Original Research Article

Clinical profile of Acute Pancreatitis in Malwa region of Punjab and its correlation with Balthazar CT Severity Index

Nitin Nagpal[1*], Salvinder Singh Toor[2], D. S. Sidhu[3]

[1]Associate Professor, [2]Junior Resident, [3]Professor and Head
Department of Surgery, GGS Medical College, Faridkot, Punjab, India
[*]Corresponding author email: **drnitinnagpal@gmail.com**

International Archives of Integrated Medicine, Vol. 3, Issue 5, May, 2016.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 11-04-2016 **Accepted on:** 19-04-2016
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: Nitin Nagpal, Salvinder Singh Toor, D. S. Sidhu. Clinical profile of Acute Pancreatitis in Malwa region of Punjab and its correlation with Balthazar CT Severity Index. IAIM, 2016; 3(5): 1-7.

Abstract

Background: Acute pancreatitis is a common condition with wide clinical variation, ranging from mild self-limiting pancreatic inflammation to extensive pancreatic necrosis with life-threatening consequences. The present study aimed to assess the clinical profile of acute pancreatitis in Malwa region of Punjab where there increased prevalence of alcoholism and gall stone disease and to assess the efficacy of Ranson's score and Balthazar Computed tomography severity index (CTSI) in predicting the prognosis.

Materials and methods: 50 patients with proven acute pancreatitis were included and data was collected to study their clinical, laboratory and radiologic profile to obtain prognostic indices Ranson's score and CTSI which were then compared with outcome.

Results: Mean age recorded was 43.40 ±12.004 years with a range of 19-64 years and male to female ratio 2.12:1. 62% of patients had alcohol induced pancreatitis and 32% had gall stone pancreatitis. Observed morbidity rate was 44% and mortality rate was 6%. Most common complications encountered were pleural effusion (18%), Hypocalcemia (20%) and sterile pancreatic necrosis (20%). 18 patients had Ranson's score more than 3, whereas 11 patients had CTSI more than 7 indicating severe acute pancreatitis. On correlation Ranson's score was found to be more sensitive while CTSI was more specific for an adverse outcome.

Conclusion: Severe acute pancreatitis remains a significant cause of morbidity and mortality due to increased prevalence both alcoholism and gall stone disease in Malwa region of Punjab. In our setup Ranson's score and CTSI when used in combination showed improved sensitivity for detection severe acute pancreatitis.

Key words

Acute Pancreatitis, CT severity index, Ranson's score.

Introduction

Acute Pancreatitis is a routine cause of patients reporting to emergency department with acute abdomen. It is a reversible inflammatory process of the pancreas that is associated with little or no fibrosis. Although etiology of acute pancreatitis is a complex subject but two factors, biliary tract stone disease and alcoholism, account for majority (80-90%) of the cases. Parenchymal pancreatic injury is the pathological hallmark of this form of the disease, but the triggering physiological factors that initiate and sustain this process are not fully understood. The hallmark symptom of acute pancreatitis is the acute onset of persistent upper abdominal pain, usually with nausea and vomiting. The usual locations of the pain are the epigastric and periumbilical regions and it may radiate to the back, chest, flanks, and lower abdomen. Patient is usually restless and bend forward (knee chest position) in an effort to relieve the pain because the supine position tends to exacerbate the intensity of symptoms [1]. Clinical parameters such as tachycardia, orthostatic hypotension, shock, respiratory distress, and signs of peritonitis are consistent with a severe attack [2]. Old age, hyperlipidemia, and obesity are associated with an increased risk of death [3]. Flank ecchymosis (Grey-Turner sign) or periumbilical ecchymosis (Cullen sign) are more specific and have been associated with a 37% mortality rate [4]. Cornerstone of the diagnosis of Acute Pancreatitis is the clinical findings and an elevation of pancreatic enzymes. Most of the cases of acute pancreatitis have a mild clinical course and resolves spontaneously with supportive care but approximate mortality of 10 to 30% has been reported in patients with severe acute pancreatitis [5]. It has been estimated that experienced clinicians can correctly predict a severe attack of pancreatitis in only 34%–39% of patients at the time of admission [6]. According to a study the diagnosis of AP was missed in large number of patients with fatal necrotizing pancreatitis which was retrospectively diagnosed at the time of autopsy [7]. So, it is important to quantify the disease as per severity. Various scoring systems, having parameters based on clinical, biochemical and radiologic findings, individually or in combination are available. Ranson's score represents a major advancement in evaluation of disease severity and has been used clinically for more than four decades. Contrast Enhanced Computed Tomography (CECT) is now considered as gold standard radiological investigation for diagnosis of acute pancreatitis [8]. The morphologic severity of acute pancreatitis can be determined using a CT severity index (CTSI) that was developed by Balthazar and co-workers.and has been useful in predicting severity early in the course of disease [9]. The present study was conducted on patients of acute pancreatitis attending the Department of Surgery, GGSMCH, Faridkot to asses there clinical profile and to evaluate role of Balthazar CTSI in assessing prognosis in these patients.

