



Acute Pancreatitis: CT Severity Index, Fluid-Resuscitation Nursing Algorithms, and Lipase/CRP as Outcome Markers

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Abstract

Background: Acute pancreatitis (AP) is a common, potentially life-threatening inflammatory condition with heterogeneous etiologies and trajectories. Early risk stratification and coordinated supportive care are pivotal to reduce necrosis, organ failure, and resource use.

Aim: To synthesize evidence on three practical pillars of inpatient AP care—radiologic grading by CT Severity Index (CTSI), fluid-resuscitation nursing algorithms, and biochemical markers (lipase and C-reactive protein, CRP)—and to propose an integrated, bedside-ready framework.

Methods: Narrative review of contemporary diagnostic and management domains summarized in this article, emphasizing: (1) imaging roles (ultrasound/CECT/MRI) and CTSI for local complications; (2) nurse-led fluid protocols (targets, choice of crystalloid, ROSE phases); and (3) serial lipase/CRP as outcome markers alongside clinical severity classifications.

Results: CTSI reliably stages local pancreatic/peripancreatic complications and complements clinical systems (e.g., RAC/DBC) for prognostication and timing of interventions. Nurse-delivered resuscitation algorithms with early lactated Ringer's at 5–10 mL/kg/h, dynamic endpoints (heart rate <120 beats/min, urine output >0.5 mL/kg/h), and avoidance of overload reduce SIRS, organ failure, and length of stay. Early enteral nutrition embedded in nursing pathways improves tolerance and pain trajectories. Lipase confirms diagnosis but trends add limited prognostic value; CRP trajectories (e.g., 48–72 h) better correlate with severity and necrosis, especially when interpreted with imaging and physiology. Multidisciplinary coordination among radiology, laboratory, and nursing closes operational gaps and accelerates definitive etiologic care.

Conclusion: An integrated pathway combining CTSI-guided imaging, protocolized nurse-led fluid resuscitation, and judicious use of lipase/CRP improves risk recognition, streamlines decisions, and may reduce complications and costs in AP.

Key words: acute pancreatitis; CT Severity Index; lactated Ringer's; nursing algorithms; lipase; C-reactive protein; risk stratification; enteral nutrition.

Introduction

Acute pancreatitis is a sudden inflammatory disorder of the exocrine pancreas characterized clinically by severe epigastric pain and biochemically by elevations in serum amylase and lipase, with a subset of patients progressing to local or systemic organ dysfunction [1]. On a population level, contemporary epidemiology places the global incidence at approximately 30–40 cases per 100,000 persons annually, underscoring a substantial and rising

healthcare burden across diverse health systems [1]. Although less common in pediatric populations, acute pancreatitis in children remains clinically significant, with reported incidence estimates of 10–15 cases per 100,000 children per year and distinct etiologic spectra that require age-appropriate diagnostic frameworks and supportive care pathways [2]. Mortality typically ranges from 1–5%, but risk escalates sharply in the presence of pancreatic necrosis and multi-organ failure, highlighting the need for early risk

stratification, vigilant hemodynamic management, and timely evaluation for complications such as infected necrosis or hemorrhage [1]. From a health economics perspective, per-episode expenditures average around USD 10,000, with prolonged stays and readmissions amplifying direct costs and productivity losses, thereby magnifying the socio-economic impact on patients, families, and payers [3]. Despite a very large literature base—spanning tens of thousands of publications addressing diagnosis, classification, and management—therapeutic development has been hampered by heterogeneous etiologies, variable disease trajectories, and a relative paucity of high-quality randomized trials testing targeted interventions [1]. Consequently, contemporary care continues to emphasize precise etiologic determination (e.g., gallstones, alcohol, hypertriglyceridemia, drugs) and complication profiling, because both dimensions shape inpatient fluid resuscitation strategies, analgesia, nutritional support, timing and modality of imaging, and downstream outpatient plans, including cholecystectomy or lipid-lowering therapy where indicated [1],[3]. Accurate and timely diagnosis integrates clinical assessment with biochemical confirmation and judicious imaging to delineate severity and detect local complications, aiming to mitigate progression to necrosis, reduce resource utilization, and improve patient-centered outcomes across adult and pediatric cohorts [2],[3]. In this article, we synthesize the current state of acute pancreatitis with particular emphasis on diagnostic precision as the keystone for etiology-specific and complication-aware management across the continuum of care [1].

Diagnostic Criteria

The diagnosis of acute pancreatitis (AP) relies on the fulfillment of at least two out of three internationally recognized criteria: (1) characteristic abdominal pain radiating to the back, (2) serum lipase or amylase levels elevated to at least three times the upper limit of normal, and (3) confirmatory findings consistent with pancreatitis on imaging, typically computed tomography (CT) or magnetic resonance imaging (MRI) [4]. These diagnostic criteria have been standardized to enhance diagnostic accuracy and ensure prompt intervention, yet they are not without limitations. It is well established that relying solely on the first two criteria may lead to underdiagnosis, with up to 25% of true pancreatitis cases potentially being overlooked due to atypical biochemical or clinical presentations [5]. Consequently, a comprehensive diagnostic approach incorporating imaging and clinical evaluation remains indispensable. Imaging plays a pivotal role in the confirmation and classification of AP severity. Contrast-enhanced CT (CECT) remains the gold standard for detecting pancreatic necrosis, pseudocysts, and other local complications, but its use must be judicious. The administration of intravenous contrast carries a risk of

nephrotoxicity, especially in patients with underlying renal impairment or severe dehydration, both of which are common in the early phase of AP [6]. Moreover, CT scanning involves exposure to ionizing radiation, which, when repeated across multiple episodes or follow-ups, can impose cumulative biological risks. Therefore, MRI emerges as a valuable alternative, particularly for patients requiring serial imaging or for those in whom contrast administration is contraindicated. However, MRI's higher cost, longer acquisition time, and limited availability in certain settings may restrict its utility, particularly in resource-constrained environments [6]. Clinically, the presentation of acute pancreatitis is often variable, necessitating a high degree of suspicion by attending physicians. The predominant symptom is severe epigastric or diffuse abdominal pain, present in approximately 80–90% of cases, often radiating posteriorly and exacerbated by food intake [7]. Accompanying manifestations such as abdominal distension, nausea, vomiting, fever, tachycardia, and tachypnea occur in up to 50–80% of patients, reflecting both systemic inflammatory response and local pancreatic irritation [7]. Given this heterogeneity, a thorough assessment combining history, laboratory data, and targeted imaging is crucial for accurate diagnosis. Clinicians must not only interpret biochemical markers within their clinical context but also balance diagnostic yield against potential harm from imaging modalities. The integration of these considerations ensures timely, accurate diagnosis and forms the cornerstone for effective management of acute pancreatitis [4–7].



