CT EVALUATION OF PANCREATIC LESIONS

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By

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This is to certify, the ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved, ***Dr. ANKITA CHAUHAN*** PG student in the subject of RADIODIAGNOSIS at Sri Devaraj Urs Medical College, Kolar to take up the dissertation work titled "***CT evaluation of pancreatic lesions"*** to be submitted to Sri Devaraj Urs Academy of Higher Education and Research, Kolar.

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ABSTRACT

Background/Objectives:

To evaluate the pancreatic lesions by computed tomography (CT).

Materials and Methods:

During the period of 18 months of the study, 39 patients who fulfilled inclusion criteria were studied by using SIEMENS SOMATOM ESPRIT single slice spiral CT machine. The exposure settings used were 130 KvP and 100 mAs. Plain CT scan abdomen 10 mm axial cuts were taken for all the cases. Oral contrast (10-15mL of ultravist in 2L of flavoured water) was given. After 45-60 min, Triple-phase contrast study of CT abdomen was undertaken by administering 80mL of ultravist @ 2.5mL/s by a pressure injector.

Results:

Contrast-enhanced CT abdomen study showed acute inflammatory changes in 21 cases(53.8 %), pseudopancreatic cyst in 14 cases(35.89%), chronic pancreatitis in 5 cases(12.8%), isolated pseudopancreatic cyst in 3 cases(7.69%), and tumor in 6 cases(15.38%).

Maximum of the acute pancreatitis cases belong to Balthazar grade C (42.86%).

Pancreas was found to be bulky, altered attenuation and peripancreatic fat strandings in significant number of cases, but necrosis, parenchymal or ductal calcifications, dilated duct, peripancreatic fluid, pancreatic infiltration by adjacent tumors, and adjacent vascular thrombosis/fat infiltration was found in only few cases.

Ascites and pleural effusion were noted in quite a number of cases. Diffuse fatty infiltration of liver, gallbladder calculus and common bile duct dilatation was seen in only few cases.

Conclusion:

The results of the present study show that Helical Computed Tomography of the abdomen has improved the visualization of pancreas, more so in cases where pancreas is obscured on ultrasonography by poor acoustic window owing to excessive bowel gas. It not only enables proper assessment of pancreatic size and morphology, but also hepatobiliary system, vasculature, spleen, bowel loops, and even bases of lungs can be assessed and commented upon in the same setting.

Keywords: Pancreatic lesions, Helical CT, Triple phase, Ultravist.

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CT – Computed tomography

USG - Ultrasonography

EUS – Endosopic ulrasound

MRI – Magnetic resonance imaging

PET – Positron emission tomography

MRCP – Magnetic resonance cholangiopancreatography

ERCP – Endoscopic retrograde cholangiopancreatography

FNAC – Fine needle aspiration cytology

HU ---- Hounsfield unit

MDCT --- Multidetector computed tomography

CECT – Contrast-enhanced computed tomography

INTRODUCTION

Patients presenting with inflammatory and malignant pancreatic diseases are common in daily practice. The diagnosis of different pancreatic diseases has recently become a recurrent problem. Imaging modalities are used to identify and characterize pancreatic lesions. When a neoplasm is suspected, the main task is to judge operability.

CT is the imaging modality of choice for preoperative assessment of a pancreatic lesion. CT has fulfilled the main role of diagnostic imaging of the pancreatic disease, because the comprehensive diagnosis of existence, quality and progress of the tumor can be done non-invasive.[1, 2]

Ultrasound is limited in its usefulness as part of the early investigation of acute pancreatitis or traumatic pancreatic injury , whereas CT has been shown to be useful in helping to predict the outcome of acute pancreatic inflammation and to detect necrosis and fracture of the pancreas.[3, 4]

CT and Magnetic resonance imaging (MRI) are reasonably and similarly accurate in the characterization of cystic pancreatic masses as benign or malignant. MRI did not provide any major advantages compared with CT for the characterization of cystic pancreatic lesions**.** [5,6]

Third generation CT scans provides detailed images of the cancer and its relationship to the surrounding structures such as the major blood vessels around the pancreas. This information is crucial to make the determination whether a tumor in the pancreas is removable. Helical CT enables to recognize small pancreatic arteries, and the evaluation of these should be considered in the staging of pancreatic carcinomas.[7]

Contrast-enhanced CT is the dominant imaging modality used for the diagnosis and staging of pancreatic ductal adenocarcinoma. Computerised Tomography arterial portography (CTAP) did not confer any advantage over Intravenous Computerised Tomography (IVCT) for the preoperative evaluation of liver metastases from pancreatic carcinoma.[8]

Conventional CT is associated with a wide range of sensitivities for pancreatic tumor detection (67% to 97%).In most centers, CT has replaced transabdominal ultrasound for the evaluation of pancreatic cancer. It has been well-established that dynamic CT is useful and is probably the single best modality for the diagnosis and staging of pancreatic adenocarcinoma, although it is not fully sufficient in many cases, especially in those involving small tumors.[9]

AIMS AND OBJECTIVES

To evaluate the pancreatic lesions by computed tomography (CT).

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

- The pancreas was one of the last organs in the abdomen to receive the attention of anatomists, physiologists, physicians, and surgeons. Located in the "straggling mesenchyme" of the retroperitoneum, the pancreas has in the past been called the "hermit" or "hidden" organ of the abdomen. The pancreas was first described in the Talmud and depicted as the "finger of the liver" between 200 BC and 200 AD. Ruphos named this organ the pancreas (Greek -.*pan,* meaning "all"; *kreas,* meaning "flesh") shortly thereafter.

- Wirsung of Padua demonstrated the pancreatic duct in 1642, and Santorini of Venice described the accessory duct in 1724. The papilla of Vater was described by A. Vater in 1720. In 1887, Oddi described the complex musculature of the sphincter bearing his name. The histologic structure of the pancreas was described by Langerhans in 1869. The role of the pancreas in digestion was first suggested by Bernard in 1850, and the connection between diabetes and the pancreas was established in the 1890s. Surgery for pancreatic disease was not popular until the pioneering work of Whipple in the 1930s.

- The pancreas also remained a hidden organ for the radiologist for decades. Indirect signs on plain films and barium studies were usually present only in advanced disease. The 1970s brought endoscopic retrograde cholangiopancreatography (ERCP) and angiography to the fore. CT, ultrasound, and MRI now routinely provide superb visualization of the gland noninvasively.

EMBRYOLOGY

- The pancreatic duct develops from two buds originating from the endodermal lining of the duodenum. One is the dorsal pancreatic bud, which is in the dorsal mesentery and is seen as a diverticulum of the foregut before 28 days. It grows into the dorsal mesentery. The other is the ventral pancreatic bud located close to the bile duct and appears as an invagination at the biliary-duodenal angle between 30 and 35 days. The dorsal and ventral pancreatic buds soon grow into a pair of branching, arborized ductal systems, each with its own central duct. At day 37, the ventral pancreas rotates posterior to the duodenum and comes into contact with the dorsal pancreas. These two anlagen fuse and, together with the duodenum, fuse with the abdominal wall. In the mature organ, the ventral primordium becomes the inferior portion of the head and the uncinate process, and the dorsal pancreas becomes the body and tail. After the fusion, a new duct connects the distal portion of the dorsal pancreatic duct with the shorter duct of the ventral pancreas to form the main duct or the duct of Wirsung. This main pancreatic duct, which is present in approximately 91% of adults, enters the duodenum together with the bile duct at the major papilla. The proximal portion of the dorsal pancreatic duct is pinched off during fusion and usually atrophies and disappears but may persist as the small accessory duct of Santorini, which has a variety of appearances. This accessory duct empties into the duodenum at the minor papilla 2 to 3 cm proximal to the ampulla of Vater. In about 10% of all cases, the duct system fails to fuse and the original double system persists.

- As the duodenum grows and differentiates, the duodenal wall resorbs the distal bile duct up to its junction with the pancreatic duct. Different degrees of

resorption account for variations in the appearance and relationships of the common bile duct and pancreatic duct. If ductal resorption is minimal, a long intramural ampulla is created, and the junction is extramural. The junction becomes intramural with increasing degrees of resorption, which produces a shortened ampulla. Maximal resorption produces separate orifices for the pancreatic duct and common bile duct, which no longer share a common ampulla.

- Beginning in the third month of fetal life, the islets of Langerhans develop as clusters of cells from the terminal ductules. They become intimately associated with the capillary plexus and finally separate from the ductules to become the endocrine portion of the pancreas. Insulin secretion begins at approximately the fifth month. The acini develop from terminal ductal cells. The ductal system and the acini collectively become the exocrine portion of the pancreas. Secretory activity probably becomes established in the pancreas during the second trimester, although this has been disputed.

NORMAL ANATOMY

The pancreas is an unpaired accessory digestive gland that has both exocrine and endocrine functions. It is a slender, soft, lobulated organ that in the adult measures approximately 15 to 25 cm in length, 3 to 5 cm in height, and 1.5 to 3.5 cm in thickness and weighs 70 to 110 g. It has a pale, yellow-tan surface that is finely nodular and firm to palpation.

Topography

- The pancreas lies within the anterior pararenal space. The pancreatic head has a constant relationship with the duodenum, with its right lateral border nestled in the duodenal sweep. The head is the thickest portion of the gland; it gives rise to the uncinate process, which projects similar to a hook, dorsal to the superior mesenteric vein. The pancreatic neck lies immediately anterior to the confluence of the splenic and superior mesenteric veins. The neck narrows behind the pylorus, then widens as it becomes the body. The body arches anteriorly and laterally to cross the spine and may be thinner than the pancreatic head and tail as it does. The body bulges up in the pancreatic tubercle almost to the level of the celiac axis. The tail is not well demarcated from the body as it extends to the splenic hilum.
- The shape, position, and axis of the pancreas are quite variable and are influenced by age, body habitus, previous surgery, and organomegaly. The head usually lies at the level of Ll-2, and the body crosses the spine at L1; the tail is located more superiorly in the region of the splenic hilum. The longi-tudinal axis of the pancreas is about 20 degrees in relationship to the transverse plane. The axis of the pancreas is occasionally transverse, and even less commonly the tail may lie caudal to the head. If the left kidney is congenitally or surgically absent, the tail often lies in a posteromedial position, adjacent to the spine. The pancreas can be shaped like an L, S, or inverted V.
- The gastric body and antrum lie anterior to the body and tail of the pancreas, and the pylorus is located ventral to the pancreatic neck. The duodenum lies along the right lateral border of the pancreatic head; it also passes inferior to

the head, body, and tail. The spleen lies along the lateral and superior aspect of the pancreatic tail. The right kidney and adrenal gland are located posterior to the pancreatic head, and the left kidney and adrenal gland are dorsal and occasionally caudal to the tail. Depending on its size, the left lobe of the liver may lie anterior to the pancreatic body. The gallbladder is positioned ventral to the pancreatic head. The transverse colon lies anterior and generally inferior to the pancreas. The small bowel usually lies inferior to the level of the pancreas but occasionally can lie ventral to the tail.

- Nearly all of the pancreas is retroperitoneal; a nonperitonealized bare area results from the reflection of the posterior parietal peritoneum to form the two leaves of the transverse mesocolon and the posterior-inferior margin of the lesser sac. The transverse mesocolon originates where the hepatic flexure of the colon crosses ventral to the second portion of the duodenum. The bare area begins as a broad strip across the infra-ampullary portion of the descending duodenum and continues across the head, body, and tail of the pancreas. The pancreatic tail, after extending across the left kidney, is actually an intraperitoneal structure incorporated within the leaves of the splenorenal ligament. The root of the small bowel mesentery originates inferior to the pancreatic body and is contiguous with the transverse mesocolon. Therefore, pancreatic processes may affect the stomach and duodenum by direct spread and the small bowel loops and colon via the small bowel mesentery and transverse mesocolon.

Common Bile Duct

- The common bile duct enters the head of the pancreas after it passes posterior to the first part of the duodenum in the hepatoduodenal ligament. It passes inferiorly and dorsally, embedded in the posterior surface of the pancreatic head to join the pancreatic duct of Wirsung. This segment runs a short intramural course before it enters the posteromedial aspect of the duodenum through the major papilla of Vater. The common bile duct is 7 cm long and has an average diameter of 7.4 mm.

Pancreatic Duct

- The pancreatic duct arises from the pancreatic tail and receives 20 to 35 short tributaries entering at right angles to its long axis as it courses toward the head. The duct lies midway between the superior and inferior margins of the pancreas and slightly more dorsally than ventrally. At the level of the major papilla, the main pancreatic duct (duct of Wirsung) courses horizontally to join the caudal surface of the common bile duct forming the ampulla of Vater. The accessory pancreatic duct of Santorini drains the anterior and superior portion of the head of the pancreas either into the duodenum at the minor papilla or into the main pancreatic duct. The minor papilla is often not patent, and the accessory duct may be partially or completely obliterated or have an anomalous connection with the duct of Wirsung.

Arterial Supply

- The arterial blood supply of the pancreas arises from the celiac trunk and the superior mesenteric artery. After originating from the celiac artery, the common hepatic artery courses to the right in proximity to the neck and subsequently the head of the pancreas. At this point, it divides into the proper

hepatic artery, which enters the free edge of the hepatoduodenal ligament, and the gastroduodenal artery, which courses caudally to lie ventral and lateral to the pancreatic head. The gastroduodenal artery gives rise to the anterior and posterior- superior pancreaticoduodenal arteries, which supply the head of the pancreas. These vessels help form the pancreatic arcade when they join the anterior and posterior-inferior mesenteric arteries that arise separately or as a common trunk from the proximal portion of the superior mesenteric artery.

- The splenic artery arises from the celiac artery and loops like a snake above and below the superior margin of the pancreas. It becomes more tortuous with age and occasionally becomes embedded within the pancreatic parenchyma. The pancreatic body and tail are supplied by the dorsal pancreatic artery, which arises from the splenic artery or as a fourth branch of the celiac trunk, as well as by the splenic, hepatic, or superior mesenteric arteries. The pancreatica magna is the largest of the series of superior pancreatic branches of the splenic artery.
- The superior mesenteric artery arises from the anterior surface of the aorta, 1 to 2 cm below the celiac trunk. It courses caudal and dorsal to the neck of the pancreas, passing anterior to the uncinate process, where it serves as a major landmark for cross-sectional imaging. Displacement of the artery to the right of the aorta is a normal variant. The most frequent arterial anomaly of the upper abdomen is partial or complete replacement of either the right hepatic or the proper hepatic artery to the superior mesenteric artery. In these patients, the replaced hepatic artery passes cephalad and ventral to the pancreas, then anterior or posterior to the portal vein to reach the liver.

Venous Drainage

- The venous drainage of the pancreas is constant; the portal system serves as an essential landmark for localizing the pancreas on cross-sectional imaging. In general, the veins of the pancreas parallel the arteries and lie inferior to them. Four pancreaticoduodenal veins form venous arcades that drain the pancreatic head and the duodenum. The inferior pancreaticoduodenal veins drain into the first jejunal branch of the superior mesenteric vein. The inferior pancreaticoduodenal veins are smaller than their superior counterparts; they are not commonly visualized on cross-sectional imaging. The posterior-superior pancreaticoduodenal vein extends cephalad to join the caudal aspect of the portal vein directly. The anterior-superior pancreaticoduodenal vein runs horizontally to drain into either the gastrocolic trunk or the right gastroepiploic vein, both of which | extend to the superior mesenteric vein. Three to 13 small veins from the body and tail empty directly into the splenic vein, which follows a smooth arching course from the spleen to its junction with the superior mesenteric vein. The splenic vein parallels the splenic artery and lies in a groove along the dorsal and superior margin of the pancreas. It is a superb landmark for the posterior aspect of the pancreas, and long segments of this vessel can be seen on CT and ultrasound studies on a single image. Rarely the distal tip of the pancreatic tail can lie posterior to the vein, adjacent to the adrenal gland. In these cases, it may be difficult to differentiate a mass arising in the pancreatic tail from those originating in the left adrenal gland.
- The splenic vein joins the superior mesenteric vein posterior to the neck of the pancreas and is seen as a round or oval dilatation to the right of the midline, posterior to the neck of the pancreas. The superior mesenteric vein courses

anterior to the uncinate process just to the right of the superior mesenteric artery.

- The portal vein is formed behind the pancreatic neck by the union of the splenic and superior mesenteric veins and continues superiorly and laterally toward the porta hepatis as the main extrahepatic portal vein, dorsal to the common bile duct and hepatic artery. In one third of the population, the inferior mesenteric vein enters at the confluence; in another third, it joins the splenic vein close to the junction; and in the remainder, it joins the superior mesenteric vein.

Lymphatics

- The lymph nodes of the pancreas are distributed along the major vascular pathways. The lymphatic channels of the pancreas form a richly branched plexus that empties in multiple directions. The anatomy of the lymphatics suggests that partial removal of the pancreas for cancer may not be sufficient because of the direct connections between different lymphatic chains.
- The suprapancreatic and infrapancreatic lymphatic chains receive branches from the neck, body, and portions of the pancreatic tail. Branches from the posterior surface of the head and neck drain into the pancreaticoduodenal and juxta-aortic nodes. The posterior surface of the pancreatic body drains into the suprapancreatic and infrapancreatic nodes. The pancreatic tail drains into the nodes of the splenic hilum and gastropancreatic fold. Some drainage from the pancreatic head and proximal body enters the nodes in the porta hepatis and may extend inferiorly toward the superior mesenteric, mesocolic, and para-aortic nodal chains. The suprapancreatic nodes are closely related to the

splenic artery and vein; the infrapancreatic chain is adjacent to the leaves of the transverse mesocolon.

Nerve Supply

- It is important to appreciate the nerve supply of the pancreas in planning celiac nerve blocks for control of pain resulting from pancreatic carcinoma or chronic pancreatitis. The pancreas receives sympathetic innervation by way of the splanchnic nerves and parasympathetic innervation from the vagus nerve. The sympathetic nerves carry the pain (visceral afferent) fibers. They pierce the diaphragmatic crura to enter the celiac plexus and celiac ganglion that surround the celiac artery. The superior mesenteric ganglia and plexus surround the superior mesenteric artery. Chemical extirpation of the celiac ganglion interrupts afferent pain fibers from both the sympathetic and the parasympathetic systems and can be accomplished by injection of the chemical agent between the celiac artery and the superior mesenteric artery either antecrurally or retrocrurally.

VARIANTS OF NORMAL ANATOMY

- The lateral aspect of the head and neck of the pancreas can have varieties of shapes and occasionally look prominent. A deep cleft separating two distinct pancreatic moieties may be identified on CT scans in the head and neck of the pancreas in patients with pancreas divisum.

- The pancreas is surrounded by fat that clearly defines its margin. However, owing to the lack of any pancreatic capsule, the pancreatic lobules can be outlined by fat. Such fatty infiltration can be diffuse or focal. Focal fatty

infiltration can mimic a mass on CT, particularly if there is associated lobulation. Therefore, MRI may become necessary to differentiate this benign process from a neoplasm. Fatty infiltration may be associated with focal sparing of pancreatic parenchyma and that should not be mistaken for tumor.

- The position and configuration of the pancreas are quite variable, and these variations may simulate pathologic conditions. For example, the pancreatic head is not fixed in position, although it almost invariably maintains a fixed relationship medial to the second portion 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. Although the splenic vein usually marks the dorsal margin of the body and tail of the pancreas, the tip of the gland may rarely curve dorsal to the splenic vein to simulate adrenal abnormalities. Occasionally, even in normal persons, the pancreatic tail may be anterior-lateral to the left kidney, where it may appear as a pseudomass on excretory urography. In patients with prior left nephrectomy or in those with congenital absence of the left kidney, the pancreatic tail is often displaced into the renal fossa, which may simulate recurrent tumor or a primary retroperitoneal lesion.[10]

- Kreel and colleagues published a set of in vivo and in vitro measurements of pancreatic dimensions and concluded that the normal diameter of the head was up to 3 cm, that of the neck and body up to 2.5 cm, and that of the tail up to 2 cm. However, the size, shape, and position of the normal pancreas are highly variable, and there is usually a gradual tapering from the head to the tail without abrupt alterations in size or contour. There is gradual decrease in the size of the pancreas with advancing age, sometimes becoming very small

beyond the seventh decade. Fatty lobulations are more commonly observed in obese and elderly individuals. Rarely, an accessory spleen can be embedded in the tail of the pancreas, and MRI or a nuclear medicine study may be needed to differentiate this variant from a mass.

- Bifid pancreatic duct is a rare anatomic anomaly in which the main pancreatic duct is bifurcated along its length. It is associated with high incidence of pancreatitis.

RADIOLOGIC TECHNIQUES

- **Plain Radiographs**

  Plain radiographs are obtained on patients with suspected pancreatic disease chiefly to exclude other conditions, such as obstruction or a perforated duodenal ulcer that may simulate pancreatitis. Oblique views are often helpful in patients with chronic pancreatitis to detect calcifications that may be obscured by the spine on the anteroposterior view.

- **Contrast Studies - Barium**

  Before the advent of cross-sectional imaging, angiography, ERCP, and barium studies were the major means of evaluating the pancreas. The posterior gastric wall, the distal duodenum, and the duodenojejunal junction can be abnormal with lesions of the pancreatic tail and body; the greater curvature of the gastric antrum and medial aspect of the descending duodenum can provide clues for lesions arising in the pancreatic head and neck. Barium enema examination may reveal abnormalities of the colon caused by disease spread via the trans-

verse mesocolon or at the splenic flexure caused by disease carried by the phrenicocolic ligament.

- **Ultrasound**

  Sonography is a superb means of noninvasively evaluating the pancreas, particularly in thin patients. It is fast, safe, and inexpensive; it can be done portably; and it requires little preparation or cooperation of the patient and no administration of contrast medium. Ultrasound examination of the pancreas is best performed on the fasting patient to reduce the amount of gas and food in overlying bowel. Real-time equipment with the highest frequency transducer (5-8 MHz) possible should be used.

- **Intraoperative Ultrasound**

  Intraoperative sonography is a time-consuming but accurate means of localizing small islet cell tumors of the pancreas. It can also be used to guide open biopsy and aspiration. With the increased use of laparoscopic techniques, probes for use during this procedure have also been introduced.

- **Endoscopic Ultrasound**

  Ultrasound transducers have been modified so that they can be incorporated into the tip of flexible endoscopes. These transducers have the advantage of higher frequency and spatial resolution, which may be of use in diagnosing small pancreatic tumors, in demonstrating subtle pancreatitis, and in evaluating islet cell tumors preoperatively. This higher resolution comes at the expense of limited sound penetration and a limited field of view.

- **Computed Tomography**

  Computed tomography is the best single technique for the noninvasive imaging of the pancreas. It is unaffected by bowel gas or large body habitus, is widely available, and is relatively easily performed. CT has greatly diminished the need for diagnostic endoscopic retrograde cholangiopancreatography (ERCP) and angiography. Multidetector row CT techniques have improved CT examination of the pancreas, allowing thinner contiguous images, overlapping images without increased radiation exposure, and freedom from respiratory misregistration. The rapidity of scanning allows several acquisitions to be made through the pancreas during different phases of a single contrast bolus.

  Size, Shape, and Density on Computed Tomography

  The morphologic appearance of the pancreas on CT depends on the amount of fat within the intralobular septa that separate the acinar lobules of the gland. In younger patients, the contour of the gland is smooth; the parenchyma is homogeneous, with an attenuation similar to that of spleen and muscle but less than that of liver on noncontrast scans. With age and progressive fatty deposition, the pancreas becomes lobulated, irregular, and inhomogeneous.

  Although the size of the pancreatic head, neck, body, and tail generally correspond to normal measurements found sonographically, absolute numbers should not be used alone in diagnosing pancreatic enlargement. It is important to observe symmetry within the gland, with the head (maximal normal diameter of 3 cm) being slightly larger in anterior- posterior dimension than the body (2.2 cm) and tail (2.8 cm). The pancreatic tail can be bulbous in some normal patients and measure larger than the head. The thickness of the head of

the pancreas should be less than the transverse diameter of the adjacent vertebral body; the body and tail should be less than two thirds of the size. The pancreatic body may be thinner where it crosses the spine. The lateral contour of the pancreatic head may have discrete lobulations lateral to the gastroduodenal or anterior superior pancreaticoduodenal artery in approximately one third of normal examinations. The uncinate process has a triangular appearance on cross section as it projects behind the mesenteric vessels. The pancreas moves an average of 3.2 cm craniocaudally between phases of respiration on CT scans. In patients with a surgically or congenitally absent left kidney, the pancreatic tail lies dorsomedial, adjacent to the spine, and occupies the empty renal fossa along with bowel and spleen. The entire pancreas may rarely lie to the left of the aorta.

The pancreatic vascular anatomy is well demonstrated by MDCT. The splenic vein makes an excellent landmark for the body and tail of the pancreas. The anterior and posterior-superior pancreaticoduodenal veins are seen on 98% and 88% of scans, and the gastrocolic trunk may be visualized on 89% of scans. Frequently visualized are the gastroduodenal, anterior and posterior-superior pancreaticoduodenal, and right gastroepiploic arteries. Occasionally visualized are the dorsal pancreatic, pancreatica magna, and anterior and posterior inferior pancreaticoduodenal arteries.

Pancreatic Duct on Computed Tomography

The normal pancreatic duct appears as a thin, tubular region of low attenuation coursing in the center of the pancreas on CT scans. The duct can be seen at least partially in 70% of normal patients; usually, short segments are visualized on a limited number of scans. The duct is most frequently seen in

the region of the pancreatic body as it arches over the spine and mesenteric vessels or in the region of the head.

The normal pancreatic duct should be no wider than 2 to 3 mm, but it can occasionally be larger in older individuals. The normal fat plane between the splenic vein and pancreatic parenchyma should not be mistaken for the pancreatic duct.

When CT is performed within 30 minutes of ERCP, valuable information about duct morphology and ductal obstruction can be learned by use of thin collimation. It is useful in demonstrating communication between the gut and the pancreas and in patients with prior pancreatectomy who are being considered for additional surgery. This technique should not be used routinely, however, because it adds little additional useful information in patients with a normal ERCP.

Common Bile Duct on Computed Tomography

The internal diameter of the distal common bile duct is less than 6 to 7 mm. In older patients or those with a history of previous biliary tract disease or surgery, the duct may be as large as 10 mm in the absence of obstruction. Thus, the diagnostic significance of ducts 7 to 10 mm in size is often difficult to assess. On contrast scans, the vasa vasorum of the duct may enhance.

- **Magnetic Resonance Imaging**

The pancreas had been one of the most difficult organs to image reliably on MR imaging until the development of techniques such as breath-hold imaging, chemically selective fat saturation, and gadolinium enhancement. One study compared helical CT to MR imaging and found MR imaging

superior in identification of small pancreatic adenocarcinomas. The inability to demonstrate small calcifications remains a relative drawback.

- **Magnetic Resonance Cholangiopancreatography**

An important new technique to examine the pancreatic and biliary tree is MR cholangiopancreatography. The normal and abnormal pancreatic duct can be visualized without the invasiveness of ERCP. Several variations of the technique have emerged. All have in common a heavily T2-weighted pulse sequence in which the fluid-filled ducts stand out from the surrounding low signal intensity tissues. Many of these are breath-hold sequences that can easily be added to routine pancreatic imaging with little increase in overall examination time. Postprocessing using a maximum-intensity profile technique allows visualization from multiple perspectives, giving an appearance similar to ERCP. Secretin administered before the dynamic acquisition of images has been used to improve visualization of the pancreatic duct, diagnose papillary stenosis or dysfunction, and evaluate reduced pancreatic exocrine reserve. MR cholangiopancreatography may be particularly valuable in postoperative patients in whom ERCP is technically impossible.

