the presence of a large pancreatic mass which shows calcification, contrast

enhancement of the primary tumour and metastasis, without involvement of the celiac axis and proximal superior mesenteric artery are characteristic features of the latter.

S. Wyatt & E. Fishman (1994)[103] stated that differentiating islet cell tumours from a pancreatic adenocarcinoma was important because the former generally shows a more favorable response to chemotherapy. Functioning tumours are usually less than 2cms and tend not to deform the pancreatic contour, so that administration of adequate IV contrast enhancement is critical for detection. Because of the high level of contrast enhancement offered by spiral CT, the technique is especially applicable to localization of these very small hypervascular tumours. Spiral CT also uses contiguous thin sections, which is critical for detection of such small tumours.

Levine van Hoe et al (1995)[104] in a study, of 10 patients with surgically proven islet cell tumours, performed arterial phase and parenchymal phase helical scans with 5mm collimation and overlapping image reconstruction. Nine of Eleven tumours could he located using two phase helical CT ;(sensitivity 82%), including one 4mm gastrinoma. Two lesions smaller than 5mm could not be visualized. They concluded that CT scans obtained in both arterial and parenchymal phases lead to improved detection of pancreatic islet cell tumours.

A. D. King et al (1998)[105] showed dual phase contrast enhanced spiral computed tomography has potential to improve detection of small insulin secreting islet cell tumour of pancreas.

Sheila Sheth et al (2002)[106] reported classic and most common enhancement pattern of islet cell tumours is hyper attenuating lesion in the arterial and venous phases and many small lesions enhance more prominently in the arterial phase or become inconspicuous in the venous phase.

Gallotti A et al (2013)[107] conducted a study on 60 patients “to evaluate the MDCT

features of incidentally detected neuroendocrine tumors (NETs) of the pancreas. Various MDCT features such as size, morphology, enhancement, and presence of calcifications were evaluated and were correlated with tumor biology on histopathology. A total of 32 of 60 (53%) NETs were nonbenign with a solid or complex pattern. The presence of calcification, local invasion, main pancreatic duct dilatation, vascular invasion, and lymph node enlargement along with angioinvasion and a Ki-67 index greater than 2% on histology were associated with a nonbenign diagnosis and a higher risk of recurrence. They concluded that approximately 50% of incidental NETs show uncertain or malignant behavior, solid tumors 3 cm or larger are commonly nonbenign; however, about 30% of tumors smaller than that size cutoff can be malignant. Nonbenign tumors and those with invasive features on MDCT have a higher incidence of recurrence".

Tadros MY et al[101](2016) in his study of 30 adult patients who underwent triple-phase MDCT using a 16-slice machine reported that 01 patient had neuroendocrine tumor (insulinoma) that appeared as well defined small lesion which enhanced more intensely than the normal pancreatic parenchyma in all phases. They concluded "multiphase pancreatic imaging by MDCT with its post processing techniques represents the imaging modality of choice for diagnosis of adult pancreatic endocrine tumors".

- **LYMPHOMA:**

Elmar M. Merkle1 et al[108] (2000) in their paper about "imaging findings of lymphoma in various modalities stated that in patients with primary pancreatic lymphoma, no marked pancreatic ductal dilatation is present even with ductal invasion as opposed to adenocarcinoma. Lymph node involvement below the level of the renal veins was another finding not seen with adenocarcinoma. Clinical and

imaging findings are otherwise not specific in the differentiation of pancreatic lymphoma and pancreatic cancer, but a bulky homogeneous tumoral mass without alteration of Wirsung's duct or the peripancreatic vessels should suggest the diagnosis. In patients with diffuse infiltration of the pancreatic gland without clinical signs of pancreatitis, the radiologist should be alert to the possibility of pancreatic lymphoma".

Enrico Boninsegna et al[109] (2018) in their study on 14 pathologically proven cases of lymphoma described the imaging characteristics of lymphoma using contrast enhanced MDCT as large mass lesion with delayed homogeneous enhancement; peri-pancreatic fat stranding and vessel encasement without vascular infiltration ,enlarged lymph nodes & pancreatic duct dilatation rarely.

- **ACINAR CELL CARCINOMA:**

Servet Tatli et al[38] (2005) stated that pure acinar cell carcinoma of the pancreas is usually an exophytic, oval or round, well-marginated, and hypovascular mass. It typically is completely solid when small and contains cystic areas due to necrosis when large.

Li Tian et al[110] (2015) in their study involving 17 patients of "pathologically proven PAAC they retrospectively reviewed clinical features, CT/MRI findings to improve the accuracy of imaging diagnosis. The median age of the patients was 56 years .The tumors were located in any part of the pancreas or exophyitc growth, with a median maximal diameter of 68 mm. Thirteen masses presented with ovoid shape. Nine masses had less clear boundaries. Eleven masses showed a variable degree of intratumoral hypodense or necrosis before contrast administration on CT images. Five masses showed hypointense on unenhanced T1 weighted images and hyperintense on T2 weighted images. After contrast administration, the most common enhancement pattern was slight enhancement on arterial phase and persistent enhancement on portal

vein phase. Infiltration of tumor into duct and vessels was not common. Five and 2 patients developed hepatic metastasis and local lymphadenopathy, respectively. By the end of the last follow-up, 11 patients survived free of disease.

PAAC should be included in the differential diagnosis when a bulky, ovoid, heterogeneous mass, with clear or less clear margins, in the pancreas or peripancreas, with slight and persistent enhancement after contrast administration on CT or MRI images is seen, particularly in elder men".

Wang Q et al[111] (2016) conducted a study "involving 43 patients to evaluate and describe the computed tomography (CT) features of ACC and compare with pancreatic ductal adenocarcinoma (DAC) for improving preoperative diagnosis. The control group consisted of 34 patients with DAC. The CT imaging from nine patients with pathologically confirmed ACC was retrospectively reviewed. The tumor location, size, texture, and enhancement patterns are analyzed. They found that 64.3% (9/14) of ACC tumors were homogeneous and 35.7% (5/14) had necrosis. The percentage of common bile duct and pancreatic ductal dilation was 14.3% (2/14) and 7.1% (1/14), respectively. The mean size of ACC was 50.1±24.2 mm. The mean attenuation of ACC was 35.4±3.9 Hounsfield unit (HU) before enhancement, 73.1±42.9 HU in arterial phase, and 71.8±15.6 HU in port venous phase. It is difficult to distinguish ACC from DAC preoperatively only based on CT findings. However, compared with DAC, ACC tumors are likely to be larger and contain more heterogeneous intratumoral necrotic hypovascular regions, and less pancreatic ductal and common biliary dilation".

- **INTRAPANCREATIC METASTASES:**

Ioannis Tsitouridis et al[112] (2010) in their study of 11 patients found male predominance , lung carcinoma in 7 patients, breast carcinoma in 3 patients and renal

cell carcinoma in 1 patient with Ct characterisation involving solid and cystic attenuation, metastases to other organs in 7 cases, lymph node involvement, few showing homogenous and rim enhancement in few cases. Thus stating that CT can be used for proper characterisation and diagnosis of pancreatic metastases.

Hong-yuan Shi et al[113] (2015) in their "retrospective study of 18 patients with 36 histopathologically proven pancreatic metastases aimed to identify the computed tomography (CT) imaging findings. The primary malignancy included lung (n=7), gastrointestinal (n=5), renal (RCC) (n=3), osteosarcoma (n=1), cardiac sarcomas (n=1), and neuroendocrine ethmoid sinus carcinoma (n=1). Tumor markers were elevated for 8 patients.

- Metastases from NSCLC and gastrointestinal carcinoma frequently presented as small well-circumscribed lesions, with homogeneous or rim enhancement, and or local pancreatic infiltration instead of focal mass, mimicking local pancreatitis.
- Neuroendocrine ethmoid sinus carcinoma affecting the pancreas also exhibited local pancreatic infiltration.
- Metastases from RCC and cardiac sarcomas had typical characteristics of hypervascular lesions.
- Osteosarcoma metastasizing to pancreas had special manifestation, that is, cystic lesion with thick wall and calcification".

Hossain MS et al[114] (2016) conducted a study of 47 patients to assess "Role of MDCT scan in the evaluation of pancreatic mass with histopathological correlation. Of which 33 are malignant and 14 are benign masses. Out of 33 malignant cases, 17 (36.2%) had carcinoma pancreas, 13 (27.7%) had carcinoma with metastasis and 3 (6.4%) patients had extrapancreatic malignancy (lymphoma and ampullary growth). They concluded that excellent soft tissue resolution, better evaluation of

peripancreatic fat plane disruption or fascial plane thickening and extension or invasion of growth proved CT scan may be a useful tool for assessing and characterization of pancreatic mass lesions".

- **CYSTADENOMAS (SEROUS & MUCINOUS) OF PANCREAS :**

Wyatt & Fishmann et al[103] (1994) found that "cystic neoplasms of the pancreas, which are relatively uncommon, are either microcystic (formerly serous) or macrocystic (mucinous) cystadenoma or cystadenocarcinoma.

- ❖ Microcystic tumours are approximately 5cm in size, have no malignant potential, predominate in the pancreatic head, vary in appearance from a solid mass to multiple small cysts to a multilocular mass and may have a central scar with stellate calcifications.
- ❖ Macrocystic tumours are potentially malignant and found in the pancreatic body and tail. They are of size 10cm or greater and tend to be multiloculated with lager cysts and thicker septae, which may have calcification or mural nodules.

Because of the optimal parenchymal enhancement spiral CT can clearly depict the tumour spread and tumour margins within the gland. These tumours may show displacement or invasion of neighbouring structure and which are evaluated clearly with spiral technique. Hence spiral CT finding really enable improved surgical planning".

S Y Back et al[115] (2000) reported a case of serous cystadenoma showing diffuse involvement of pancreas except part of head which is replaced by islet cell tumour. On imaging serous cystadenoma appears as multiple cysts while islet cell tumour appears as solid mass with calcification.

Sahani DV et al[116] (2005) in their study of evaluation of cystic masses of pancreas using MDCT and MRI characterized cystic lesions morphologically into four types

viz unilocular cyst, microcystic cyst(serous), macrocystic (mucinous) and cyst with solid component. They further described the characteristic features of serous and mucinous cystadenoma and their appearances using MDCT & MRI.

Dawoud MA et al[99] (2014) in their study of 20 patients to "determine the role of MDCT in evaluation and prediction of pancreatic tumors resectability reported 14 malignant cases and 4 mucinous cystadenoma and 2 pseudocyst cases. As per MDCT criteria 6 patients were considered suitable for tumor resection and 14 patients were considered inoperable with unresectable tumor. They concluded that multiphase MDCT imaging is the choice for diagnosis and prediction of pancreatic tumor resection".

Botch et al[117] (2015) in their review article on pancreatic masses using "64 slice MDCT described characteristic appearance of various solid & cystic pancreatic neoplasm and stated that acquisition of images in arterial, venous and delayed phases improves the accuracy of diagnosing unresectable pancreatic carcinoma and also helps in identifying indirect signs of a mass with no visible pancreatic contrast in the form of atrophic distal parenchyma, interrupted duct sign and mass effect". Findings of mucinous cystic neoplasm includes female preponderance of 40-60 years, large size cysts, common in head, internal septations and calcifications with no connection with MPD.

- **INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS (IPMN):**

Ling Tan et al[118] (2009) in their study of "20 pathologically proven IPMN cases (12-malignant & 8- benign) using MDCT stated that diameters of the cystic lesions and main pancreatic ducts (MPDs) were significantly larger in malignant IPMNs ($P < 0.05$). The combined-type IPMNs had a higher rate of malignancy than the other two types of IPMNs ($P < 0.05$). Tumors with mural nodules and thick septa had a

significantly higher incidence of malignancy ($P < 0.05$). From comparison with the pathological diagnosis, the sensitivity, specificity, and accuracy of MDCT in characterizing the malignancy of IPMN of the pancreas were determined to be 100%, 87.5% and 95%, respectively. They concluded MDCT with contrast study and reconstruction techniques can elucidate the imaging features of IPMNs and help predict the malignancy of these tumors".

Dushyant V. Sahani et al[119] (2011) conducted a study for "characterization of pancreatic cystic lesions in 114 patients using contrast enhanced MDCT . The radiologic accuracy (reader 1 and reader 2) for stratifying lesions into mucinous and nonmucinous subtypes was 85% and 82% and for recognizing cysts with aggressive biology was 86% and 85%, respectively.

Predictive values of MDCT were superior for

- Lesions > 30 mm and non mucinous lesions.

Features favoring aggressive biology were

- Main pancreatic duct dilation > 10 mm ($p < 0.0001$)
- Biliary obstruction ($p=0.01$), mural nodule ($p < 0.0001$)
- Main-duct intraductal papillary mucinous neoplasm ($p < 0.0001$)
- advanced age ($p = 0.0001$).

They concluded that morphologic features of pancreatic cystic lesions using contrast enhanced MDCT allow reliable characterization into mucinous and nonmucinous subtypes and enable prediction of biologic aggressiveness".

Jung Hoon Kim et al[120] (2013) carried a study of 38 patients with "surgically proven IPMNs with an associated invasive carcinoma (IPMC) using MDCT to assess the diagnostic accuracy of MDCT for determining the prognostic factors, including the T category, lymph node metastasis, tumor size, and perineural invasion. The

morphologic types of IPMC included the main-duct type (n = 11, 29%), combined type (n = 18, 47%), and branch-duct type (n = 9, 24%). The diagnostic accuracy for the T category was 73.7% (n = 28) and 68.4% (n = 26) and for the lymph node metastasis was 68.4% (n = 26) and 76.3% (n = 29), respectively. The areas under the receiver operating characteristic curve for perineural invasion were 0.868 and 0.821. The sensitivity, specificity, and positive predictive value were 100%, 71.4%, 55.5%, and 90%, 71.4%, 52.9%, respectively. Interobserver agreement was moderate (κ = 0.659). The main duct size was 11.5 + 6.2 mm. Mural nodules were detected in 74% (n = 28) of patients".

Hyo-Jin Kang et al[121] (2016) in their study of 129 patients with pathologically proved pancreatic IPMNs stated that diagnostic performance of MDCT and MRI with MRCP for identifying the malignant potential of pancreatic IPMNs was similar and showed good intermodality agreement, suggesting that follow-up with either modality may be used. Both modalities showed showed similar diagnostic performance in depicting the malignant potential of pancreatic IPMNs using features suggesting "higher malignant stigmata"(MPD size >10 mm and an enhancing mural nodule), "worrisome features"(MPD size of 5–9 mm, a cyst size >30 mm, a nonenhancing mural nodule, a thickened and enhanced cyst wall, and an abrupt pancreatic duct change with parenchyma atrophy), presence of a parenchymal mass and local-regional extension as overt signs of malignancy similar to high-risk stigmata can increase the overall diagnostic performance of invasive IPMN.

- **LITERATURE ON PANCREATIC TRAUMA:**

Bradley EL et al[122] (1998) in their retrospective study of blunt pancreatic trauma involving 237 data fields observed that isolated pancreatic injuries are rare, and associated injuries to other solid organs occur in over 90% of cases. Serum amylase

levels are neither sensitive nor specific. Involvement of MPD injury is associated with higher morbidity than without. They stated CT plays a better role in detection of parenchymal injuries however it is unreliable in diagnosing MPD injuries and should not be used to guide therpay.

Ulrich Linsenmaier et al[123] (2008) in their study on blunt trauma of pancreas and duodenum classified and graded the pancreatic & duodenal traumas, suggested MDCT protocols for evaluation of trauma cases, mechanism & patterns of injuries using MDCT, efficiency and reliability of MDCT in identifying subtle signs of injury and in potential complications of duodenal and pancreatic injuries.

Gordon RW et al[124] (2013) in their study of 53 patients with "suspected pancreatic injury having history of blunt abdominal trauma stated the MDCT imaging findings suggestive of pancreatic injury included low attenuation peripancreatic fluid (n = 51), hyperattenuating peripancreatic fluid (n = 13), pancreatic contusion (n = 7), active hemorrhage (n = 2), and pancreatic laceration (n = 16). There were highly sensitive, nonspecific imaging findings such as the presence of low attenuation peripancreatic fluid (sensitivity, 100 %; specificity 4.9 %) as well as insensitive, specific findings such as visualizing a pancreatic laceration involving >50 % of the parenchymal width (sensitivity, 50 %; specificity, 95.1 %). MDCT imaging findings can be grouped into two categories for determining integrity of the main pancreatic duct: indirect, highly sensitive but nonspecific findings and direct, specific but insensitive findings".

Shadab Maqsood et al[125] (2018) in their study involving 46 patients of blunt trauma using MDCT stated increase in incidence of pancreatic injuries(6.5%) compared to previous literature(2%) of blunt abdominal trauma. In his study 66% of pancreatic injuries were grade II injuries and 33.3% were grade I. The pancreatic injuries were associated with injuries to liver in 66.6% (2/3) of cases, injuries to kidneys in 66.6%

(2/3) of cases involving right and left kidney separately and injuries to spleen in 33.3% (1/3) of cases. They concluded that MDCT with reconstruction images helped in better diagnosis of complex injuries with improved accuracy, vascular involvement and also in course of management.

- **TECHNICAL ASPECT:**

In 1967, Josef Rosch published an article in AJR in which he mentioned that the pancreas was a difficult organ to image since it is situated deep in the retroperitoneal space.

HISTORY OF CT, SPIRAL CT & MDCT:

Wider availability and technical advances over the last two decades have made CT the imaging modality of choice for the evaluation of pancreatic pathologies[5]. "Since the introduction of computed tomography (CT) scan in late 1970s, there has been dramatic improvement in pancreatic imaging. With early conventional CT scanners, only 10-mm thick slices with a large acquisition time of 1 minute/slice were obtained; this resulted in motion artifacts and limited resolution[126].

Helical (spiral) CT scanners, introduced in late 1980s, allowed much faster data acquisition with a slice thickness of 1–2 mm and a volume data set for three-dimensional imaging. Power injectors were introduced allowing bolus contrast administration for fast dynamic scanning[126].

The better spatial resolution and dedicated pancreatic and portal venous phase (dual-phase helical CT) dynamic scanning increased the tumor conspicuity and allowed better detection and staging of pancreatic neoplasms. However, the multiplanar imaging still suffered from stair-stepping artifacts.

This drawback was overcome with the introduction of multidetector computed tomography (MDCT) in late 1990s. In contrast to single-detector helical CT scanners,

these scanners use multiple detector rows, are 10 times faster, and can obtain 16–256 slices per rotation at a slice thickness of 0.5 mm. The MDCT has improved volume coverage speed and spatial resolution along z-axis, and allows three-dimensional reformatting due to isotropic voxels and exquisite multiplanar reconstruction of pancreatic anatomy. High speed of MDCT also allows organ imaging in clearly defined perfusion phase"[126].

By late 1998, "all major CT manufacturers launched multiple row detector CT (MDCT) scanners capable of at least four slices per x-ray tube rotation. Major advantages of these scanners appear to be improved volume coverage and/or longitudinal spatial resolution.

Introduction of 16 section scanners with decreasing scan time, have yielded more thinner sections, improved z-axis spatial resolution. These scanners have led to number of new clinical applications especially in cardiac imaging. Different vendors have chosen different technological approaches to achieve their designs and, as a result, offer different sets of options to the user .

The following sections discuss key concepts, common to most multiple-row detector CT (MDCT) scanners, required to understand their functions and capabilities".

- Detector and Data Acquisition System (DAS)
- Sequential Scan Mode
- Helical Multisection Mode
- Helical pitch

Advantages of Multiple-row detector CT:

- Obtain large number of thin slices in both axial and longitudinal direction.
- Higher spatial resolution
- Improved reconstruction technique

- Fast imaging of large volume of tissue with variable slice thickness.
- Better utilization of x-ray tube.
- Minimal artefacts.

- **TRIPLE PHASE STUDY**

Multiphase CT is a commonly used "imaging technique for detection and preoperative staging of pancreatic carcinoma, identifying complications of acute pancreatitis, such as exudates, obstuction in chronic pancreatitis, pseudocysts, necrosis and abscesses.

Accurate preoperative evaluations of the degree of local tumor extension and peripancreatic vascular involvement are crucial for estimating the likelihood of benefit from surgical resection and the prognosis of patients with malignant pancreatic neoplasms[127].

Although it is suggested that single-phase scanning is effective for the diagnosis and assessment of resectability of suspected pancreatic carcinoma[127].CT images of the pancreas often are acquired at different phases of contrast enhancement—that is, at peak enhancement of the pancreas and peak enhancement of the peripancreatic vessels—to maximize the conspicuity of pancreatic tumors and visualization of peripancreatic vessels".

Triple phase study of pancreas includes image acquisition in arterial, pancreatic and portal phases with scan delay of 20,40,70 sec at an injection rate of 3-5ml/sec. Its uses are:

- *Arterial phase* - mainly peripancreatic vascularity, invasion and endocrine tumors.
- *Parenchymal phase* – mainly tumor detection and characterization.
- *Portal vein phase* - mainly for liver metastasis, vascular invasion.

With MDCT, a scan can be acquired at each phase within a few seconds, allowing completion of the entire scan while a substantial amount of contrast medium circulates and remains in the blood vessels and visceral parenchyma.[127]

Thus MDCT is well suited for multiphasic imaging of the pancreas.

- **ROLE OF IODINE CONCENTRATION IN CONTRAST:**

When imaging the abdomen, the depiction of parenchymal or soft tissue organs requires a certain amount of total iodine for appropriate scanning. The optimal amount of iodine should be approximately 35-45 g. When considering a median rate of 40 g iodine per imaging, the following volumes should be used: for lower-concentrated contrast agent (300 mgI/ml) an overall volume of 130 ml is necessary for adequate imaging quality; whereas for a concentration of 350 mgI/ml, 115 ml of contrast medium is needed; while for 400 mg I/ml, 100 ml is used.

To ensure adequate vessel opacification as well as soft tissue imaging with fast MDCT acquisitions, the iodine administration rate needs to be increased. This can be achieved either by an increase of injection flow rate or, more conveniently, by using a higher iodine concentration contrast medium.

Balthazar EJ, et al[17] (1994) in their review article stated that dynamic contrast material enhanced CT is the current standard imaging modality for the diagnosis and evaluation of acute pancreatitis. To enhance visualization of the pancreas and peripancreatic arterial architecture CT must be performed at the peak of the pancreatic arterial perfusion.

McNulty et al[128](2001) reported that “the multiphase imaging capability, increased speed of acquisition, and greater anatomic coverage achieved with MDCT have resulted in the need to redesign imaging protocols and pay more attention to bolus timing. Appropriate timing to achieve adequate contrast enhancement at each phase of

scanning is more difficult and critical in MDCT than in single-detector CT. Inappropriate timing reduces tumor conspicuity".

Shinagawa M et al[129](2003) conducted a study on "assessment of pancreatic CT enhancement using a high concentration of contrast material and stated that the pancreatic parenchymal phase and the portal venous phase are both significantly superior to the early arterial phase for maximum tumor-to-parenchymal contrast and tumor detection, and that the early arterial phase is not necessary to evaluate vascular encasement. The authors concluded that enhancement of the pancreatic parenchyma relies more on iodine dose delivered per second than the total iodine load".

Fenchel S et al[130] (2004) in their study compared "the effect of a high-concentration contrast material (400 mg I/mL) to a moderate concentration (300 mg I/mL) in 50 patients. The 50 patients were evenly divided between the two concentrations; six pancreatic cancers were included in the 300 mg I/mL group and five in the 400 mg I/mL group.

Equal iodine loads (39 g) were infused at 5 mL/sec. Arterial phase imaging was initiated at the time to peak aortic enhancement (mean, 17 sec) as dictated by a test bolus, followed by an acquisition between 50 and 70 sec from the initiation of contrast infusion.

They concluded that

- The higher concentration resulted in significantly higher pancreatic and tumor enhancement in the arterial and hepatic venous phases

- Improved delineation of tumor from surrounding tissue is found by using the 300 mg I/mL concentration and is likely the result of the fact that the mean attenuation difference of pancreatic parenchyma to tumor (tumor-to-pancreas contrast) was slightly higher with the lower concentration during both the arterial phase (35 H with

300 mg I/mL vs 29 H with 400 mg I/mL) and the 50- to 70-sec phase, where the difference in mean tumor-to-pancreas contrast between the two contrast concentrations was even greater (39 H with 300 mg I/mL vs 17 H with 400 mg I/mL).

