



ACUTE PANCREATITIS: PROGNOSTIC VALUE OF COMPUTED TOMOGRAPHY SEVERITY INDEX, A PROSPECTIVE STUDY

Prof.(Dr.)Biant Singh	Professor And Head, Department Of Surgery VCSG Govt. MS&RI Srinagar-Garhwal Uttarakhand 246174
Dr. Gurpreet Kour	Senior Registrar, Department Of Pedodontics And Preventive Dentistry SDD HDC, Panchkula, Haryana
Dr. Manpreet Kour	Resident, VCSG Govt. MS&RI Srinagar-Garhwal Uttarakhand 246174
Dr. Navajyoti Bora*	Associate Professor, Department of Obstetrics and Gynaecology, VCSGMS&RI, Srinagar, Uttarakhand *Corresponding Author

ABSTRACT

BACKGROUND : Acute pancreatitis is an inflammatory process of pancreas with variable course involving other regional tissues and remote organ systems. It is therefore essential to recognise the severity of disease and plan focussed management

METHODS: A prospective study on 50 patients of acute pancreatitis admitted in our teaching hospital in Garhwal from June 2017 to June 2019 was done to recognise the severity, to predict the outcome and to plan a focussed early management with the help of Contrast enhanced computed tomography in identifying and staging the disease, making justified use of limited hospital resources, thus aimed at reducing the morbidity and mortality rate.

RESULTS: We found that with increase in severity of disease, mortality rate was highest in severe grade as against nil in mild grade cases. Conservative measures, critical care management in severe cases and standard operative procedure when required was necrosectomy with closed peritoneal lavage in severe cases with 50% operative mortality.

CONCLUSION: We conclude that CECT can prognosticate patients of acute pancreatitis and predict the morbidity, hospital stay and mortality rate. It can recognise the patients who need early intervention to prevent progression of disease.

KEYWORDS : Acute Pancreatitis, Contrast Enhanced Computed Tomography, Ct Severity Index, Organ Failure.

INTRODUCTION:

Acute pancreatitis is most complex and clinically challenging of all acute abdominal disorders. "It is inflammatory process of pancreas with variable course involving other regional tissues and remote organ systems."^[1] The condition shows a wide spectrum of disease severity ranging from mild self limiting having mortality of less than 2% to severe having high incidence of complication and running a protracted course in gravely ill patients leading to multi organ failure ranging from 20 to 50%.

It is essential to recognise the severity of disease and plan a focussed management. World wide gall stones are the most common cause of occurrence of this condition, in 45% cases.^[2] Alcohol being second most common factor accounting for 35% cases and other rare causes include idiopathic, various drugs, trauma (accidental or iatrogenic), ERCP, metabolic abnormalities (hypertriglyceridemia, hypercalcemia), obstruction (pancreatic divisum and ductal anomalies), infections (bacterial, viral and parasitic infestations), vascular anomalies (emboli and vasculitis) and hereditary mutations of trypsinogen 1 gene. Premature activation of trypsinogen to trypsin in acinar cells is a key event leading to autodigestion of pancreas.^[3] What is triggering is still unresolved. In severe cases extensive interstitial necrosis, necrotising vasculitis leading to devitalisation of pancreatic tissue occurs. Inflammatory process may extend to retroperitoneal fatty tissue. Toxic biologically inflammatory mediators like IL1, TNF, oxygen free radicals get liberated into blood stream and ascitic fluid. Post inflammatory cascade leads to distal organ failure and sometimes severe fatal necrotising pancreatitis cases are missed till autopsy.

Our knowledge of pathogenesis is still fragmentary and scoring systems available to identify and predict the severity of disease are too cumbersome and unsatisfactory. Rapid severity assessment is a challenge and obvious need for simple system to predict severity and plan focussed management is looked for. Clinical assessment alone fails to identify two- third of patients who eventually develop compli-

cations or die but supportive lab investigations and imaging systems have proved useful.^[4]

Serum amylase, serum lipase and serum and urinary trypsinogen levels measurements have proved useful but have limitations. Other markers like serum elastase, phospholipase A2, pancreas specific protein (procarboxypeptidase) and pancreatic isolipase have nothing advantageous to offer.^[5] Imaging by USG is basic investigation but non visualisation of pancreas, in acute pancreatitis, by obscuring bowel gas is an issue in 30-40% cases.