Fig. 1: Severe Necrotizing Pancreatitis.

Etiologies

A precise understanding of the etiologies of acute pancreatitis (AP) is foundational to effective management and secondary prevention, and it begins with a meticulous clinical history at the time of first presentation. Because the exocrine pancreas can be injured by mechanical obstruction, toxic-metabolic insults, immune dysregulation, and iatrogenic or traumatic mechanisms, causal attribution is often multifactorial, and the dominant driver may change over a patient's lifetime. Accordingly, clinicians

should take a structured history covering biliary symptoms, alcohol and tobacco use, metabolic risk factors, prior procedures, medication and supplement exposure, and relevant infectious or traumatic events, while simultaneously integrating laboratory indices and imaging features to triangulate etiology. Gallstones, alcohol misuse, and hypertriglyceridemia constitute the most common global causes, but autoimmune conditions, anatomic variants such as pancreas divisum, obesity-related metabolic perturbations, drug-induced injury, and post-endoscopic retrograde cholangiopancreatography (ERCP) pancreatitis are also important contributors in specific populations and settings. Even when a single cause appears likely, clinicians should actively consider coexisting or compounding factors—for example, hypertriglyceridemia in an individual who also binges alcohol—because recognizing such interactions informs tailored inpatient care and targeted outpatient strategies to reduce recurrence risk. In practice, the etiologic evaluation should proceed in parallel with resuscitation and early supportive care, since timely attribution enables definitive interventions such as biliary decompression, lipid-lowering therapy, alcohol cessation support, or medication withdrawal when indicated [8–11], [20–24], [25–31], [32–33].

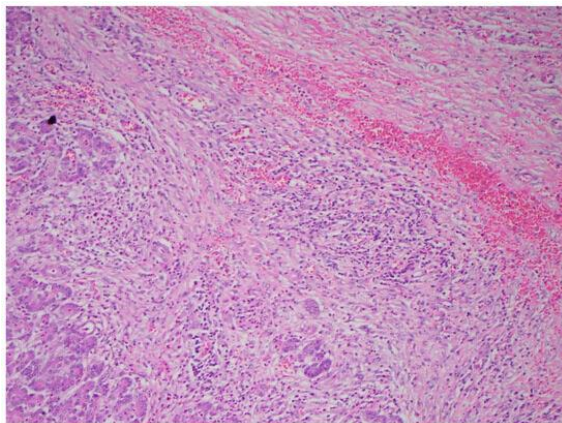


Fig. 2: Acute Pancreatitis with hemorrhage.

Gallstones

Gallstone disease is the leading etiology of AP worldwide and accounts for approximately half of cases in many Western cohorts, reflecting the high prevalence of cholelithiasis and the potential for transient or persistent obstruction of the ampulla by stones or sludge [8], [9]. Epidemiologic analyses consistently demonstrate that cholelithiasis prevalence increases with age, which naturally translates into a rising burden of biliary pancreatitis in older adults; at the same time, sex-related differences persist, with women comprising about two-thirds of biliary pancreatitis cases in large Western series, likely related to hormonal and reproductive influences on bile composition and gallbladder motility [9], [10]. The pathophysiologic mechanism centers on obstruction of the pancreatic duct outflow by a migrating stone or microlithiasis, which raises

intraductal pressures, disrupts acinar cell zymogen trafficking, and precipitates intrapancreatic enzyme activation. In many patients the obstructive episode is brief and self-limited, yet even transient obstruction can trigger a clinically significant inflammatory cascade. Because biliary pancreatitis carries implications for early intervention and recurrence prevention, the initial diagnostic work-up should nearly always incorporate right upper-quadrant ultrasonography to detect gallstones, gallbladder wall changes, or common bile duct (CBD) dilation that might signal choledocholithiasis or cholangitis [11].

Decisions about advanced imaging and endoscopic management hinge on pretest probability of persistent CBD stones and the presence of systemic toxicity or sepsis. When ultrasonography reveals CBD dilation or laboratory evidence suggests cholestasis, further evaluation with magnetic resonance cholangiopancreatography (MRCP) or endoscopic ultrasound (EUS) may noninvasively define the biliary tree and guide the need for therapeutic ERCP [11]. In the subset with ascending cholangitis, urgent ERCP is indicated to decompress the biliary system and remove obstructing stones, an intervention that can be life-saving and also curtail the pancreatic inflammatory drive [11]. After recovery from the index event, early cholecystectomy is recommended in most patients with gallstone pancreatitis to prevent recurrence, given the high short-term risk of repeat biliary events; timing is individualized to clinical stability and the presence of local complications. The overall trajectory of biliary pancreatitis is often favorable with rapid improvement once obstruction resolves, but vigilance is required for local collections or necrosis, particularly in older patients with comorbidities [8], [9], [11]. In health systems terms, the predominance of this etiology underscores the population-level benefits of timely imaging, appropriate triage for ERCP, and definitive gallbladder surgery to reduce recurrence and downstream costs [10], [11].

