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The role of diagnostic radiology in pancreatitis

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Abstract

Acute pancreatitis is a frequent inflammatory and necrotic process of pancreas and peripancreatic field. To detect the presence of infected or sterile necrotic components and hemorrhage of the pancreatic paranchyma is important for therapeutic approach. Chronic pancreatitis is characterized by irreversible exocrine dysfunction, progressive loss of pancreatic tissue and morphological changes of the pancreatic canal. Imaging modalities play a primary role in the management of both acute and cronic pancreatitis. CT and MR imaging confirm the diagnosis and detect the severity of disease. In chronic pancreatitis, MRCP after Secretin administration, Spiral CT and endoscopic US seems to replace diagnostic ERCP. However differentiation of pseudotumor of chronic pancreatitis from the pancreatic carcinoma is difficult with either imaging modalities. © 2001 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Pancreas; Acute pancreatitis; Chronic Pancreatitis; Computed tomography; Magnetic Resonance Imaging

1. Acute pancreatitis

Acute pancreatitis is one of the most complex and dramatic pathology of the abdomen, requiring urgent therapy. The process may include supuration, necrosis and hemorrhage of pancreatic tissue [1,2]. The most common etiologic factors of acute pancreatitis are cholelithiasis and alcohol abuse. Other causes include metabolic, iatrogenic, vascular, infectious, and toxic factors. Although not significant, it has been shown that, in ERCP, the occurrence of acute pancreatitis increases when isotonic contrast agents with high osmolarity are used compared with those with low osmolarity [3].

The clinical spectrum of acute pancreatitis varies from mild to fulminant disease, which may lead to death [2]. The degree, stage and preferable therapy are based on the early evaluation of first exacerbation of the disease [4]. Diagnosis can be obtained commonly, with serum amylase and lipase levels. With the advent of cross-sectional imaging modalities, the imaging approach to acute pancreatitis has also changed. Imaging techniques performed on admission will focus on confirmation of the diagnosis, identification of the cause of pancreatitis, and assessment of the extent and complications of disease [5,6].

The wide variations in clinical findings require different therapeutic methods and multidisciplinary approach. A group of 40 experts on anatomy, internal medicine, gastroenterology, pathology, radiology, and surgery has made the last clinically based classification of acute pancreatitis in the Atlanta Symposium, 1992. According to this classification, acute pancreatitis is classified as mild and severe acute pancreatitis in the light of clinical and laboratory findings, and severity of pathologic changes [7].

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1.1. Mild acute pancreatitis

In the mild form of acute pancreatitis, the interstitial edema of the pancreatic tissue predominates in association with only a few foci of necrosis [8]. Morphological changes of this form are diffuse enlargement of the gland (Fig. 1) with or without heterogenicity, peripancreatic fluid collections (Fig. 2), and thickening of the anterior pararenal fascial plane predominantly on the left.

1.2. Severe acute pancreatitis

In the severe form of acute pancreatitis, extensive pancreatic necrosis and inflammation of the peripancreatic fat tissues are present that may evolve towards the formation of fluid collections [8]. In this form, morphological changes can be described as follows:

- Pancreatic tissue necrosis.
- Pancreatic or peripancreatic abscess.
- Parenchymal hemorrhage.

Therapeutic approach in acute pancreatitis depends on the severity of the first attack. The severity of the acute attack and prognosis are appraised by the quantification system, which uses clinical and laboratory parameters. The most commonly used criteria are the Ranson criteria (Table 1) [9] and Apache II criteria [10]. Patients with severe acute pancreatitis exceed three Ranson criteria at 48 h, or five Apache II criteria at any time during the disease. The severity of acute pancreatitis is directly related to the extent and intensity of inflammation [8].

1.3. Complications of acute pancreatitis

In the Atlanta Symposium, the terminology of complications was defined on the basis of clinical and morphological nomenclature [7].

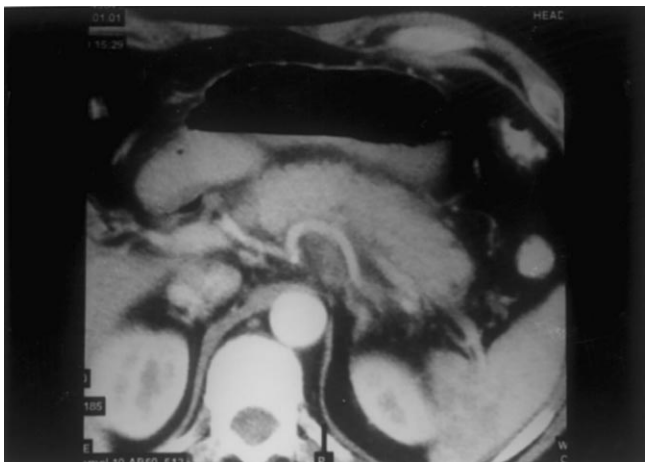

Fig. 1. Acute pancreatitis (mild form). Arterial phase spiral CT. Diffuse enlargement of pancreas without fluid accumulation.

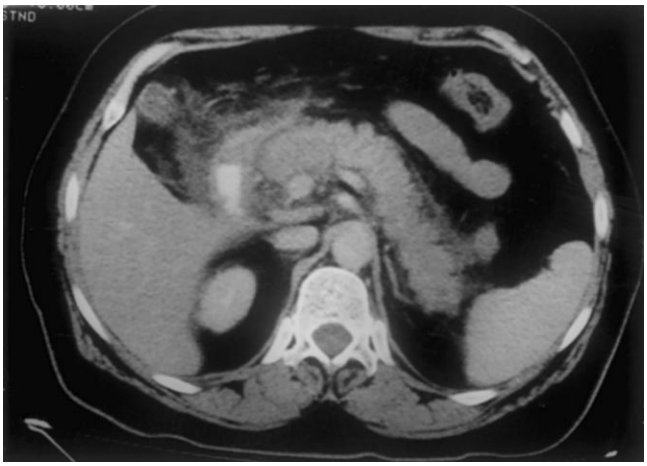

Fig. 2. Acute pancreatitis (mild form). Diffuse edema of pancreatic gland with peripancreatic fluid.

1.3.1. Pancreatic necrosis

Pancreatic gland necrosis is defined as focal or diffuse areas of nonviable parenchyma, which is typically associated with peripancreatic fat necrosis (Fig. 3a–f).

1.3.2. Peripancreatic fluid collections

Acute fluid collections are collections of enzyme-rich pancreatic juice that occur early in the course of acute pancreatitis (Fig. 3e). These collections develop in about 40–50% of patients with acute pancreatitis [11]. Usually, only small fluid collections resolve spontaneously within 4–6 weeks.

1.3.3. Pseudocyst

If peripancreatic fluid collections remain without resolvement, they may evolve into pancreatic pseudocyst. These are round, encapsulated (with fibrous-tissue wall) collections of pancreatic fluid (Fig. 4a, b and Fig. 5).

1.3.4. Pancreatic abscesses

They correspond to circumscribed intra-abdominal collections of pus located in peripancreatic space (Fig.