- **Endoscopic Retrograde Cholangiopancreatography**

ERCP affords opacification of both the pancreatic and the bile ducts. In addition, the endoscopist can inspect and perform a biopsy of suspicious periampullary lesions, perform a sphincterotomy with or without stone extraction, and place internal biliary stents. The indications for ERCP include the following: to determine the cause of idiopathic pancreatitis (i.e., pancreas

divisum); to provide a road map for the surgeon in patients with dilated ducts and chronic pancreatitis before the Puestow procedure; to identify communication of the pancreatic duct with pseudocysts and fistulas; to detect small intraductal pancreatic neoplasms that distort ductal anatomy but do not yet cause a mass effect that can be detected by CT and ultrasound; and to identify the cause of extrahepatic biliary obstruction (e.g., stone, tumor, benign stricture, or inflammation).

- **PET/CT**

Minimal physiological uptake is identified on PET/CT. In cases of pancreatitis, the pattern of increased FDG activity may be diffuse or focal in nature and may be difficult to distinguish from malignancy. Studies have demonstrated the relatively high sensitivity and specificity of PET in distinguishing benign and malignant lesions in the pancreas – 92% and 85% in comparison to 65% and 62% for CT, respectively. PET is also less dependent on lesion size for diagnostic accuracy. PET/CT is a critical preoperative staging modality for resection of pancreatic cancer, as it significantly improves patient selection and is ultimately cost-effective. FDG-PET is not clearly superior to CT for N staging, likely due to the proximity of lymph nodes to the primary mass, which may become obscured. However, in the case of anatomically small lymph nodes(less than 1cm), with increased metabolic activity, PET/CT would have an advantage. PET/CT is more accurate than CT alone in the detection of distant metastatic disease.

The primary challenge faced by PET/CT in the imaging of pancreatic cancer pertains to altered glucose metabolism created by glucose intolerance and

diabetes seen in these patients. This setting may create false-negative findings in patients who are hyperglycemic or have inadequately controlled blood glucose levels. False-negatives may also result when the tumor is less than 1cm, such as in small ampullary carcinomas. False-positives are mainly the result of inflammation secondary to pancreatitis.

PET/CT is an important imaging adjunct to CT and may provide earlier detection of metastatic or recurrent disease, assisting in treatment planning as well as potentially improving outcome.

CONGENITAL ANOMALIES OF THE PANCREAS

Pancreas Divisum

- In this anomaly, the pancreas is divided in two separate parts as a result of an absent or incomplete fusion of the ventral and dorsal anlagen. As a consequence, the pancreatic head and uncinate process are drained by the duct of Wirsung through the major papilla; the body and tail are drained by the duct of Santorini through the minor papilla.

- Most cases of pancreas divisum are asymptomatic. This anomaly may contribute to recurrent episodes of idiopathic pancreatitis in younger patients with no risk factors. The age at presentation varies widely but is most commonly between ages 30 and 50 years. Cases of pancreatitis, multiple neuroendocrine tumors of the pancreas and intestinal malrotation have been reported in association with pancreas divisum.

- Endoscopic retrograde cholangiopancreatography (ERCP) is often considered the effective modality for confirming a diagnosis of pancreas divisum.

Diagnosis can also be made by secretin stimulation test, linear-array EUS, sonographic secretin test, MDCT with high-resolution oblique coronal image reconstruction, Magnetic resonance cholangiopancreatography (MRCP) and Secretin-stimulated MRCP.

Annular and Semiannular Pancreas

- Annular pancreas is a rare congenital anomaly occurring in 1 of every 12,000 to 15,000 live births. In this anomaly, the annulus is often a flat band of pancreatic tissue completely encircling the second portion of the duodenum.
- Occasionally, the duodenum may not be completely encircled with pancreatic tissue. This anomaly is called semiannular pancreas.
- Annular pancreas may be quiescent and not cause symptoms until adulthood. Its most commonly associated with intestinal malrotation. It is recommended to evaluate the pancreas for malignancy in cases of annular pancreas presenting with obstructive jaundice.
- Pediatric patients with annular pancreas often have diagnostic findings on plain radiographs with the "double-bubble" sign; the proximal bubble is caused by gastric distention and the distal bubble by a dilated duodenal bulb.
- Diagnosis of annular pancreas is most commonly suggested by ERCP, which shows typical features in about 85% of cases. CT and sonographic examination reveals nonspecific enlargement of the pancreatic head. Endoscopic ultrasound is more accurate in making the diagnosis. Because CT may only show an enlarged pancreatic head, fat-suppressed T1-weighted MR image can clearly discriminate the pancreas from the duodenum. Celiac angiography

may demonstrate an anomalous branch from the posterior pancreaticoduodenal artery that courses in a right and inferior direction to supply the annular moiety.

Ectopic Pancreatic Tissue

- Heterotopic pancreas is the result of heteroplastic differentiation of parts of embryonic endoderm that do not normally produce pancreatic tissue. Although the embryonic origins of the pancreas and liver are close anatomically, ectopic pancreas is much more common than heterotopic liver. These ectopic rests of pancreatic tissue most commonly occur in the gastric antrum (25.5%) or proximal portion of the duodenum (27.7%). Less frequent sites include jejunum (15.9%), ileum, Meckel's diverticulum, colon, appendix, mesentery, omentum, liver, gallbladder, spleen, bile ducts, esophagus, ediastinal cyst, fallopian tube, and broncho- esophageal fistula. When present in the walls of the duodenum and stomach, heterotopic pancreas is usually composed of normal pancreatic tissue, including islet cells, and a small pancreatic duct. Islet cells are usually absent at other sites. The ectopic pancreatic tissue usually lies submucosally (73%), although it can be located in the muscularis mucosae or on the serosal surface of the gut.

- Ectopic pancreatic tissue is functional and subject to the same inflammatory and neoplastic disorders that afflict the normal pancreas.

- Ectopic pancreas in barium study characteristically appears as a broad-based, smooth, extramucosal or intramural lesion either along the greater curvature of the gastric antrum or in the proximal duodenum. A small collection of barium appearing as a central niche or umbilication is diagnostic and present in 45%

of cases. This represents the orifice of the rudimentary duct into which the ectopic pancreas empties. This pit may be as large as 5 mm,in diameter and 10 mm in length. If this feature is absent, the lesion cannot be differentiated from other submucosal tumors.

Agenesis, Hypoplasia, and Hyperplasia of the Pancreas

- Agenesis of the pancreas is rare and typically incompatible with life. A casual relationship with intrauterine growth retardation has been noted and is presumed to be due to the lack of fetal insulin necessary for development.

- Partial pancreatic agenesis and hypoplasia are also rare; however, cases of agenesis of the dorsal pancreas have been reported along with malrotation. Abnormal rotation of the intestine may interrupt the normal rotation of the pancreatic primordium, with malpositioning of the pancreatic buds resulting in abnormal morphology of the uncinate process.

Ductal Abnormalities

- The single main pancreatic duct normally connects with the accessory duct of Santorini, and then they open in conjunction with the common bile duct at the ampulla of Vater. This arrangement is seen in 60% to 70% of normal individuals.

- The common variations of pancreatic ductal anatomy include (1) junction of the ventral duct and common bile duct at the ampulla with complete regression of the dorsal duct(40%-50%); (2) junction of the ventral and common bile ducts at the ampulla but with persistence of the dorsal duct(35%); (3) persistence of both the dorsal and the ventral ducts without communication, or pancreas divisum (5%-10%); (4) common channel with a retroduct entering

the common duct from 5 to 15 mm from the ampulla (5%-10%) and (5) separate entrance of the ventral duct into the duodenum with variable persistence of the dorsal duct (5%). Variations in ductal configurations include a sigmoid configuration of the duct and descending course of the duct. One may rarely encounter with a loop at the point of the embryologic fusion of the ducts.

PANCREATITIS

- Pancreatitis is one of the most complex and clinically challenging of all abdominal disorders. It remains a major diagnostic challenge because its clinical manifestations are as protean as its causes. Indeed, only one in three severe cases of acute pancreatitis is recognized to be severe at initial presentation, and 42% of fatal cases of acute pancreatitis do not have a correct diagnosis before autopsy.

CLASSIFICATION OF PANCREATITIS

- Pancreatitis is classified according to clinical, morphologic, and histologic criteria on the basis of the Marseille and Cambridge symposia and the 1992 International Symposium in Atlanta, Georgia.[11-12]

ACUTE PANCREATITIS

Etiology

- Although the causes of pancreatitis are diverse, alcoholism and biliary tract disease (gallstones) account for approximately 90% of cases in the United States. The relative frequency of these causes varies with the country and population of patients examined. Alcoholic pancreatitis is more common in urban and Veterans

Administration hospitals, whereas gallstone pancreatitis predominates in suburban and rural hospitals. The incidence of acute pancreatitis is 0.005% to 0.01% in the general population. Approximately 10% to 30% of patients with acute pancreatitis will never have the cause established and are given the diagnosis of "idiopathic"pancreatitis. Biliary sludge and microlithiasis are probably responsible for the development of pancreatitis in the majority of these cases.

Classification of Pancreatitis

Acute Pancreatitis

- **Diagnostic Criteria**
  - **An elevation of plasma levels of pancreatic enzymes >10 SDs above normal laboratory values**
  - **Evidence of acute pancreatitis from laparotomy, imaging, or autopsy**
- **Clinical Classification**
  - **Etiology when known(e.g., gallstones, alcohol abuse)**
  - **Degree of severity:**

    (a) mild, with no multisystem failure and an uncomplicated recovery

    (b) severe (multisystem failure, pancreatic necrosis, and/or development of a complication, e.g., pseudocyst)

Chronic Pancreatitis

- **Diagnostic Criteria**
  - **Permanently impaired exocrine pancreatic function tests >2 SDs below normal levels**
  - **Permanent morphologic change in the gland**
- **Clinical Classification**
  - **Etiology, if known**
  - **Absence or presence of pain**
  - **Complications: cysts, portal hypertension, diabetes mellitus**

Pathophysiology

- The precise mechanisms of pathogenesis of acute pancreatitis are not completely understood. Alcohol increases the risk for pancreatitis through multiple mechanisms. More than 95% of heavy alcohol users never develop significant pancreatitis, however, which suggests that this is a complex syndrome, with other risk factors. Alcoholic pancreatitis has been explained by a number of mechanisms: necrosis-fibrosis sequence, duct obstruction, leakage of enzymes from the pancreatic duct, abnormal synthesis of digestive enzymes, toxic metabolites, altered pancreatic blood flow, mitochondrial damage, and activation of pancreatic stellate cells, which produce collagen in the fibrotic pancreas. The toxic effect and chemical alterations of the exocrine secretions produced by alcohol lead to protein precipitates that obstruct the pancreatic ducts. In addition, alcohol can lead to duodenitis, edema, and spasm of the papilla of Vater, further contributing to ductal obstruction. Alcohol also indirectly stimulates pancreatic secretion by inducing an increase in gastrin and secretin levels and decreases the

level of zymogen inhibitor, resulting in the premature activation of trypsin. In acute pancreatitis, these activated pancreatic enzymes are extravasated, causing pancreatic auto-digestion and necrosis.

- In cholelithiasis-induced pancreatitis, there is obstruction of the common biliopancreatic channel by a stone (Opie stone), with resultant reflux of bile into the pancreatic duct and activation of pancreatic enzymes. Gallstones are found in the stool of most of these patients. This is the rationale for emergency chole-cystectomy or endoscopic retrograde cholangiopancreatography (ERCP)-directed sphincterotomies in the treatment of gallstone pancreatitis. It is interesting to note that reflux of contrast material into the pancreatic duct during intraoperative cholangiography is much more common in patients with gallstone pancreatitis than in patients undergoing cholecystectomy who do not have pancreatitis.

- Pancreas divisum induce pancreatitis by functional obstruction at the accessory papilla of Santorini.

- A variety of other causes, such as drugs, calcium, endotoxins, hyperlipidemia, viral infections, ischemia, anoxia, and trauma, can also activate proteolytic enzymes in the pancreas that lead to autodigestion and stimulate other enzymes such as bradykinin and vasoactive substances that cause vasodilatation, increased vascular permeability, and edema.

- Leukocytes attracted by the pancreatic injury release inflammatory mediators called cytokines, which have an important role in disease progression and multisystem complications of acute pancreatitis. Biologically active compounds such as phospholipase A2, tumor necrosis factor, polymorphonuclear cell elastaste, complement factor, interleukins, and leukotrienes are released into the

systemic circulation, stimulating the production of other mediators and leading to distant organ failure. Some of these mediators, such as tumor necrosis factor, are also toxic to acinar cells and may contribute to pancreatic injury and necrosis. These inflammatory products occur early in the course of the disease and can be used as indicators of severity and prognosis in acute pancreatitis.

- Pancreatitis has been shown to have an increased prevalence in AIDS patients, explained either by the effects of medication (pentamidine and 2',3'-dideoxyinosine [ddl]) or by secondary opportunistic infections.

Major pathologic categories of acute pancreatitis –

Acute interstitial pancreatitis is the mildest form of pancreatitis and is also called ***edematous pancreatitis***. It is characterized by absent or minimal pancreatic and systemic dysfunction and a rapid response to medical therapy without complications. Only pancreatic edema and mild cellular infiltrate are present. The gland may enlarge to three times its normal size and become firm. There may be a few small, scattered foci of necrosis and saponification in the peripancreatic fatty tissue.

Necrotizing pancreatitis is a far more severe form of pancreatitis in which varying degrees of systemic and distal organ failure and potentially lethal complications can occur. Extensive fat necrosis, hemorrhage, and necrotic liquefaction of the pancreatic parenchyma and adjacent peripancreatic tissues and fascial planes are seen.

Definitions

- **Phlegmon:** Descriptive term, formerly used, implying the presence of a heterogeneous mass-like enlargement of the pancreas and retroperitoneal tissues. It does not correlate with the presence of infection or necrosis, and hence, its use should be discouraged.

- **Fluid collection:** A localized collection of pancreatic fluid, homogeneous or heterogeneous in attenuation, which is seen in 30% to 50% of patients with acute pancreatitis and may be located in the pancreas, lesser sac, anterior pararenal space, or retroperitoneal space or as distant as the neck, mediastinum, pleura, pericardium, or groin. Nearly two thirds of these collections resolve spontaneously; others become infected (abscess) or may persist as pseudocysts.

- **Pseudocysts:** Localized collections of pancreatic fluid confined by a capsule of fibrous or granulation tissue. Pseudocysts develop 4 to 6 weeks after an episode of acute pancreatitis or are associated with stigmata of chronic pancreatitis. They lack a true epithelial lining, a feature that differentiates them from a cyst or cystic neoplasm. Pseudocysts smaller than 4 cm often resolve spontaneously. Fluid collections greater than 7 cm in diameter are likely to require intervention, particularly in patients with alcoholic pancreatitis. Persistent pseudocysts often communicate with the pancreatic duct and have the potential to rupture, bleed, become infected, or cause a pseudoaneurysm.

- **Abscess:** Pancreatic abscess develops from infected, extravasated pancreatic secretions. It usually occurs 3 or more weeks after the initial attack or may develop as a secondary infection of a pseudocyst. The term should be used to

define an encapsulated collection of pus in proximity to the pancreas without associated pancreatic necrosis.

- **Infected pancreatic necrosis:** Infected necrotic pancreatic tissue is partially or totally liquefied. It should be differentiated from an abscess because it leads to a 48% mortality rate, compared with 25% from abscess, and is often not amenable to percutaneous drainage.

- **Hemorrhagic pancreatitis:** This term has been used synonymously with "pancreatic necrosis". It should be restricted to the intraoperative and postmortem appearance of the gland. Massive hemorrhage is a rare manifestation of acute pancreatitis, usually occurring as a late complication from a ruptured pseudoaneurysm. Gastrointestinal hemorrhage can occur if a pseudoaneurysm ruptures into the pancreatic duct directly or by way of a pseudocyst. Pseudocysts also rupture and erode into the gastrointestinal tract.

- **Pseudoaneurysm:** A focal area of dilatation of a splanchnic artery that may occur as a result of inflammatory weakening of the arterial wall by enzymes liberated in acute pancreatitis. Pseudoaneurysms usually are found in the splenic, gastroduodenal, and pancreaticoduodenal arteries and can be free standing or associated with a pseudocyst. Intermittent or life-threatening hemorrhage into a pseudocyst, retroperitoneum, or peritoneal cavity may occur.

- **Pancreatic ascites**: It develops when there is outpouring of pancreatic fluid from a disrupted pancreatic duct into the peritoneal cavity by way of a fistula. Pancreatic ascites usually occurs as a complication of chronic pancreatitis but may be noted after any episode of acute pancreatitis.

Clinical Findings

- The clinical diagnosis of pancreatitis is often difficult. Patients' symptoms range from mild abdominal pain, nausea, vomiting, fever, tachycardia, and abdominal distention to severe abdominal pain and shock. These findings are nonspecific, and the differential diagnosis usually includes acute cholecystitis, bowel obstruction or infarction, perforated viscus, renal colic, duodenal diverticulitis, aortic dissection, appendicitis, and ruptured abdominal aortic aneurysm. In very severe cases, flank ecchymosis (Grey-Turner's sign) or periumbilical hematoma (Cullen's sign) may be present.

- Consequently, a battery of laboratory tests has been developed to diagnose and grade pancreatitis. These tests include evaluation of serum amylase, lipase, serum/urinary amylase ratio, pisoamylase, immunoreactive trypsin, chymotrypsin, elastase, serum cyclic adenosine monophosphate, C-reactive protein, urinary trypsinogen-2, and methemalbumin. Serum amylase and lipase levels are the most commonly used measures to diagnose pancreatitis. Unfortunately, these values are elevated in only 80% to 90% of patients with acute pancreatitis. Amylase is rapidly secreted by the kidneys and may return to normal levels during the first 48 to 72 hours. Pancreatitis caused by gallstones, microlithiasis, or drugs is often associated with a greater elevation in amylase than in lipase. The amylase level relative to lipase tends to be lower in alcoholic pancreatitis, hypertriglyceridemia- induced pancreatitis, neoplasia, and chronic pancreatitis. There is no correlation between the levels of serum amylase and the severity of acute pancreatitis; patients with mild forms of disease may exhibit levels of over 1000 IU, whereas patients with severe necrotizing pancreatitis may

have normal or low amylase levels. Furthermore, hyperamylasemia may be seen in other acute abdominal conditions such as bowel obstruction, bowel infarction, gangrenous cholecystitis, and perforated ulcer. An elevated lipase is more specific for pancreatitis but the level does not predict the etiology.

- The clinical course of acute pancreatitis varies from mild and self-limited disease to shock, overwhelming sepsis, and death. Approximately 50% of patients have mild interstitial pancreatitis and require a limited hospital stay with minor supportive therapy, 40% have a stormier course but ultimately survive, and 10% die of shock associated with early respiratory and renal failure or later sepsis. In an attempt to predict which course the patient may take, Ranson has described a number of criteria linked to a poor prognosis. In a study of more than 450 patients, there was good correlation between the number of risk factors and the severity of acute pancreatitis. Other scoring tests, including the Acute Physiology and Chronic Health Evaluation II (APACHE II) grading system, are similarly based on measuring physiologic and laboratory parameters routinely available in most hospitals. It should be stressed, however, that these scoring tests reflect only systemic alterations (renal, pulmonary, cardiovascular) and not local disease. The scoring tests do not have any diagnostic specificity, the physiologic alterations being seen in a variety of other conditions.

Radiologic Findings

- Radiologic imaging of patients with suspected pancreatitis has four major objectives:

  (1) To exclude other abdominal disorders that can mimic acute pancreatitis,

  (2) To confirm the clinical diagnosis of acute pancreatitis

(3) To evaluate the extent and nature of pancreatic injury and peripancreatic inflammation in an attempt to stage the severity of disease

(4) To determine the etiology of acute pancreatitis, if possible.

Abdominal Plain Radiographs

Abnormalities in the abdominal gas pattern are the most frequent findings on abdominal plain radiographs and range from a gasless abdomen to an ileus pattern. The most common findings are a small bowel ileus (42%), where an adynamic sentinel loop is seen, and the "colon cutoff" sign (56%), related to spasm of the splenic flexure with distal paucity of gas caused by spread of the pancreatic inflammation into the phrenocolic ligament. Although originally described in abdominal plain films, the "colon cutoff"sign can also be seen on CT.

Chest Radiographs

Approximately one third of patients with acute pancreatitis demonstrate pulmonary and chest abnormalities: elevated diaphragms, pleural and pericardial effusions, basal atelectasis, pulmonary infiltrates, and the adult respiratory distress syndrome. Abnormal chest radiographs can be useful to determine the severity of acute pancreatitis.

Barium Studies

- Meyers and Evans emphasized the importance of the ligaments and mesenteries of the retroperitoneal space in the spread of pancreatitis.

Extravasated pancreatic enzymes commonly enter the lesser sac and spread to

the retroperitoneal space of the transverse mesocolon, phrenicocolic ligament, and small bowel mesentery, causing serosal inflammation and irritation of the gastrointestinal tract. Thickening and spiculation of mucosal folds in the stomach and duodenum can be seen on CT and barium studies. Varying degrees of atony associated with spastic segments of the duodenum, jejunum, and transverse colon can also be present.

Cholangiography

- Occasionally, the enlarged edematous pancreas causes compression and obstruction of the distal common bile duct, resulting in jaundice. The narrowing is typically smooth and symmetric.
- Some gastroenterologists advocate endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy to alter the course of pancreatitis when it appears that multiple stones are passing through the common bile duct. The pancreatic duct is usually normal but can be compressed. ERCP can also demonstrate other causes of pancreatitis, such as pancreas divisum, choledochocele, choledochal cyst, perivaterian duodenal diverticulum, pancreatic or bile duct carcinoma, and ampullary carcinoma.
- Magnetic resonance cholangiopancreatography (MRCP) is often performed to determine the cause of pancreatitis, because it is accurate and noninvasive and avoids the complications associated with ERCP.

Angiography

- Angiography is not performed in patients with acute pancreatitis unless the presence of a pseudoaneurysm that can be treated with transcatheter embolization is suspected. It can also be helpful to elucidate vascular causes of pancreatitis such as vasculitis, polyarteritis nodosum, postaortic aneurysm resection, after

transplantation, Ortner's syndrome, systemic lupus erythematosus, low-flow states, shock, and diabetes. In edematous pancreatitis, the vessels are stretched and displaced with increased parenchymal staining. The vessels may be beaded with an irregular caliber or show thrombosis or pseudoaneurysm formation.

Ultrasonography

- Abnormal findings are seen at sonography in 33% to 90% of patients with acute pancreatitis. The classic sonographic appearance of pancreatitis is a diffusely enlarged, hypoechoic pancreas. Less commonly, focal enlargement is present.
- The echogenicity of the pancreas in acute pancreatitis is extremely variable and depends on a number of factors:

  (1) Timing of the sonographic study, with maximal decrease in echogenicity occurring 2 to 5 days after the initial episode of acute abdominal pain

  (2) Amount of intrapancreatic fat (with age, the pancreas is replaced by fat and becomes more echogenic)

  (3) Presence of hemorrhage

  (4) Presence of underlying chronic pancreatitis with calcification

  (5) Degree of extrapancreatic spread of acute pancreatitis.
- The pancreatic duct may dilate, particularly if the inflammation is confined to the pancreatic head. Focal intrapancreatic masses may be due to an acute fluid collection, hemorrhage, or ill-defined and hypoechoic pancreatic enlargement that may sonographically simulate carcinoma. Cystic masses should be scrutinized with Doppler ultrasound studies to exclude pancreatic pseudoaneurysms. Lesser sac fluid collections are often seen and may produce a "butterfly" appearance.

- In acute pancreatitis, bowel gas and other factors will limit visualization of the gland in one fourth to one third of patients. This is one of four major limitations of ultrasound in this clinical setting. The second is the inability of ultrasound to completely define the complex extrapancreatic spread of infection along fascial planes and within the peripancreatic compartments. It is particularly limited in visualizing spread into the transverse mesocolon. Third, ultrasound cannot specifically reveal areas of pancreatic necrosis in patients with severe pancreatitis, information that provides important prognostic information. Finally, only CT or angiography can diagnose many gastrointestinal and vascular complications of acute pancreatitis.
- What, then, is the role of sonography in acute pancreatitis? It is a good screening test in patients with suspected biliary pancreatitis and a mild clinical course. It is also useful in thin patients with mild edematous pancreatitis that promptly responds to conservative therapy.
- CT is preferred for patients with clinically severe pancreatitis, those whose disease fails to respond to conservative therapy, acutely ill patients who pose a diagnostic dilemma, and patients with complications such as infected pseudocyst, hemorrhage, pseudoaneurysm formation, and pancreatic necrosis.
- The use of tissue harmonic imaging improves image quality, delineation of the pancreatic tail, lesion conspicuity and fluid-solid differentiation relative to conventional B-mode sonography.[13] In a recent study, tissue harmonic sonography was able to detect abnormalities in 91% of patients with acute pancreatitis. The authors found that the two most useful sonographic findings were extrapancreatic inflammation and parenchymal inhomogeneity. It was difficult, however, to distinguish between fluid collections and extrapancreatic

inflammation. Even with tissue harmonics, sonography does not depict necrosis or other complications as well as CT or MRI.

Computed Tomography and Magnetic Resonance Imaging

- Computed tomography is the premier imaging test in the diagnosis and management of patients with acute pancreatitis. It visualizes the gland, bowel, retroperitoneum, abdominal ligaments, mesenteries, and omenta in their entirety. In addition, it may help determine the etiology, stage severity, and detect complications of pancreatitis and can be used to guide interventional procedures such as fine needle aspiration biopsy or catheter placement. However, the radiation dose delivered by CT may be a significant factor for young patients with pancreatitis who will require multiple examinations. MRI is an option for these patients, but it is more costly than CT. Other advantages of MRI include its high soft tissue contrast resolution and ability to evaluate the common bile duct, pancreatic duct, and parenchyma in a single examination. In addition, MRI does not require iodinated contrast and thus is preferred in patients with renal insufficiency or severe pancreatitis with an increased risk for renal disease. MRI, however, is limited in the more acute setting when the patient is very ill, because patient cooperation and breath-holding may be compromised during this longer examination.

Diagnosis

- The imaging findings in acute pancreatitis are similar, regardless of etiology, with the exception of traumatic pancreatitis, in which pancreatic lacerations cause high-density (50-90 Hounsfield units [HU]) hematomas. MRI can be helpful in determining the etiology of pancreatitis, including

choledocholithiasis, pancreas divisum, and underlying tumors. In the acute setting, however, the etiology may be difficulty to determine due to the significant inflammation, which may obscure stones, the pancreaticobiliary ducts, or underlying masses.

- In mild forms of pancreatitis, CT and MRI may be normal or may show a slight to moderate increase in gland size. Mild inflammation may surround an otherwise normal-appearing gland. Alternatively, the pancreas maybe diffusely enlarged, with a shaggy and irregular contour, and slightly hypoenhancing heterogeneous parenchyma. On MRI, there may be decreased pancreatic signal intensity on Tl-weighted fat-suppressed sequences before and following gadolinium administration, depending on the degree of inflammation, edema, and necrosis.
- In more advanced cases, extravasation of pancreatic fluid leads to the formation of intrapancreatic and extrapancreatic fluid collections. Because the pancreas does not have a well-developed fibrous capsule, pancreatic secretions commonly extravasate in the retroperitoneum, most often in the anterior pararenal space, retroperitoneal space, and interfascial spaces. CT superbly depicts peripancreatic inflammation, fluid, and, occasionally, mild thickening of the adjacent fascial planes. Peripancreatic inflammatory stranding is also well seen on MRI.
- A more unusual **segmental form of acute pancreatitis** occurs in as many as 18% of patients. The CT findings are similar to those in diffuse pancreatitis; however, only part of the gland is involved either exclusively or predominantly. The segment most often involved is the pancreatic head. This form of pancreatitis is usually mild and is most

often associated with stone disease. However, patients with segmental pancreatitis should be carefully evaluated to exclude an adenocarcinoma simulating pancreatitis or a mass causing pancreatitis. It may be appropriate to further investigate these patients with endoscopic ultrasound, ERCP, or biopsy or to perform short-term follow-up CT or MRI, especially if they are older or do not have risk factors for pancreatitis.