- Higher pancreatic parenchymal and pancreatic tumor enhancement from the 400 mg I/mL concentration did not result in improved tumor conspicuity".

Hiroshi Kondo et al[127] (2007) conducted a "Three-phase MDCT study of the pancreas in 170 patients after administration of 2 mL/kg of 300 mg I/mL contrast medium injected at 4 mL/s to determine the optimal MDCT scanning delay for peripancreatic arterial, pancreatic parenchymal and venous contrast enhancement with a bolus-tracking technique.

Patients were prospectively randomized into three groups with different scanning delays for the three phases (arterial, pancreatic, and venous) after bolus tracking was triggered at 50 H of aortic contrast enhancement. The results were :

-Mean contrast enhancement in the aorta (change in attenuation, 321–327 H) and the superior mesenteric artery (change in attenuation, 304–307 H) approached peak enhancement 5–10 seconds after bolus tracking was triggered.

-Pancreatic parenchyma became most intensely enhanced (change in attenuation, 84–85 H) 15–20 seconds after triggering, and then the enhancement gradually decreased. Enhancement of the splenic vein and portal vein peaked 25 seconds and that of the superior mesenteric vein peaked 30 seconds after triggering.

- Liver parenchyma reached 52 H 30 seconds after triggering and reached a plateau (change in attenuation, 58–61 H) at a further scanning delay of 45–55 seconds.

The study concluded that protocol for optimal scanning delay after triggering of bolus tracking at 50 H of aortic contrast enhancement was 5–10 seconds for the

peripancreatic arterial phase, 15–20 seconds for the pancreatic parenchymal phase, and 45–55 seconds for the portal venous phase".

RESULTS & ANALYSIS

TABLE 6:
AGE WISE DISTRIBUTION OF ALL CASES

Age (years)	Total	Percentage (%)
<20	6	7.7
20-39	**30**	**38.5**
40-59	23	29.5
>60	19	24.3
Total	78	100

The ages of patients included in the study were in the range of 15 - 92 years with a mean age of 45 years. Maximum number of patients were under the age group of 20-39 years which consisted of 30 patients, accounting for ~38.5%. Patients in the age group of 20-59 years were 53 in number (68%) accounting for more than half the cases.

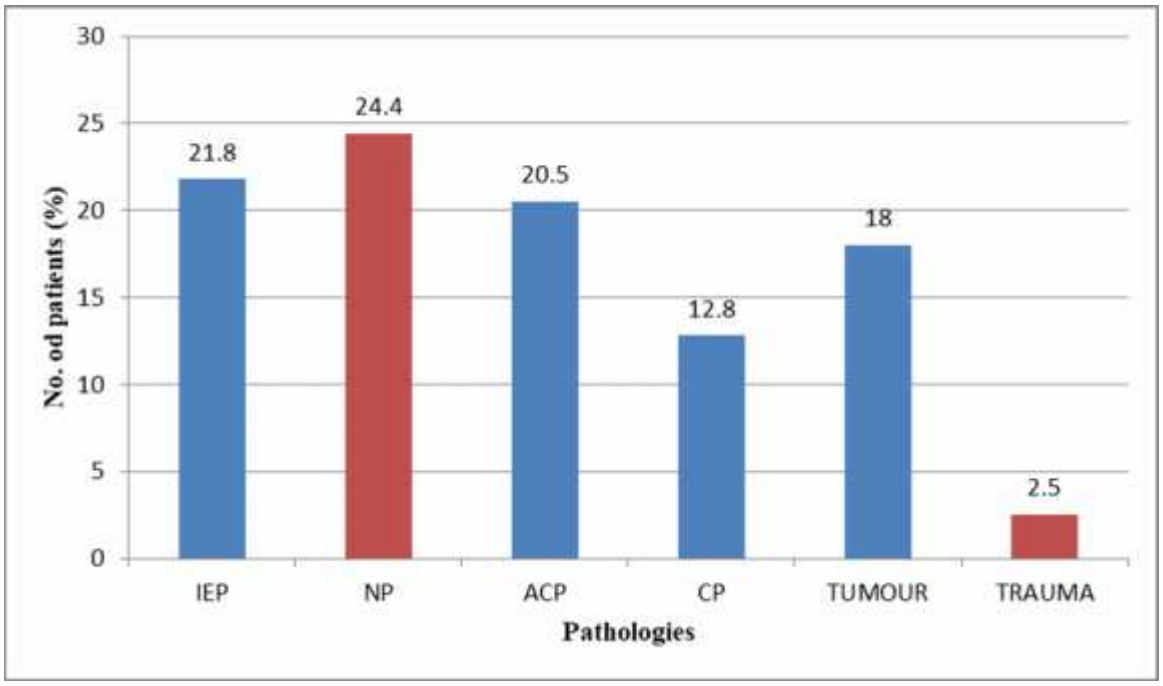
TABLE 7:
GENDER WISE DISTRIBUTION OF ALL CASES

Gender	Total	Percentage (%)
Male	**54**	**69.3**
Female	24	30.7

Of the 78 cases included in the study, males were 54 in number (~69%) , showing a strong male predilection, as opposed to only 31% of cases in the female category, with Male: Female ratio of ~2.2 :1.

FIGURE 6:

PATHOLOGY DISTRIBUTION OF ALL CASES



Of the 78 cases included in this study, Acute pancreatitis (n=36) i.e. 46% which includes 17 cases of interstitial oedematous pancreatitis (IEP) (22%) and 19 cases of necrotizing pancreatitis (NP) (24%), Acute on chronic pancreatitis (ACP) includes 16 cases (20.5%), Chronic pancreatitis includes 10 cases (12.8%), Tumour includes 14 cases (18%) and 2 cases of trauma (2.5%).

Hence acute pancreatitis was found to be the most common pancreatic pathology in our study accounting for ~46% of the cases and subtype of necrotizing pancreatitis (24%).

TABLE 8:
AGE WITH PATHOLOGY DISTRIBUTION

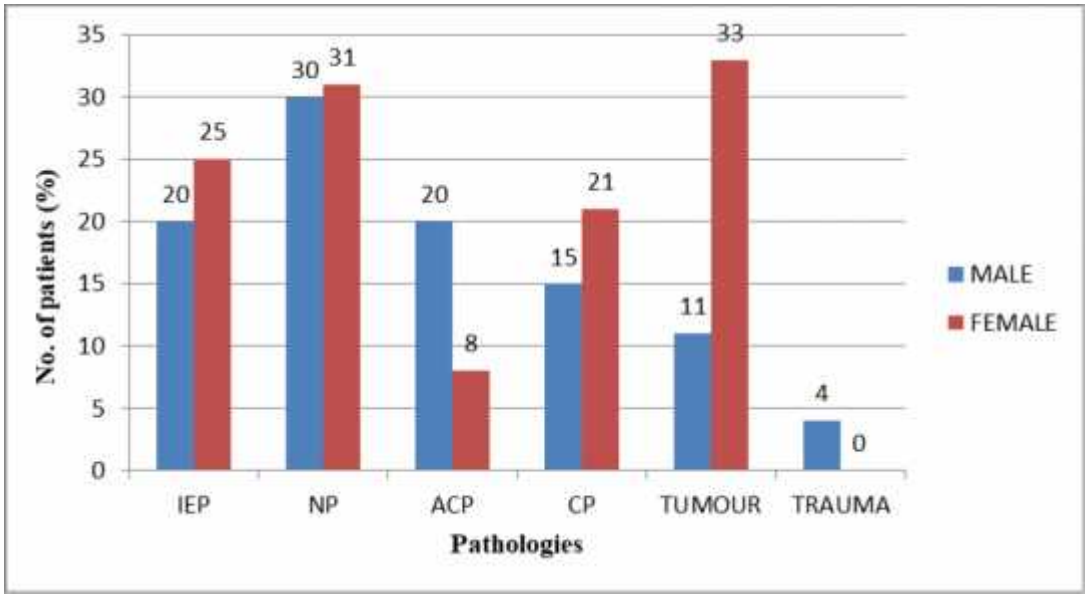
Age (years)	IEP	NP	ACP	CP	Tumour	Trauma
<20	2 (11%)	0	1 (6%)	2 (20%)	0	1 (50%)
20-39	**7 (41%)**	**12 (63%)**	**10 (63%)**	0 (0)	1 (7%)	0 (0)
40-59	4 (24%)	5 (26%)	4 (25%)	**4 (40%)**	5 (36%)	1 (50%)
>60	4 (24%)	2 (11%)	1 (6%)	**4 (40%)**	**8 (57%)**	0 (0%)
Total	17	19	16	10	14	2

Of the 78 patients in the study, maximum cases of Acute pancreatitis & Acute on chronic pancreatitis were under age group of 20-39 years, Chronic pancreatitis & Tumours were seen in age group of >60 years.

Hence in our study we found that increase in age increases the percentage of tumour occurrence predominantly after 60 years.

FIGURE 7:

GENDER WISE PATHOLOGY DISTRIBUTION



Of the 78 patients in the study, male predominance was noted in inflammatory and traumatic aetiology whereas female predominance was noted in neoplastic lesions.

Hence in our study females are more prone to develop neoplasm than males.

PANCREATITIS:

TABLE 9:

SERUM AMYLASE & LIPASE DISTRIBUTION IN PANCREATITIS:

Diagnosis	Serum Amylase & Lipase comparison					
	Increased Amylase	Normal (<125U/L)	Increased Lipase	Normal (<100U/L)	Total	Chi square test
IEP	**14 (82%)**	3 (18%)	**17 (100%)**	0(0%)	17	
NP	**18 (95%)**	1 (5%)	**19(100%)**	0(0%)	19	**P<0.001***
ACP	**11 (69%)**	5 (31%)	**15 (93.7%)**	1 (6.3%)	16	
CP	1 (10%)	**10 (90%)**	1 (10%)	**9 (90%)**	10	

Chi square test p<0.001(highly significant) stating that in interstitial oedematous, necrotizing pancreatitis and acute on chronic pancreatitis the values tend to increase whereas in cases of chronic pancreatitis the serum amylase and lipase value tends to be normal.

Among serum amylase and lipase, Lipase was seen elevated (more than three times) in more number of cases than amylase and thus stating serum lipase is more accurate than serum amylase for acute pancreatitis.

MDCT CHARACTERISTIC FEATURES IN PANCREATITIS

TABLE 10:

SIZE WISE DISTRIBUTION OF PANCREAS

Diagnosis	Size				Chi square test
	Bulky	Atrophy	Normal	Total	
IEP	**17 (100%)**	0 (0)	0	17	P<0.001*.
NP	**17 (89%)**	0	2 (11%)	19	
ACP	**11 (69%)**	1 (6%)	4 (25%)	16	
CP	0	**8 (80%)**	2 (20%)	10	
Total	45	9	8	62	

Chi square test p<0.001(highly significant) stating that in interstitial oedematous, necrotizing pancreatitis and acute on chronic pancreatitis the size of pancreas in bulky whereas in cases of chronic pancreatitis the predominantly tends to be atrophic. In patients with interstitial oedematous pancreatitis, necrotizing pancreatitis and acute on chronic pancreatitis 100%, 89% and 69% respectively, have bulky pancreas. In cases of chronic pancreatitis 80% of the patients have atrophic pancreas.

TABLE 11:

DISTRIBUTION OF FAT STRANDING, NECROSIS & PERIPANCREATIC COLLECTION IN PANCREATITIS

Diagnosis	Fat stranding		Necrosis		Peri pancreatic collection		
	Yes	No	Yes	No	Yes	No	Total
IEP	**17 (100*)**	0 (0%)	0 (0%)	17 (100%)	**14 (88%)**	3 (12%)	17
NP	**19 (100*)**	0 (0%)	**19 (100%)**	0 (0%)	**16 (84%)**	3 (16%)	19
ACP	**14 (88%)**	2 (12%)	**10 (63%)**	6 (37%)	8 (50%)	8 (50%)	16
CP	0 (0%)	**10 (100%)**	0 (0%)	**10 (100%)**	0 (0%)	**10 (100%)**	10
Total	52	10	29	33	38	24	62
Chi square test	P<0.001*		P<0.001*		P<0.001*		

Chi square test p<0.001(highly significant) stating that in patients of interstitial oedematous, necrotizing pancreatitis and acute on chronic pancreatitis signs of fat stranding, necrosis and peri pancreatic collections are noted whereas in cases of chronic pancreatitis they are not found.

In patients with interstitial oedematous pancreatitis, necrotizing pancreatitis and acute on chronic pancreatitis:

- 100%, 100% and 88% respectively have fat stranding.
- 0%, 100% and 63% respectively have necrosis.
- 88%, 84% and 50% respectively have peri pancreatic collections.

In cases of chronic pancreatitis no patients were found to have fat stranding/necrosis/peri pancreatic collection.

TABLE 12:
PSEUDOCYST & WALLED OFF NECROSIS DISTRIBUTION IN PANCREATITIS

Diagnosis	Pseudo cyst		Walled off necrosis		
	Yes	No	Yes	No	Total
IEP	1 (6%)	16 (94%)	0	17 (100%)	17
NP	2 (11%)	17 (89%)	**4 (21%)**	15 (79%)	19
ACP	**11 (69%)**	5 (31%)	0	16 (100%)	16
CP	**7 (70%)**	3 (30%)	0	10(100%)	10
Total	21	41	4	58	62
Chi square test	P<0.0001*		P=0.0215*		

Chi square test p<0.0001(highly significant) stating that in acute on chronic pancreatitis and chronic pancreatitis there is increase in occurrence of pseudocyst accounting to 69% and 70% of their cases respectively whereas fewer cases of interstitial oedematous and necrotizing pancreatitis were found to have pseudocyst.

Chi square test p=0.0215(highly significant) stating that walled off necrosis (n=4) is seen only in necrotizing pancreatitis whereas not seen in any case of interstitial oedematous, acute on chronic and chronic pancreatitis.

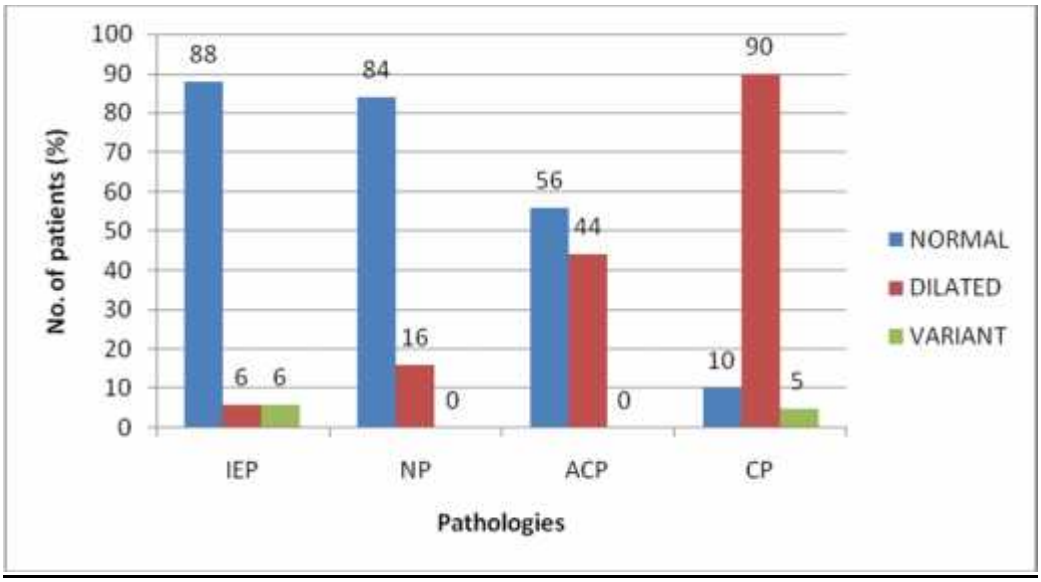
TABLE 13:
CALCIFICATION PATTERN DISTRIBUTION IN PANCREATITIS

Diagnosis	Calcification			
	IP	ID	IP AND ID	Total
IEP	0 (0%)	0	0	0
NP	0 (0%)	0	0	0
ACP	**5 (71%)**	0	2 (33%)	7 (54%)
CP	2 (29%)	0	**4 (57%)**	6 (46%)
Total	7 (54%)	0	6 (46%)	13

Of 62 cases of pancreatitis about 13 cases (7-ACP & 6-CP) showed calcification. Of these intra parenchymal (IP) calcification alone was seen in 7 cases (54%) whereas intra ductal (ID) & intra parenchymal together was seen in 6 cases (46%).

No cases of interstitial oedematous /necrotizing pancreatitis were found to have calcifications.

FIGURE 8:
DISTRIBUTION OF MAIN PANCREATIC DUCT FINDINGS IN PANCREATITIS



Chi square test p=0.002(highly significant) stating that Main pancreatic duct (MPD) is dilated 90% cases of chronic pancreatitis followed by 44% acute on chronic pancreatitis as opposed to very few cases of acute oedematous & necrotizing pancreatitis.

Of the 62 cases of pancreatitis in the study, MPD is dilated in 20 cases of which 45% cases are of chronic pancreatitis and variant ductal anatomy is noted in 1 case of interstitial oedematous pancreatitis.

TABLE 14:

MODIFIED CT SEVERITY INDEX (MCTSI) SCORE GRADING IN ACUTE PANCREATITIS

Diagnosis	Modified CT Severity Index				Chi square test
	0-2 (Mild)	4-6 (Moderate)	8-10 (Severe)	Total	
IEP	1(100%)	**16 (88.8%)**	0	17	P=0.001*
NP	0 (0%)	2 (11.2%)	**17 (100%)**	19	
Total	1	**18**	17	36	

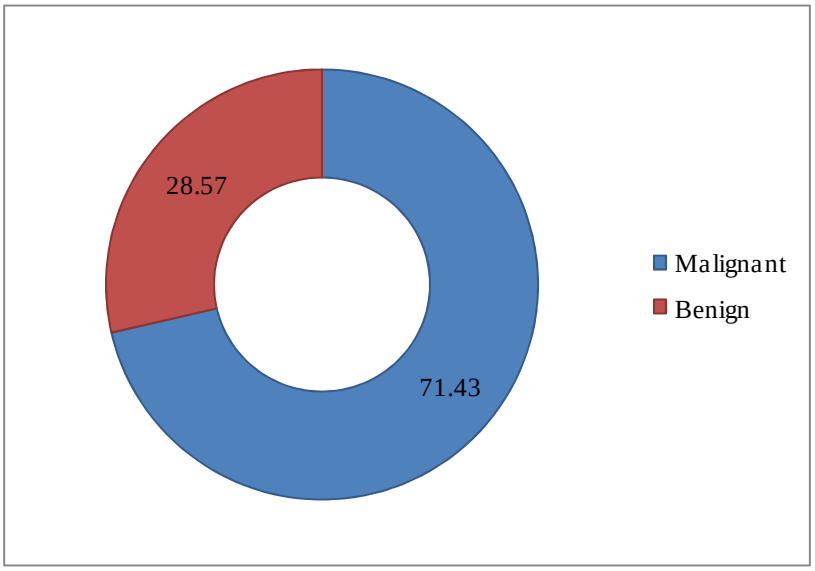
Chi square test p=0.001(highly significant) stating that majority of interstitial oedematous pancreatitis (94.1%) cases have fallen under moderate grade and majority of necrotizing pancreatitis (89.5%) under severe grade.

Of the 36 cases of acute pancreatitis in the study, majority of cases (50%) were found to be of moderate grade (MCTSI=4-6) and 47.2% were found to be of severe grade and 2.8% of mild grade.

PANCREATIC NEOPLASM:

FIGURE 09:

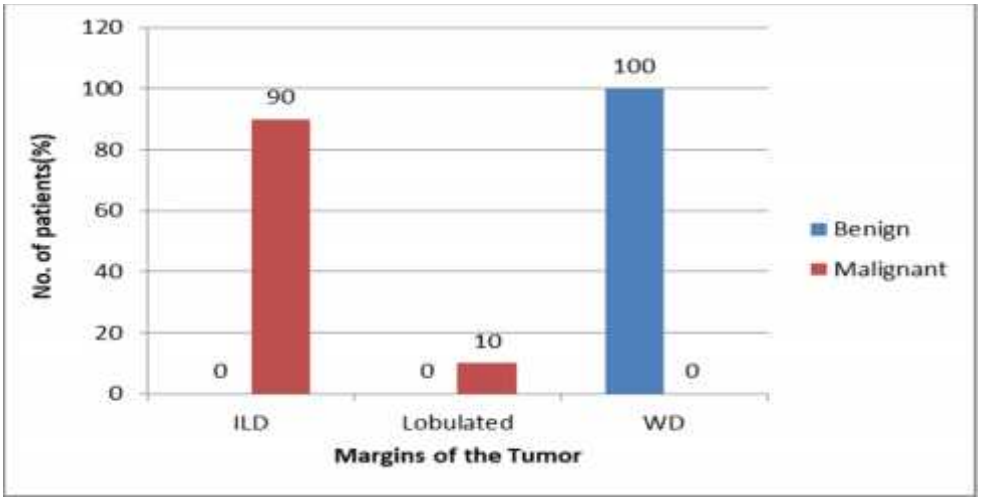
DISTRIBUTION OF TUMOUR NATURE PATTERN



Of the 14 patients of neoplasms in the study, majority (n=10) are of malignant nature (71.4%) and benign cases (n=4) accounted for 28.6%.

FIGURE 10:

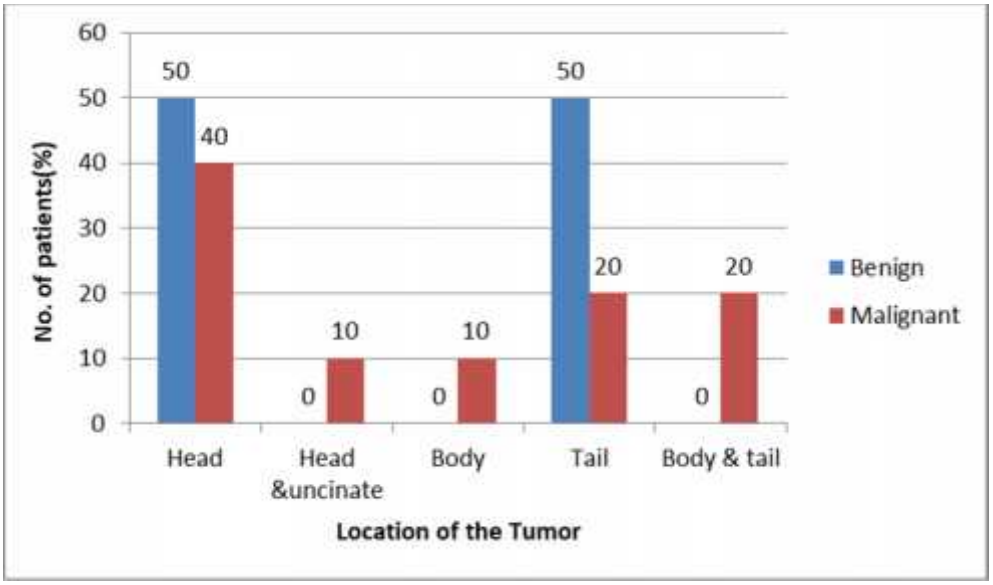
DISTRIBUTION OF TUMOUR MARGIN PATTERN



Chi square test p=0.0009 (highly significant) stating that majority of malignant cases (90%) have ill-defined margins, 10% cases show lobulated margins and all the benign cases (100%) have well defined margins.

FIGURE 11:

DISTRIBUTION OF TUMOUR LOCATION



Of the 14 neoplastic patients in the study, majority of cases (50%) involved head of pancreas followed by tail region (28%). Majority of malignant cases were seen involving head of pancreas (40%) whereas no specific pattern is seen in benign cases.