Table 1
Ranson criteria for predicting the severity of acute pancreatitis [9]

At admission	During initial 48 h
Age > 55 years	Decrease hematocrit > 10%
White cells > 16 000/mm3	Increase blood urea > 5 mg/100 ml
Blood glucose > 11 mmol/l (200 mg/dl)	Calcium < 8 mg/100 ml
Lactic dehydrogenase (LDH) > 350 IU/l	pO2 < 60 mmHg
Glutamic oxaloacetic transaminase (SGOT) > 250 IU/l	Base deficit > 4 mEq/l
	Fluid deficit > 6000 ml

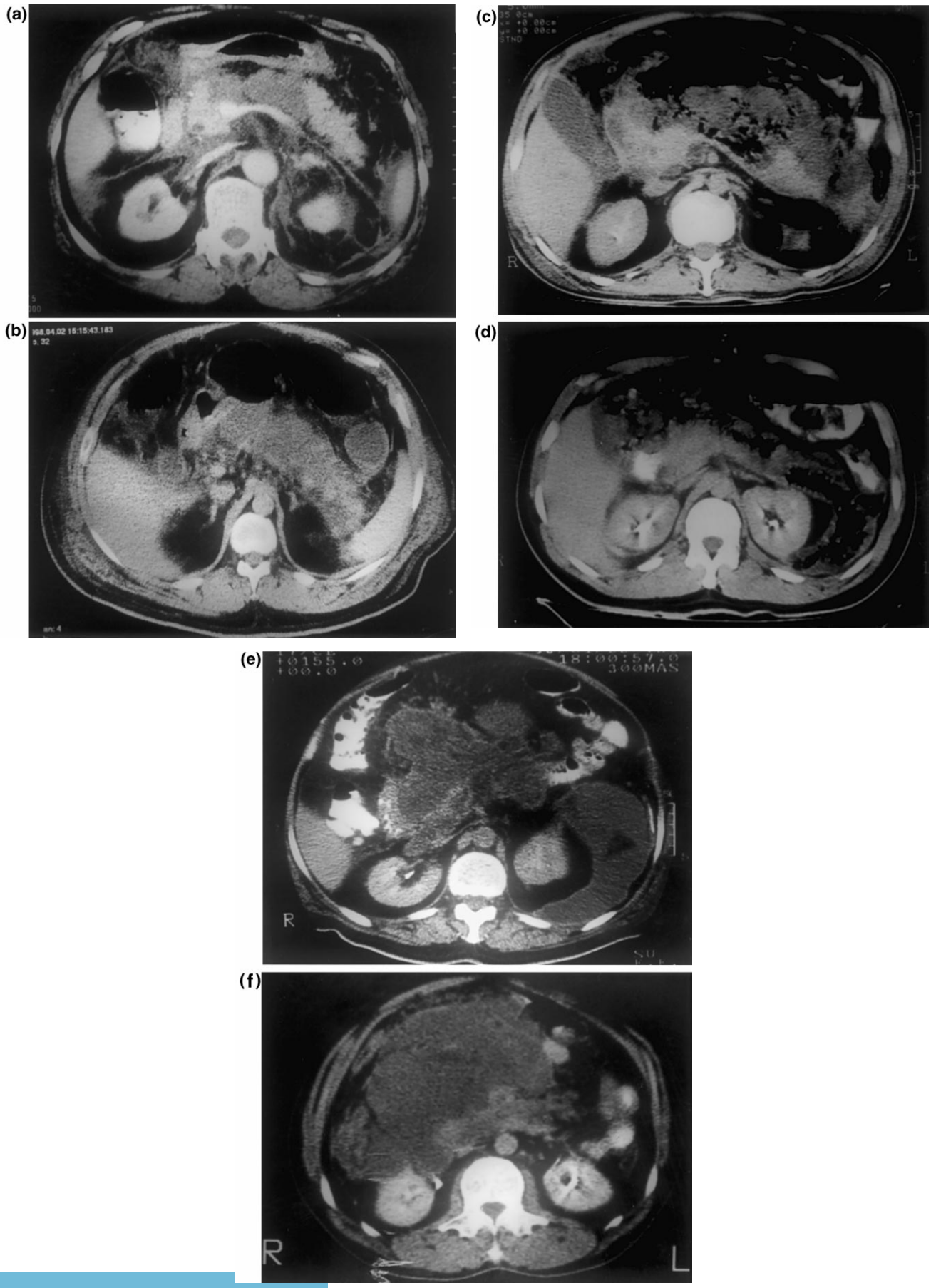

Fig. 3. (*Continued*)

3f and Fig. 4a). These abscesses occur at areas of limited necrosis with secondary infections and occur after 4 weeks or later following the onset of attack [12].

1.3.5. Sepsis

Acute pancreatitis promotes translocation of gut-derived organisms to the inflamed pancreas and peripancreatic region [13]. In severe acute pancreatitis, the frequency of development of sepsis is overall between 40 and 70%, and increases with time after onset of symptoms [14].

1.3.6. Infected necrosis

This entity is described as infected pancreatic or peripancreatic necrotic tissue (Fig. 3c and d). Mortality rate of the patients with infected necrosis rises to around 60% and requires surgical debridement [15–17].

The benefits of ultrasound, computerized tomography (CT) and magnetic resonance imaging (MRI) are accepted as imaging modalities for diagnosis and management of acute pancreatitis.

1.4. Ultrasound

Ultrasound is usually the first modality of choice in a patient with acute abdomen. Ultrasound precludes other diseases such as acute cholesistitis, hepatic abscess, can depict coexisting biliary and gallbladder diseases; however, associated paralytic ileus can limit ultrasound diagnosis of acute pancreatitis during the first 48 h. Pancreas is more easily visualized by ultrasound during the convalescent phase [18].

A normal gland can be observed in the mild form. However, in one third of the cases, due to the interstitial edema, pancreatic parenchyma may show diffuse enlargement and hypoechoic texture [18]. Focal intrapancreatic ill-defined masses can be observed in acute pancreatitis. These are areas of hemorrhage and pancreatic necrosis. Peripancreatic fluid collections may be identified as anechoic areas. Free intraperitoneal fluid should be searched for in the pouches of Morrison and Douglas [8]. After the attack of the acute pancreatitis, intra and extraperitoneal fluid collections can be followed-up by serial US examinations. These fluid collections may resolve with time or pseudocysts may develop.

Color Doppler ultrasound visualizes the vascular complications such as gastroduodenal or splenic artery pseudoaneurysm, thrombosis of the portal system and venous collateral pathways due to portal venous thrombosis [19].

1.5. Computerized tomography

Spiral CT is the preferable technique in suspected pancreatic diseases. CT is the most commonly used to evaluate acute and chronic pancreatitis. Spiral CT permits scanning the entire pancreas during a single breath hold. The peak parenchymal and vascular enhancement can be evaluated by spiral CT study. Contrast-enhanced CT has a critical role in acute pancreatitis. CT scan provides early diagnosis of the disease, designates the severity of illness, detects associated complications, and ensures efficacy in percutaneous therapy.