Materials and methods

After getting approval from institutional ethical and research committee, this study was conducted prospectively from March 2014 to October 2015 to include 50 patients who were admitted with diagnosis of Acute pancreatitis on the basis of more than 3 fold elevated serum amylase levels, presenting within 48 hours of initiation of symptoms. Patients with pregnancy, immunocompromised patients or associated with other infectious conditions like inflammatory bowel disease, pneumonia, cholangitis, appendicitis were not included in the study. Detailed history and clinical findings were recorded in proforma after taking informed written consent. Serial laboratory investigations were recorded and Ranson's score was calculated for each patient as per indices in **Table - 1**. Patients with Ranson's score of 3 were labelled as Mild AP whereas those with Ranson's score more than 3 were labelled as Severe AP. After

initial resuscitation and stabilization all patients underwent CECT scan within 48 hours of admission. Based on findings on radiology, Balthazar CT severity index was calculated for each patient as illustrated in **Table – 2**.

Table - 1: Ranson's score.

For Non-Gallstone pancreatitis	For Gallstone pancreatitis
At Admission	
1. Age in years > 55 years 2. White blood cell count > 16000 cells/mm^3 3. Blood glucose > 11 mmol/L (> 200 mg/dL) 4. Serum AST > 250 IU/L 5. Serum LDH > 350 IU/L	1. Age in years > 70 years 2. White blood cell count > 18000 cells/mm^3 3. Blood glucose > 12.2 mmol/L (> 220 mg/dL) 4. Serum AST > 250 IU/L 5. Serum LDH > 400 IU/L
Within 48 hours:	
1. Serum calcium < 2.0 mmol/L (< 8.0 mg/dL) 2. Hematocrit fall > 10% 3. Oxygen (hypoxemia PaO_2 < 60 mmHg) 4. BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration 5. Base deficit (negative base excess) > 4 mEq/L 6. Sequestration of fluids > 6 L	1. Serum calcium < 2.0 mmol/L (< 8.0 mg/dL) 2. Hematocrit fall > 10% 3. Oxygen (hypoxemia PaO_2 < 60 mmHg) 4. BUN increased by 0.7 or more mmol/L (2 or more mg/dL) after IV fluid hydration 5. Base deficit (negative base excess) > 5 mEq/L 6. Sequestration of fluids > 4 L

Table – 2: Balthazar CT severity index.

Criteria	Finding on CECT	Points
Pancreatic Inflammation	Normal pancreas	0
	Focal or diffuse pancreatic enlargement	1
	Intrinsic pancreatic alterations with peri pancreatic fat stranding	2
	Single fluid collection/ phlegmon	3
	Two or more fluid collection or gas, in or adjacent to pancreas	4
Pancreatic Necrosis	None	0
	≤30%	2
	30%-50%	4
	≥50%	6

Further course of stay in the hospital and finding of associated morbidity (complications) and mortality were recorded. Using IBM SPSS 20 software descriptive statistics were reported as frequencies and percentage, or as mean and standard deviation. Pearson correlation and linear regression model was used to examine the relation between Ranson's scoring, CTSI and outcome in terms of morbidity and mortality.

Results

50 patients with Acute Pancreatitis were studied in a prospective manner to obtain following observations. Majority of patients, 22 patients (44%) were in the age group of 31-45 years with mean age 43.40 ±12.004 years and range of 19-64 years. Of the 50 patients, 34 (68%) were male, and 16 (32%) were female with male to

female ratio 2.12:1. Most patients (58%) had BMI between 18.5-24.9 with a mean BMI of 25.38 ±0.54. In the present study, majority of patients, 31 patients (62%) had alcohol induced pancreatitis or had gall stone pancreatitis (32%). Other minor aetiologies were post ERCP (6%), post surgical (gastrectomy) complications (2%), post traumatic (4%) and no definite etiology was found in 2 patients. Etiology wise distribution was as per **Table – 3**.

