

Role of Computed Tomography in Acute Pancreatitis and its Complications among Age Groups

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Abstract

Objective: To determine value of CT scan in diagnosis of acute pancreatitis, its complications and to correlate with severity among different age groups.

Methods: The study was carried out from August 2001 to August 2002 at the Radiology Department, Aga Khan University Hospital. A total of 40 patients (33 male and 7 female) with age range from 16-71 years were divided in three groups. Group I was less than 40 years (12 patients), Group II was between 40-60 years (17 patients), and Group III was more than 60 years (11 patients). CT scans were assessed for pancreatic necrosis and its complications. CT Severity Index (CTSI) was calculated according to Balthazar's method.

Results: In 17 patients with mild pancreatitis, 5 had necrosis involving one-third of pancreas. In 13 patients with severe pancreatitis, 8 had necrosis involving more than half of the pancreas and 5 had necrosis involving half of the pancreas. No significant correlation was demonstrated between moderate pancreatitis and degree of necrosis. Thirty patients had complications, 8 had mild CTSI, 9 had moderate CTSI and 13 patients had severe CTSI.

Conclusion: The study demonstrated a relationship between CTSI and severity of pancreatic damage and incidence of complications (JPMA 55:431;2005).

Introduction

Acute pancreatitis is a protean disease with a broad clinical spectrum of findings varying from mild to severe.¹ It typically presents with abdominal pain and is usually

associated with raised pancreatic enzymes in blood or urine. Presenting complaints are different in different age groups and also mortality rate is higher in older age group.² It may remain localized within the pancreas, spread to regional

tissues, or involve adjacent or remote organs. Alcoholism and biliary tract disease account for 90% of all cases of acute pancreatitis. It occurs most often in middle life. Gallstones are present in 35-60% of cases of pancreatitis, and about 5% of patients with gallstones develop pancreatitis.³ The male to female ratio is 1:3 with biliary tract disease, and 6:1 in alcoholics.

On ultrasound, pancreatic visualization is 60-78%. Acute pancreatitis may appear as hypoechoic diffuse or focal enlargement of pancreas with dilatation of duct if head is focally involved.⁴ Fluid collection may be seen in the lesser sac in approximately 60% of cases. Cholangiography in acute pancreatitis shows, long gently tapered narrowing of the CBD with prestenotic biliary dilatation. CBD may show smooth or irregular mucosal surface.^{5,6}

To date, MRI has not played a major role in the evaluation of pancreatitis. MRI can detect changes of pancreatitis and distinguish acute from chronic forms.⁷ CT scan is useful not only for the diagnosis of acute pancreatitis but also for evaluating the severity and delineating pancreatic and extra-pancreatic complications, such as, peripancreatic fluid collection, pseudocyst and pancreatic abscess. The prognostic value of computed tomography (CT) in acute pancreatitis has been previously investigated, mainly by correlating the presence and extent of peripancreatic fluid collection with the clinical severity of the disease, development of complications, and death.⁸⁻¹² Balthazar showed that patients without peripancreatic inflammation (grade A and B) have a mild uncomplicated clinical course, while patients with one or several peripancreatic collections (grade D and E) often exhibit a protracted clinical illness and high frequency of abscesses and death. Grading system of Balthazar⁹ allows identification of a subgroup of patients with acute pancreatitis in whom most serious complications will occur. The shortcoming of this system is that within this subgroup some patients (54% in his series) show spontaneous resolution of these fluid collections, whereas the other 46% of individuals who could not be identified develop complications.

Finnish and German investigators have focused on the appearance of the pancreatic gland during CT examinations with bolus administration of contrast material.^{2,13} They have shown that lack of enhancement or low CT numbers correlate well with areas of pancreatic necrosis found at surgery.

This study was conducted to determine the value of CT scan in the diagnosis of acute pancreatitis and its complications.

Patients and Methods

All patients referred to the radiology department for suspected acute pancreatitis during August 2001 to August 2002, were included in this study. Patients less than thirteen years of age, trauma and post-operative

patients were excluded.

Forty patients underwent clinical, laboratory and radiologic evaluation for acute pancreatitis including 33 males (82.5%) and 7 females (17.5%). Age range was 16 to 71 years.