- In severe forms of **necrotizing pancreatitis**, the gland may become enlarged, and it is commonly enveloped by high-attenuation heterogeneous fluid collections. Because of high-attenuation exudates, the presence of pancreatic necrosis cannot be assessed on CT unless the gland is imaged during the late arterial-early portal venous phase of a rapid bolus intravenous injection of contrast medium. On MRI, peripancreatic high signal intensity may be seen on Tl-weighted fat-suppressed sequences related to fat necrosis and hemorrhage. This finding is a poor prognostic sign.[14] Patchy areas of absence of enhancement, fragmentation, and liquefaction necrosis can be detected on CT and MRI. Poorly defined peripancreatic exudates obliterate the peripancreatic fat, envelop the pancreas, dissect the interfascial planes, and penetrate through fascial and peritoneal boundaries and ligaments. These collections most often occur in the anterior pararenal space around the body and tail of the pancreas and within the lesser peritoneal sac. When fluid collections are massive, they tend to extend inferiorly along the pararenal spaces in the left flank or bilaterally. Exudates can invade the small bowel mesentery, the transverse mesocolon, and the posterior

pararenal spaces. Fluid can penetrate into solid organs such as the spleen or the liver and even the mediastinum. Splenic involvement is most common due to the close anatomic relationship of the pancreatic tail and splenic hilum. Subcapsular or intrasplenic fluid collections or pseudocysts, infarcts, and splenic hemorrhage may be seen with pancreatitis.

- In about 7% to 12% of patients with acute pancreatitis, CT and MRI reveal a small amount of free intraperitoneal fluid. Massive pancreatic ascites, caused by communication of a disrupted pancreatic duct with the peritoneal cavity, is rarely seen. This presentation is usually associated with a more severe form of pancreatitis. In most of these patients, characteristic pancreatic and peripancreatic abnormalities are recognized..

Diagnostic Sensitivity

- The accuracy of CT in the diagnosis of acute pancreatitis depends to a large extent on the severity of the disease. The reported CT sensitivity for the diagnosis of acute pancreatitis ranges from 77% to 92%. The usefulness of CT is further supported by its high specificity. In most series, there are few false-positive findings, and CT specificity as high as 100% has been reported.[15] In addition, by examining the entire abdomen, CT can reveal a variety of other abdominal conditions in patients with clinically suspected acute pancreatitis.

Limitations in Diagnosis

- Limitations in the CT diagnosis of acute pancreatitis are related to suboptimal examinations resulting from poor technique, lack of

intravenous contrast medium, or inability of the patient to cooperate. Motion or streak artifacts and paucity of retroperitoneal fat are limiting factors in some patients. In addition, in mild forms of clinical pancreatitis, morphologic parenchymal or retroperitoneal abnormalities do not develop and the gland may appear normal on CT. It occurs in patients with mild symptoms and transitory elevation of serum amylase levels. The incidence of normal CT scans in these persons is not well established because surgical or pathologic correlation is lacking, but it has been estimated to be 14% to 28%. Experience has shown that given a good-quality CT scan, all patients with moderate or severe pancreatitis will exhibit some CT abnormality. In patients with a normal CT, pancreatitis is either absent or of minimal clinical significance.

Staging

- By virtue of its ability to accurately and rapidly evaluate the pancreas, peritoneum, and retroperitoneum, CT can be used to predict the severity of acute pancreatitis. As early as 1982, Hill and coworkers correlated the initial CT findings with the clinical type of edematous or necrotizing pancreatitis. Other investigations prospectively correlated the CT appearance with the severity of disease, development of complications, and death. Earlier studies using slower scanners, 10-mm collimation, and slow intravenous drip infusions staged pancreatitis on the basis of size, configuration of the gland, and the presence and degree of fluid collections. With the use of helical scanners and power injection of intravenous contrast material, the CT evaluation has concentrated on the

appearance and density of the pancreatic gland in an attempt to detect or exclude pancreatic necrosis.[16,17]

Grading System

- Balthazar and colleagues have initially classified the type and severity of acute pancreatitis into five grades :

  Grade A: Normal pancreas

  Grade B: Focal or diffuse enlargement of the gland including contour irregularities and inhomogeneous attenuation but without peripancreatic inflammation

  Grade C: Intrinsic pancreatic abnormalities associated with inflammatory changes in the peripancreatic fat

  Grade D: Small and usually single, ill-defined fluid collection

  Grade E: Two or more large fluid collections or the presence of gas in the pancreas or retroperitoneum

- On the basis of two consecutive prospective studies including 171 patients, it was found that patients with grade A or B pancreatitis had a mild uncomplicated clinical course, whereas patients with grade D or E disease developed a protracted clinical illness and significantly higher incidence of abscess and death. Abscesses developed in 17% of patients with grade D pancreatitis and in 61% of those with grade E pancreatitis. Patients with grades D and E pancreatitis had 14% mortality and 54% morbidity compared with 0% mortality and 4% morbidity in patients with grades A, B, and C. Studies of patients with minimal extrapancreatic inflammation showed no deaths, whereas small areas of fluid collections led to a 4% mortality, and extensive fluid involvement resulted in a mortality of 42%. This initial CT

grading system had limitations in predicting morbidity and mortality in patients with retroperitoneal fluid collections because half of these patients did not develop complications. In addition, this grading system did not include pancreatic necrosis, which has significant prognostic implications.

Delineation of Necrosis

- During the arterial phase of bolus intravenous administration of contrast medium, the normal pancreas should enhance homogeneously. Mild inflammation and interstitial edema do not interfere with the expected homogeneous enhancement of the gland. When necrosis is present, there is absence of contrast medium enhancement together with liquefaction and a change in the density or signal intensity of the gland. Gadolinium-enhanced T1-weighted gradient-echo MR images demonstrate pancreatic necrosis as low signal areas of nonenhancing parenchyma. The process can be focal or segmental or can affect the entire pancreatic gland. It can be grossly quantified as involving 30% or 50% or all of the gland. In Kivisaari and associates' original series using CT, mild pancreatitis was associated with a rapid rise in the density of the gland by 40 to 50 HU after contrast administration. Pancreatic necrosis was found at surgery in all patients who exhibited absence of enhancement or low enhancing values of less than 30 HU. There is good correlation between necrosis and length of hospitalization, development of complications, and death. Garg and colleagues[13] found a mortality rate of 12% for patients with sterile necrosis and 50% for those with infected necrosis.

Limitations: Staging

- Extensive pancreatic necrosis is detected mostly in patients with grades D and E pancreatitis. Pancreatic necrosis, however, develops early, within the first 24 to 48 hours in the course of the disease. Necrosis may be the initial CT manifestation and may precede the development of peripancreatic fluid collections. Furthermore, small patchy areas of pancreatic necrosis can be missed, particularly on the initial CT examination. These are much better defined on a follow-up examination performed a few days after the initial acute attack. Therefore, the accuracy of CT to detect and quantify pancreatic necrosis is higher 2 to 3 days after the clinical onset of disease, when there is liquefaction of the ischemic pancreatic tissue. A combined CT evaluation of the pancreatitis grade plus degree of pancreatic necrosis improves the ability of CT to predict complications.

Severity Index

- On a scale of 1 to 10, patients are assigned 0 to 4 points based on the A-to-E grading. To this, 2, 4, or 6 points are added if the initial CT scan detects up to 30%, 30% to 50%, or more than 50% necrosis. A severity index classification into three distinct categories (0-3, 4-6, and 7-10 points) more accurately reflects the prognostic value of CT as judged by the initial CT examination. There is a statistically significant increase in morbidity and mortality rates in these three groups of patients.

  Severity index (0 or 1) -- no mortality and morbidity.

  Severity index (2) -- no mortality and only 4% morbidity.

  Severity index (7 to 10) -- 17% mortality and a 92% complication rate.

Computed Tomography versus Clinical Prognostic Signs

- The average number of prognostic signs (Ranson's grave signs) is higher in patients with grades D and E pancreatitis than in those with lower grades of disease. All patients with more than five prognostic signs have grade E pancreatitis. The correlation, however, is poor in patients with up to five prognostic signs. A study comparing Ranson criteria with the Balthazar CT criteria for detection of severe acute pancreatitis found that the CT prognostic indicators had greater sensitivity and specificity. Other studies have shown that the CT severity index is superior to the APACHE II score to predict necrotizing pancreatitis and superior to the Simplified Acute Physiology score for prediction of a favorable outcome. It should be emphasized, however, that a close correlation between CT findings and clinical scoring systems should not be expected. This is because CT evaluates local abdominal complications, whereas clinical prognostic signs are indicative of systemic complications.
- A **modified CT severity index** has been proposed, which includes a simplified assessment of pancreatic inflammation and necrosis as well as an evaluation of extrapancreatic complications. The modified CT severity index differentiates only between the *presence and absence of acute fluid collections*, not taking into account the number of collections. In addition, it scores pancreatic necrosis into three levels only, which are “no necrosis”, “minimal necrosis”, or “substantial necrosis”. Last, the presence of extrapancreatic findings such as pleural effusion, ascites, vascular and parenchymal complications, and gastrointestinal tract involvement is included in the analysis. Unlike the current CT severity

index, the modified index was able to predict the development of organ failure and the length of hospital stay when comparing patients with moderate pancreatitis to those with severe pancreatitis. The modified index also correlated better than the current CT severity index with the need for surgical or percutaneous procedures and the occurrence of infection.

Indications for Examination

- Many patients with edematous pancreatitis have a typical clinical presentation and show rapid amelioration with limited supportive therapy. They do not require imaging examinations for diagnosis or management.
- On the other hand, **CT imaging is needed and should be performed early when**

(1) The clinical diagnosis is in doubt

(2) There is failure to respond to medical treatment within 48 to 72 hours

(3) Acute abdominal symptoms (distention, tenderness), leukocytosis, or fever is present

(4) Patients have more than two of Ranson's grave signs

(5) There is a change in clinical status suggesting a developing complication.

- **Specific indications for performing MRI over CT would include**

(1) Iodinated contrast allergy or poor renal function

(2)To detect the etiology of pancreatitis such as choledocholithiasis, pancreas divisum, and pancreatic tumors

(3)To characterize complex fluid collections and to determine their drainability.

Complications

Fluid Collections and Pseudocysts

- Fluid collections develop in up to 50% of patients with acute pancreatitis. The fluid may be pancreatic juice, serum, or blood. They develop either from actual rupture of the pancreatic duct with liberation of enzymes and pancreatic juice or secondary to exudation of fluid from the surface of the pancreas owing to activation of pancreatic enzymes within the gland. The fluid is contained by whatever structures happen to be adjacent to the collection. Most fluid collections are absorbed within 2 to 3 weeks. Unabsorbed fluid collections can organize and, within 4 to 6 weeks, develop a fibrous capsule, forming a pseudocyst. The lack of resorption of extravasated secretions and a communicating tract with the pancreatic duct are implicated in the development of pseudocysts. The main causes of pancreatic pseudocysts are chronic alcoholism (75%) and abdominal trauma (13%), with cholelithiasis, pancreatic carcinoma, and idiopathic causes composing the remainder. If the patient with a suspected pseudocyst has no history of pancreatitis, pancreatic trauma, or pancreatic surgery, a follow-up imaging study or aspiration biopsy may be required to exclude a pancreatic cystic neoplasm.
- The clinical significance of a pseudocyst is related to its size and the potentially lethal complications that may occur. Spontaneous rupture in the peritoneal cavity leads to pancreatic ascites or peritonitis. Secondary infection results in abscess formation. Erosion into an adjacent vessel leads to massive and sudden hemorrhage. Most of these complications, however, occur in pseudocysts larger than 4 to 5 cm. Small pseudocysts are often seen on CT in asymptomatic patients, have a low incidence of morbidity, and can be

followed expectantly with clinical and CT examinations. Surgical, endoscopic, or percutaneous drainage is indicated for pseudocysts that increase in size, become symptomatic, or develop complications. Success rates of over 90% have been reported with percutaneous drainage of pseudocysts guided by CT or sonography.[18]

- Pseudocysts vary greatly in size, are round or oval, and are located either within the pancreatic parenchyma or outside it. On CT scans, they are characterized by low fluid density (<15 HU) contents and by a peripheral fibrous capsule. Higher attenuation values are indicative of secondary infection or the presence of necrotic tissue. A density greater than 40 to 50 HU are suggestive of intracystic hemorrhage. On MRI, uncomplicated pseudocysts typically show low signal intensity on Tl-weighted sequences and high signal intensity on T2-weighted sequences. Complicated pseudocysts may have high signal intensity on Tl-weighted images due to hemorrhagic or proteinaceous fluid and may contain solid debris, which are best depicted on T2-weighted images. MRI is superior to CT and ultrasound in its ability to characterize fluid collections.
- ERCP is less accurate than CT or MRI in demonstrating pseudocysts, as less than 50% of pseudocysts fill with contrast at ERCP. Conversely, MRCP is able to detect pseudocysts that do not communicate with the pancreatic duct. ERCP, however, may be required to demonstrate communication between a pseudocyst and the pancjreatic duct, although sometimes it can be seen on MRI. Communicating pseudocysts may require prolonged catheter drainage until the communication to the pancreatic duct closes and the cyst collapses. Decompression of concomitant pancreatic duct obstruction may be required.

- Although many of the initial poorly defined fluid collections seen in acute pancreatitis resolve spontaneously, the natural history of pseudocysts is difficult to predict. They may persist, they can resolve, or they can even continue to grow over time. Spontaneous resolution even of large pseudocysts can occur and is explained by drainage into the pancreatic duct, erosion into an adjacent hollow organ (small bowel, colon), or rupture with spillage into the peritoneal cavity.

Infected Pancreatic Necrosis

- Bacterial superinfection of necrotic and hemorrhagic pancreatic and peripancreatic tissue complicates severe forms of pancreatitis. Secondary infection of the necrotic tissue occurs in 40% to 70% of patients with necrotizing pancreatitis and is often the cause of death in this patient population. The frequency of bacterial infection is proportionate to the extent of pancreatic necrosis and duration of disease. Cultures usually reveal gram-negative organisms derived from the intestinal tract, particularly the colon, but an increase in gram-positive and fungal organisms has been observed in association with the prophylactic use of antibiotics. On CT, sterile, partially liquefied pancreatic necrosis cannot be differentiated from infected necrosis unless gas bubbles (seen in about 12%-18% of cases) are present in the necrotic tissue. In the absence of gas, if infected necrosis is suspected, a CT-guided percutaneous aspiration for Gram stain and culture should be performed. This technique is safe and has a sensitivity and specificity of more than 90%. Aggressive surgical treatment of infected pancreatic necrosis is recommended

because it can reduce the mortality rate significantly. If possible, surgery should be delayed for at least 2 weeks after the onset of illness to allow better demarcation between viable and necrotic tissue, thus avoiding loss of viable pancreatic parenchyma.

Abscess

- Fluid collections that fail to resorb represent an ideal medium for bacterial growth, explaining the development of abscesses. Abscesses occur in about 3% of patients after an attack of acute pancreatitis. They develop within several weeks after the onset of symptoms in patients with severe forms of pancreatitis. Their presence is usually heralded by deterioration in the clinical course with septic systemic symptoms. They may have an inconspicuous clinical presentation initially.
- Abscesses are located in the peripancreatic tissues and have different sizes and configurations. On CT scans, they appear as poorly defined or partially encapsulated fluid collections of different densities (20-50 HU) and are often indistinguishable from residual noninfected fluid collections. A more characteristic appearance, seen in about 20% of abscesses, is the presence of gas bubbles produced by gas-forming bacteria. Retroperitoneal gas may be seen in patients with enteric fistulas; however, it is always strongly suggestive of an abscess. CT is more sensitive than MRI for detection of small gas bubbles, although large collections of gas or gas-fluid levels can be detected on T2-weighted MR images. CT is quite accurate in depicting the location and extent of small collections of retroperitoneal fluid and gas. Infection should be suspected

in all patients with pancreatitis in whom poorly encapsulated fluid collections are still present 2 to 4 weeks after the initial attack. MRI may be able to differentiate abscesses with complex internal content from simple collections in these patients. It is important to differentiate infected necrosis from abscesses, because the mortality risk for infected necrosis is double that of pancreatic abscesses. Percutaneous drainage is the therapeutic procedure of choice for pancreatic abscesses.

Hemorrhage

- Although small patchy areas of hemorrhage combined with necrotic tissue are common findings in acute pancreatitis, massive life-threatening intra-abdominal hemorrhage is seldom reported. This complication may occur within 2 to 3 weeks to several years after an acute episode of pancreatitis. It is the result of erosion of peripancreatic vessels with the formation of a pseudoaneurysm and subsequent retroperitoneal bleeding. Commonly, the site of bleeding is located along the pancreaticoduodenal arcade or along the splenic vessels adjacent to the tail of the pancreas. Pseudo-aneurysms can be located within a pancreatic pseudocyst. Hemorrhage occurs when a slowly enlarging pseudoaneurysm ruptures into the peritoneum, or erodes into an adjacent hollow viscus or into the pancreatic duct producing hemosuccus pancreaticus. CT examination can identify retroperitoneal bleeding by the presence of high-density (50-100 HU) fluid collections. MRI is more sensitive than CT for the detection of hemorrhage, which is depicted as high signal intensity on T1-weighted fat-suppressed images because of the presence of methemoglobin.

Dynamic enhanced MDCT or T1-weighted gadolinium-enhanced MR images show a pseudoaneurysm as a rapidly enhancing mass similar in contrast density to the adjacent arteries and aorta. Spillage of contrast material into the retroperitoneum due to active bleeding, as well as fresh blood in the peritoneal cavity, can also be diagnosed. It is important to have a high index of suspicion for pseudoaneurysm in patients with history of pancreatitis and a suspected mass in the pancreas or its vicinity. Accordingly, biopsy of these lesions should be performed only after a contrast-enhanced CT, MRI, or Doppler sonography study has excluded a pseudoaneurysm. If the source of bleeding is obscured by the surrounding hemorrhage, angiography is crucial in identifying the presence and precise location of the bleeding pseudoaneurysm. Therapeutic arterial embolization of the bleeding vessel is required; it is an emergency, lifesaving procedure. Massive hemorrhage from ruptured vessels or pseudo-aneurysms requires surgical treatment.

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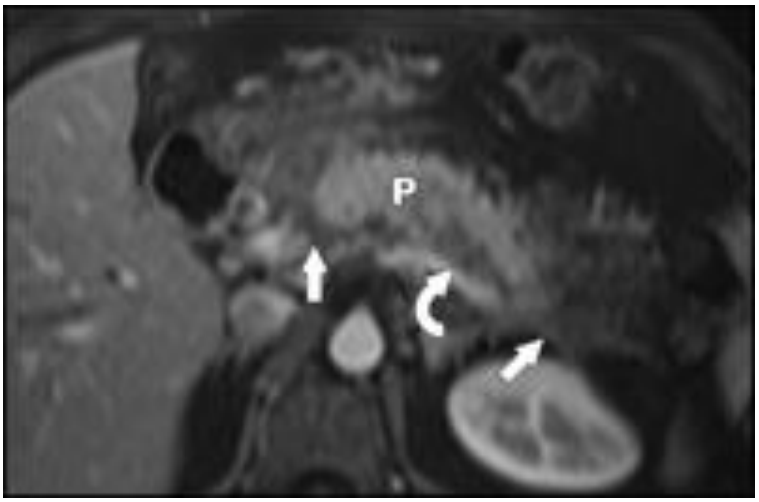
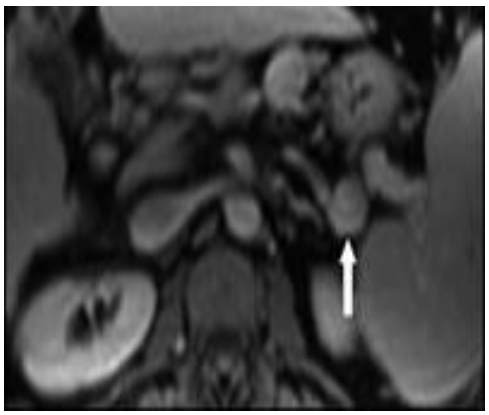
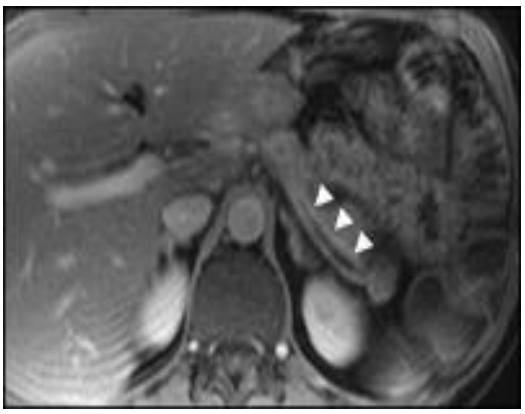


PANCREATITIS AFTER ERCP. Axial enhanced T1-weighted fat-suppressed spoiled gradient-echo image obtained during venous phase shows heterogeneous pancreatic enhancement due to pancreatitis with peripancreatic fluid and inflammation (*straight arrows*). Fluid (*curved arrow*) is seen between pancreas and splenic vein. P = pancreas.





ACUTE PANCREATITIS. Axial enhanced T1-weighted fat-suppressed spoiled gradient-echo images showing splenic artery pseudoaneurysm (*arrow*) enhancing similar to arteries and low-signal-intensity thrombus (*arrowheads*) in splenic vein.

CHRONIC PANCREATITIS

- Chronic pancreatitis is a relatively uncommon disease of prolonged pancreatic inflammation characterized by irreversible morphologic and functional damage to the pancreas. The incidence of pancreatic cancer is significantly elevated in patients with chronic pancreatitis.

Etiology

- In the United States, approximately 75% of cases of chronic pancreatitis are due to alcoholism. Continued consumption of alcohol for a 3- to 12-year period is necessary before the manifestations of chronic pancreatitis develop. In contrast to their major role in the development of acute pancreatitis, gallstones play little role in the etiology of chronic pancreatitis. Hyperlipidemia, hyperparathyroidism, trauma, and pancreas divisum have all been implicated as risk factors for developing chronic pancreatitis.

Clinical Findings

- Pain is the predominant clinical finding in 95% of patients with chronic pancreatitis. The pain typically radiates from the epigastrium through the back and can be constant or intermittent and extremely difficult to palliate, frequently requiring narcotics or neurolysis. Weight loss often accompanies the pain, and these two findings raise the clinical suspicion of a malignancy. Endocrine and exocrine deficiencies occur with progressive destruction of the gland. Diabetes and malabsorption with steatorrhea eventually develop in approximately half the patients with chronic pancreatitis.

- The clinical diagnosis of chronic pancreatitis, especially in its early stages, is often difficult. Histopathologic diagnosis is rarely available, because of the risks associated with pancreatic biopsy, including acute pancreatitis, fistula, and hemorrhage. Therefore, the diagnosis is based on clinical, morphologic, and functional abnormalities. ERCP and pancreatic function tests are considered the gold standard diagnostic procedures, but they have limitations, and prolonged clinical follow-up is sometimes required to confirm the diagnosis.

Radiologic Findings

Plain Radiographs -

- Typical pancreatic calcifications are diagnostic of chronic pancreatitis. They develop in 40% to 60% of patients with alcoholic pancreatitis, and approximately 90% of calcific pancreatitis is caused by alcoholism. Unfortunately, calcifications occur late in the course of chronic pancreatitis, being associated with severe disease. Most pancreatic calculi are small, irregular calcifications that may be diffuse or confined to a specific region of the pancreas. Although they can appear on plain radiographs, CT is the most specific and accurate imaging modality for depicting pancreatic calcifications.

UItrasonography

- Although its contribution in acute pancreatitis is modest, sonography is often used in patients with suspected chronic pancreatitis and its complications. Sonography shows a 60% to 80% diagnostic accuracy rate and provides a noninvasive, inexpensive, and rapid method of evaluating morphologic changes in the pancreas. Sonographic findings include abnormalities in gland

size, irregular margins, inhomogeneous or heterogeneous echogenicity of parenchyma, and dilatation of the pancreatic duct. Calcifications are recognized as shadowing echogenic foci within the parenchyma or the main pancreatic duct. Pseudocysts are often present in chronic pancreatitis, and they are usually unilocular, anechoic, and sharply defined. Other complications of chronic pancreatitis such as biliary dilatation and splenic vein thrombosis can also be detected with sonography.

- Abnormalities in size and contour of the pancreas are the least sensitive indicators of chronic pancreatitis on ultrasound studies. Decreased echogenicity occurs when there are acute exacerbations with parenchymal edema. Dilatation of the pancreatic duct is one of the most common sonographic abnormalities of chronic pancreatitis and is seen in up to 90% of cases as a tubular, anechoic structure in the pancreatic body.

Endoscopic Sonography

- In many cases, EUS is helpful to elucidate the cause of pancreatitis, being able to detect small pancreatic tumors and microlithiasis not seen by other imaging modalities. EUS findings indicative of chronic pancreatitis include parenchymal calcifications, hyperechoic foci or strands, pseudocysts, heterogeneous echotexture, and lobular contour of the gland. Ductal abnormalities include dilatation and irregularity, hyperechoic walls, intraductal stones, and visible branches. Chronic pancreatitis is likely when more than two of these findings are present. With more than six findings, the disease is probably moderate to severe. Unlike ERCP, EUS has the advantage of being able to evaluate simultaneously the pancreatic ductal system and the

parenchyma. However, the diagnosis of chronic pancreatitis based on EUS changes alone is controversial. There is usually a good correlation of EUS with ERCP in patients with moderate or severe chronic pancreatitis but not in those with mild disease. Others studies reported abnormal findings at EUS in patients with normal ERCP and pancreatic function tests, suggesting that EUS may overdiagnose chronic pancreatitis or, alternatively, it may be more sensitive than ERCP and function tests to detect subtle pancreatic changes.

Endoscopic Retrograde Cholangiopancreatography

- Endoscopic retrograde cholangiopancreatography (ERCP) is the standard imaging modality to evaluate the pancreaticobiliary tract, being a diagnostic and therapeutic procedure. Because of its invasiveness and potentially serious complications, however, ERCP is ideally reserved for patients who need intervention.
- In early chronic pancreatitis, the pancreatic duct is often normal, limiting the sensitivity of ERCP. The earliest changes involve the first-order and second-order side branches of the main pancreatic duct and include dilatation and contour irregularity, clubbing, stenosis of the side branches, and opacification of small cavities. Some of these changes, however, can be seen in elderly normal patients and must be interpreted with caution in this age group.
- With disease progression, the involvement of the main pancreatic duct increases with more dilatation, mural irregularities, loss of normal tapering, and areas of stenosis or occlusion. If a solitary stricture is seen in the main pancreatic duct, the differential considerations include

neoplasm or pseudocyst. Stenoses are usually shorter, smoother, and more symmetric in pancreatitis than those associated with neoplasm. Biopsy of suspicious lesions or brushing and collection of pancreatic secretions can be performed with ERCP and may be helpful in the diagnosis of pancreatic carcinoma. In advanced chronic pancreatitis, the dilatation is more marked, and intraductal calculi can be seen. The pancreatic duct and side branches may have a "chain of lakes" appearance.

- According to the Cambridge Classification or its modifications, chronic pancreatitis is considered mild if the main pancreatic duct is normal but at least three side branches are abnormal. Moderate disease requires abnormalities in the main pancreatic duct and in more than three side branches. Severe disease includes the abnormalities of moderate disease plus one of the following: a large cavity, ductal obstruction, filling defects, severe dilatation, or irregularity.

