Original Article

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Contrast-enhanced Computed Tomographic Evaluation of Acute Pancreatitis: An Exploratory Study

U S Vinayaka[1], G Ravichandra[1], K Devadasa Acharya[2], K N Muralidhara[3]

[1]Associate Professor, Department of Radiodiagnosis, Yenepoya Medical College, Deralakatte, Mangalore, Karnataka, India, [2]Professor and HOD, Department of Radiodiagnosis, Yenepoya Medical College, Deralakatte, Mangalore, Karnataka, India, [3]Senior Resident, Department of Radiodiagnosis, Yenepoya Medical College, Deralakatte, Mangalore, Karnataka, India

Abstract

Introduction: Diseases of the pancreas have a very variable presentation and hence imaging plays an important role in the diagnosis and management of pancreatic disease, especially in acute pancreatitis. Computed tomography (CT) is the modality of choice as an evaluation of the pancreas.

Materials and Methods: A total of 30 patients who were clinically suspected of having acute pancreatitis attending our hospital were our study participants.

Results: The present study consisted of 30 patients who were suspected to have acute pancreatitis by clinical examination and laboratory parameters and referred for contrast-enhanced CT (CECT) examination of the abdomen. The peak age of incidence was noted in 30-40 years. 25 out of 30 patients had enlargement of the pancreas. 11 of these showed focal enlargement and the rest (14 patients) showed diffuse enlargement. Peripancreatic fat stranding was noted in 21 cases. Phlegmonous changes were evident in 14 patients with involvement of the lesser sac, mesentery transverse mesocolon, and anterior pararenal spaces. 7 cases showed the involvement of more than one anatomical site. In our study, 3 patients had Grade "A," 7 patients had Grade "B," 15 patients had Grade "C," 1 patient had Grade "D," and 4 patients had Grade "E" pancreatitis. In our study, CT severity index (CTSI) of 0-3 as seen in 16 patients, 4-6 was seen in 8 patients, and 7-10 was seen in 6 patients. Out of 4 patients who expired during the course of study, 3 had CTSI of more than 7 and those patients with CTSI of <3 had no complications, and there was no need of ICU stay for these patients.

Conclusion: In all the cases, CT scan revealed the exact morphological appearance of the disease. CECT was very useful in staging acute pancreatitis using various CT numerical grading systems.

Key words: Contrast-enhanced computed tomography, Grading of pancreatitis, Pancreas

INTRODUCTION

The pancreas was one of the last organs in the abdomen to receive the attention of anatomists, physiologists, physicians, and surgeons.[1] Diseases of the pancreas have a very variable presentation, and hence imaging plays an important role in the diagnosis and management of pancreatic disease, especially in acute pancreatitis. Modalities for imaging of pancreas range from plain radiographs to contrast studies, ultrasonography, endoscopic ultrasound, endoscopic retrograde cholangio-pancreatography (ERCP), computed tomography (CT), and magnetic resonance imaging. CT scan is the modality of choice as a non-invasive method of evaluation of the pancreas because it is unaffected by bowel gas or large body habitus. Among the diseases of the pancreas, pancreatitis is one of the most complex and clinically challenging of all abdominal disorders.[2,3] During development of the pancreas, due to the differential growth of the gut wall, the ventral bud (along with the bile duct) shifts to the left side. Pancreatic tissue formed with respect to these two buds now fuses to form one mass. The ducts of the dorsal and

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Corresponding Author: Dr. U S Vinayaka, Department of Radiodiagnosis, Yenepoya Medical College Hospital, Deralakatte, Mangalore - 575 018, Karnataka, India. Phone: +91-9845270001. E-mail: drvinayaka@yahoo.com

ventral buds anastomose with each other and open into the duodenum at ampulla of Vater.[3] The most commonly seen developmental anomalies of the pancreas are - agenesis of dorsal or ventral pancreas, annular pancreas, pancreas divisum, and left sided pancreas.[3] Out of these anomalies, pancreas divisum is commonly associated with recurrent pancreatitis.[3-5] CT scan is the most useful modality in imaging a suspected case of pancreatitis. Ever since Mr. Hounsfield applied reconstruction technique to produce the world's first clinically useful CT image, it is extensively used/developed for imaging pancreatitis.[4]