Alcohol

Alcohol-related pancreatitis represents the second most common cause of AP across North America and Europe, accounting for roughly one-third of cases in aggregate datasets [12], [13]. The risk is driven both by cumulative exposure—typically years of heavy consumption that remodels acinar cell physiology, sensitizes the pancreas to injurious stimuli, and alters ductal secretion—and by acute binge patterns that can precipitate an episode in a susceptible pancreas [16]. Binge drinking, defined in many studies as five or more standard drinks per occasion (approximately 70 g of ethanol for men and 56 g for women), has been linked to seasonal and event-driven spikes in incidence, with recognizable surges during holiday periods when alcohol intake is more intense and clustered [14], [15]. The clinical penetrance of alcohol as an etiologic factor is amplified by co-exposures—especially cigarette smoking—which independently elevates the risk of

AP, recurrent acute pancreatitis, and progression to chronic pancreatitis, likely through synergistic oxidative and inflammatory mechanisms within the acinar microenvironment [17].

Intriguingly, cohort analyses suggest that very low levels of alcohol intake may exert modest protective ductal secretory effects in non-smokers with respect to a first AP episode, a nuance that does not translate into protection against recurrences and certainly does not extend to individuals who smoke, underlining the complex, dose- and context-dependent biology of ethanol on the pancreas [18], [19]. From a diagnostic standpoint, attributing etiology to alcohol requires careful interviewing to quantify both average weekly intake and binge frequency, while screening for withdrawal risk and coexisting liver disease. Laboratory tests are non-specific but may reveal macrocytosis or transaminase patterns consistent with alcohol use; imaging is often deployed to exclude biliary causes and to assess for complications, including peripancreatic collections. Management must go beyond index hospitalization to encompass structured alcohol cessation interventions, linkage to addiction services, and smoking cessation support, because recurrent alcohol-related AP is a potent driver of chronic pain, exocrine insufficiency, and healthcare utilization [12], [13], [17]. In systems with integrated care pathways, coordinated addiction medicine, nursing counseling, and primary care follow-up reduce relapse and readmission, translating etiologic insight into durable outcome benefits [14–16], [18], [19].

Hypertriglyceridemia

Hypertriglyceridemia (HTG) has emerged as the third most common cause of AP globally, implicated in roughly 9% of cases in pooled analyses, with even higher proportions reported in certain regions and referral centers [20]. A prominent high-volume tertiary hospital series from China observed that approximately one-third of AP presentations were attributable to HTG, underscoring geographic and demographic variability linked to diet, metabolic syndrome prevalence, and genetic predispositions [21]. The Endocrine Society categorizes HTG as mild (150–500 mg/dL), moderate (500–1000 mg/dL), and severe (>1000 mg/dL), a framework that maps to pancreatitis risk and guides acute and longitudinal management [22]. Above about 1000 mg/dL, the risk of AP rises sharply, and epidemiologic modeling suggests an approximately 4% increase in AP incidence for every additional 100 mg/dL increment beyond that threshold, a striking gradient that emphasizes the need for aggressive risk factor control in very high triglyceride states [23].

The pathogenesis of HTG-AP is thought to involve hydrolysis of triglyceride-rich lipoproteins within pancreatic capillaries, generating free fatty acids that are directly toxic to acinar and endothelial cells and that exacerbate local ischemia and inflammation. Because triglyceride levels may decline

rapidly with fasting and initial resuscitation, it is essential to obtain a lipid panel upon admission—ideally before significant intravenous fluid therapy—to avoid underestimating the magnitude of HTG and misclassifying etiology [24]. Clinical suspicion should be particularly high in patients with poorly controlled diabetes, obesity, metabolic syndrome, pregnancy, or familial lipid disorders. During the acute episode, standard supportive care remains the cornerstone, but in severe or refractory HTG-AP, therapies such as insulin infusions (to enhance lipoprotein lipase activity) and, in select cases, plasmapheresis may be considered to accelerate triglyceride clearance; downstream strategies include fibrate therapy, high-dose omega-3 fatty acids, optimization of glycemic control, weight reduction, and alcohol avoidance. Prognostically, HTG as the primary driver of AP is associated with more severe systemic inflammation and higher complication rates than secondary or mixed causes, reaffirming the importance of early identification and tailored interventions [20–24]. Preventive cardiometabolic care after discharge—integrating dietary counseling, pharmacotherapy, and monitoring—can meaningfully reduce recurrence and improve global cardiovascular risk profiles [22], [24].

Drugs

Drug-induced pancreatitis (DIP) is less common than biliary, alcohol, or HTG causes but remains a crucial diagnostic category because recognition enables curative management through withdrawal of the offending agent. Overall, medications account for approximately 5% of AP cases, a figure that likely underestimates the true burden given under-reporting and challenges in establishing causality [25]. A careful medication history—including over-the-counter products and herbal supplements—is therefore indispensable, as temporal associations can be subtle: while some drugs trigger pancreatitis within days, others exert cumulative or idiosyncratic effects that only manifest after weeks to months of exposure [25], [28]. To systematize the evidence base, drugs have been classified into tiers, with Class I agents defined by at least one case report documenting recurrence upon rechallenge, a stringent criterion that increases confidence in causality [26]. Representative Class I drugs span diverse therapeutic classes, including tetracyclines and cotrimoxazole among antibiotics; prednisone, dexamethasone, and estradiol among steroids and hormones; carbamazepine and valproic acid among antiepileptics; lisinopril, losartan, and furosemide among cardiovascular agents; and codeine among opioids [27].

The mechanistic underpinnings of DIP vary and may include hypersensitivity reactions, direct cytotoxicity, metabolic effects such as hypertriglyceridemia, and sphincter of Oddi dysfunction, depending on the agent. Some compounds demonstrate dose-dependent toxicity or

toxic metabolites that disproportionately affect acinar cells or the pancreatic microcirculation, lending biological plausibility to clinical observations [28]. In practice, causality assessment integrates chronology, dechallenge response, exclusion of alternative etiologies, and, when ethically permissible and clinically necessary, cautious rechallenge in controlled settings. Because many DIP cases occur in patients with complex comorbidities receiving polypharmacy, collaboration with pharmacists to identify candidate agents and safer alternatives is essential. When a likely agent is identified, discontinuation should be prompt, with clear documentation to prevent inadvertent re-exposure. Patient education at discharge should emphasize avoiding the culprit drug and recognizing early warning symptoms of recurrence. At a population level, pharmacovigilance programs and high-quality case adjudication continue to refine the causal list, improve risk stratification, and inform prescribing practices [25–27]. As precision therapeutics proliferate, maintaining vigilance for DIP remains a key component of modern pancreatology [26], [28].