In this study the patients were divided into three age groups. First group was less than 40 years (12 patients), second was 40-60 years (17 patients) and last group was more than 60 years (11 patients). Thirty three patients presented with epigastric pain, 7 had generalized abdominal pain, 13 had nausea and vomiting in addition to epigastric pain, 5 had abdominal pain and fever, 18 patients had cholelithiasis, 7 had history of alcohol intake and 15 had no known cause. Serum amylase and lipase levels were raised in all patients.

Chest X-Rays were available in 38 patients, 10 had bilateral pleural effusion, 18 unilateral effusion and 10 were normal.

Computer Tomographic Severity Index (CTSI)

The CTSI in acute pancreatitis devised by Balthazar et al⁹ was used in this study. This index analyzes the initial CT finding as a prognostic indicator of morbidity and mortality. CTSI was created by combining the two prognostic indicators, grade and degree of acute pancreatitis. In grading system, patients with grades A-E of acute pancreatitis have been assigned zero to four points. In degree system, zero point for no necrosis, two points for 30%, four points for 50% and six points for more than 50% of pancreatic necrosis (Table 1).

Table 1. CT severity index of Acute Pancreatitis.

The Index was calculated by grade+degree of necrosis points. Patients were divided into three categories: mild

	Points
Grade of acute pancreatitis	
A=Normal pancreas	0
B=Pancreatic enlargement alone	1
C=Inflammation confined to the pancreas and peripancreatic fat	2
D=One pancreatic fluid collection	3
E=Two or more fluid collection	4
Degree of pancreatic necrosis	
No necrosis	0
Necrosis of one-third of pancreas (30%)	2
Necrosis of one-half of pancreas (50%)	4
Necrosis of more than one-half of pancreas (>50%)	6

(0-3 points), moderate (4-6 points) and severe (7-10 points). A patient with CT grade D was assigned three points, if in addition, the patient had more than 50% pancreatic necrosis, an additional six points were assigned, for a total index score of 9.

CT Technique

All CT scans were performed by GE Medical System, HiSpeed CT/i. All patients received 1000 ml of oral contrast material (Gastrografin) 45-60 minutes prior to study. An additional 200-250 ml of oral contrast was given just prior to scanning. The area of scanning was from the diaphragm to iliac crest and was performed with suspended expiration following hyperventilation.

Eighty milliliter of intravenous non-ionic iodinated (300-350 mg iodine/ml) contrast material was delivered with power injector with a rate of 2-3 ml/second. Scanning was started after 60 seconds delay. The kVp was 120-140 and mAs in the range of 210-330, collimation was 7 mm with pitch of 1.5.

The CT scan was assessed for normal enhancement and non-enhancement of pancreas. Non-enhancement of pancreas represents pancreatic necrosis and is defined as a definite focal area of decreased enhancement compared with normal enhancing pancreatic parenchyma. The location of necrosis was categorized as involving the pancreatic head, body or tail. The extent of pancreatic necrosis was estimated as less than 30%, more than 30% but less than 50% and more than 50%.

CT scans were also assessed for peripancreatic inflammation, mesenteric stranding, transverse mesocolon infiltration, peripancreatic fluid collection, pseudocyst, pancreatic abscess, splenomegaly, splenic vein thrombosis, intra-splenic pseudocyst, splenic infarction, splenic necrosis, peri-renal fat stranding, peri-renal/subcapsular fluid collection and renal vein thrombosis.

Results

Of 40 patients in the study, 17 (42.5%) had mild pancreatitis (CTSI within range of 1-3), 10 (25%) had moderate pancreatitis (CTSI within range of 4-6), and 13 (32.5%) patients had severe acute pancreatitis (CTSI range of 7-10). Of 40 patients, 12 were below 40 years, 17 between 40-60 years and 11 above 60 years age. The relationship between CT severity index (CTSI) and age of patients, hospital stay and complications are summarized in Table 2.

Table 2. Correlation of CTSI with Age, Hospital stay, and Complications.

