

Assessment of Severity of Acute Pancreatitis on Multidetector Computed Tomography and its Correlation with Serum C-Reactive Protein Levels

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A B S T R A C T

Introduction: The pathological spectrum of acute pancreatitis varies from edematous pancreatitis, which is usually a mild and self-limiting disorder, to necrotizing pancreatitis, in which the degree of pancreatic necrosis correlates with the severity of the attack and its systemic manifestations. Contrast enhanced computed tomography (CECT) is the investigation of choice for detection of pancreatic necrosis and other complications of acute pancreatitis. The purpose of present study was to study the role of Multidetector Computed Tomography (MDCT) in assessing the severity of acute pancreatitis by computed tomography severity index and its correlation with serum C-reactive protein levels at 48 hrs of admission.

Material and Methods: The present study was conducted on a total of 60 patients with clinical diagnosis of acute pancreatitis. Contrast enhanced MDCT was performed in all patients and severity of pancreatitis was graded according to Balthazar score and patients were categorised into mild, moderate and severe pancreatitis according to CT severity index. Radiological severity was then compared with the CRP levels measured at 48 hours of admission.

Results: There were no patients in grade A, 1 patient was in grade B, 7 patients were in grade C and 11 patients were in grade D. Out of 41 patients of grade E pancreatitis, 20 patients (48.8%) showed evidence of necrosis. 17 patients were having CTSI between (0-3), and 26 patients were having CTSI between (4-6). The remaining 17 patients were categorised as having severe pancreatitis with CTSI between (7-10). Out of 60 patients, 37 patients (61.7%) had CRP levels more than 150 mg/l and remaining 23 patients (38.3%) had CRP levels less than 150 mg/l. Out of 26 patients with CTSI 4-6 (moderate pancreatitis), 20 (77%) patients had CRP levels more than 150 mg/l. Among the remaining 17 patients with CTSI 7-10 (severe pancreatitis), all patients (100%) had CRP levels more than 150 mg/l.

Conclusion: Contrast enhanced MDCT examination is very helpful in diagnostic evaluation of acute pancreatitis and for detection of necrosis and associated complications. CCRP is an easily measurable serological marker which has good correlation with severity of pancreatitis.

Keywords: MDCT, Pancreatitis, Necrosis, CRP

INTRODUCTION

Diseases of pancreas have very variable presentation and hence imaging plays an important role in the diagnosis and management of pancreatic disease. Modalities for imaging the pancreas range from plain x-rays to contrast studies, ultrasonography (USG), endoscopic ultrasound, endoscopic retrograde cholangio-pancreaticography (ERCP), computed tomography (CT) and magnetic resonance imaging. USG is a good non-invasive method for evaluating pancreatitis secondary to the presence of gall stones or common bile duct (CBD) stones, however it is of limited value in patients, who are obese and bulky, and in whom there is extensive bowel

gas. Ultrasonographically the diagnosis of acute pancreatitis includes the increase in the volume of pancreas, structural changes in the parenchyma and significant decrease in echoes.¹ Abnormal USG findings are seen in 33% to 90% of patients with acute pancreatitis.²

However, in recent years endoscopic ultrasound (EUS) has emerged as a very useful diagnostic modality in the evaluation of patients with acute pancreatitis (AP). Studies have shown EUS to be highly accurate in the diagnosis of gallstone related pancreatitis (including microlithiasis), chronic pancreatitis, pancreatic tumors and other causes of idiopathic AP which have negative or inconclusive results as assessed by other imaging methods.³

CECT of abdomen and pelvis is the standard imaging modality for evaluating acute pancreatitis and its complications. Both intravenous and oral contrast are administered. CT findings in acute pancreatitis include diffuse or segmental enlargement of pancreas, irregularity of pancreatic contour with obliteration of peripancreatic fat planes, heterogenous appearance and areas of decreased density within the pancreas or outside the gland in lesser sac and in pararenal spaces.⁴ Multidetector CT (MDCT) offers further advantages with rapid scanning and excellent multiplanar reconstructions.

