A Retrospective Analysis between Various Indices of Acute Pancreatitis

Naveen Kumar Tirkey¹, Pradeep Beck^{2*}

Abstract

Introduction: Acute pancreatitis (AP) is the sudden inflammation of the pancreas and, to a varied degree, involves local tissues or distant organs. The severity of AP can be predicted based on 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. The objective of the study was to determine the relation between various indices of AP. **Methods:** A retrospective analysis of diagnosed cases of AP of total 200 patients was done. Subjects included both the genders, all age groups including pediatric and geriatric age groups and all classes of socioeconomic strata. **Results:** There was a significant male preponderance. Most common cause was alcohol abuse in males and gallstone disease in females. There was a good correlation between Balthazar computed tomography 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 was similar quantitatively, but the average lipase value was higher than the average amylase value in all the etiologies. Sensitivity of the ultrasound was good while the specificity was still low. Most common complication was pseudocyst of the pancreas. In the present study, we had the objective of analyzing the various severity indices. **Conclusion:** The initial management for an AP 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 AP 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.

Keyword: Acute pancreatitis, Ranson's score, Conservative management *Asian Pac. J. Health Sci.*, (2021); DOI: 10.21276/apjhs.2021.8.2.11

INTRODUCTION

Acute pancreatitis (AP) is the sudden inflammation of the pancreas and, to a varied degree, involves local tissues or distant organs.

AP is a relatively common disease with wide clinical variation and its incidence is increasing. The average mortality rate in severe AP 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 AP into two broad categories: Mild (edematous and interstitial) AP and severe (usually synonymous with necrotizing) AP.^[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 AP is alcoholism, gallstone, ischemia, drug induced, hyperparathyroidism, hypercalcemia, trauma, endoscopic retrograde cholangiopancreatography, mechanical obstruction, pancreas divisum, autoimmune, hereditary, infectious, malnutrition, scorpion bite, hyperlipoprotenemia, and pregnancy. Gallstones and chronic ethanol abuse account for 70% of cases of AP.^[5]

The severity of AP can be predicted based on 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 48–72 h or later.^[6]

Pathophysiologic mechanisms include microcirculatory injury, leukocyte chemoattraction, 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 AP is conversion of trypsinogen to trypsin within acinar cells in

¹Associate Professor, Department of General Medicine, Bharat Ratna Late Shree Atal Bihari Vajpayee Memorial Medical College, Rajnandgaon, Chhattisgarh, India

²Professor, Department of General Medicine, Bharat Ratna Late Shree Atal Bihari Vajpayee Memorial Medical College, Rajnandgaon, Chhattisgarh, India

Corresponding Author: Dr. Pradeep Beck, Department of General Medicine, Bharat Ratna Late Shree Atal Bihari Vajpayee Memorial Medical College, Rajnandgaon, Chhattisgarh, India. E-mail: dr_pradeepbeck@yahoo.co.in

How to cite this article: Tirkey NV, Beck P. A Retrospective Analysis between Various Indices of Acute Pancreatitis. Asian Pac. J. Health Sci., 2021;8(2):57-60.

Source of support: Nil

Conflicts of interest: None.

Received: 10-01-2021 Revised: 20-02-2021 Accepted: 25-03-2021

sufficient quantities to overwhelm normal mechanisms to remove active trypsin.^[8]

Clinical features of AP 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. Location of pain is epigastrium in most 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 is 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 AP. $^{\left[9,10\right]}$

^{©2021} The Author(s). This is an open access article distributed under the terms of the Creative Commons Attribution License (http:// creativecommons.org/ licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

About one-fourth of the deaths occur within 24 h of admission.^[11] After the 2nd week of illness, patients succumb to pancreatic infection associated with multiorgan failure.^[12]

Many patients referred to as AP 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 AP contain precipitates of various descriptions in approximately 60% of the cases.^[15-17] Abnormalities hepatic bile secretion and changes in the contractile function and mucosal properties of gallbladder contribute to the formation of gallbladder sludge.^[2,18]

CT, as used to aid the diagnosis and staging of AP, has greatly improved and has changed the clinical management of this condition.[19,20] CT performed during the initial 12 h may show only equivocal findings, with a slight heterogeneous decrease in attenuation of the pancreas (ischemia) but a normal parenchymal texture.^[21] The CT severity index is an attempt to improve the early prognostic value of CT in cases of AP.^[22] The utility of the Ranson criteria compared with that of the Balthazar CT criteria for the detection of severe pancreatitis, contrast-enhanced computed tomography (CECT) results were found to be better prognostic indicators, due to greater sensitivity and specificity.^[23] Extravasation of activated pancreatic enzymes induces the development of retroperitoneal fat necrosis.[24,25] There has been much interest in early surgical and endoscopic removal of gallstones retained in the common bile duct.^[26] There is good evidence that early endoscopic intervention is the procedure of choice in patients with stone impaction and cholangitis.^[26]

The objective of the study was to determine the relation between various severity indices of AP such as Ranson's score, computed tomography (CT) severity index, and pancreatic enzymes.

