

Review Article



Advantages of Modified Computed Tomography Severity Index of Acute Pancreatitis Over Other Scoring System

Md. Mofazzal Sharif¹, Khaleda Parvin Rekha², Umme Iffat Siddiqua³, Mst. Monira Khatun⁴, AKM Enamul Haque⁵, Saiful Arifin⁶.

Abstract

Pancreatitis is one of most complex and clinically challenging of all abdominal disorders. USG and abdominal Computed Tomography (CT) are the most commonly used diagnostic imaging modalities for the evaluation of pancreas. Computed Tomography (CT) is highly accurate and sensitive than USG in both diagnosing as well as demonstrating the extent. Early assessment of the cause and severity of acute pancreatitis is of utmost importance for prompt treatment and close monitoring of patient with severe disease. CT is the imaging method of choice for assessing the extent of acute pancreatitis and for evaluating complications. CT severity index is used to assess prognostic correlation and clinical outcome of acute pancreatitis. Modified CT severity index makes the score easier to calculate and reduces the inter-observer variation.

Key words: Abdominal Computed Tomography, Complications, Pancreatic diseases

Date of received: 08.11.2018.

Date of acceptance: 26.03.2019.

DOI: <https://doi.org/10.3329/kyamej.v10i2.42790>

KYAMC Journal.2019;10(2): 110-113.

Introduction

Pancreatitis is an inflammatory process in which pancreatic enzymes auto-digest the gland. The gland sometimes heals without any impairment of function or any morphologic changes; this process is known as acute pancreatitis. Pancreatitis can also recur intermittently, contributing to the functional and morphologic loss of the gland; recurrent attacks are referred to as chronic pancreatitis.¹⁻³ Once a working diagnosis of acute pancreatitis is reached, laboratory tests are obtained to support the clinical impression, to help define the etiology, and to look for complications. Diagnostic imaging is unnecessary in most cases but may be obtained when the diagnosis is in doubt, when severe pancreatitis is present, or when a given imaging study might provide specific information needed to answer a clinical question. Image-

guided aspiration may be useful. Genetic testing may be considered.⁴ Modalities for imaging pancreas range from plain x-ray to Ultrasonography (USG), endoscopic ultrasound, Endoscopic Retrograde Cholangiopancreatography (ERCP), Computed Tomography (CT), Magnetic Resonance Imaging (MRI), Magnetic Resonance Cholangiopancreatography (MRCP). Computed Tomography (CT) is highly accurate, and sensitive than USG in both diagnosing as well as demonstrating the extent. CT is a key diagnostic tool in understanding the cause of endocrine and exocrine pancreatic insufficiency in most patients. Pancreatitis is one of most complex and clinically challenging of all abdominal disorders. Management depends largely on severity. Medical treatment of mild acute pancreatitis is relatively straightforward.^{5,6} Treatment of severe acute pancreatitis involves intensive care;

1. Associate Professor, Department of Radiology & Imaging, KhwajaYounus Ali medical college and Hospital, Sirajganj, Bangladesh.
2. Assistant Professor, Department of Radiology & Imaging, KhwajaYounus Ali medical college and Hospital, Sirajganj, Bangladesh.
3. Assistant Professor, Department of Radiology & Imaging, KhwajaYounus Ali medical college and Hospital, Sirajganj, Bangladesh.
4. Lecturer, Department of Microbiology, North Bengal Medical College, Sirajganj, Bangladesh.
5. Assistant Professor of Community Medicine, ShaheedTajuddin Ahmed Medical College, Gazipur, Bangladesh.
6. Associate Professor of Biochemistry, Faridpur Diabetic Association Medical College, Faridpur, Bangladesh.

