ORIGINAL RESEARCH PAPER

Volume-7 | Issue-10 | October-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

ACUTE PANCREATITIS - REVIEW ARTICLE

Surgery

Dr Puneet Kumar Agarwal* Associate Professor, Dept of Surgery, AIIMS Bhopal *Corresponding Author

Rajesh Malik Professor, Dept of Radiodiagnosis, AIIMS Bhopal

Dr Richa Goyal Medical officer, Dept of Radiodiagnosis, AIIMS Bhopal

KEYWORDS

INTRODUCTION

Acute Pancreatitis includes a spectrum of clinical illness that ranges from mild self-limiting symptoms to rapid deterioration and death. Disease is currently defined as pancreatic inflammation that may he followed by clinical & biochemical restitution of the gland if primary cause is eliminated. (1) Severe episodes, however, may involve a progression to extensive pancreatic necrosis, development of the systemic inflammatory response syndrome (SIRS), multiorganfailure, rapid clinical deterioration, and even death. (2, 3)

ETIOLOGY

Acute pancreatitis may be attributed to a wide range of etiological factors which Include metabolic factors (alcoholism, hyperlipoproteinaemia,) mechanical (as cholelethiasis, pancreas divisum, pancreatic injury, pancreatic & duodenal obstruction & infective (mumps, cytomegalovirus). Intra-acinar activation of trypsinogen, with subsequent activation of other pancreatic enzymes, is thought to play a central role in the pathogenesis of the disease. Furthermore, ischemia-reperfusion injury is believed to be critical to disease progression. A local inflammatory response in the pancreas is associated with the liberation of oxygen-derived free radicals and cytokines including interleukin (IL)-1, IL 6,IL-8, tumor necrosis factor alpha (TNF-α), and platelet activating factor; these mediators play an important role in the transformation from a local inflammatory response to a systemic illness. (4)

ASSESSMENT

Clinical scoring system for acute pancreatitis such as Ranson's criteria and Glasgow scores use multiple variables to predict outcomes, patients are evaluated at the time of admission and after forty eight hours.(5,6,7). The Acute Physiologic and Chronic Health Evaluation II (APACHE II) SCORE is another physiological scoring system though cumbersome and not specific for acute pancreatitis but APACHE II system is as accurate at twenty four hours as other systems at forty eight hours and is now therefore regarded as perhaps the optimal scoring system to assess disease severity in acute pancreatitis. (7,8). Twelve physiological variables are measured and weighed based on their degree of abnormality: TEMP, SERUM PH, MEAN ARTERIAL PRESSURE, HEART RATE, RESPIRATORY RATE,ARTERIAL OXYGEN TENSION,SERUM SODIM ,SERUM POTTASIUM , SERUM CREATININE , HAEMATOCRIT , WHITE BLOOD CUELL COUNT AND GLASGOW COMA SCALE (9,10). Prominent initial symptom is upper abdominal pain, which radiates to back (in 50% of patients) and often start after a heavy meal or during a drinking binge. Nausea and vomiting is also present in about ninety two percent of patients. Findings on physical examination include Tachycardia, Tachypnea, and abdominal distension with epigastric fullness with generalized abdominal tenderness more marked in upper abdomen. Periumblical and Flank bruising may be seen with severe and hemorrhagic pancreatitis (Cullen s and Grey Turners sign) Lab findings include elevated serum Amylase (95% of patients at the time of admission), elevated serum lipase levels (more specific than amylase), hyperglycemia and hypocalcaemia.

ROLE OF IMAGING

Imaging studies serve as tool as CT scan, it provides better definition of pancreatic anatomy .CT findings in pancreatitis include enlargement of pancreas with loss of peri pancreatic fat planes, areas of decreased density and occasionally the presence of fluid collection. The sensitivity for identifying pancreatic necrosis with CT scan approaches 100%, four days from the diagnosis. CT scan also has been instrumental in facilitating the early diagnosis of infected pancreatic necrosis. Using image guided, precise aspiration of necrotic pancreas however, infected pancreatic necrosis can be diagnosed with high degree of accuracy.(11-16)

MANAGEMENT

While the treatment of mild pancreatitis has changed little in recent years, advances in the management of severe pancreatitis have been associated with significantly reduced morbidity and mortality. Improvements in the recognition of severe disease with scoring system and CT Scanning has allowed early goal therapy in appropriate patients.

Timely resuscitation and invasive monitoring are standard and there is an increased recognition of the role of prophylactic antibiotics for pancreatic necrosis and image guided FNA to diagnose infection.