CTSI	Number of Patients	Age (Years)			Hospital Stay (Average Days)	Complication (number of Patients)
		<40	40-60	>60		
Mild	17	7	8	2	10.05	8
Moderate	10	5	4	1	12.6	9
Severe	13	0	5	8	25.12	13

Pancreatic necrosis was detected in 28 patients on the basis of CT assessment. In patients with mild acute pancreatitis, 5 out of 17 had one third necrosis of the pancreas. No patient with mild CTSI had greater than half

necrosis of the pancreas. In patients with moderate acute pancreatitis all 10 had pancreatic necrosis, 4 patients had one-third necrosis, 3 one-half necrosis and 3 more than one-half necrosis. In severe acute pancreatitis, according to CTSI, all 13 patients had pancreatic necrosis. Of these 5 had one-half necrosis and 8 had more than one-half of pancreatic necrosis.

Table 3 shows the relation of pancreatic necrosis with different age groups. No patient in the less than 40 years age group had more than half of pancreatic necrosis, while in the greater than sixty years age group most of the patients had more than half of pancreatic necrosis.

Table 3. Correlation of Age with Degree of Necrosis.

Age (Years)	Degree of Pancreatic Necrosis			No Pancreatic Necrosis
	One-third	One-half	>One-half	
Less than 40 years	3	5	0	4
Between 40-60 years	4	2	3	8
More than 60 years	2	1	8	0
TOTAL	9	8	11	12

The cause of necrosis was not associated with presence and degree of pancreatic necrosis. Of 28 patients with pancreatic necrosis 25 showed pancreatic and extra-pancreatic complications and 3 had no complications. Of 12 patients without pancreatic necrosis only 2 had complications and 10 were without complications.

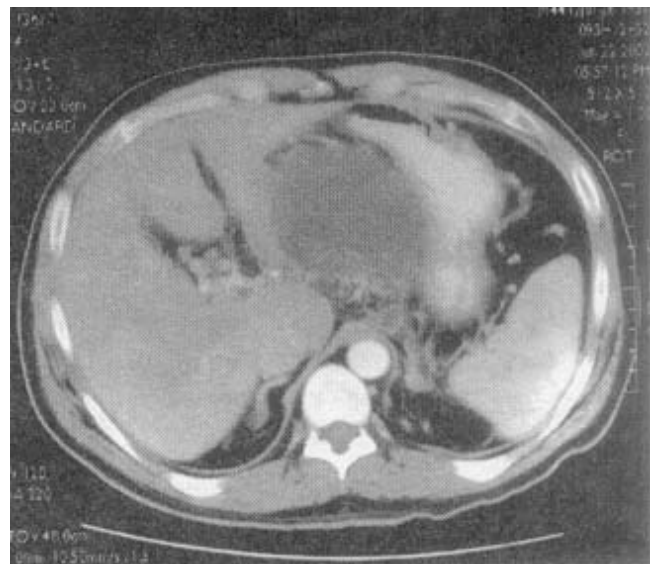


Figure 1. Pseudocyst formation lesser sac following Acute Pancreatitis.

There was a relationship between age group of patients and pancreatic and extra-pancreatic complications. Patients more than 60 years age had 90% complications compared to patients less than 40 years of age who had 66% of complications. Among 30 patients with

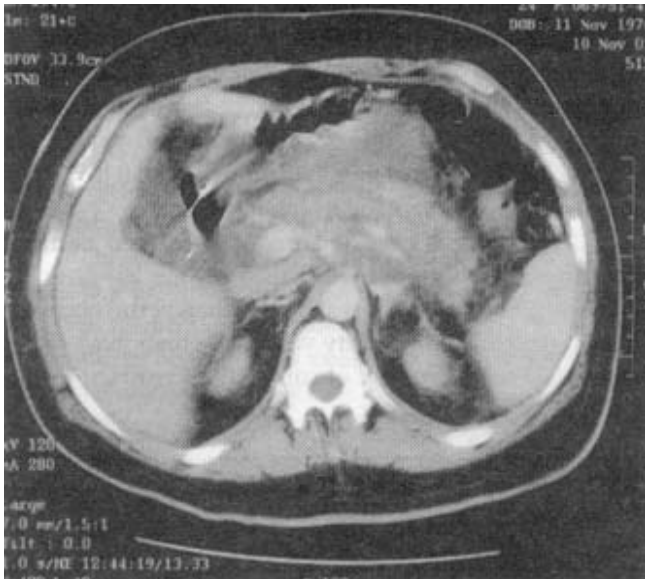


Figure 2. Edematous and swollen pancreas with peripancreatic inflammatory changes and fluid collection.