C-reactive protein is an important prognostic marker of pancreatic necrosis with the highest sensitivity and negative prognostic value (94.1% and 95.7% respectively) given the cut-off is 110 mg/l. The patients with C-reactive protein below 110 mg/l are low risk to develop pancreatic necrosis.⁵ The present study was conducted to assess the role of MDCT in assessing the severity of acute pancreatitis by computed tomography severity index/ modified computed tomography severity index and its correlation with serum C-reactive protein levels.

MATERIAL AND METHODS

The present study was conducted in Department of Radiodiagnosis, Rajindra Hospital, Patiala on 60 patients with clinical diagnosis of acute pancreatitis referred from various departments of hospital.

Inclusion criteria

1. Patients of all ages and both sexes were included.
2. Only indoor patients with typical clinical symptoms were included.

Exclusion criteria

1. Cases having any other inflammatory disease.
2. Outdoor cases
3. Patients who did not give consent or whose serum creatinine value >1.5mg%

A detailed clinical history was taken from all patients. Clinical diagnosis was based on typical features such as epigastric pain radiating to back and associated nausea and vomiting. Patients were examined for associated physical signs such as tenderness in the epigastrium.

Hematological investigations Hb, TLC, DLC, Blood Sugar, Serum Calcium, Blood Urea, Serum Creatinine and liver function tests were done in all patients.

Biochemical investigations relevant to the diagnosis of acute pancreatitis i.e. Serum Amylase were also done.

Technique

The CECT scan examination was conducted on Siemens Somatom Emotion, six slice, third generation spiral CT machine. All patients were called with at least 6 hours of fasting. A written consent was obtained from each patient after explaining the possibility of contrast reaction. Orally or through Ryle's tube water soluble iodinated contrast (Diatrizoate Meglumine and Diatrizole sodium injection USP) diluted in 700ml to 1000ml of water was given before CT examination.

The patient was placed on the gantry table in supine position with both arms above the head, 6mm to 8mm sections were

obtained. Contrast scan was obtained by injecting 80ml iodinated contrast at the rate of 2.5ml per second using a pressure injector via 20 G angiocath placed in antecubital vein. Patients were scanned in the parenchymal phase which was around 50-60 sec after the start of injection.

The topogram was taken first and from topogram, the region of pancreas was selected. A reference section was taken to confirm the pancreatic position and plain sections were taken. After contrast administration, scan was done from the domes of diaphragm to the lower poles of kidneys. If pancreatic inflammation appeared to extend inferior to the lower pole of kidneys, the remainder of abdomen and whole of the pelvic area was included to see the extent of spreading inflammation or any collection. Multiplanar reconstructions were performed wherever applicable.

The axial sections thus obtained were studied in detail for the size of pancreas, contour of pancreas, enhancement or areas of necrosis, intrapancreatic fluid collection, peripancreatic inflammation, Gerota's fascia, lateroconal fascia, mesentery, mesocolon and extrapancreatic fluid collection. Peripancreatic vessels i.e. splenic vein, portal vein, splenic artery, superior mesenteric artery and vein were studied carefully for evidence of any thrombosis or pseudoaneurysm.

Based on CT findings, the pancreatic inflammation was divided into five grades (grade A-E) according to Balthazar classification.⁶

Then CT severity index (CTSI)⁷ was calculated by assigning 0-4 points to Grade A-E pancreatitis plus

2 points for less than 30% necrosis

4 points for 30-50% necrosis

6 points for more than 50% necrosis

The CT findings of acute pancreatitis were graded according to CT severity index and categorized as: mild (0-3), moderate (3-6) and severe (7-10)

The CT findings of acute pancreatitis were graded according to modified CT severity index and categorized as mild (0-2 points), moderate (4-6 points), or severe (8-10 points) pancreatitis.

Radiological severity was then compared with the CRP levels measured at 48 hours of admission using appropriate statistical methods.

RESULTS

The present study was conducted on 60 patients having a strong clinical suspicion of acute pancreatitis. Contrast enhanced computed tomography of abdomen for the assessment of disease severity. The radiological severity was then correlated with serum CRP levels taken at 48 hours of admission.