Post-ERCP Pancreatitis

Post-ERCP pancreatitis (PEP) is the most common serious adverse event of ERCP and a leading iatrogenic cause of AP. While ERCP is indispensable for biliary and pancreatic ductal therapy, it carries an inherent pancreatitis risk that, in contemporary literature, contributes to a meaningful fraction of all AP presentations [29]. A comprehensive systematic review spanning more than 100 randomized clinical trials estimated that approximately 9% of AP episodes in pooled analyses were PEP, with rates rising to as high as 14% among high-risk subgroups, emphasizing the need for risk assessment and prophylactic strategies [29]. Procedural factors that elevate risk include multiple efforts at biliary cannulation, unintended or repeated pancreatic duct cannulation, difficult anatomy necessitating prolonged manipulation, and sphincterotomy in certain contexts. Patient-related risk is particularly pronounced in younger women, those with suspected sphincter of Oddi dysfunction, and individuals with small bile ducts, each of which is associated with higher PEP incidence, possibly through heightened papillary sensitivity and altered sphincter dynamics [30], [31].

Mitigation strategies span pre-, intra-, and post-procedural domains. Pre-procedurally, accurate selection of candidates and use of noninvasive alternatives (MRCP, EUS) to answer purely diagnostic questions can reduce unnecessary ERCPs; for therapeutic indications, a plan that minimizes pancreatic duct instrumentation is preferred [29]. Intra-procedurally, wire-guided cannulation techniques, early adoption of alternative access (e.g., precut) by experienced endoscopists when cannulation is difficult, and placement of prophylactic pancreatic duct stents in high-risk patients have all been associated with lower PEP rates. Pharmacologic

prophylaxis with rectal nonsteroidal anti-inflammatory drugs is widely used as an evidence-based, low-cost intervention to reduce PEP across risk strata. Post-procedurally, early recognition of abdominal pain and biochemical changes consistent with pancreatitis should prompt aggressive hydration and standard AP care pathways. The decision to proceed with ERCP in any given patient must balance the therapeutic imperative—such as urgent biliary decompression in cholangitis—against the procedural risk profile, with informed consent that explicitly addresses PEP. Institutional quality programs that track PEP rates and promote adherence to best practices can meaningfully diminish iatrogenic AP burden over time [29–31].

Other Causes of Acute Pancreatitis

Beyond the major etiologies, a spectrum of less common causes can precipitate AP and should enter the differential diagnosis when the initial evaluation is unrevealing or when specific clinical clues are present. Traumatic pancreatic injury—whether blunt abdominal trauma from motor vehicle collisions or iatrogenic injury during surgery—can disrupt the pancreatic duct or parenchyma and lead to AP; cross-sectional imaging and, in select cases, pancreatography aid in delineating the extent of damage [32]. Hypercalcemia, often in the context of hyperparathyroidism or malignancy, is a recognized metabolic trigger that facilitates intraductal stone formation and acinar injury; identifying and correcting the calcium disorder is essential to prevent recurrence [32]. Viral infections, including coxsackie B virus, cytomegalovirus, mumps, and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), have all been implicated in AP, likely through direct cytopathic effects and immune-mediated inflammation; in such cases, supportive pancreatitis care must be integrated with pathogen-specific therapies or antiviral stewardship as appropriate to disease severity and host factors [32]. Cardiac bypass surgery has been associated with postoperative AP, with ischemia-reperfusion injury, hypoperfusion, and microembolization proposed as mechanistic drivers; importantly, these cases often abate as overall cardiac function and perfusion improve, reinforcing the primacy of hemodynamic optimization in management [33].

Environmental and envenomation-related causes, though rare in many regions, should be considered where epidemiologically relevant. Scorpion envenomation, for instance, can trigger autonomic storms that perturb pancreatic secretion and ductal motility, culminating in AP; when suspected, notification of local poison control authorities and consideration of antivenom may be appropriate, albeit high-quality trial data remain limited and supportive care is the mainstay [32].

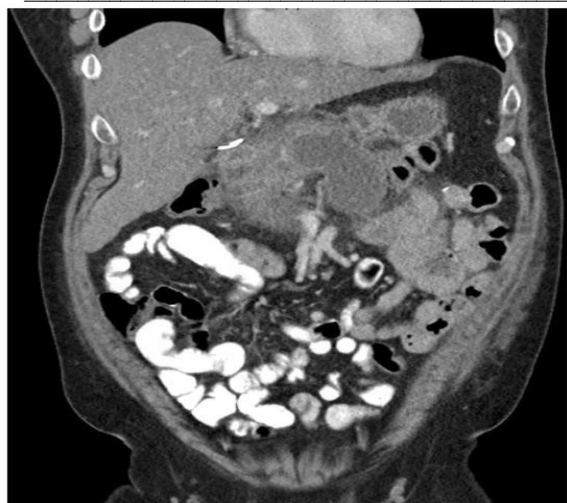


Fig. 3: CT Showing acute pancreatitis.

Autoimmune pancreatitis and anatomic variants such as pancreas divisum occupy an etiologic intersection between structural and immune-mediated disease; while not enumerated among the major categories above, their recognition is vital because treatment (e.g., corticosteroids for autoimmune pancreatitis) can be dramatically effective when correctly applied, and because ductal stenting or minor papilla therapy may alleviate obstruction in select anatomical scenarios. Similarly, obesity functions both as a risk modifier for other etiologies (e.g., HTG) and as a disease severity amplifier due to proinflammatory adipokine milieu; its presence should prompt aggressive risk factor modification after recovery. Across these “other” causes, the shared clinical principle is the same: careful attention to context, targeted testing to confirm the suspected etiology, and etiologic therapy alongside best-practice supportive care to reduce recurrence and long-term sequelae [32], [33].