Magnetic Resonance Cholangiopancreatography

- MRCP has been increasingly used in patients with suspected pancreatitis or pancreaticobiliary abnormalities because it lacks ionizing radiation, does not require iodinated contrast material, and is noninvasive, sparing the patient potential complications. MRCP is also helpful in patients with anatomic abnormalities that impede cannulation of the common bile duct or pancreatic duct. Using heavily T2-weighted sequences, MRCP is able to depict fluid-filled structures such as pseudocysts and detect abnormalities of the pancreatic duct including dilatation, irregularity,

intraductal stones, and multiple or severe obstructions, which may be a limitation for ERCP. MRCP has the advantage of showing the ductal segments proximal and distal to an obstruction, being able to evaluate its character, extent, location and cause.

Computed Tomography and Magnetic Resonance Imaging

- The diagnostic criteria for chronic pancreatitis on CT and MRI examinations are based on assessment of the size and contour of the gland, dilatation and shape of the pancreatic duct, and presence of ductal calcifications. CT has reported sensitivities of 50% to 90% and specificities of 55% to 85% in the detection of chronic pancreatitis.[19]
- Although CT correctly detects the morphologic alterations of chronic pancreatitis, its ability to evaluate severity of disease is more limited. These shortcomings are related to (1) the inability of CT to accurately diagnose incipient forms of chronic pancreatitis that do not exhibit gross morphologic changes and (2) poor correlation between functional exocrine and endocrine deficit and pancreatic morphology on imaging studies. When compared to ERCP and pancreatic function tests, CT is not sensitive in the diagnosis of early chronic pancreatitis. Some experts believe that MRI can detect findings of chronic pancreatitis prior to CT based on signal intensity abnormalities of the pancreas. These changes are best visualized on nonenhanced and gadolinium-enhanced T1-weighted fat-suppressed images. The normal pancreas has high signal intensity due to the presence of acinar proteins and enhances markedly following gadolinium administration. Chronic pancreatitis and associated

fibrosis result in loss of proteinaceous material and decreased signal intensity of the pancreas.

- Several studies have demonstrated that focal pancreatitis and pancreatic carcinoma may have similar appearance and pattern of enhancement on CT and MRI, because of the presence of fibrosis in both types of lesions. Both conditions may also cause dilatation of the pancreatic duct and common bile duct (double-duct sign), ductal strictures, arterial encasement, and peripancreatic venous obstruction. In addition, carcinoma develops in 2% to 3% of cases of chronic pancreatitis, and this malignant degeneration is often difficult to appreciate on CT. A new or rapidly increasing homogeneous and ill-defined pancreatic mass, which can be detected by comparison with previous CT examinations or short-term follow-up, is indicative of malignancy. Imaging features that favor the diagnosis of inflammatory mass related to chronic pancreatitis over adenocarcinoma include a non-dilated or smooth-tapering pancreatic duct coursing through the mass (duct penetrating sign), the presence of pancreatic calcifications, a lower ratio of duct caliber to pancreatic gland width, and irregular ductal dilation. The imaging features of adenocarcinoma and focal pancreatitis often overlap, however, and complementary studies including ERCP and EUS with fine needle aspiration biopsy may be required for definite diagnosis.
- Dilatation of the pancreatic duct and its secondary radicles (>2 to 3 mm in size) is characteristic for chronic pancreatitis. In advanced disease, the main duct appears beaded, irregular, or smooth, often containing stones. These pancreatic duct abnormalities are better demonstrated on MRI than

on CT. Subtle changes in side branches seen in early chronic pancreatitis, however, are best depicted on ERCP The pancreatic duct should be visualized entirely to the level of the papilla because small tumors of the pancreatic head may produce similar findings. The pancreatic duct may be dilated in patients with senile atrophy of the pancreas, mimicking chronic pancreatitis.

- CT demonstrates pancreatic calcifications in approximately 50% of patients with chronic pancreatitis. These are the most reliable imaging features of the disease. They can be scattered throughout the gland, isolated, or focal in the head or body of the pancreas. Calcifications vary from innumerable to single and small.
- Pseudocysts of various sizes may be present within the pancreas or in an extrapancreatic location. They occur in 25% to 60% of patients with chronic pancreatitis and are usually stable.
- Pseudoaneurysms and thrombosis of the splenic vein with extensive collateral circulation and gastric varices are sometimes encountered in chronic pancreatitis and can be readily diagnosed by CT and MRI. Additionally, patients with repeated episodes of acute exacerbation may present with an acute abdominal catastrophe, dropping hematocrit, and evidence of massive intra-abdominal bleeding.

Pancreaticopleural Fistula

- Pancreaticopleural fistula can occur in acute or chronic pancreatitis or as a result of pancreatic trauma. Pancreatic secretions from a ruptured pancreatic duct dissect through the aortic or esophageal hiatus or directly

through the diaphragm and reach the mediastinum, pleural cavities, pericardium, or bronchial tree. Patients usually present with large pleural effusions and dyspnea. A high index of suspicion for pancreaticopleural fistula is required. CT and MRI may identify the fistula, especially when coronal reformatted images in are used, as well as the changes of chronic pancreatitis. ERCP has been considered the best imaging modality for evaluation of pancreaticopleural fistulas, but technical failures may result from incomplete opacification of the pancreatic duct or a long fistulous tract. MRCP may depict the fistula and the ductal anatomy and can be helpful in the surgical planning of these patients, potentially replacing ERCP.

Groove Pancreatitis

- Patients who have repeated episodes of pancreatitis or acute exacerbations of chronic pancreatitis may develop a form of segmental pancreatitis described as “groove pancreatitis”, in which the inflammatory reaction and fluid collection dissect into the “groove” between the duodenum and the head of the pancreas. Because of the enzymatic action of pancreatic secretions, as many as 50% of patients develop duodenal stenosis and/or strictures of the common bile duct. The recognition of groove pancreatitis is important to distinguish from pancreatic and duodenal carcinoma.
- Contrast-enhanced CT shows a poorly enhancing lesion extending between the duodenum and the pancreatic head. Cysts in the groove or

duodenal wall and duodenal stenosis are often seen. The head of the pancreas enhances normally.

- MRI findings of groove pancreatitis include a sheetlike fibrotic mass between the pancreatic head and a thickened duodenal wall associated with cystic changes in the duodenal wall. This fibrotic mass has a low signal intensity on Tl- and T2-weighted images. The cystic component is low signal intensity on Tl-weighted images and high signal intensity on T2-weighted images and does not enhance due to its fluid content. The fibrotic component demonstrates delayed enhancement following gadolinium and is easily demarcated from the normal pancreas, which has marked enhancement initially and less intense enhancement on more delayed images.
- Similar duodenal changes, referred to as **cystic dystrophy of the duodenal wall**, have been reported in patients with *acute pancreatitis* and *heterotopic pancreatic tissue in the duodenum*.

Autoimmune Chronic Pancreatitis

- Autoimmune chronic pancreatitis has an autoimmune mechanism and may coexist with diabetes mellitus and autoimmune diseases such as Sjogren's syndrome, primary sclerosing cholangitis, and primary biliary cirrhosis. It is relatively asymptomatic, predominates in middle-aged or older men, and usually shows a remarkable response to steroids. Laboratory findings may include increased levels of serum gammaglobulin and/or IgG, or the presence of autoantibodies. Pancreatic

fibrosis with infiltration of lymphocytes and plasma cells is seen on histopathological examination.

- Imaging studies show a rare association of diffuse enlargement of the pancreas with irregular narrowing of the pancreatic duct. On CT and MRI, the pancreas appears diffusely enlarged, with a "sausage-like" appearance, usually without calcifications or stones. As in chronic pancreatitis of other etiologies, the pancreas shows decreased enhancement in the arterial phase and increased enhancement in delayed phases after contrast administration. A characteristic low-density rim, likely composed of fibrous tissue, is often seen surrounding the pancreas. This capsule-like rim shows delayed enhancement on dynamic imaging and low signal intensity on Tl- and T2-weighted MR images. Diffuse or segmental irregular narrowing of the main pancreatic duct associated with strictures of the intrahepatic and extrahepatic biliary tract is typically demonstrated on ERCP. MRCP shows well the biliary tract abnormalities but does not show the stenosis of the pancreatic duct as well as does ERCP.

PANCREATIC NEOPLASMS

The U.S. Armed Forces Institute of Pathology (AFIP) classification includes benign, borderline, and malignant exocrine tumors; benign, borderline, and malignant endocrine tumors; intraductal neoplasms; soft tissue neoplasms; secondary (metastatic) neoplasms; and non-neoplastic tumorlike conditions (e.g., mass-forming chronic pancreatitis).

DUCTAL ADENOCARCINOMA

Epidemiology and Pathology

- The estimated frequency of ductal adenocarcinoma of the pancreas is 9 per 100,000 patients, 11th among all cancers. Common risk factors for developing pancreatic adenocarcinoma include cigarette smoking, black male, BRCA2 gene positivity, hereditary pancreatitis, cirrhosis, diabetes mellitus, chronic pancreatitis, hypercholesterolemia, obesity, and certain occupations and carcinogens.

- From 80% to 90% of patients with newly diagnosed pancreatic cancer will have advanced disease at the time of discovery. Unfortunately, sensitive, specific, noninvasive, and widely available screening methods have remained elusive. An elevated serum CA 19-9 is the only clinically available blood test to suggest the presence of pancreatic malignancy; however, because 15% to 20% of patients do not secrete this marker and the marker may not be detectable when the tumor is small (and potentially curable), its utility is limited. Continued advances in the detection of abnormal gene expression and identification of other biomarkers may lead to effective molecular screening leading to effective nanobiotic genetic "bullet" therapy.

- The American Joint Committee on Cancer has developed a tumor-node-metastases (TNM)-based staging system for pancreatic adenocarcinoma. Unfortunately, only 15% to 20% of patients are candidates for surgery at the time of diagnosis. Thus the majority of patients will be staged solely by imaging.

- Ductal adenocarcinoma accounts for 85% to 90% of all pancreatic tumors—60% to 70% arising in the head, 5% to 10% in the body, and 10% to 15% in the tail of the gland. Up to 22% of tumors can affect multiple regions of the gland (diffuse). By the time the tumors are discovered, they usually measure 2 to 3 cm in diameter. Pancreatic adenocarcinoma has scant cellular elements and elicits an intense desmoplastic response, encasing intrapancreatic blood vessels, which explains why 90% of these masses are hypodense compared with background pancreas. The upstream main pancreatic duct may be obstructed, and the surrounding parenchyma is atrophic. Tumors in the head of the pancreas will also eventually obstruct the common bile duct. The tumor rapidly grows through lymphatics, along peripancreatic vessels and along nerves; they may also grow along tissue planes into the surrounding duodenum and posterior wall of the stomach. Lesions in the tail of the pancreas infiltrate into the splenic hilum and into the left renal hilum, via the gastrosplenic and splenorenal ligaments respectively. Distant metastases are most frequently found in the liver and peritoneal cavity.

- The major blood vessels involved by pancreatic ductal adenocarcinoma are the superior mesenteric artery, the celiac axis, and their branches. More frequently, the tumor will involve the superior mesenteric, splenic, and portal veins. Once the major arterial branches are involved with tumor, most surgeons agree they are unresectable.

Imaging

Goals of Imaging

- Although the diagnosis of ductal pancreatic adenocarcinoma may be clinically suspected, the diagnosis is established by an imaging examination, MDCT, MRI, and/or endoscopic ultrasound (EUS), with needle aspiration biopsy. Other imaging modalities such as transabdominal ultrasound (US), diagnostic endoscopic retrograde cholangiopancreatography (ERCP), ^{18}F-fluorodeoxyglucose-PET (FDG-PET), and receptor (somatostatin) specific nuclear medicine studies are secondary procedures.

- The protocol for the imaging test is designed to detect the presence of a pancreatic neoplasm and to determine whether an individual patient has truly resectable disease. Timing of the imaging acquisition must be optimized to maximize lesion-to-background pancreas contrast differences, evaluate the integrity of major peripancreatic arteries and veins, and detect extrapancreatic metastases.

Imaging Findings

Multidetector Computed Tomography

- Pancreatic-directed MDCT is the primary imaging test for patients with suspected pancreatic adenocarcinoma. The neoplasm is most frequently hypodense compared with the background parenchyma and its borders, poorly defined some 11% of lesions may be isoattenuating. MDCT can detect lesions at a small size before they deform the contour of the gland. This underscores the importance of imaging the pancreas during the phase of maximal background pancreatic enhancement, where the tumor-to-gland attenuation

difference is greatest. Secondary findings include dilatation of the upstream pancreatic duct and dilatation of the common bile duct when the tumor is located within the pancreatic head. Use of curved-multiplanar reformatting or 3D volume rendering can improve detection of ductal dilatation.

- CT findings of major arterial encasement include obliteration of the normal fat between the pancreatic margin and the adjacent vessel, greater than 180-degree contact between the tumor and the vessel, and morphologic changes in the artery that include narrowing or encasement of the affected artery. Using 3D volume rendering, images of the pancreatic arterial supply can be created that rival conventional catheter angiography, making recognition of the vascular changes straightforward. CT angiographic images have been shown to be more accurate for detecting arterial involvement from pancreatic adenocarcinoma than by looking at traditional axial slices alone.

- Criteria for venous invasion include greater than 180- degree contact with a soft tissue mass and the vein. When the superior mesenteric vein is involved with tumor, it may display a “teardrop” configuration. Although soft tissue contact with the venous system has a high predictive value for nonresectability, significant involvement of the vein may be found at surgery when the imaging study fails to reveal any direct contact with the vein. It is therefore critical to evaluate the presence and pattern of collateral venous channels surrounding the pancreatic head. Common collateral channels include prominent short gastric varices, gastrohepatic ligament varices, and gastroepiploic to gastrocolic trunk. It is important to look for presence of the small posterior pancreaticoduodenal veins; when these collaterals are present, there is a high likelihood that the tumor has involved the superior mesenteric

vein to a degree that would preclude the ability to obtain a negative tumor margin.

Magnetic Resonance Imaging

- Because the pancreatic mass is hypovascular and elicits a dense desmoplastic response, most masses will have a shorter relaxation time than the surrounding normal parenchyma and therefore appear as a "dark" region on T1-weighted sequences. Fat suppression will increase the conspicuity of the mass by significantly decreasing the signal from intraperitoneal and retroperitoneal fat, thereby increasing the signal from the pancreas. This heightens the contrast between normal parenchyma, the mass, and the pancreatic duct. Multiphasic gadolinium-enhanced sequences using fat suppression are the most valuable for lesion detection and assessment of resectability. Following the intravenous administration of gadolinium, pancreatic adenocarcinoma enhances less than the background pancreatic parenchyma. As with CT, pancreatic enhancement is maximal approximately 40 to 50 seconds following intravenous contrast administration (pancreatic phase), making this phase the most valuable for mass detection. Liver metastases are best seen against a background of densely enhanced liver, which occurs in the portal venous phase. Metastases have longer Tl and T2 relaxation times. MRCP images are also obtained to assess bile and pancreatic duct involvement.

Endoscopic Ultrasound

- EUS is excellent for the detection of tumors less than 2 cm and can provide an excellent guide for fine-needle aspiration biopsy. EUS can detect vascular invasion of the portal venous system. EUS is superb for local staging of the neoplasm; however, its utility decreases in evaluating distant metastases.

Considerable literature suggests that EUS can aid in the differentiation of pancreatic adenocarcinoma from chronic pancreatitis. Pancreatic neoplasm is suggested by a hypoechoic irregular mass and hypoechoic enlarged nodes. Vascular invasion is suspected when there is loss of the normal vessel border, but the true accuracy of this finding is controversial. EUS is the preferred method for obtaining histologic confirmation of malignancy. There is a statistically significantly lower chance of developing peritoneal carcinomatosis following EUS biopsy compared with percutaneous fine-needle aspiration biopsy.

- The most frequent cause of imaging understaging pancreatic adenocarcinoma is the failure to detect small metastases to the liver and peritoneal cavity. Small peritoneal implants may be present without ascites or other more flagrant signs of peritoneal carcinomatosis, making them extremely difficult to diagnose. To detect these small lesions, laparoscopic ultrasound is typically performed immediately prior to surgery in those patients deemed resectable by imaging. In most clinical practice, MDCT is the primary imaging modality, with MRI reserved for patients with contrast allergy. EUS is used in cases where ductal obstruction is visualized without a mass visible on the cross-sectional study and as the best method for obtaining tissue to confirm malignancy.

Other Imaging Modalities

- **Transabdominal ultrasound** is commonly used in jaundiced patients to rapidly identify a dilated biliary tract and to evaluate the gallbladder. While biliary dilation and gallbladder stones are well demonstrated, the specific etiology of extra-hepatic bile duct dilatation is frequently difficult to establish.

The most frequent reason for this is bowel gas obscuring the region of the pancreatic head and common bile duct. In selected patients, however, an echogenic mass obstructing either the pancreatic or common bile ducts will be depicted. Promising results from contrast-enhanced transabdominal ultrasound are under investigation.

- Demonstration of an obstruction or stricture in an otherwise normal main pancreatic duct is indicative of ductal adenocarcinoma at **ERCP**. ERCP has no role in the preoperative assessment of pancreatic adenocarcinoma when the diagnosis is established by other imaging modalities. ERCP is often used preoperatively to place a drainage stent within an obstructed CBD. Several studies have shown that this preoperative stent placement should not be performed before surgical resection as there is clear increase in complication rate. Most leading pancreatic surgeons will request CBD stenting when surgery will be delayed more than 14 days following diagnostic evaluation. Expandable metallic endoscopically placed endobiliary stents should be reserved for those patients with unresectable disease needing palliation.

CYSTIC NEOPLASMS

- Cystic pancreatic neoplasms have recently become a major focus of imaging and clinical research due to a number of factors: (1) the increasing recognition of these masses paralleling the wide use of imaging, (2) the potential that these lesions may serve as a model for pancreatic tumorogenesis, and (3) these cystic tumors have a better prognosis than pancreatic ductal adenocarcinoma.

- Cystic pancreatic neoplasms can be divided into four distinct categories: (1) serous cystadenoma, (2) mucinous cystic neoplasm, (3) intraductal papillary mucinous tumor (IPMT), and (4) other.

Serous Cystadenoma

- Serous cystadenomas (SCAs) account for approximately 1% to 2% of all exocrine pancreatic neoplasms. The World Health Organization (WHO) classification of pancreatic tumors considers SCA to be a benign lesion. From 25% to 50% of patients will be symptomatic at the time of diagnosis. The tumor occurs more frequently in women, with a mean age of 57 ages, and most often occurs in the pancreatic head. The tumor contains glycogen-rich periodic acid-Schiff (PAS)-positive epithelial cells delimiting cysts separated by varying amounts of fibrous septations. They most frequently occur sporadically; 60% to 80% of patients with Von Hippel-Lindau syndrome have pancreatic SCA.
- SCA has two morphologic appearances: microcystic or classic type, and the macrocystic or oligocystic type. The microcystic form, accounting for two thirds of SCAs, is a spongelike lesion formed from innumerable cysts containing clear, watery fluid. The cysts range from 1 to 5 mm in the center of the mass; larger cysts (up to 2 cm) are present in the periphery. A central fibrous stellate nidus is present, giving rise to radially oriented fibrous bands. This central nidus frequently calcifies. The oligocystic type has scant locules or be unilocular. There is no central scar, and the cyst fluid may be clear but often is hemorrhagic.

- The imaging features of SCA reflect the morphologic appearance of the mass. In the "classic" variety, there is a solitary mass that displays central calcification and a stellate arrangement of dense tissue delimiting varying numbers of cysts. In some tumors, the cysts are so small and the fibrous component so dense that the lesion may actually appear "solid". On MDCT, the cysts are near water density and the surrounding fibrous network appears dense. The presence of central calcification best detected by MDCT establishes the diagnosis of SCA but is present in fewer than 30% of cases. The tumors may encase vessels and obstruct the pancreatic and/or biliary duct systems. Nevertheless, even these seemingly aggressive tumors may remain indolent.
- On T1-weighted fat-suppressed MRI sequences, the fluid component is darker than the fibrous matrix, whereas on T2-weighted acquisitions, the fluid component becomes more conspicuous, appearing bright due to the longer T2 relaxation time. EUS may be particularly useful in displaying the honeycombed internal structure in lesions less than 2 cm. EUS provides an excellent means to sample cyst fluid.
- The oligocystic variety of SCA should be suspected when there is a unilocular nonenhancing cystic mass in the pancreatic head with a lobulated contour. Despite the high reported specificity of this constellation of findings, differentiation of this lesion from mucinous cystic tumor or pseudocyst may be impossible.

Mucinous Cystic Tumor

- These rare neoplasms are thought to all be potentially malignant; therefore, the terms "mucinous cystadenoma" and "mucinous cystadenocarcinoma" should not be used. Rather, this lesion is more properly called a mucinous cystic tumor (MCT). MCTs are formed from variably atypical epithelial cells that produce mucin and are supported by an ovarian-type stroma that does *not* communicate with the pancreatic duct system. They account for 2% to 6% of all exocrine pancreatic neoplasms. These tumors occur almost exclusively in women, with peak incidence in the fifth decade. The female preponderance is similar to that of mucinous tumors of the hepatobiliary system, retroperitoneum, and ovary. The stromal component stains for cytokeratin markers indicative of cellular luteinization.
- At MDCT, the lesions will display large cysts with thin septae, best seen following intravenous contrast administration. When calcification occurs, it is lamellated (as opposed to the "starburst" pattern seen in SCA) and in the periphery of the lesion (as opposed to the central location of calcification in SCA). Lesions with a higher degree of epithelial atypia will display nodules on the wall, peripheral calcification, and a more disorganized internal architecture. Malignant lesions tend to be larger than benign lesions. On MRI, the lesion will appear bright on T2-weighted sequences. On T1-weighted sequences, intravenous gadolinium is necessary to image the septations that become more apparent the longer the imaging sequence is carried out. Mucin within the lesion can produce decreased signal within the center of the lesion that should not be confused with the radiating septae seen in SCA. On EUS, the mural nodularity is easily recognized and can be differentiated from the

honeycomb appearance of an SCA. Endoscopic ultrasound is particularly valuable for aspirating cyst fluid. Presence of carcinoembryonic antigen (CEA) has a high predictive value for the presence of a mucinous cystic tumor.

Intraductal Papillary Mucinous Neoplasm

- The intraductal papillary mucinous neoplasm (IPMN) was first described at ERCP by Ohhashi and colleagues in 1982. The entity has been reported under a wide variety of names, including ductectatic mucinous cyst- adenoma, villous adenoma of the main pancreatic duct, and intraductal mucinous hypersecreting. The 1997 AFIP fascicle suggested “intraductal papillary mucinous neoplasm” as the best single term for this lesion. The pathologic hallmarks of the lesion are (1) diffuse or segmental dilatation of the main and/or branch pancreatic ducts, (2) intraductal growth of the mucin-producing epithelial lining cells, and (3) protrusion and dilatation of the major and minor papilla with mucus excretion. In slightly more than 50% of the cases, the pancreatic duct bulges into the ampulla of Vater with marked hypersecretion of mucus seen in the duodenum. Two morphologic forms are distinguished: (1) those involving the main pancreatic duct with or without side branch involvement and (2) those exclusively involving the branch ducts. Cases in which the main duct is involved have a significantly higher likelihood of harboring more malignant epithelium than do those restricted to the branch ducts. Branch duct IPMNs can arise anywhere within the pancreas, although head, neck, and uncinate process are the most common locations. Individual lesions contain a wide spectrum of epithelial dysplasia, but an individual lesion's biologic behavior is variable. Factors correlating with malignancy

include (1) advanced age, (2) main duct involvement, (3) concurrent diabetes or other pancreas related abdominal symptoms, (4) lesion size (>3 cm) and multiplicity, and (5) the presence of visible mural nodules or any other solid elements. As opposed to SCA and MCT, IPMN occurs with a slight increased frequency in male patients.

- At imaging, the lesion is characterized by increased diameter of a portion of the pancreatic duct system. Visualization is optimized by using contrast-enhanced thin-section MDCT or T2-weighted MRI. The diagnosis is confirmed by demonstrating communication of the cystic lesion with the pancreatic duct. This is best visualized by the use of 3D techniques including CT cholangiopancreatography and MRCP. Careful attention to protocol will allow the radiologist to demonstrate the bulging papilla into the duodenal lumen; a finding that, when present, allows distinction from chronic pancreatitis. Mucin is intensely echogenic and is well suited to EUS evaluation. EUS is particularly useful for evaluating main duct tumors, particularly the longitudinal extent of main duct involvement, aiding surgical planning.
- ERCP is useful for aspirating mucin and confirming the diagnosis. Cross-sectional techniques are more sensitive than ERCP in visualizing branch duct involvement. ERCP aspiration of duct contents aids in the differentiation of main duct IPMN from chronic pancreatitis.
- Serial observation suggests that small tumors (<3 cm) confined to branch ducts follow an indolent clinical course. Several large studies conclude that for patients with branch duct IPMN, no solid elements, and size less than 3.0 cm, serial imaging without intervention may be sufficient.

Other Cystic Tumors

- The solid and pseudopapillary epithelial neoplasm (SPEN) is most frequently found in female patients, with a mean reported age in the mid 20s. This lesion has been reported in patients as young as 10 years and as old as 79 years. Review of the literature suggest that approximately 15% of these tumors are malignant, with the likelihood of malignancy increasing with the patient's age.
- The proportion of solid and cystic elements within the tumor is directly related to the degree of hemorrhage the tumor has undergone. The lesion appears as a round, encapsulated mass with varying amounts of necrotic and soft tissue foci. The presence of blood products within the tumor results in hyperintense signal on T1-weighted MRI sequences. No septations are visualized; central and/or rim calcifications have been reported in 29% of patients. The recommended treatment is surgery. Localized lesions are treated as low-grade neoplasms. Median survival rates of 5.2 years are reported for patients with metastatic disease.
- Any pancreatic neoplasm can display a “cystic morphology”. Cystic islet cell tumors and cystic ductal adenocarcinomas have been reported. True epithelial pancreatic cysts are extremely rare in the absence of systemic cystic disease such as adult polycystic liver and kidney disease or von Hippel-Lindau syndrome. When a cystic tumor of the pancreas is recognized, every effort should be made to exclude a history of acute or pancreatitis because pancreatic pseudocysts remain the single most common cystic pancreatic mass.

ISLET CELL TUMORS (FUNCTIONING AND NONFUNCTIONING)

- Pancreatic endocrine tumors (islet cell tumor) can be divided into either nonfunctioning (nonsecreting, inactive) and functioning (secreting, active) types. Benign or malignant tumors can exist within each group. Even when both groups are combined, islet cell tumo'rs account for only 1% to 2% of all pancreatic neoplasms. They occur with equal frequency in men and women, peak age in the 50s and 60s, and can be located anywhere within the gland. They can occur sporadically or be inherited as in patients with the autosomal dominant multiple endocrine neoplasia (MEN I) syndrome.
- Functioning islet cell tumors include insulinoma, gastrinoma (the most common form seen in patients with MEN I), glucagonoma, VIPoma, and somato- statinoma.
- Nonfunctioning endocrine tumors may be clinically silent but do secrete hormones; these hormones may be (1) secreted in tiny amounts, (2) substances that are rapidly degraded, and (3) biologically inactive.
- Islet cell tumors have similar imaging features regardless of their hormonal output. The most typical appearance is that of a hypervascular focus best seen in the arterial phase of enhancement on MDCT and fat-suppressed, gadolinium-enhanced Tl-weighted MRI. In patients with Zollinger-Ellison syndrome or MEN I, the second portion of the duodenum must be carefully evaluated. Current imaging techniques will detect up to 90% of functioning endocrine neoplasms. Nonfunctioning islet cell tumors display a wide variety of imaging appearances, but the most common appearance is that of a hyperdense pancreatic mass with hypervascular hepatic metastases.