complications, 8 had mild CTSI, 9 moderate CTSI and 13 severe CTSI. Of 8 patients with mild CTSI, 4 had acute pancreatic fluid collection, peripancreatic and mesenteric inflammation and 3 patients had swollen pancreas with peri-renal fluid collection and peripancreatic inflammation. One patient had only peripancreatic inflammation, mesenteric and perirenal fat stranding.

All 9 patients with moderate CTSI had pancreatic necrosis and peripancreatic inflammation; 3 had acute pancreatic fluid collection, 3 had pseudocyst, one of them had



Figure 3. Inflammatory changes around the tail of pancreas and thickening of Gerota's fascia and latero-conal fascia.

splenic vein thrombosis and 2 had pancreatic abscess. Of the 13 patients with severe CTSI, all had pancreatic necrosis with peripancreatic and mesenteric inflammation and

perirenal fat stranding. Five had acute perirenal fluid collection, 4 had pancreatic abscess and 4 patients developed a pseudocyst.

Discussion

Acute pancreatitis is a life threatening condition with significant morbidity and mortality. The treatment depends on the accurate assessment of severity. The assessment of disease severity must be objective and early detection of pancreatic necrosis is the most important aspect in its management. Mortality rate of <1% is associated with the interstitial pancreatitis and it rises to >20% in patients with necrotizing pancreatitis. Almost all life-threatening complications occur with necrotizing pancreatitis, including secondary bacterial contamination and multi-organ failure. Secondary bacterial infection has mortality of 67% in patients with infectious necrosis of >50% of pancreas.¹⁴ Patients with pancreatic necrosis are closely monitored in the intensive care unit and follow up laboratory and imaging studies are routinely performed. An ideal or desirable detection system should have high sensitivity and positive predictive value. It should be able to detect necrosis early in the course of disease. Of all the available means for assessing the disease severity, only CT scan can reliably detect the pancreatic necrosis.

Recognition of severe pancreatic injury by means of clinical examination is unreliable. It is experienced that a good clinician can clinically diagnose acute pancreatitis in only 34% cases and with plasma levels of lipase and amylase available, in 39% of patients.¹⁵ Furthermore, the diagnosis is missed in 30-40% of patients with fatal necrotizing pancreatitis.¹⁶ Detection of urinary trypsinogen activated peptide levels has been shown to be promising in identifying patients with severe pancreatitis.¹⁷ But its clinical accuracy as an indicator of pancreatic necrosis has yet to be determined.

The criteria developed by Ranson et al¹⁸ Acute Physiology and Chronic Health Evaluation (APACHE2) are popular in clinical practice, but none of these is sufficiently sensitive or specific in identifying most patients with necrotizing pancreatitis. Conventional abdominal X-rays, barium studies and chest X-rays show indirect signs of pancreatitis but have only limited role in the early evaluation of disease severity.

Ultrasound is helpful in identifying gallstones and common duct stones; however, it is not sensitive in the early detection of pancreatic necrosis. Ultrasound has limitations because visualization of pancreas is often impaired because of overlying bowel gases. A diffusely enlarged and hypoechoic gland is consistent with interstitial edema, while extrapancreatic fluid collections in the lesser sac and anterior pararenal space can be detected in severe disease.

CT scan is sensitive in the detection of early pancreatic necrosis. Pancreatic gland necrosis is a diffuse or local area of nonviable pancreatic parenchyma that typically is associated with peripancreatic fat necrosis. Normal unenhanced pancreas has CT attenuation of 30-50 Hounsfield units and shows homogeneous enhancement with post contrast attenuation of 100-150 Hounsfield units. A focal or diffuse well-marginated zone of un-enhanced pancreas, larger than 3cm in diameter or larger than 30% of the area of pancreas, is considered a reliable CT finding for diagnosis of necrosis. CT is 80-90% accurate in the detection of pancreatic necrosis. Specificity of CT increases with increasing percentage of pancreatic necrosis. Specificity of CT is about 50%, if there are only small areas of necrosis however, in more than 30% necrosis; specificity of CT is 100%. In addition to early detection of pancreatic necrosis, there is other CT staging criteria of acute pancreatitis including grades of acute pancreatitis. There are 5 grades of acute pancreatitis from A to E. CTSI or CT severity index of acute pancreatitis is then calculated from grade of acute pancreatitis and degree of pancreatic necrosis.