Methods

This retrospective analytical study involved prior consent from hospital authorities/medical superintendents of the local randomly selected secondary and tertiary care hospitals having surgical ward and intensive care unit (ICU), records to see the records of the patients from medical records department. The study was conducted within ethical standards. The patients who were admitted in randomly selected tertiary care hospitals including our teaching hospital in the city were selected for the study. Randomization was done using computer tables in selecting data. It was observed in the records that all patients underwent standard clinical examinations, routine biochemical and hematological investigations, ultrasonography of whole abdomen, and received treatment. Medical record numbers were used to generate the data for analysis.

For the purpose of the present study, data of 200 of the randomly selected patients (candidates/study subjects) who sought care between March 1, 2015, and March 1, 2020, were retrospectively identified. A total of 200 patients were selected which were proven cases of AP and conservatively managed during a period of 5 years. Study Subjects which were retrospectively identified included both adult Male and Female gender, having different socioeconomic status.

It was seen that detailed clinical examination was done and all the patients went through routine investigations. Mild cases are usually successfully treated with conservative measures: Hospitalization, pain control, nothing by mouth, intravenous nutritional support, and intravenous fluid rehydration. Severe cases often require admission to an ICU to monitor and manage complications of the disease. Complications are associated with a high mortality, even with optimal management.

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 3-fold 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 the form of different modalities such as (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, and CECT 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 Belthazar⁽²⁰] and CT Severity Index (CTSI)⁽²¹⁾ was used for analysis.

1. Details of Belthazar Grade are :

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.
- 2. Details of CTSI are

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 centers and other surgical units of local tertiary care hospitals as diagnosed cases of AP were included in this study.

Data were filled in Microsoft Excel and continuous data were expressed as mean \pm standard deviation. The data were analyzed by IBM SPSS Statistics 23. Overall, P < 0.05 was proposed to represent statistical significance after correction.

RESULTS

Age and Sex Distribution

With the study conducted on 200 patients, 53 patients were female and 147 were male. Majority of the patients were in the age group of 18–40 constituting 52% with a mean of 39.62 years. There was a clear sex predilection toward males.

Among the pancreatic enzymes levels, both serum amylase and lipase were analyzed in all the 200 patients and all of them showed more than 3-fold higher than the upper limit of normal.

In the assessment of Ranson's score of the 200 patients included in the study, 83% (166) were mild and 11% (22) were severe as per Ranson's score.

CECT abdomen was done in all the 200 patients. The most common finding was diffuse swelling of the pancreas and peripancreatic fat stranding. Maximum cases were mild in CTSI.

DISCUSSION

Two hundred patients were included in the study. Majority of the patients were in the age group of 18-40 constituting 66% of the total. Minimum age of the study group was 18 and eldest was 79 years. 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 et al.)^[27] which said that patients of alcoholism group were relatively younger to the gallstone disease. In the present study, alcohol abuse was the most common cause in males and gallstone disease was common cause in females [Table 1]. It was consistent with the Indian textbooks and opposing western textbooks in which it is usually guoted 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 antibodies. The present study did not show any difference between the elevations in these etiologies. This was not consistent with the previous studies which showed that the enzymes were relatively more elevated in gallstone disease as compared to ethanol abuse. In an Indian study done at Bangalore St. John's Medical College,^[1] 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 AP. The natural history of the disease varied from mild to severe necrotizing pancreatitis. All the patients were treated conservatively.

There was no gold standard treatment for AP; all the cases were managed conservatively. Henceforth, prediction of severity has not yet been important for the treatment of AP. However, this will be an important task for the assessment of prognostication and treatment complications. Among 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 ≤ 3 [Table 2]. CECT was done in all the cases and as per them only four cases were severe with CT severity index score of 8. These cases were also severe as per Ranson's score. According to Kim et al., [27] the estimation of severity through the Ranson's criteria is not precise and is not an appropriate method because it needs 48 h to complete and has a low specificity (77%) and sensitivity (75%).[27] 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

Table 1: Distribution of patients of acute pancreatitis with etiology
--

Etiology	Percentage of patients found $(n=200)$		
Alcoholism	73%		
Biliary sludge	19%		
Idiopathic	08%		

Table 2: Distribution of cases as per CT severity index

Grading	Mild	Moderate	Severe
CTSI score	0-3	4–6	7–10
No. of cases ($n=200$)	188	8	4
Percentage	94%	04%	02%

study, the utility of the Ranson analytic criteria compared with that of the Balthazar CT criteria for the detection of severe pancreatitis, CECT results were found to be better prognostic indicators, due to greater sensitivity and specificity.^[27]

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 pseudocyst of the pancreas the most common (in 14% 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 gallstone 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 was good while the specificity was still low. Most common complication was pseudocyst of the pancreas. In the present study, we had the objective of analyzing the various severity indices.