- Insulinoma is the most common islet cell tumor, occurring with an incidence of approximately 1:100,000. Fifty percent of the lesions are found in the head of the pancreas; the remainder are found elsewhere within the gland; 85% are solitary; and 0.5% of the lesions are extrapancreatic. Most lesions are 1 to 2 cm. Ten percent of the lesions are malignant; malignant insulinomas are larger (measuring up to 8 cm), produce extremely high levels of insulin or proinsulin. Metastases are often present at the time of diagnosis. When the lesion is solitary and localized, simple excision or enucleation is sufficient for treatment. In advanced cases, surgery must attempt to completely remove the tumor and debulk metastases for adequate control of the hypoglycemia.
- Gastinoma is the second most common islet cell tumor. Most are located in the anatomic region between the pancreatic head and common bile duct and within the first or second portion of the duodenum, the "gastrinoma triangle".[20] The excessive gastrin production leads to the Zollinger-Ellison syndrome either as a sporadic entity or as a manifestation of MEN I. From 50% to 70% of sporadic cases of Zollinger-Ellison syndrome are usually the result of tumors within the pancreas, whereas the remaining cases are secondary td tumors located within the duodenum. Most of the gastrinomas found in MEN I are located within the duodenum.
- Glucagonoma is a malignant tumor in over 60% of patients at the time of diagnosis. These lesions are usually found in the tail of the pancreas. These tumors give rise to a clinical syndrome characterized by a migratory necrolytic erythema, angular stomatitis, chelits, and atrophic glossitis.
- VIPomas produce watery diarrhea, hypokalemia, hypochlorhydria, and metabolic acidosis, the Verner-Morrison syndrome. The masses are large,

solitary masses in the pancreatic tail; 80% of these tumors are malignant at the time of diagnosis. Approximately 35% of somatostatinomas will present in the pancreas, 52% in the duodenum, and the remaining 13% anywhere else. Seventy percent of tumors arising in the pancreas are malignant, whereas 50% of duodenal lesions are malignant. These tumors are extremely rare.

Secondary Pancreatic Neoplasms

- The pancreas can be secondarily involved by neoplasm via either (1) direct extension from a contiguous primary tumor (e.g., gastric carcinoma), (2) invasion from local metastatic lymphnodes (e.g., invasion of the pancreatic head by metastatic peripancreatic lymph nodes draining a primary tumor of the right colon), or (3) hematogenous metastases. Renal cell carcinoma is the most common primary neoplasm to metastasize to the pancreas. The lesions appear as hypervascular foci within the pancreatic parenchyma. Other common sources of metastases to the pancreas include melanoma and lung cancer; however, metastases to the pancreas can originate from virtually any primary neoplasm.

PANCREATIC TRAUMA

- There is considerable truth to the surgical aphorism that "the pancreas is not your friend" in terms of the unforgiving nature of pancreatic injuries. Owing to its retroperitoneal location, pancreatic injuries are not easily diagnosed by diagnostic peritoneal lavage, which has a high false-negative rate.
  Furthermore, the classic presentation of upper abdominal pain, leukocytosis,

and hyperamylasemia common in acute pancreatitis is far less reliable in the setting of pancreatic trauma. Multidetector row computed tomography (MDCT), ultrasound, magnetic resonance imaging (MRI), and endoscopic retrograde cholangiopancreatography (ERCP) can expedite the diagnosis and management of these patients.

- The degree of serum hyperamylasemia does not correlate with the severity of pancreatic injury and may actually be greater in patients with contusions than in those with frank transections. The retroperitoneal location of the pancreas often masks symptoms of significant injury. In some cases, the patient may be asymptomatic initially, with pain, tenderness, and abdominal distention developing gradually in a 12- to 24-hour period. Peritoneal lavage results are usually negative if the peritoneum overlying the pancreas remains intact.

- Another clinical sign recently shown to correlate with the presence of pancreatic injury in children is the "seat belt sign." Pancreatic injury was 22 times more commonly present in children with this sign (erythema, ecchymoses, and abrasions across the patient's abdominal wall resulting from the vehicle's seat belt restraint).

The American Association for the Surgery of Trauma Pancreatic Injury Grading Scale

Grade		Injury description
I	Haematoma	Minor contusion without ductal injury
	Laceration	Superficial laceration without ductal injury
II	Haematoma	Major contusion without ductal injury or tissue loss
	Laceration	Major laceration without ductal injury or tissue loss
III	Laceration	Distal transection or pancreatic parenchymal injury with ductal injury
IV	Laceration	Proximal transection or pancreatic parenchymal injury involving the ampulla
V	Laceration	Massive disruption of the pancreatic head

Radiologic Findings

Computed Tomography

• CT is the preferred means of evaluating patients with blunt abdominal trauma. Pancreatic lacerations are often subtle relative to injuries involving the liver, spleen, or kidneys and difficult to visualize on imaging studies immediately after trauma. Focal pancreatic enlargement and peripancreatic fluid are suggestive of pancreatic injury, and faint lacerations/ fracture lines may be seen on closer inspection. The radiologist must rely on secondary findings because many injuries manifest as pancreatitis with diffuse or focal swelling of the pancreas. Minor thickening of the anterior limb of Gerota's fascia and fluid or increased attenuation in the anterior pararenal space, small bowel mesentery, or transverse mesocolon may be the only CT abnormalities. Fluid between the pancreas and the splenic vein was initially thought to be a useful sign of pancreatic injury. This sign was subsequently found to be neither sensitive nor specific in a later study.

• A major limitation of CT in pancreatic injury is that it often cannot reliably assess the integrity of the pancreatic duct. As CT may not directly show the pancreatic duct injury, such injury may be suspected based on the degree of parenchymal injury. In addition, the pancreas may appear relatively normal on initial examinations. Later examinations may demonstrate injuries not seen on the initial examination; however, this may relate to the characteristic evolution of pancreatic injury rather than an inaccurate initial examination. The tear progressively increases in size and becomes more well defined, often associated with extrapancreatic fluid collections. Thin sections or repeat scanning in 12 to 24 hours can be helpful if there is a strong suspicion for fracture in the face of a

negative CT result. Some of the prior reports of poor results with CT examination may, in part, relate to the studies being performed without helical CT scanners, thin slices, and optimal intravenous and/or lack of oral contrast material.

•Pancreatic contusions most often are isodense but occasionally may present as hyperdense masses because of intra- parenchymal hemorrhage or maybe hypodense due to edema. A laceration through the pancreas is usually manifested by a thin linear lucency. Pancreatic fractures may be seen as well-demarcated lucent defects traversing the gland, generally involving the neck of the pancreas perpendicular to its long axis**.**

- There is often little or no separation of the pancreatic fragments, and the density change can be subtle but may be obscured by surrounding hematomas and fluid collections or streak artifacts. Helical CT has greatly improved the ability to identify pancreatic injuries by obtaining images without respiratory misregistration and allowing optimal delivery of intravenous contrast material and thinner slices than conventional CT. Pseudocysts may form within a few days after pancreatic duct laceration and appear similar on CT to pseudocysts from pancreatitis.

Ultrasound

- Although ultrasound is very sensitive to demonstrate intraabdominal fluid (hemoperitoneum), it has very limited value to demonstrate pancreatic injuries. The actual pancreatic parenchymal injury maybe difficult to visualize sonographically. In addition, many of these patients have an associated ileus limiting sonographic evaluation of the pancreas. The sonographic findings

may be relatively subtle, usually demonstrating nonspecific gland enlargement caused by pancreatitis or contusion.

Magnetic Resonance Pancreatography

- MRCP may identify abnormalities that may not be visualized on ERCP, including fluid collections and the presence of any additional post-traumatic abnormalities upstream from the site of duct disruption/transaction. The exact role of MRCP in the setting of acute pancreatic injury has not yet been established.

Conventional Pancreatography

- The main pancreatic duct is a rigid and brittle structure compared with the pancreatic parenchyma. Accordingly, the duct may be lacerated without producing cross-sectional imaging abnormalities or surgically detectable peripancreatic hemorrhage or capsular disruption. ERCP is not an appropriate screening test for pancreatic trauma; however, in cases in which the CT scan or MRCP study is equivocal or technically inadequate, emergent ERCP is useful in identifying the presence and location of pancreatic duct disruption. Demonstration of a normal duct can obviate the need for exploratory surgery in these patients. ERCP is also useful in detecting post-traumatic pancreatic ductal strictures and pseudocyst communication.

ROLE OF INTERVENTIONAL RADIOLOGY IN THE MANAGEMENT OF PANCREATIC LESIONS

- Percutaneous biopsy of a pancreatic neoplasm identified by CT scan, MR imaging, or sonography.

- Management of Infected pancreatic necrosis and pseudocysts - Percutaneous catheter drainage

Indications for Pancreatic Mass Biopsy

- **Patients with Imaging Findings That Suggest an Unresectable Pancreatic Cancer**

  In patients with unresectable pancreatic cancer and other extrapancreatic lesions, such as hepatic or peritoneal/omental masses, biopsy of the extrapancreatic mass could potentially result in less morbidity than biopsy of the pancreatic tumor.

- **Patients with a Known Extrapancreatic Primary Cancer**

  Percutaneous pancreatic mass biopsy is indicated in these patients to differentiate a surgically resectable pancreatic ductal adenocarcinoma from a metastasis. A pretreatment diagnosis is needed because virtually all metastases are treated medically while resectable pancreatic carcinomas are treated surgically. The importance of a pretreatment tissue diagnosis in these patients is especially emphasized in patients with lymphoma, renal cell carcinoma, and lung cancer, three tumors that spread not uncommonly to the pancreas. Therefore, when a pancreatic mass is detected in a patient with an extrapancreatic primary malignancy, the mass should not be presumed to represent a metastasis; biopsy should be performed.

- **Patients with a Pancreatic Mass That May Be Caused by Inflammation**

  Chronic pancreatitis (including autoimmune and groove pancreatitis subtypes) can appear masslike and mimic a pancreatic neoplasm.

- **Patients with a Cystic Pancreatic Mass**

- **Multiple Solid Pancreatic Masses**

MATERIALS AND METHODS

The study was conducted at R.L.Jalappa Hospital and Research Centre which is attached to Sri Devaraj Urs Medical College, Kolar, Karnataka. All patients presenting with pancreatic pathology and suspected by ultrasonogram in R.L. Jalappa Hospital were included in the study. The consent of the patients was taken prior to the investigation. The proposed prospective study include evaluation of cases over a period of one-and-half years i.e. from Dec 2008 –MAY 2010.

CT scan abdomen study was done on the 39 patients who fulfilled the inclusion criteria by using SIEMENS SOMATOM ESPRIT single slice spiral CT machine. The exposure settings used were 130 KvP and 80 to 100 mAs. Topographic image was taken from base of lungs to the pubic symphysis.

Plain CT scan abdomen 10 mm axial cuts were taken for all the cases. Except in one road-traffic case, oral contrast (10-15mL of ultravist in 2L of flavoured water) was given. After 45-60 min, contrast study of CT abdomen was undertaken by administering 80mL of ultravist @ 2.5mL/s by a pressure injector. Triple-phase study was done by doing arterial phase at a delay of 5-10s, venous phase at delay of 25-30s, and delayed phase was done 30-45min after contrast administration. Patient were asked to hold their breath during the arterial and venous phases.

For all the cases, pancreatic size, morphology and duct was assessed. Calcifications in pancreatic parenchyma or duct were taken into account. Peripancreatic fluid whether present or not was analysed. Whether the peripancreatic fat planes were maintained or obliterated were taken into account. Any loculated collections in pancreatic parenchyma itself or elsewhere in relation to the pancreas were noted.

Peripancreatic vasculature was also assessed for any thrombus or perivascular fat infiltration.

Other than pancreas, hepatobiliary system and spleen were assessed ---regarding fatty infiltration of liver, any space-occupying lesion, gallbladder calculi, common bile duct dilatation, hepatic or splenic infarct, etc.

Any free fluid in the peritoneal or pleural cavity was also taken into account.

CT findings were correlated with FNAC in two cases.

Excel software was used to analyze the statistical data.

RESULTS

During the period of 18 months of the study, 39 patients who fulfilled inclusion criteria were studied, out of which 31 were male and 8 were female **(Chart - 1).**

Majority of the patient cohort belongs to 36-45 years age group(n= 16), followed by 15-25 years group(n=7), 26-35 years(n=6), 46-55 years(n=4), 56-65 years(n=3), 66-75 years(n=2) and >75 years(n=1), respectively **(Chart - 2).**

Only 12 patients gave history of alcoholism **(Chart - 3)**. 16 patients went ahead for ultrasonographic examination of abdomen with clinical suspicion of pancreatic pathology **(Chart - 4)**. Pancreas was seen normal in 3 cases, completely obscured by bowel gas in 16 cases and partially obscured in 12 cases**(Chart - 5).**

USG diagnosed acute pancreatitis in 10, chronic pancreatitis in 4, pancreatic pseudocyst in 4 and carcinoma head of pancreas in 2 cases. Pancreatic pathology was not diagnosed by USG in 19 cases. **(Chart - 6)**

Serum amylase was found to be raised in 13 cases, normal in 8 cases, but was not done in 18 cases**(Chart - 7).**

Plain CT evaluation of abdomen showed calcification in 11 cases**(Chart - 8).**

Contrast study showed acute inflammatory changes in 21 cases, out of which 16 were found to have acute pancreatitis, 1 having associated pseudocyst formation, and 4 with acute-on-chronic pancreatitis. Chronic pancreatitis was found in 5 cases with associated pseudocyst formation in another 3 cases. Pseudopancreatic cyst was found in 3 cases. Malignant pathology involving pancreas was found in total 6 cases, with 2 cases having underlying chronic pancreatitis changes. One road-traffic-accident case was evaluated for having epigastric tenderness, and was found to have contusions involving head of pancreas**(Chart - 9).**

Acute pancreatitis cases were graded according to Balthazar grading, and majority of cases were found to have grade C(n=9) followed by grade E(n=6), grade D(n=5) and grade B(n=1), respectively **(Chart - 10).**

Pancreas was found to be diffusely bulky in 12 cases, followed by bulky head(n=7), bulky body-tail(n=3), bulky tail(n=2) and bulky head-body(n=1), respectively. It was found to be of normal size in 6 cases, and atrophic in 4 cases **(Chart - 11).**

Pancreatic morphology was found to be altered in 23 cases, was normal in 14 cases, and was not commentable in 2 cases **(Chart - 12).**

Pancreatic parenchyma showed necrotic components in 9 cases **(Chart - 13)**.

Peripancreatic fat strandings were noted in 31 cases **(Chart - 14)**, and peripancreatic fluid collection is noted in 9 cases **(Chart - 15)**.

Pancreatic duct was dilated in 9 cases, out of which 2 cases were tortuous and 2 showed ductal calculi. Duct was not made out in 2 cases **(Chart - 16).**

Pseudopancreatic cyst was found in 14 cases **(Chart - 17).**

In 4 cases, pancreas was found to be infiltrated by adjacent malignancies(cholangiocarcinoma(n=2), retroperitoneal tumor(n=1), lymphoma(n=1)), if only by fat infiltration **(Chart - 18).**

In 8 cases adjacent vasculature was found to be involved. Thrombosis was seen in 5 cases (Inferior venacava thrombus(n=2), portal vein thrombosis(n=2), and portal vein-superior mesenteric vein thrombosis(n=1)). Caeliac arterial fat infiltration was found in 2 cases, and that of superior mesenteric artery in 1 case. Portal vein and Common hepatic arterial fat infiltration was noted in 2 cases, with cuffing of splenic and hepatic artery in 1 case **(Chart - 19).**

Ascites was found in 17 cases**(Chart - 20)**, and pleural effusion in 14 cases **(Chart - 21).**

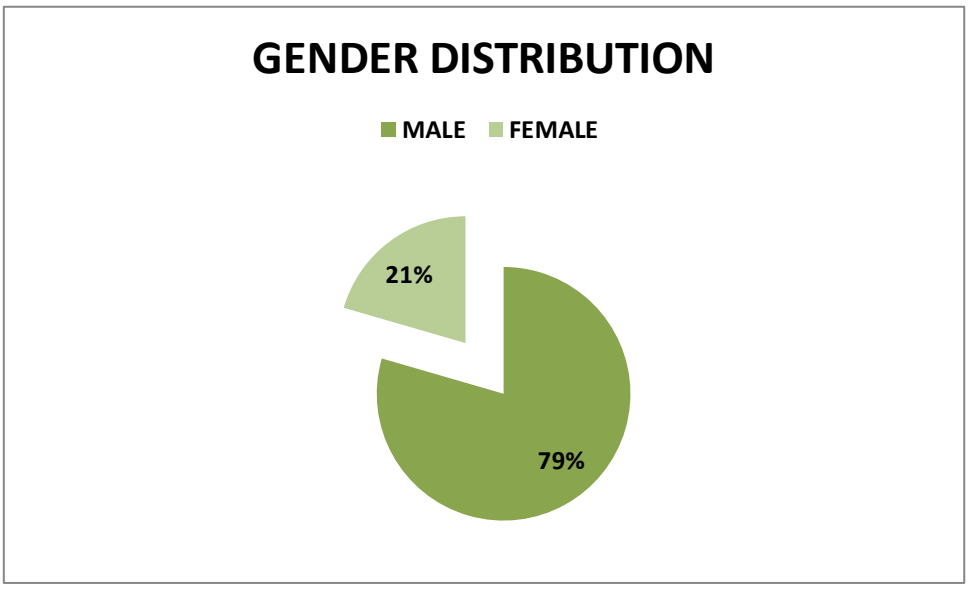
Gallbladder calculus was seen in only 6 cases. Gallbladder was not seen in 2 cases, one case being post-operative state**(Chart - 22).**

Common bile duct was found to be dilated in only 5 cases **(Chart - 23).**

Liver was found to be normal in 25 cases, had diffuse fatty infiltration in 8 cases, had metastases in 3 cases, and 1 case each of hepatocellular carcinoma, carcinoma gallbladder and hepatic infarct **(Chart - 24).**

OBSERVATIONS

CHART : 1



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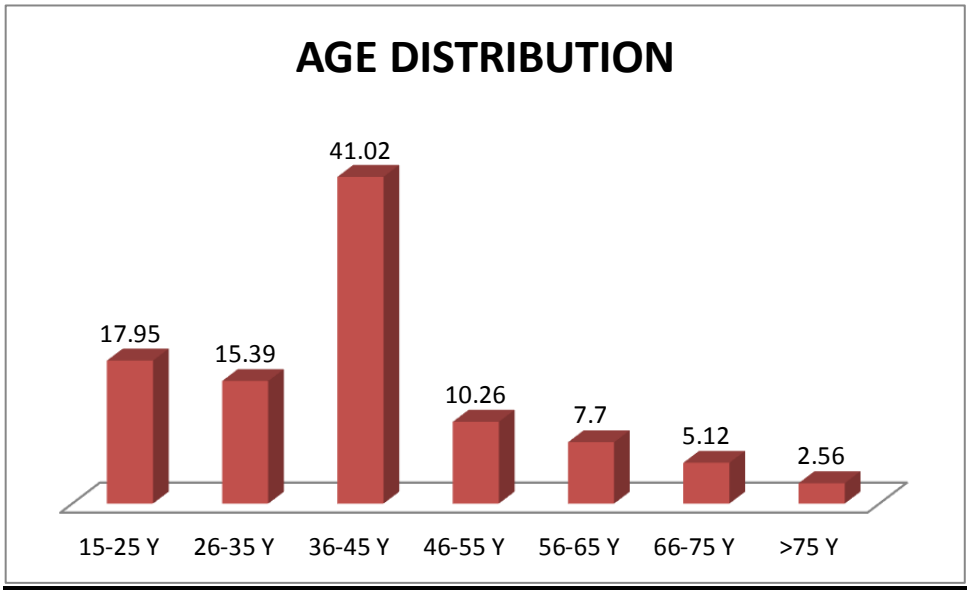
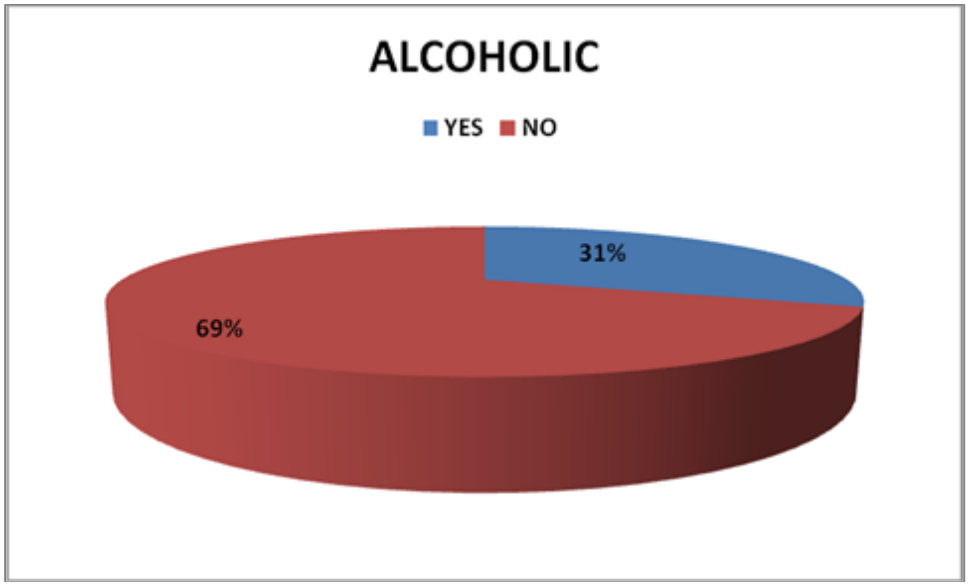


CHART : 3



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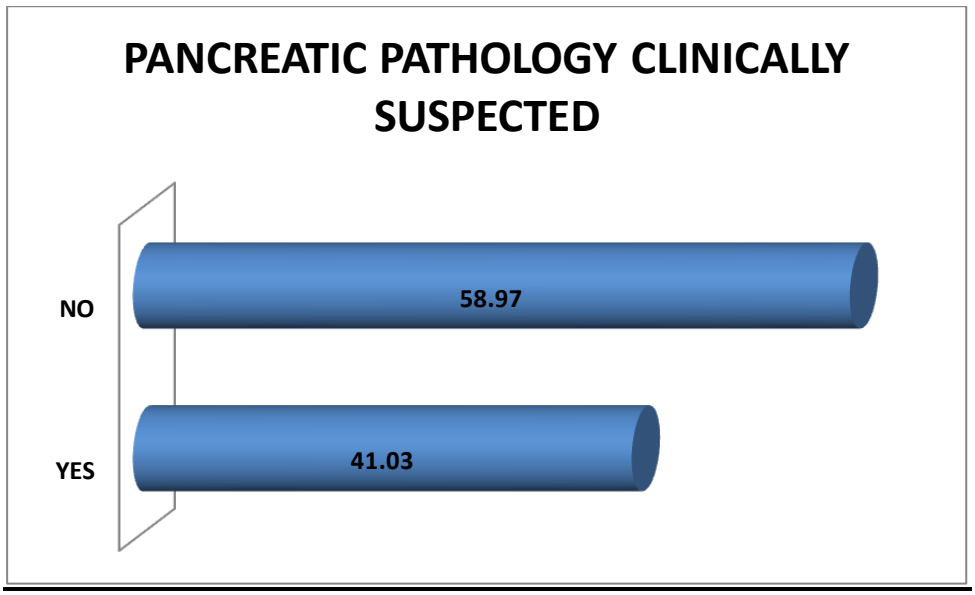

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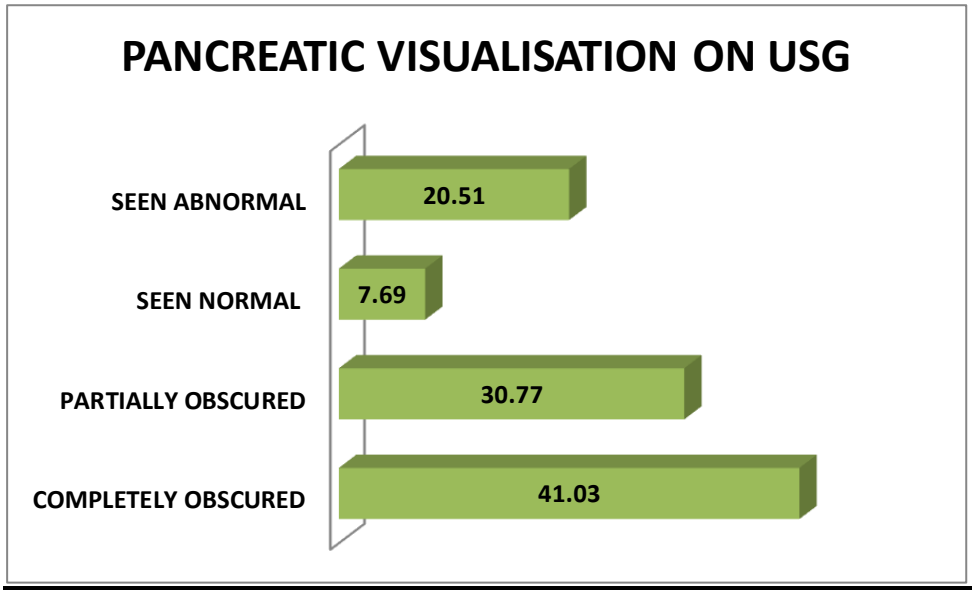

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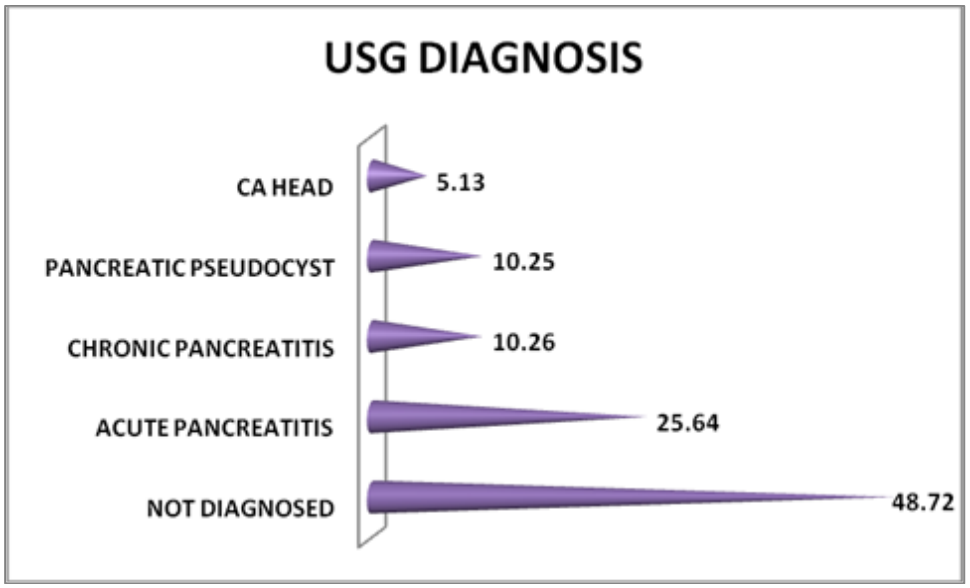

