



E-ISSN: 2616-3470
P-ISSN: 2616-3462
© Surgery Science
www.surgeryscience.com
2020; 4(1): 13-16
Received: 10-11-2019
Accepted: 12-12-2019

Dr. Subhash Chander Grover
Assistant Professor,
Department of General Surgery,
Dr. Radhakrishnan Government
Medical College, Hamirpur,
Himachal Pradesh, India

Dr. Gambheer Singh
Professor, Department of General
Surgery, Raipur Institute of
Medical Sciences, Raipur,
Chhattisgarh, India

An observational study to determine relation between various indices of acute pancreatitis

Dr. Subhash Chander Grover and Dr. Gambheer Singh

DOI: <https://doi.org/10.33545/surgery.2020.v4.i1.a.303>

Abstract

Introduction: Acute pancreatitis is a relatively common disease with wide clinical variation and its incidence is increasing. The severity of acute pancreatitis can be predicted based upon various severity grading systems. Some of these can be performed on admission to assist in triage of patients while others can only be obtained during the first 48 to 72 hours or later. The objective of the study was to determine the relation between various indices of acute pancreatitis.

Methods: This observational study involved Prior Consent from the patients & was found to be within ethical standards. A total of 100 patients were selected which were proven cases of Acute Pancreatitis during a period of 2.5 years from year 2016 to 2019. Subjects included both the genders, all age groups including pediatric and geriatric age group and all classes of socio economic strata.

Results: Majority of the patients were in the age group of 21-40. There was a clear gender predilection towards males with M:F of 4:1. In all cases, Pancreatic enzymes showed more than threefold higher than the upper limit of normal. 72% of the cases were related to alcoholism. 88% cases were having mild ranson's score. As per CT Severity index maximum cases were mild in severity.

Conclusion: There was a significant male preponderance. Most common cause was alcohol abuse in males and gall stone disease in females. There was a good correlation between Balthazar CT severity index and Ranson's score. Magnitude of enzyme elevation had no relation to the severity of the disease. Irrespective of the cause enzyme elevations were similar quantitatively.

Keywords: Acute pancreatitis, ranson's score, CT severity index, Balthazar grade

Introduction

Acute pancreatitis is a relatively common disease with wide clinical variation and its incidence is increasing. The average mortality rate in severe acute pancreatitis approaches 2–10 %^[1]. It is an acute inflammatory process of the pancreas with varying involvement of other regional tissues or remote organ systems^[2].

A commonly used classification system (the Atlanta classification) divided acute pancreatitis into two broad categories: Mild (edematous and interstitial) acute pancreatitis and Severe (usually synonymous with necrotizing) acute pancreatitis^[3].

About 80% of the attacks are mild, 20% are severe and they are commonly accompanied by necrosis of the pancreas and or organ failure^[4]. Etiology of Acute Pancreatitis are alcoholism, gall stone, ischemia, drug induced, hyperparathyroidism, hypercalcaemia, trauma, ERCP, mechanical Obstruction, pancreas divisum, autoimmune, hereditary, infectious, malnutrition, scorpion bite, hyperlipoproteinemia, pregnancy. Gall stones and chronic ethanol abuse account for 70% of cases of acute pancreatitis^[5].

The severity of acute pancreatitis can be predicted based upon clinical, laboratory, and radiologic risk factors, various severity grading systems, and serum markers. Some of these can be performed on admission to assist in triage of patients while others can only be obtained during the first 48 to 72 hours or later^[6].

Pathophysiologic mechanisms include micro-circulatory injury, leucocyte chemo-attraction, release of pro and anti-inflammatory cytokines, oxidative stress, leakage of pancreatic fluid into the region of pancreas, bacterial translocation to the pancreas and systemic circulation^[7]. The initial step in the pathogenesis of acute pancreatitis is conversion of trypsinogen to trypsin within acinar cells in sufficient quantities to overwhelm normal mechanisms to remove active trypsin^[8].

Corresponding Author:
Dr. Gambheer Singh
Professor, Department of General
Surgery, Raipur Institute of
Medical Sciences, Raipur,
Chhattisgarh, India

Clinical Features of Acute Pancreatitis are abdominal pain, nausea, vomiting, hiccoughs and abdominal distention [3]. Abdominal pain develops quickly, reaching maximum intensity within minute and persist for hours or even days. Constant in nature, experience first in epigastrium, radiating to back in 50% of cases. It is refractory to the usual dose of analgesics and constant in nature and intensity. Nausea and vomiting are usually marked accompaniments. Vomiting is often frequent and persistence. Hiccoughs can be troublesome and may due to gastric distention or irritation of diaphragm. Abdominal distention as a result of ileus, bowel sounds are usually diminished during an attack of pancreatitis and the abdomen may become distended and tympanic [3, 6].

