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Clinical Reviews

ACUTE PANCREATITIS: WHAT'S THE SCORE?

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☐ **Abstract—Background: Acute pancreatitis (AP) is a common presentation in the emergency department (ED). Severity of pancreatitis is an important consideration for ED clinicians making admission judgments. Validated scoring systems can be a helpful tool in this process. Objective: The aim of this review is to give a general outline on the subject of AP and compare different criteria used to predict severity of disease for use in the ED. Discussion: This review updates the classifications and scoring systems for AP and the relevant parameters of each. This article assesses past and current scoring systems for AP, including Ranson criteria, Glasgow criteria, Acute Physiology and Chronic Health Evaluation II (APACHE II), computed tomography imaging scoring systems, Bedside Index of Severity in Acute Pancreatitis (BISAP) score, Panc 3, Harmless Acute Pancreatitis Score (HAPS), and the Japanese Severity Score. This article also describes the potential use of single variable predictors. Finally, this article discusses risk factors for early readmission, an outcome pertinent to emergency physicians. These parameters may be used to risk-stratify patients presenting to the ED into mild, moderate, and severe pancreatitis for determination of appropriate disposition. Conclusion: Rapid, reliable, and validated means of predicting patient outcome from rapid clinical assessment are of value to the emergency physician. Scoring systems such as BISAP, HAPS, and single-variable predictors may assist in decision-making due to their simplicity of use and applicability within the first 24 h.** © 2015 Elsevier Inc.

☐ **Keywords—Acute pancreatitis; Ranson's criteria; BISAP score; scoring systems; disease severity**

INTRODUCTION

The incidence of acute pancreatitis (AP) has been on the rise worldwide (1). AP has a yearly incidence of approximately 13–45 new cases per 100,000 per year, with an estimated annual cost of $2.2 billion each year for admissions (2,3). Although a number of guidelines and scoring systems exist, there is a considerable amount of inconsistency in the diagnosis and management of this disease process, and few physicians are familiar with any tools for risk stratification beyond Ranson criteria.

In 1997, the American College of Gastroenterology produced guidelines for the diagnosis and treatment of AP that have since been updated in 2006 and 2013 (4,5). These guidelines advocate for the rapid determination of hemodynamic status and initiation of resuscitative efforts and risk assessment for the appropriate stratification of patients with AP. Classic risk assessment tools such as Ranson criteria can predict disease severity and dictate treatment in AP, but they have a significant limitation. Many of the criteria are not obtainable at presentation to the emergency department (ED), which makes the job of ascertaining how the patient's disease will progress difficult for emergency physicians. Evidence-based guidelines need to be in place to best risk-stratify patients early in the disease course. This article will briefly review the clinical features, diagnosis, and management of AP and familiarize readers with predictors

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of severe disease and readmission other than Ranson criteria.

DISCUSSION

Pathophysiology

The disease process in AP is described in three phases (6). The first phase is due to leakage of pancreatic enzymes into pancreatic tissue secondary to injury or disruption of the pancreatic acini. The leaked enzyme, trypsinogen, becomes activated into trypsin, causing subsequent edema, vascular damage, hemorrhage, and necrosis of the pancreas. The second and third phases of AP are characterized by intrapancreatic and extrapancreatic inflammation, respectively.

The two most common and important causes of AP are gallstones (40–70%) and alcohol (25–35%) (5,7). Gallstone pancreatitis is usually due to an obstructing stone in the pancreatic duct near the sphincter of Oddi (6). In alcohol-related pancreatitis, it is believed that the acinar cells of the pancreas are susceptible to damage by ethanol and underlie the etiology of the disease (8). Another common cause, iatrogenic pancreatitis, may occur after endoscopic retrograde cholangiopancreatography (ERCP) in up to 5% of patients. Other etiologies of AP include medications, infections, trauma, hereditary, and autoimmune disease (Table 1).

Diagnostic Guidelines

According to the American College of Gastroenterology, a patient must have two of the following three features present to make a diagnosis of AP: characteristic abdominal pain, an elevated serum amylase or lipase ≥ 3 times the upper limit of normal, and characteristic findings of AP on computed tomography (CT) scan (5). The decision to obtain relevant laboratory studies and imaging is based on interpretation of the clinical presentation. Classically, patients with AP will report mild to severe epigastric abdominal pain that persists for more than 24 h. The pain may be characterized as constant, dull, and radiating to the back. The pain tends to be more severe in the supine position; sitting up and leaning forward may provide partial relief. Nausea, vomiting, and exacerbation with oral intake are other common symptoms (5). Patients may also complain of shortness of breath or impaired respiratory function secondary to pleural effusion, diaphragm irritation, or in severe cases, acute respiratory distress syndrome.

Physical examination in mild AP reveals normal or slightly elevated vital signs and mild epigastric tenderness with the absence of peritoneal signs. Patients with severe AP will display abnormal vital signs, including fever in 50% of patients. Significant abdominal tenderness, rebound, and guarding may be present in severe AP. Breath and bowel sounds may both be decreased. Cutaneous findings, like Cullen's and Grey-Turner's signs, are rare (9).

Current guidelines state that serum amylase or lipase elevation ≥ 3 times the upper limit of normal is one of the diagnostic criteria for AP. Both are typically elevated within the first 24 h of symptoms. Lipase is the preferred test, but the criteria allow for an elevation of either enzyme for diagnosis. Serum lipase, when elevated to ≥ 3 times the upper limit of normal, has been found to have a specificity of 99% for AP and a sensitivity of 100%. Amylase has been shown to have a specificity of up to 99% when ≥ 3 times the upper limit of normal, but is less sensitive and may be elevated in unrelated disease states such as pregnancy,

Table 1. Etiologies of Pancreatitis

Most Common	Medications	Infections	Hereditary	Other
Gallstones	Azathioprine	Bacterial:	Familial hypercalcemia	Idiopathic
Alcohol	Sulfonamides	Mycoplasma pneumoniae	Hypertriglyceridemia	Autoimmune
Triglycerides	Sulindac Tetracycline	Salmonella typhosa	Mutations:	Trauma
Iatrogenic (ERCP)	Valproic acid Didanosine	*Leptospira*	CFTR gene mutations	
	Methyldopa	*Campylobacter*	PRSS1	
	Estrogens	*Mycobacterium tuberculosis*	SPINK1	
	Furosemide	Viral:	Pancreas divisum	
	6-Mercaptopurine Pentamidine	EBV	Sphincter of Oddi dysfunction	
	5-Aminosalicylic acid Compounds	Coxsackie virus		
	Corticosteroids	Echovirus		
	Octreotide	Varicella-zoster		
		Measles		
		Other:		
		Ascariasis		

ERCP = endoscopic retrograde cholangiopancreatography; EBV = Epstein-Barr virus; CFTR = cystic fibrosis transmembrane conductance regulator; PRSS1 = protease, serine, 1 (trypsin 1); SPINK1 = serine protease inhibitor Kazal-type 1.