Review of Literature

The pancreas is an exocrine and endocrine organ situated retroperitoneally in the left hypochondrium. It is descriptively divided into four parts *viz.* the head, neck, body, and tail (Figure 1).[1,2,4] It is situated in the anterior pararenal space, by the posterior parietal peritoneum and dorsally by the anterior pararenal (Gerota's) fascia. The main pancreatic duct runs along the length of the pancreas and joins the common duct at the ampulla of Vater. In the head region, ranges 1-3 mm in diameter. The accessory pancreatic duct is more horizontal than the main duct. The CBD is seen within the pancreatic head close to its lateral and posterior surface, as a round or oval near water density structure.[3,4] The attenuation of the pancreas is normally the same as that of soft tissue (30-50 HU). The normal pancreas increases in density after intravenous (IV) contrast administration. Pancreas is supplied by branches arising from the celiac and superior mesenteric arteries and drained by the tributaries of the superior mesenteric vein.

Acute pancreatitis is a common illness characterized by non-specific pancreatic inflammation associated with diverse etiologic factors, which include the following:[5] (a) Metabolics such as alcoholic, hyperlipoproteinemia, hypercalcemia, drugs, scorpion venom, and genetic; (b) Mechanical such as cholelithiasis, post-operative (gastric/biliary), post-traumatic, retrograde pancreatography, pancreatic duct obstruction, pancreatic tumor, ascaris infestation, and duodenal obstruction; (c) Vascular such as post-operative (cardiopulmonary bypass), poly arteritis nodosa, and atheroembolism; and (d) Infections such as mumps and coxsackie virus. The most common etiologies found in 80% of patients are heavy alcohol abuse and cholelithiasis.[5]

The pathophysiology is still controversial but appears to be related to a temporary or permanent blockage of the pancreatic duct leading to a sudden release of enzymes into adjacent interstitial tissue. The activated extravasated enzymes lead to autodigestive fat necrosis and non-specific inflammation of the pancreas and peripancreatic tissues.[6] The need for reliable imaging modality to diagnose and confirm the clinical diagnosis of acute pancreatitis is evident when alternate methods of diagnosis are reviewed.[7,8]

CT scanning is a reliable and non-invasive modality able to adequately evaluate the pancreas and the adjacent retroperitoneal structures in all most all individuals. The CT findings in acute pancreatitis reflect the presence and extent of the retroperitoneal inflammatory process.[9-12]

In patients presenting with milder clinical forms of pancreatitis, CT shows a relatively normal pancreas or a slight to moderate increase in the size of the gland. In most cases, the entire pancreas irregular and the parenchyma appears heterogeneous with areas of abnormal enhancement. Since pancreas does not have a well-developed fibrous capsule, extravasation of pancreatic secretions in and around the pancreatic gland occurs early. On CT, the peripancreatic fat becomes hazy and dirty, showing a slight increase in density and often mild thickening of adjacent facial planes.[12]

In the more severe forms of acute pancreatitis, small fluid collections are seen in the gland, and the amount of peripancreatic inflammatory exudates is increased. The gland may be massively enlarged and may show patchy areas of lack of enhancement, necrosis, and fragmentation.[13,14] There is the total obliteration of the peripancreatic fat by large amounts of solid elements mixed with high density (20-40 Hu) fluid collections.

The sensitivity of CT to diagnose pancreatitis has been shown to be as high as 92%. The specificity of CT for acute pancreatitis is as high as 100%.[13,14] Contrast-enhanced CT (CECT) accurately depicts the infected necrotic tissue and infected fluid collections and other complications of acute pancreatitis.[15-19]

Aims and Objectives

To study the use of CT for the detection and evaluation of acute pancreatitis.

To differentiate between acute edematous and acute necrotizing pancreatitis and to grade the severity of the disease using IV contrast-enhanced CT imaging features.

