



EARLY DETECTION OF ACUTE PANCREATITIS AND OPTIMIZATION OF ITS MANAGEMENT: CURRENT EVIDENCE AND PRACTICAL RECOMMENDATIONS

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Acute pancreatitis ranges from a self-limited illness to multi-organ-failure with infected pancreatic necrosis and long-term sequelae

ABSTRACT

Acute pancreatitis (AP) is a common, potentially life-threatening inflammatory condition of the pancreas. Early detection of AP and accurate early risk stratification are essential to prevent progression to severe disease and guide resource-intensive interventions. Recent guideline updates and trials (2022–2025) have clarified several aspects of early diagnosis, biomarker research, fluid resuscitation, nutritional support, timing of biliary interventions, and minimally invasive management of necrotizing disease. This article reviews contemporary diagnostic tools (clinical, biochemical, imaging, biomarker, and AI/EHR approaches), synthesizes evidence for early management strategies that reduce complications, and highlights promising research directions including multi-omics biomarkers and machine-learning risk prediction.

Introduction. Acute pancreatitis ranges from a self-limited illness to multi-organ-failure with infected pancreatic necrosis and long-term sequelae. Early recognition and targeted initial care — within the first 24–72 hours — strongly influence patient trajectory, length of stay, and mortality. Recent international guideline revisions and randomized trials have refined best practice recommendations and challenged some long-standing assumptions.

2. Early detection: clinical and laboratory signals. Clinical presentation and red flags

Typical presentation remains acute epigastric pain radiating to the back, nausea/vomiting, and elevated pancreatic enzymes. Early red flags for possible severe disease include persistent tachycardia, hypotension, oliguria, early organ dysfunction, and signs of SIRS. Risk stratification should begin immediately at presentation.

Standard biochemical tests/ Serum amylase and lipase are the mainstay for diagnosis; lipase has greater sensitivity and specificity. However, enzyme elevation alone does not predict severity. Basic labs (hematocrit, BUN/creatinine), lactate, and early organ-function markers remain crucial for early triage.

Emerging biomarkers and multi-omics.

Research in 2023–2025 has focused on early pancreatic-specific markers (trypsinogen isoforms and trypsinogen activation peptide), inflammatory markers (procalcitonin), and

high-throughput “omics” (proteomics, metabolomics, circulating microRNA) to detect pancreatitis and predict severity earlier than traditional tests. Recent systematic reviews show promise for serum trypsinogen-2 and certain proteomic/metabolomic signatures, but results remain heterogeneous and not yet ready for universal clinical deployment. Ongoing multi-centre validation and standardized assays are required before routine adoption.

Imaging for early diagnosis. Transabdominal ultrasound is often the first imaging test to identify biliary etiology or gallstones. Contrast-enhanced CT remains the main imaging to stage pancreatic necrosis but is usually reserved >48–72 hours after onset unless diagnosis is unclear or there is clinical deterioration. Endoscopic ultrasound (EUS) and MRI/MRCP provide high sensitivity for biliary obstruction, choledocholithiasis, and small pancreatic collections. Electronic health records and AI for early detection. Machine-learning models applied to EHR data and combined biomarker panels have demonstrated improved early prediction of severe AP compared with traditional scores in retrospective and prospective cohorts (2023–2025). These tools can flag high-risk patients early, enabling prioritization for critical monitoring and early interventions; however, external validation and prospective implementation studies are still limited and required before routine clinical use.

3. Early risk stratification: scoring systems vs. ML Traditional scoring systems BISAP, APACHE II, and the Balthazar CTSI are widely used. BISAP is simple, bedside-friendly, and performs reasonably well for early risk stratification. However, single scores have limited sensitivity and should not replace dynamic clinical judgment. **Machine learning and ensemble methods** Recent studies (2023–2025) show that ML models that combine demographic data, vital signs, trends in routine labs, and imaging features outperform single traditional scores in discrimination for severe outcomes. When deployed carefully (with transparency about inputs and periodic recalibration), these models can enhance early triage and resource allocation. Prospective trials demonstrating outcome improvements are still needed.

