ejpmr, 2021,8(6), 474-478

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Research Article

ISSN 2394-3211

EJPMR

ACUTE PANCREATITIS AND ITS COMPLICATIONS: ROLE OF CONTRAST ENHANCED COMPUTED TOMOGRAPHY

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Article Received on 12/04/2021 **Article Revised on 02/05/2021** **Article Accepted on 23/05/2021**

ABSTRACT

Background*:* Serum amylase and lipase levels are traditionally used in the diagnosis of acute pancreatitis. However clinical course and complications of acute pancreatitis cannot be determined by enzymes levels. Contrast enhanced computed tomography is vital in assessing the course and complications of acute pancreatitis. **Methods:** In this study, total 37 patients were included who presented with clinical suspicion of acute pancreatitis. These patients were evaluated clinically, biochemical analysis was done and then CECT was done in all cases after 72 hours. CECT findings were evaluated in all the patients. Patients were classified into AIEP and ANP groups. Patients in both the groups were followed up for complications at two months interval. **Results:** 75.7% of the study population had acute interstitial edematous pancreatitis(AIEP) and 24.3% had acute necrotizing pancreatitis(ANP).On follow up, complications were significantly more in ANP group as compared to AIEP group (22.2% vs 17.8%).). 29.8% of patients had less than three fold rise in serum lipase levels at presentation despite signs of acute pancreatitis on CECT. **Conclusion:** Contrast enhanced computed tomography (CECT) is standard imaging modality for the evaluation of acute pancreatitis and its complications. Using enzyme assay of serum amylase and lipase, clinicians can establish the diagnosis but cannot evaluate for pancreatic necrosis and its vascular complications. Moreover significant subset of patients can have less than three fold rise in serum lipase and amylase level.

KEYWORDS: Acute pancreatitis; complications; contrast enhanced computed tomography.

INTRODUCTION

Pancreatitis defined as the inflammation of the pancreas and is always, associated with acinar cell injury. Acute pancreatitis is clinically characterized by acute onset of abdominal pain and a rise in the activity of pancreatic enzymes in the blood and urine.[1]

The most common etiology in adults is cholelithiasis followed by alcohol consumption. Other causes include hypertriglyceredemia, drug induced, trauma etc.[2] Majority of patients with acute pancreatitis (approximately 80% to 85%) has no complications. Only 15% to 20% will develop a complicated clinical course characterized by organ failure and or local complications.[3]

The initial phase of acute pancreatitis is characterized by intra-pancreatic digestive enzyme activation and acinar cell injury which involves the first few hours of AP. The second phase is characterized by an intrapancreatic inflammatory reaction and varying degrees of acinar cell necrosis which starts approximately 12-72 hours after the initial phase. Finally, the third phase is characterized by further progression of the pancreatic injury and the appearance of extrapancreatic changes including SIRS and ARDS.[4]

Acute pancreatitis is clinically defined by at least the first two of three features.
(a) Abdominal pain suggestive of pancreatitis (epigastric pain often radiating to the back).
(b) Serum amylase and lipase levels three or more times of normal(imaging is to be used if the elevated values are, 3 times normal i; e more than 180 U/L).
(c) Characteristic findings on CT, magnetic resonance (MR) imaging, or trans-abdominal ultrasonographic (US) studies.

If acute pancreatitis is diagnosed on the basis of the first two criteria with no systemic sign of severe systemic inflammatory response syndrome or persistent organ failure, contrast material–enhanced CT may not be necessary for determining patient care.[5]

Baseline ultrasonography should be performed in all patients to evaluate the biliary tract and in particular to

determine if the patient has gallstones and/or a stone in the common bile duct (CBD).

Computed tomography (CT) should be performed selectively when.
1) A patient presents with substantial abdominal pain and a broad differential diagnosis that includes acute pancreatitis.
2) In patients with suspected local complications of acute pancreatitis (e.g., peritonitis, signs of shock, suggestive ultrasound findings).

Acute pancreatitis can be accompanied by pancreatic parenchymal or peripancreatic collections. The acute collections are referred to as either APFCs (acute peripancreatic fluid collections) or as ANCs (acute necrotic collections), depending on the absence or presence, respectively, of necrosis. All types of pancreatic fluid collections can be sterile or infected. Other complications include extrahepatic biliary dilatation; splenic, portal, and mesenteric venous thrombosis; varices; arterial pseudoaneurysm; pleural effusion; and ascites.[6]

Computed tomography for the assessment of local complications is most useful 48–72 hours after the onset of symptoms rather than at the time of admission. Unless contraindicated (e.g., renal dysfunction), intravenous contrast should be given in order to assess for pancreatic necrosis once patients are adequately fluid replenished and normovolemia is restored.