By follow-up imaging to detect any complications such as (a) Pseudocyst formation, (b) Pancreatic abscess formation, (c) Pancreatic phlegmon formation, (d) Vascular complications such as pseudoaneurysm of splenic, hepatic or pancreatico-duodenal arteries. To look for any associated conditions such as (a) Fatty liver, (b) Cholelithiasis, (c) Pancreatic calcifications, and (d) Pleural effusion; to plan the surgical intervention if indicated and CT guided aspiration of the abscess if any.

MATERIALS AND METHODS

A total of 30 patients who were clinically suspected of having acute pancreatitis attending Yenepoya Medical College Hospital, Mangalore were our study participants (Figure 2). The study was conducted for a period of 1-year from November 2014 to October 2015. Computed tomographic examinations were performed in the Department of Radio-diagnosis, Yenepoya Medical College Hospital, Mangalore. All cases referred for CT scan with clinical suspicion of acute pancreatitis were included in this study. Patients were selected on the basis of Clinical history, laboratory data suggestive of acute pancreatitis or findings of acute pancreatitis on other imaging modalities, especially ultrasounds scan.

Each patient underwent a thorough clinical evaluation including a detailed history and physical examination. All the patients underwent routine baseline blood investigations, which, however, did not form a part of the study. All the study participants were made to undergo CECT scan as the radiologic examination after taking proper informed consent for the same.

The study was performed using GE's 16 slice MDCT CT machine.

RESULTS

In our study, a total 30 patients were studied using CT scan, who were suspected to have acute pancreatitis. Among them, 24 (80%) were males and 6 (20%) were females (Table 1). In our study, 25 out of 30 (83.3%) patients had enlargement of the pancreas with focal enlargement seen in 11 patients (36.6%) (Figure 3) while the 14 patients (46.6%) showed diffuse enlargement. The contour of the pancreatic gland was irregular in 20 (66.6%) patients while in 10 (33.3%) it was regular. The density of the pancreatic gland was normal in 3 (10.0%) patients; focally hypodense in 20 (66.6%) of patients, generalized hypodensities in 5 (16.6%) patients, and the entire gland was distorted in 2 patients (6.6%). 21 of 30 patients (70%) showed peripancreatic fat stranding with or without phlegmonous changes (Figure 4).

Necrosis of the pancreatic gland parenchyma was seen in 14 (46.6%) patients. 8 patients (26.6%) showed <30% necrosis. 3 patients (10%) showed 30-50% necrosis, and 3 patients (10%) showed more than 50% necrosis (Figure 5 and Table 2).

By considering the grading and the extent of pancreatic necrosis CT severity index (CTSI) was calculated. CTSI = Grades A to E patients were assigned 0-4 points plus 2 points for 30% necrosis, 4 points for 30-50% necrosis, and 6 points for more than 50% patients (Table 3). CTSI score of 0-3 was seen in 16 patients (53.3%), CTSI of 4-6 was seen in 8 patients (26.6%), and CTSI of 7-10 was seen in 6 patients (20%). 22 cases showed ascites and pleural effusion. However, the quantity of free fluid was more in

Table 1: Age and sex distribution of acute pancreatitis

Age (years)	Male (%)	Female (%)
0-10	0 (0)	0 (0)
10-20	2 (6.66)	0 (0)
20-30	4 (13.33)	0 (0)
30-40	7 (23.33)	0 (0)
40-50	4 (13.33)	1 (3.33)
50-60	4 (13.33)	2 (6.66)
60 and above	3 (10)	3 (10)

Table 2: CT signs of acute pancreatitis

Sign	*N* (%)
Gland	
Normal	5 (16.6)
Diffuse enlargement	14 (46.6)
Focal enlargement	11 (36.6)
Contour	
Regular	10 (33.3)
Irregular	20 (66.6)
Density	
Isodense	3 (10)
Focal hypodensity	20 (66.6)
Generalized hypodensities	5 (16.6)
Distorted architecture	2 (6.6)
Necrosis (%)	
<30	8 (26.6)
30-50	3 (10)
>50	3 (10)
Peripancreatic changes	21 (70)
Presence of gas/abscess	4 (13.3)
Phlegmonous changes	14 (46.6)
Pseudocyst formation	4 (13.3)
Pseudoaneurysm	1 (3.3)
Ascites	22 (73.3)
Pleural effusion	22 (73.3)