CECT of abdomen and pelvis being in use for last more than 30 years has greatly improved and changed the management of acute pancreatitis by quantifying parenchymal injury being 87% accurate and 100% sensitive for detecting pancreatic necrosis.^[6] Besides being useful for therapeutic, follow-up modalities and evaluating complications. It helps in prompt focused management of high risk patients by offering therapeutic window to change patient outcome where in beneficial results with close monitoring, early management in ICU, offering therapeutic ERCP (selected cases), prophylactic antibiotics and surgery is offered.^[7]

CECT evaluation of acute pancreatitis patients also avoids unnecessary use of invasive and risky procedures in mild to moderate cases facilitating optimal use of limited health care resources in government run hospitals like ours located in peripheral hilly rural terrain of Uttarakhand state. Improvement in unenhanced CT, grading was made in 1990 when CTSI was introduced based on contrast enhancement in acute pancreatitis. An excellent correlation was documented between pancreatic necrosis duration of hospitalisation, development of complications and death.

MATERIAL AND METHODS:

Present study was conducted in the department of general surgery VCSG govt MS & RI teaching hospital in Srinagar, Garhwal to assess the prognostic correlation and clinical outcome of acute pancreatitis on the basis of CTSI. Prosp

ective study included 50 consecutive cases of acute pancreatitis admitted in the hospital from June 17 to June 19. Clinical history was taken and salient features with reference to abdominal pain, site, radiation and duration was done. Associated symptoms like nausea, vomiting, loss of appetite, jaundice and fever were noted. Personal history with special reference to alcohol use and drug intake besides family history of hyperlipidemia was taken into account

EXCLUSION CRITERIA:

- 1- Patients with allergy to contrast
- 2- Pregnant female patients with acute pancreatitis
- 3- Patients of pancreatic malignancy
- 4- Patients with chronic pancreatitis, pancreatic calcifications, intraductal strictures and calculi
- 5- Any previous pancreatitis surgery and post operative cases.

Clinical examination with special reference to general condition, vital signs, hemodynamic stability, CVS, respiratory and urinary systems were made. Abdominal signs of tenderness, guarding, free fluid in peritoneal cavity and bowel sound status were recorded. Routine lab investigations like HB, TLC, Coagulogram, serum lipase, serum amylase, serum calcium, serum phosphorus and hematocrit were done in all cases within 48 hours. Xray chest, ECG, USG abdomen and pelvis was done routinely in all cases to rule out conditions mimicking acute pancreatitis. Patients were managed on conservative protocol nil orally, IV fluids, antispasmodics, PPIs and antibiotics routinely and put under close monitoring. Ryle tube suctioning was done in patients with vomiting. All vitals were monitored and managed as per protocol. CECT was done after 72 hours of admission in all the 50 cases on 40 slice CT scanner (Phillips Brilliance). [Figure 1-7] Non-ionic iodinated contrast material (Iopamide-ultravist 370) 70-100 ml at a dose of 1.5 ml/kg was administered intravenously by using an injector at the rate of 3ml/sec, followed by saline chase of 20 ml normal saline at the rate of 2.5ml/sec. Post contrast scanning was done in porto-venous phase (70s), and the scans were obtained in the cranio-caudal direction from the domes of diaphragm to the level of pubic symphysis in the supine position. Scan parameters used were as follows: 120 kVp, 200 Ma/Slice. Axial CT sections were taken at a collimation of 40* 0.625 and a pitch of 0.9, and were reconstructed at 3mm thickness, increment of --1.5 mm. Images were analysed and reported as per scoring system vis a vis CT SEVERITY INDEX. [Table 1]

Table 1: Scoring system according to the CT SEVERITY INDEX for every patient.

ELEMENT	FINDINGS	POINTS
GRADE OF ACUTE PANCREATITIS	Normal pancreas	0
	Pancreatic enlargement	1
	Inflammation involving pancreas and peri-pancreatic	2
	single fluid collection or phlegmon	3
	two or more fluid collections or phlegmon	4
DEGREE OF NECROSIS	No necrosis	0
	necrosis of 1/3 rd of pancreas	2
	necrosis of 1/2 of pancreas	4
	necrosis of more than 1/2 of pancreas	6

Computed tomography severity index was calculated as points for grading of Acute Pancreatitis (Balthazar score) + points of degree of pancreatic necrosis
 Group A – MILD (0-3 points)

Group B- MODERATE(4-6 points)
 Group C- SEVERE (7-10 points)

Patients were observed for any complications. CT severity index was used to predict the morbidity, duration of hospital stay and mortality of patients under study. Any intervention if needed in the form of laprotomy, pancreatic necrosectomy and closed peritoneal lavage etc was performed. The results were tabulated and subjected to appropriate statistical analysis which was done using graph-pad in stat version 3.10. P value of <0.05 was taken as significant.