Table – 3: Etiology for Acute Pancreatitis.

Etiology	No. of cases	%
Alcohol	31	62
Gall Stone Disease	16	32
Post ERCP	3	6
Others	5	10

In the present study, following an attack of AP complications were observed in 22 (44%) patients and most common complications encountered were pleural effusion (18%), Hypocalcemia (20%) and sterile pancreatic necrosis (20%) while others include ARDS, infected pancreatic necrosis, pancreatic abscess, Ascites, MODS, splenic vein thrombosis. Mortality was encountered in 3 cases i.e. 6%. Complications associated were as per **Table – 4**.

Table – 4: Morbidity and Mortality associated with AP.

Complication	No. of cases	%
Pleural Effusion	9	18
Hypocalcemia	10	20
Sterile Pancreatic Necrosis	10	20
Infective Pancreatic Necrosis	4	8
ARDS	6	12
MODS	2	4
Ascites	2	4
Pancreatic Abscess	3	6
Splenic Vein Thrombosis	1	2
Death	3	6

Out of 50 patients, 18 patients (36%) had Ranson's score more than 3 and were classified as severe AP based on it. Whereas 11 patients (22%) had CTSI more than 7 indicating severe AP. Distribution of both scores was as per **Table - 5**.

Table – 5: Distribution of Ranson's score and Balthzar CTSI.

Scoring system	Score	No of patients (%)
Ranson's	<3 (Mild AP)	32 (64)
	≥3 (Severe AP)	18 (36)
CTSI	0-3 (Mild)	34 (68)
	4-6 (Moderate)	5 (10)
	7 or > 7 (Severe)	11 (22)

In the present study, it was observed that more number of complications was associated with high RANSON's score. Statistically significant association (p value <0.05) was found between high RANSON's score and complications like pleural effusion (0.034), hypocalcemia (0.01), sterile pancreatic necrosis (0.000), ARDS (0.000) and infected pancreatic necrosis (0.05). Also it was found that more number of complications were associated with very high CTSI score (≥7). Statistically significant association (p value <0.05) was found between high CTSI score and complications like pleural effusion (0.014), sterile pancreatic necrosis (0.000), ARDS (0.000) and infective pancreatic necrosis (0.000). On comparison of adverse level of the two indices, it was found that 16.66% of patients with high RANSON's score had died while 25% patients with high CTSI had died (**Table – 6**). So we concluded on CTSI being a more specific predictor of prognosis and Ranson's score is more sensitive in prediction of adverse outcome.

Discussion

Historically, acute pancreatitis was reported to be the cause of death of Alexander the great (323 BC) [10]. Reginald Fitz famous pathologist from Massachusetts, in his paper presented details of clinical presentation, distinguishing between

hemorrhagic, suppurative and gangrenous forms of disease. Fitz believed that pancreatitis originates by the extension of gastroduodenal inflammation [10]. It was Hans Chiari who in 1896, postulated that pancreas succumbs to its own digestive properties meaning that the mechanism of the disease was pancreatic autodigestion. Opie EL proposed that a gall stone lodged in the ampulla might occlude both the common bile duct and the pancreatic duct that would allow reflux of bile into the pancreatic duct with the activation of pancreatic enzymes and pancreatitis [10]. Surgical treatment mostly involved pancreatectomy or necrosectomy was historically associated with a mortality rate of more than 80%. In 1970s Beger and colleagues' described the characteristics of pancreatic necrosis (infected vs sterile) and based on this surgical intervention (open vs closed drainage) could be planned [11]. This led to considerable improvement in outcome. Incidence of acute pancreatitis has been reported to be 40 per lakh population [12]. No population based data on prevalence of acute pancreatitis is available from India, however some idea of incidence can be obtained from patients admitted in tertiary care centres, a report from AIIMS found approximate incidence to be 55 patients per year [13].

Table – 6: Morbidity and Mortality distribution based on Ranson's score and Balthazar CTSI.