CT: Computed tomography

Table 3: Distribution of patient of acute pancreatitis according to the Grade of pancreatitis

Grade	No of patients (%)
A	3 (10)
B	7 (23.3)
C	15 (50)
D	1 (3.3)
E	4 (13.3)

Grade A: Normal pancreas, Grade B: Focal or diffuse enlargement of the gland, including contour irregularity, non-homogenous attenuation of the gland, dilatation of the pancreatic duct. Grade C: Intrinsic pancreatic abnormality associated with haziness and streaky densities representing inflammatory changes in the peripancreatic fat. Grade D: Single ill-defined fluid collection. Grade E: Two or multiple poorly defined fluid collections or presence of gas within the pancreas

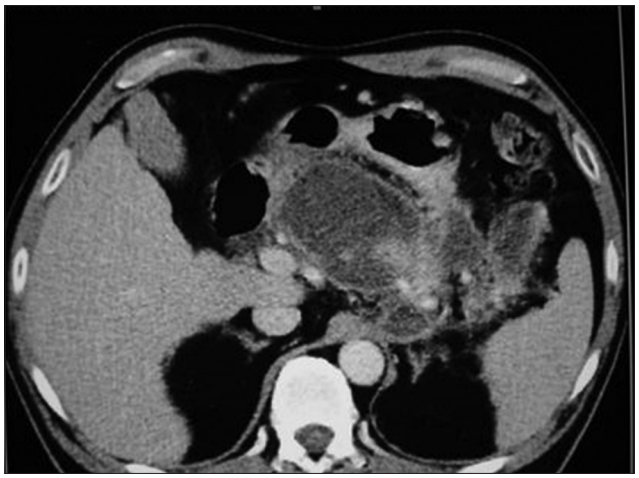

Figure 1: A case of resolving acute pancreatitis with developing pseudocyst in the body and tail regions of the pancreas

Figure 2: Mesenteric fat stranding with phlegmon formation in ongoing acute pancreatitis

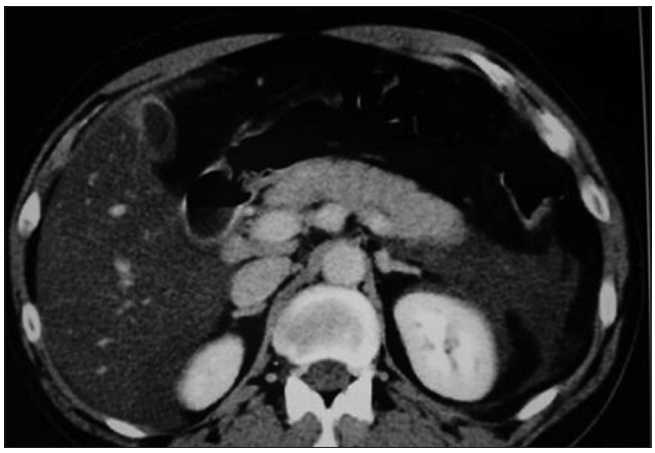

Figure 3: Case of Grade "B" pancreatitis with fatty liver with focal inflammation of peripancreatic fat around the tail of the pancreas due to alcoholic pancreatitis

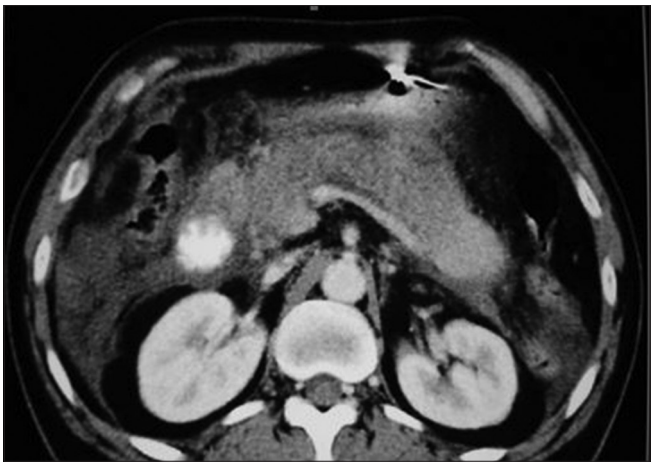

Figure 4: Mesenteric fat stranding with thickening of Gerota's fascia secondary to pancreatitis

Figure 5: Acute necrotizing pancreatitis with complete necrosis of pancreas

severe cases, i.e., Grades C-E. Phlegmonous changes were seen in 14 cases, the lesser sac was involved in 12 cases, pararenal space was involved in 5 cases, and involvement of mesocolon and mesentery was seen in 4 cases. Out of these 14 cases, 7 cases had shown the involvement of more than one anatomical site.