OBSERVATIONS AND RESULTS:

Acute pancreatitis being a common ailment forms sizable proportion of emergency surgical admissions. Early recognition of cases for most suitable treatment to reduce morbidity & mortality, while making justified use of hospital resources is need of the hour. In our study CTSI scoring is used to determine the desired goals. In our prospective study 50 consecutive cases of acute pancreatitis were admitted. Patients were taken irrespective of age. There were 29 males and 21 females, average age for females was 47.71 yrs and for males was 54.48 yrs. [Table 2] Almost all of our patients were from rural background. Most common etiological factor was gall stones (50%). Second most common cause was alcohol (30%) based on personal history. [Table 3]

Table 2: Age and sex, Male : female = 1.38:1

AGE (years)	No.	Mean age	SD	SEM	T test	P value*
Females	21	47.71	12.346	2.694	1.044	0.8880
Males	29	54.48	12.777	2.373	1.044	0.8880

SD- standard deviation, SEM- standard error of mean, *unpaired t test

Table 3: Etiology:

ETIOLOGY	NO. OF PATIENTS
Gallstones	25
Alcohol	14
Idiopathic	9
hyperlipidemia	2

Common symptom observed at presentation was pain upper abdomen followed by nausea and vomiting. All the patients had epigastric tenderness 100%, 70% had guarding, 24% had distention, & 14% had shifting dullness. USG findings in our study showed inability to visualise pancreas in 32% cases because of overlying bowel gas shadows. 56% had diffused pancreatic edema and 12% had focal pancreatic edema. 22% of our patients who were diagnosed to have pancreatitis by other methods had normal serum amylase on admission. CECT findings showed diffuse enlargement of pancreas in 80% cases. 88% cases had peri-pancreatic fluid collections, 6 cases (12%) had pleural effusion, 10% cases had thickened root of mesentery, 6% cases had pancreatic ascitis, 4% (2 cases) had emphysematous pancreatitis. Pancreatic necrosis was seen in 31 cases. 19 cases (38%) had < 30% necrosis, 9 cases (18%) had 50% necrosis & 3 cases (6%) had more than 50% pancreatic necrosis. [Table 4] Morbidity was highest in group C patients (91.67%), 6.25% in group A (mild) and 36.37% in group B as per CTSI scoring. [Table 5]

Table 4: CECT findings:

CECT Findings	No. of cases
Pancreatic enlargement	40
Peripancreatic fat stranding	30
Peripancreatic fluid collection	30
	14

Pancreatic necrosis	
nil	19
< 30%	19
30-50%	9
>50%	3

Emphysematous pancreatitis	2
Pancreatic ascitis	3
Thickened root of mesentery	5
Pleural effusion	6

Table 5: Morbidity pattern

Complications	No. of patients	Patients with complications	percentage	P value	MORBIDITY PATTERN					
					Pleural effusion	Acute fluid collection	Acute renal failure	Pancreatic ascitis	ARDS	sepsis
MILD (A)	16	1	6.25%	A vs B 0.0525	1 (6.25%)	0	0	0	0	0
MODERATE(B)	22	8	36.37%	B vs C 0.0031	3 (13.64%)	3 (13.64%)	1 (4.55%)	1 (4.55%)	0	0
SEVERE(C)	12	11	91.67%	A vs C <0.0001	2 (16.66%)	2 (16.66%)	3 (25%)	2 (16.66%)	1 (8.33%)	1 (8.33%)

P value is significant on comparing group A & C.

Most common complication noted was pleural effusion and maximum complications were noted in group C patients. Mortality was found highest in group C patients(16.67%) and no mortality was seen in group A patients.[Table6] Mean duration of hospital stay in mild (group A) was 9.25 days, moderate (group B) was 12 days and in severe cases (group C) was 24.58 days.[Table 7] Surgical intervention was required in 4 cases of group C(25% of group) with 2 post-operative deaths(50% of operated cases). No surgical intervention was needed in group A and group B cases.[Table8]

Table 6:Mortality

Mortality	No. of patients	Patients expired	percentage	P value
Mild (A)	16	0	0%	A vs B 1.000
Moderate (B)	22	1	4.5%	B vs C 0.2794
Severe (C)	12	2	16.67%	A vs C 0.1746

Table 7: Hospital stay

Mortality	No. of patients	MEAN	SD	SEM	'P' value
Mild (A)	16	9.250	3.000	0.7500	A vs B <0.05
Moderate (B)	22	12.000	1.877	0.4002	B vs C <0.001
Severe (C)	12	24.583	4.441	1.282	A vs C 0.001

P' value was significant statistically on comparing group A & C.