The initial management for an AP 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 AP 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 AP. This study of AP is an effort to recount and record each battle which the surgeon fought to treat the illness.

ACKNOWLEDGMENTS

We would like to thank all the participants of the study.

REFERENCES

- Singh VK, Bollen TL, Wu BU, Repas K, Maurer R, Yu S, *et al*. An assessment of the severity of interstitial pancreatitis. Clin Gastroenterol Hepatol 2011;9:1098-103.
- Bradley EL 3rd. A clinically based classification system for acute pancreatitis. Summary of the international symposium on acute pancreatitis, Atlanta, Ga, September 11 through 13, 1992. Arch Surg 1993;128:586-90.
- Russell RC. The pancreas. In: Russell RC, Williams NS, Bulstrode CJ, editors. Bailey and Loves' Short Practice of Surgery. 24th ed. London: Hodder Arnold; 2004. p. 1115-32.
- McKay CJ, Evans S, Sinclair M, Carter CR, Imrie CW. High early mortality rale from acute pancreatitis in Scotland 1984-1995. Br J Surg 1999;86:1302-5.
- Corfield AP, Cooper MJ, Williamson RC. Acute pancreatitis: A lethal disease of increasing significance. Gut 1985;26:724-9.
- 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. p. 939-82.
- Lerch MM, Adler G. Experimental animal models of acute pancreatitis. Int J Pancreatol 1994;15:159-70.
- 8. Grady T, Saluja A, Kaiser A. Pancreatic edema and intra-pancreatic

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;10:247-52.
- 10. Funnell IC, Bomman PC, Weakley SP. Obesity: An important prognostic factor in acute pancreatitis. Br J Surg 1993;80:484-6.
- 11. McKay CJ, Buter A. Natural history of organ failure in acute pancreatitis. Pancreatology 2003;3:111-4.
- Gloor B, Muller GA, Worni M, Martignoni ME, Uhl W, Büchler MW. Later mortality in patients with severe acute pancreatitis. Br J Surg 2001;88:975-9.
- Bolondi L, Gaiani S, Testas, Labo G. Gallbladder sludge formation during prolonged fasting after gastro-intestinal surgery. Gut 1985;26:734-8.
- Ros E, Navarro S, Bru C, Garcia-Puges A, Valderrama R. Occult microlithiasis in idiopathic pancreatitis: Prevention of relapses by cholecystectomy or ursodeoxycholic acid therapy. Gastroenterology 1991;101:1701-9.
- 15. Freund H, Pfeffiermann 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-5.
- Dervenis C, Johnson CD, Bassi C, Bradley E, Imrie CW, McMahon MJ, et al. Diagnosis, objective assessment of severity, and management of acute pancreatitis. Int J Pancreatol 1999;25:195-210.
- 20. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JH. Acute pancreatitis: Value of CT in establishing prognosis. Radiology 1990;174:331-6.
- 21. Balthazar EJ, Freeny PC, Van Sonnenberg E. Imaging and intervention in acute pancreatitis. Radiology 1994;193:297-306.
- Basterra G, Alvarez M, Marcaide A, DelgadoE, de Otazu RD, Campos FG. Acute pancreatitis: Evaluation of the prognostic criteria of the latest Balthazar tomographic classification. Rev Esp Enferm Dig 1999;91:433-8.
- Beger HG, Rau B, Mayer J, Pralle U. Naturalcourse of acute pancreatitis. World J Surg 1997;21:130-5.
- Kloppel G. Pathology of severe acute pancreatitis. In: Bradley EL 3rd editor. Acute Pancreatitis: Diagnosis and Therapy: New York: Raven; 1994. p. 35-46.
- 25. Kelly TR, Wagner DS. Gallstone pancreatitis: A prospective randomized trial of the timing of surgery. Surgery 1988;104:600-5.
- 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.
- 27. Kim YS, Lee BS, Kim SH, Seong JK, Jeong HY, Lee HY. Is there correlation between pancreatic enzyme and radiological severity in acute pancreatitis? World J Gastroenterol 2008;14:2401-2405.