In cases of acute pancreatitis who had persistent symptoms or who were suspected to have pseudocyst, a repeat scan was performed. In our study, 4 cases showed pseudocyst formation. All the cases of pseudocyst were a complication of Grade "D" or Grade "E" pancreatitis. One case of Grade "C" pancreatitis had a complication in the form of pseudoaneurysm of the splenic artery. This case presented with persistence of pain abdomen following diagnosis of acute pancreatitis with an initial CT scan. 4 cases showed significant fatty liver. 5 cases showed gall bladder calculus. One case was a renal transplant recipient. This patient was thought to have azathioprine-induced acute pancreatitis. One case had pancreatic calcification. Abscess was seen within the pancreatic tissue in 4 cases (13.3%). In the plain and contrast-enhanced CT scan, all these findings were shown conclusively. All the cases in our study were followed till

recovery. The number of days of hospitalization was noted. Out of 30 patients, in our study, 4 cases were expired due to the complications of the disease. Out of these 4 cases, 3 were of Grade "E" pancreatitis. One case was of Grade "C" pancreatitis which was a case of post renal transplant recipient who was on azathioprine. In four cases, ERCP was performed, and removal of the bile duct calculus was performed. In 3 cases, surgical drainage of the collection was performed. One case of pseudoaneurysm of splenic artery was lost for follow-up (Figure 6). One patient developed partial thrombosis of the portal vein as a complication of acute pancreatitis (Figure 7). Though chronic pancreatitis was not part of our study, we had one case of acute pancreatitis that had preexisting asymptomatic pancreatic calcifications. It was a 28-year-old male, who was asymptomatic till then, presented with acute severe pain abdomen of 2 days duration. CT scan of the abdomen revealed normal sized pancreas with peripancreatic fat stranding, thickening of Gerota's fascia, Ascites, and bilateral pleural effusion (Figure 8). Furthermore, there was a minimal area of necrosis. Multiple small areas of calcifications were noted in the body and tail region of the gland. The patient was hospitalized for 12 days and treated conservatively. The patient recovered completely without any residual exocrine or endocrine insufficiency.

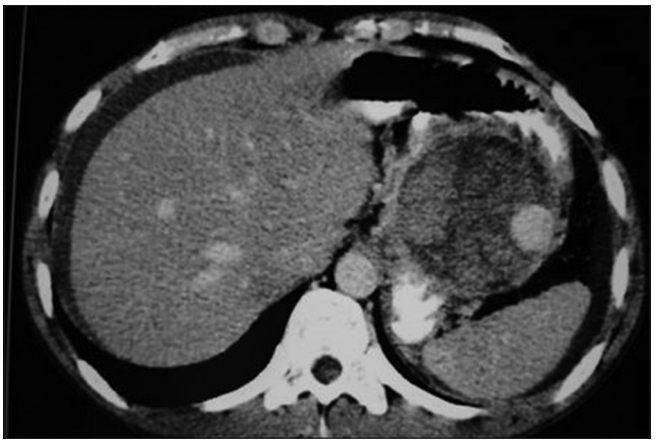

Figure 6: Pseudoaneurysm of splenic artery - a delayed complication of acute pancreatitis

Figure 7: Focal pancreatitis involving the head of the pancreas with dilated pancreatic duct. Note was made about portal vein thrombosis, as a complication of acute pancreatitis

Figure 8: Acute exacerbation of chronic pancreatitis

DISCUSSION

Our study consisted of 30 patients who were suspected to have acute pancreatitis by clinical examination and laboratory parameters and referred for CECT examination of the abdomen.