In this study group (n=60), there were 41 males and 19 females with M:F ratio=2. Out of 60 patients included in our study, 15 patients (25%) were below the age of 30 years, 27 patients (45%) were between 31-40 years, 10 patients between 41-50 years, 7 patients between 51-60 years.

The most common cause of acute pancreatitis in our study was alcohol abuse, seen in 27 patients (45%). In 11 patients, acute pancreatitis was associated with biliary calculi and in 2 patients, trauma was the etiological factor for pancreatitis. However, in the remaining 20 patients (33.3%), no cause of

CTSI	Patients (n=60)	Patients with CRP>150 mg/l	% Age
0-3 (Mild)	17	0	0
4-6 (Moderate)	26	20	77%
7-10 (Severe)	17	17	100%
Total	60	37	62%

Table-1: Showing distribution of cases according or CTSI and CRP levels

	Patients (n=60)	PAntients with complications (% age)	Mortality (% age)
CTSI (0-3)	17	2 (11.7%)	0 (0%)
CTSI (4-6)	26	10 (38.4%)	1(3.8%)
CTSI (7-10)	17	13 (76.4%)	7(41%)
Total	60	25 (42%)	8(13.3%)

Table-2: Showing incidence of complications, mortality and CTSI score

pancreatitis could be found and was treated as idiopathic. Depending upon the morphological/inflammatory changes in pancreatic, peripancreatic and extrapancreatic regions on CT scan, the patients in this study group were categorized according to Balthazar's grading system into five grades i.e. grades A,B,C,D and E. Maximum number of patients (41, 68.3%) included in the study were of grade E. There were no patients in grade A, 1 patient was in grade B, 7 patients were in grade C and 11 patients were in grade D. Out of 41 patients of grade E pancreatitis, 20 patients (48.8%) showed evidence of necrosis. Pancreatic necrosis was present in 2 of 11 patients of grade D pancreatitis and no necrosis was seen in grade B (1) and C (7) patients. Out of total 20 patients of grade E with necrosis, in 8 patients degree of necrosis was more than 50%, 9 patients had necrosis between 30% to 50% and remaining 3 patients had necrosis less than 30%. All patients (n=2) of grade D pancreatitis with necrosis had less than 30% parenchymal necrosis.

On the basis of CTSI patients were divided into three categories i.e. mild (0-3), moderate (4-6) and severe (7-10). In our study, 17 patients were having CTSI between (0-3), and 26 patients were having CTSI between (4-6). The remaining 17 patients were categorised as having severe pancreatitis with CTSI between (7-10) (Figures 1 & 2). Pleural effusion was seen in 37 (62%) patients. It was bilateral in 23 (38%) patients and exclusively left sided in 10 (17%) patients. Exclusively right sided pleural effusion was seen in 3 (5%) patients only.

Out of 60 patients, 37 patients (61.7%) had CRP levels more than 150 mg/l and remaining 23 patients (38.3%) had CRP levels less than 150 mg/l. None of the patients with CTSI 0-3 (mild pancreatitis) had CRP levels more than 150 mg/l. Out of 26 patients with CTSI 4-6 (moderate pancreatitis), 20 (77%) patients had CRP levels more than 150 mg/l . Among the remaining 17 patients with CTSI 7-10 (severe pancreatitis), all patients (100%) had CRP levels more than

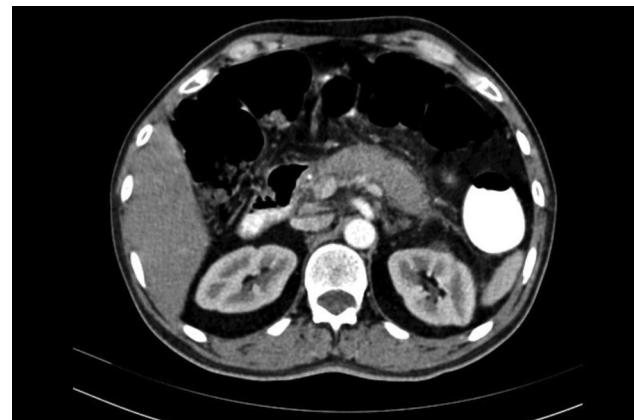


Figure-1: Case of mild acute pancreatitis (CTSI-2). Axial CECT image showing homogeneously enhancing pancreas. Peripancreatic fat stranding is seen and left Gerota's fascia is thickened.