TABLE 15:

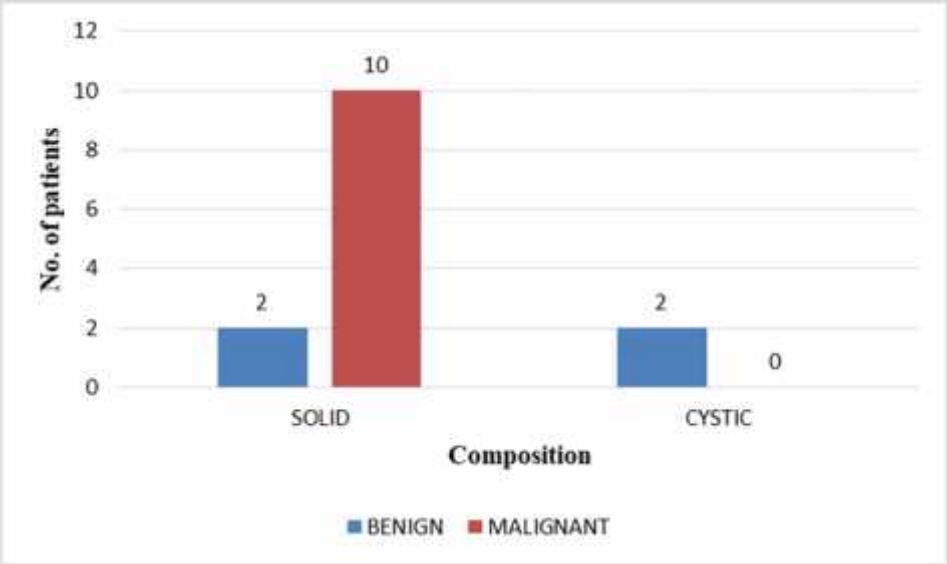
TUMOUR SIZE DISTRIBUTION

Size of the tumour (cm)	Benign	Malignant	Total
<2	0 (0%)	4 (40%)	4 (28.5%)
2-5	**3 (75%)**	2 (20%)	5 (36%)
5-10	1 (25%)	3 (30%)	4 (28.5%)
.>10	0 (0%)	1 (10%)	1 (7%)
Total	4 (29%)	10 (71%)	14 (100%)

Of the 14 neoplastic patients in the study, maximum cases (n=9) are of less than 5cm in size accounting to 65%.Majority of benign cases (75%) were of less than 5cm in size and no specific pattern of distribution is noted in malignant cases. However 4 out of 5 cases larger than 5cm in size are of malignant nature.

FIGURE 12:

TUMOUR COMPOSITION DISTRIBUTION



Chi square test p=0.0028 (highly significant) stating that majority of solid tumours (n=10) are malignant (83.3%) in nature compared to 16.7% benign cases (n=2).

Chi square test p=0.0455 (highly significant) stating that all cystic tumours (100%) are benign in nature.

All malignant tumours have solid composition in them whereas 50% of benign tumours are solid and 50% are cystic in composition.

TABLE 16:

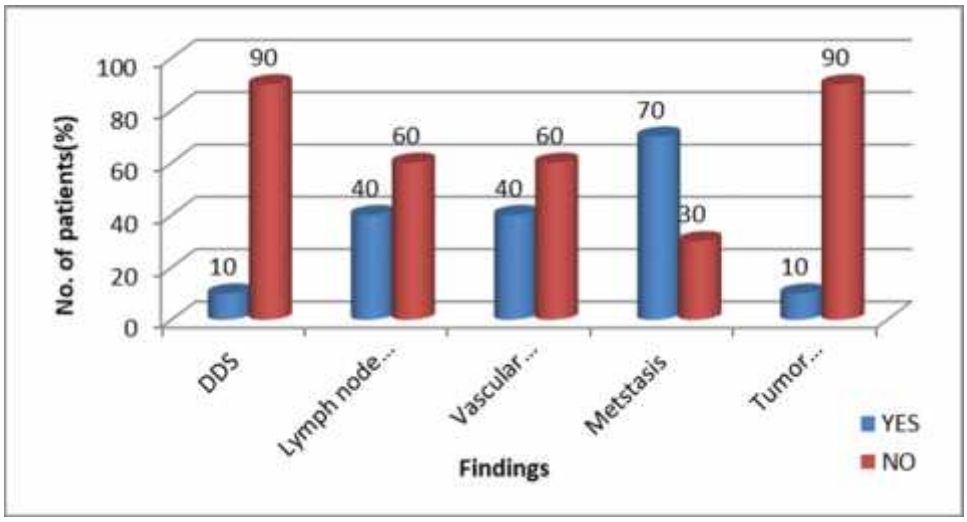
DISTRIBUTION OF CALCIFICATION PATTERN TUMOUR

Calcification pattern	Benign	Malignant	Total
Intra lesional	0 (0)	1 (10%)	1
Intra parenchymal	0 (0)	1 (10%)	1
Wall calcification	**2 (50%)**	0 (0%)	2
None	2 (50%)	8 (80%)	10
Total	4 (29%)	10 (71%)	14

About 50% cases of benign tumours showed wall calcifications whereas malignant cases showed intralesional (10%) and intra parenchymal (10%) calcifications with no calcifications in 80% cases.

FIGURE 13:

DISTRIBUTION OF OTHER MALIGNANT SIGNS



Of 10 malignant cases in the study, metastasis was seen in 70% cases, lymph node spread & vascular involvement in 40% each, double duct sign in 10% and non resectability in 90% cases.

TABLE 17:

DISTRIBUTION OF PROBABLE IMAGING DIAGNOSIS OF VARIOUS NEOPLASMS

Diagnosis	No. of patients	Percentage(%)
Benign		
Mucinous cystadenoma	2	14.3
Neuroendocrine tumour	1	7.1
Solid benign tumour	1	7.1
Malignant		
Adenocarcinoma	5	35.8
Lymphoma	1	7.1
Metastasis		
Liver	3	21.5
Gall bladder	1	7.1
Total	**14**	**100**

Of the 14 cases, adenocarcinoma is most common (35.8%) followed by liver metastasis (21.5%). In benign neoplasms mucinous cystadenoma was more common (14.3%).

TABLE 18:

MDCT FINDINGS IN TRAUMA CASES

CT Findings	No. of patients	Percentage(%)
Number of lesions		
1	1	50
>1	1	50
Location		
Head	1	50
Body/Tail	1	50
Contusion	2	100
Laceration	0	0
Other organ Injuries	2	100
Ascites	1	50
Vascular injury	1	50
AAST Grading		
I	0	0
II	1	50
III	1	50
IV	0	0

Of the 2 trauma cases in the study, multiplicity (>1 lesion) is seen in 50%, contusions in 100% cases, other organ injuries in 100% cases, ascites and vascular injury in 50% of cases each. 1 case was included under Grade II and 1 case under Grade III of AAST grading.

TABLE 19:

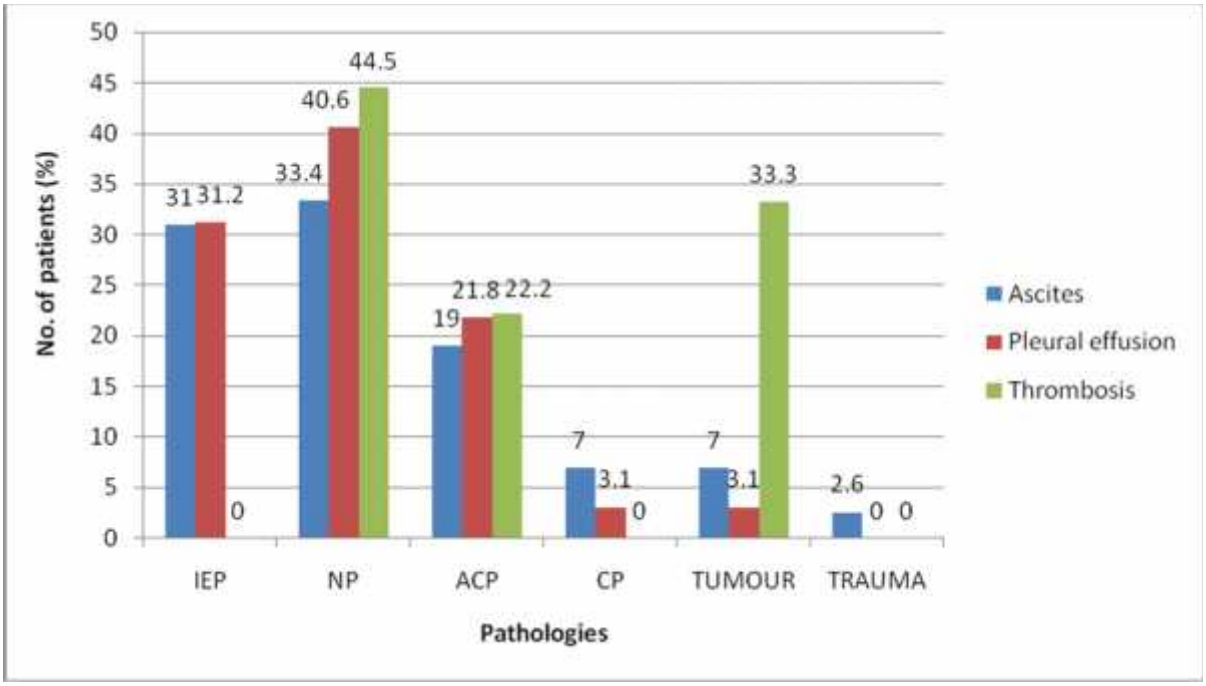
DISTRIBUTION PATTERN OF ABNORMAL MPD SIZE IN ALL PATHOLOGIES

Diagnosis	Main Pancreatic Duct size					
	3-8 mm	>8 mm	Obstruction with dilatation	Disruption	Variant	Total
Tumour						
Benign	0	0	0	0	0	0
Malignant	2 (29%)	1 (14%)	**4 (57%)**	0	0	**7**
Trauma	0	0	0	1 (100%)		1
Pancreatitis						
IEP	1 (50%)	0	0	0	1 (50%)	2
NP	3 (100%)	0	0	0	0	3
ACP	**6 (86%)**	1 (14%)	0	0	0	7
CP	3 (66%)	**6 (33%)**	0	0	0	**9**
Total	**15**	8	4	1	1	29

Of the 78 patients in the study, abnormal MPD was noted in 29 cases. Majority of cases (n=15) accounting to 52% were included under the size group of 3-8mm, followed by 8 cases (27.5%) under >8mm group, obstruction with dilatation of MPD due to tumour in 4 cases, disruption in 1 case and 1 case showed variant ductal anatomy.

FIGURE 14:

DISTRIBUTION PATTERN OF COMPLICATIONS IN ALL PATHOLOGIES



Of the 78 patients in the study, ascites was seen in 42 cases (54%), pleural effusion in 32 cases (41%) and thrombosis in 9 cases (11.5%). Among all, the cases of necrotizing pancreatitis showed maximum cases with ascites (33.4%), pleural effusion (40.6%) and thrombosis (44.5%).

ATTENUATION TABLES (HU VALUES):

TABLE 20:

ATTENUATION VALUES IN DIFFERENT PHASES OF ACUTE PANCREATITIS

Acute Pancreatitis (n=36)	Min	Max	Mean	SD	95% Confidence Interval for Mean		Kruskal Wallis Test
					Lower Bound	Upper Bound	
AP	100	121	112.61	5.55	110.73	114.49	p=0.001*
PPP	126	140	133.39	4.13	131.99	134.79	
PVP	118	132	123.75	3.54	122.55	124.95	

In acute pancreatitis the enhancement attenuation (HU) of the parenchyma increased from AP to PPP, whereas a mild drop was noted in the PVP with significant ‘p value’.

TABLE 21:

ATTENUATION VALUES IN DIFFERENT PHASES OF ACUTE ON CHRONIC PANCREATITIS

Acute on Chronic Pancreatitis (n=16)	Min	Max	Mean	SD	95% Confidence Interval for Mean		ANOVA Test
					Lower Bound	Upper Bound	
AP	98	112	107.75	3.85	105.69	109.80	p=0.001*
PPP	120	134	127.87	4.28	125.59	130.15	
PVP	114	128	121.75	4.05	119.58	123.91	

In acute on chronic pancreatitis the enhancement attenuation (HU) of the parenchyma increased from AP to PPP, whereas a mild drop was noted in the PVP with significant ‘p value’.

TABLE 22:

ATTENUATION VALUES IN DIFFERENT PHASES OF CHRONIC PANCREATITIS

Chronic Pancreatitis (n=10)	Min	Max	Mean	SD	95% Confidence Interval for Mean		ANOVA Test
					Lower Bound	Upper Bound	
AP	98	116	105.60	6.65	100.84	110.36	p=0.001 *
PPP	114	126	119.00	3.43	116.54	121.46	
PVP	108	120	113.00	4.24	109.96	116.04	

In chronic pancreatitis the enhancement attenuation (HU) of the parenchyma increased from AP to PPP, whereas a mild drop was noted in the PVP with significant 'p value'.

TABLE 23:

ATTENUATION VALUES IN DIFFERENT PHASES OF PANCREATIC NEOPLASMS

Neoplasms (N=14)	Min	Max	Mean	SD	95% Confidence Interval for Mean		Kruskal Wallis Test
					Lower Bound	Upper Bound	
APR	73.42	105	73.42	12.64	66.12	80.73	p=0.027 *
PPP	85.14	109	85.14	12.59	77.87	92.41	
PVP	93.42	137	93.42	17.83	83.13	103.72	

In Pancreatic neoplasm the enhancement attenuation (HU) of the parenchyma increased from AP to PPP and persistently increased in PVP with significant 'p value'.

TABLE 24:

ATTENUATION VALUES OF ARTERIAL PHASE (AP) IN VARIOUS PATHOLOGIES

Arterial Phase (AP)	Min	Max	Mean	SD	95% Confidence Interval for Mean		Kruskal Wallis Test
					Lower Bound	Upper Bound	
Acute Pancreatitis	100	121	112.61	5.55	110.73	114.49	p=0.0001*
ACP	98	112	107.75	3.85	105.70	109.80	
CP	98	116	105.60	6.65	100.84	110.36	
TUMOUR	45	105	73.43	12.64	66.13	80.73	

TABLE 25:

MULTIPLE COMPARISONS BY POST HOC TEST IN ARTERIAL PHASE

Multiple Comparisons By Post hoc Test		p	Statistical significance
Acute Pancreatitis	Pancreatic neoplasm	0.001	**Highly Significant**
	Acute on Chronic Pancreatitis	0.096	Not Significant
Pancreatic neoplasm	Acute on Chronic Pancreatitis	0.0001	**Highly Significant**

- The mean attenuation (HU) of acute pancreatitis, acute on chronic pancreatitis and chronic pancreatitis is more than of pancreatic neoplasm in arterial phase.
- Multiple Comparisons of arterial phase using Post hoc Test states that there is high significant difference in the attenuation among acute pancreatitis with pancreatic neoplasm and pancreatic neoplasm with acute on chronic pancreatitis.
- However there is no significant difference in attenuation among acute pancreatitis and acute on chronic pancreatitis.

TABLE 26:

ATTENUATION VALUES OF PANCREATIC PARENCHYMAL PHASE (PPP) IN VARIOUS PATHOLOGIES

Pancreatic Parenchymal phase (PPP)	Min	Max	Mean	SD	95% Confidence Interval for Mean		Kruskal Wallis Test
					Lower Bound	Upper Bound	
Acute Pancreatitis	126	140	133.39	4.13	131.99	134.79	
ACP	120	134	127.88	4.28	125.59	130.16	p=0.0001*
CP	114	126	119.00	3.43	116.54	121.46	
TUMOUR	57	109	85.14	12.59	77.87	92.41	

TABLE 27:

MULTIPLE COMPARISONS BY POST HOC TEST IN PANCREATIC PARENCHYMAL PHASE (PPP)

Multiple Comparisons By Post hoc Test		p	Statistical significance
Acute Pancreatitis	Pancreatic neoplasm	0.033	**Highly Significant**
	Acute on Chronic Pancreatitis	0.001	**Highly Significant**
Pancreatic neoplasm	Acute on Chronic Pancreatitis	0.0001	**Highly Significant**

- The mean attenuation (HU) of acute pancreatitis, acute on chronic pancreatitis and chronic pancreatitis is more than of pancreatic neoplasm in pancreatic parenchymal phase.
- Multiple Comparisons of PPP using Post hoc Test states that there is high significant difference in the attenuation among acute pancreatitis with pancreatic neoplasm, acute pancreatitis and acute on chronic pancreatitis and pancreatic neoplasm with acute on chronic pancreatitis.

TABLE 28:

ATTENUATION VALUES OF PORTAL VENOUS PHASE (PVP) IN VARIOUS PATHOLOGIES

Portal Venous Phase (PVP)	Min	Max	Mean	SD	95% Confidence Interval for Mean		Kruskal Wallis Test
					Lower Bound	Upper Bound	
Acute Pancreatitis	118	132	123.75	3.54	122.55	124.95	p=0.0001*
ACP	114	128	121.75	4.05	119.59	123.91	
CP	108	120	113.00	4.24	109.96	116.04	
TUMOUR	65	137	93.43	17.83	83.13	103.72	

TABLE 29:

MULTIPLE COMPARISONS BY POST HOC TEST IN PORTAL VENOUS PHASE (PVP)

Multiple Comparisons By Post hoc Test		p	Statistical significance
Acute Pancreatitis	Pancreatic neoplasm	0.001	**Highly Significant**
	Acute on Chronic Pancreatitis	0.750	Not Significant
Pancreatic neoplasm	Acute on Chronic Pancreatitis	0.001	**Highly Significant**

- The mean attenuation (HU) of acute pancreatitis, acute on chronic pancreatitis and chronic pancreatitis is more than of pancreatic neoplasm in portal venous phase.
- Multiple Comparisons of PVP using Post hoc Test states that there is high significant difference in the attenuation among acute pancreatitis with pancreatic neoplasm and pancreatic neoplasm with acute on chronic pancreatitis.
- However there is no significant difference in attenuation among acute pancreatitis and acute on chronic pancreatitis.

TABLE 30:

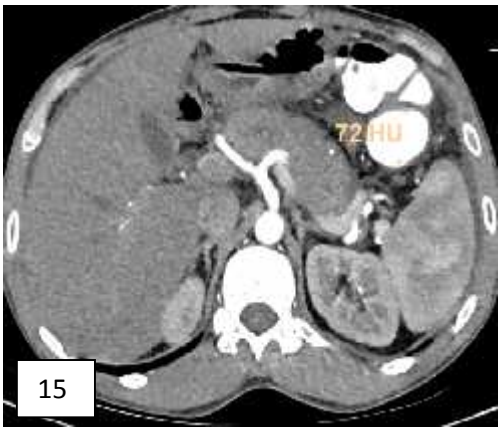
COMPARISON OF P VALUES OF ALL PATHOLOGIES IN DIFFERENT PHASES

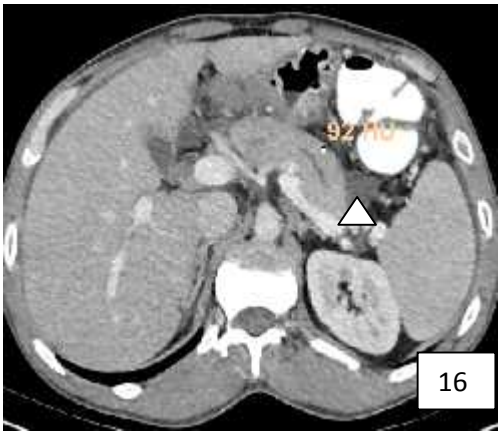
Type			'p' Value	Statistical significance
Acute Pancreatitis	AP	PPP	0.001	*Highly Significant
	AP	PVP	0.001	*Highly Significant
	PPP	PVP	0.0001	*Highly Significant
Acute on Chronic Pancreatitis	AP	PPP	0.001	*Highly Significant
	AP	PVP	0.001	*Highly Significant
	PPP	PVP	0.0001	*Highly Significant
Chronic Pancreatitis	AP	PPP	0.001	*Highly Significant
	AP	PVP	0.010	*Highly Significant
	PPP	PVP	0.040	*Highly Significant
Pancreatic neoplasm	AP	PPP	0.024	*Highly Significant
	AP	PVP	0.003	*Highly Significant
	PPP	PVP	0.333	Not Significant

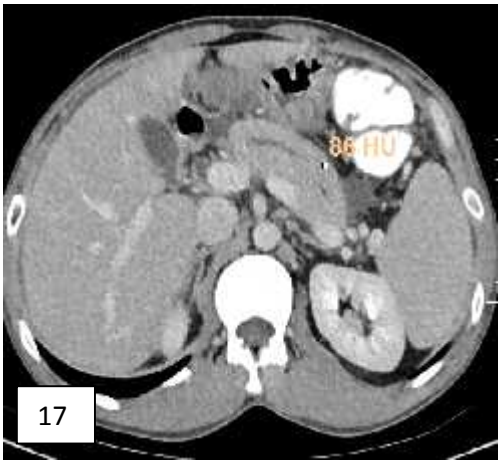
- In acute pancreatitis there is high significant difference in attenuation value among all phases.
- In acute on chronic pancreatitis there is high significant difference in attenuation value among all phases.
- In chronic pancreatitis there is high significant difference in attenuation value among all phases.
- In pancreatic neoplasm there is significant difference in attenuation value among AP with PPP and AP with PVP, however not significant between PPP & PVP.

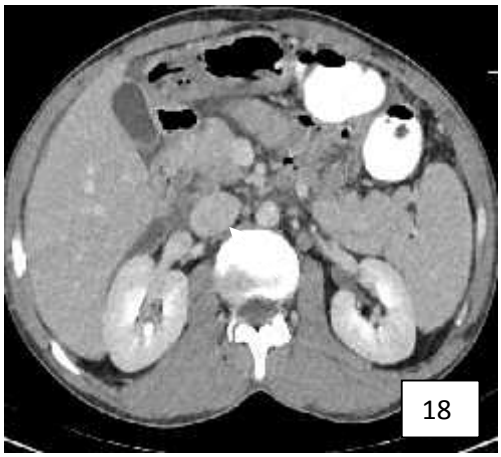
IMAGING GALLERY

INTERSTITIAL OEDEMATOUS PANCREATITIS (Figures 15,16,17&18):





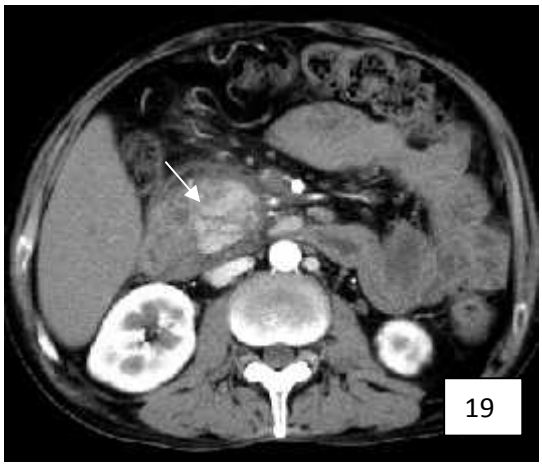




- Above figures showing bulky edematous pancreas with mild peripancreatic fat stranding and acute peripancreatic fluid collection at the tail region (arrow head) & MCTSI – 6 (moderate grade).
- Attenuation value in AP is 72 HU (Fig.**15**) PPP is 92HU (Fig.16) & PVP is 86HU (Fig.17).
- Fig. 18 shows evidence of fluid in pericholecystic & hepatorenal spaces suggestive of ascites (arrow).