In sum, etiologic determination in AP is neither academic nor optional; it is a practical imperative that shapes acute decision-making, informs definitive interventions, and drives prevention. Gallstones predominate globally and demand early biliary imaging and, when indicated, urgent ERCP for cholangitis followed by timely cholecystectomy to forestall recurrence [8], [9], [11]. Alcohol-related disease underscores the need to assess both cumulative exposure and binge patterns, to address the synergistic harm of smoking, and to embed addiction and cessation support into post-discharge care [12–19]. Hypertriglyceridemia highlights the importance of an admission lipid panel—before dilution by fluids—to avoid missed diagnoses and to trigger triglyceride-lowering strategies that prevent severe recurrences [20–24]. Drug-induced pancreatitis requires exacting medication reconciliation, alertness to delayed presentations, and decisive dechallenge, guided by evolving causal classifications and mechanistic insights [25–28]. Post-ERCP pancreatitis, the leading iatrogenic cause, compels procedural prudence, risk

stratification, and prophylaxis to curtail harm while preserving the life-saving benefits of therapeutic ERCP [29–31]. Finally, less common but clinically meaningful causes—from trauma and hypercalcemia to viral infections, cardiac bypass-related ischemia, and envenomation—must remain on the diagnostic radar, particularly when first-line evaluations are negative, because identifying and addressing these drivers can be the difference between recurrent disease and durable remission [32], [33]. As health systems seek to reduce AP morbidity, mortality, and cost, an etiology-first paradigm—grounded in careful history-taking, context-aware testing, and targeted interventions—offers the most reliable path to improved outcomes across diverse patient populations and care settings.

Pathophysiology

The pathophysiology of acute pancreatitis (AP) represents a complex interplay between premature enzyme activation, inflammatory signaling, and systemic immune dysregulation. Understanding these mechanisms is essential for the development of targeted therapies to mitigate both pancreatic and multi-organ injury [34]. The initiating event—whether triggered by gallstones, alcohol, toxins, or metabolic disturbances—results in direct injury to pancreatic acinar and ductal cells. This cellular insult disrupts the finely regulated intracellular calcium signaling responsible for stimulus–secretion coupling, leading to uncontrolled activation of digestive enzymes within the pancreatic parenchyma [35]. Under physiological conditions, calcium oscillations are transient and tightly regulated, but in AP, sustained elevations of cytosolic calcium overwhelm cellular homeostasis, deplete adenosine triphosphate (ATP) stores, and trigger mitochondrial dysfunction. High toxin exposure or prolonged ischemia amplifies this effect, exacerbating oxidative stress, impairing ATP production, and promoting necrotic rather than apoptotic cell death. At the subcellular level, defective autophagy and abnormal endolysosomal trafficking ensue, leading to the accumulation of damaged organelles and zymogen granules that further fuel intracellular inflammation [34,36]. The inflammasome complex, particularly NLRP3, becomes activated and catalyzes the release of potent pro-inflammatory cytokines such as tumor necrosis factor- α (TNF- α), interleukin (IL)-1 α , IL-1 β , IL-6, and IL-18, amplifying the inflammatory milieu [37]. Microscopically, this manifests as interstitial edema, vascular congestion, and hemorrhage, with extensive leukocyte infiltration and necrosis visible in severe forms. The initial local inflammatory response rapidly escalates into a systemic inflammatory cascade that spreads via lymphatic and vascular channels to distant organs, including the liver, lungs, heart, kidneys, and gastrointestinal (GI) tract, ultimately predisposing to multi-organ dysfunction syndrome (MODS) [38].

One of the earliest systemic manifestations is the systemic inflammatory response syndrome (SIRS), characterized clinically by fever, tachycardia, tachypnea, and leukocytosis. Persistent SIRS reflects a hyperinflammatory state that predicts severe AP and worse outcomes. Involvement of the GI tract contributes to mucosal barrier dysfunction and bacterial translocation, with subsequent seeding of pancreatic necrosis by enteric organisms [39]. The bacterial species identified often correlate with disease severity, with Enterococcidae showing a strong association with severe and necrotizing disease [40]. Obesity further intensifies the inflammatory cascade through excess adipose tissue lipolysis, which releases unsaturated fatty acids that are toxic to acinar cells and perpetuate local and systemic inflammation [41,42]. This lipotoxic injury, combined with cytokine-driven microvascular damage, accelerates pancreatic necrosis and systemic complications. Thus, acute pancreatitis evolves from an initially local enzymatic injury to a whole-body inflammatory disorder, in which early intracellular derangements, immune activation, and metabolic factors converge to determine clinical severity and patient outcomes [34–42].

Severity of Pancreatitis

The assessment of severity in acute pancreatitis (AP) is both prognostic and operational, guiding triage, monitoring intensity, and the timing of interventions. Although an initial prediction of severity is typically attempted at admission using clinical profile, laboratory derangements, and early physiologic trends, the definitive categorization requires temporal evolution of the disease to reveal the trajectory of organ function and the emergence of local complications. The most widely adopted framework is the Revised Atlanta Classification (RAC), which stratifies AP into three categories: mild disease, defined by the absence of local complications and organ failure; moderately severe disease, characterized by transient organ failure lasting less than 48 hours and/or local complications; and severe disease, marked by persistent organ failure beyond 48 hours [4]. In routine practice, this taxonomy clarifies expectations for resource utilization and outcomes: approximately 65–70% of patients experience an uncomplicated, self-limited course with symptom resolution over several days, while 20–25% develop moderately severe disease with local pancreatic injury such as acute peripancreatic fluid collections or necrosis, prolonging hospitalization and necessitating closer surveillance [4,43]. A smaller but clinically crucial subset—about 10%—progress to severe AP, with sustained organ dysfunction, refractory pain, nutritional compromise, and hospital stays exceeding four weeks, frequently requiring intensive care unit support and multidisciplinary intervention.