We used non-ionic water-soluble contrast medium and were able to get good contrast enhancement of the normal pancreas. Since ours was an MDCT scan machine, our results were better than the results Zwicher *et al*.[20-24]

None of the 30 patients developed any adverse reaction to the IV contrast medium. All patients were observed for 3 h after injecting IV contrast medium.

Among these 30 patients, 24 were males and 6 were females. Thus, an increase in the percentage of males in the study could be attributed to alcoholism, which was the most common cause of pancreatitis.[25-28]

The peak age of incidence was noted in 30-40 years. This correlates with other studies[29-33] in which mean age was 38 years. Two patients were in the age group of 10-20 years. Out of these, one was the recipient of transplant kidney and was on azathioprine. He had developed azathioprine-induced pancreatitis. The other patient was a 16-year-old boy who had developed post-traumatic pancreatitis.

25 out of 30 (83.3%) patients had enlargement of the pancreas. 11 of these (36.6%) showed focal enlargement and

the rest (14 patients - 46.6%) showed diffuse enlargement. This correlated with the previous studies[14,34,35] that reported pancreatic gland edema in 90% of their patient (Table 4).

Peripancreatic fat stranding was noted in 21 cases (70%). Phlegmonous changes were evident in 14 patients (46.6%) with the involvement of the lesser sac, mesentery transverse mesocolon, and anterior Pararenal spaces. 7 cases showed the involvement of more than one anatomical site. These statistics are consistent with phlegmonous spread of pancreatitis described by other workers in 2/3rd of their patients (Table 5).[14,35]

In our study, 3 patients (6.6%) had Grade "A,"(Figure 9) 7 patients had Grade "B" (23.3%) (Figure 10), 15 patients (50%) had Grade "C," (Figure 11) 1 patient (3.3%) had Grade "D," (Figure 12) and 4 patients (13.3%) had Grade "E" pancreatitis (Figure 13 and Table 6).

Balthazar *et al.* (1985)[13] reported the following Grade "A" in 14.5%, Grade "B" in 29.9%, Grade "C" in 25%, Grade "D" in 14.5%, and Grade "E" in 27.7% of cases.

We calculated the CTSI as given by Balthazar *et al.* in their 1990 series.[36] Grades A to E patients were assigned 0-4 points plus 2 points for necrosis of <30%, 4 points for necrosis 30–50%, and 6 points for >50% necrosis of the pancreatic gland. This calculated CTSI grading into three categories (0-3, 4-6, and 7-10 points) more accurately reflects the early prognostic value of CT. They found that patients with a CTSI of 0-2 had no mortality and 4% morbidity. In contrast, a CTSI of 7-10 yields a 17% mortality and 92% complication rate.

In our study, CTSI of 0-3 as seen in 16 (53.3%) patients, 4-6 was seen in 8 patients (26.6%), and 7-10 was seen in 6 patients (20%).

Out of 4 patients, who expired, 3 had CTSI of more than 7. The other one was a boy with CTSI of 2 was organ transplant recipient. The correlation between CTSI and mortality is consistent with the results of Balthazar *et al.*

All the patients with CTSI of <6 recovered well (except the one who was the recipient of organ transplant who was on azathioprine - who expired).

Patients with CTSI of <3 had no complications and the number of days of hospitalization was less.

Ascites and pleural effusion of various severities were seen in 22 patients. Quantity of ascites and pleural effusion were more in Grades C, D, and E pancreatitis.

Table 4: Distribution of patients of acute pancreatitis according to CTSI

CISI	No of patients (%)
0-3	16 (53.3)
4-6	8 (26.6)
7-10	6 (20)