	Score	**Complications(no. of patients)**						
		Pleural Effusion	**Hypo-Calcemia**	**Sterile Pancreatic Necrosis**	**Infected Pancreatic Necrosis**	**ARDS**	**Death**	**Others**
Ranson Score	<3	3	2	0	0	0	0	0
	>3	6	8	10	4	6	3	5
CTSI	0-3	3	4	0	0	0	0	0
	4-6	3	1	3	0	1	0	0
	≥7	3	5	7	4	5	3	5

World over gallstones have largely been implicated as a common cause of acute pancreatitis, in contrast present study found alcoholism as the main etiological factor. It has been said that relative rate of gallstone disease versus alcoholism as a cause critically depends upon the age, sex and geographical distribution of the patients [14]. As in our study, majority of the patients are middle aged men, so alcohol is attributed as the most common etiological factor accounting for 62% of the cases. Also higher prevalence of alcohol intake in Malwa region of Punjab [15] may be attributed to, even if there is high prevalence gall stone disease also. Acute pancreatitis in women is more frequently related to gallstone disease.

In 1992, Atlanta, GA, International Symposium on Acute Pancreatitis has classified this entity into mild acute pancreatitis and severe acute pancreatitis [12]. While this is not a perfect classification system, since intermediate forms of disease do occur, but it has provided a more reliable basis for experimental studies and for clinical management of acute pancreatitis. The rationale of previous classifications [16, 17] has been based on the extent and degree of pancreatic injury, which could only be assumed at the time of diagnosis and which could sometimes be confirmed later during surgical exploration or post-mortem examination. In 2012, the Atlanta classification was revised with an emphasis on persistent organ failure. The new classification is based on the presence of multi-organ failure (clinical and laboratory parameters) and on the morphology of the pancreatic gland as depicted by contrast enhanced CT scan. Severe pancreatitis is defined by the presence of any evidence of organ failure or presence of systemic or a local complication. Organ failure was

defined as shock (systolic blood pressure < 90 mmHg), pulmonary insufficiency (Pao_2 < 60 mm hg), Renal failure (S. creatinine level > 2 mg/dl after fluid resuscitation), Gastrointestinal bleeding (>500 ml/24 hr). Systemic Complications like Disseminated intravascular coagulation (platelet count ≤ 100,000, Fibrinogen < 1 gr/L, Fibrin split products > 80 µg/dL) and metabolic disturbance (calcium level ≤ 7.5 mg/dL). Local Complications like Necrosis, Abscess, Pseudocyst [18].

Acute pancreatitis is a complex disease that can vary from a mild self-limiting presentation, in approximately 80-90% of patients, to a clinically severe form in 10-20% with multiple complications and high mortality rate [12, 14]. Mild acute pancreatitis usually has a very low mortality rate (less than 1%) [6, 17] whereas the death rate for severe acute pancreatitis can be 10 to 30% depending on the presence of sterile versus infective necrosis [6]. The present study observed a morbidity rate of 44% and mortality rate of 6%. Mortality in acute pancreatitis has bimodal distribution: an early phase (first 2 weeks of onset of symptoms) in which the multi organ dysfunction syndrome (most common cause of death) is the final result of an intense inflammatory cascade triggered initially by pancreatic inflammation and late phase (after 2 weeks) which is due to septic complications [5]. The most crucial point is the development of pancreatic necrosis, which often progresses to a systemic inflammatory response syndrome (SIRS). Thus, multi organ dysfunction syndrome, the extent of pancreatic necrosis, infection, and sepsis are the major determinants of mortality in acute pancreatitis [18].

Individual preference and available institutional facilities influence the method chosen for prognostic assessment of acute pancreatitis [19]. A milestone achievement in assessing the severity of acute pancreatitis occurred in 1974, when Ranson developed his prognostic scoring system. He examined relationship of 43 different measurements made during the first 48 hours of treatment, finding 11 variables that significantly correlated with overall morbidity and mortality. Originally Ranson's criteria were created for alcohol-induced pancreatitis and were revised in 1979 for gallstone-induced acute pancreatitis. Original Ranson's score was used by default as alcohol-induced acute pancreatitis was the most prevalent etiology. When gallstones were found, revised Ranson's score was used. The cut-off value accepted in the literature is 3. Patients with Ranson's score more than 3 is considered to have severe acute pancreatitis [7, 12]. As for patients presenting to our setup often belong to low socioeconomic strata and have financial constrains, score like Ranson's is heavily depended upon for predicting outcome and is also relevant in developing countries. In our study, 22 cases developed complications out of which 18 patients (i.e. 81%) had a score ≥3, implying that occurrence of complications is more in patients with high Ranson's score showing that it is highly sensitive in prediction of a severe disease. CT severity index (CTSI) was developed by Balthazar and co-workers and then simplified and extended to monitor organ failure by Silverman, Banks, and colleagues in 2004 [9]. The CT severity index is an attempt to improve the early prognostic value of CT in cases of acute pancreatitis [18, 19]. In the present study there was a statistically significant correlation between a continuous increasing incidence of morbidity and mortality and patients stratified according to CT severity index groups.