CT has also its role in the management of patients with acute pancreatitis in addition to diagnosis and assessment of disease severity and assessing prognosis. CT along with ultrasound can be used for percutaneous drainage procedures. CT however, provides more information about the extent, number of peripancreatic collections and location of adjacent structures.

Conclusion

Prognosis of acute pancreatitis can be depicted by computed tomography severity index (CTSI). CT severity index has excellent correlation with development of local complications and incidence of death in patients with acute pancreatitis.

References

1. Tsushima Y, Tamura T, Tomioka K, Okada C, Kusano S, Endo K. Transient splenomegaly in acute pancreatitis. *BJR* 1999;72:637-43.
2. Fan ST, Choi TK, Lai EC, Wong J. Acute Pancreatitis in aged. *Australian & New Zealand journal of surgery* 1988;58:717-21.
3. Stear ML. Recent insights into the etiology and pathogenesis of acute biliary pancreatitis. *AJR* 1995;164:811-14.
4. Jeffery RB Jr. Sonography in acute pancreatitis. *Radiologic Clinics of North America* 1989;27: 5.
5. Jeffery RB Jr, Laing FC, Wing VW. Extrapaneatic spread of acute pancreatitis: New observations with real-time US. *Radiology* 1986;159: 707.
6. Lawson TL. Acute pancreatitis and its complications: Computed Tomography and Sonography. *Radiologic Clinics of North America* 1983;21:495-513.
7. Mitchell D. MR imaging of the pancreas. *MRI Clinics of North America* 1995;3:51-71.
8. Balthazar EJ, Freeny PC, Van Sonnenberg E. Imaging and intervention in acute pancreatitis. *Radiology* 1994;193:297-306.
9. Balthazar EJ, Robinson D, Megibow A. Acute pancreatitis: Value of CT in establishing prognosis. *Radiology* 1990;174:331-6.
10. Morteale KJ, Mergo PJ, Taylor HM, Ernst MD, Ros PR. Renal and perirenal space involvement in acute pancreatitis: Spiral CT findings. *Abdominal Imaging* 2000;25:272-8.
11. Morteale KJ, Mergo PJ, Taylor HM, Ernst MD, Ros PR. Splenic and perisplenic involvement in acute pancreatitis: Determination of prevalence and morphologic helical CT features. *J Computer Assisted Tomography* 2001;25:50-4.
12. Tsushima Y, Tamura T, Tomioka K, Okada C, Kusano S, Endo K. Transient splenomegaly in acute pancreatitis. *BJR* 1999;72:637-43.
13. Gastano DLH, Antolin S, Salovnil D, Pena D, Garcia J, Romero P. Relationship between the presenting symptoms and age in diagnosis in alcoholic and non-alcoholic chronic pancreatitis. *Rev Esp Enferm Dig* 1997;89:269-79.
14. Berger HG, Rau B, Mayer J, Pralle U. Natural course of acute pancreatitis. *World J Surgery* 1997;21:130-5.
15. McMahon MJ, Playforth MJ, Pickforth IR. A comparative study of methods for the prediction of severity of attack of acute pancreatitis. *BJS* 1980;67:22-25.
16. Corfield AP, Cooper MJ, Williamson RCN. Prediction of severity in acute pancreatitis: Prospective comparison of three prognostic indices. *Lancet* 1985;2:403-7.
17. Tenner S, Fernandez-del Castillo C, Warshaw A. Urinary trypsinogen activation peptide (TAP) predicts severity in patients with acute pancreatitis. *Int J Pancreatol* 1997;21:105-10.
18. Ranson JHC, Rifkind KM, Rose DF. Objective early identification of severe acute pancreatitis. *Am J Gastroenterology* 1974;61:443-51.