4. Initial management optimization (first 24–72 hours) Fluid resuscitation: balance is essential Large randomized evidence (WATERFALL trial and subsequent guideline syntheses) shows that **aggressive early fluid resuscitation** increases the risk of fluid overload without improving outcomes versus more moderate, goal-directed therapy. Contemporary recommendations favor individualized, goal-directed fluid management (using clinical endpoints, urine output, hematocrit/BUN trends, and frequent reassessment) with preference for balanced crystalloids (e.g., lactated Ringer’s) over normal saline when fluids are indicated. Pain control and supportive care. Early, adequate analgesia (opioid or multimodal analgesia as appropriate), antiemetics, oxygen/ventilatory support if hypoxic, and monitoring for early organ dysfunction are foundational. Early involvement of a multidisciplinary team (gastroenterology, surgery, critical care, nutrition) is recommended for patients at risk of severe disease.

Early enteral nutrition. Early enteral nutrition — preferably oral if tolerated, or nasoenteric feeding if not — reduces infectious complications and length of stay compared with prolonged fasting or parenteral nutrition. ESPEN and recent clinical guidance support initiating oral intake as soon as pain and nausea permit, rather than waiting for complete normalization of pancreatic enzymes. Avoid routine total parenteral nutrition (TPN) unless

strictly indicated. Antibiotics and infection prevention. Prophylactic systemic antibiotics are not recommended to prevent infected pancreatic necrosis. Antibiotics should be reserved for proven or strongly suspected extrapancreatic infection or documented infected necrosis. Source control (drainage/debridement) should be guided by culture results and multidisciplinary assessment. Biliary pancreatitis: ERCP timing. For biliary pancreatitis, **urgent ERCP within 24 hours is indicated in patients with cholangitis or persistent biliary obstruction**. In the absence of cholangitis or persistent obstruction, routine early ERCP (within 72 hours) is generally not recommended and medical management is preferred; timing should be individualized. Cholecystectomy during the same admission for gallstone pancreatitis is usually recommended once the patient's condition stabilizes to reduce recurrence.

5. Management of necrotizing and infected pancreatitis. Step-up approach and minimally invasive techniques. For necrotizing pancreatitis complicated by infection or organ failure, a step-up strategy—beginning with percutaneous or endoscopic drainage, followed by minimally invasive necrosectomy if needed—improves outcomes relative to open surgical necrosectomy. Timing is important: delayed interventions (after demarcation of necrosis, often ≥ 4 weeks) reduce morbidity when clinically feasible. **Role of endoscopic transluminal necrosectomy.** Transluminal endoscopic necrosectomy (endoscopic drainage followed by direct endoscopic necrosectomy) is an established option for suitable walled-off necrosis and is associated with shorter recovery and fewer fistulas compared with open surgery, when performed in experienced centers. Prevention, follow-up, and rehabilitation. Identify and treat underlying causes (gallstones — cholecystectomy, alcohol use — structured cessation interventions, hypertriglyceridemia — lipid management). Screen and manage early endocrine/exocrine insufficiency after severe or necrotizing pancreatitis. Patient education, vaccination where appropriate, and planned outpatient follow-up for nutrition and glycemic control reduce readmissions and long-term complications.

Research priorities and future directions

1. Validated, standardized biomarker panels and point-of-care assays (trypsinogen isoforms, proteomics, microRNA) to enable reliable early detection and severity prediction.

2. Prospective trials of ML decision-support embedded in EHRs to test whether AI-guided early triage improves patient-centred outcomes.

3. Personalized fluid and hemodynamic protocols using dynamic monitoring (biomarkers + ultrasound/cardiac output) rather than fixed aggressive regimens.

4. Continued refinement of minimally invasive therapies and optimal timing for interventions in necrotizing disease.

Conclusion. Early detection of AP and optimization of initial care reduce progression to severe disease and improve outcomes. While standard clinical evaluation and routine labs remain essential, incorporation of validated biomarkers, multi-omics, and responsibly deployed ML tools promises earlier and more accurate prognostication. Management has shifted toward individualized, conservative resuscitation, early enteral nutrition, targeted biliary intervention only when indicated, and minimally invasive strategies for necrotizing disease. Continued large-scale validation studies and pragmatic trials are required to translate novel diagnostics into routine practice.

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