*Features are suggestive of ***Interstitial oedematous pancreatitis with ascites.***

NECROTIZING PANCREATITIS (Figures 19,20&21):



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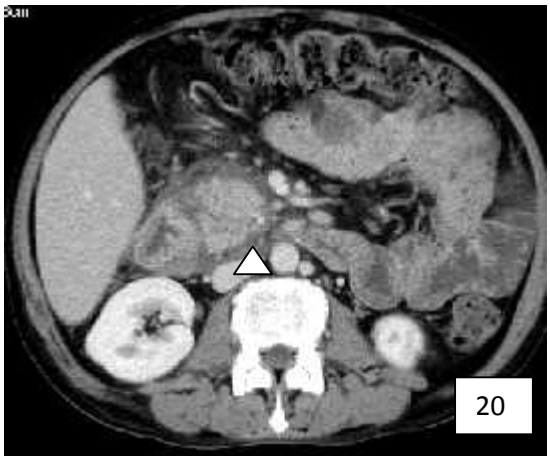

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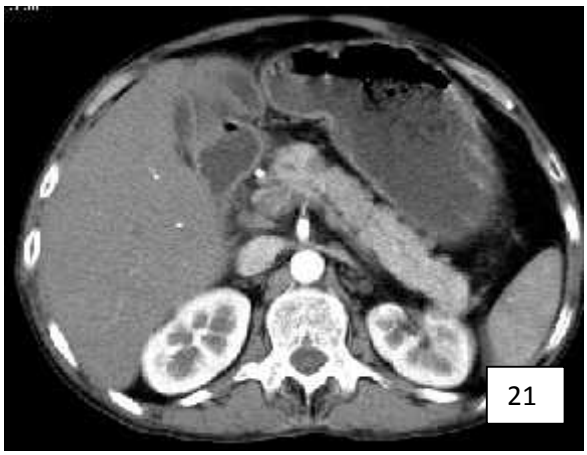


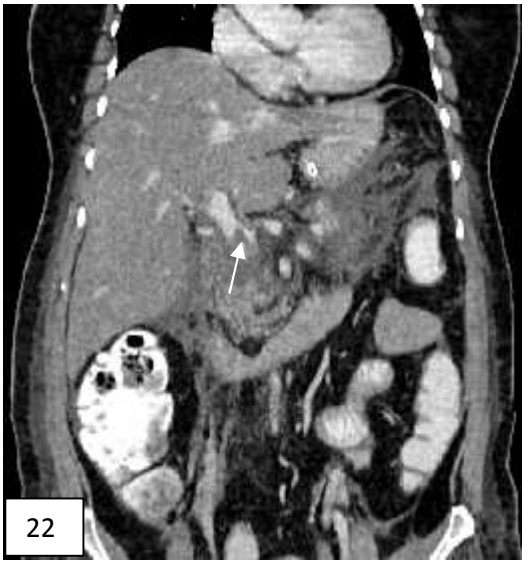
21

- Above figures showing bulky head of pancreas with parenchymal necrosis(short arrow), peripancreatic fat stranding and acute necrotic collection (arrow head) & MCTSI – 8 (severe grade).
- Attenuation value in AP is 90HU (Fig.19), and PVP is 93HU (Fig.20)
- Fig.21: showing normal body and tail of pancreas**.**

*Features are suggestive of ***Necrotizing pancreatitis***.

COMPLICATIONS OF NECROTIZING PANCREATITIS:

CASE 01 (Figures 22 & 23)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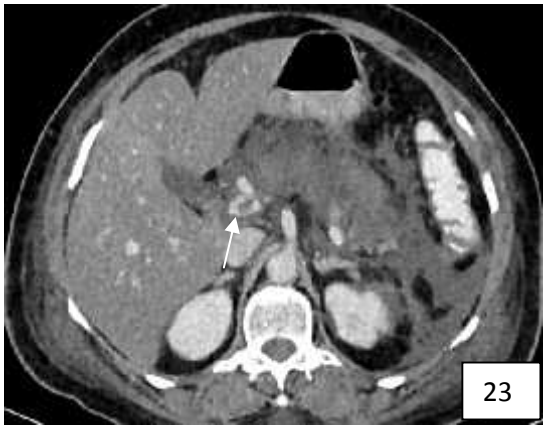
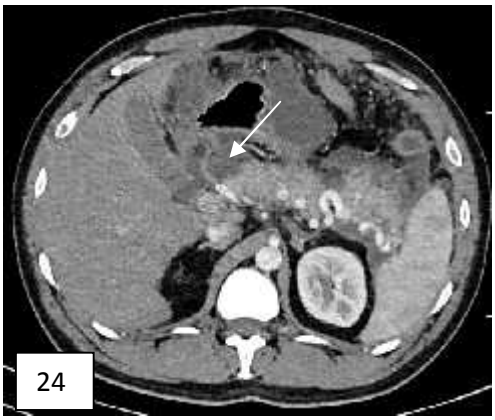

23

Above figures show a bulky pancreas with severe necrosis, peripancreatic fat stranding & acute necrotic collection (ANC) with ***portal vein thrombus (arrow)*** in coronal section (Fig.22) & axial section (Fig.23)
*****Features are suggestive of ***Necrotizing pancreatitis with complications***.

CASE 02 (Figures 24 & 25)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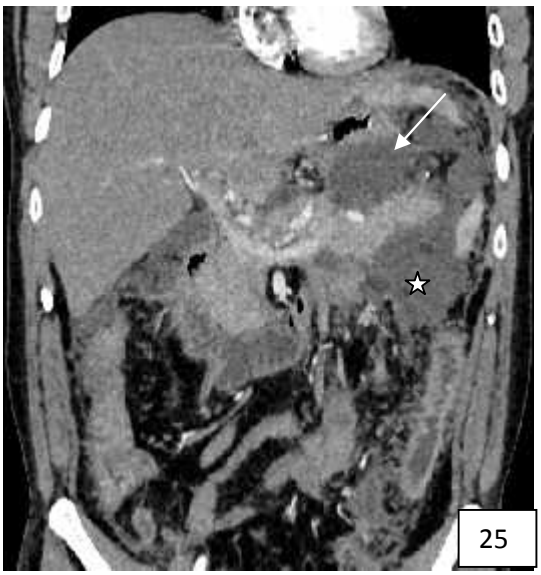
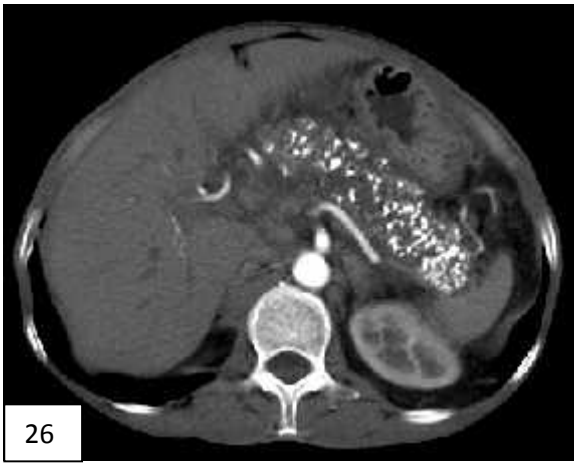

25

- Above figures show a well defined hypodense lesion with enhancing wall (arrow) in the head of pancreas (Fig.24).
- Another similar lesion larger in size is seen in tail (arrow) with peripancreatic fat stranding & fluid collection (star) (Fig.25)

*****Features are suggestive of ***Necrotizing pancreatitis with walled off necrosis***

ACUTE ON CHRONIC PANCREATITIS (Figures 26,27&28):



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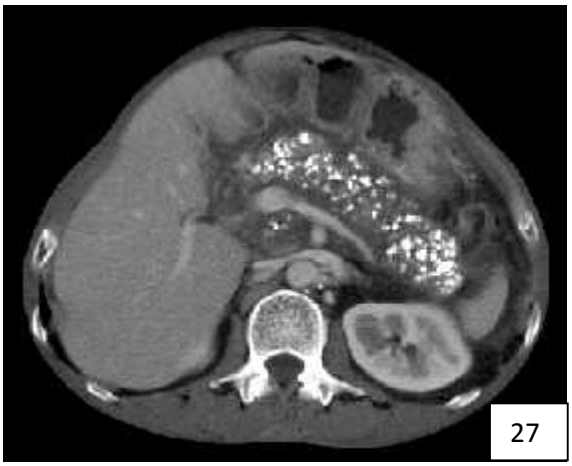

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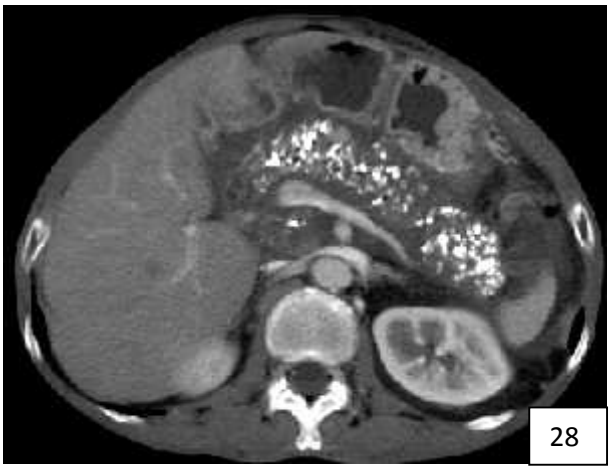
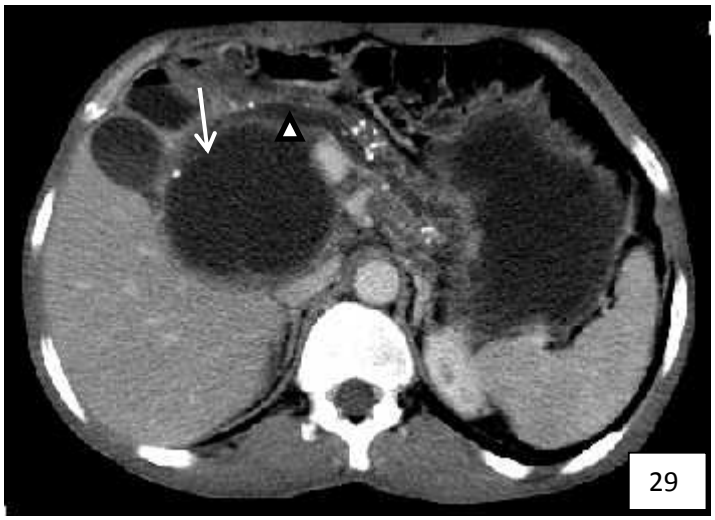


28

- Above figures showing AP (Fig.26), PPP (Fig.27) and PVP (Fig.28) of diffuse enlargement of the pancreas with multiple pancreatic parenchymal calcification, along with peripancreatic fat stranding and collection

  *****Features are suggestive of ***Acute on chronic pancreatitis.***

CHRONIC PANCREATITIS (CASE 01-Figure 29 & CASE 02-Figure 30):



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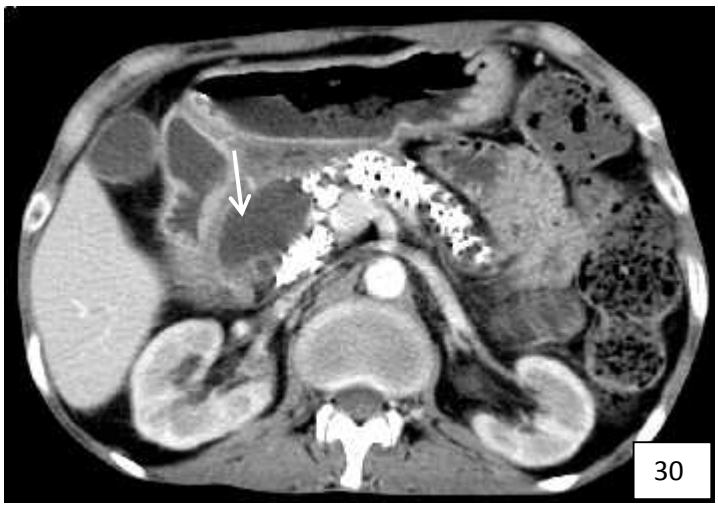
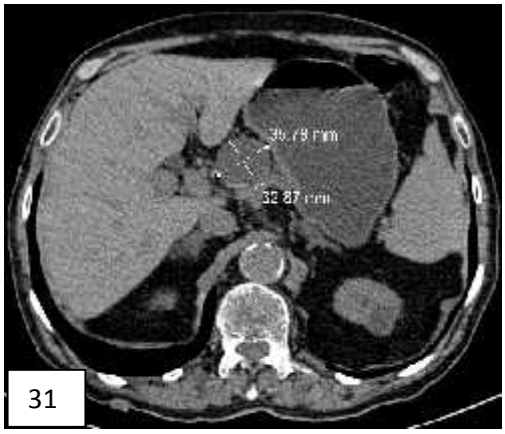


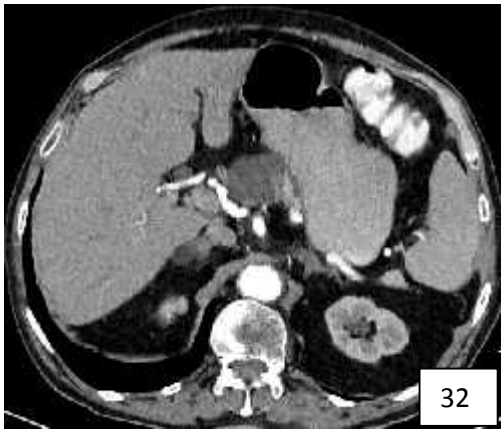
30

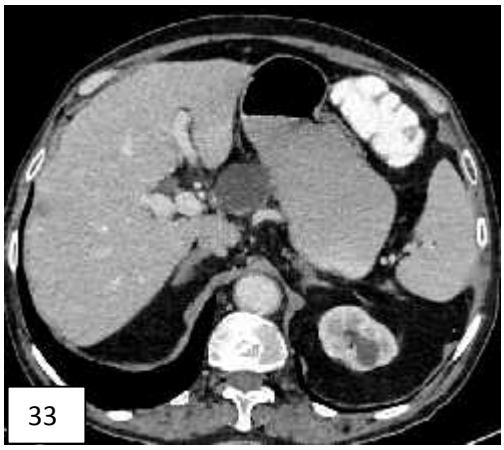
- **Fig.29** shows thinning of pancreatic parenchyma with dilatation of the main pancreatic duct (arrow head). **Fig.30** shows atrophy of pancreatic parenchyma.
- Both figures show multiple calcifications of the pancreatic parenchyma with pseudo cyst of pancreas (arrow).

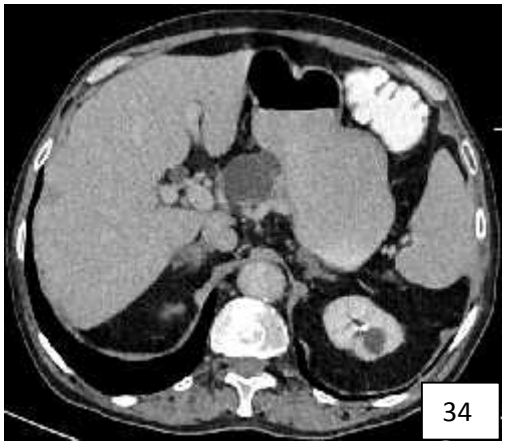
*Features are suggestive of ***Chronic pancreatitis with calcifications, dilated MPD & pseudo cysts.***

MUCINOUS CYSTADENOMA (Fig 31,32,33 &34):





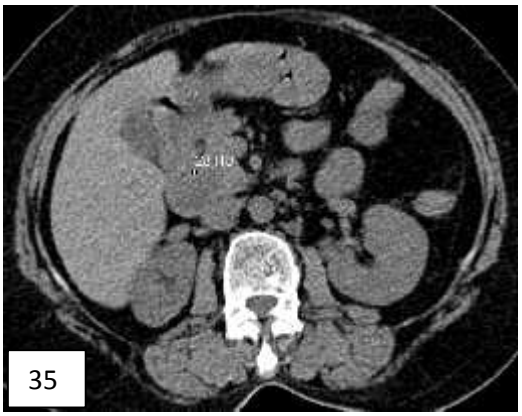




- Above figures show a large well-defined thin walled cystic lesion (3.5cm) in head of pancreas having few calcific foci in the wall (Fig.31) and no evidence of enhancement on AP (Fig.32) , PPP (Fig.33) & PVP (Fig.34)

*Features are suggestive of ***Benign cystic neoplasm of pancreas likely Mucinous cystadenoma***

NEURO ENDOCRINE TUMOUR (Fig 35,36,37&38):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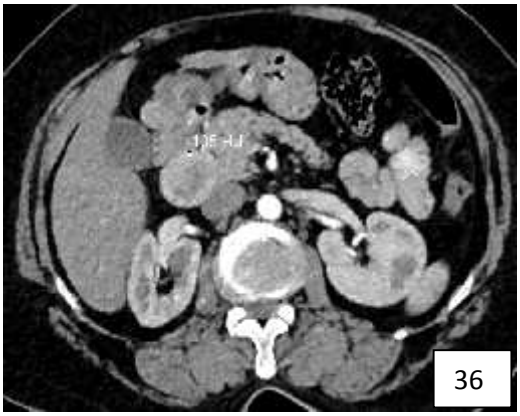
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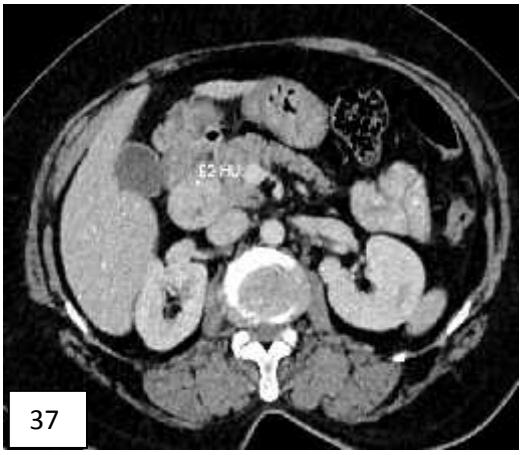
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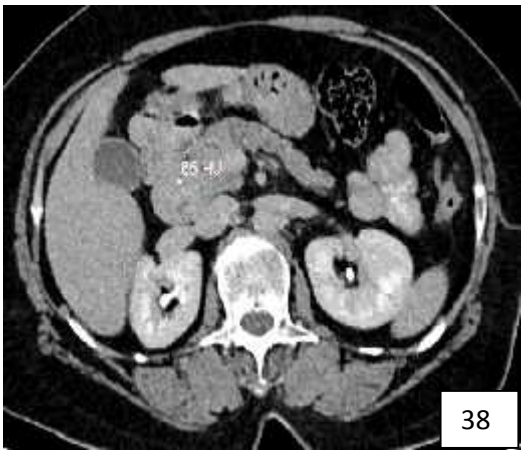
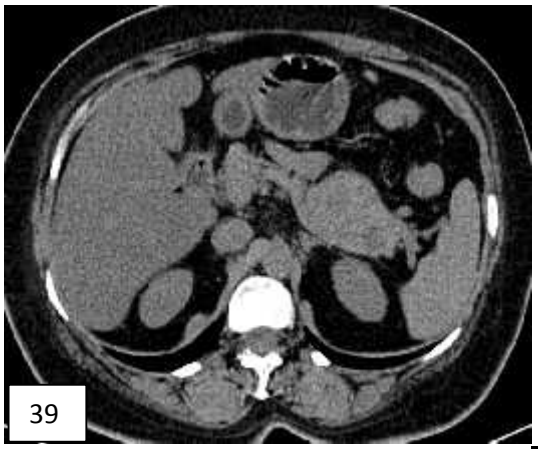

38

- Above figures show a well-defined solid lesion in head of pancreas with HU=28 in NECT (Fig.35) which shows intense enhancement in AP (HU=105) with few necrotic foci within (Fig.36), mild drop of HU=92 in PPP (Fig.37) & washout in PVP (HU=65) (Fig.38)

*Features are suggestive of ***Benign enhancing solid neoplasm of pancreas likely Neuro endocrine tumour.***

BENIGN SOLID PSEUDOPAPILLARY TUMOUR (Fig 39,40,41&42):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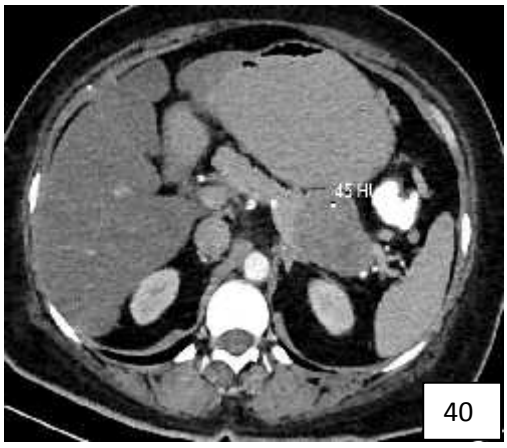
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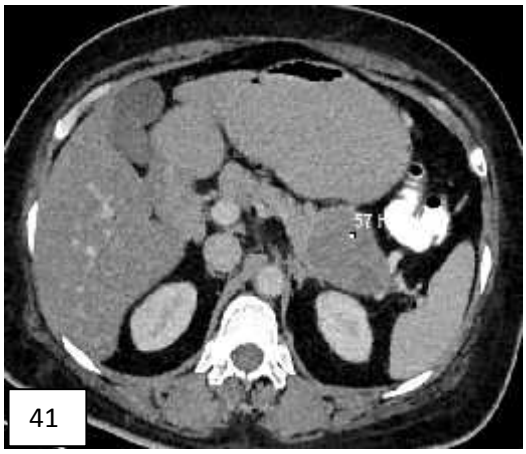
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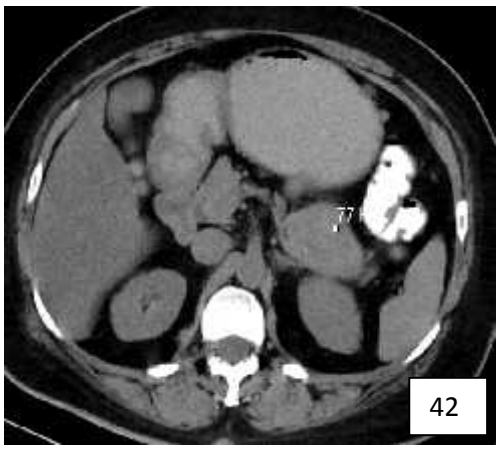
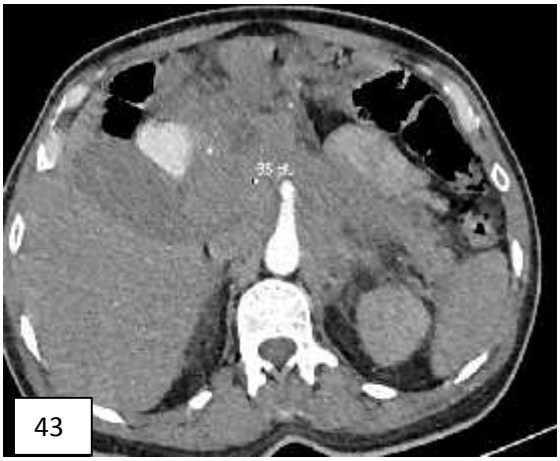

42

- Above figures show a well-defined heterogeneous solid mass in tail of pancreas in NECT (Fig.39) which shows mild enhancement in AP (HU=45) with multiple necrotic foci within (Fig.40), increase in enhancement from PPP (HU=57) (Fig.41) to PVP (HU=77) (Fig.42).