CTSI: Computed tomography severity index

Table 5: Distribution of phlegmonous changes according to anatomical site

Site	No
Mesentery/mesocolon	4
Pararenal space	5
Lesser sac	12
Psoas muscle	0

Table 6: Distribution of mortality among cases of pancreatitis

Grade	Cases
A	0
B	0
C	1
D	0
E	3

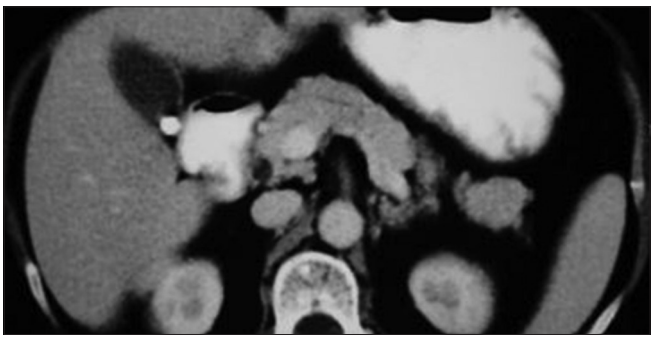

Figure 9: Grade "A" pancreatitis; normal contrast-enhanced computed tomography morphology of pancreas with elevated serum amylase. Incidental gallbladder calculus noted

Though chronic pancreatitis was not part of our study, we had one patient who had presented with acute severe pain abdomen for the first time in his life who did not have any risk factor for developing pancreatitis. He was diagnosed to have pancreatitis based on clinical parameters and serum amylase and lipase parameters. CT scan of the abdomen showed normal sized pancreas with peripancreatic fat stranding, ascites, and pleural effusion. There were multiple tiny areas of calcification in the body and tail region of the pancreas. Based on clinical and imaging parameters, he was diagnosed to have acute on chronic pancreatitis. According to various studies, pancreatic calcifications were the most consistent feature of chronic pancreatitis.[37-46]

Figure 10: Grade "B" pancreatitis with cholelithiasis

Figure 12: Grade "D" pancreatitis

Figure 11: Grade "C" pancreatitis

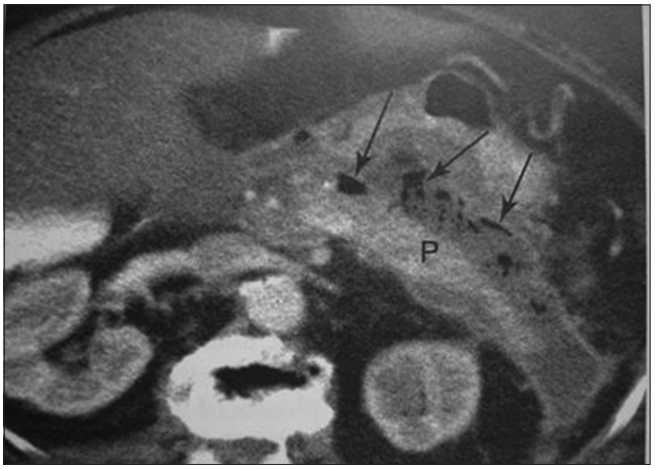

Figure 13: Grade "E" (emphysematous) pancreatitis

Limitations of the Study

Since ours was a tertiary care hospital, most of the cases had some data suggestive of pancreatitis before admission. So, all the cases, which are part of our study, had pancreatitis of various severities. In primary and secondary care centers, the clinically suspected cases of acute pancreatitis may turn out to some other diagnosis.

Our sample size was 30, which is relatively small in number.

Because we received most of our cases after a latent period of 24 h after the onset of symptoms, in our study, the CT scan was performed about 48-72 h after the onset of symptoms. If imaging was performed before 24 h of onset of symptoms, the changes of acute pancreatitis might not have developed.[45]

CONCLUSION

This descriptive study of patients with clinically suspected acute pancreatitis using CT depicted full spectrum of appearances. In all the cases, CT scan revealed the exact morphological appearance of the disease. It also helped in diagnosing other associated findings such as fatty liver, gall bladder calculus, common bile duct calculus, pancreatic duct calculus, ascites, pleural effusion, portal vein thrombosis, and pancreatic duct dilatation. CT scan of the abdomen also revealed most of the local complications such as peripancreatic fat stranding, phlegmonous changes pancreatic pseudocyst, and pseudoanuerysm of the splenic artery. Furthermore, CT scan helped to rule out any other associated disease or complication suspected. CECT was very useful in staging acute pancreatitis using

various CT numerical grading systems. All the patients in our study were categorized into various stages based on Balthazar criteria and CTSI. The CT scan classification of the patients with acute pancreatitis into various grades helped in accurate prediction of prognosis in these patients.

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