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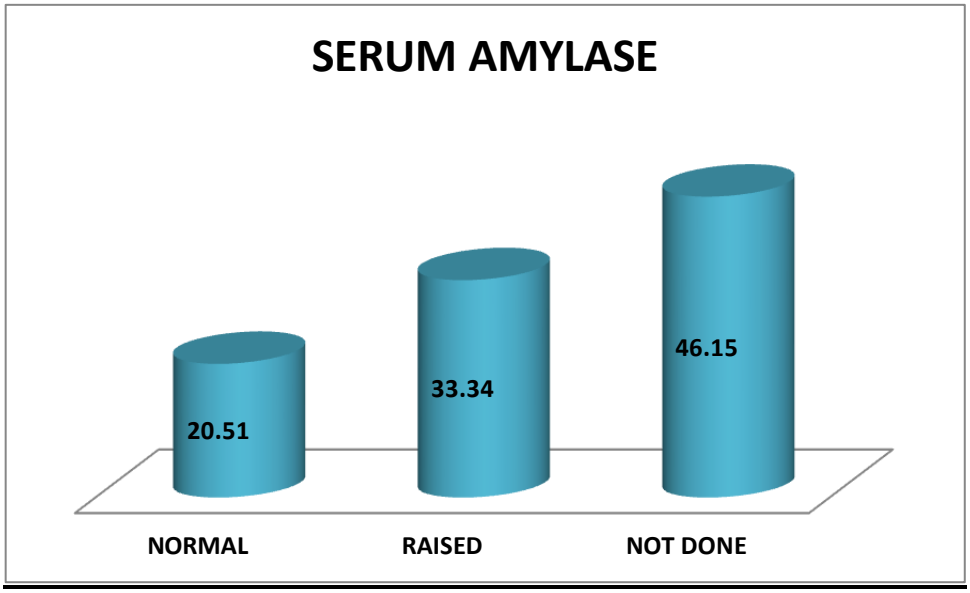

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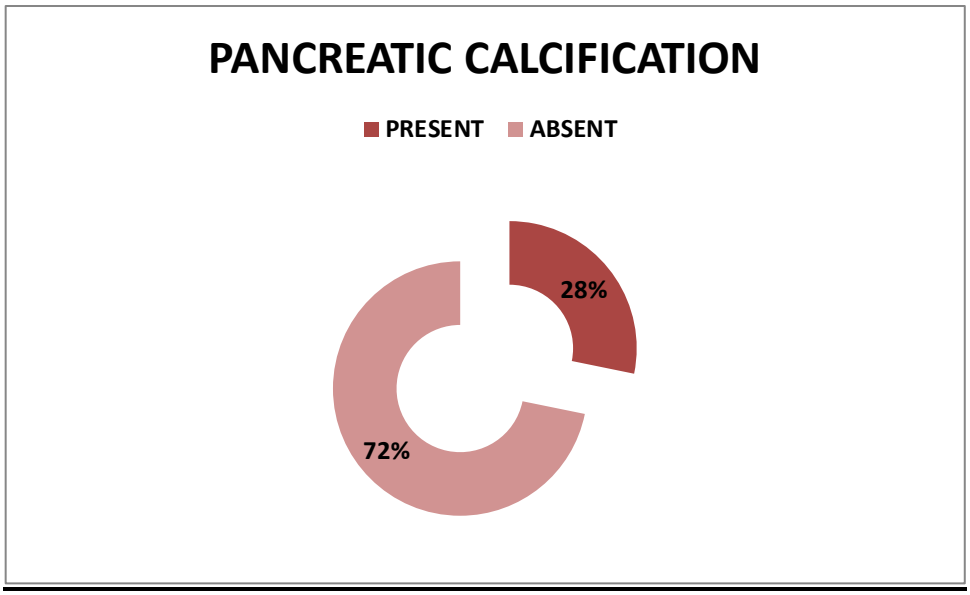

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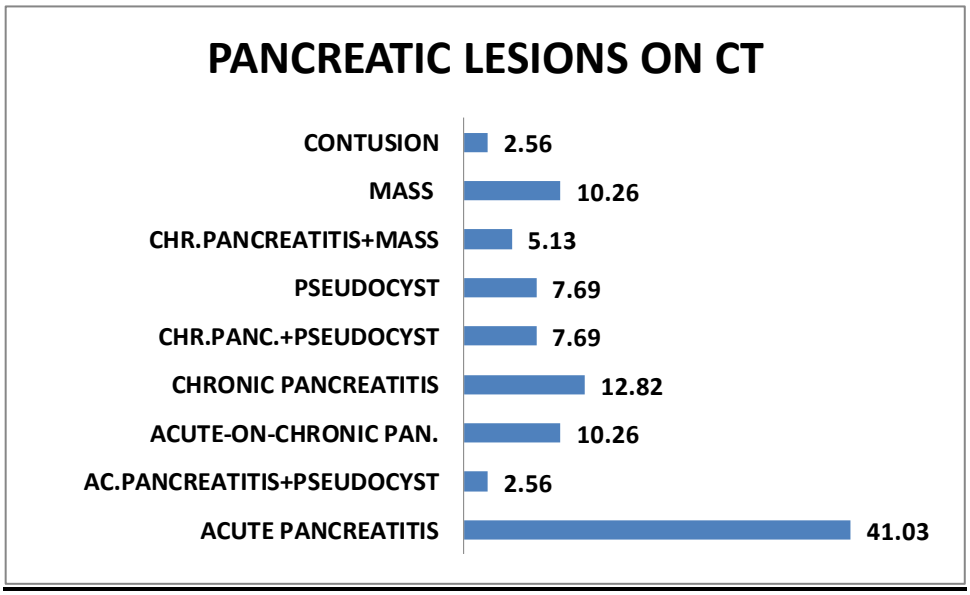

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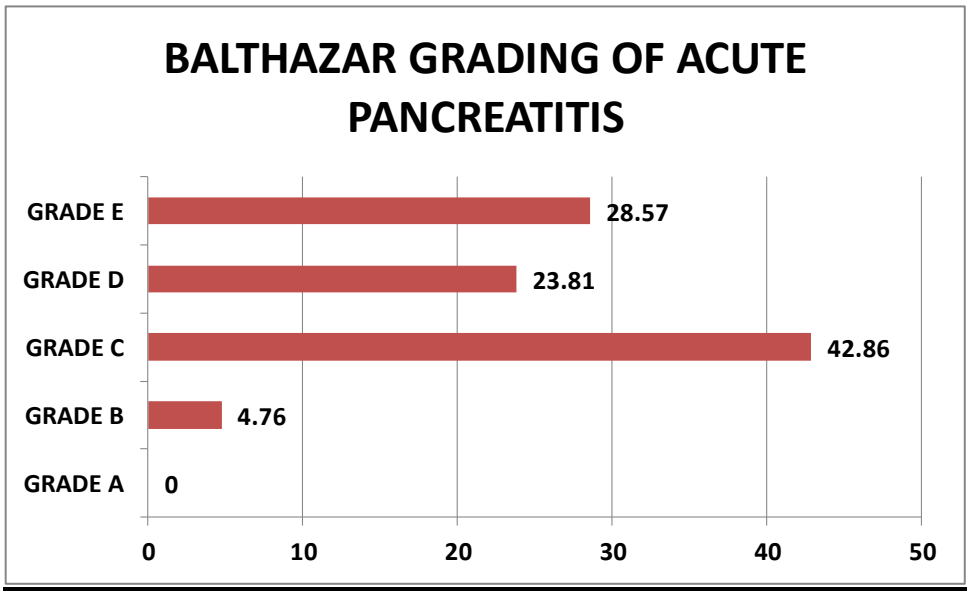

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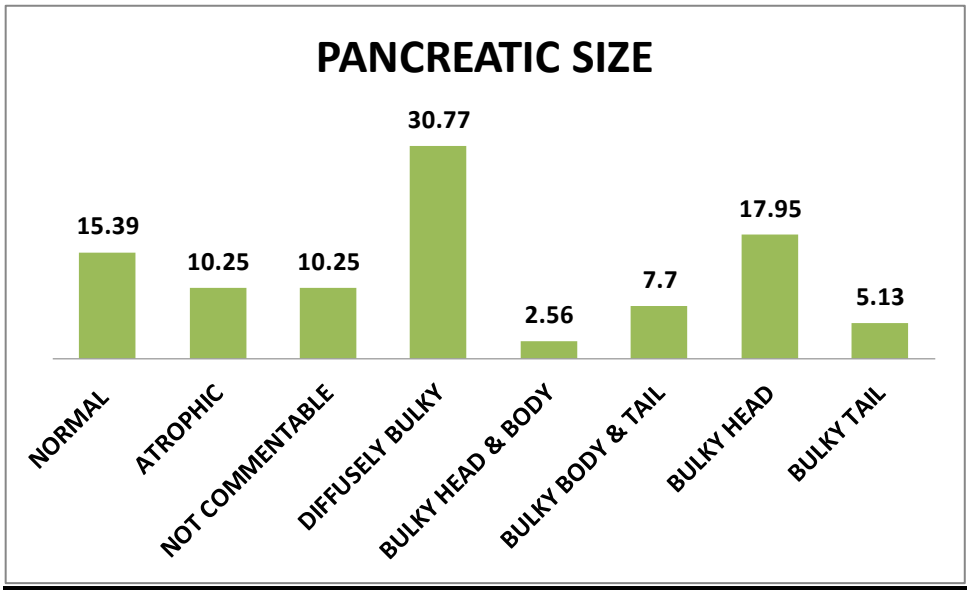

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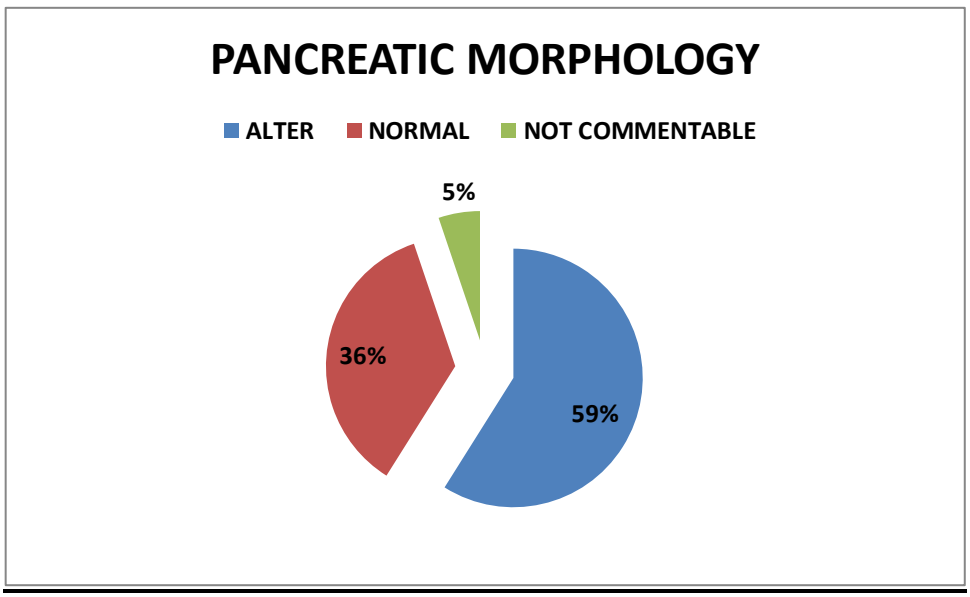

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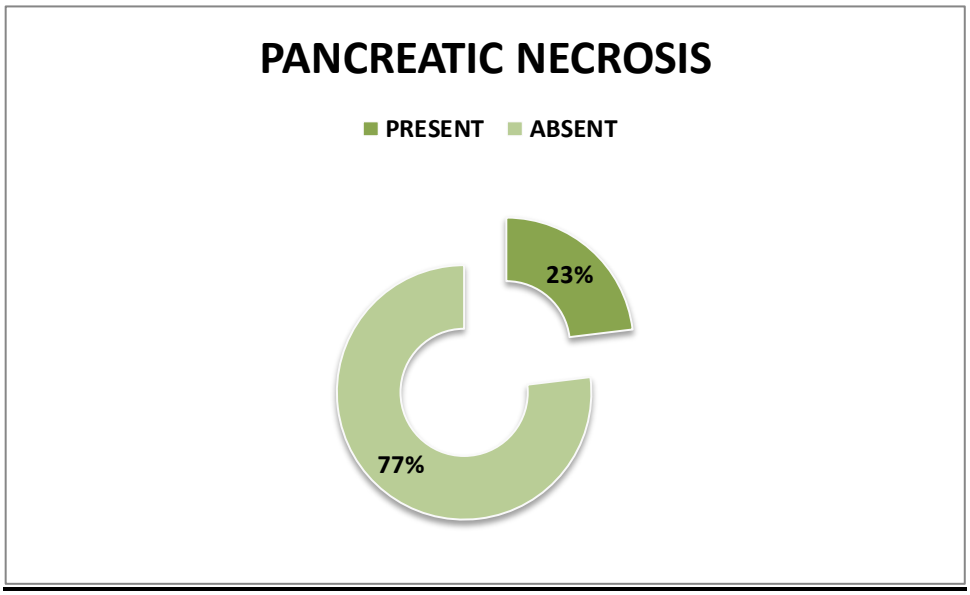

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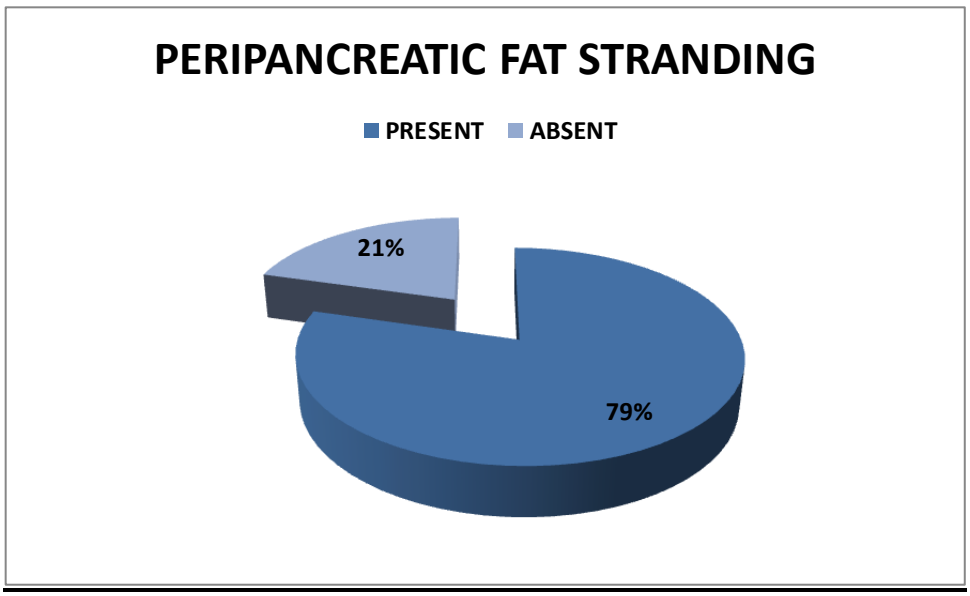

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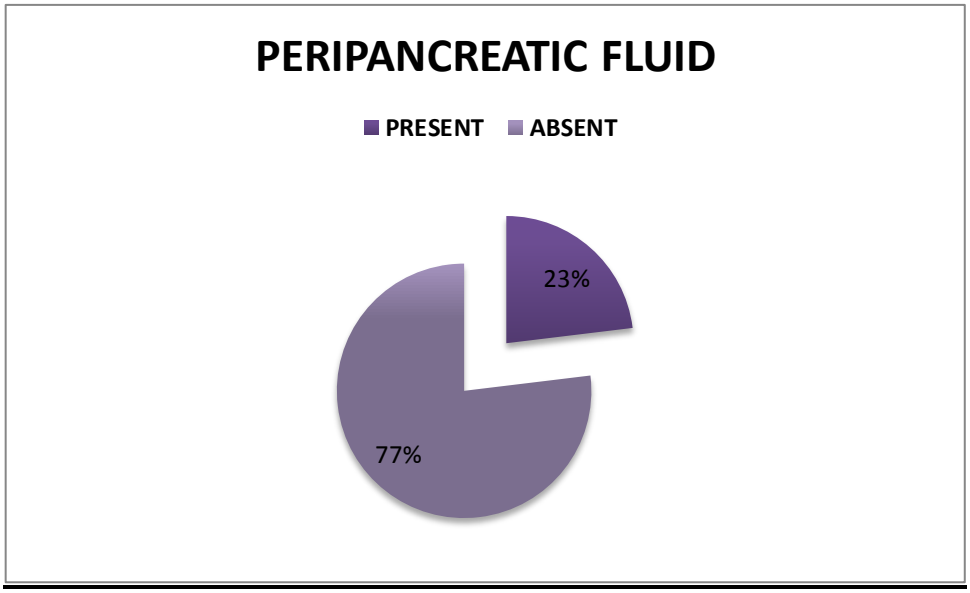

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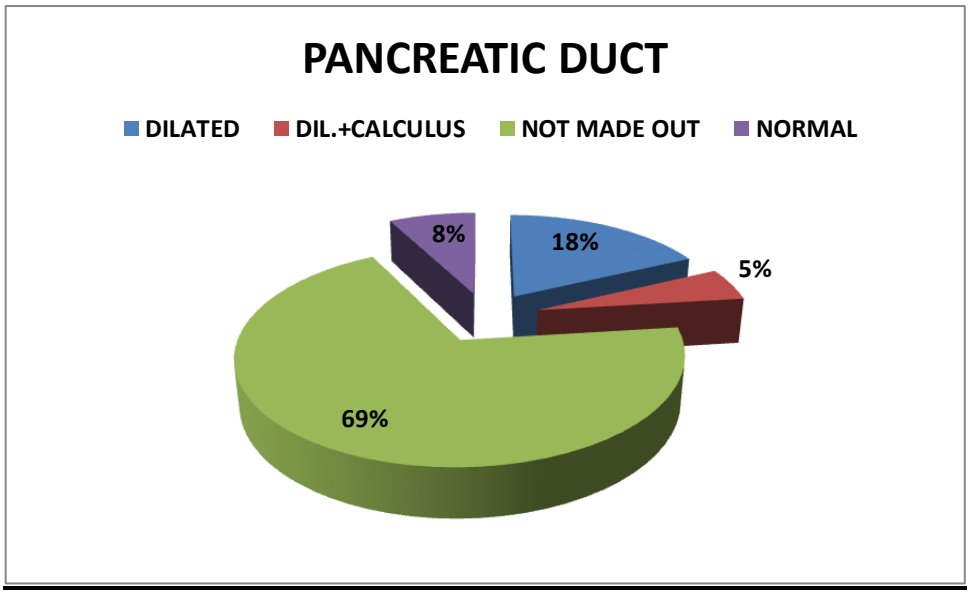

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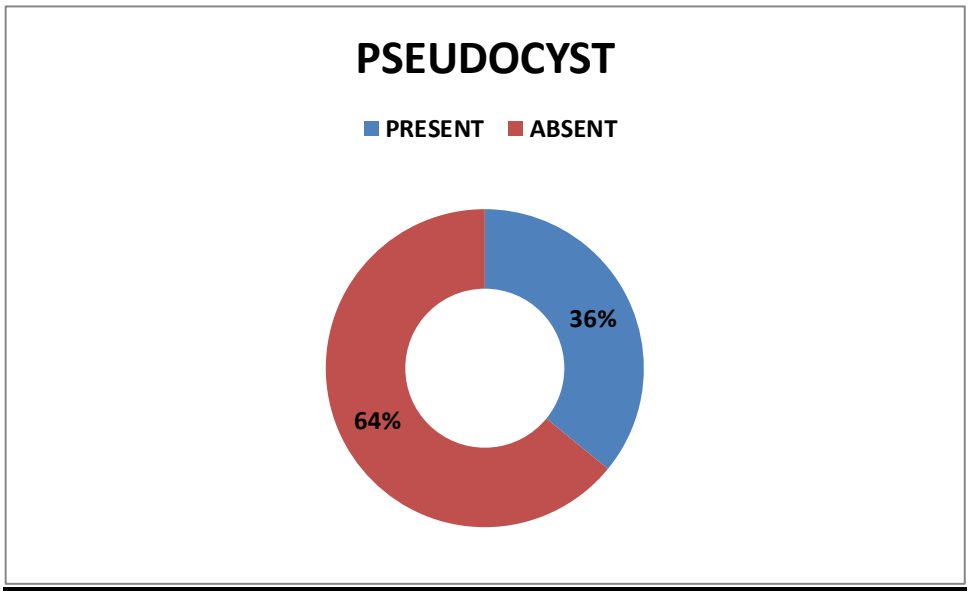

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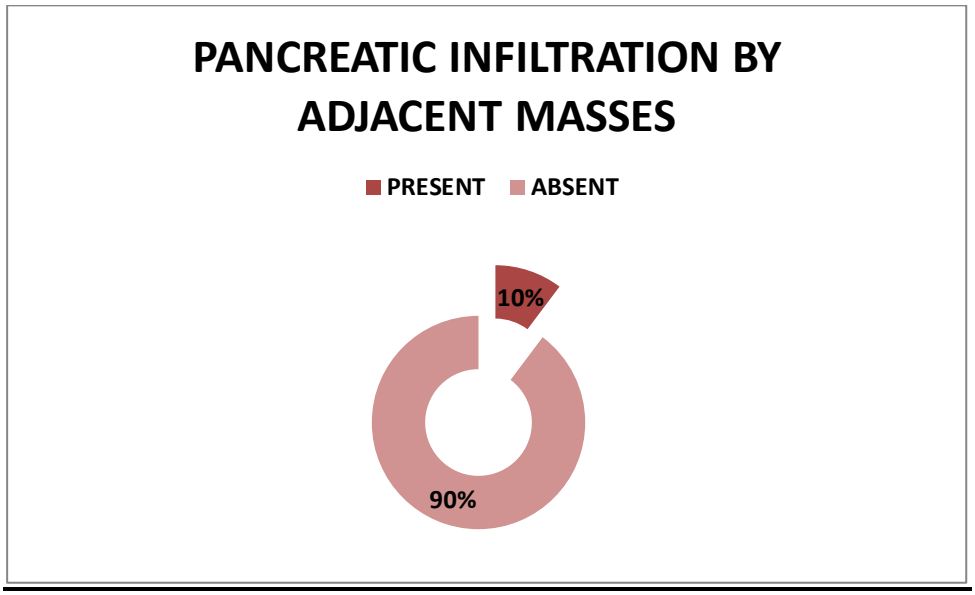

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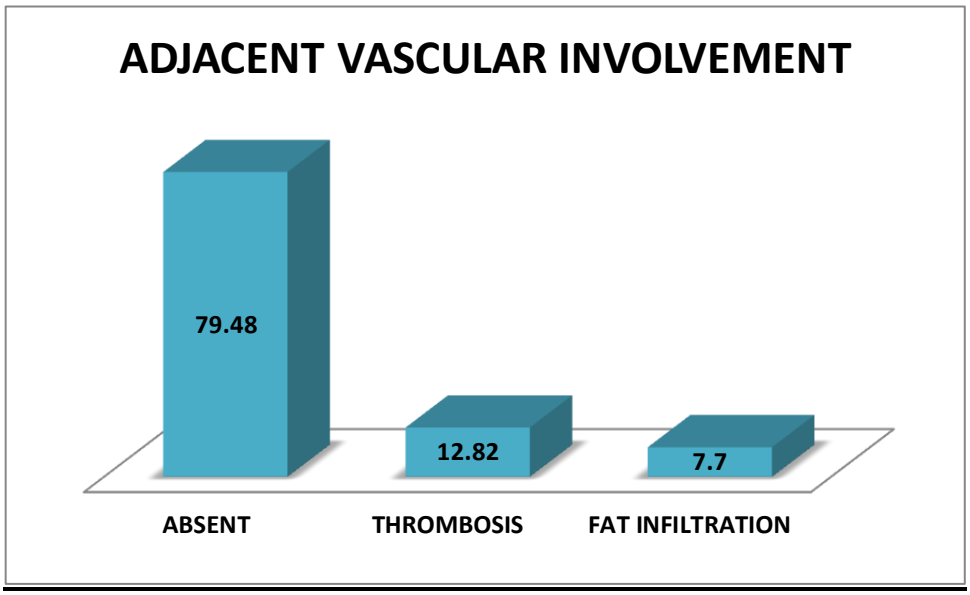

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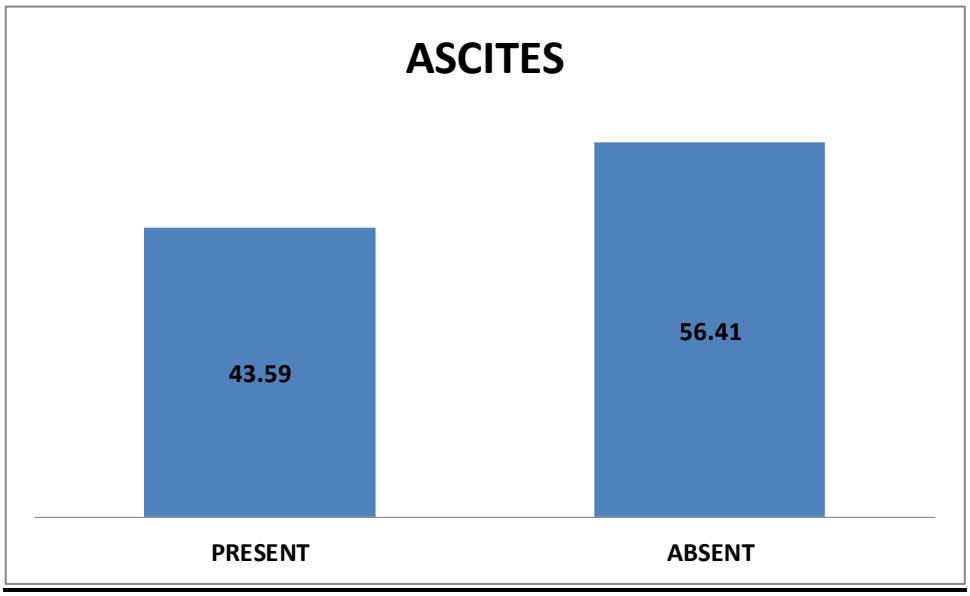

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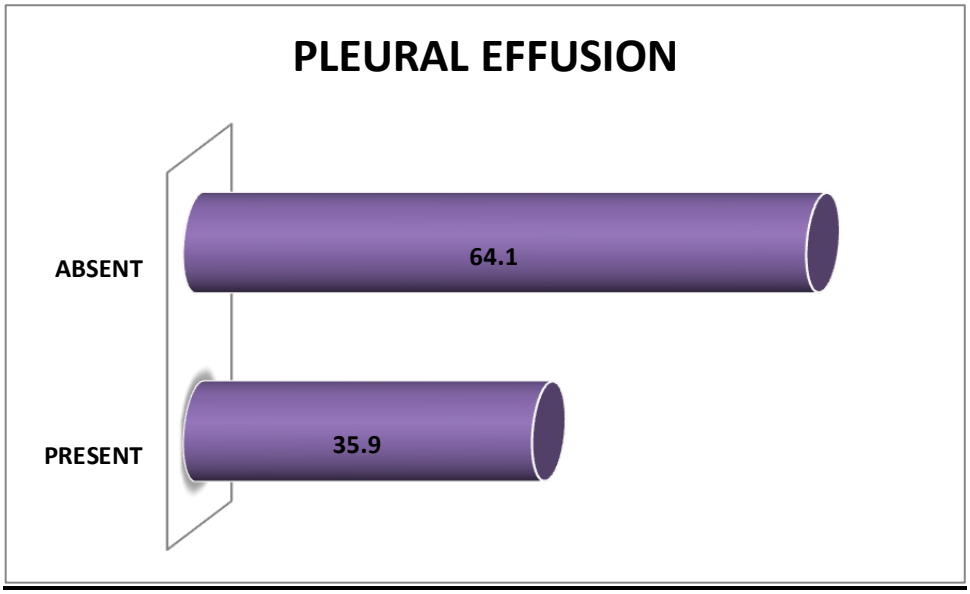