Magnetic resonance cholangiopancreatography (MRCP) is recommended only in patients in whom there is elevation of liver enzymes and in whom the CBD is either not visualized adequately or is found to be normal on ultrasound.[7]

Although the overall mortality of acute pancreatitis is static at 1 to 2%, in severe acute pancreatitis mortality is 10 to 30%. Early diagnosis of acute pancreatitis is therefore important to start immediate and proper treatment.

Among the various imaging modalities, CT offers higher sensitivity and accuracy in diagnosing acute pancreatitis.[8] CECT is considered as the gold standard in diagnosing acute pancreatitis as it is also sensitive in evaluating pancreatic necrosis and extrapancreatic complications.[9] So, CECT is necessary in acute pancreatitis patients for correct diagnosis, its complications and follow up.

MATERIAL AND METHODS

This study was aimed to study CT findings of acute pancreatitis and its complications irrespective of enzymes levels. Study was carried in the Department of Radio-diagnosis, Indira Gandhi Medical College and Hospital, Shimla. The research procedure was in accordance with the approved ethical standards of Indira Gandhi Medical College and Hospital, Shimla, Ethics Committee.
Study Design: Hospital based cross sectional study.
Study duration: July 2018 to June 2019.
Sample Size: 37 patients.

Inclusion Criteria

- Age ≥ 18 years.
- Admitted patients in various clinical departments having clinical, and/or biochemical (or both) suspicion of acute pancreatitis after 72 hours of onset of symptoms.
- Willingness to participate in prospective study.

Exclusion Criteria

- Patients who had allergy to Iodine CT-contrast agents.
- Impaired creatinine clearance.
- Pregnant patients.
- Patients with signs of chronic pancreatitis.
- Absence of peripheral venous access.
- Who were not willing to participate in the study.

Steps of Study

1. Serum lipase level less than 180 units/L was referred as Lipase negative patient and lipase level more than 180 units/L was referred as Lipase positive patient.
2. CECT abdomen was performed after 72 hours in all patients.
3. CECT findings were interpreted.
4. Patients were followed up for complications of acute pancreatitis. Complications were classified according to Revised Atlanta classification.[6]
5. Statistical analysis and conclusions were derived on completion of the study.

Procedure methodology

CT acquisition and image reconstruction

CECT was performed in all patients of acute pancreatitis after 72 hours after onset of symptoms using a MDCT 64 slice MODEL VCT XTE GE Medical System CT scanner. All patients were given omnipaque (Iohexol) 1.5 ml/kg body weight with concentration of 350 miligram iodine/mililitre intravenously (i.v.) at a rate of 3 ml/second via the antecubital vein. No enteric (oral or rectal) contrast material was given. Breath-hold imaging in the portal venous phase starting 60 seconds after i.v. contrast agent injection was done to allow for proper discrimination between edematous and necrotic pancreatitis. The scan region covered the area from the diaphragm to the pubic symphysis.

CECT Diagnosis of acute pancreatitis

Acute pancreatitis was diagnosed on the basis of presence of at least one of the following findings.

- Edematous pancreatic swelling indicated by the increase in size of pancreas. (Normally maximum anteroposterior diameter of head, body, tail of pancreas measures 2.7cm, 2.5cm, 2.8cm respectively).

- Peripancreatic fat stranding and/or fluid collection.
- Parenchymal and/or peripancreatic necrosis.

Necrosis was indicated by absent pancreatic parenchymal contrast enhancement and/or heterogeneous peripancreatic fluid collection containing both liquefied and solid necrotic material defined as a CT attenuation value more than 20 HU (Hounsfield unit).

Depending upon the presence or absence of necrosis Acute pancreatitis was classified into two types.
1. Acute interstitial edematous pancreatitis (AIEP)-no necrotic component present.
2. Acute necrotizing pancreatitis (ANP)-pancreatic or peripancreatic necrosis or both present.
Pancreatic and peri-pancreatic fluid collections were classified according to Revised Atlanta classification.[6]

STATISTICAL ANALYSIS
We had analyzed data using EPI-INFO 7.2.2.6 version for windows. Patients were divided into AIEP and ANP groups. Data was entered in Microsoft excel spreadsheet. It was presented in form of percentages, means, median, standard deviation and interquartile range. The means of two groups were compared using independent t' test for normally distributed data and Mann Whitney test for skewed data. Categorical data was analyzed using Chi square test or Fisher exact test where appropriate. P value of < 0.05 was taken as statistically significant.