*Features are suggestive of ***Benign heterodense solid neoplasm in tail of pancreas of a middle aged female - likely Solid pseudo papillary tumour.***

LYMPHOMA (Fig 43,44,45,46&47):



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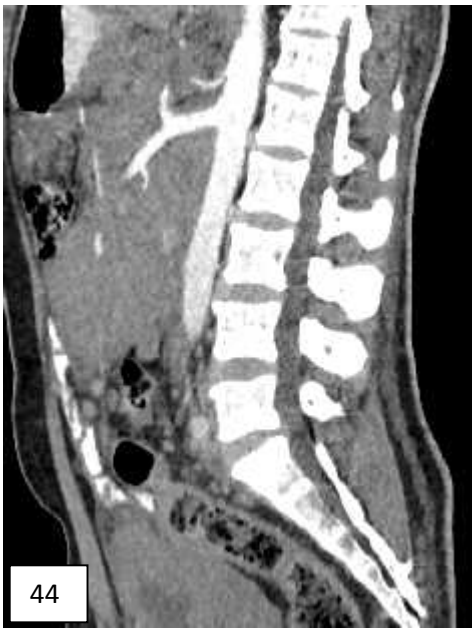

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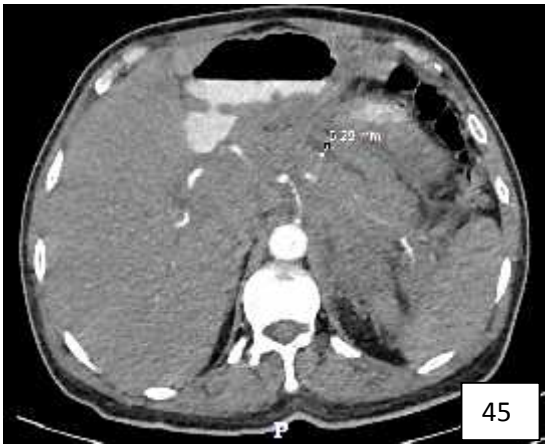
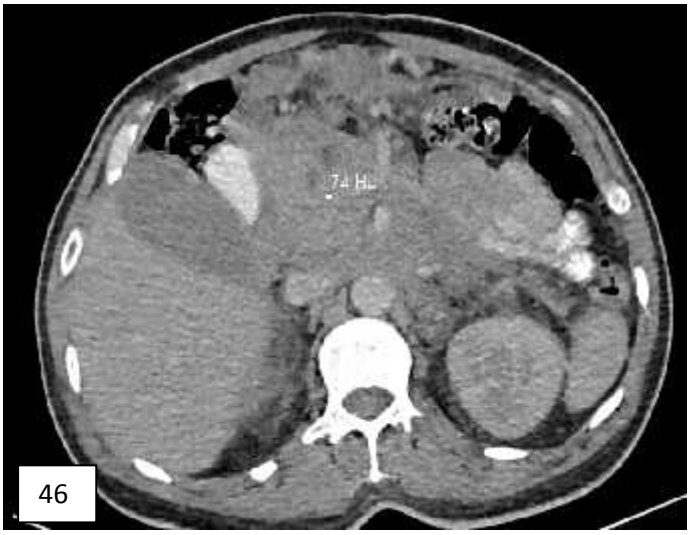
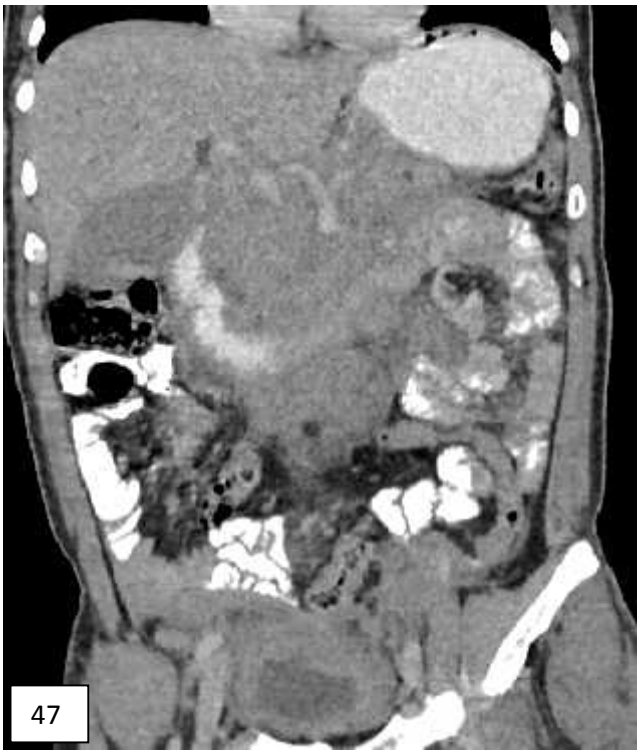


45

Above figures show ill-defined lobulated mass lesion in the peripancreatic region predominantly of head with HU =65 in AP which is seen encasing celiac axis (Fig.43) its branches & SMA can be well depicted in sagittal reconstruction image (Fig.44). MPD dilatation with mesenteric haziness is seen (Fig.45)

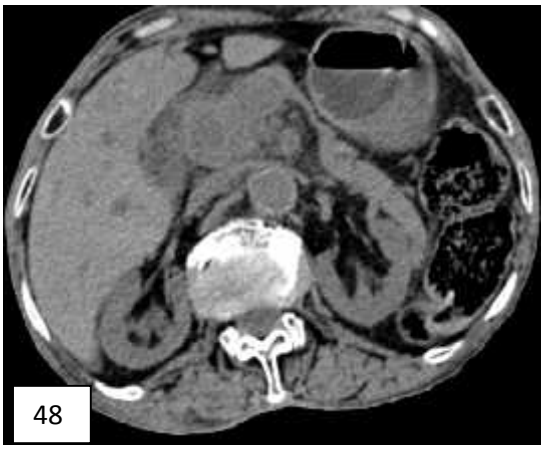




Above figures show increase in HU=74 in PVP with homogenous enhancement (Fig.46). The mass is seen encasing the portal vein, abutting and displacing the C loop of duodenum inferio-laterally as is seen in coronal reconstructed image (Fig.47)

*Features are suggestive of ***Malignant pancreatic lesion likely lymphoma***

METASTASES (FROM GALL BLADDER CARCINOMA) (Fig 48,49,50&51):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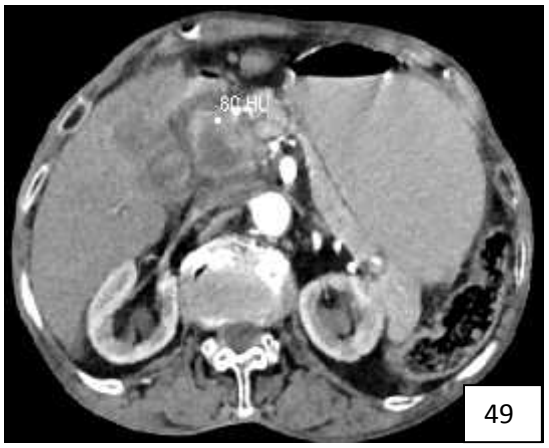
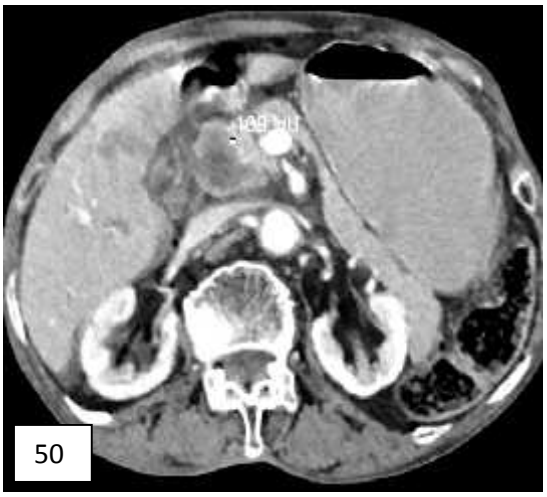
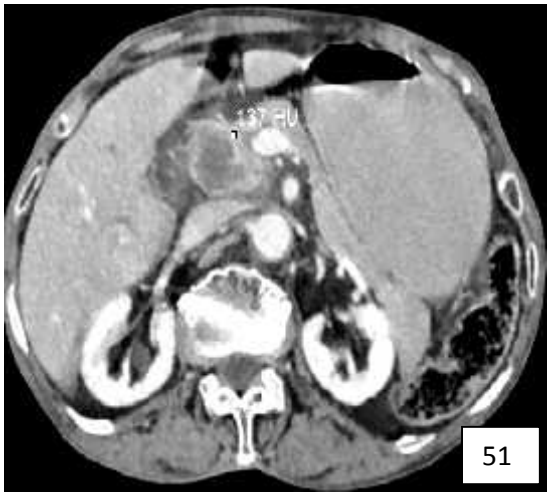


49



50

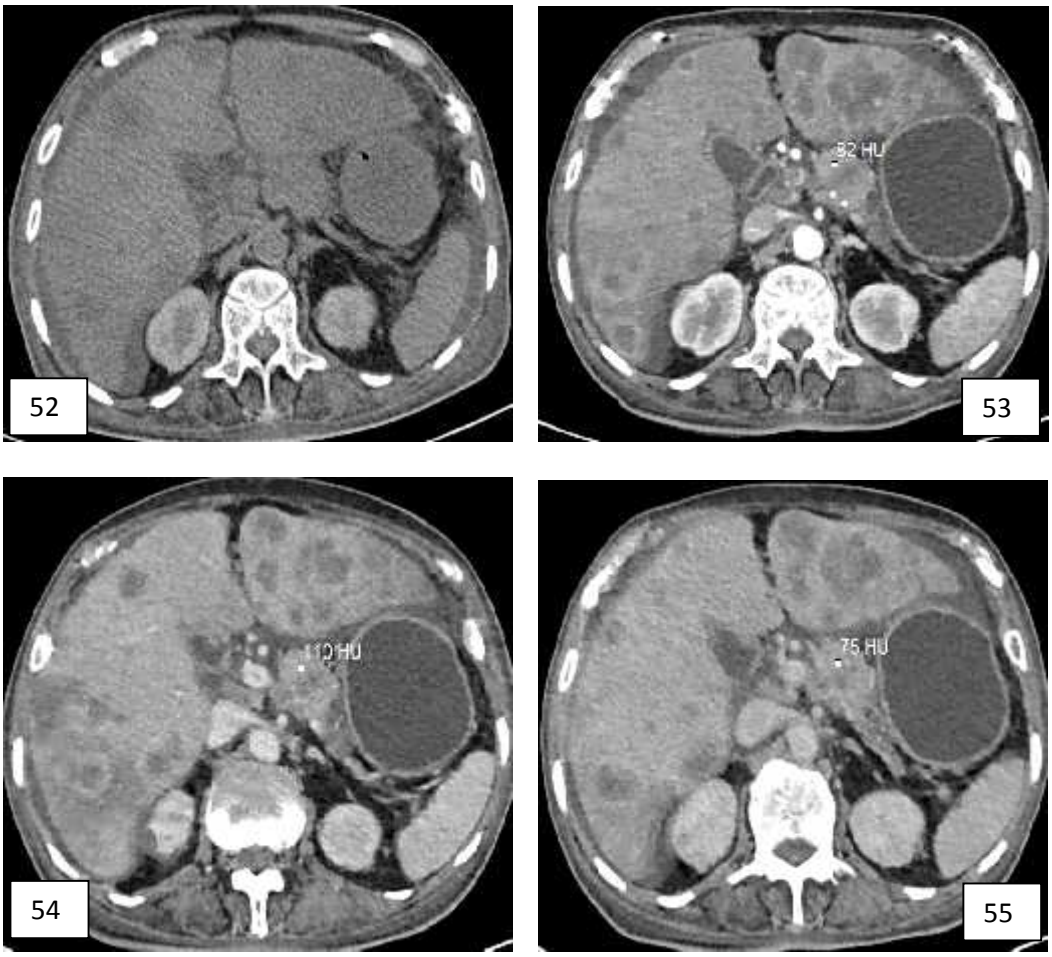


51

- In a case of Carcinoma of Gall bladder the above figures show an ill-defined soft tissue density lesion in the head of pancreas (Fig.48) which shows heterogeneous peripheral enhancement with central non enhancing necrotic areas with increase in attenuation values from AP (HU=80) (Fig.49) to PPP (HU=109) (Fig.50) to PVP(HU=137) (Fig.51).
- Multiple ill-defined hypodense areas are noted in liver likely metastatic deposits.

*Features are suggestive of ***Pancreatic metastatic deposit.***

METASTASES (FROM HEPATOCELLULAR CARCINOMA) (Fig 52,53,54&55):

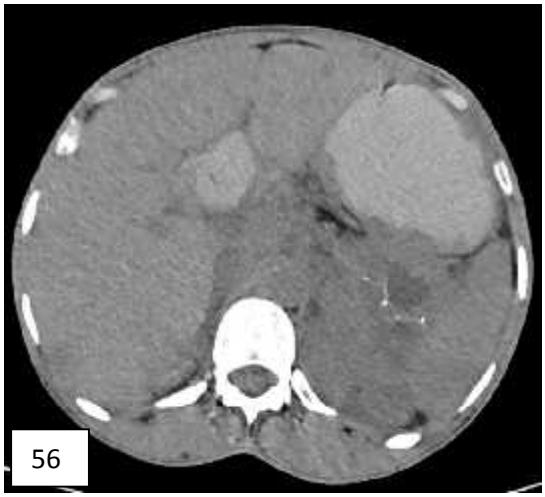


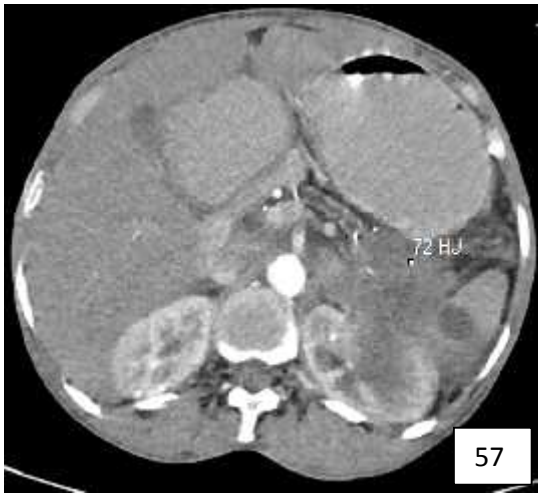
- In a case of Hepato cellular carcinoma the above figures show an ill-defined soft tissue density lesion in the body of pancreas (Fig.52) which shows heterogeneous peripheral enhancement with central non enhancing necrotic areas with increase in attenuation values from AP (HU=82) (Fig.53) to PPP (HU=110) (Fig.54) and drop in PVP (HU=75) (Fig.55).
- Multiple ill-defined peripherally enhancing areas are noted in liver likely metastatic deposits.

*Features are suggestive of ***Pancreatic metastatic deposit.***

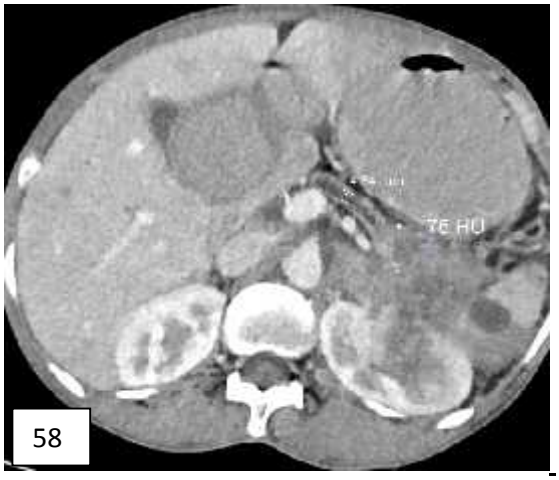
ADENOCARCINOMA

CASE 01(Fig 56,57,58&59):

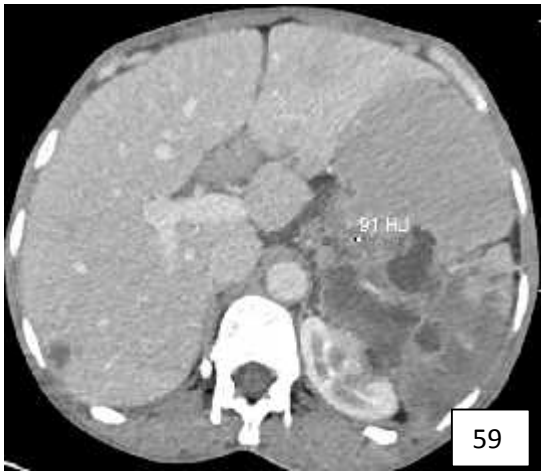

56



57



58



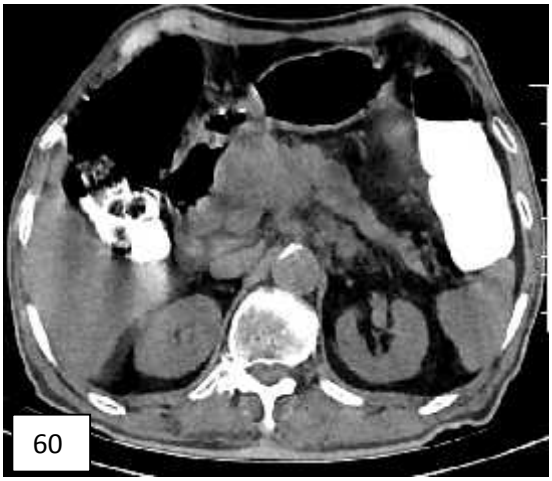
59

- Above figures show an ill-defined heterogeneous solid mass in the tail of pancreas causing parenchymal destruction with obstruction of MPD leading to proximal dilatation and few specs of calcification within (Fig 56). There is increase in attenuation values from AP (HU=72) (Fig.57) to PPP (HU=76) (Fig.58) & PVP (HU=91) (Fig.59) and shows heterogeneous enhancement with non enhancing necrotic areas within.
- The mass lesion is seen infiltration into the lesser curvature of stomach, invading the left kidney causing its structural distortion, encasing & infiltrating the splenic vessels causing thrombosis and leading to multiple infarcts, few non enhancing areas are noted in liver likely metastatic deposits.

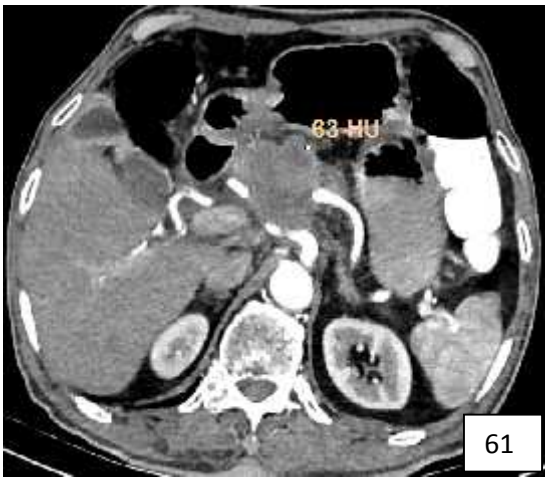
*Features are suggestive of ***Malignant pancreatic tumour likely Adenocarcinoma*** with infiltration of left kidney, stomach, splenic vessels and liver metastasis. Hence, it is unresectable mass.

ADENOCARCINOMA

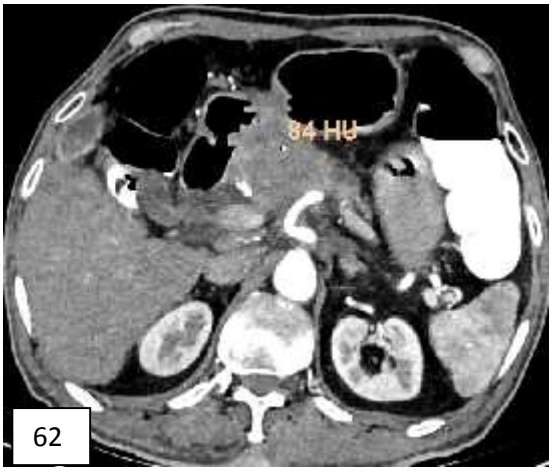
CASE 02 (Fig 60,61,62&63):



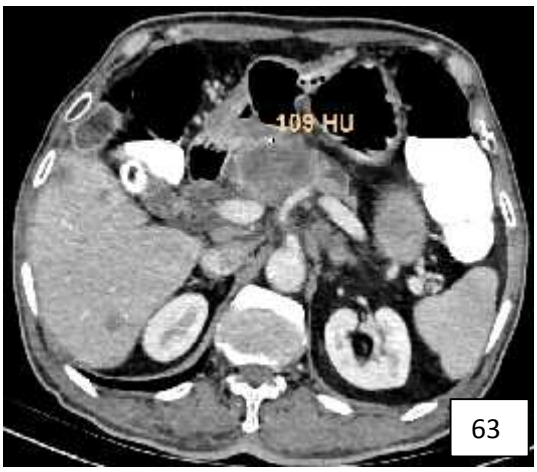
60



61



62



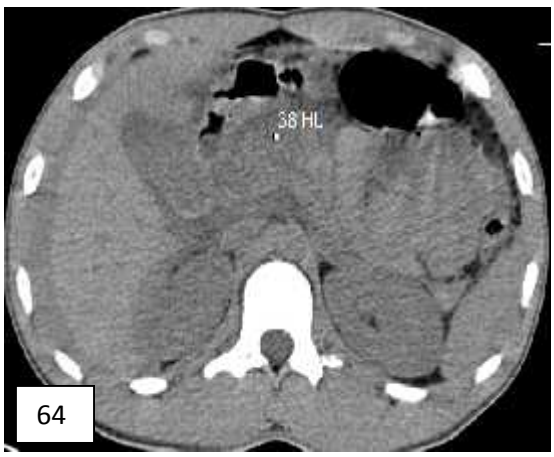
63

- Above figures show an ill-defined heterogeneous solid mass in the head of pancreas causing parenchymal destruction with obstruction of MPD leading to distal dilatation (Fig 60). There is increase in attenuation values from AP (HU=63) (Fig.61) to PPP (HU=84) (Fig.62) & PVP (HU=109) (Fig.63) and shows heterogeneous enhancement with non enhancing necrotic areas within.
- Few non enhancing areas are noted in liver likely metastatic deposits.

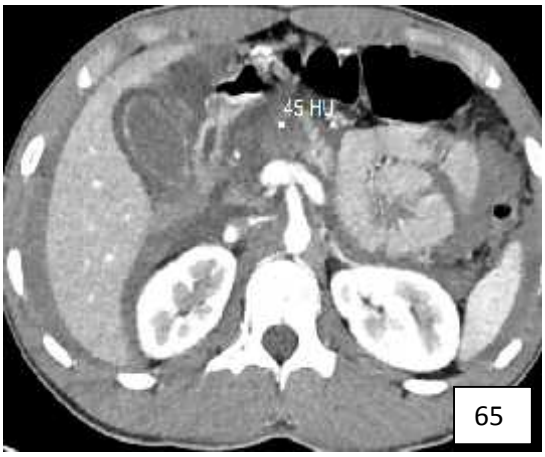
*Features are suggestive of ***Malignant pancreatic tumour likely Adenocarcinoma*** with liver metastasis. Hence, it is unresectable mass.

PANCREATIC TRAUMA

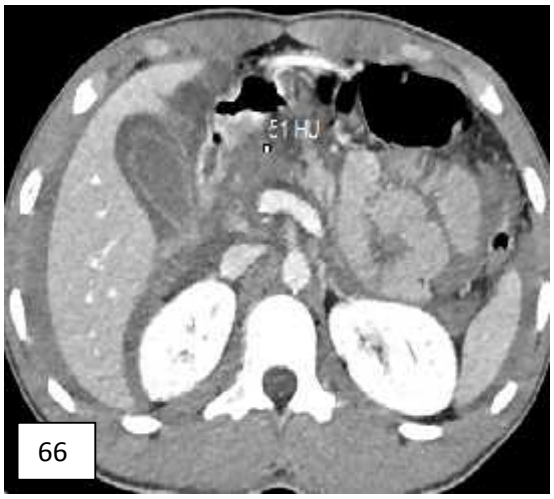
CASE 01 (Fig 64,65,66&67):



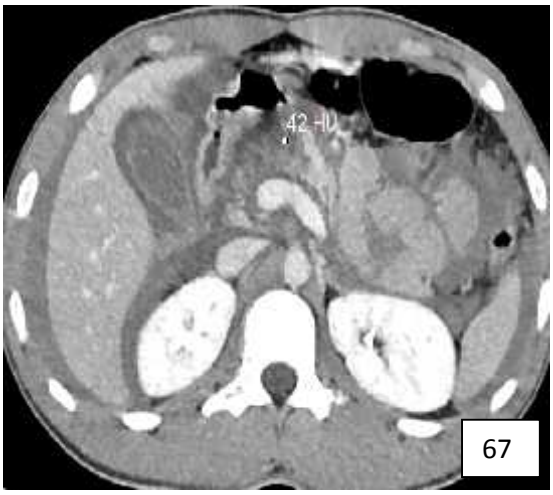
64



65



66



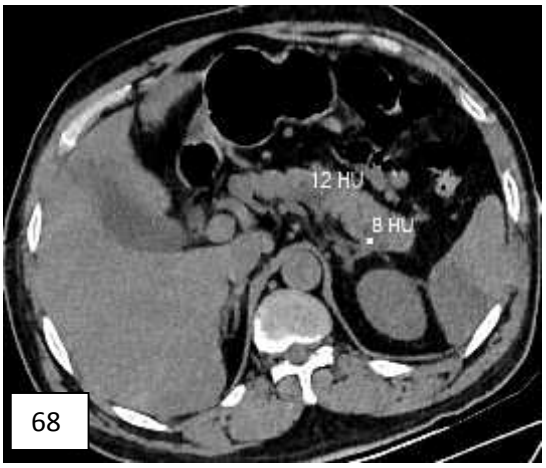
67

- Above figures show ill-defined hypodense area in the head & neck of pancreas with non visualization of MPD in the same region (Fig.64).
- Minimal enhancement with increase in attenuation values from AP (HU=45) (Fig.65) to PPP (HU=51) (Fig.66) and drop in PVP (HU=42) (Fig.67) is noted. Gross Ascites can be noted in the images.