Figure-2: Case of severe acute pancreatitis. Axial CECT image showing enlarged pancreas with irregular margins and non-enhancing areas (>50%) in the head, body and tail region, peripancreatic fat stranding is present. Left Gerota's fascia and lateroconal fascia are thickened.

150 mg/l. (Table 1)

In this study group, 42% patients had morbidity in the form of various complications. 7 patients (11.6%) had pseudocysts, 10 patients (16.6%) had walled-off necrosis (WON) which became infected in 2 patients. 6 patients (11%) had multiple organ failure (MOF) and 2 patients (3.3%) had vascular complications in form of splenic vein thrombosis.

2 patients (11.7%) with mild pancreatitis, CTSI (0-3) developed pseudocysts and both of them recovered. 10 patients (38.4%) of moderate pancreatitis CTSI (4-6) developed complications (5 pseudocysts, 3 walled-off necrosis, 1 infected necrosis, 1 vascular) and the patient with infected necrosis expired (3.8% mortality). Incidence of complications and mortality rate, both were highest in severe pancreatitis (7-10). 13 patients (76.4%) developed complications (5 WON, 1 infected necrosis, 6 MOF, 1 vascular). Out of these it proved fatal in 7 patients i.e. 41% mortality. Total incidence of mortality in this study group was 13.3%.

DISCUSSION

68% of patients in our study were males. This can be explained by prevalence of alcoholism amongst males in our series. This is in agreement with study by Balthazar et al⁶ in which there were 75% male patients. Alcoholism and biliary calculi were the most common identifiable causes of acute pancreatitis as has been seen in many studies.⁸⁻¹⁰

On the basis of severity of pancreatitis on CT scan patients were categorized into 5 grades, A to E (Balthazar's grades). In our study, 68.3% patients were categorized as grade E. The presence of higher number of patients in grade E in our study could be attributed to the fact that our hospital being a tertiary centre, very sick patients having severe pancreatitis were referred to us.

In the present study, in 17 patients pancreatitis was mild and CTSI was between 0-3, in 26 patients CTSI was between 4-6 indicating pancreatitis to be of moderate severity. In remaining 17 patients CTSI was between 7-10, indicating severe pancreatitis. Patients with CTSI of 0-3 exhibited no mortality and 11.7% morbidity. Those with CTSI of 4-6 exhibited 3.8% mortality and 38.4% morbidity. The incidence of mortality and morbidity was very high in patients with CTSI of 7-10. They exhibited 41% mortality and 76.4% morbidity. This is similar to study to other studies^{7, 11} except for higher mortality on severe pancreatitis in our study. This could be attributed to the fact that our hospital being a tertiary centre, clinically very sick patients having severe pancreatitis were referred to us. Thus, CTSI is a good prognostic indicator in predicting the final outcome in acute pancreatitis.

In our study, pancreatic necrosis was found in 22 patients (36.6%) which is higher than that in other studies.^{7, 12, 13} This difference is due to higher proportion of severe pancreatitis in our study group. Patients with necrosis had 36% mortality and 77% complication rate while patients without necrosis had 0% mortality and 21% morbidity. This was in comparison to the study by Balthazar et al⁷ which showed that patients with necrosis had 23% mortality and 82% of complication rate while patients without necrosis had 0% mortality and

6% morbidity.