Table 8:Surgical intervention

Severity grade	patients	Operative intervention	Post operative deaths
Mild(A)	16	0	0
Moderate(B)	22	0	0
Severe (C)	12 (25%)	4(25%)	2(50%)

"p" value – 0.024 , relative risk-1.50 ,remarks- significant.

DISCUSSION:

In our study we evaluated the role of Contrast enhanced severity index as a predictor of outcome of acute pancreatitis. In our study We found males predominating . with males to females ratio, 1.38:1. This falls in concordance with other studies done by Balthazar EJ, W Uhl , Minguez M , K Choi, AC de Beaux^[8-12] Average age of males was 47.71 and females 57.48 years. Balthazar EJ in his study found mean age of 52 years and Antonio in his study has a range of 18-93 years and Median age of 61.5 years^[13,14] The study of W Uhl and A C de Beaux had a median age of patients similar to ours^[8,12]

Majority of our patients were from rural areas and few were travellers (yatris). Since the Garhwal terrain is spread in hilly rural areas and there is no city catering to our hospital. The commonest etiological factor in our study was gallstones (50%) and second common was alcohol(28%). W Uhl et al, in their study had a biliary tract pathology in range of 36-38%.^[9] Marshall JB found biliary pathology and alcohol abuse as a cause of acute pancreatitis 60-80%.^[15] Minguez found biliary tract pathologies causative 52%.^[10] Alcoholism which forms a major etiology in Western world has also found a place in our study as the male folk of this hilly terrain is a significant consumer of alcohol. Presenting symptom in our study was Abdominal pain(100%), Nausea and vomiting(76%), Abdominal Distension(20%), fever(12%), constipation(6%) , breathlessness (2%). Webster PD and Shah SSH et al reported similar results in their study^[16,17] In 32% of patients with acute pancreatitis pancreas could not be visualized during sonographic assessment due to overlying bowel gas at first instance. Similar findings were observed Silverstein et al and Gamaste^[18,19]

In 22% patients of acute pancreatitis ,diagnosed by computed tomography, Serum amylase levels at admission were normal. Clavein et al in his study found normal amylase values in 19% patients of Acute Pancreatitis on admission.^[3] With regards to morbidity , patients with CTSI of 0-3 (MILD) had complications of 6.25% whereas CTSI 4-6 (MODERATE) had complications 36.37% and CTSI 7-10 (SEVERE) had complications 91.67%. Our study is comparable to the studies made by Balthazar EJ, Vriens PW and Chisty IA .^[8,20,21] Various complications in our patients belonging to severe group in order of frequency were azotemia(3/12) , pleural effusion (2/12), pancreatic ascites(2/12),acute fluid collection(2/12), ARDS(2/12) and sepsis (2/12). Overall , The most common complication was pleural effusion(12%).

Our study had results comparable to that of Beger et al , Viedma et al ,Lankisch et al , Toh et al who noted respiratory failure as the most common type of organ failure in acute pancreatitis^[22-25] Wongnai A et al has pleural effusion as the most common extrapancreatic complication in their study. Our study had results comparable to the most of mentioned. In our study we found mortality of 0% in patients of Mild CTSI 0-3, 4.5% in patients with Moderate CTSI 4-6 and 16.67% in patients with Severe (CTSI 7-10) Thus revealing increasing trend of mortality with increase in CTSI. Balthazar EJ and Simchuk et al have mentioned the same trend . Bradley E showed that CTSI >8 is an index of death.^[26,14,27,28]

The mean duration of hospital stay of patients in Group A (CTSI 0-3) was 9.25 +-3 days , Group B (CTSI 4-6)12 DAYS +- 1.87 days and Group C (CTSI 7-10) 24.58 DAYS +- 4.44 DAYS. Balthazar EJ , Chisty IA and Wongnai A have also reported a

prolonged hospital stay in severe group similar to that of our study.^[14,21,26]

Operative intervention in the form of Laprotomy with Pancreatic Necrosectomy and closed lavage was required in 4(25%) of our patients belonging to CTSI 7-10. The need for surgical intervention was due to findings suggestive of emphysematous pancreatitis (infected necrosis) on CECT in 2 patients. Rest of the two patients indication was clinical deterioration and presence of pancreatic necrosis on CECT. There were two patients who expired, one due to uncontrolled sepsis and other due to ARDS. Shah SSH et al noted that 16% of their patients with severe pancreatitis underwent laprotomy, washout and drainage.^[17] Sivasankar S in his study noted 34.6% of patients with severe pancreatitis required surgical intervention of whom 27.8% patients died.^[29] Our observations were similar to their study.