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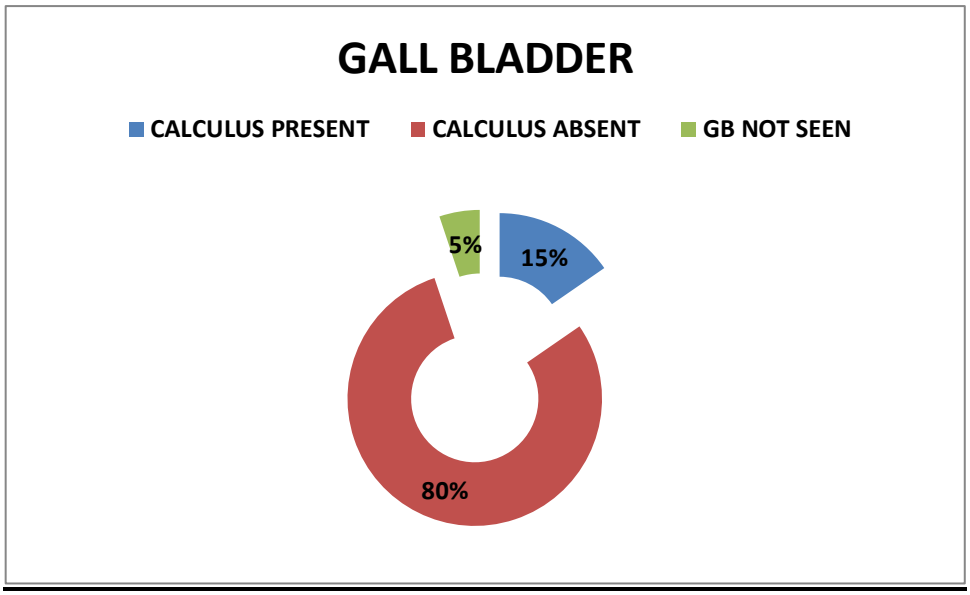

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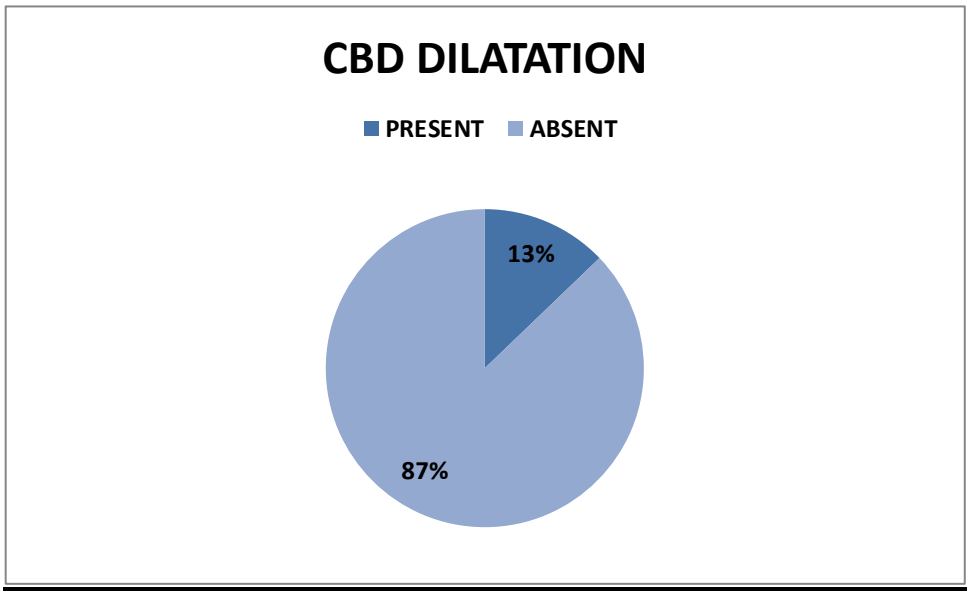

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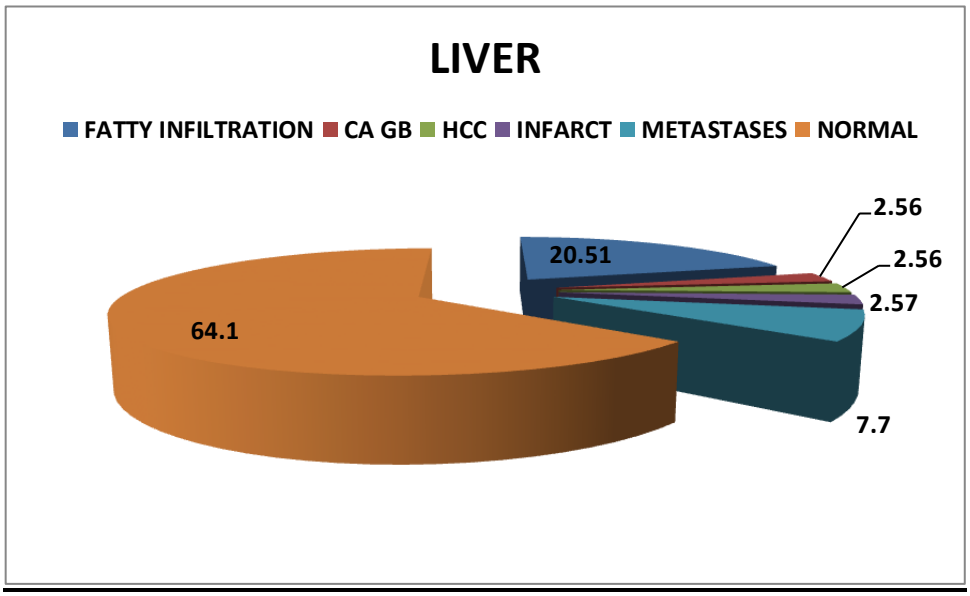


FIGURES

FIGURE # 1 : TOMOGRAPHIC FILM OF CT ABDOMEN

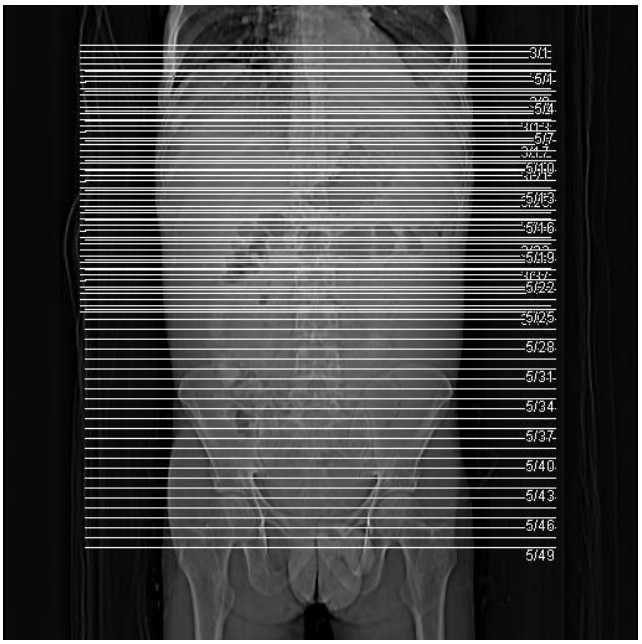


FIGURE # 2 : PLAIN CT ABDOMEN AXIAL SECTION SHOWING CALCIFICATIONS IN PANCREATIC TAIL(ARROW).

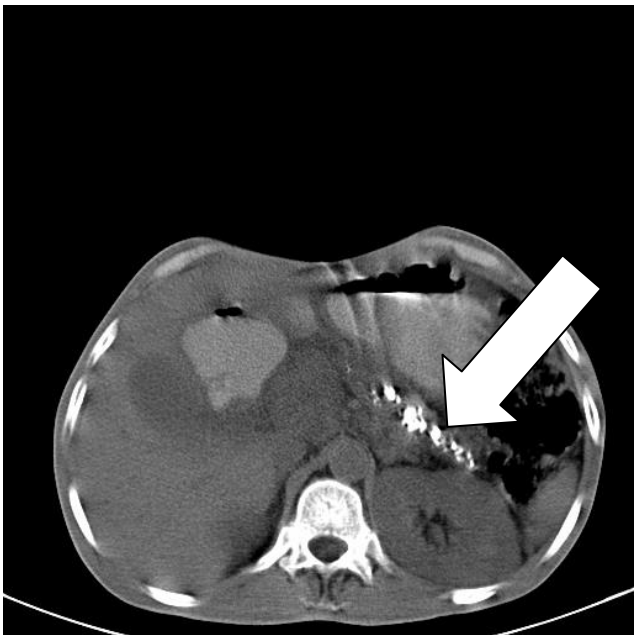


FIGURE # 3 : CT ABDOMEN POST-ORAL CONTRAST AXIAL SECTION SHOWING CALCIFICATIONS IN PANCREATIC HEAD(ARROW).

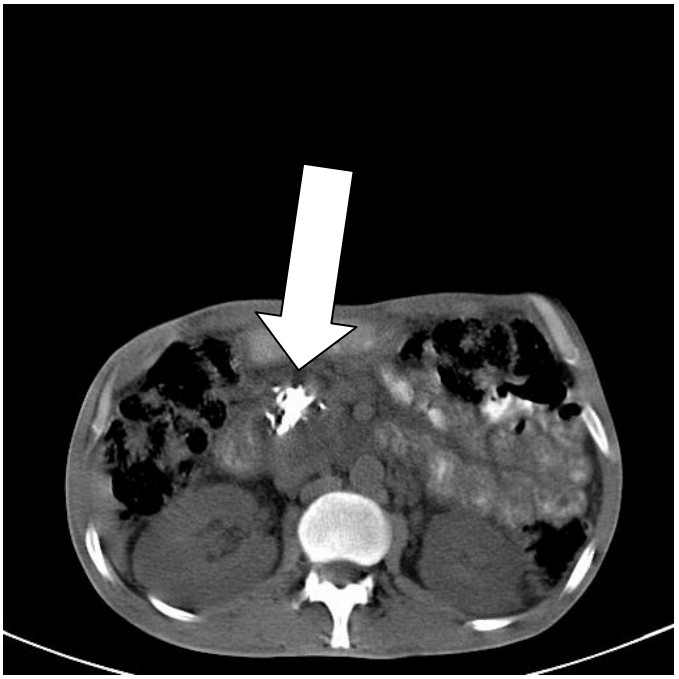


FIGURE # 4 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF DELAYED PHASE SHOWING CALCIFICATIONS IN PANCREAS(ARROW).

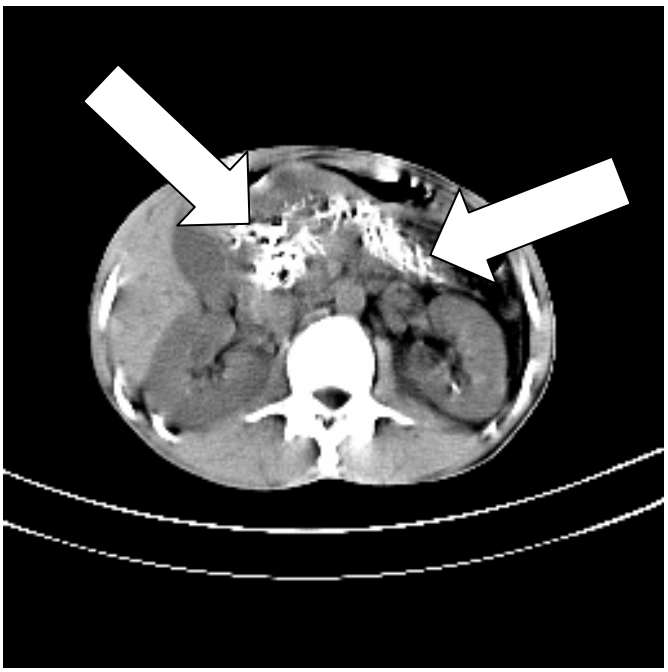


FIGURE # 5 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF ARTERIAL PHASE SHOWING PSEUDOCYST IN PANCREATIC HEAD(ARROW).

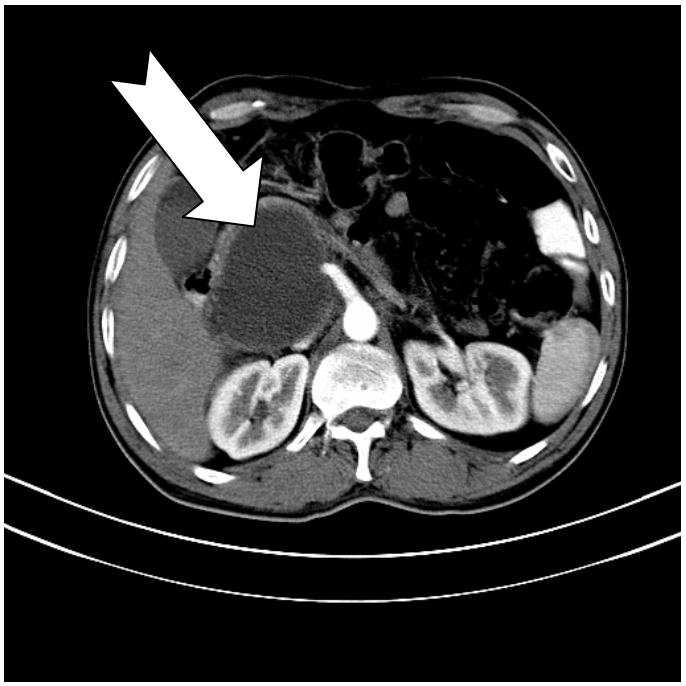


FIGURE # 6 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF ARTERIAL PHASE SHOWING MULTIPLE LOCULATED COLLECTIONS IN PANCREATIC BODY AND TAIL REGION(ARROW).

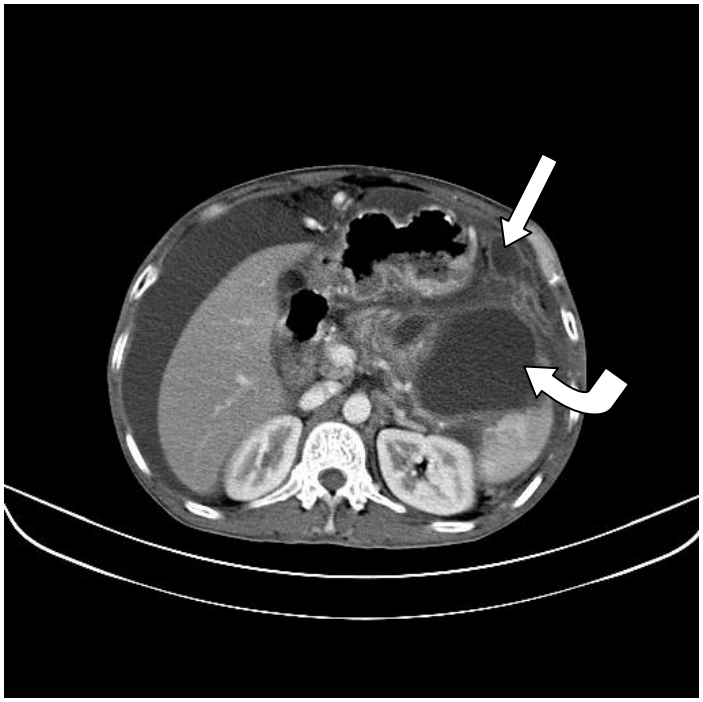


FIGURE # 7 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTIONS OF ARTERIAL PHASE SHOWING MULTILOCULATED COLLECTION IN RELATION TO PANCREATIC HEAD(ARROW).

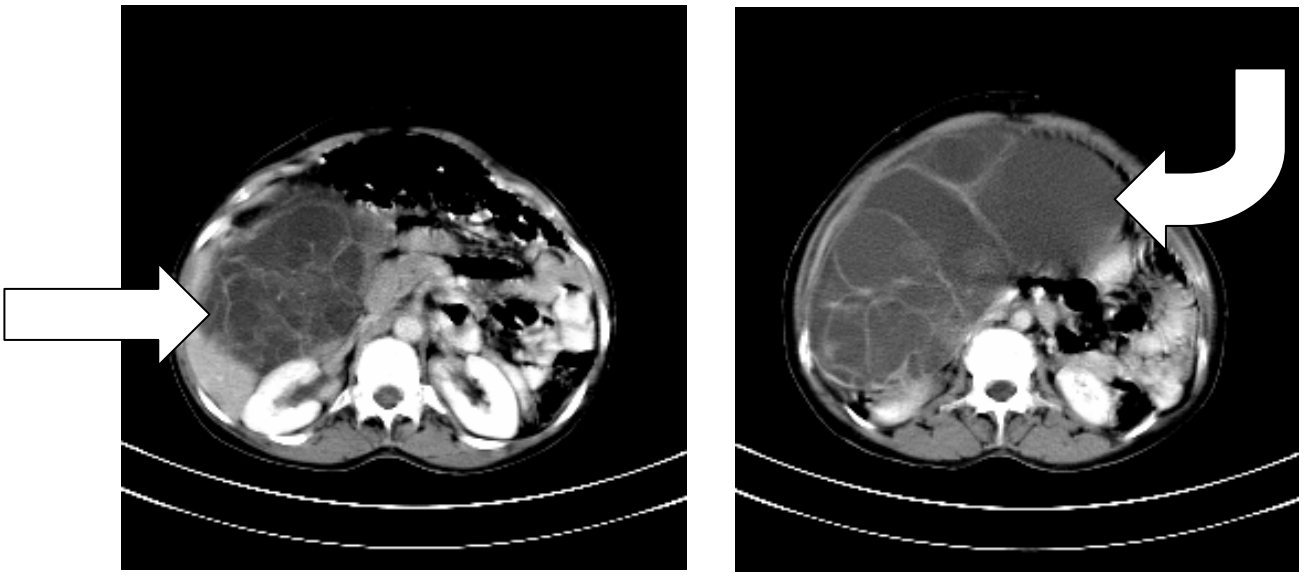


FIGURE # 8 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF VENOUS PHASE SHOWING BULKY PANCREATIC BODY AND TAIL(ARROW).

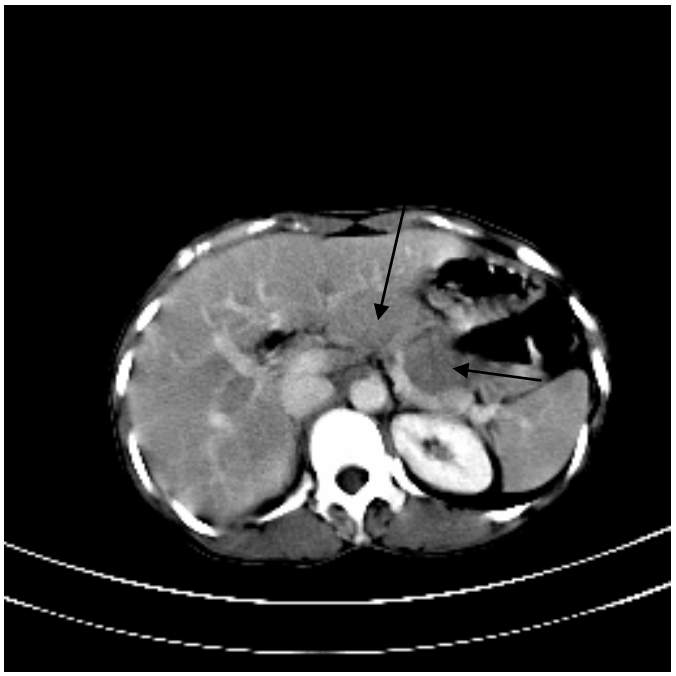


FIGURE # 9 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF DELAYED PHASE SHOWING LOCULATED COLLECTIONS IN PANCREATIC TAIL(ARROW) AND ALSO ASCITES(CURVED ARROW).

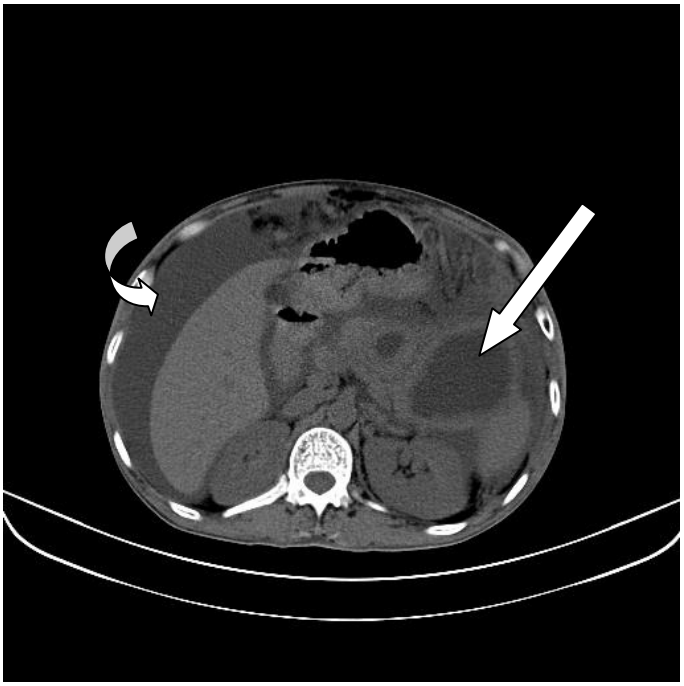


FIGURE # 10 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTIONS OF ARTERIAL PHASE SHOWING MULTILOCULATED HYPODENSE LESION IN PANCREATIC HEAD(ARROW) AND ALSO IHBRD (CURVED ARROW).

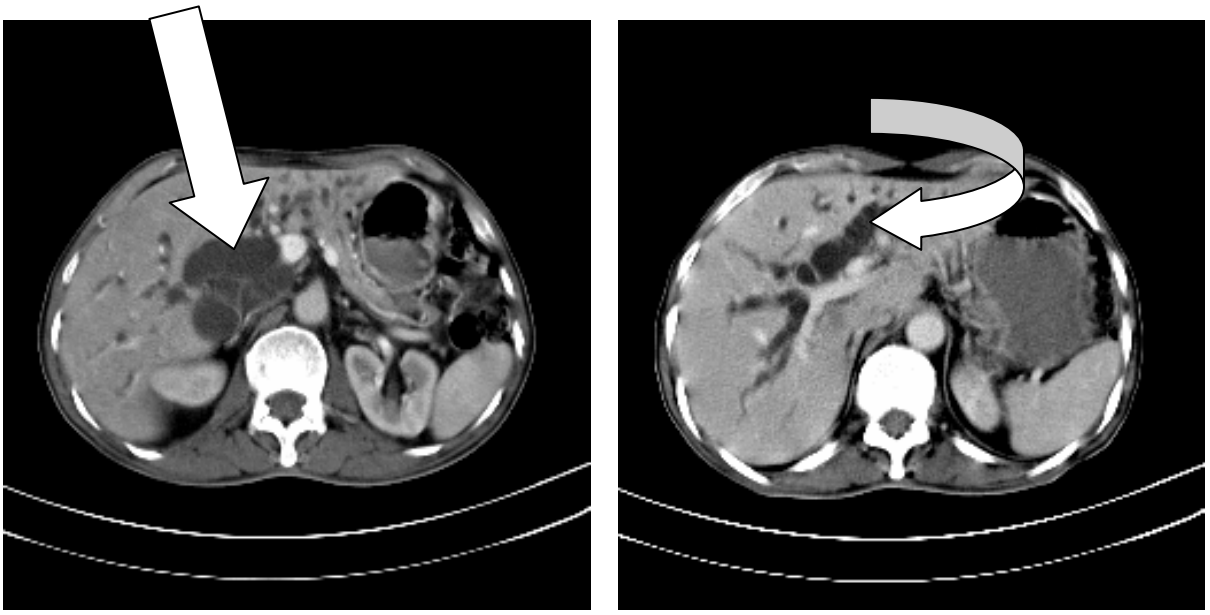


FIGURE # 11 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF VENOUS PHASE SHOWING NECROTIC COMPONENTS IN PANCREATIC HEAD AND BODY(ARROW) WITH LOSS OF PERIPANCREATIC FAT PLANES (CURVED ARROW).

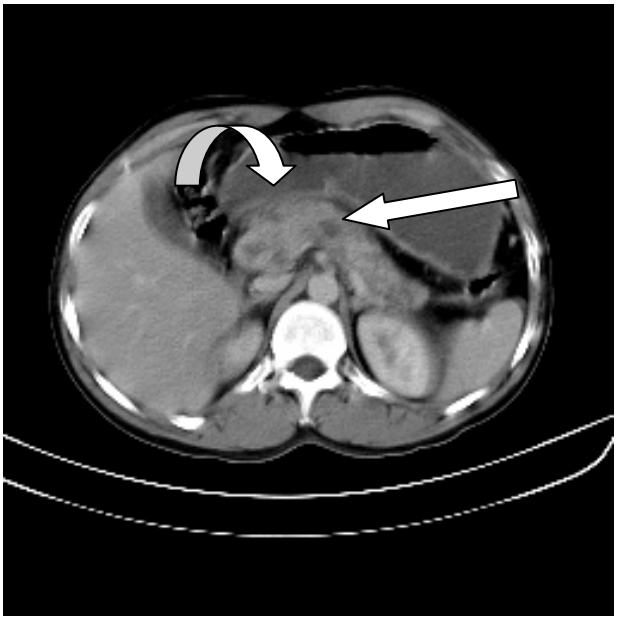


FIGURE # 12 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF ARTERIAL PHASE SHOWING BULKY IRREGULAR PANCREATIC BODY AND TAIL(ARROW) WITH PERIPANCREATIC FAT STRANDINGS IN THE TAIL REGION.

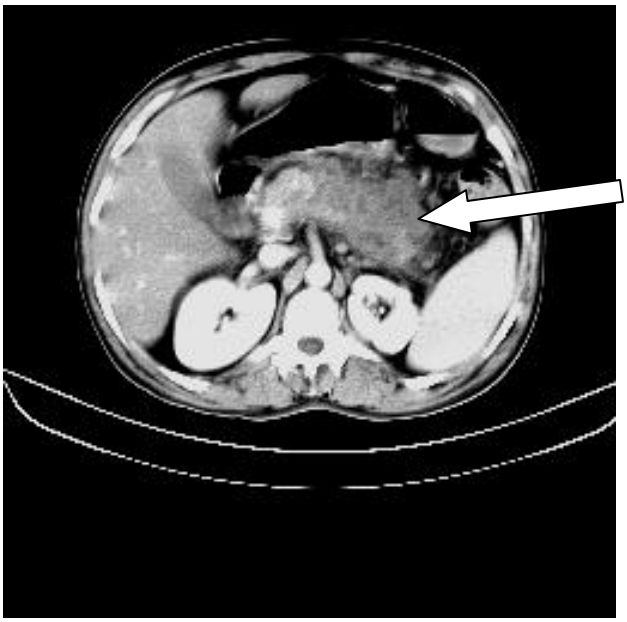


FIGURE # 13 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTIONS SHOWING DIFFUSELY BULKY PANCREAS WITH NECROTIC COMPONENTS WITHIN(SMALL ARROW), LOCULATED COLLECTIONS (ARROWHEAD), IRREGULAR MASS LESION INVOLVING PANCREATIC HEAD(ARROW) WITH PERIPANCREATIC FAT STRANDINGS.