Complementing RAC, the Determinant-based Classification (DBC) was proposed to foreground two key pathobiologic determinants—organ failure and infected necrosis—and to arrange

severity into four categories: mild (no necrosis or organ failure), moderate (sterile necrosis or transient organ failure <48 hours), severe (infected necrosis or persistent organ failure >48 hours), and critical (infected necrosis with persistent organ failure) [43,44]. This determinant-centric perspective aligns closely with bedside decision-making, because persistent organ failure carries the strongest prognostic weight, while infected necrosis historically compounded risk. Notably, more recent observations indicate that infected pancreatic necrosis may exert a smaller independent influence on mortality than previously believed, potentially reflecting advances in step-up drainage strategies, minimally invasive necrosectomy, and optimized antimicrobial stewardship [45]. Even so, both RAC and DBC underscore the same clinical reality: early physiologic instability and the dynamics of organ support needs are the dominant signals of severe disease, whereas local morphology refines, but does not replace, systemic risk assessment.

A central limitation of all severity schemas is their reliance on data that crystallize over time, often beyond the first 24 hours. This temporal lag can be discordant with the clinical imperative to allocate high-acuity resources preemptively. The pathophysiologic substrate of early deterioration—diffuse endothelial activation with capillary leak—drives intravascular volume depletion, hemoconcentration, and tissue hypoperfusion, priming the cascade to multi-organ failure in susceptible patients [46]. The ability to forecast which patients will develop clinically meaningful capillary leak has direct therapeutic implications, from tailoring the intensity of resuscitation to anticipating respiratory support and renal monitoring. Biomarkers that mirror oncotic reserve and endothelial integrity are therefore attractive as early risk indicators. In this context, serum albumin—long appreciated as a composite signal of nutritional status, hepatic synthetic capacity, and capillary permeability—has shown pragmatic value: lower levels correlate with the subsequent development of multi-organ failure and worse outcomes, offering an easily obtainable, low-cost adjunct to dynamic clinical assessment [47,48]. While albumin is not a mechanistic biomarker per se, its integration into early risk stratification complements physiologic indices and helps bridge the informational gap before organ failure becomes entrenched.

Looking forward, the promise of precision medicine introduces the prospect that germline and somatic genomic signals could refine severity prediction at presentation. Prior literature has suggested associations between specific gene variants and the severity phenotype of pancreatitis, raising the possibility that genetically mediated differences in calcium handling, inflammatory signaling, autophagy, or lipid metabolism modulate the threshold for organ failure or necrosis [49]. At present, these observations remain preliminary and have not yet translated into

routine clinical risk models; nevertheless, they delineate a research frontier in which genomic, transcriptomic, and proteomic signatures might be combined with bedside clinical variables to produce personalized risk trajectories and tailored interventions. Such advances could decisively shorten the window of uncertainty that currently compels clinicians to “wait and see” before final severity classification.

In practical terms, severity assessment should never be a one-time label but a serial process. Early prediction informs where the patient is cared for and how aggressively fluids, analgesia, and nutrition are delivered; repeated reassessment at 24–48 hours captures the evolution toward persistent organ failure, the development of local complications such as necrosis or walled-off collections, and the onset of secondary infections. For patients with moderately severe disease, the presence of sterile necrosis or evolving peripancreatic collections prompts planned imaging follow-up and careful nutritional support, recognizing that enteral nutrition mitigates infectious complications and may forestall deterioration. For those with severe disease, meticulous ICU management focusing on hemodynamics, oxygenation, early recognition of abdominal compartment physiology, and judicious, staged intervention for necrosis is paramount. Across these strata, the synergy of RAC and DBC constructs supports a common bedside objective: identify early who is likely to worsen, deliver the right level of care before irreversible injury accrues, and reserve invasive therapies for the subset in whom nonoperative strategies fail. Despite incremental advances in classification and supportive care, an optimal categorization that perfectly balances immediacy, accuracy, and therapeutic relevance remains elusive, reinforcing the need for biomarkers and models that can front-load prognostic clarity while still aligning with the dynamic biology of acute pancreatitis [4,43–45,46–49].

Acute Pancreatitis in the Elderly, Children, and Pregnancy:

Acute pancreatitis (AP) in the elderly presents distinct epidemiological, etiological, and clinical features compared to younger populations, largely due to age-associated physiological changes, polypharmacy, and the accumulation of comorbidities. The incidence of gallstones increases markedly with age, and biliary pancreatitis consequently represents the predominant cause of AP in this demographic [50,51]. With advancing age, gallbladder motility decreases and bile cholesterol saturation rises, promoting lithogenesis and recurrent biliary obstruction, which are key triggers for pancreatic inflammation. These mechanical and biochemical alterations, coupled with delayed presentation and atypical symptoms, complicate early diagnosis and may delay definitive intervention. Frailty, diminished

physiological reserve, and multiple chronic illnesses—including cardiovascular disease, diabetes, and renal impairment—are critical modifiers of disease trajectory in elderly patients. These comorbidities predispose to poorer hemodynamic stability and reduced tolerance to systemic inflammation, thus increasing the likelihood of multi-organ dysfunction and mortality [52]. Moreover, the pharmacologic complexity associated with polypharmacy introduces the risk of drug-induced pancreatitis, which often remains unrecognized due to overlapping clinical pictures and underreporting. Drugs such as diuretics, corticosteroids, and certain antibiotics are common culprits in this context, necessitating careful medication review upon presentation.