OBSERVATIONS AND RESULTS
In this study, total 37 patients were included who presented with clinical suspicion of acute pancreatitis to the various Departments of IGMC, Shimla. These patients were evaluated clinically, biochemical analysis was done and then CECT was done in all cases after 72 hours. CECT findings were evaluated in all the patients. Patients were followed up for complications at two months interval.

The patients were divided into Acute Interstitial Edematous Pancreatitis (AIEP) and Acute Necrotizing Pancreatitis (ANP) groups on the basis of CT findings. 29.7%(11 out 37) of the study population (Lip−) had less than three fold rise in serum lipase levels at presentation despite abdominal pain and signs of Acute pancreatitis on CECT.

Twenty Eight out of 37 (75.7%) patients had AIEP and 9 (24.3%) had ANP. This has been summarized in Table-1.

Table 1.

AIEP	28(75.7%)
ANP	9(24.3%)

GENDER DISTRIBUTION
In our study of 37 patients, there was male predominance, as twenty three (62.2%) were male patients and 14(37.8%) were females. This is summarized in Table -2.

Table 2.

Gender		
	N	**%**
Male	23	62.2
Female	14	37.8
Total	37	100.0

AGE DISTRIBUTION
The age group varied from 19-90 years in this study. In age wise distribution, the highest number of patients were in age group 30-60 years.

ETIOLOGY
Fourteen out of 37 patients (37.9%) had alcohol as the etiological factor and in 56.7 % patients (21 out of 37), gall stones was identified as the etiological factor.

In 2.7% patients (1 out of 37) the cause of pancreatitis could not be ascertained. One patient (2.7%) etiology was post ERCP. It has been summarized in Table 3.

Table 3.

Etiology	**N**	**%**
Alcohol	14	37.9
Gall Stones	21	56.7
Post ERCP or idiopathic	2	5.4
Total	37	100.0

CECT FINDINGS
In our study, 75.7% of patients had AIEP and 24.3% had ANP. CECT findings has been summarized in Table-4.

Table 4.

	Total number of patients
	n = 37 (%)
CECT findings	
Morphology n (%)	
Acute interstitial pancreatitis	28 (75.7)
Acute necrotizing pancreatitis	9(24.3)
Pancreatic findings n(%)	
Edematous organ swelling	34 (91.9)
Peripancreatic fat stranding	34(91.9)
Acute peripancreatic fluid collection	18 (48.5)

Parenchymal or extra pancreatic necrosis	8 (21.6)
Extrapancreatic findings n (%)	
Pleural effusion	22 (59.5)
Ascites	19 (51.3)
Mesenteric inflammation	29 (78.4)

Complications On Follow Up

On follow up after 2 month interval, 5 out of 28(17.8%) patients in AIEP group had complication in the form of pseudocyst formation.

On other hand, complications were present in 2 out of 9(22.2%) patients in ANP group. One patient (11.1%) died in this group. One patient (2.7%) had emphysematous pancreatitis. One patient had (2.7%) had pseudo aneurysm formation. This has been summarized in Table -5.

Table 5: Cross tabulation between Follow up complications in two groups.

Follow up	AIEP		ANP				P value
	N	%	N	%	N	%	
Eventful	5	17.8	2	22.2	7	18.9	0.399 NS
Uneventful	23	82.2	7	77.8	30	81.1	
Total	28	100.0	9	100.0	37	100.0	

Statistical Analysis: Fisher's exact test. P<0.05: Statistically significant.

DISCUSSION

The present study was undertaken to evaluate the role of CECT in acute pancreatitis and its complications. Acute pancreatitis (AP) is caused most commonly by various biliary tract diseases or heavy alcohol intake. In our study, 56.7% of patients had gall stones and 37.8% patients had alcohol as the etiological factor. The diagnosis is most commonly based upon characteristic history of acute abdomen accompanied with rise in levels of pancreatic enzymes namely amylase and lipase in serum.