Conclusion

Severe form of acute pancreatitis remains a debilitating illness associated with significant morbidity and mortality. Often, the surgeon/physician faces a challenge to differentiate between mild acute pancreatitis and severe acute pancreatitis at the time of admission. Thus it is critical to have clinical, laboratory and radiologic indicators of severe disease so that aggressive management can be administered. Routine use of CT scan can also help in predicting the need of therapeutic intervention. We recommend routine use of CTSI score with Ranson's score to improve sensitivity

and specificity of detection of severe acute pancreatitis.

References

1. Silen W. Acute pancreatitis. In: Silen W, Cope Z. Cope's Early Diagnosis of the Acute Abdomen. 18th edition, New York, N.Y.: Oxford University Press, 1999, p. 123-31.
2. Agarwal N, Pitchumoni CS. Assessment of severity in acute pancreatitis. Am J Gastroenterol., 1991; 86: 1385–1391.
3. Porter KA, Banks PA. Obesity as a predictor of severity in acute pancreatitis. Int J Pancreatol., 1991; 10: 247–252.
4. Dickson AP, Imrie CW. The incidence and prognosis of body wall ecchymosis in acute pancreatitis. Surg Gynaecol Obstet., 1984; 159: 343–347.
5. Senn H. The surgery of pancreas. Philadelphia, WJ Dormon; 1886, p. 71.
6. Wilson C, Heath DI, Imrie CW. Prediction of outcome in acute pancreatitis: a comparative study of APACHE II, clinical assessment and multiple factor scoring systems. Br J Surg., 1990; 77: 1260–1264.
7. Corfield AP, Cooper MJ, Williamson RCN, et al. Prediction of severity in acute pancreatitis: prospective comparison of three prognostic indices. Lancet, 1985; 2: 403–407.
8. Matos C, Cappeliez O, Winant C, Coppens E, Deviere J, Metens T. MR imaging of the pancreas: a pictorial tour. Radiographics, 2002; 22: e2.
9. Balthazar, Emil J. Acute Pancreatitis: Assessment of Severity With Clinical And CT Evaluation. Radiology, 2002; 223(3): 603-613.
10. D A O'Reilly, A N Kingsnorth. A Brief history of pancreatitis. J R Soc Med., 2001; 94: 130-132.
11. Beger HG, Buchler M, Bittner R, Nevalainen T, et al. Necrosectomy and post operative local lavage in necrotising pancreatitis. Br j Surg., 1988; 75: 207-12.
12. Toouli J, Brooke-Smith M, bassi C, et al. Guidelines for the management of acute pancreatitis. J Gastroenterol Hepatol., 2002; 17 Suppl: S15-39.
13. Tandon RK. Management of Acute Pancreatitis: Indian Guidelines and Protocols. J Gastroenterol Hepatol., 2013; 16: 267-70.
14. Cappell MS. Acute pancreatitis: etiology, clinical presentation, diagnosis, and therapy. Med Clin North Am., 2008; 92(4): 889-923.
15. V Khosla, KR Thankappan, GK Mini, PS Sarma. Prevalence & predictors of alcohol use among college students in Ludhiana, Punjab, India. Indian J Med Res., 2008; 128(1): 79-81.
16. Kloppel G. Pathology of severe acute pancreatitis. In: Bradley EL III, ed. Acute pancreatitis: diagnosis and therapy. NewYork, NY: Raven, 1994; p. 35–46.
17. Beger HG, Rau B, Mayer J, Pralle U. Natural course of acute pancreatitis. World J Surg., 1997; 21: 130–135.
18. Peter A Banks, Thomas L Bollen, Christos Dervenis, Hein G Gooszen, et al. Classification of acute pancreatitis - 2012: revision of the Atlanta classification and definitions by international consensus. Gut, 2013; 62: 102–11
19. Khanna AK, Meher S, Prakash S, Tiwary SK, Singh U, Srivastava A, Dixit VK. 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 Surg., 2013; 2013: 3675-81.