Spiral CT scanners improve the diagnostic capabilities of CT evaluation of pancreas. Unenhanced scanning is necessary for detection of recent hemorrhage. Contrast enhanced CT scan is performed after intravenous injection of iodinated contrast agent (300-mg iodine/ml) using a power injector. I recommend a dual-

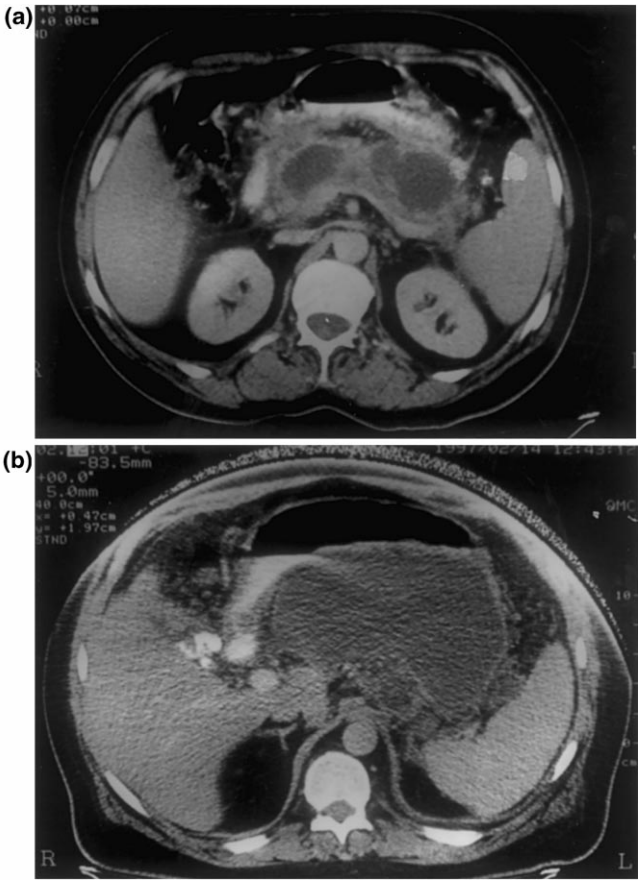

Fig. 4. Pancreatic pseudocyst. (a) A round fluid collection with thin capsule is seen within the lesser sac. (b) Other psedocyst formations in the pancreatic gland parenchyma.

Fig. 3. Patients with severe acute pancreatitis. (a) Contrast enhanced CT. Focal unenhanced area of pancreatic necrosis. (b) Lack of enhancement of the pancreatic parenchyma due to the necrosis of the entire pancreatic gland. (c) Infected pancreatic necrosis. Hypodense, necrotic area at pancreatic corpus with bubbles of gas. (d) Infected peripancreatic fluid collection with abundant gas bubbles at the anterior pararenal compartment. (e) Infected necrosis of the entire pancreatic parenchyma with peripancreatic abscess ventral to the pancreatic head and fluid collection on left pararenal space. (f) Ill-defined fluid collection located in the mesocolon and right anterior pararenal space.

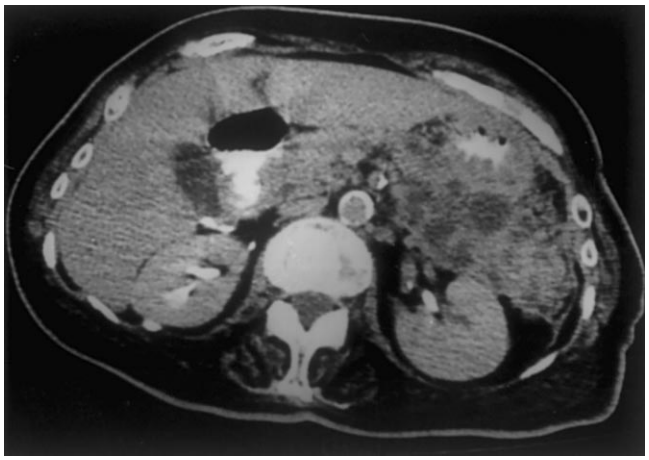

Fig. 5. Acute severe pancreatitis and peripancreatic abscess formation. Peripancreatic abscess formation is observed within the peripancreatic and the left anterior pararenal space.

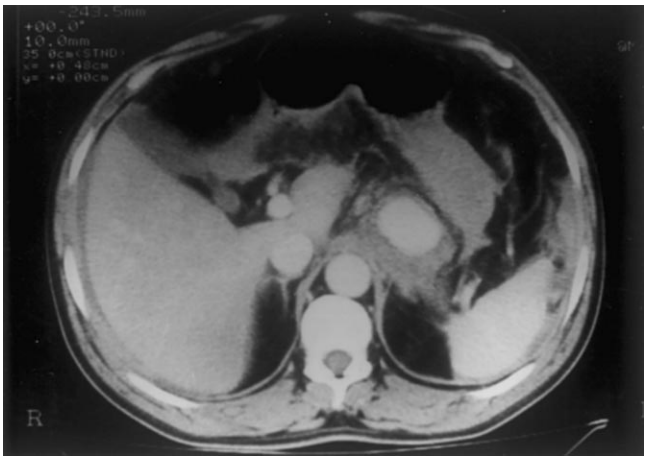

Fig. 6. Splenic artery pseudoaneurysm. Late complication of acute pancreatitis after 6 weeks of acute attack.

phased spiral acquisition, the first phase obtained at 25 s for the arterial, and the second at 50 s for portal venous phase. The rate of contrast medium injection was 4 ml/s.

CT findings in mild acute pancreatitis vary between a normal pancreas or slight to moderate diffuse hypertrophy of the gland (Fig. 1a and b). In the mild form, peripancreatic fatty planes show increased density with mild thickening of the adjacent fascial planes. These changes result in a hazy appearance of the pancreatic contour. In more severe forms, acute fluid collections can be seen as ill-defined masses of low attenuation within the peripancreatic areas. The most common spaces involved are the left anterior pararenal space and the lesser sac [20]. Inferior extension occurs towards the pelvis and upward extension to the mediastinum. Infection of fluid collections develops in 3–21% of patients with acute pancreatitis [15,21]. A focal or diffuse, well marginated area of unenhanced pancreatic parenchyma suggests the pancreatic necrosis [1]. In acute pancreatitis, 80% of deaths occur as a result of infected necrosis.

Severe retroperitoneal hemorrhage is due to the erosion of vessels by extravasation of proteolytic enzymes, resulting in bleeding or in the formation of pseudoaneurysms (Fig. 6) [22,23].

Evaluating the obliteration of the fat surrounding the superior mesenteric vein (SMV) and artery (SMA) has been considered in differentiating pancreatic carcinoma and pancreatitis. However, some patients with acute pancreatitis can demonstrate perivascular fat plane infiltration, mimicking appearances of carcinoma [24,25]. There is a significant increase in the diameter of SMV in pancreatitis. Venous diameter and blood flow increases in pancreatitis due to release of vasoactive substances in inflammation. Since veins have distensible walls and the tissue resistance of an organ does not differ significantly in inflammation, the SMV could freely be distended to compensate for the increased blood flow through the SMA. This may be the reason for SMV dilatation in pancreatitis [26].

1.6. CT staging

The severity of CT findings correlates with the prognosis of acute pancreatitis. A variety of classifications have been made by different groups in acute pancreatitis. Balthazar et al. classified the severity of pancreatitis according to CT appearance into five categories (Table 1). In their results, patients with grade A–C pancreatitis suffered a mild, uncomplicated clinical course, while grade D–E pancreatitis had prolonged morbidity with a higher incidence of abscess and mortality [5,8,27,28].