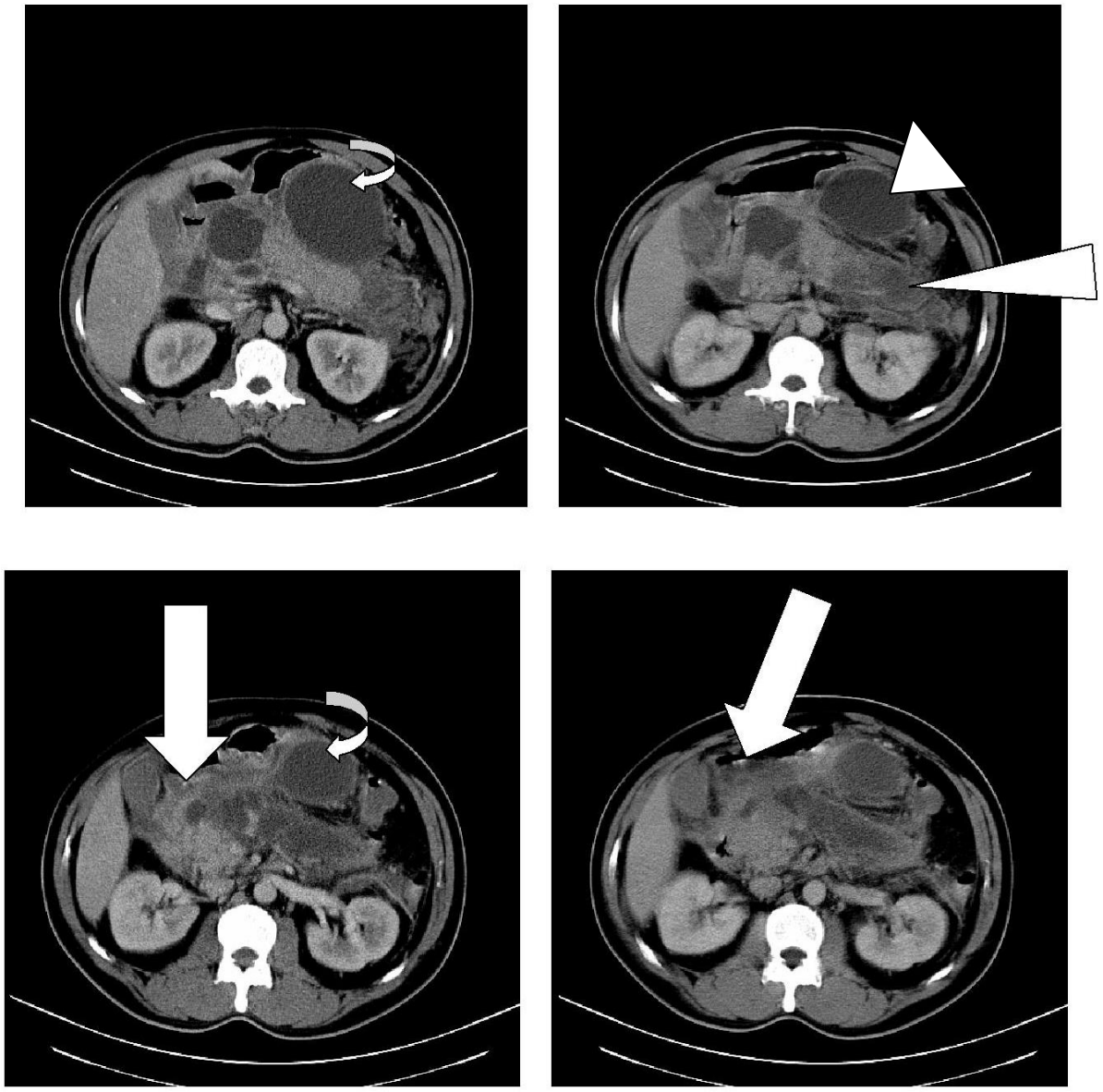


FIGURE # 14 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF ARTERIAL PHASE SHOWING BULKY PANCREATIC BODY AND TAIL WITH A SUBTLE MASS LESION MASS LESION INVOLVING PANCREATIC TAIL(ARROW) WITH PERIPANCREATIC FAT STRANDINGS.

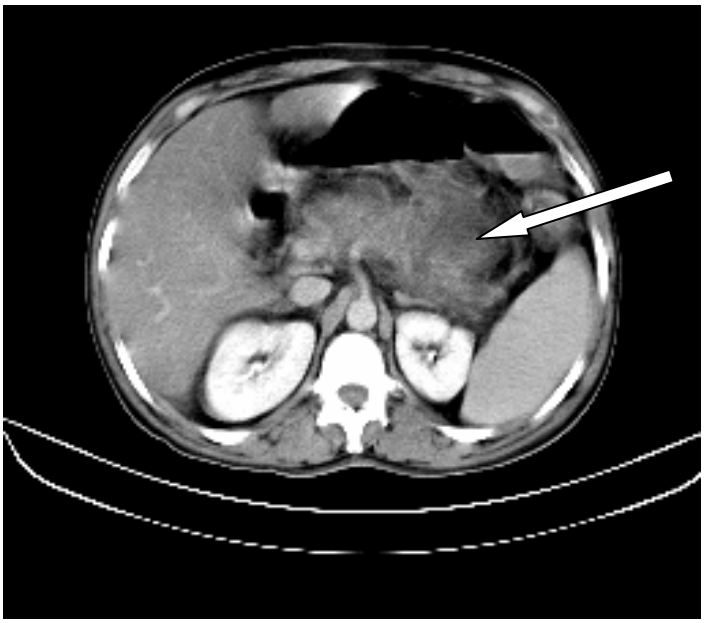


FIGURE # 15 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF DELAYED PHASE SHOWING A SUBTLE MASS LESION MASS LESION INVOLVING PANCREATIC HEAD(ARROW) WITH PERIPANCREATIC FAT STRANDINGS AND WITH CALCIFICATIONS IN THE TAIL REGION(CURVED ARROW).

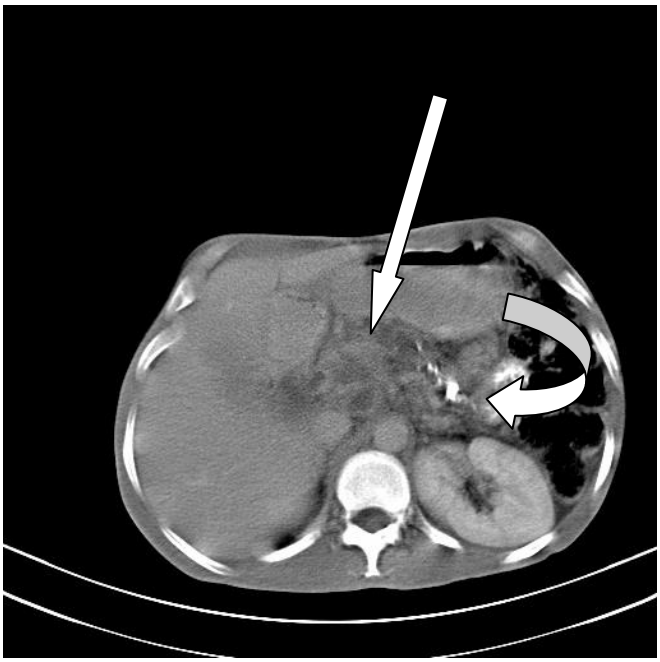


FIGURE # 16: CONTRAST ENHANCED CT ABDOMEN AXIAL SECTION OF PORTAL PHASE SHOWING NON-ENHANCING HYPODENSE LESIONS IN RIGHT LOBE(ARROW).

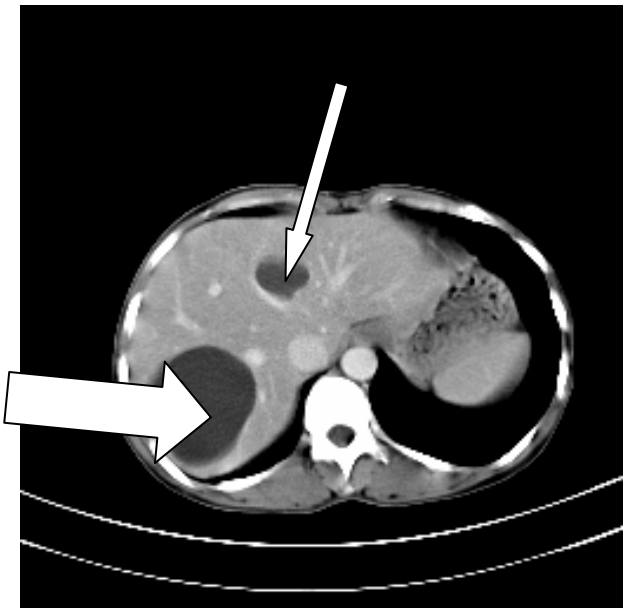
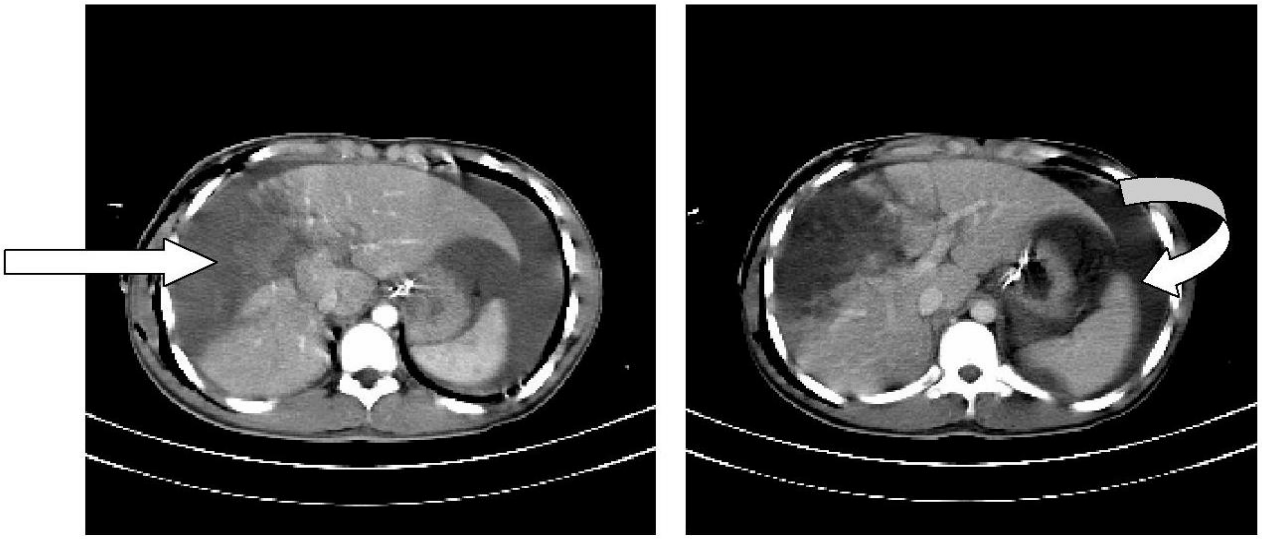


FIGURE # 17 : CONTRAST ENHANCED CT ABDOMEN AXIAL SECTIONS OF ARTERIAL AND VENOUS PHASE SHOWING LARGE ILL-DEFINED NON-ENHANCING HYPODENSE LESION INVOLVING RIGHT LOBE OF LIVER(ARROW) WITH ASCITES(CURVED ARROW



DISCUSSION

Whether the pancreas is adorable or not is a debatable issue; however, advanced cross-sectional imaging has permitted generation of excellent and exquisite images of the pancreas, which in turn has contributed immensely to the assessment of various pancreatic diseases. Ultrasonography has been used to evaluate abdominal pathologies, and its advantages include wide availability, low cost, and lack of radiation. Despite significant improvements and refinements in ultrasound technology, there are still inherent problems when imaging deep in the abdomen, especially in large patients.[21]

Spiral CT scanning brings to CT a concept that forms the basis for all advanced medical imaging. By scanning an entire area or volume, we are no longer limited to single slices of data; instead, we gain a volume of data.[22] This allows us to manipulate and view the additional data in any plane or perspective that may seem of value. Evaluation of the pancreas would seem to be a natural use for spiral CT scanning. This organ lies deep within the abdominal cavity and has a close relationship to key vascular structures such as the portal, splenic, and superior mesenteric veins; the aorta; and the superior mesenteric artery. Opacification of these structures provides important information both in detecting pancreatic abnormalities and in defining their extent. Although the normal pancreas routinely shows only minimal enhancement, the high contrast levels achieved during spiral CT help accentuate the difference between normal pancreas and tumor.[23]

In the present study, 79% of the patient cohort comprises of male population, compared to 69.7% of study done by Kim et al. Mean age group in my study belongs to 36-45 year age group, whereas it was 57 ± 15.7 years in the study done by Kim et

al. 31% of patients in my study were known alcoholic, compared to 36.8% of those in study of Kim et al.[24]

Pancreatic pathology was not diagnosed by ultrasonography in little less than 50% cases.

Plasma levels of the pancreatic enzymes amylase and lipase, while useful diagnostic indicators, have no role in the assessment of disease severity.[25,26] In the current study, serum amylase levels were raised only in 33.3% cases.

Acute pancreatitis is generally classified into mild and severe forms: mild pancreatitis, so-called interstitial or edematous pancreatitis, is associated with minimal organ failure and an uneventful recovery. Severe pancreatitis, also referred to as necrotizing pancreatitis, occurs in approximately 20% of patients and is associated with organ failure or local complications, including necrosis, infection, or pseudocyst formation.[25] The rationale for assessing the severity of acute pancreatitis is mainly practical: mild pancreatitis responds well to supportive therapy, whereas severe pancreatitis requires intensive monitoring and specific therapies and has a more guarded prognosis.[27,28]

Ultrasonography (US) is indicated early in an acute episode of pancreatitis, to help evaluate for the presence of gallbladder and/or common duct stones. US however, has limited applications in the early staging of the disease. Visualization of the pancreas is often impaired because of overlying bowel gas, and the detection of intraparenchymal and retroperitoneal fluid collections correlates poorly with pancreatic necrosis. Abnormal US findings are seen in 33%–90% of patients with acute pancreatitis. A diffusely enlarged and hypoechoic gland is consistent with interstitial edema, while extrapancreatic fluid collections (lesser sac, anterior pararenal space) are detected in patients with severe disease.[29,30] Pancreatic pathology was not diagnosed by

ultrasonography in little less than 50% cases, and acute pancreatitis could be diagnosed only in 25.6% cases.

Helical CT allows quick and accurate diagnosis and staging of pancreatitis.[31] CT can assess the degree of involvement and enables detection of complications including development of pseudocysts, abscess, necrosis, hemorrhage, and vascular occlusion.[32]

Most cases of acute pancreatitis are diagnosed clinically and do not rely on imaging.[33] However, often the history and presentation of the patient may not be straight forward, and a reliable imaging modality is needed to establish the diagnosis.[34-37] For nearly two decades, CT has been the imaging procedure of choice in the initial evaluation and follow-up patients with suspected pancreatitis. The sensitivity of helical CT for the diagnosis of acute pancreatitis is not known, especially in very mild cases, but it is reasonable to assume that a good-quality contrast-enhanced helical CT scan will demonstrate definite changes in the vast majority of patients with moderate to severe involvement. Helical CT depicts all but the mildest forms of acute pancreatitis, demonstrates most major complications, and can help guide percutaneous aspirations and drainages. Helical CT is also indicated when there is failure of clinical response to treatment. In addition, helical CT can confidently detect other causes of abdominal pain in patients initially thought to have symptoms of acute pancreatitis.

Diagnostic evaluation of the pancreas, with either single or multidetector row helical CT, has always required careful attention to study technique and protocols. Dual-phase helical scanning during both the arterial and portal venous phases has several advantages when imaging the pancreas. The arterial phase is superior to the portal venous phase visualization mesenteric and peripancreatic arteries. Opacification of the splenic vein, superior mesenteric vein, and portal venous confluence is better obtained

during the more delayed portal venous phase. The pancreatic parenchyma is best seen in the parenchymal phase, which is around 50 seconds after the start of the injection.[38-39]

The extent of pancreatic enhancement will vary based on a number of technical parameters and study design features. Kim et al.[26] found the optimal time for imaging for necrosis is in the venous phase or 50-60 seconds after contrast injection. Injection rate should be in the 3-cc/sec range. The pancreatic enhancement is normally in the 100-150 HU range. Lack of enhancement of less than 30 HU is representative of decreased blood perfusion of the gland and correlated well with necrosis. Caution in defining pancreatic necrosis is important as areas of peripancreatic fluid can simulate areas of necrosis. Pancreatic necrosis is ideally detected on scans performed 48-72 hours after the onset of an attack of acute pancreatitis. Scans done within the first 24 hours may be falsely negative or equivocal. Although scans are commonly done at time of admission the need for a second study should be kept in mind for patients without rapid improvement initially. Lack of contrast enhancement or minimal contrast enhancement of less than 30 HU of a portion of the pancreas or of the entire pancreas indicates decreased blood perfusion (ischemia) and correlates with the development of necrosis. In this regard, however, several factors and potential pitfalls should be kept in mind. ***First***, enhancement values of the pancreas during examination with a bolus of contrast material can be substantially decreased in healthy patients with fatty infiltration of the pancreas, as well as in patients with interstitial pancreatitis, due to parenchymal edema. Furthermore, a slight variation in the enhancement values of the head, body, and tail of the pancreas (usually <30 HU) is sometimes seen in healthy individuals. Pancreatic necrosis should not be diagnosed in these cases unless a localized or diffuse change in the texture of the gland is

recognized. Whether presumed pancreatic ischemia manifesting as areas of decreased attenuation on the initial CT study is a reversible or inconsequential phenomenon is not known but is subject to speculation. Additionally, small intrapancreatic fluid collections sometimes seen in acute pancreatitis should not be confused with small areas of parenchymal necrosis. The distinction is difficult and sometimes impossible to make unless previous or follow-up CT images are available for review. ***Second***, it has been determined that pancreatic necrosis develops early, within the first 24–48 hours after the onset of clinical symptoms. CT performed during the initial 12 hours may show only equivocal findings, with a slight heterogeneous decrease in attenuation of the pancreas (ischemia) but a normal parenchymal texture. When pancreatic necrosis develops, zones of tissue liquefaction become better defined and more easily recognized 2–3 days after the initial onset. Thus CT scans obtained 3 days after clinical onset yield higher accuracy in the depiction of necrotizing pancreatitis and in the discrimination of normal variants or equivocal zones of ischemia from pancreatic necrosis. This phenomenon is probably responsible for the few reported cases of late development of necrosis[24] or enlarging areas of pancreatic necrosis on follow-up CT scans.[40] When the clinical diagnosis of pancreatitis is in doubt, an initial early CT study is used to confirm the clinical suspicion or to help detect alternative acute abdominal conditions that mimic acute pancreatitis. For staging purposes, however, more reliable results are obtained with the use of bolus contrast-enhanced CT performed 48–72 hours after the onset of an acute attack of pancreatitis. ***Third***, extravasation of activated pancreatic enzymes induces the development of retroperitoneal fat necrosis, a common phenomenon that occurs in patients with or without parenchymal necrosis.[41,42] The retroperitoneal chemical injury results in multiple areas of fat necrosis, which interfere with fluid absorption and favor

secondary bacterial contamination. This phenomenon probably explains the 22% incidence of local complications in patients without pancreatic necrosis but with peripancreatic fluid collections.[24] CT cannot be used to help reliably diagnose, nor can it help accurately quantify, retroperitoneal fat necrosis. It has been suggested, therefore, that in clinical practice all heterogeneous peripancreatic collections should be considered areas of fat necrosis until proven otherwise.[43] Necrotic fatty tissue can manifest as small heterogeneous collections on follow-up CT scans. Infection cannot be excluded in these patchy areas of fat necrosis.

In this present study, necrosis was found in only 23% of the cases with maximum acute pancreatitis cases being grade C, as compared to 80% and combined grade D+E, respectively in a study done by Balthazar in 2002.[44]

Despite the advances in cross-sectional imaging, the characterization of pancreatic masses, and particularly of cystic lesions, is a common problem.

Pseudocysts represent the most commonly encountered pancreatic cystic lesions, and misdiagnosis and mismanagement still occur frequently.[45] Pseudocysts typically occur with acute pancreatitis or may develop insidiously in the setting of chronic pancreatitis.

Since there are patients (although very few) with pancreatitis who do not have abdominal pain[46] or an increase in enzyme levels[47], pseudocyst should be considered in the differential diagnosis of unilocular cystic pancreatic lesions.

CT provides limited assistance in the differentiation between serous and mucinous neoplasms. Cohen-Scali et al. found that CT was helpful for differentiating serous oligocystic adenoma and mucinous cystadenoma, and they described specific CT

findings indicating serous oligocystic adenoma—namely, location in the pancreatic head, a lobulated contour, and the absence of wall enhancement.[31]

Pseudocysts and chronic pancreatitis can result in vascular occlusion of the splenic vein, superior mesenteric vein, and/or portal vein. Splenic vein thrombosis is most common, and results in complications such as gastric varices. Helical CT can accurately depict sites of vascular thrombosis and demonstrate collateral vascular pathways.

Classically, pancreatitis is a disease process where spread is not limited by adjacent organs, mesenteries or the omentum. While pancreatitis most commonly involves the pararenal spaces and lesser sac it can extend to and involve adjacent organs. For example, renal involvement is typically inflammatory extension into the anterior and sometimes posterior pararenal space. The left pararenal space is most commonly involved except when the cause of pancreatitis is post procedural like post-ERCP pancreatitis where right-sided involvement is more common. On occasion a pseudocyst can tract into the perirenal space and even beneath the renal capsule. This pseudocyst can at times, on select images, even simulate a renal cyst. When pancreatic fluid tracts beneath the capsule it can result in a Page kidney due to compressive forces on the renal parenchyma. Percutaneous drainage may be needed. Other unusual complications include renal vascular abnormalities such as narrowing of the renal vein, renal vein thrombosis, perirenal varices and asymmetric renal enhancement due to extrinsic pressure on one of the renal arteries. In one series of acute pancreatitis 7% of cases had renal and perirenal complications.[48]

Splenic involvement by pancreatitis is not uncommon especially when one considers the intimate relationship of the tail of the pancreas and the splenic hilum.[49-50] In addition to vascular complications ranging from splenic artery pseudoaneurysm to splenic vein occlusion, pseudocysts may tract deep into the spleen. This can result in complications ranging form intrasplenic pseudocysts to splenic infarction to intrasplenic hemorrhage. Intrasplenic pseudocyst can weaken the pancreatic gland so that with even minor trauma splenic rupture may occur. Splenic abscess may develop as a rare complication of pancreatitis. Although rare (frequency of 1-5%) splenic involvement by pancreatitis can be life threatening and CT can be used as a guide to monitor these patients and to determine when aggressive intervention is necessary to avoid catastrophic clinical outcomes. Portal vein thrombosis is another complication of acute pancreatitis.

Computed tomography (CT) was the most effective method of detecting neoplastic and inflammatory disease. It was found to be a reliable, often specific, and noninvasive method for detecting pancreatic neoplasms and pseudocysts.[32] The full extent of the disease process, including involvement of the retroperitoneum and metastasis to the liver, was visualized with one examination. Calcification and cystic collections associated with pancreatitis were also clearly seen.[51] The use of CT has resulted in the diminished use of pancreatic angiography.[32]

In the current study, one case of chronic pancreatitis underwent malignant transformation within the course of the study. The patient initially was diagnosed as chronic pancreatitis with associated pseudocyst formation, which in due course of time showed a cystic mass with calcifications in the head region of pancreas with multiple inhomogenously-enhancing mixed density lesions in liver with associated thrombosis of portal vein and common hepatic artery. It was proved by USG-guided

FNAC to be adenocarcinoma of head of pancreas with adenocarcinomatous metastatic deposits in the liver.

Diagnostic accuracy of modern ultrasonographic techniques is comparable to helical CT in detecting pancreatic carcinoma. 3-D is superior to the other methods in local staging (T1-T4) of pancreatic cancer. The best method of assessing regional lymph node metastases is helical computed tomography. All imaging methods enable the correct diagnosis of neoplastic dissemination. Modern ultrasonographic techniques and CT are useful to predict the possibility of resection. Because of their relatively low cost and accessibility, modern ultrasonographic modalities are recommended as diagnostic tools in detecting and staging pancreatic carcinoma. It is important, however, that examinations should be performed and evaluated by experienced and competent physicians or techniques.[52]

SUMMARY

- This study was undertaken to evaluate the pancreatic lesions by CT scan in adult patients.
- The study includes 39 patients referred to R.L. Jalappa Hospital and Research Center for CT abdomen contrast study with suspicion of pancreatic pathology.
- Serum amylase and history of alcoholism was not found to be much significant to predict pancreatic pathology in my study.
- Plain and contrast study of abdomen was undertaken with SIEMENS Esprit single slice Spiral CT unit.
- Contrast-enhanced CT abdomen study showed acute inflammatory changes in 21 cases(53.8 %), pseudopancreatic cyst in 14 cases(35.89%), chronic pancreatitis in 5 cases(12.8%), isolated pseudopancreatic cyst in 3 cases(7.69%), tumor in 6 cases(15.38%), and post-road-traffic-accident contusion in 1 case(2.56%).
- Maximum of the acute pancreatitis cases belong to Balthazar grade C (42.86%).
- Pancreas was found to be bulky, altered attenuation and peripancreatic fat strandings in significant number of cases, but necrosis, parenchymal or ductal calcifications, dilated duct, peripancreatic fluid, pancreatic infiltration by adjacent tumors, and adjacent vascular thrombosis/fat infiltration was found in only few cases.
- Ascites and pleural effusion were noted in quite a number of cases. Diffuse fatty infiltration of liver, gallbladder calculus and common bile duct dilatation was seen in only few cases.

CONCLUSION

Helical Computed Tomography of the abdomen with high dose contrast has improved the visualization of pancreas, more so in cases where pancreas is obscured on ultrasonography by poor acoustic window owing to excessive bowel gas.

It not only enables proper assessment of pancreatic size and morphology, but also hepatobiliary system, spleen, bowel loops, and even surrounding vasculature bed and the celiac and pre/para aortic group of lymphnodes can be assessed and commented upon in the same setting. Infiltration of the peripancreatic structures and possible pancreatic infiltration by other tumors can be well appreciated.

CT plays an important role in grading of pancreatitis and trauma cases, which correlate fairly well with the clinical outcome and also decides the appropriate mode of treatment.

Serum amylase levels and alcohol history were not found to be “the diagnostic criteria” for predicting pancreatic pathology in the current study.

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PROFORMA

NAME ---

HOSPITAL NO. ---

AGE --- YEARS

IP/OP

SEX --- MALE

RELIGION ---

CT SCAN NO. ---

ADDRESS ---

CLINICAL DATA ---

PAST HISTORY –

CLINICAL EXAMINATION ---

PERSONAL HISTORY ---

CLINICAL DIAGNOSIS ---

INVESTIGATIONS --SERUM AMYLASE ---

USG ABDOMEN ---

USG DIAGNOSIS –

CT SCAN ABDOMEN CONTRAST STUDY ----

CT DIAGNOSIS

	ULTRASONOGRAPHY		COMPUTED TOMOGRAPHY	
ORGANS	**NORMAL/ NIL SIGNIFICANT**	**ABNORMAL**	**NORMAL/ NIL SIGNIFICANT**	**ABNORMAL**
LIVER				
GALL BLADDER				
SPLEEN				
FREE FLUID				
ENCYSTED FLUID				
PANCREAS **SIZE** **MORPHOLOGY** **NECROSIS** **FAT STRANDING** **CALCIFICATIONS** **PERIPANCREATIC FLUID** **PANCREATIC DUCT**				

INTRODUCTION

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ANNEXURES

ABBREVIATIONS USED IN MASTER CHART

Sr. N. – Serial number

ALC. – Alcoholic

CL.Δ -- Clinical diagnosis

S.AMYL – Serum amylase levels

USG Δ -- Ultrasound diagnosis

P-OBSC --- Pancreas obscured by bowel gas

CT Δ -- CT diagnosis

NECR. --- Pancreatic necrosis

CAL. --- Pancreatic calcifications

PERIP. FL. --- Peripancreatic fluid

P.DUCT --- Pancreatic duct

PSEUDO ---- Pseudopancreatic cyst

ASCIT. --- Ascites

PL.EFF. --- Pleural effusion

GB CAL ---- Gall bladder calculus

P.INFILTR --- Pancreatic infiltration by other mass lesions

AD.VASC.INVOLVT. --- Adjacent vasculature involvement

CHLNG CA – Cholangiocarcinoma

LYMPH – Lymphoma

RTP TU – Retroperitoneal tumor

CEL & SP. A – Celiac trunk and splenic artery

HEP A – Heaptic artery

PV – Portal vein

ND – Not done