In our study, majority of the patients had mild form of pancreatitis on the basis of Modified CT severity index.[10] Few had moderate form of acute pancreatitis (10%). Majority of these patients i.e. 75.7% (28 out of 37) had acute interstitial edematous pancreatitis (AIEP). Whereas 24.3% (9 out of 37) had acute necrotizing pancreatitis (ANP).

In our study, another finding was the presence of less than three fold increase in serum lipase levels in 11 out of 37(29.7%){with p value <0.05} of the study population (Lip−) despite abdominal pain and signs of AP on CECT. In previously published data by Lankish et al, 18% of patients with first attack of acute pancreatitis had less than three fold increase of serum lipase.[11] The diagnosis of AP would be missed in these patients by serum lipase levels alone without confirming by CECT.

Different extrapancreatic inflammatory changes, pleural effusions, ascites, and mesenteric inflammation can be assessed accurately by CECT. Pleural effusion was present in 59.5% of patients, ascites in 51.3% and mesenteric inflammation in 78.4% of patients. These findings are of importance as patients with abdominal pain but without threefold increase of serum lipase and without possible accompanying clinical manifestations of AP like pleural effusions and ascites are prone to be under diagnosed without being confirmed by CECT.

In our study, 62.2%(28 out of 37) were male. The etiological factor was alcohol in 37.9% of patients and gall stone in 56.7% of patients. These observations were in line to those in the study by Imrie CW and Whyte AS where the biliary disease was found in (51 %) of cases, alcoholism in (26%) of cases and idiopathic in (13%) of cases as etiological factors.[12] Another study by Blarney SL et al have shown gall stone as etiological factor in 44% of cases, while alcohol accounted for 33% of cases and rest 24% being idiopathic.[13]

Both study groups were followed up for complications. On follow up after 2 month interval, 5 out of 28(17.8%) patients in AIEP group had complication in the form of pseudocyst formation. Complications were present in 2 out of 9(22.2%) patients in ANP group. One patient (11.1%) died in this group. This patient had vascular complications in form of thrombosis of portal vein and splenic vein.

Another finding in our study was that 2 out 37 (5.4%) patients presented with first episode of pain abdomen. However on imaging, pseudocyst and walled off necrosis was present in these patients. These imaging findings are suggestive of complications of acute pancreatitis that develop after 4 weeks of an attack of acute pancreatitis. Serum lipase levels were raised in these patients. These features could imply that first episode of pancreatitis whether interstitial edematous or necrotizing type might have been of milder form causing none or negligible clinical complaints.

One of the patient (2.7%) of acute pancreatitis in our study had pseudo aneurysm of gastro-duodenal artery which was comparable to studies by Fagi G et al[14] and Sharma PK et al.[15] Major vascular complications of

pancreatitis occur with a frequency of 1.2-14%, with a greater incidence seen in chronic pancreatitis (7-10%) than acute pancreatitis (1-6%).[14-15] The overall mortality rate due to hemorrhage in acute pancreatitis has been reported to reach ranges as high as 34-52%, and is significantly higher than in cases of patients without bleeding.[15] However, deaths in pancreatitis patients often occur due to multi-organ failure. Direct vascular injuries are the most feared complications as they can cause rapid blood loss and clinical deterioration.

Emphysematous pancreatitis is a rare variant of severe acute pancreatitis characterized by gas formation within and around the pancreas. Though the outcome of EP is reported to be poor, there are very few reports on clinical characteristic of EP.2.7% of patients (1 out of 37) had emphysematous pancreatitis. Air foci were present within pancreatic parenchyma and not around the pancreas. There are only scattered case reports in the literature about emphysematous pancreatitis, most of them dealing with radiological features. In a study by Mendez et al[16], 2% of patients had intrapancreatic air. In a recent report of 14 necrosectomies over 4 years, three patients had emphysematous pancreatitis.[17]

STUDY LIMITATIONS

Our study had its limitations. It was a small study conducted on 37 patients and thus the results need to be interpreted in a larger study for validation. Another limitation of our study was short duration of follow up so complications beyond this duration might have been missed.

CONCLUSION

Contrast enhanced computed tomography (CECT) is standard imaging modality for the evaluation of acute pancreatitis and its complications. Using enzyme assay of serum amylase and lipase, clinicians can establish the diagnosis but cannot evaluate for pancreatic necrosis and its vascular complications. Moreover significant subset of patients with acute pancreatitis can have less than three fold rise in serum lipase and amylase level.

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