Correspondence: Dr. Khaleda Parvin Rekha, Assistant Professor, Department of Radiology & Imaging, Khwaja Yunus Ali Medical Collage & Hospital, Enayetpur, Sirajganj, Bangladesh. Phone: +8801748622229, Email: parvinrekha79@gmail.com

the goals of medical management are to provide aggressive supportive care, to decrease inflammation, to limit infection or superinfection, and to identify and treat complications as appropriate. Surgical intervention (open or minimally invasive) is indicated in selected cases.^{7,8}

Epidemiology

Acute pancreatitis has an incidence of approximately 40 cases per year per 100,000 adults. In recent years, nearly 220,000 patients with acute pancreatitis are expected to be admitted to non-federally funded hospitals. In Europe and other developed nations, such as Hong Kong, India more patients tend to have gallstone pancreatitis, whereas in the United States, alcoholic pancreatitis is most common.^{3,5,6} Generally, acute pancreatitis affects males more often than females. In males, the etiology is more often related to alcohol; in females, it is more often related to biliary tract disease. Idiopathic pancreatitis has no clear predilection for either sex.⁴

Pathophysiology

Acute pancreatitis may occur when factors involved in maintaining cellular homeostasis are out of balance. The initiating event may be anything that injures the acinar cell and impairs the secretion of zymogen granules; examples include alcohol use, gallstones, and certain drugs. At present, it is unclear exactly what pathophysiologic event triggers the onset of acute pancreatitis. It is believed, however, that both extracellular factors (eg, neural and vascular response) and intracellular factors (eg, intracellular digestive enzyme activation, increased calcium signaling, and heat shock protein activation) play a role. In addition, acute pancreatitis can develop when ductal cell injury leads to delayed or absent enzymatic secretion, as with the CFTR gene mutation.⁸⁻¹⁰

Assessment of severity and advantages of modified CT severity index

Overall mortality in patients with acute pancreatitis is 10-15%. Patients with biliary pancreatitis tend to have a higher mortality than patients with alcoholic pancreatitis. This rate has been falling over the last 2 decades as improvements in supportive care have been initiated. In patients with severe disease (organ failure), who account for about 20% of presentations, mortality is approximately 30%.^{2,5} This figure has not decreased in the past 10 years. In 1974, Ranson developed his prognostic signs. He examined the relationship of 43 different measurements made during the first 48 h of treatment, finding 11 variables that significantly correlated with overall morbidity and mortality. Imrie et al. later modified Ranson's criteria.³⁻⁶ However, the Ranson and Imrie criteria cannot be calculated until data from admission and 48 h after admission are compared. Larvin and McMahon applied the APACHE II score in the setting of acute pancreatitis. An advantage of the APACHE II score was flexibility, as it could be recalculated at any time during a hospital stay. APACHE II score had just a 67% PPV at 24 h after admission. They also showed that the APACHE II score was even less accurate for

identifying patients with specific complications including peripancreatic fluid collections or major organ failure. Thus, better prognostic tools are needed. Following the correct diagnosis of acute pancreatitis, severity stratification should be performed promptly and repeatedly after the onset, in particular for the first 48 hour.^{5,6} Several scoring systems can predict the severity of pancreatitis. Ranson's criteria, the Imrie scoring system, the Acute Physiology and Chronic Health Evaluation (APACHE II) scale, the Computed Tomography Severity Index (CTSI), Modified CTSI and Extra Pancreatic Inflammation on CT (EPIC) have been developed and validated to predict adverse outcomes, including mortality, in patients with pancreatitis.^{9,11}

Acute pancreatitis patients recover in maximum cases. Some might develop abscess, pseudocyst or duodenal obstruction. In 5 percent cases, it might result in ARDS (acute respiratory distress syndrome), DIC (disseminated intravascular coagulation), etc. Acute pancreatitis could be further divided into mild and severe pancreatitis. Mostly the Ranson's Criteria was used to determine severity of acute pancreatitis. In severe pancreatitis serious amount of necrosis determine the further clinical outcome. About 20% of the acute pancreatitis was severe with a mortality of about 20%. This was an important classification as severe pancreatitis would need intensive care therapy whereas mild pancreatitis could be treated on the common ward. Necrosis would be followed by a systemic inflammatory response syndrome (SIRS) and would determine the immediate clinical course. The further clinical course was then determined by bacterial infection. SIRS is the cause of bacterial (Gram negative) translocation from the patient's colon.^{1,2,8,12}