According to this table (Table 1), presence of necrosis and acute inflammatory process are the two most important CT prognostic factors in the assessment of severity of acute pancreatitis. Balthazar developed a grading system using these two CT prognostic factors called the 'CT severity index (CTSI)' (Tables 2 and 3)

Table 2
CT classification of the severity of acute pancreatitis [4]

Grade A	Normal pancreas
Grade B	Focal or diffuse pancreas enlargement with contour irregularities, paranchymal inhomogeneous attenuation of gland, dilatation of pancreatic duct and foci of small fluid collections within gland without peripancreatic inflammation
Grade C	Intrinsic pancreatic abnormalities with haziness and streaky densities representing inflammatory changes in peripancreatic fat
Grade D	Single, ill defined fluid collections with no recognizable capsule or wall
Grade E	Two or more poorly defined fluid collections or presence of gas in or adjacent to pancreas

Table 3
Calculation of CT severity index (CTSI) [16]

Inflammatory process		
Grade A	Normal pancreas	0
Grade B	Focal or diffuse enlargement of pancreas	1
Grade C	Pancreatic gland abnormalities associated with peripancreatic inflammation	2
Grade D	Fluid collection in a single location	3
Grade E	Two or more fluid collections and/or the presence of gas in/or adjacent to pancreas	4
Gland necrosis		
	No necrosis	0
	Less than 30%	2
	30–50%	4
	Greater than 50%	6
CT severity index		a+b

[16]. He reported that patients with a CTSI of 0–3 show a 3% mortality rate and an 8% morbidity rate and where as in patients with a CTSI of 7–10, mortality and morbidity rates were 17 and 92%.

The purpose of CT in acute pancreatitis is the verification of diagnosis and the determination of severity of the disease. CT may be repeated in cases with unusual findings like fever, pain, hypotension, or decreasing haematocrite level. Although ultrasound is the modality of choice in the follow-up, CT may be repeated 10 days after the first attack for the determination of late complications.

1.7. Magnetic resonance imaging (MRI)

MRI can depict the presence and extent of necrosis and peripancreatic fluid collections, as well as CT. MRI in severe acute pancreatitis requires administration of gadolinium (Gd) to detect necrosis. [29].

The drawback of CT in acute pancreatitis is the use of iodinized contrast agents. There are several studies reporting increased rate of complications and delay of recovery after contrast-enhanced CT in patients with acute pancreatitis [29–33]. Iodinized contrast media used in CT are potentially nephrotoxic, especially in dehydrated patients [34]. However, intravenous Gd used in MRI has good renal tolerance hence the use of MRI in acute pancreatitis has become a current issue.

Advances in MRI technique such as fat-suppression sequences and Fast SE supplemented by breath-hold images with phased-array coils may allow increasingly reliable MRI evaluations and allows excellent contrast resolution of the pancreatic and peripancreatic tissues [35](Fig. 7a, b and Fig. 8a, b).

High field MRI is more successful in differentiating between mild and severe acute pancreatitis than mid and low field MRI [36].

T2-weighted images and Gd enhanced T1 sequences are currently the most valuable imaging sequences for the assessment of patients with acute pancreatitis. Normal pancreatic tissue display homogenous hyperintense signal intensity relative to liver on a fat saturated T1 Fast SE sequence, and is defined as Grade A. Grade B is defined as a heterogeneous pancreatic signal without peripancreatic fat involvement and Grade C as the presence of strands in the peripancreatic fat [37,38]. Grade D and E demonstrate acute fluid collections as ill-defined confluent areas without wall or capsule. Gas can be recognized as hypointense areas on both T1 and T2 weighted images.

Routine pancreas MRI protocol must include T2-W Fast SE, fat-suppressed T1-W Fast SE, and a series of T1-W Gradient echo sequences prior and immediately after Gd-DTPA injection. With this protocol, Lecesne et al. reported that MRI is a reliable method for staging pancreatitis and is at least as accurate as CT in establishing the prognosis of disease [39].

Enlargement of the gland can be demonstrated on any sequence. Parenchymal edema is well shown on

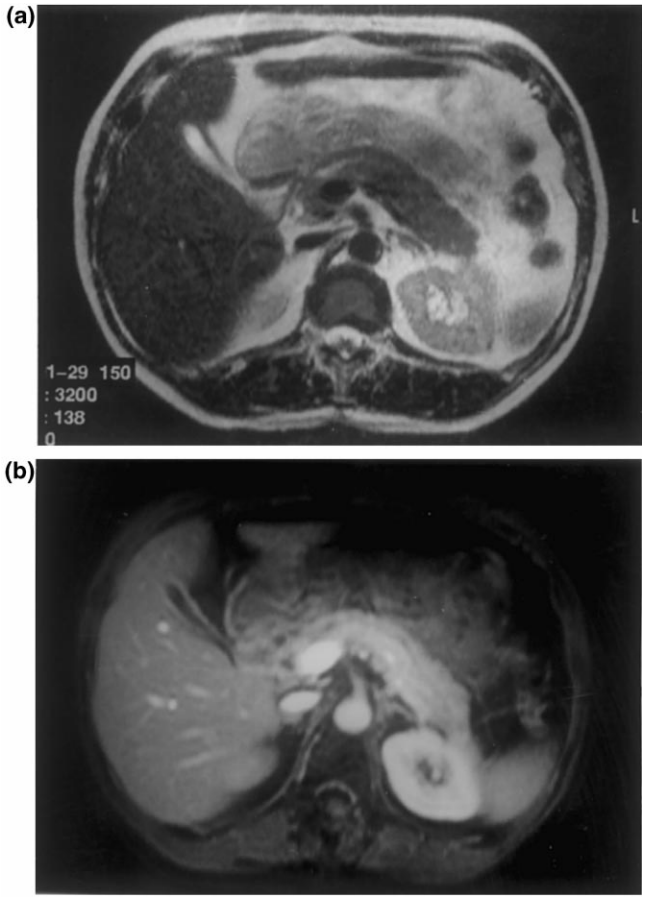

Fig. 7. Acute mild pancreatitis. T2W and T1W (fat saturated) axial Fast SE Gd. DTPA enhanced MR (a) hypointense (b) hyperintense pancreatic parenchyma without necrosis or peripancreatic fluid collection.

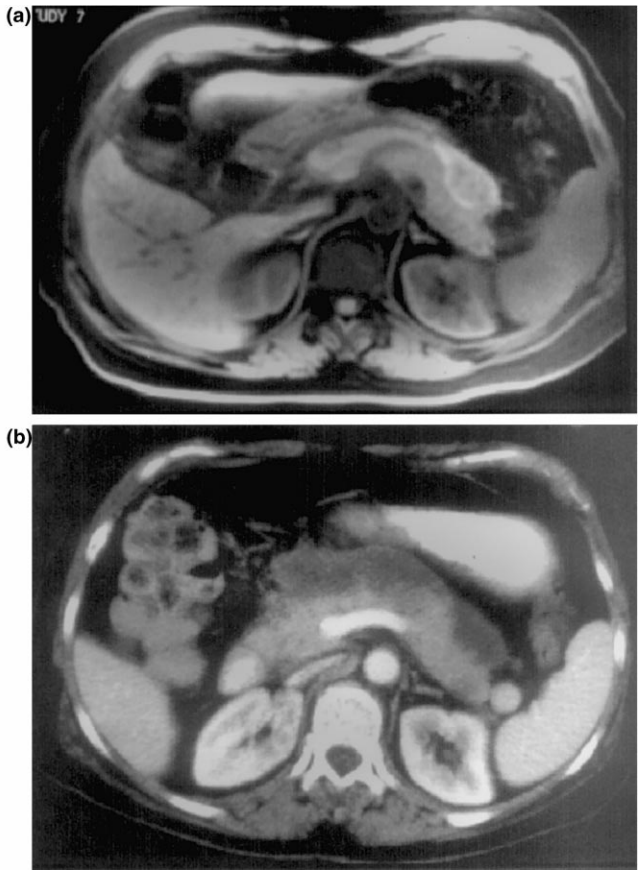

Fig. 8. Acute pancreatitis with hemorrhage. On CT (a) image prepancreatic fluid collection is visualized, which is hyperintense on T1-weighted fat saturated MR image (b) consistent with subacute hemorrhage (Courtesy Professor Semelka, UNC, Chapel Hill).

unenhanced T1-W images. Pancreatic enhancement with Gd-DTPA is maximal within 20–40 s after injection. The presence and extent of parenchymal necrosis is well demonstrated on sequential multi-slice acquisitions obtained during the first 1–2 min after injection of Gd-DTPA [40].