CONCLUSION:

From this study, it can be concluded that contrast enhanced computed tomography severity index can clearly prognosticate patients of acute pancreatitis and can predict morbidity, duration of hospital stay and mortality rate in patients of acute pancreatitis, thus predicting which patients may require intensive critical care and surgical intervention to prevent progression of disease. It can help in avoiding unnecessary use of costly management in mild cases, thus making optimal use of limited costly health care facilities. Since improved outcome in the severe form of Acute pancreatitis is based on early identification of disease severity and subsequent focused management of these high-risk patients, offering early therapeutic window, we advocate the use of contrast enhanced computed tomography in all cases of acute pancreatitis.

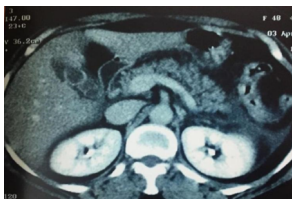


Figure 1: Bulky pancreas with marked peri-pancreatic fat stranding and thickening of latero-coanal fascia (CTSI=2)

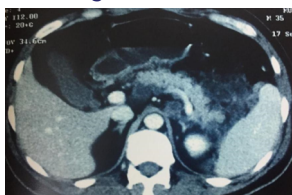


Figure 2: Inflamed pancreas with irregular outline and necrosis of the head of pancreas (CTSI=3)

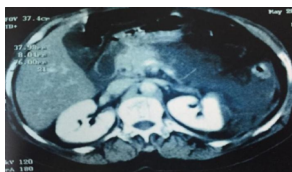


Figure 3: Marked necrosis of the body and tail of the pancreas with thickening of latero-coanal fascia (CTSI=8)



Figure 4: Necrosis of the head, body and tail of the pancreas

with peripancreatic fluid collection. (CTSI=10)

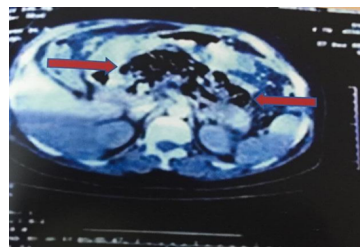


Figure 5: Picture revealing acute emphysematous pancreatitis (depicted by gas in and around the pancreatic parenchyma). A rare entity.

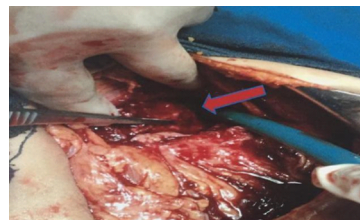


Figure 6: Necrotic pancreatic tissue in situ after exposing the lesser sac through gastrocolic ligament.



Figure 7: Inflamed head of the pancreas left behind after debridement (marked by arrow head)

AUTHOR'S CONTRIBUTION:

PROF. (DR.) BIANI SINGH:

Conceptualized the study, principal investigator, prepared the study protocol, participated in data collection, entry, preparation and editing of all drafts.

DR. GURPREET KOUR:

Manuscript preparation, review of literature and preparation of the drafts.

DR. MANPREET KOUR:

Coinvestigator, manuscript preparation, participated in data collection, entry, preparation and editing of all drafts.

DR. NAVAJYOTI BORA :

Coinvestigator, manuscript preparation, participated in data collection, preparation and editing of all drafts.