Interestingly, idiopathic pancreatitis remains a frequent diagnosis among older adults, accounting for approximately 30–40% of cases despite advances in high-resolution imaging and endoscopic modalities [53]. This residual category underscores the challenge of identifying subtle etiologies such as microlithiasis, sphincter of Oddi dysfunction, or occult neoplasia. Indeed, malignancy-associated pancreatitis warrants particular vigilance in this group; pancreatic head tumors may cause ductal obstruction and secondary inflammation, rendering pancreatitis an early manifestation of underlying pancreatic cancer [54]. Hence, any idiopathic or recurrent pancreatitis in the elderly should prompt comprehensive imaging, including contrast-enhanced computed tomography (CT), magnetic resonance cholangiopancreatography (MRCP), or endoscopic ultrasound (EUS) to exclude neoplastic obstruction. Autoimmune pancreatitis (AIP) also gains prominence with age and represents an important yet underdiagnosed cause of pancreatitis in elderly populations. Characterized by lymphoplasmacytic infiltration and elevated serum IgG4 levels, AIP may mimic malignancy both clinically and radiologically [55]. Early recognition is essential, as corticosteroid therapy can induce rapid remission and prevent irreversible fibrosis. Collectively, the management of AP in the elderly demands a nuanced, multidisciplinary approach—balancing diagnostic thoroughness with procedural prudence, minimizing iatrogenic risks, and tailoring interventions to the patient’s comorbid and functional profile. Prognosis in this cohort is closely linked to early etiology identification, optimized supportive care, and vigilant monitoring for complications, all of which are pivotal to improving outcomes in this vulnerable and growing patient population [50–55].

Inpatient Management of Acute Pancreatitis

Inpatient care for acute pancreatitis (AP) is anchored in meticulous monitoring, early risk stratification, and prompt institution of supportive therapies that attenuate pancreatic injury and forestall systemic complications. On admission, patients should undergo frequent assessments of respiratory status,

hemodynamics, mental state, and urine output, with nursing documentation synchronized to clinical milestones. The initial laboratory evaluation typically includes serum amylase and lipase to confirm the diagnosis, a lipid panel emphasizing triglycerides to detect hypertriglyceridemia, a complete blood count to gauge hemoconcentration and leukocytosis, and a comprehensive metabolic panel to track renal function and electrolyte disturbances; hemoglobin A1c can provide context for glycemic control and metabolic risk. Transabdominal ultrasound is recommended to evaluate for gallstones and biliary dilation, thereby identifying patients who might benefit from early endoscopic intervention. Foundational therapies—supplemental oxygen, intravenous fluid resuscitation, opioid-sparing analgesia strategies, and early nutrition—should be initiated in parallel, with close attention to evolving severity so that care intensity can be escalated or de-escalated safely [62–64], [71,72].

Oxygen

Respiratory support in AP aims to correct hypoxemia and prevent secondary organ injury while minimizing iatrogenic harm. A target oxygen saturation (SpO₂) of 94–99% is generally appropriate for most patients, recognizing that lower target ranges (88–92%) may be safer for those with chronic obstructive pulmonary disease or morbid obesity due to the risk of oxygen-induced hypercapnia [62]. Documentation should include the delivery interface (nasal cannula, simple face mask, reservoir mask), inspired oxygen fraction or flow rate, and serial saturation data to reveal trajectory rather than isolated values. In cases where the initial saturation is <85%, the immediate administration of high-flow oxygen—such as 1 L/min via a reservoir mask—can acutely raise arterial oxygen content and should be down-titrated as the patient stabilizes and respiratory mechanics improve [62]. Episodes of desaturation warrant arterial blood gas analysis to detect concurrent hypercapnia or metabolic acidosis; findings should guide escalation along a stepwise pathway that includes optimizing patient positioning, encouraging pulmonary hygiene, and adjusting oxygen delivery devices. The interplay between systemic inflammation, capillary leak, and noncardiogenic pulmonary edema in severe AP underscores the need for vigilant respiratory surveillance, as early intervention can mitigate the progression to acute hypoxemic respiratory failure [63].

Intravenous Fluid Resuscitation

Intravenous fluid therapy is a cornerstone of early AP management and should be prioritized within the first 24 hours to blunt the inflammatory cascade and restore effective circulating volume. Adequate resuscitation reduces the incidence of systemic inflammatory response syndrome (SIRS) and organ failure, with recommended infusion rates typically ranging from 5–10 mL/kg/h during the initial phase, titrated to dynamic clinical endpoints rather than fixed volumes [24]. The principal therapeutic objective is to

correct third-space losses and tissue hypoperfusion that arise from endothelial dysfunction and microcirculatory impairment—pathophysiologic hallmarks of AP that exacerbate acinar injury and propagate systemic dysfunction [63]. Practical bedside targets include reducing heart rate to <120 beats/min, maintaining urine output >0.5 mL/kg/h, and improving markers of hemoconcentration, all while avoiding excessive fluid administration that can worsen pulmonary status, precipitate abdominal compartment physiology, and heighten the risk of multi-organ failure [64]. The choice of crystalloid also merits attention. A randomized trial involving 40 patients suggested that lactated Ringer's solution may be preferable to normal saline during early resuscitation, potentially due to its buffered composition and lower propensity to induce hyperchloremic acidosis [65]. These findings were corroborated in a subsequent smaller trial, providing convergent evidence for lactated Ringer's as the initial fluid of choice in many patients [66]. Nonetheless, fluid strategy should evolve with clinical phase and phenotype, aligning with the conceptual ROSE framework—Resuscitation, Optimization, Stabilization, and Evacuation—to balance perfusion needs against the risks of fluid overload [64]. Serial reassessment should integrate clinical examination, bedside ultrasound (when available) to appraise intravascular volume and venous congestion, and laboratory indices that reflect renal function and acid–base status. In severe cases, particularly when capillary leak is profound, the threshold for critical care consultation should be low to facilitate invasive monitoring and timely adjustments in fluid and vasopressor therapy [63,64].