T2-weighted SE imaging offers the most sensitive demonstration of fluid collections. It is more effective than CT in demonstrating the internal structure and content of larger fluid collections allowing the decision of percutaneous drainage [40,41].

Imaging in severe acute pancreatitis requires intravenous contrast enhancement for assessment of pancreatic parenchymal perfusion and presence of necrosis. In addition, it is necessary to know the presence, location, size and extent of fluid collections within or around of the gland. Recent studies have confirmed that MRI can accomplish these aims, as well as CT. However, CT retains several significant advantages. These are, CT is widely accessible and less costly than MRI; it is more sensitive in detecting small gas bubbles and calcifications and CT allows interventional therapeutic procedures [29].

2. Chronic pancreatitis

Incidence of chronic pancreatitis is less frequent than acute pancreatitis. It is a chronic inflammatory process of the pancreas, which results in irreversible exocrine dysfunction and irreversible morphologic changes of the pancreas and pancreatic duct [42]. It is characterized by a relentless and progressive damage and loss of pancreatic parenchymal tissue [43,44]. Chronic pancreatitis has various causes. The most common cause is chronic alcoholism for 6–12 years [45]. Other causes can be encountered as chronic ulcerative colitis, Sjörgen's syndrome, primary sclerosing cholangitis. These diseases can be described as non-alcoholic duct-destructive chronic pancreatitis.

Patients present with recurrent abdominal pain and exocrine or endocrine dysfunction after the subclinic phase [46]. Most common complications are obstructive jaundice, ileus, pseudocyst formation, pancreatic abscess, and gastrointestinal bleeding. Vascular complications as pseudoaneurysm occurs as a late complication of vascular injuries produced by extravasated pancreatic enzymes. Pancreaticoduodenal and splenic arterial branches are most frequently involved [5,19]. Pseudoaneurysm may result with bleeding (Fig. 9a–d and Fig. 10) [23]. Another vascular complication is the thrombosis of portal venous system (Fig. 11). Pancreatic cancer develops in approximately 4% of patients with chronic pancreatitis within 20 years [47].

The morphological changes include irregular sclerosis of the parenchyma. These histological changes are seen as parenchymal atrophy in cross-sectional images. Findings accompanying glandular atrophy include the irregularity of the pancreatic duct, and the presence of narrowed and widened segments along the pancreatic duct. Pancreatic duct calcifications and calculi may be observed. Pancreatic edema or necrosis, which may be focal, segmental or diffuse may be present at acute exacerbations of pancreatitis.

'Obstructive chronic pancreatitis' is characterized by dilatation of the pancreatic duct proximal to an obstruction, atrophy of the acinar parenchyma, and uniformly diffuse fibrosis. Many of these changes may regress if the obstruction is relieved [48]. Criteria for the diagnosis of chronic pancreatitis are represented by tapered structure of the common bile duct and irregular dilatation with dilated and stenotic segments (beaded appearance). Other common findings include filling defects such as stones or casts within the pancreatic duct and pseudocyst [49].

Diagnosis of chronic pancreatitis requires clinical evaluation, laboratory studies, and interventional and/or cross-sectional imaging modalities.

2.1. Ultrasound

Alterations in size, shape, and contour, changes of parenchymal texture, dilatation of the main pancreatic and biliary ducts, calcifications, patency of portal venous system, and presence of fluid collections can be evaluated with ultrasound. Atrophy is the late feature of chronic disease. Both peripancreatic fat and fibrotic gland tissue are visualized as hyperechoic structures on ultrasound study, hence atrophy of the gland may not be apparent.

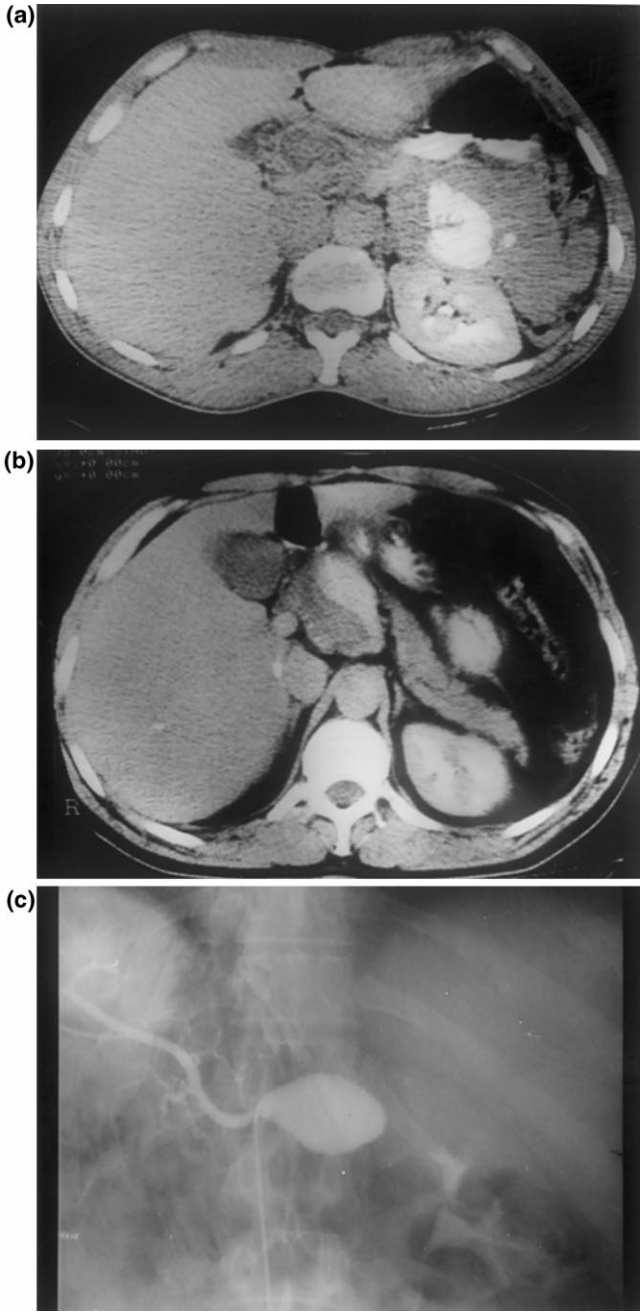

Fig. 9. (a) CT scan study after acute attack of a patient with chronic pancreatitis shows hypodense area located at the pancreatic head. (b) Two years after the onset of the earlier attack, CT demonstrates the partially thrombosed hepatic artery pseudoaneurysm located above and causing downward displacement of the pancreatic head. (c) Superselective angiograms clearly demonstrating the non-thrombosed lumen of the aneurysm at the origin of hepatic artery.