In present study, all the 22 patients with necrosis had serum CRP levels in excess of 150 mg/l taken at 48 hours. It showed that chances of pancreatic necrosis are less with CRP levels < 150 mg/l. Similar observations have been made by other authors using slightly different cutoff values of CRP.^{5, 14}

In this study group, none of the patients with CTSI (0-3) had CRP levels more than 150 mg/l taken at 48 hours. Out of 26 patients with CTSI (4-6), 20 (77%) had CRP levels more than 150 mg/l. Among the remaining 17 patients with CTSI (7-10), all patients (100%) had CRP levels more than 150 mg/l. Thus 86% (37/43) of patients having CTSI score greater than 3 had a serum CRP level in excess of 150 mg/l taken at 48 hours. A similar correlation was found between CTSI and serum CRP levels in another study by Gurley et al.¹⁵

CONCLUSION

In conclusion, contrast enhanced MDCT examination is very helpful in establishing or confirming the clinical diagnosis of acute pancreatitis. It is very sensitive in depicting morphological / inflammatory changes in pancreatic, peripancreatic and extrapancreatic region. C-reactive protein has a good correlation with the radiological severity of acute pancreatitis. CRP is an easily measurable simple method which can be used to estimate the severity of pancreatitis.

REFERENCES

1. Badea R. Ultrasonography of Acute Pancreatitis – An Essay in images. Romanian Journal of Gastroenterology 2005;14(1):83-9.
2. Jeffery RB. Sonography in acute pancreatitis. Radiol Clin North Am. 1989;27(1):5-17.
3. Kotwal V, Talukdar R, Levy M, Vege SS. Role of endoscopic ultrasound during hospitalization for acute pancreatitis. World J Gastroenterol 2010;16(39):4888-91.
4. Clavien PA, Hauser H, Meyer P, Rohner A. Value of contrast enhanced computerized Tomography in the Early Diagnosis and Prognosis of Acute Pancreatitis. American Journal of Surgery 1988;155:457-66.
5. Barauskas G, Svagzdys S, Maleckas A. C-reactive protein in early prediction of pancreatic necrosis. Medicina (Kaunas) 2004;40(2):135-40.
6. Balthazar EJ, Ranson JHC, Naidich DP, Megibow AJ, Caccavale R, Cooper MM. Acute pancreatitis: Prognostic Value of CT. Radiology 1985;156:767-72.
7. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JHC. Acute pancreatitis: value of CT in Establishing Prognosis. Radiology 1990;174(2):331-6.
8. Kim YS, Lee BS, Kim SH, Seong JK, Jeong HY, Lee HY. Is there correlation between pancreatic enzyme and radiological severity in acute pancreatitis? World J Gastroenterol 2008;14(15):2401-05.
9. Simmons MZ, Miller JA, Zurlo JV, Levine CD. Pleural effusions associated with acute pancreatitis: Incidence and appearance based on computed tomography. Emergency Radiology 1997;4(5):287-89.
10. Bohidar NP, Garg PK, Khanna S, Tandon RK. Incidence, etiology, and impact of Fever in patients with

- acute pancreatitis. *Pancreatology* 2003;3(1):9-13.
11. Mir MA, Bali BS, Mir RA, Wani H. Assessment of the severity of acute pancreatitis by contrast-enhanced computerized tomography in 350 patients. *UlusTravmaAcilCerrahiDerg.* 2013;19(2):103-08.
 12. Khanna AK, Meher S, Prakash S, Tiwary SJ, Singh U, Srivastava A et al. Comparison of Ranson, Glasgow, MOSS, SIRS, BISAP, APACHE-II, CTSI Scores, IL-6, CRP, and Procalcitonin in Predicting Severity, Organ Failure, Pancreatic Necrosis, and Mortality in Acute Pancreatitis. *HPB Surgery* 2013;367581(2):1-10.
 13. Finley JW. Respiratory complications of acute pancreatitis. *Am Surg*1969;35(1):591-98.
 14. Alfonso V, Gómez F, López A, Moreno-Osset E, del Valle R, Antón MD, et al. Value of C-reactive protein level in the detection of necrosis in acute pancreatitis. *Gastroenterol Hepatol.* 2003;26(5):288-93.
 15. Gurleyik G, Emir S, Kilicoglu G, Arman A, Saglam A. Computed tomography severity index, APACHE II score, and serum CRP concentration for predicting the severity of acute pancreatitis. *JOP* 2005;6(3):562-67.

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