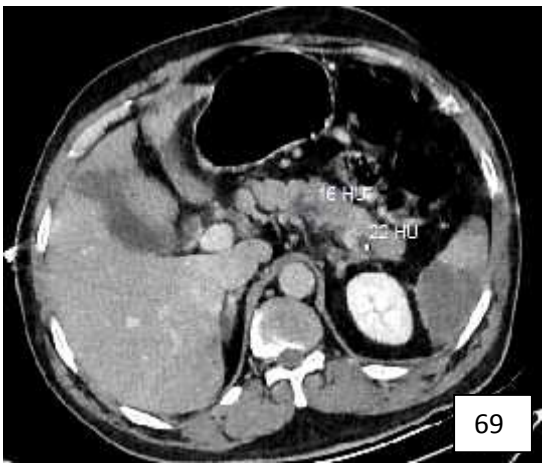
*Features are suggestive of ***Pancreatic contusion with MPD disruption (Grade III AAST injury)***

PANCREATIC TRAUMA

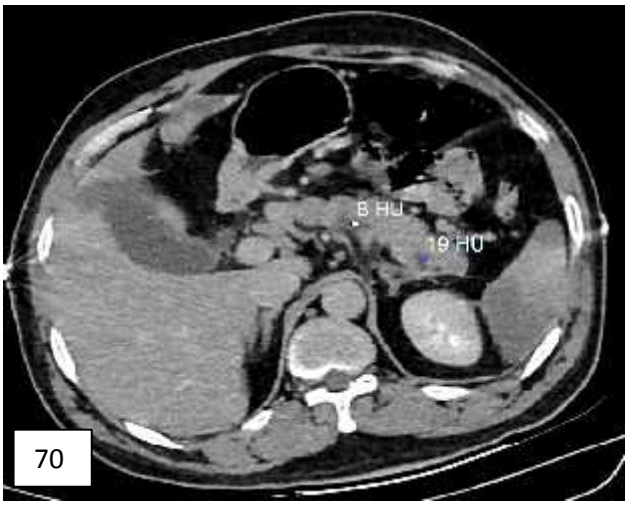
CASE 02 (Fig 68,69&70):



68



69



70

- Above figures show two ill-defined hypodense areas in the body & tail of pancreas with normal MPD (Fig.68).
- Minimal enhancement with increase in attenuation values from NECT (HU=12 & 8) (Fig.68) to PPP (HU=16 & 22) (Fig.69) and drop in PVP (HU=8 & 19) (Fig.70) is noted. Multiple non enhancing areas are noted in liver & spleen suggestive contusions.

*Features are suggestive of ***Multiple pancreatic contusions (Grade II AAST injury) with associated liver (Grade II) & splenic injuries (Grade III AAST injury)***

HISTO-PATHOLOGICAL IMAGES:

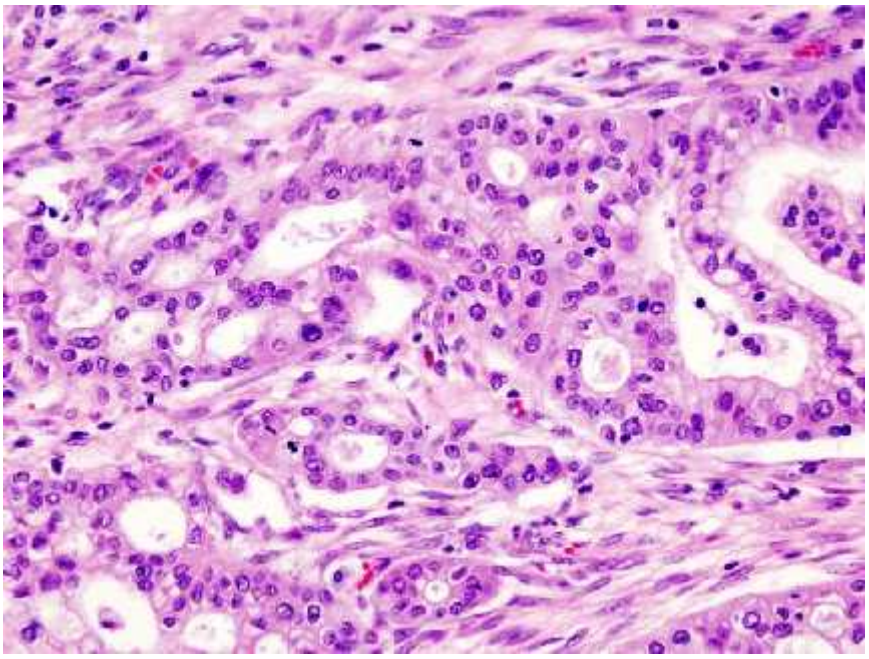


Fig.71 ADENOCARCINOMA SLIDE

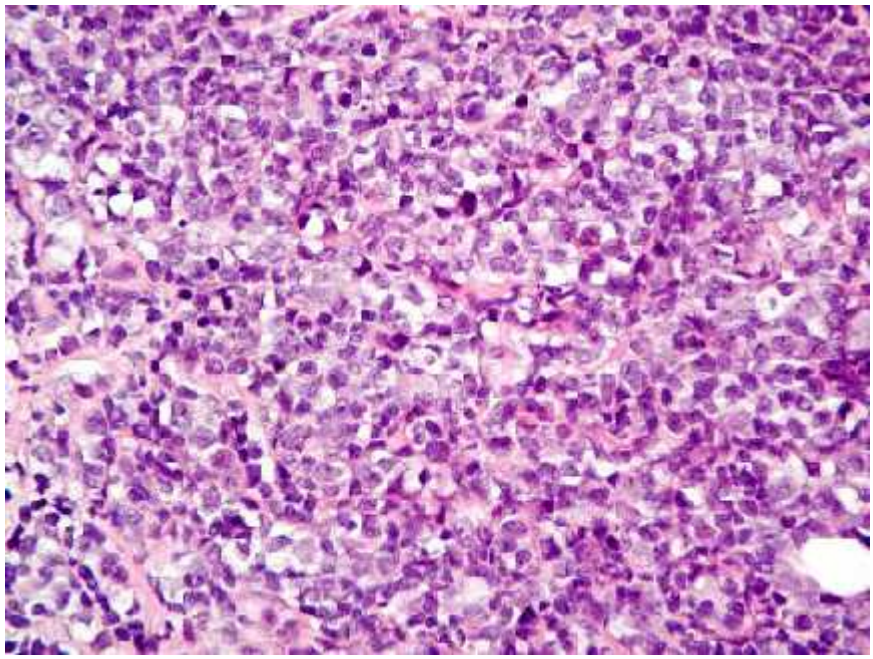


Fig.72 LYMPHOMA SLIDE

DISCUSSION

A total of 78 patients referred for pancreatic pathology were studied using MDCT-triple phase which included inflammatory, neoplasm and trauma cases. The study was done using Multidetector CT, which allowed acquisition of images within a single breath hold, without any motion artefacts caused by respiratory movement. This was comparable to **Fletcher Joel G et all (2003)[90]** in which respiratory motion artefact was absent due to faster scanning in Multidetector row CT.

This technology permits thinner slices to be acquired during multiphasic scanning, with improved spatial resolution[131]. Advent of multi phasic imaging helps in optimal pancreatic enhancement and ability to analyse the attenuation value in each phase and the disease entity. Triple phase analysis was done in our study.

Contrast scan was obtained in three phases after obtaining unenhanced MDCT which includes arterial phase (AP), pancreatic parenchymal phase (PPP), and portal venous phase (PVP) as described under methodology section. **Nancy J. et al**[131] used similar multi phasic imaging methodology in 77 cases with 20sec delay in AP, 35sec delay for PPP and 60sec delay for PVP.

DEMOGRAPHICS:

Majority of study population (68%) are in the age group of 20 – 59 years. Mean age of patients in this study was ~45years (15-92years). These findings are comparable to study by **Shalab Jain et al[61] &** slightly higher in study by **Avanesov M et al[64]**

STUDY SERIES	AGE IN YEARS	MEAN AGE
Present study	15-92	45
Shalab Jain et al[61]	18-80	43.6
Avanesov M et al[64]	18-64	55

Maximum cases of Acute pancreatitis & Acute on chronic pancreatitis were in age group of 20-39 years, majority of chronic pancreatitis & Tumours were seen in age group of >60 years.

About 69.3% of the study population were males and remaining 30.7 % were female with ~2.2:1 (M: F) ratio. These findings correlated with studies by **Shalab Jain et al[61] & Avanesov M et al[64]** whereas the study by **Freeney et al[81]** showed much higher M: F ratio.

STUDY SERIES	MALE	FEMALE	M:F
Present Study	54	24	2.2:1
Shalab Jain et al[61]	99	51	2:1
Avanesov M et al[64]	73	29	2.5:1
Freeney et al[81]	26	8	3.2:1

Male predominance was noted in inflammatory and traumatic aetiology whereas female predominance was noted in neoplastic lesions.

Of the total cases (n=78), maximum cases are of acute pancreatitis (46%), followed by acute on chronic pancreatitis (20.5%), pancreatic neoplasm (18%), chronic pancreatitis (13%) and least were trauma cases (2.5%).

ACUTE PANCREATITIS:

In this study the “Revised Atlanta classification of acute pancreatitis” is used with standardised terminology of pancreatitis and its complications.

In our study 46% of the cases (n=36) are of acute pancreatitis which includes about 27 males (50%) and 9 females (38%).

Of the 36 cases of acute pancreatitis serum amylase was elevated in 32 cases (14- IEP & 18-NP) accounting for 88.9% and serum lipase was elevated in with 36 cases (17- IEP & 19-NP) with Chi square test p<0.001 (highly significant) suggesting it is specific for acute pancreatitis and serum lipase is more specific than amylase. This were comparable to studies by **HS Batra et al**[62] & **Gomez D et al**[60]

Study series	No.of patients	Increased Serum lipase level	Increased Serum Amylase level	Normal serum amylase levels.
Present study	36	36	32	04
HS Batra et al[62]	50	50	42	08
Gomez D et al[60]	117	113	96	21

➢ INTERSTITIAL OEDEMATOUS PANCREATITIS (IEP):

In our study about 17 cases are of interstitial oedematous pancreatitis (22%). About 11% are below 20 years, 41% are in 20-39 years, 24% are in 40-59 years and 24% are above 60 years. Majority are males (n=11) and rest females (n=6).

Bulky enlarged pancreas (p<0.001) was seen in all the cases (100%), Fat stranding (p<0.001) in 100%, acute peri pancreatic fluid collection (p<0.001) in 88%, with 1 case each having pseudo cyst, dilated main pancreatic duct and variant ductal anatomy. No cases showed any necrosis. These findings were comparable to study by **Bharat et al**[63] **& Avanesov M et al**[64]

Variable	Present study (n=17)	**Bharat et al**[63] (n=20)	**Avanesov M et al**[64] (n=52)
M: F	1.8 :1 (11/6)	-	2.2 : 1 (36/16)
Bulky pancreas	100 % (17)	85 % (17)	69 % (36)
Fat stranding	100 % (17)	95 % (19)	100 % (52)
Acute peri pancreatic fluid collection	82% (14)	90 % (18)	56 % (29)
Necrosis	0%	0%	0%
Ascites	76.4% (13)	60 % (12)	25 % (13)
Pleural effusion	58.8% (10)	60 % (12)	35 % (18)
Thrombosis	0	5% (1)	10 % (5)

➢ NECROTIZING PANCREATITIS (NP):

In our study about 19 cases are of necrotizing pancreatitis (24.4%). No cases are seen below 20 years, 63% are in 20-39 years, 26% are in 40-59 years and 11% are above 60 years. Majority are males (n=16) and rest females (n=3).

Bulky enlarged pancreas ($p<0.001$) was seen in majority of the cases (89%), Fat stranding ($p<0.001$) in 100%, Parenchymal necrosis ($p<0.001$) in 100%, acute necrotic collection ($p<0.001$) in 84% and few cases showed pseudo cyst (2) and dilated main pancreatic duct (3). No cases showed any calcifications. These findings were more comparable to study by **Bharat et al**[63] **&** to study of **Avanesov M et al**[64] with few differences in the results as mentioned below:

Variable	Present study (n=17)	**Bharat et al**[63] (n=15)	**Avanesov M et al**[64] (n=50)
M: F	5.3 :1 (16/3)	-	2.8 : 1 (37/13)
Bulky pancreas	89 % (17)	100 % (15)	0 %
Fat stranding	100 % (19)	100 % (15)	100 % (50)
Acute necrotic collection	84% (16)	93.3 % (14)	0 %
Necrosis	100% (19)	93.3 % (14)	84% (42)
Ascites	73.6% (14)	86.6 % (13)	32% (16)
Pleural effusion	68.4% (13)	73.3 % (11)	52 % (26)
Thrombosis	21% (4)	20% (3)	14 % (7)

Out of 78 patients only 4 cases showed walled off necrosis and all are of necrotising pancreatitis group (p=0.0215) highly significant stating that it is specific for NP[32].

Modified CT Severity Index (MCTSI) score:

MDCTSI was assessed for all the cases (n=36) of which 1 case came under mild grade (IEP), 18 cases under moderate grade (IEP -16 & NP -2) and 17 cases under severe grade (NP) with statistical significance (p=0.001) between IEP showing maximum cases under moderate grade and NP showing maximum under severe grade.

As per this study majority of cases are under moderate grade. These findings are comparable to study of **Bharat et al**[63] **&** as opposed to study by **Shalabh J et al**[61] with maximum cases in severe grade.

MDCTSI Grading	PRESENT STUDY				
	IEP	NP	Total	**Bharat et al[63]**	**Shalabh J et al[61]**
Mild (0-2)	1	0	1	4	27
Moderate (4-6)	16	2	18	18	57
Severe (8-10)	0	17	17	12	66
Total			**36**	**34**	**150**

ACUTE ON CHRONIC PANCREATITIS:

In our study about 16 cases are of acute on chronic pancreatitis (20.5%). About 6% are seen below 20 years, 63% are in 20-39 years, 25% are in 40-59 years and 6% are above 60 years. Majority are males (n=11) and rest females (n=5).

Elevated serum amylase level was noted in 11 cases accounting to 69% whereas elevated serum lipase was noted in 15 cases accounting to 93.7%. These findings are comparable to study by **Bhatt A et al[70]**.

STUDY SERIES	No. of patients	Increased Serum lipase	Increased Serum amylase
Present Study	16	15	11
Bhatt A et al[70]	06	04	05

Statistically significant ‘p values’ (<0.001) are noted in bulky size (69%), fat stranding (88%), Parenchymal necrosis (63%), peri pancreatic collection (50%) and pseudocyst (69%) suggesting these characters are specific for acute nature of pathology. Intra parenchymal & intra ductal calcifications (43.7%) with significant p

value (p=0.002) for dilated MPD (44%) shows it is specific for the underlying chronic pathology.

CHRONIC PANCREATITIS:

In our study about 10 cases are of chronic pancreatitis (12.8%). About 20% are seen below 20 years, 0% are in 20-39 years, 40% are in 40-59 years and 40% are above 60 years. Majority are males (n=8) and rest females (n=2).

Normal serum amylase (<125 IU/L) & lipase level (<150 IU/L) was noted in majority (n=10) (p<0.001) of the cases accounting to 90%. This is comparable to study of **Oh HC et al[71]** who stated that low to normal serum lipase and amylase levels are seen in 88.8% and stating it is specific finding for Chronic pancreatitis.

STUDY SERIES	Serum Amylase/lipase levels (Normal/Low)
Present study	90% cases
Oh HC et al[71]	88.8% cases

Majority of the cases (80%) showed atrophic pancreas (p<0.001) while 20% showed normal pancreas, 70% cases showed pseudo cyst (p<0.0001) and 60% cases showed calcifications (intra parenchymal & intra ductal). Dilated main pancreatic duct (p=0.002) was seen in 90% cases with a mean value of 8.3mm stating that it is specific for chronic pancreatitis. These findings were comparable to studies of **Bharat et al[63] & Patrick Luetmer et al**[69].

VARIABLE	Present Study	**Bharat et al[63]**	**Patrick Luetmer et al**[69]
Atrophy of pancreas	80%	50%	54%
Calcifications	60%	60%	56%
MPD dilatation	90%	90%	68%
Pseudocysts	70%	50%	30%

Thus features like atrophic pancreas, dilated MPD, calcifications, pseudo cysts with normal serum amylase characterise chronic pancreatitis.

ATTENUATION PATTERN IN PANCREATITIS (HU Values) -

Hounsfield units (HU) were calculated in all the three phases [Arterial Phase (AP), Pancreatic Parenchymal Phase (PPP), and Portal Venous Phase (PVP)] of all cases of pancreatitis. The findings are as follows :

- In Acute pancreatitis the mean in AP, PPP and PVP were 112.61, 133.39 and 123.75 HU respectively.
- In acute on chronic pancreatitis the mean in AP, PPP and PVP were 107.75, 127.87 and 121.75 HU respectively.
- In chronic pancreatitis the mean in AP, PPP and PVP were 105.60, 119.00 and 113.00 HU respectively.

All conditions showed increase in HU values from AP to PPP, whereas a mild drop was noted in the PVP with higher values in acute then acute on chronic and least in chronic pancreatitis.

Pair wise comparisons was done using Post hoc Wilcoxon Signed Rank Test and results were as follows :

- In Acute pancreatitis p=0.001, p=0.001 and p=0.0001 was noted among AP v/s PPP, AP v/s PVP and PPP v/s PVP respectively.
- In acute on chronic pancreatitis, p=0.001, p=0.001 and p=0.0001 was noted among AP v/s PPP, AP v/s PVP and PPP v/s PVP respectively.
- In chronic pancreatitis, p=0.001, p=0.010 and p=0.040 was noted among AP v/s PPP, AP v/s PVP and PPP v/s PVP respectively.

All comparisons are highly significant, stating that there is significant difference in attenuation value among different phases in each type of pancreatitis.

Among all phases in all pancreatitis cases maximum value is seen in pancreatic parenchymal phase (PPP) thus stating that Parenchymal phase is optimal phase for evaluation of necrosis of pancreas. This finding was comparable to study by **Bharat et al[63]** who concluded the same.

PANCREATIC NEOPLASM:

In our study 17.9% of the cases (n=14) are of pancreatic neoplasm; of these 7% are in 20-39years, 36% in 40-59years and 57% above 60years of age. Majority are females (n=8) accounting to 57% and rest are males (n=6) accounting to 43%.

Therefore in our study pancreatic neoplasms are more common in females than males. These findings are comparable to study by **Ichikawa T et al[95]** As opposed to studies by **Dawoud MA et al[99]** & **Hossain MS et al[114].**

STUDY SERIES	MALE	FEMALE	TOTAL
Present study	6	8	14
Ichikawa T et al[95]	13	18	31
Dawoud MA et al[99]	16	4	20
Hossain MS et al[114]	37	10	47

Out of 14 neoplasms in our study, 4 cases were found to be benign (28.5%) and 10 cases malignant (71.5%) based on imaging findings.

STUDY SERIES	BENIGN	MALIGNANT	TOTAL
Present study	4	10	14
Dawoud MA et al[99]	6	14	20
Hossain MS et al[114]	14	33	47

SIZE:

Of the 4 benign cases majority (n=3) were in range of 2-5cm in size accounting to 75% and 1 case was in 5-10cm range (9cm). Mean size was about 5.4cm.

Of the 10 malignant cases 40% were of <2cm, 20% in 2-5cm, 30% in 5-10cm and 10% are >10cm in size. All the malignant lesions <2cm size are metastatic lesions. A size >10cm suggests high possibility of malignancy as per the study. These findings are comparable to study by **Dawoud MA et al[99].**

MARGINS:

All the benign cases (n=4) showed well defined margins in the study whereas majority (n=9) of malignant cases showed ill-defined margins accounting to 90% with 1 case showing lobulated margins (10%) with a Chi square test p=0.0009(highly significant) stating that ill-defined/ lobulated margins are specific for malignant nature of neoplasms.

STUDY SERIES	Well-defined	Ill-defined	TOTAL
Present study	4	10	14
Dawoud MA et al[99]	6	14	20

LOCATION:

Of the 14 cases of neoplasms in the study, majority of cases (n=7) accounting to 50% are seen involving head of pancreas followed by tail (28%). Among benign cases 50% involved head and 50% the tail. In malignant cases 40% were seen involving head, 10% head & uncinated process, 10% body, 20% tail, 20% body & tail with a Chi square test p=0.6327(not significant) stating that no specific pattern of

distribution is seen among benign & malignant neoplasms. This correlated with the studies by **Dawoud MA et al[99]** & **Hossain MS et al[114].**

STUDY SERIES	No.of cases	Maximum location	No. & %
Present study	14	Head	7/50%
Dawoud MA et al[99]	20	Head	9/45%
Hossain MS et al[114]	47	Head	30/64%

COMPOSITION:

Of the 14 neoplasm cases majority of the cases (n=12) were of solid composition accounting to 85.7%, of which 2 cases (16.7%) are benign and 10 cases (83.3%) are malignant with Chi square test p=0.0028 (highly significant) stating that solid composition of masses indicates more of malignant nature.

About 2 cases (14.3%) showed cystic composition and are seen only in benign cases with Chi square test p=0.045 (highly significant) stating that cystic composition of masses indicates more of benign nature.

STUDY SERIES	Solid	Cystic	Solid & Cystic
Present study (n=14)	12	02	-
Dawoud MA et al[99](n=20)	10	08	02

MAIN PANCREATIC DUCT (MPD):

Of the 14 cases of neoplasm in the study, abnormal MPD was seen in 7 cases (50%). Of these majority of cases (n=4) accounting to 57% showed obstruction with dilatation of MPD, 29% in 3-8mm and 14% are seen having >8mm sized MPD. The mean diameter is 6.2mm

MPD was normal in all benign cases (100%). All the 7 abnormal MPD cases are of malignant group with majority of them showing obstruction of duct due to tumour growth with dilatation of remaining duct stating that MPD obstruction with dilatation & a size criteria of >8mm are more indicative of malignancy. Similar findings are noted in study of **K Takeshita et al[98]**.

STUDY SERIES	Abnormal MPD	Mean diameter (mm)
Present study (n=14)	7	6.2
K Takeshita et al[98] (n=8)	8	6.6 +/- 0.5

CALCIFICATIONS:

Of the 14 cases of neoplasm in the study, calcifications are seen only in 4 cases (28.5%). Of these 1 case showed intra lesional calcification, 1 showed intra parenchymal calcification both are of malignant neoplasms. 2 cases of benign neoplasms showed wall calcifications in the lesions. Chi square test p=0.104 stating that there is no specific pattern of calcification in neoplasms. These findings are comparable to study by **Gallotti A et al[107].**

STUDY SERIES	Solid	Cystic	Total
Present study	02	02	-
Gallotti A et al[107]	05	05	10

OTHER MALIGNANT SIGNS:

Of the 10 malignant cases in the study, double duct sign (dilated main pancreatic duct and common bile duct) was seen in 1 case (10%), regional lymph node spread was seen in 4 cases (40%), metastasis was seen in 7 cases (70%), vascular involvement was seen in 4 cases (40%) and in 9 cases (90%) tumour was deemed un resectable due

to the tumour infiltration and spread to vessels or distant metastasis. These findings were similar to study by **Mahmoud A D et al[99].**

Variable	Present Study (%)	Mahmoud A D et al[99] (%)
Double duct sign	10	25
Lymph node involvement	40	20
Metastasis	70	50
Vascular involvement	40	10

- Lymph nodes predominantly involved are peri pancreatic, porta hepatis, pre & para aortic and aorto caval groups in our study.
- Vascular involvement included encasement of celiac axis, hepatic arteries, superior mesenteric vessels, left renal vein, portal vein, splenic vessels and thrombosis of splenic vein leading to few cases of splenic infarct in our study.
- Distant metastasis of primary pancreatic malignant tumour was predominantly to liver, invasion into left kidney, left adrenal gland, posterior wall of stomach and proximal jejunum was also involved in our study. Distant metastasis of secondary malignant cases included rectal wall thickening, vertebral bodies, hip bones and spleen.
- In this study 9 out of 10 malignant cases were deemed un resectable owing to extensive infiltration to adjacent organs, distant metastasis, lymph nodal involvement and vascular involvement in few cases predominantly the celiac axis, superior mesenteric vessels, portal vein and renal vein. In fewer cases the arch of contact between the lesion and vessel was more than 180^0 (>180 degrees) which is specific finding for non resectability of tumour. These findings were similar to studies of **Mahmoud A D et al[99] &** opposed by study of **Anuraj A et al[100]** in which resectable tumours were more.