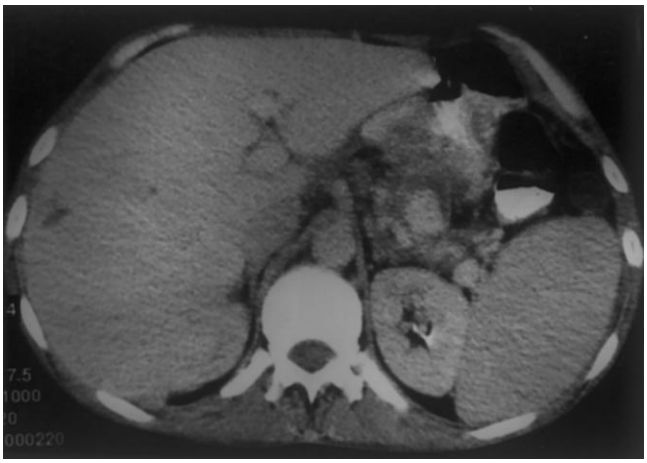

Fig. 10. Contrast enhanced CT study. (a) Partially thrombosed splenic artery pseudoaneurysm.

Color Doppler ultrasound allows the detection of vascular complications such as pseudoaneurysm, thromboses of portal system, presence of collateral pathways [45].

2.2. ERCP

The diagnosis of chronic pancreatitis is based on demonstration of radiomorphological changes of main pancreatic duct and side-branches. The indications for ERCP in chronic pancreatitis are diagnostic (Table 4), preoperative staging and occasionally endoscopic therapy [50]. Side-branch ectasia of pancreatic duct is the most prominent and specific feature of this disease process. Other features are multifocal dilatations and

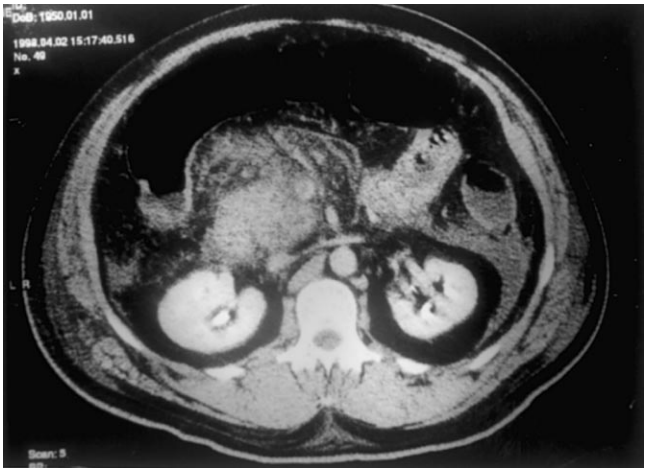

Fig. 11. Thrombosis of superior mesenteric vein is seen as another vascular complication of acute pancreatitis.

Table 4
ERCP anomalies in chronic pancreatitis [51]

Side branches
Dilatation with or without downstream stenosis
Irregular lining
Obstruction
Intraluminal filling defects or calcified stones
Main pancreatic duct (MPD)
Dilatation with or without downstream stenosis
Irregular lining
Obstruction:seldom
Intraluminal filling defects or calcified stones
Pancreatic parenchyma
Pseudocyst(s) communating with MPD or side branches
Abscess:seldom
Common bile duct (pancreatic segment)
Long smooth stenosis (Caroli I)
Short smooth preterminal stenosis (Caroli III)
Deviation (pseudocyst)
Obstruction, asymmetric stenosis:seldom

structures and irregular contours of the main duct and side branches; filling defects from calculi, mucinous plugs, or debris and pseudocysts (Table 5) [45].

The earliest findings of chronic pancreatitis are side branch dilatation with or without stenosis, irregular lining, intraluminal filling defects, and small calcified stones (Fig. 12a and b). In the later stages, the main pancreatic duct becomes abnormal. Changes of main pancreatic duct may be diffuse, focal or multisegmental [48]. Regularly lined cavities filling with contrast are mostly caused by pseudocysts. Pseudocysts may size from 1 to 20 cm, or occasionally larger. They may be located in or around the pancreas. Such cavities are only filled with contrast when they communicate with the main pancreatic duct or a side branch [39].

Axon et al. classified chronic pancreatitis into three types according to the morphologic abnormalities (Cambridge system), mild; moderate; severe (Table 5) [51–53]. In the Cambridge classification, ultrasound, CT and ERCP are used to classify chronic pancreatitis.

Table 5
Cambridge classification of chronic pancreatitis [51,52]

Category	Pancreatogram
Normal	Normal MPD (main pancreatic duct) and normal side branches
Equivocal	Normal MPD, <3 abnormal side branches
Mild	Normal MPD, >3 abnormal side branches
Moderate	Abnormal MPD, >3 abnormal side branches
Marked	As in category moderate, with one or more of the following Large cavity (>10 mm) Intraductal filling defect or calculus Severe irregularity Dilatation (>10 mm) or obstruction of MPD

The differentiation between chronic pancreatitis and pancreatic carcinoma can be made with ERCP. The side branches and main pancreatic duct show focal upstream dilatation in pancreatic carcinoma. Calcifications and cavities are seldom in neoplasia. However, when chronic pancreatitis and pancreatic carcinoma occur together in the same patient, it is very difficult to make the correct diagnosis. Regression of calcifications in earlier identified chronic pancreatitis may be a sign of superimposed carcinoma [52].

2.3. Computerized tomography

CT findings of chronic pancreatitis are dilatation of the main pancreatic duct, parenchymal atrophy, pancreatic calcifications and pseudocysts (Figs. 13 and 14). While diffuse enlargement is common in acute pancreatitis, this finding is rare in chronic pancreatitis and parenchymal atrophy is more frequent than enlargement. Occasionally pancreatic enlargement can occur due to extensive interlobular and periductal fibrosis [54,55]. Carcinomas are relatively uncommon in-patients with known chronic pancreatitis but is hard to diagnose when they coexist. Moreover, tumors are hard to differentiate from focal areas of chronic pancreatitis. The presence of ductal calcification or stone is suggestive of a benign lesion. Although obliteration of fat and enhancement of superior mesenteric vessels or celiac truncus have been described in-patients with pancreatic carcinoma, these findings are nonspecific [26,56].

On unenhanced CT, both inflammatory masses and small carcinomas are usually isodense with normal pancreatic tissue. Carcinoma is hypovascular and shows much less contrast enhancement than normal pancreatic tissue in the arterial phase of biphasic CT study [29].

In the differentiation between chronic pancreatitis and pancreatic carcinoma, the sensitivities of ultrasound and CT are 98 and 94%; and specificities are 90 and 95%, respectively [57].

2.4. Magnetic resonance imaging

Until recently, ERCP and PTC were the only techniques capable of providing images of pancreatic duct. Both techniques are invasive and they have mortality and morbidity rates of 1 and 7%, respectively [58]. Acute pancreatitis is the most common complication of ERCP.