Obese patients with pancreatitis have a higher incidence of local complications, severe acute pancreatitis [9, 10].

About one fourth of the deaths occur within 24 hrs of admission [11]. After the second week of illness patients succumb to pancreatic infection associated with multi-organ failure [12].

Many patients referred to as acute pancreatitis have precipitates commonly referred to as biliary sludge [13]. Biliary sludge accounted for approximately 67% of acute idiopathic pancreatitis [14]. Tissue specimens obtained during cholecystectomy from patients with acute pancreatitis contain precipitates of various descriptions in approximately 60% of the cases [15, 16, 17]. Abnormalities hepatic bile secretion and changes in the contractile function and mucosal properties of gall bladder contribute to formation of gall bladder sludge [18, 19].

CT, as used to aid the diagnosis and staging of acute pancreatitis, has greatly improved and has changed the clinical management of this condition [20, 21]. CT performed during the initial 12 hours may show only equivocal findings, with a slight heterogeneous decrease in attenuation of the pancreas (ischemia) but a normal parenchymal texture [22]. The CT severity index is an attempt to improve the early prognostic value of CT in cases of acute pancreatitis [23]. The utility of the Ranson criteria compared with that of the Balthazar CT criteria for detection of severe pancreatitis, CECT results were found to be better prognostic indicators, owing to greater sensitivity and specificity [24]. Extravasation of activated pancreatic enzymes induces the development of retroperitoneal fat necrosis [25, 26]. There has been much interest in early surgical and endoscopic removal of gallstones retained in the common bile duct [27]. There is good evidence that early endoscopic intervention is the procedure of choice in patients with stone impaction and cholangitis [27].

The objective of the study was to determine the relation between various severity indices of acute pancreatitis like Ranson's score, CT severity index and pancreatic enzymes.

Methodology

This observational study involved Prior Consent from the patients & was found to be within ethical standards. A total of 100 patients were selected which were proven cases of Acute Pancreatitis during a period of 2.5 years from year 2016 to 2019. Subjects included both the genders, all age groups including pediatric and geriatric age group and all classes of socio economic strata.

Detailed Clinical Examination were done & all the patients went through routine investigations. The selection criterions for the patient were based on (1) complain of abdominal pain; (2) on examination- abdominal tenderness; (3) Ultrasonography finding; and (4) serum amylase more than threefold rise than normal. Ultrasonography is a very safe investigation which can be repeated. CT scan used in cases when there is clinical deterioration. The selected patients had been treated thereafter in

form of different modalities like, (a) simple analgesic; (b) fluid resuscitation; and (c) surgical treatment as and when required.

Patients with chronic pancreatitis and with acute exacerbations of the same were excluded from the study. Age, sex, complete blood picture, liver function tests, serum calcium, serum amylase and lipase, contrast enhanced computed tomography of the abdomen were performed for the patients. Patients were treated with conservative management. Serum enzymes were repeated when and where required. Computed Tomography (CT) grading system of Balthazar and CT Severity index scoring system (CTSI)

Balthazar Grade: Grade A: Normal pancreas consistent with mild Pancreatitis. Grade B: Focal or diffuse enlargement of the gland, including contour irregularities and inhomogeneous attenuation Grade C: Abnormalities seen in grade B plus peripancreatic Inflammation. Grade D: Grade C plus single fluid collection. Grade E: Grade C plus two or more peripancreatic fluid collections or gas in the Pancreas. CTSI = Balthazar grade score plus necrosis score. Balthazar grade score A = 0 B = 1 C = 2 D = 3 E = 4 Necrosis score: Absence of necrosis = 0 Necrosis of up to 1/3% of pancreas = 2 Necrosis of 1/3 to 50% = 4 Necrosis of >50% = 6. Patients admitted in the various centres and other surgical units of local tertiary care hospitals as diagnosed cases of Acute Pancreatitis were included in this study. The operating team / Surgeons were the same in all cases. Data was filled in Microsoft Excel & analysed using a computer software Epi Info version 6.2 (Atlanta, Georgia, USA) & SPSS. P value of 0.05 and less was considered as statistically significant.

Results

Age and sex distribution

With the study conducted on 100 patients, 20 patients were female and 80 were males. Majority of the patients were in the age group of (21-40) constituting 52 % with a mean of 38.42 yrs. There was a clear sex predilection towards males with M:F of 4:1

Table 1: showing distribution of patients of Acute Pancreatitis with Etiology

Etiology	Number of Patients / Percentage
Alcoholism	72
Biliary Sludge	20
Idiopathic	08

Among the pancreatic enzymes levels, both serum amylase and lipase were analysed in all the 100 patients and all of them showed more than threefold higher than the upper limit of normal. In the assessment of Ranson's score of the 100 patients included in the study, 88 were mild and 12 were severe as per Ranson's score.