STUDY SERIES	Resectable tumours	Unresectable tumours	Total
Present Study	01	09	10
Mahmoud A D et al[99]	06	14	20
Anuraj A et al[100]	25	06	31

PROPABLE IMAGING DIAGNOSIS OF NEOPLASMS:

Based on various findings using MDCT triple phase the 14 neoplasms in the study were diagnosed as 4 cases of benign and 10 cases of malignant neoplasms. 2 cases are of mucinous cystadenoma (14.3%), 1 case of Neuro endocrine tumour (7.1%) 1 case of benign solid pseudo papillary tumour (7.1%), 5 cases are adenocarcinomas (35.8%), 4 cases are secondary metastasis from other organs (28.6%) and 1 case of primary lymphoma was noted (7.1%).

These findings are comparable to study by **Tadros MY et al[101] & Hossain MS et al[114].**

❖ **ADENOCARCINOMA:**

5 out of 10 malignant cases in the study are found probably to be adenocarcinoma. 60% of cases are of >60 years age and rest 40% below 60 years. 80% cases are seen in females whereas only 20% in males with M:F ratio of 1:4 stating that females are four times more prone to develop adenocarcinoma than males and old age (>60years) are more prone to develop adenocarcinoma in our study. These findings are comparable to study by **Ichikawa T et al[95].**

CT findings included hypo attenuating, >5cm in size (80%), ill-defined solid mass (100%), involving head & tail (40% each) & body of pancreas (20%), parenchymal destruction with necrosis (100%), obstruction of MPD with dilatation of duct (80%), and heterogeneous /peripheral enhancement on contrast administration

(100%), calcifications in 40%, double duct sign in 20% cases, vascular invasion in 60% with splenic thrombosis in 60%, distant metastasis in 80% with non resectability of tumour in 80% and ascites was noted in 40% cases. These findings are comparable to studies by **Patric Freeny et al (1988)**[82] & **Mahmoud A D et al**[99]**.**

Among these 5 cases one of the case was proven to be adenocarcinoma using biopsy correlation. Since remaining cases have similar findings the provisional diagnosis of adenocarcinoma has been made for all.

❖ **SECONDARY METASTASIS:**

4 out of 10 malignant cases in the study were found to be metastasis of other primaries. Of these 75% (n=3) are from primary liver malignancy and 25% (n=1) from primary gall bladder malignancy. 75% cases are >60 years age and rest 25% below 60 years. 75% cases are seen in males whereas 25% in females with M: F ratio of 3:1.

CT findings included hypo attenuating, <2cm in size (100%), ill-defined solid mass (100%), involving head (50%), body & tail (25% each), necrosis (100%), dilated MPD (25%), and peripheral rim enhancement with central non enhancing areas on contrast administration (75%), distant metastasis in 75% cases is seen involving structures like spleen, vertebral bodies, hip bones and rectum, lymph node involvement is seen in 75%, 25% cases showed ascites and pleural effusion with non resectability of tumour in 100% cases. No cases showed calcifications, double duct sign and vascular invasion.

These findings are similar to studies by **Ioannis Tsitouridis et al**[112] **& Hong-yuan Shi et al**[113] except lung being the most common site of primary in their studies as opposed to our study where liver is most common primary.

❖ LYMPHOMA:

1 out of 10 malignant cases in the study was found to be primary pancreatic lymphoma which is proven by biopsy & is seen in a male patient of age <60 years.

CT findings included iso-hypo attenuating ill-defined lobulated solid mass , >10cm in size involving the head and uncinate process, dilated MPD (5mm) with minimal homogenous enhancement on contrast administration, seen encasing the celiac trunk & its branches, portal vein, splenic vessels, superior mesenteric artery and its branches, renal vessels & left iliac vein with no obvious evidence of infiltration/invasion.

These findings were similar to **Elmar M. Merkle1 et al[108] & Enrico Boninsegna et al[109]** study on biopsy proven pancreatic lymphomas which showed homogenous enhancement & vascular encasement without any infiltration.

❖ MUCINOUS CYSTADENOMA:

2 out of 4 benign cases (50%) in the study were found probably to be mucinous cystadenoma. All the cases are of >60 years age with M: F ratio of 1:1 in our study.

CT findings included well defined hypo attenuating cystic mass (100%), >5cm in size (50%) & <5cm (50%), involving head (50%) & tail (50%) of pancreas), no enhancement on contrast administration (100%), peripheral wall calcifications in 100%, resectability of tumour in 100%. No evidence of necrosis, MPD dilatation, lymph node & distant metastasis, vascular involvement & extra pancreatic complications are seen.

These findings are comparable to study by **Botcha S et al**[117] with female preponderance of 40-60 years, large size cysts, common in head, internal septations and calcifications are common with normal MPD.

- **NEUROENDOCRINE TUMOR:**

1 out of 4 benign cases (25%) in the study was found probably to be neuro endocrine tumour. It is seen in a female patient of age <60 years (52yrs).

CT findings included well defined iso attenuating solid mass, <5cm in size, involving head of pancreas, showing heterogeneous hyper enhancement in arterial phase with washout in portal phases post contrast administration with few central necrotic foci within & tumour is resectable. No evidence of calcific foci, MPD dilatation, lymph node & distant metastasis, vascular involvement & extra pancreatic complications are seen.

These findings were similar to **Tadros MY et al**[101] & **S. Wyatt & E. Fishman (1994)**[103] study where the well-defined small lesion which enhanced more intensely than the normal pancreatic parenchyma in all phases.

- **BENIGN SOLID PSEUDO PAPILLARY TUMOUR:**

1 out of 4 benign cases (25%) in the study was found probably to be benign solid pseudo papillary tumour. It is seen in an adult female patient of age 45 years.

CT findings included well defined heterogeneously hypo attenuating solid mass, 5cm in size, involving tail of pancreas, showing enhancement of solid component with non-enhancing necrotic areas within on contrast administration & tumour is resectable. No evidence of calcific foci, MPD dilatation, lymph node & distant metastasis, vascular involvement & extra pancreatic complications are seen.

These findings are comparable to study by **Estrella JS et al**[132] with 53 patients of pathologically proven SPT showing female preponderance, mean age of 35.4 years,

mostly solid mass in body or tail with mean size of 6.4cm of tumour and may show calcifications.

ATTENUATION PATTERN IN NEOPLASMS (HU Values) –

Hounsfield units (HU) were calculated in all the three phases of solid neoplasms [Arterial Phase (AP), Pancreatic Parenchymal Phase (PPP), and Portal Venous Phase (PVP)]. The mean in AP, PPP and PVP were 73.42, 85.14 and 93.42 HU respectively, subsequent increase in the attenuation value was noted from AP, PPP to PVP except in case of NET where there is wash out in portal phase with reduction of HU in PVP compared to AP & PPP.

Pairwise comparisons was done using Wilcoxon Signed Rank Test; p=0.024 and 0.003 of AP v/s PPP and AP v/s PVP respectively, which is significant, stating that there is significant difference in attenuation value. Whereas p=0.333 noted among PPP v/s PVP, which is not significant.

Thus stating that maximum lesion enhancement is seen in PPP & PVP compared to AP and is better in detection and characterisation of tumour. These findings are comparable to studies by **Tomoaki Ichikawa et al[95] & Tadros MY et al[101]** stating that combination of pancreatic parenchymal phase and PVP imaging is necessary and efficient for the assessment of pancreatic neoplasms as opposed to.

TRAUMA:

Of the 78 patients in the study, 2 cases are of trauma (4%). 1 case is under <20 years age group and 1 in 40-59 years. Both the cases are of males.

CT findings include ill-defined low attenuating areas involving head (50%), body & tail (50%). These lesions showed no enhancement on contrast administration suggestive of contusions. Number of lesions was one in 1 case and two in 1 case (body & tail), MPD was disrupted in 1 case (50%). Associated other organ injuries are noted in both the cases (100%) which included liver laceration, splenic laceration,

lung contusions and rib fractures. Vascular injury was seen in 1 case (50%). Ascites with hemo-peritoneum is seen in 1 case (50%). These findings were similar to studies of **Gordon RW et al[124] & Shadab Maqsood et al[125]**.

Variables	Present Study (n=2)	Gordon RW et al[124] (n=53)	Shadab Maqsood et al[125] (n=46)
Contusion	02	07	02
Laceration	00	16	01
Other organ injury	02	00	34
Ascites	01	02	-

As per AAST guidelines 1 case with parenchymal contusion and MPD disruption is classified under Grade III injury and 1 case with only parenchymal contusions and normal MPD is classified under Grade II injury.

AAST Grading	Present Study (n=2)	Shadab Maqsood et al[125] (n=46)
Grade I	00	01
Grade II	01	02
Grade III	01	00
Grade IV	00	00
Grade V	00	00
TOTAL	**02**	**03**

EXTRA PANCREATIC COMPLICATIONS IN ALL PATHOLOGIES:

Of the 78 patients in the study:

42 cases showed ascites (53.8%). Majority of cases are of acute pancreatitis (n=27) accounting for 64.4%, which includes 13 cases of interstitial oedematous pancreatitis (31%) and 14 cases of necrotizing pancreatitis (33.4%). 8 cases of Acute

on chronic pancreatitis (19%), 3 cases of chronic pancreatitis (7%), 3 cases of neoplasms (7%) and 1 case of trauma (2.6%) showed ascites.

32 cases showed pleural effusion (41%). Majority of cases are of acute pancreatitis (n=23) accounting for 71.8%, which includes 10 cases of interstitial oedematous pancreatitis (31.2%) and 13 cases of necrotizing pancreatitis (40.6%). 7 cases of Acute on chronic pancreatitis (21.8%), 1 case of chronic pancreatitis (3.1%), 1 case of neoplasms (3.1%) and no case of trauma (0%) showed pleural effusion.

9 cases showed thrombosis (11.5%). Majority of cases are of acute pancreatitis (n=4) accounting for 44.5%, all are cases of necrotizing pancreatitis, 2 cases of Acute on chronic pancreatitis (22.2%), 1 case of chronic pancreatitis (3.1%), 3 cases of neoplasms (33.3%) and no case of trauma and chronic pancreatitis (0%) showed thrombosis.

Among all pathologies, the extra pancreatic complications were more common in acute pancreatitis and in particular necrotizing pancreatitis accounting to 33.4%, 40.6% & 44.5% of cases with ascites, pleural effusion & thrombosis respectively. These findings were comparable with the findings in the study by **Shalab Jain et al[61] & Avanesov M et al[64]**

Study series	**Total patients**	**Ascites**	**Pleural effusion**	**Thrombosis**
Present Study	78	42	32	9
Shalab Jain et al[61]	150	54	84	12
Avanesov M et al[64]	102	28	43	12

COMPARISON OF PANCREATITIS WITH NEOPLASM

Phase wise [i.e. arterial phase (AP), Pancreatic Parenchymal Phase (PPP) and Portal Venous Phase (PVP)] comparison was done in acute pancreatitis, acute on chronic pancreatitis, chronic pancreatitis and solid pancreatic neoplasm.

In arterial phase the mean attenuation values of acute pancreatitis, acute on chronic pancreatitis and chronic pancreatitis (112.61, 107.75 and 105.60 HU respectively) are higher than pancreatic neoplasm (73.43 HU). Kruskal Wallis test (p=0.0001) which is highly significant. Multiple comparison was done using post hoc test, there is high significant difference in attenuation value among acute pancreatitis v/s pancreatic neoplasm (p=0.001) and pancreatic neoplasm v/s acute on chronic pancreatitis (p=0.0001). No significant difference among acute pancreatitis v/s acute on chronic pancreatitis (p=0.096). Similar results were obtained in pancreatic parenchymal phase and portal venous phase, as described below.

In pancreatic parenchymal phase the mean attenuation values of acute pancreatitis, acute on chronic pancreatitis and chronic pancreatitis (133.39, 127.88 and 119.00 HU respectively) are higher than pancreatic neoplasm (85.14 HU). Kruskal Wallis test (p=0.0001) which is highly significant. Multiple comparison was done using post hoc test, there is high significant difference in attenuation value among acute pancreatitis v/s pancreatic neoplasm (p=0.033) and pancreatic neoplasm v/s acute on chronic pancreatitis (p=0.0001) and acute pancreatitis v/s acute on chronic pancreatitis (p=0.001).

In portal venous phase the mean attenuation values of acute pancreatitis, acute on chronic pancreatitis and chronic pancreatitis (123.75, 121.75 and 113.00 HU respectively) are higher than pancreatic neoplasm (93.43 HU). Kruskal Wallis test (p=0.0001) which is highly significant. Multiple comparison was done using post hoc

test, there is high significant difference in attenuation value among acute pancreatitis v/s pancreatic neoplasm (p=0.001) and pancreatic neoplasm v/s acute on chronic pancreatitis (p=0.001). No significant difference among acute pancreatitis v/s acute on chronic pancreatitis (p=0.750).

These findings are comparable to study by **Bharat et al**[63] for pancreatitis & to studies by **Tomoaki Ichikawa et al**[95] **& Tadros MY et al**[101] for neoplasms.

To conclude, the maximum attenuation (HU) is seen in PPP in all cases of pancreatitis and in PVP in cases of tumours. The mean attenuation values of pancreatitis are higher than tumours in all phases. Hence PPP is the optimal phase for evaluation of parenchyma in pancreatitis whereas PPP & PVP are useful over AP for evaluation of lesion characters and extra pancreatic findings in neoplasms.

SUMMARY

A prospective study of 78 patients was carried out over the period of two years in suspected pancreatic abnormality patients and to evaluate the role of triple phase [arterial phase(AP), pancreatic parenchymal phase(PPP), and portal venous phase(PVP)] multi–detector row Computed Tomographic (MDCT) using a specific protocol as mentioned in the methodology.

Mean age of the patients was ~45years, majority belonged to the age group of 20 – 39 years (n=30) and majority of them being males (n=54). Maximum numbers of cases were of acute pancreatitis (n=36).Male predominance was noted in inflammatory and traumatic pathologies whereas female predominance was noted in neoplasms. Maximum cases of Acute pancreatitis & Acute on chronic pancreatitis were seen in <40years age whereas Chronic pancreatitis & Tumours are seen in age group of >60 years.

In **Acute pancreatitis** (n=36) [IEP-17 & NP-19] all of the cases had elevated serum lipase levels (n=36) whereas elevated serum amylase was seen in 32 cases (14-IEP & 18-NP). Modified CT Severity Index (MCTSI) was assessed, and most of the cases were of moderate grade (4-6) in IEP (94.1%) and severe grade (8-10) in NP (89.5%).

Significant ‘p value’(<0.001) is seen in IEP & NP cases in reference to bulky pancreas (100% & 89%), fat stranding (100% & 100%), acute peri pancreatic fluid collection in IEP (88%), acute necrotic collection in NP (84%) suggesting these are specific for acute pancreatitis. Walled off necrosis were seen only in cases of NP with significant ‘p value’ (0.0215) stating its specificity.

Calculated Hounsfield units (HU) in all the three phases of acute pancreatitis showed increased attenuation in AP to PPP, whereas a mild drop was noted in the

PVP. Pairwise comparisons showed high significant difference in attenuation value among AP vs PPP, AP vs PVP & PPP vs PVP phases with p values 0.001, 0.001 & 0.0001 respectively.

In **Acute on chronic pancreatitis** (n=16) most of the cases (n=11) had elevated serum amylase (69%). Majority of the cases had pancreatic calcifications (43.7%), dilated main pancreatic duct (44%) and pseudo cysts (69%) with acute findings like bulky pancreas (69%), fat stranding (88%), necrosis (63%) and peri pancreatic fluid (50%), all showing significant 'p values' suggesting the presence of specific acute ($p<0.001$) and chronic ($p=0.002$) findings. Attenuation value evaluated in all the three phases revealed increased attenuation in AP to PPP, whereas a mild drop was noted in the PVP. Pairwise comparisons showed high significant difference in attenuation value among AP vs PPP, AP vs PVP & PPP vs PVP phases with p values 0.001, 0.001 & 0.0001 respectively.

In **Chronic pancreatitis** (n=10) most of the cases (n=9) had normal serum amylase ($p<0.001$), 80% with atrophic pancreas ($p<0.001$), pancreatic calcifications (60%), pseudo cysts (70%) ($p<0.0001$) and dilated MPD (90%) ($p=0.002$) with mean MPD diameter was 8.3mm. All of these showing significant 'p value' stating they are more specific for chronic pancreatitis. Attenuation value evaluated in all the three phases revealed increased attenuation in AP to PPP, whereas a mild drop was noted in the PVP. Pairwise comparisons showed high significant difference in attenuation value among AP vs PPP, AP vs PVP & PPP vs PVP phases with p values 0.001, 0.010 & 0.040 respectively.

Serum Amylase & lipase correlation among pancreatitis showed elevated serum lipase & amylase levels in patients with interstitial oedematous pancreatitis (100% & 82.3%), necrotizing pancreatitis (100% & 94.7%), and acute on chronic pancreatitis

(93.7% & 68.7%) and normal serum amylase & lipase levels in chronic pancreatitis (90%) with Chi square test value of p<0.001(highly significant) stating that increase in serum lipase & amylase levels is more specific for acute and acute on chronic pancreatitis with serum lipase more reliable than serum amylase evaluation.

MPD correlation among pancreatitis showed main pancreatic duct (MPD) is dilated in 90% cases of chronic pancreatitis followed by 44% acute on chronic pancreatitis as opposed to very few cases of acute oedematous & necrotizing pancreatitis with Chi square test value of p=0.002 (highly significant) stating that MPD dilatation is more specific for chronic followed by acute on chronic pancreatitis.

In **Pancreatic neoplasms** (n=14), majority are seen in females (57%), age of >60 years (57%), majority are malignant neoplasms (71.5%), solid tumours (n=12) contributed the majority (86%), majority involved head of pancreas (50%), in malignant cases 40% had lymph node spread, metastasis in 70% and 90% of malignant are un resectable whereas all benign masses are resectable at the time of imaging.

- **Adenocarcinoma** constitutes majority of malignant cases (50%) with 4:1 (F: M) ratio presenting as ill-defined hypo attenuating solid mass of >5cm in size with parenchymal destruction (100%), causing obstruction of MPD with dilatation (80%), vascular invasion (60%), distant metastasis (80%) and non resectability in 80% cases.
- **Secondary metastases** are second most common malignant neoplasms (n=4) in the study constituting 40%. Among these 75% (n=3) are from liver as primary and 25% (n=1) from gall bladder. They presented as ill-defined hypo attenuating solid masses of <2cm in size with peripheral rim enhancement, distant metastasis (75%) and non respectability in all cases.

- **Lymphoma** of pancreas was seen in 1 case involving the head region as an ill-defined lobulated iso attenuating solid mass >10cm in size, seen encasing multiple vessels like celiac trunk, superior mesenteric vessels & branches, splenic, iliac vessels with no invasion/infiltration and shows mild homogenous enhancement on contrast administration.
- **Mucinous cystadenoma** constitutes majority of benign cases (50%) with 1:1 (M: F) ratio and >60 years. They presented as well defined hypo attenuating cystic lesions >5cm in size with wall calcifications and no enhancement on contrast administration.
- **Neuro Endocrine tumour** of pancreas was seen in 1 case involving head of pancreas, well defined iso attenuating solid mass hyper enhancing with washout on portal phase & no infiltration/duct dilatation.
- **Benign pseudo papillary neoplasm** of pancreas was seen in an adult female patient involving predominantly tail of pancreas, appearing well defined heterogeneously hypo attenuating solid mass of 5cm size with heterogeneous enhancement due to necrotic areas and no calcification, duct involvement, invasion or metastasis is seen.

Attenuation value calculated in all the three phases of pancreatic neoplasms, there was subsequent increase in the attenuation from AP, PPP to PVP. Pair wise comparisons of AP v/s PPP and AP v/s PVP displayed significant difference (p=0.024 and p=0.003 respectively).and no significant difference between PPP vs PVP (p=0.333).

The attenuation values (i.e. HU) of all the three phases were compared among pancreatitis and neoplasm:

- In **arterial phase,** attenuation values of all types of pancreatitis are higher than pancreatic neoplasm (Kruskal Wallis test, p=0.0001). Multiple comparison (Post

hoc test) in this phase revealed high significant difference in attenuation among acute pancreatitis v/s pancreatic neoplasm (p=0.001) and pancreatic neoplasm v/s acute on chronic pancreatitis (p=0.0001).

- In **pancreatic parenchymal phase,** the attenuation values of all types of pancreatitis are higher than pancreatic neoplasm (Kruskal Wallis test, p=0.0001). Multiple comparison (Post hoc test) in this phase revealed high significant difference in attenuation among acute pancreatitis v/s pancreatic neoplasm (p=0.033), acute pancreatitis v/s acute on chronic pancreatitis (p=0.001) and pancreatic neoplasm v/s acute on chronic pancreatitis (p=0.0001).
- In **portal venous phase,** the attenuation values of all types of pancreatitis are higher than pancreatic neoplasm (Kruskal Wallis test, p=0.0001). Multiple comparison (Post hoc test) in this phase revealed high significant difference in attenuation value among acute pancreatitis v/s pancreatic neoplasm (p=0.001) and pancreatic neoplasm v/s acute on chronic pancreatitis (p=0.001).

Comparison of complications of all pathologies showed that ascites is the most common complication accounting to 54% of the cases. Among various pathologies, maximum cases of ascites (33.4%), pleural effusion (40.6%) and splenic thrombosis (44.5%) are noted in Necrotizing pancreatitis followed by oedematous pancreatitis in ascites, pleural effusion and tumours in thrombosis.

Limitations encountered in the study were:

- Measurement of attenuation HU with the lesion was varying; this was taken care of by placing the ROI in the area of maximum density of the lesion.
- Fewer number of trauma cases limited better evaluation of patterns of pancreatic trauma.

- Pathological correlation of all the solid pancreatic neoplasm would give more weightage to imaging diagnosis.
- Follow up scans, for all the lesions would have added post treatment changes / resolution of the lesion / residual changes / changing pattern of the disease entity and is beyond the period of study.

CONCLUSION:

A prospective study of 78 patients was carried out in suspected cases of pancreatic abnormality with MDCT in triple phase using a specific protocol.

The aims of the study, were to evaluate and characterize various inflammatory, neoplastic and traumatic cases of pancreas with measurement of lesion attenuation (Hounsfield Unit) values in triple phase contrast study.

The results were as follows:

- Neoplasms are more common in females with an M: F ratio of 1:4 in adenocarcinomas particularly. Inflammatory pathologies & trauma are common in males.
- With increase in age there is shift in frequency of cases from inflammatory to neoplasm (above 60 years).
- Elevated serum lipase & amylase is more specific to acute pancreatitis (necrotizing pancreatitis followed by interstitial oedematous pancreatitis), acute on chronic pancreatitis and is low to normal in chronic pancreatitis. Serum lipase is more reliable than amylase for assessment of pancreatitis.
- MDCT characters like bulky pancreas, fat stranding are noted in almost all cases of acute pancreatitis, acute peri pancreatic fluid collection in IEP (88%) & acute necrotic collection in NP (84%) with high significance suggesting these are specific.
- Walled off necrosis was seen only in cases of Necrotizing Pancreatitis with high significance stating its specificity.