MRI combines the advantages of cross-sectional imaging techniques such as ultrasound and CT, with the ability to visualize the pancreatic duct as in ERCP and PTC [45]. A pancreatic MR examination should include T1W and T2W images and MR cholangiograms (Figs. 15 and 16a, b). Magnetic Resonance Cholangiopancreatography (MRCP) is a non-invasive

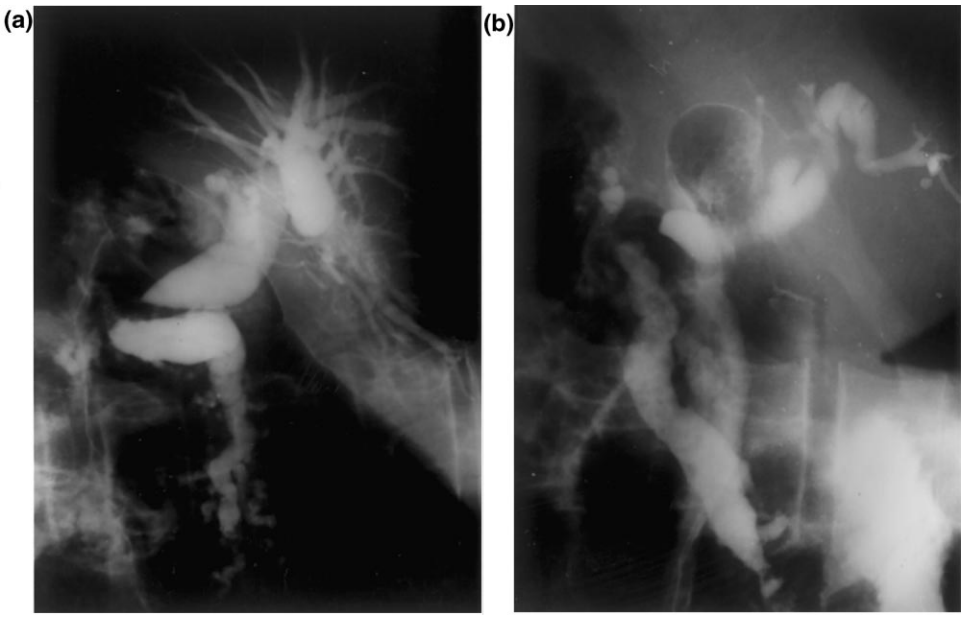

Fig. 12. ERCP features of chronic pancreatitis. (a) Dilatation of biliary and main pancreatic ducts with side branch ectasia (b) and numerous filling defects in the main duct due to multiple calculi (Courtesy of the Gastroenterology Department, Ege University Medical Faculty).

imaging technique that provide projectional images of the biliary and pancreatic ducts (Fig. 17a, b) [49]. Normal non-dilated pancreatic duct can be on 69% of MIP-reconstructed images and 81% of source images [59].

The role of MRCP in chronic pancreatitis is still controversial. Due to lower spatial resolution, abnormalities of mild pancreatitis can not be assessed. During retrograde pancreatography, contrast medium injection creates overdistention of the ducts, whereas MRCP reflects the physiological condition [49]. Matos et al. proposed the use of secretin administration for improving the delineation of ductal morphological features in both normal and diseased patients presenting with suspected chronic pancreatitis with no ductal alteration at CT and ultrasound. Moreover, secretin stimulation provided additional functional data regarding the presence of papillary stenosis and duodenal filling, which is correlated to the exocrine pancreatic function [60]. Pancreatic ductal abnormalities in pancreatitis can be readily demonstrated by MRCP, however, because contract agent is not used, communication between a cyst and the ductal system is not discernible when it exists together with pancreatitis [49].

Stones as small as 2 mm in diameter can be detected by MRCP [60]. However, MRCP is less sensitive in the detection of subtle ductal abnormalities and, in comparison with ERCP, may over or underestimate the length and severity of stenoses [58]. Comparisons between MRCP and ERCP in cases of chronic pancreatitis have revealed agreement of 83–100% for identification of ductal dilatation, 70–92% for identification of narrowing, and 92–100% for identification of filling defects, respectively [58,61]. ERCP is more sensitive to early side-branch changes because of its increased spatial resolution [42].

MR can help differentiatiation of pancreatic carcinoma and chronic pancreatitis with regard to focal or diffuse changes in signal intensity and contrast enhancement features. On T1-W fat-saturated MRI, areas of chronic pancreatitis show decreased signal intensity compared with normal pancreas. But the degree of signal reduction is generally less than that is associated with carcinoma [62,63]. The signal from inflammatory masses in chronic pancreatitis may be the same as that of the liver, but carcinoma is often hypo-intense to liver [63]. Even in the presence of typical changes of chronic pancreatitis, the presence of a focal hypointensity on

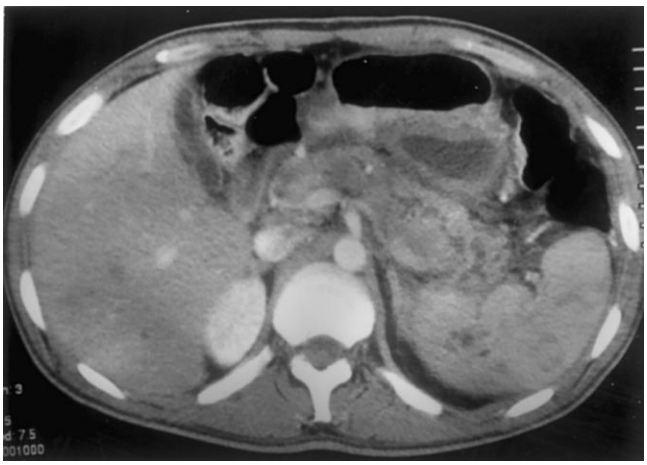

Fig. 13. Chronic pancreatitis. Contrast enhanced CT. Pancreatic duct calcifications.

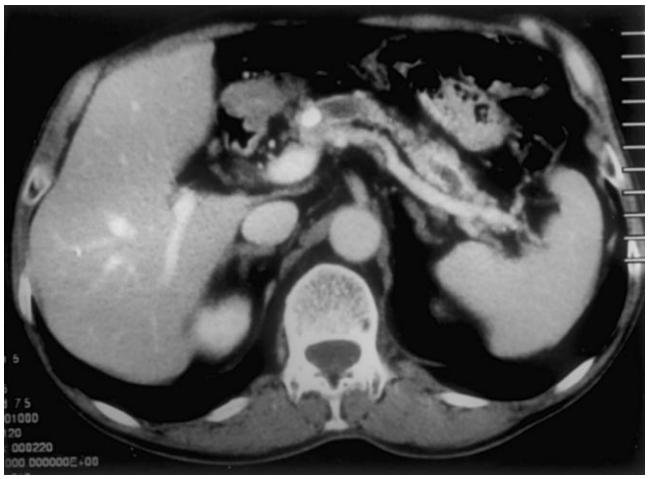

Fig. 14. Chronic pancreatitis. Contrast enhanced CT. Pancreatic duct calculus and dilatation of the duct proximal to the calculus.

turbo FLASH images should be considered as diagnostic of pancreatic carcinoma until proved otherwise [45].