CECT abdomen was done in all the 150 patients. The most common finding was diffuse swelling of the pancreas and peri-pancreatic fat stranding. Maximum cases were mild in CTSI.

Table 2: showing distribution of cases as per CT Severity index

CTSI Score	0-3	4-6	7-10
No of cases	90	6	4
Percentage	90%	06%	04%
Grading	Mild	Moderate	Severe

Discussion

100 patients were included in the study. Majority of the patients were in the age group of 21-40 constituting 52% of the total.

Minimum age of the study group was 11 and eldest was 78 yrs. There was no significant difference between the mean age of patients between the alcoholism and biliary stone disease groups. This was not consistent with the previous study (Korean study KIM YS *et al.*, St. John's Bangalore), which said patients of alcoholism group were relatively younger to the gall stone disease. In the present study alcohol abuse was commonest cause in males and gall stone disease was common cause in females. It was consistent with the Indian textbook Tandon and opposing western textbook (Sleissenger) which quoted that alcohol abuse is common cause in both males and females. Patients were designated to have idiopathic cause after performing serum triglycerides, serum calcium, serum electrolytes and anti-nuclear anti bodies. Present study did not show any difference between the elevations in the these etiologies. This was not consistent with the previous studies which showed that the enzymes were relatively more elevated in gall stone disease as compared to ethanol abuse. In an Indian study done at Bangalore St. John's medical college mean amylase and mean lipase levels were significantly lower in ethanol abuse than the biliary group. Magnitude of enzyme elevation did not bear any relation with the severity of the acute pancreatitis. The natural history of the disease varied from mild to severe necrotizing pancreatitis. All the patients were treated conservatively. Of the biliary cases sent for ERCP, 2 patients came for follow up, of them 1 patient had undergone laparoscopic cholecystectomy.

There was no gold standard treatment for acute pancreatitis; all the cases were managed conservatively. Henceforth, prediction of severity has not yet been important for the treatment of acute pancreatitis. However, this will be an important task for assessment of prognostication and treatment complications. Amongst the various clinical scoring systems, the most feasible one Ranson's score was calculated and was compared with Balthazar's CT severity index. Majority of the cases were mild with a score of less than or equal to 3. CECT was done in all the cases and as per them only 4 cases were severe with CT severity index score of 8. These cases were also severe as per Ranson's score. According to KIM YS *et al.* [28] the estimation of severity through the Ranson's criteria is not precise and is not an appropriate method because it needs 48 hours to complete and has a low specificity (77%) and sensitivity (75%) [28]. They did not find any correlation between Ranson's score and radiological grade in their study. In the present study there was a fair correlation between Ranson's and Balthazar's score. According to another study the utility of the Ranson analytic criteria compared with that of the Balthazar CT criteria for detection of severe pancreatitis, CECT results were found to be better prognostic indicators, owing to greater sensitivity and specificity [28].

All the cases were subjected to ultrasonography. The most common finding was diffusely swollen pancreas with increase in size. Higher score on CT severity score was associated with a more prolonged stay. No systemic complications were encountered in the patients. Local complications were encountered, of which Pseudo cyst of the pancreas the most common (in 16% of the patients) was followed by splenic vein thrombosis, pleural effusion, and necrosis.

Conclusion

The following were the notable findings. There was a significant male preponderance. Most common cause was alcohol abuse in males and gall stone disease in females. There was a good correlation between Balthazar CT severity index and Ranson's

score. Magnitude of enzyme elevation had no relation to the severity of the disease. Irrespective of the cause enzyme elevations were similar quantitatively, but the average lipase value was higher than the average amylase value in all the etiologies. Sensitivity of the ultrasound is about 61% while the specificity was still low. Most common complication was pseudo cyst of the pancreas. In the present study we had the objective of analysing the various severity indices.

The initial management for an acute pancreatitis attack should be conservative; with surgery reserved for cases having uncertainty of diagnosis, trauma, very severe attacks not responding to medical therapy and complications of the disease. The severity of acute pancreatitis is variable. The ability to predict the severity can help identify patients at increased risk for morbidity and mortality, thereby assisting in appropriate triage and selection of patients for specific interventions. This topic review will summarize methods for predicting the severity of acute pancreatitis. This study of Acute Pancreatitis is an effort to recount and record each battle which the surgeon fought to treat the illness.

Acknowledgements

We would like to thank all the participants of the study. Compliance with Ethical Standards.

Conflict of interest: None.

Funding: None.

Informed Consent Obtained.