While the need for aggressive intervention in infected pancreatic necrosis remain unchanged, initial conservative management of most patients with sterile pancreatic necrosis has gained wide spread acceptance; some patients with sterile necrosis eventually may require delayed debridement either for persistent systemic illness or for failure to thrive, although accurate prospective identification on these patients has not been possible. (17-28)

For patients needing debridement open surgical technique remains the gold standard of management. Advances in minimally invasive technology hold promise as adjuncts to open procedure in the future; particularly as a means of delaying surgery to facilitate debridement when the necrotic pancreas become more organized. (29)

REFERENCES

1. Banks PA. Acute pancreatitis: medical and surgical management. Am JGastroenterol. 1994 Aug;89(8 suppl):S78–S85.
2. Beger HG, Rau B, Mayer J, Pralle U. Natural course of acute pancreatitis.World J Surg. 1997 Feb;21(2):130–135.
3. Yousaf M, McCallion K, Diamond T. Management of severe acute pancreatitis. Br J Surg. 2003 Apr;90(4):407–420.
4. Sakorafas GH, Tsiotou AG. Etiology and pathogenesis of acute pancreatitis: current concepts. J Clin Gastroenterol. 2000 Jun;30(4):343–356.
5. Ranson JH, Rifkind KM, Roses DF, Fink SD, Eng K, Spencer FC. Prognostic signs and the role of operative management in acute pancreatitis.Surg Gynecol Obstet. 1974 Jul;139(1):69–81.
6. Blamey SL, Imrie CW, O'Neill J, Gilmour WH, Carter DC. Prognostic factors in acute pancreatitis. Gut. 1984 Dec;25(12):1340–1346.
7. Eachempati SR, Hydo LJ, Barie PS. Severity scoring for prognostication in patients with severe acute pancreatitis: comparative analysis of the Ranson score and the APACHE III score. Arch Surg. 2002 Jun;137(6):730–736.
8. Connor S, Ghaneh P, Raraty M, et al. Increasing age and APACHE II scores are the main determinants of outcome from pancreatic necrosectomy. Br J Surg. 2003 Dec;90(12):1542–1548.
9. Williams M, Simms HH. Prognostic usefulness of scoring systems in critically ill patients with severe acute pancreatitis. Crit Care Med. 1999 May;27(5):901–907.
10 Triester SL, Kowdley KV. Prognostic factors in acute pancreatitis. J Clin Gastroenterol. 2002 Feb;34(2):167–176.
11. Balthazar EJ, Ranson JH, Naidich DP, Megibow AJ, Caccavale R, Cooper MM. Acute pancreatitis: prognostic value of CT. Radiology. 1985 Sep;156(3):767–772.
12. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JH. Acute pancreatitis: value of CT in establishing prognosis. Radiology. 1990 Feb;174(2): 331–336.
13. 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 May; 128(5):586–590.
14. Arvanitakis M, Delhaye M, De Maertelaere V, et al. Computed tomographyand

magnetic resonance imaging in the assessment of acute pancreatitis. Gastroenterology. 2004 Mar;126(3):715–723.
15. Schmidt J, Hotz HG, Foitzik T, et al. Intravenous contrast medium aggravatesthe impairment of pancreatic microcirculation in necrotizing pancreatitis in the rat. Ann Surg. 1995 Mar;221(3):257–264.
16. Banks PA, Gerzof SG, Langevin RE, Silverman SG, Sica GT, Hughes MD.CT-guided aspiration of suspected pancreatic infection: bacteriology andclinical outcome. Int J Pancreatol. 1995 Dec; 18(3):265–270.
17. Toouli J, Brooke-Smith M, Bassi C, et al. Guidelines for the management of acute pancreatitis. J Gastroenterol Hepatol. 2002 Feb;17(suppl): S15–S39.
18. Uhl W, Warshaw A, Imrie C, et al. IAP Guidelines for the Surgical Managementof Acute Pancreatitis. Pancreatology. 2002;2(6):565–573.
19. Tenner S, Banks PA. Acute pancreatitis: nonsurgical management. World J Surg. 1997 Feb;21(2):143–148.
20. Rau B, Uhl W, Buchler MW, Beger HG. Surgical treatment of infected necrosis. World J Surg. 1997 Feb;21(2):155–161.
21. Bradley EL, 3rd, Allen K. A prospective longitudinal study of observation versus surgical intervention in the management of necrotizing pancreatitis. Am J Surg. 1991 Jan;161(1):19–24; discussion 24–15.
22. Lumsden A, Bradley EL, 3rd. Secondary pancreatic infections. Surg GynecolObstet. 1990 May;170(5):459–467.
23. Bassi C, Falconi M, Talamini G, et al. Controlled clinical trial of pefloxacin versus imipenem in severe acute pancreatitis. Gastroenterology. 1998 Dec;115(6):1513–1517.
24. Fernandez-del Castillo C, Rattner DW, Makary MA, Mostafavi A, McGrath D, Warshaw AL. Debridement and closed packing for the treatment of necrotizing pancreatitis. Ann Surg. 1998 Nov;228(5):676–684.
25. Rattner DW, Legermate DA, Lee MJ, Mueller PR, Warshaw AL. Early surgical debridement of symptomatic pancreatic necrosis is beneficial irrespective of infection. Am J Surg. 1992 Jan;163(1):105–109; discussion 109–110.
26. McFadden DW, Reber HA. Indications for surgery in severe acute pancreatitis. Int J Pancreatol. 1994 Apr;15(2):83–90.
27. Rau B, Pralle U, Uhl W, Schoenberg MH, Beger HG. Management of sterile necrosis in instances of severe acute pancreatitis. J Am Coll Surg. 1995 Oct;181(4):279–288.
28. Ashley SW. Sterile pancreatic necrosis: is operation necessary? J Am Coll Surg. 1995 Oct;181(4):363–364.
29. Carter R. Management of infected necrosis secondary to acute pancreatitis: a balanced role for minimal access techniques. Pancreatology. 2003; 3(2):133–138