MRCP has the following advantages over ERCP. First of all, it is a non-invasive technique, requiring no anesthesia or premedication, and no contrast agent or ionizing radiation is used. There is no increased risk of acute pancreatitis. MRCP can be performed on patients with altered pancreaticobiliary system morphology for earlier surgery. Conventional MR sequences can be combined for complete study of liver and pancreas. In-patient with complete occlusion of main pancreatic duct, MRCP can demonstrate the upstream anatomy and periductal abnormalities.

The advantages of ERCP over MRCP are, superior demonstration of main pancreatic ducts and side-branches, direct visual inspection of papilla, cytologic sampling of pancreatic juice. ERCP can demonstrate changes in the ductal system earlier than MRCP. Moreover, ERCP allows therapeutic maneuvers as papillotomy and/or stent insertion [29].

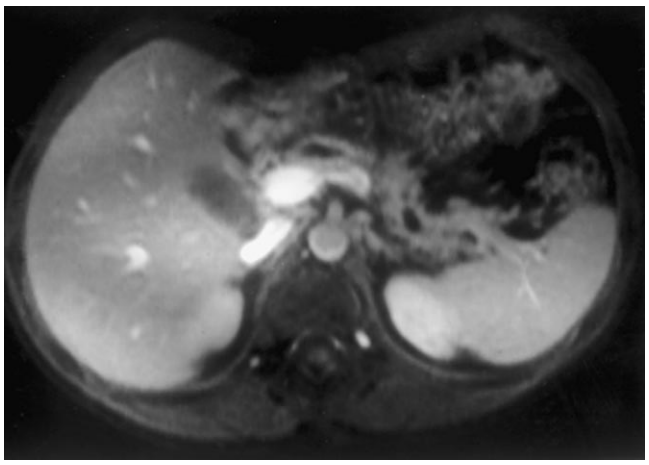

Fig. 15. Chronic pancreatitis. Contrast enhanced MRI. Dilatation of the main pancreatic duct.

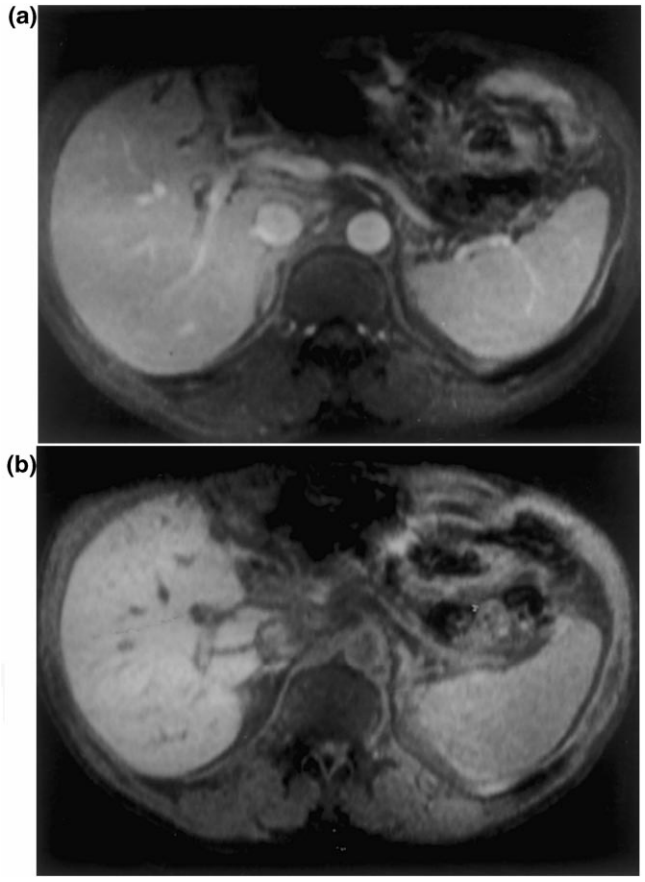

Fig. 16. (a) Non enhanced and (b) enhanced T1W fat saturated Fast SE MR. Chronic pancreatitis with pancreatic parenchymal atrophy and pancreatic duct dilatation.

Ultrasound, CT and MRI are accepted as imaging modalities for diagnosis and management of acute pancreatitis. Ultrasound has some limitations due to intestinal loops, which cover the pancreatic and peripancreatic tissue, mesenteric fat, and the other anatomic structures. However ultrasound has been used as the first imaging modality for detecting the etiologic factor and follow-up of the amount of fluid collections. The diagnosis of acute pancreatitis with laboratory findings can be confirmed by CT. In-patients with suspected acute pancreatitis, early CT study is suggested by many authors for confirmation of the diagnosis and detection of severity of disease.

Contrast agent toxicity of CT has been accused for the delay of recovery and prolongation of hospitalization. It has been reported that fat-suppressed-Fast SE sequences after Gd injection protocols are preferable in the establishment of severity of disease and determination of prognosis. The superiority of MRI in hemorrhage pancreatitis, which is the most severe form of pancreatitis with highest mortality rates, is beyond question.

MRCP have been accepted as the primary imaging technique in the diagnosis of chronic pancreatitis at

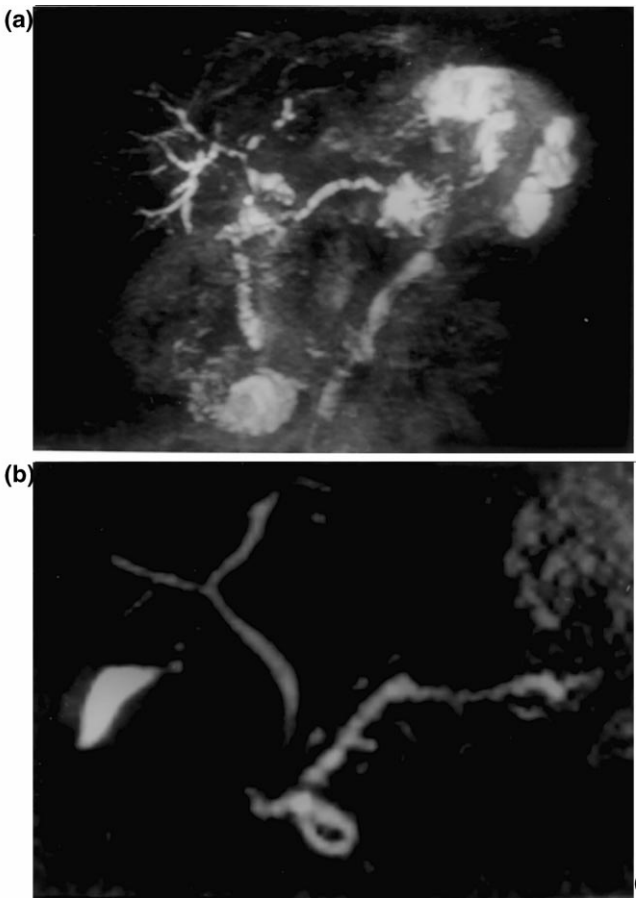

Fig. 17. MRCP examples of chronic pancreatitis. (a) Mild dilatation of main pancreatic duct with side branch ectasia. (b) MIP image display dilatation and irregularity of main pancreatic duct associated with biliary duct irregularity corresponding to sclerosing cholangitis.

several centers. Parenchymal findings can be evaluated by dynamic spiral CT or fat saturated T1-W; T2-W Fast SE MR sequences. Meanwhile ERCP can be accepted as the gold standard technique in the early period of disease, due to capabilities of demonstration of the main and side branch pancreatic ducts; and ERCP is essential for therapeutic procedures.

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