References

1. Singh VK, Bollen TL, Wu BU *et al.* An assessment of the severity of interstitial pancreatitis. *Clin Gastroenterol Hepatol.* 2011; 9:1098.
2. Bradley EL 3rd: A clinically based classification system for acute pancreatitis, Atlanta, Ga, September 11 through 13, 1992. *Arch surg.* 1993; 128:586.
3. Russell RCG. The Pancreas. In: Bailey and Loves' Short Practice of Surgery. Russell RCG, Williams NS, Bulstrode CJK, editor. 24th ed. London: Hodder Arnold, 2004, pp. 1115-1132.
4. McKay CJ, Evans S, Sinclair M. High early mortality rate from acute pancreatitis in Scotland 1984-1995. *Br J Surg.* 1999; 86:1302.
5. Corfield AP, Cooper MI, Williamson RCN. Acute pancreatitis: A lethal disease of increasing significance. *Gut.* 1985; 26:724.
6. Nagle AP, Soper NJ, Hines JR. Management of Acute Pancreatitis. In: Zinner MJ, Ashley SW, editors. *Maingot's Abdominal Operations.* 11th Ed. New York: McGraw Hill, 2007, 939-82.
7. Lerch MM, Adler G. Experimental animal models of acute pancreatitis. *Int. J Pancreatol.* 1994; 15:159.
8. Grady T, Saluja A, Kaiser A. Pancreatic edema and intrapancreatic activation of trypsinogen during secretagogue-induced pancreatitis precedes glutathione depletion. *Am J Physiol.* 1996; 271:G20.
9. Porter KA, Banks PA. Obesity as a predictor of severity in acute pancreatitis. *Int. J Pancreatol.* 1991, 247.
10. Funnell IC, Bomman PC, Weakley SP. Obesity: An important prognostic factor in acute pancreatitis. *Br I Surg.* 1993; 80:484.
11. McKay CJ, Buter A. Natural history of organ failure in acute pancreatitis. *Pancreatology.* 2003; 3:111.

12. Gloor B, Muller GA, VV'orni M. Later mortality in patients with severe acute pancreatitis. *Br J Surg*. 2001; 88:975.
13. Bolondi L, Gaiani S, Testas, Labo G. Gallbladder sludge formation during prolonged fasting after gastro-intestinal surgery. *Gut*. 1985; 26:734-8.
14. Rose E, Navvaro S, Bruce C, Garcia-Pughes A, Valderman R. Occult microlithiasis in idiopathic pancreatitis: prevention of relapses by cholecystectomy or ursodeoxycholic acid therapy. *Gastroenterology*. 1991; 101:1701-9.
15. Freund H, Pfeffierman R, Durst AL, Rabinovici N. Gallstone pancreatitis: exploration of biliary tract in recurrent pancreatitis. *Arch surg*. 1976; 111:1106-7.
16. Houssin D, Castain GD, Lemoine J, Bismuth H. Microlithiasis of gall bladder. *Surg Gynecol Obstet*. 1983; 157:20-4.
17. Mayor AD, McMahon MJ. Gallstones and acute pancreatitis- is the association underestimated? *Br J Surg*. 1984; 71:905.
18. Lee SP. Pathogenesis of biliary sludge. *Hepatology*. 1990; 12:200s-5s. 20. Carey MC, Cahalane MJ, whither biliary sludge? *Gastroenterology*. 1988; 95:508-23.
19. Bradley EL III. A clinically based classification system for acute pancreatitis. *Arch Surg*. 1993; 128:586-90.
20. Dervenis C, Johnson CD, Bassi C. Diagnosis, objective assessment of severity, and management of acute pancreatitis. *Int. J Pancreatol*. 1999; 25:195-210.
21. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JHC. Acute pancreatitis: value of CT in establishing prognosis. *Radiology*. 1990; 174:331-6.
22. Balthazar EJ, Freeny PC, van Sonnenberg E. Imaging and intervention in acute pancreatitis. *Radiology*. 1994; 193:297-306.
23. Basterra G, Alvarez M, Marcaide A, DelgadoE, Diaz de Otazu R, Garcia Campos F. Acute pancreatitis: evaluation of the prognostic criteria of the latest Balthazar tomographic classification. *Rev Esp Enferm Dig*. 1999; 91:433-8.
24. Beger HG, Rau B, Mayer J, Pralle U. Naturalcourse of acute pancreatitis. *World J Surg*. 1997; 21:130-5.
25. Kloppel G. Pathology of severe acute pancreatitis. In: Bradley EL III, ed. *Acute pancreatitis: diagnosis and therapy*. New York, NY: Raven, 1994, 35-46.
26. Kelly TR, Wagner DS. Gallstone pancreatitis: a prospective randomizedtrial of the timing of surgery. *Surgery*. 1988; 104:600-5.
27. Leese T, Neoptolemos JP, Baker AR, Carr-Locke DL. Management of acute cholangitis and the impact of endoscopic sphincterotomy. *Br J Surg*. 1986; 73:988-92.
28. Kim YS. *World journal of gastroenterology*. 2008; 21:507-8.