FUNDING: No funding sources

CONFLICT OF INTEREST: None declared

ETHICAL APPROVAL:

This study was approved by the Institutional Ethics Committee

REFERENCES:

1. Brivet FG, Emlie D, Galanaud P Pro and anti-inflammatory cytokines during acute pancreatitis: An early and sustained response, although unpredictable of death. Persian group on acute pancreatitis. Crit care med.1999;27(4): 749-55
2. Symers WSC. Acute alcoholic Pancreatitis. Dublin J Med Science.1917;14(3):244-47
3. Clavien PA, Burgan S, Moosa AR. Serum enzymes and other laboratory tests in acute pancreatitis. Br J Surg. 1989;76(12):1234-43.
4. Wilson C, Health DJ, Imrie CW. Prediction of outcome in acute pancreatitis: a comparative study of APACHE II, clinical assessment and multiple factor scoring system. Br J Surg.1990;77(11):1260-4

5. Steinburg W, Tenner S. Acute Pancreatitis. *N Eng J Med.*1994;330(17):1198-1210
6. Balthazar AJ. Acute Pancreatitis. Assessment of severity with clinical and CT evaluation. *Radiology*2002;223:603-613.
7. Nordback IH, Sand J, Saaristo R, Pacjanen H. Early treatment with antibiotics reduces the need for surgery in acute necrotising pancreatitis- a single centre randomised study. *J Gastrointest Surg.* 2001;5:113-120
8. Balthazar AJ, Robinson DL, Megibow AJ, Ranson JH. Acute pancreatitis. Value of CT in establishing prognosis. *Radiology* 1990;174:331-336
9. W Uhl.A randomised double blind, multicentric trial of octreotide in moderate to severe acute pancreatitis. *Gut.*1999;45:97-104
10. Minguez. Acute pancreatitis. A prospective epidemiological study in province of alicanta. *Rev ESP Enferm Dig.* 1995;87(12) 869-873.
11. Tkchoi. Somatostatin in treatment of acute pancreatitis. *Gut* 1989;30:223-227.
12. AC de Beaux. Factors influencing morbidity and mortality in acute pancreatitis. *Gut* 1995; 37:121-126.
13. Antorio Carnvole. Mortality in acute Pancreatitis. Is it an early or late event? *JOP* 2005;6(5):438-444
14. Balthazar AJ, Ranson JHC, Nardick DMegibow AJ, Caccaiale R, Cooper MM. Acute pancreatitis. Prognostic value of CT. *Radiology* 1985;156:767-772
15. Marshall JB. Acute Pancreatitis. A review with emphasis on new development. *Arch int Medicine* 1993;153:1185-1198
16. Webster PD. Pathophysiology and management of acute pancreatitis. *Hospital Practie* 1974; 56-66
17. Shah SSH, Ansari MA, Ali S. Early prediction of severity and outcome of acute severe pancreatitis. *Pak J Med Sciences* 2009;25:619-23
18. Silverstein W. Diagnostic imaging of acute pancreatitis. Prospective study using CT and sonography. *AJR* 1981;137(3)497-502.
19. Gamaste VV. Diagnosti tests for acute pancreatitis . *Gastroenterologist.* 1994;2:119-30
20. Vreins PW, Linde P, Warmendon PE. Computed tomography severity index is an early prognostic tool for acute pancreatitis. *Journal of American college of surgeons.* 2004;201:497-502
21. Chisly IA, Vaqar B, Sajida P, Dawar B, Zishan H. Role of CT in acute pancreatitis and its complications among age groups. *Journal of Pakistan Med Association* 2005;55:431-35
22. Beger HG, Rai B, Meyer J. National course of acute pancreatitis. *World J of Surgery.* 1997;21:130-135
23. Viedma J, Perez-Meteo Agullo J, Dominguez J, Carballo F. Inflammatory response in early prediction of severity in human acute pancreatitis. *Gut.* 1994;35:822-27
24. Lankisch P, Pflichthofer D, Lehnick D. Acute pancreatitis: which patients most at risk? *Pancreas.*1999;19:321-24
25. Toh S, Phillips S, Johnson C.A prospective audit against management of acute pancreatitis in the south of England. *Gut* 2000;46:239-243
26. Wongani A, Mai WNC. Computed tomography finding of acute pancreatitis in Maharaj Nakom Chiang Mai Hospital. *Chiang Mai Med Journal* 2007;46:45-91
27. Simchuk EJ, Traveso LW, Nukui Y, Kozarek RA. CTSI is a prediction of outcomes for severe pancreatitis. *Ann J Surg.* 2000;189:352-5
28. Bradley EL, Murphy F, Ferguson C. Prediction of pancreatic necrosis by dynamic pancreatography. *Ann Surg* 1989;210:495-504
29. Sivsankar A, Kanan DG, Ravichandran, Teswanths, Balachander TG, Surendran R. Outcome of severe acute pancreatitis. Is there a role for conservative management of infected pancreatic necrosis? *Hepatobiliary and pancreatic disease international* 2006;5:599-604.