No single laboratory or clinical sign is pathognomonic for acute pancreatitis. Many bio-markers and inflammatory mediators for predicting the severity of acute pancreatitis are being evaluated. The initial laboratory evaluation should include amylase and lipase levels; complete blood count with differential; metabolic panel (blood urea nitrogen, creatinine, glucose, and calcium levels); triglyceride level; urinalysis and arterial blood gases. The most accurate serum indicator for acute pancreatitis is trypsin elevation. Recently a rapid urinary trypsinogen-2 test strip as screening test for acute pancreatitis has been developed.⁵⁻⁷ In predicting the prognosis, there were several scoring indices that had been used as predictors of survival. Two such scoring systems are the Ranson criteria and APACHE II (Acute Physiology and Chronic Health Evaluation) indices. The two tests that were most helpful at admission in distinguishing mild from severe acute pancreatitis were APACHE-II score and serum hematocrit. It is recommended that APACHE-II scores be generated during the first 3 days of hospitalization and thereafter as needed to help in this distinction. It was also recommended that serum hematocrit be obtained at admission, 12 h after admission, and 24 h after admission to help measure adequacy of fluid resuscitation.¹³

For the computed tomographic classification of acute pancreatitis, the Balthazar score and the CTSI were reported

by Balthazar et al in 1985 and 1990, respectively. The Balthazar score needs the assessments of both the pancreatic and the peripancreatic changes and the CTSI needs moreover the degree of pancreatic necrosis. However, the classification system needs physicians to assess many aspects, such as the presence of pancreatic enlargement, peripancreatic inflammation and the degree of fluid collections and pancreatic necrosis. Especially, regarding fluid collection, there are many locations to evaluate and it is difficult to assess all of them promptly.^{4,6} The extrapancreatic score (EP) was first reported by authors also recommended early CT scan and assessment using the EP score. Although the EP score is useful and correlates with patients' prognoses, relatively many points have to be assessed.¹⁴ In addition, some author also suggested that the EP changes especially in pararenal space paralleled the severity of acute pancreatitis. However, they also indicated that the extension of peripancreatic fluid to the splenic area did not correlate with mortality. Recently, other authors also focused on the relationship between retroperitoneal inflammation and acute pancreatitis.¹⁵ Ishikawa et al. classified the patients with acute pancreatitis into 5 grades.¹⁶ De Waele et al. reported a new scoring system based on the systemic inflammation signs on CT as EP inflammation on CT score.¹⁵ Although these classifications have a good predictive power of the outcomes, physicians need to understand the complicated retroperitoneal anatomy and evaluate many aspects. Necrosis of the pancreas develops in 5-20% of patients with acute pancreatitis. This parameter is considered by some authors to be the most important for predicting morbidity and mortality because it has been related to duration of hospitalization, local complications and mortality. For this reason, assessment of pancreatic necrosis has been added to the CT grade, resulting in the CT severity index.^{11,13} Organ failure (and particularly multisystem organ failure) rather than the extent of necrosis is more important in the morbidity and mortality of acute pancreatitis.^{4,6,7} A number of studies have demonstrated that infectious (peri) pancreatic complications (IPCs), rather than the presence of necrosis, are a key determinant of the high morbidity and mortality in patients with acute pancreatitis. Since the full extent of the necrotic process occurs at least 4 days after the onset of symptoms and an early CT may therefore underestimate the final severity of the disease, it is desirable to perform CT on admission and repeated CT for reevaluation 2 or 3 days later.^{9,13}

Conclusion

There is no simple and reliable index to predict aggravation of acute pancreatitis in the early stages. Early recognition of severe disease and application of appropriate therapy require vigilance as decisions regarding management need to be made shortly after admission, often within the first twenty for hour.¹³ Ranson's score is criticized because it requires 48 hours of observation for the judgement of severity, thus delaying the proper treatment after the onset of pain. Contrast enhanced Computed Tomography is excellent diagnostic modality to stage the severity of inflammatory process, detect the pancreatic necrosis and depict local complications and grading

of severity of acute pancreatitis within 24 hours after onset of pain.^{3,4,6,9}

Acknowledgements

We would like to give thanks the Doctors and medical staff of the department of internal medicine and pathology, Khwaja Yunus Ali Medical Collage & Hospital for their valuable support. We also grateful to the authority who allowed us to permission for publication.