- In Chronic pancreatitis, majority of the cases had atrophic pancreas, dilated MPD, pancreatic parenchymal calcification and pseudo cysts with high significance stating their specificity.
- Acute, acute on chronic & chronic pancreatitis showed increased attenuation in AP to PPP, whereas a mild drop was noted in the PVP.
- In pancreatic neoplasm, head of pancreas is most common location, solid tumours were more common and majority of them are malignant.
- Mucinous cystadenoma was the most common benign tumour whereas Adenocarcinoma is the most common malignant tumour followed by secondary metastasis predominantly from liver.
- A benign solid tumour which is well defined, seen involving tail of pancreas when occurs in an adult female with heterogeneous enhancement and no ductal involvement, metastasis is likely to be solid pseudo papillary tumour.
- All the tumours were of low attenuation except Lymphoma and Neuroendocrine tumour (NET) which were iso attenuating mass lesions.
- All metastatic tumours showed peripheral rim enhancement, Lymphoma showed mild homogenous enhancement, significant hyper enhancement in NET with washout in portal phase and no enhancement in cystic lesions of mucinous cystadenoma.
- Majority of adenocarcinoma cases showed parenchymal destruction, MPD obstruction with dilatation, vascular involvement and distant metastasis.
- Almost all malignant neoplasms are unresectable owing to infiltration of adjacent organs, lymph nodal involvement, distant metastasis and vascular involvement (>180^{o} arch of contact of lesion with vessel)

- Pancreatic neoplasm showed subsequent increase in the attenuation from AP, PPP to PVP.
- In pancreatic neoplasm, among pairwise comparisons of attenuation only AP v/s PPP and AP v/s PVP projected significant difference but not in PPP v/s PVP. Hence PPP & PVP are useful over AP for evaluation of lesion characters and extra pancreatic findings in neoplasms.
- In all the three phases, attenuation of acute pancreatitis, acute on chronic pancreatitis and chronic pancreatitis is higher than pancreatic neoplasm. Hence would help to differentiate focal chronic pancreatitis from mass.
- Trauma cases showed predominantly contusions, with MPD disruption in one case, no enhancement on contrast study with one case each under Grade II & III as per AAST guidelines.
- Ascites, pleural effusion and thrombosis are the complications observed among various pathologies with ascites being the most common pathology and maximum complications are registered among the cases of Necrotizing pancreatitis.

***To conclude*:** As per our study, MDCT with triple phase imaging protocol of pancreas helps in better evaluation of various pancreatic pathologies with phase wise characterization, detection of lesions as small as 1.5cm in size and assessment of resectability of a neoplastic lesion. Thus aiding in better, accurate diagnosis of pathologies and in further treatment planning.

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ANNEXURE-I

ETHICAL COMMITTEE CLEARANCE CERTIFICATE

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04-10-2016 *at* 3-00pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title "Role of Multi detector computed Tomography in evaluation of pancreatic pathologies"

Name of P.G. student Dr. Adiraju Karthik.
Dept of Radiology & Imaging

Name of Guide/Co-investigator Dr Bhushan. N. Lakhkar.
Prof & HOD. Radiology & Imaging.

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE-II

CASE SHEET PROFORMA

- NAME:
- AGE:
- SEX:
- IP/OP NO:

- CHIEF COMPLAINTS:
  - Pain Abdomen
  - Nausea/Vomiting
  - Fever
  - Jaundice
  - Trauma

- PAST HISTORY :
  - Diabetes
  - Hypertension
  - Similar episodes in past
  - Previous Surgical history

- FAMILY HISTORY
  - Similar complaints in family
  - Cancer history

- TREATMENT HISTORY

- RELEVANT PER ABDOMEN EXAMINATION
  - Inspection -Cullen's sign
    -Gray Turner sign
  - Palpation - Tenderness ,guarding ,distension.
  - Auscultation

- PROVISIONAL CLINICAL DIAGNOSIS

- RADIOLOGICAL FINDINGS :

PANCREATIC CHARACTERISTICS			
		YES	NO
SIZE	NORMAL		
	ATROPHY		
	BULKY		
CONTOUR	REGULAR		
	IRREGULAR		
ATTENUATION	HOMOGENOUS		
	INHOMOGENOUS		
DUCT	NORMAL		
	DILATED		
	DESTROYED		
NECROSIS	<30 %		
	>30%		
CALCIFICATION	PARENCHYMAL		
	DUCTAL		
MASS LESION	HEAD		
	BODY		
	TAIL		

EXTRA PANCREATIC CHANGES			
S.NO	FINDING	YES	NO
01	PERIPANCREATIC FAT STRANDING		
02	PERIPANCREATIC FLUID COLLECTION		
03	PSEUDOCYST		
04	WALLED OFF NECROSIS		
05	HEMORRHAGE		
06	THROMBOSIS		
07	ANEURYSM		
08	ASCITES		
09	PLEURAL EFFUSION		
10	CBD DILATATION		

POST CONTRAST ENHANCEMENT STUDY (HU VALUES)			
LESION	ARTERIAL PHASE	PARENCHYMAL PHASE	PORTAL VEINOUS PHASE
ACUTE PANCREATITIS			
CHRONIC PANCREATITIS			
MALIGNANT MASS			
BENIGN MASS			
LACERATION/CONTUSION			

ANNEXURE-III

CONSENT FORM

TITLE OF RESEARCH: **ROLE OF MULTI DETECTOR COMPUTED TOMOGRAPHY IN EVALUATION OF PANCREATIC PATHOLOGIES**

GUIDE : DR. BHUSHAN N. LAKHKAR

P.G. STUDENT : DR. ADIRAJU KARTHIK

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to study inflammatory and tumor pathologies of the Pancreas along with their complications on MDCT.

I understand that I will undergo detailed history and clinical examination and investigations.

RISKS AND DISCOMFORTS:

I understand the risks involved (as informed prior to procedure viz allergic reactions, skin dryness, itching & rarely long term effects) and I may experience mild pain during the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help in determining role of Multi detector computed tomography in evaluation of pancreatic lesions.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulations of hospital. If the data is used for publications the identity of the patient will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I/my ward understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I/my ward also understand that Dr. Adiraju Karthik will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date:

Dr. Bhushan N Lakhkar (Guide)

Dr.Adiraju Karthik (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I/my ward confirm that Dr.Adiraju Karthik has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I/my ward have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

KEY TO MASTER CHART

M	-	MALE
F	-	FEMALE
B	-	BULKY
AT	-	ATROPHY
NR	-	NORMAL
PPC	-	PERIPANCREATIC COLLECTON
WON	-	WALLED OFF NECROSIS
MPD	-	MAIN PANCREATIC DUCT
DIL	-	DILATED
OBS	-	OBSTRUCTED
DSRPTN	-	DISRUPTION
IP	-	INTRAPARENCHYMAL
ID	-	INTRADUCTAL
ILC	-	INTRALESIONAL CALCIFICATION
WC		WALL CALCIFICATION
MCTSI	-	MODIFIED CT SEVERITY INDEX
NA	-	NOT APPLICABLE
PLEF	-	PLEURAL EFFUSION
ASC	-	ASCITES
THROMB	-	THROMBOSIS
S	-	SPLENIC VEIN
P	-	PORTAL VEIN
BEN	-	BENIGN
MAL	-	MALIGNANT
ILD	-	ILL-DEFINED
WD	-	WELL-DEFINED
HEAD&UN	-	HEAD AND UNCINATE PROCESS
NUM	-	NUMBER
MIN HOM	-	MINIMAL HOMOGENOUS
HTR	-	HETEROGENEOUS
PER HTR	-	PERIPHERAL HETEROGENEOUS

DDS	-	DOUBLE DUCT SIGN
VASINVASN	-	VASCULAR INVASION
LMNS	-	LYMPHNODES
METS	-	METASTASIS
MIN	-	MINIMUM
MAX	-	MAXIMUM
MDCT	-	MULTI DETECTOR COMPUTED TOMOGRAPHY
IEP	-	INTERSTITIAL OEDEMATOUS PANCREATITIS
NP	-	NECROTIZING PANCREATITIS
ACP	-	ACUTE ON CHRONIC PANCREATITIS
CP	-	CHRONIC PANCREATITIS
LMPOMA	-	LYMPHOMA
ADNCA	-	ADENOCARCINOMA
SPT	-	SOLID PSEUDO PAPILLARY TUMOUR
GB/METS	-	GALL BLADDER METASTASIS
LVR/METS	-	LIVER METASTASIS
MCN	-	MUCINOUS CYSTADENOMA
NET	-	NEURO ENDOCRINE TUMOUR

CASES OF PANCREATITIS (62 CASES)

PATIENT DETAILS					PANCREATITIS MDCT CHARACTERISTICS											COMPLICATIONS			ATTENUATION VALUES (HU)					
S.NO	NAME OF PATIENT	AGE	SEX	DIAGNOSIS	S.AMYLASE/ LIPASE	SIZE	FAT STRANDING	NECROSIS	PPC	PSEUDO CYST	WON	MPD	MPD SIZE (cm)	CALCIFICATION	MCTSI SCORE	PLEF	ASC	THROMB	ARTERIAL PHASE (AP)		PANCREATIC PARENCHYMAL PHASE (PPP)		PORTAL VENOUS PHASE (PVP)	
																			Min	Max	Min	Max	Min	Max
1	RAVI D	32	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	YES	YES	NO	72	106	86	130	76	120
2	RAMESH	32	M	ACP	INC	NR	NO	YES	YES	YES	NO	NR	NR	NO	6	YES	NO	NO	88	106	98	130	96	126
3	BIBIJAN	53	F	IEP	NR	B	YES	NO	YES	NO	NO	NR	NR	NO	4	YES	YES	NO	76	108	96	132	84	124
4	SADASHIV	31	M	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	YES	NO	NO	84	118	114	140	98	128
5	SIDDAPPA	33	M	ACP	INC	B	YES	YES	NO	YES	NO	NR	NR	NO	8	YES	YES	NO	94	108	118	134	114	128
6	PREETHI	21	F	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	YES	YES	NO	82	116	104	136	96	122
8	VILAS	35	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	NO	NO	NO	90	120	110	138	102	128
9	NEELAWW.	68	F	CP	NR	AT	NO	NO	NO	YES	NO	DIL	3	NO	NA	NO	NO	NO	78	98	88	114	84	108
10	MAHESH	32	M	NP	INC	B	YES	YES	YES	YES	NO	NR	NR	NO	8	YES	YES	NO	88	118	104	136	94	124
12	RUKMA	40	F	IEP	INC	B	YES	NO	YES	NO	NO	DIL	5	NO	6	NO	YES	NO	86	114	106	136	96	126
13	MUTAPPA	68	M	CP	INC	AT	YES	NO	NO	YES	NO	DIL	9.9	IP&ID	NA	NO	YES	NO	80	114	94	120	88	116
14	SHIVA K	42	M	ACP	NR	B	YES	NO	NO	YES	NO	DIL	4	NO	4	NO	NO	NO	92	104	104	124	98	118
16	UMESH	18	M	CP	NR	NR	NO	NO	NO	YES	NO	DIL	7	IP	NA	NO	NO	NO	82	104	98	120	94	114
17	RANIBAI	46	F	ACP	NR	NR	YES	NO	NO	NO	NO	DIL	6	NO	2	NO	NO	NO	94	108	106	124	100	118
18	HANAMAN	36	M	ACP	INC	B	YES	YES	YES	YES	NO	DIL	5	NO	6	YES	YES	YES/S	96	106	110	130	102	128
19	BASVARAJ	48	M	ACP	INC	B	YES	YES	YES	YES	NO	NR	NR	NO	6	NO	NO	NO	88	108	102	126	98	120
20	BIBI.M	25	F	ACP	INC	B	YES	YES	YES	YES	NO	DIL	8.5	IP	8	YES	YES	NO	98	112	114	132	108	124
21	MAHADEV	35	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	10	NO	YES	NO	78	112	98	130	86	122
22	DANAMMA	65	F	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	10	YES	YES	YES/S&P	80	114	104	136	92	126
23	PREETI	22	F	ACP	INC	B	NO	YES	NO	YES	NO	NR	NR	IP	8	YES	YES	NO	94	108	106	124	100	118
24	ABDUL R	20	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	YES	YES	NO	84	108	100	130	94	126
25	RAMESH	42	M	CP	NR	AT	NO	NO	NO	NO	NO	DIL	9	IP&ID	NA	NO	NO	NO	78	106	94	118	86	112
26	PRAKASH	43	M	NP	NR	NR	YES	YES	NO	NO	NO	NR	NR	NO	4	NO	NO	NO	71	102	90	126	82	118
27	**PANDURAN**	**35**	**M**	**ACP**	**INC**	**B**	**YES**	**NO**	**YES**	**YES**	**NO**	**NR**	**NR**	**NO**	**6**	**YES**	**YES**	**NO**	**92**	**104**	**104**	**124**	**98**	**118**
28	SHIVU	28	M	ACP	INC	AT	YES	YES	NO	YES	NO	NR	NR	NO	8	YES	YES	YES/S&P	88	106	102	120	94	114
30	BASAVARA	30	M	IEP	NR	B	YES	NO	YES	NO	NO	NR	NR	NO	4	NO	NO	NO	73	108	88	130	80	122
31	SUNANDA	30	F	ACP	NR	B	YES	NO	NO	NO	NO	NR	NR	IP	4	NO	YES	NO	88	98	114	124	96	120
32	KRISHNA	38	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	10	YES	NO	NO	74	110	86	128	80	118
35	MALLIKARJ	47	M	CP	NR	AT	NO	NO	NO	YES	NO	DIL	8	NO	NA	NO	NO	NO	82	112	96	122	90	118
37	RAMAGON	40	M	ACP	INC	B	YES	YES	YES	YES	NO	NR	NR	NO	6	NO	NO	NO	90	112	108	132	98	124
39	NAGAPPA	63	M	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	YES	YES	NO	82	118	98	138	90	126
41	VITTAL SB	70	M	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	4	NO	NO	NO	84	116	104	134	96	124
42	SANTOSH	32	M	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	NO	YES	NO	87	121	106	140	98	132
43	IRAPPA	90	M	CP	NR	NR	NO	NO	NO	YES	NO	DIL	7	IP&ID	NA	YES	YES	NO	76	98	84	116	80	108
44	JYOTHI	22	F	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	YES	YES	NO	87	119	108	138	92	127
45	CHANDRAS	62	M	ACP	INC	B	YES	YES	YES	NO	NO	DIL	3.5	IP	6	NO	NO	NO	90	112	108	132	98	124
46	BASVARAJ	28	M	NP	INC	NR	YES	YES	NO	NO	YES	DIL	4.8	NO	6	NO	NO	NO	82	118	98	134	92	126
47	RAJSEKHAR	38	M	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	NO	YES	NO	80	110	102	134	96	122
48	ARSIYA	18	F	IEP	NR	B	YES	NO	NO	NO	NO	NR	NR	NO	2	NO	NO	NO	76	104	94	128	88	120
49	RANIBAI	70	F	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	YES	YES	NO	78	108	96	134	86	126
50	VIJAY	32	M	ACP	NR	NR	YES	NO	YES	YES	NO	NR	NR	IP	4	NO	NO	YES/S	98	112	114	132	108	124
51	AKASH	17	M	CP	NR	AT	YES	NO	NO	YES	NO	DIL	8	IP	NA	NO	YES	NO	84	116	98	126	90	120
53	GOUSPAK	32	M	NP	INC	B	YES	YES	YES	NO	YES	NR	NR	NO	8	YES	YES	NO	82	118	98	136	90	124
54	PRABHU	45	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	NO	YES	NO	84	116	104	138	96	126
56	CHNADRAK	55	M	CP	NR	AT	NO	NO	NO	NO	NO	NR	NR	NO	NA	NO	NO	NO	76	98	84	116	80	108
58	SAFIA	70	F	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	YES	YES	NO	88	120	108	140	96	132
59	BHIMARAO	45	M	NP	INC	B	YES	YES	NO	YES	YES	NR	NR	NO	8	YES	YES	NO	84	114	102	136	94	124
61	JANAPPA	40	M	IEP	INC	B	YES	NO	NO	NO	NO	NR	NR	NO	6	YES	YES	NO	78	104	96	130	86	120
62	RAKESH	28	M	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	YES	YES	NO	76	100	102	126	92	118
63	NABILAL	52	M	CP	NR	AT	NO	NO	NO	YES	NO	DIL	10	IP&ID	NA	NO	NO	NO	78	106	94	118	86	112
64	RUKMABAI	70	F	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	YES	YES	NO	74	108	96	130	88	120
65	CHANDAM	70	F	CP	NR	AT	NO	NO	NO	NO	NO	DIL	13	NO	NA	NO	NO	NO	82	104	98	120	94	114
66	ARAVIND	32	M	IEP	INC	B	YES	NO	NO	YES	NO	NR	NR	NO	6	YES	YES	NO	80	112	102	128	96	118
67	KAVERI	15	F	ACP	NR	B	YES	NO	NO	NO	NO	DIL	4.2	IP&ID	2	NO	YES	NO	90	112	108	132	98	124
68	DATTA	27	M	NP	INC	B	YES	YES	YES	NO	NO	DIL	3.4	NO	8	YES	YES	YES/S	82	114	100	130	96	124
69	RAJENDRA	49	M	NP	INC	B	YES	YES	YES	NO	NO	DIL	3.5	NO	10	YES	YES	YES/S	84	118	98	138	90	126
70	DINESH D	34	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	NO	NO	NO	88	112	106	132	94	124
71	PARAMANA	48	M	NP	INC	B	YES	YES	YES	NO	NO	NR	NR	NO	8	YES	YES	NO	80	108	102	128	94	122

72	DINESH D	35	M	NP	INC	B	YES	YES	YES	NO	YES	NR	NR	NO	8	YES	YES	NO	76	110	98	132	84	122
73	PRASANNA	18	M	IEP	INC	B	YES	NO	YES	NO	NO	VARIANT	NR	NO	6	NO	YES	NO	80	114	102	134	90	126
76	VR PATIL	55	M	IEP	INC	B	YES	NO	YES	NO	NO	NR	NR	NO	6	YES	YES	NO	82	118	104	136	94	122
77	RAGAVEND	36	M	ACP	INC	NR	YES	YES	NO	NO	NO	DIL	6	IP&ID	6	NO	NO	NO	88	108	102	126	98	120

CASES OF PANCREATIC NEOPLASMS (14 CASES) & TRAUMA (2 CASES)

PATIENT DETAILS										NEOPLASM CHARACTERISTICS ON MDCT														COMPLICATIONS			TRAUMA FINDINGS				ATTENUATION VALUES (HU)					
S.NO	NAME OF PATIENT	AGE	SEX	DIAGNOSIS	NECROSIS	PPC	MPD	MPD.SIZE (mm)	CALCIFICATION	MASS	MEASUREMENT (cm)	BEN/MALIG	SOLID/CYSTIC	MARGINS	LOCATION	NUM	ATTENUATION	ENHANCEMENT	DDS	VASINVASN	LMNS	METS	TUMOR DIAGNOSIS	RESECTABILITY	PLEF	ASC	THROMBOSIS	CONTUSION	LACERATION	ASSOCIATED INJURIES	AAST GRADE	ARTERIAL PHASE (AP)		PANCREATIC PARENCHYMAL PHASE (PPP)		PORTAL VENOUS PHASE (PVP)	
																																Min	Max	Min	Max	Min	Max
7	RAVI DJ	44	M	TUMOUR	NO	NO	DIL	5	NO	YES	14x12	MAL	SOLID	ILD	HEAD&UN	1	ISO	MIN HOM	NO	YES	YES	NO	LMPOMA	NO	NO	NO	NO	NA	NA	NA	NA	58	65	64	70	68	74
11	HANAMAW	50	F	TUMOUR	YES	NO	DIL/OBS	3.5	NO	YES	6x5	MAL	SOLID	ILD	TAIL	1	LOW	HTR	NO	YES	NO	YES	ADNCA	NO	NO	NO	YES/S	NA	NA	NA	NA	44	71	52	76	60	82
15	SARABEE	37	F	TUMOUR	YES	NO	DIL/OBS	6	ILC	YES	6x5	MAL	SOLID	ILD	TAIL	1	LOW	HTR	NO	YES	NO	YES	ADNCA	NO	NO	YES	YES/S	NA	NA	NA	NA	60	72	64	76	70	91
29	INDUMATI	46	F	TUMOUR	NO	NO	NR	NR	NO	YES	5x4	BEN	SOLID	WD	TAIL	1	LOW	HTR	NO	NO	NO	NO	SPT	YES	NO	NO	NO	NA	NA	NA	NA	35	45	45	57	58	77
33	NAGAWW/	85	F	TUMOUR	YES	NO	NR	NR	NO	YES	2x1.9	MAL	SOLID	ILD	HEAD	1	LOW	PERHRL	NO	NO	YES	NO	GB/METS	NO	NO	NO	NO	NA	NA	NA	NA	68	80	74	109	84	137
34	CHANDRAV	85	F	TUMOUR	YES	NO	DIL	9	IP	YES	5x3.5	MAL	SOLID	ILD	HEAD	1	LOW	HTR	YES	NO	NO	NO	ADNCA	YES	NO	YES	NO	NA	NA	NA	NA	50	77	56	82	62	88
36	HABBULLI	80	F	TUMOUR	NO	NO	DIL/OBS	7	NO	YES	4.5x3.7	MAL	SOLID	ILD	HEAD	1	LOW	HTR	NO	NO	NO	YES	ADNCA	NO	NO	NO	NO	NA	NA	NA	NA	58	63	64	84	74	109
38	SHIVU S	48	M	TUMOUR	YES	YES	DIL	6.5	NO	YES	<2	MAL	SOLID	ILD	HEAD	1	LOW	PERHRL	NO	NO	YES	YES	LVR/METS	NO	NO	NO	NO	NA	NA	NA	NA	52	74	58	92	66	98
40	CHANDRAS	63	M	TUMOUR	NO	NO	NR	NR	NO	YES	<2	MAL	SOLID	ILD	BODY	1	LOW	PERHRL	NO	NO	YES	YES	LVR/METS	NO	NO	NO	NO	NA	NA	NA	NA	48	76	56	90	62	98
52	BN KUCHN	92	M	TUMOUR	NO	NO	NR	NR	WC	YES	4x3	BEN	CYSTIC	WD	HEAD	1	LOW	NO	NO	NO	NO	NO	MCN	YES	NO	NO	NO	NA	NA	NA	NA	54	76	60	93	66	92
55	IRAPPA.B	80	M	TUMOUR	YES	NO	OBS	NR	NO	YES	6x5	MAL	SOLID	ILD	BDY&TAIL	1	LOW	HTR	NO	YES	NO	YES	ADNCA	NO	NO	NO	YES/S	NA	NA	NA	NA	64	77	68	95	74	111
57	IRAPPA	65	M	TUMOUR	YES	NO	NR	NR	NO	YES	2	MAL	SOLID	ILD	BDY&TAIL	1	LOW	PERHRL	NO	NO	NO	YES	LVR/METS	NO	YES	YES	NO	NA	NA	NA	NA	48	75	54	88	60	96
60	BIBI B	70	F	TUMOUR	NO	NO	NR	NR	WC	YES	9x7.5	BEN	CYSTIC	WD	TAIL	1	LOW	NO	NO	NO	NO	NO	MCN	YES	NO	NO	NO	NA	NA	NA	NA	52	72	58	88	62	90
74	SAMEER D	19	M	TRAUMA	NO	NO	DSRPTN	NR	NO	NO	NA	NA	NA	ILD	HEAD	1	LOW	NO	NO	NO	NO	NO	NA	NO	NO	YES	NO	YES	NO	YES	III	24	45	26	51	22	42
75	RAJASEB H	45	M	TRAUMA	NO	NO	NR	NR	NO	NO	NA	NA	NA	ILD	BDY&TAIL	2	LOW	NO	NO	NO	NO	NO	NA	NO	NO	NO	NO	YES	NO	YES	II	12	22	17	26	14	19
78	KAMALABA	52	F	TUMOUR	YES	NO	NR	NR	NO	YES	4x3	BEN	SOLID	WD	HEAD	1	ISO	HYPER	NO	NO	NO	NO	NET	YES	NO	NO	NO	NA	NA	NA	NA	88	105	76	92	58	65