References

1. Li H, Qian Z, Liu Z, Liu X, Han X, Kang H. Risk factors and outcome of acute renal failure in patients with severe acute pancreatitis. *J Crit Care* 2010; 25 (2): 225-229.
2. Leung TK, Lee CM, Lin SY, Chen HC, Wang HJ, Shen LK, et al. Balthazar computed tomography severity index is superior to Ranson criteria and APACHE II scoring system in predicting acute pancreatitis outcome. *World J Gastroenterol.* 2005; 11 (38): 6049-6052.
3. Rawla P, Bandaru SS, Vellipuram AR. Review of Infectious Etiology of Acute Pancreatitis. *Gastroenterology Research.* 2017; 10 (3): 153-158.
4. Hayes PC, Simpson KJ, Garden OJ. Alimentary tract and Pancreatic Diseases. Palmer KR, Penman ID, Paterson-Brown S. Editor. *Davidson's Principles and Practical Medicine.* 20th ed. Churchill Livingstone: Edinburgh. 2006; 899-1023.
5. Gardner TB, Vege SS, Chari ST, Pearson RK, Clain JE, Topazian MD, Levy MJ, Petersen BT. The effect of age on hospital outcomes in severe acute pancreatitis. *Pancreatology* 2008; 8(3): 265-270.
6. Gipson T, Suzannep Z. Acute Pancreatitis: Diagnosis, Prognosis, and Treatment. *Am Fam Physician* 2007; 75 (10): 1513-1520.
7. Matull WR, Pereira SP, O'Donohu JW. Biochemical markers of acute pancreatitis. *J ClinPathol* 2006; 59: 340-344.
8. Mergener J, Baillie J. Acute pancreatitis. *BMJ* 1997; 316: 44-48.
9. Mortelet KJ, Wiesner W, Intriére L, Shankar S, Zou KH, Kalantari BN, et al. A modified CT severity index for evaluating acute pancreatitis: improved correlation with patient outcome. *AJR Am J Roentgenol.* 2004; 183(5): 1261-1265.
10. Whitcomb DC. Clinical practice: Acute pancreatitis. *N Engl J Med* 2006; 354(20): 2142-2150.

11. Yadav D, Lowenfels AB. Trends in the epidemiology of the first attack of acute pancreatitis: a systematic review. *Pancreas* 2006; 33 (4): 323-330.
12. Rawla P, Sunkara T, Thandra KC, Gaduputi V. Hypertriglyceridemia-induced pancreatitis: updated review of current treatment and preventive strategies. *Clinical Journal of Gastroenterology*. 2018; 11(6): 441-448.
13. Whitcomb DC, Yadav D, Adam S. Multicenter approach to recurrent acute and chronic pancreatitis in the United States: the North American Pancreatitis Study 2 (NAPS2). *Pancreatology* 2008; 8(5): 520-531.
14. Hjelmqvist B, Wattsgard C, Borgstrom A, Lasson A, Nyman U, Aspelin P, et al. Early diagnosis and classification in acute pancreatitis. *Digestion* 1989; 44: 177-183.
15. De Waele JJ, Delrue L, Hoste EA, De Vos M, Duyck P, Colardyn FA. Extrapancreaticinflammation on abdominal computed tomography as an early predictor of disease severity in acute pancreatitis: evaluation of a new scoring system, *Pancreas*, 2007; 34: 185-190.
16. Ishikawa K, Idoguchi K, Tanaka H, Tohma Y, Ukai I, Watanabe H, et al. Classification of acute pancreatitis based on retroperitoneal extension: application of the concept of interfascial planes. *Eur J Radiol*. 2006; 60(3): 445-452.