Pain Control

Pain in AP is often severe, contributes to neurohumoral stress responses, and can impede early mobilization and nutrition. Randomized trials support the safety and efficacy of opioids for initial analgesia, particularly for rapid symptom relief and to enable essential care such as respiratory exercises and enteral feeding [67]. With stabilization, an opioid-sparing approach that incorporates scheduled nonsteroidal anti-inflammatory drugs (NSAIDs) may reduce cumulative opioid exposure; however, NSAIDs should be avoided or used cautiously in the setting of renal impairment or hemodynamic instability given their potential nephrotoxicity [68]. Adjunctive measures—such as antiemetics, judicious use of neuropathic agents in select patients, and local heat—can augment analgesia without increasing opioid dose. Notably, early initiation of oral nutrition, when tolerated, is associated with reductions in pain intensity and duration, possibly by dampening intestinal permeability and inflammatory signaling, thereby complementing pharmacologic analgesia [69]. The observed correlation between pain trajectory, disease severity, and total opioid administration highlights the importance of frequent reassessment

and individualized tapering strategies as inflammation subsides [70]. Equally important is the recognition that pain and psychological distress are interdependent. Anxiety and catastrophizing can amplify pain perception, prolong hospitalization, and complicate recovery. Integrating psychological support—through reassurance, expectation setting, and, when needed, formal counseling—can improve patient experience and engagement with care. Nursing interventions that normalize sleep–wake cycles, promote mobility, and maintain therapeutic alliance contribute meaningfully to pain control and overall outcomes. A proactive, multimodal analgesia plan, tailored to renal function and disease trajectory, remains central to patient-centered AP care [67–70].

Nutrition

AP induces a hypermetabolic and catabolic state characterized by increased energy expenditure, insulin resistance, accelerated proteolysis, and lipolysis; inadequate nutrition can aggravate these derangements and predispose to infectious complications [71]. Contemporary practice favors early refeeding: if the patient can tolerate oral intake without exacerbation of pain or emesis, clear liquids can be advanced to a low-fat diet as soon as feasible, often within the first 24–48 hours [72]. When oral intake is not possible, enteral nutrition via nasogastric or nasojejunal tube should be initiated within 48 hours of admission to maintain gut integrity, support immune function, and reduce inflammatory signaling [72,73]. The salutary effects of enteral feeding are mechanistically linked to preservation of the gastrointestinal barrier, reduced bacterial translocation, and attenuation of the pro-inflammatory cascade, particularly important in preventing infected pancreatic necrosis and septic complications [73]. The choice between nasogastric and nasojejunal routes can be individualized; both are generally effective, and technical ease often favors nasogastric placement. Continuous rather than bolus feeding may enhance tolerance early on, with careful monitoring for gastric residuals, nausea, or abdominal distension. In patients with inadequate caloric delivery despite optimized enteral strategies, a supplemental parenteral component can be considered; however, current trials and meta-analyses do not demonstrate superiority of combined enteral–parenteral regimens over enteral feeding alone with respect to hard outcomes, and parenteral nutrition carries risks of catheter-related infections and metabolic complications [74]. Micronutrient optimization, including thiamine in patients with alcohol-related disease and careful electrolyte replacement during refeeding, should be integrated into the nutrition plan. Glycemic control is also critical, as stress hyperglycemia aggravates pancreatic and systemic inflammation; insulin therapy should be titrated to institutional targets while avoiding hypoglycemia [71,72]. Across all four domains—oxygenation, fluids, analgesia, and

nutrition—successful inpatient management of AP relies on coordinated, iterative care. Early, protocolized interventions provide a foundation, but responsiveness to the patient’s evolving physiology is paramount. Respiratory instability, rising creatinine, or escalating pain demands rapid recalibration of the plan; conversely, clinical improvement should prompt thoughtful de-escalation to prevent iatrogenic harm. Embedding these practices within multidisciplinary pathways that include gastroenterology, surgery, critical care, nursing, pharmacy, nutrition, and, when appropriate, addiction services and social work, maximizes the likelihood of an uncomplicated course and durable recovery. By aligning bedside actions with the evidence supporting oxygen targets, targeted fluid strategies, multimodal analgesia, and early enteral feeding, clinicians can meaningfully reduce the risk of organ failure, shorten hospital stays, and improve patient-centered outcomes in acute pancreatitis [62–66], [69–74].

Roles of healthcare professionals:

The management of acute pancreatitis (AP) is inherently multidisciplinary, requiring coordinated contributions from radiologists, laboratory professionals, and nurses to ensure timely diagnosis, appropriate monitoring, and effective supportive care. Each plays a pivotal role in improving clinical outcomes and preventing complications. Radiologists are central to both diagnosis and disease staging. They perform and interpret imaging studies such as transabdominal ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) to confirm pancreatic inflammation, identify gallstones, and assess for local complications such as necrosis, pseudocysts, or abscess formation. In critically ill patients, radiologists also guide minimally invasive interventions, including percutaneous drainage of infected collections or fluid aspiration for microbiological evaluation. Through accurate interpretation and judicious use of imaging modalities, radiologists minimize unnecessary radiation exposure while ensuring diagnostic precision and procedural safety. Laboratory professionals provide essential biochemical and hematologic data that underpin the diagnosis and monitoring of AP. Measurements of serum amylase and lipase confirm pancreatic injury, while metabolic panels reveal renal, hepatic, and electrolyte disturbances that reflect disease severity. Triglyceride assays identify hypertriglyceridemia-induced pancreatitis, and inflammatory markers such as C-reactive protein (CRP) assist in prognostication. Laboratory teams also perform cultures from aspirated collections or blood samples to detect secondary infections, thereby influencing antimicrobial therapy. Their role in ensuring the accuracy, timeliness, and reliability of results is crucial for informed clinical decision-making. Nurses form the cornerstone of continuous patient care. They monitor vital signs, oxygen saturation, and fluid balance; administer

analgesia, fluids, and nutrition; and identify early signs of deterioration or complications. Beyond physical care, nurses provide psychological support, patient education, and coordination among the multidisciplinary team. Their vigilance and holistic care approach are instrumental in preventing organ failure, ensuring patient comfort, and fostering recovery. Collectively, radiologists, laboratory professionals, and nurses form an integrated triad that translates clinical, diagnostic, and supportive strategies into cohesive, evidence-based care for patients with acute pancreatitis.

Conclusion:

Acute pancreatitis demands early, coordinated decisions that weave imaging, nursing operations, and laboratory data into a coherent plan. CT Severity Index adds granular anatomic context to clinical classifications, informing the timing of interventions for collections and necrosis while avoiding unnecessary radiation or contrast exposure when alternatives suffice. Nurse-led fluid-resuscitation algorithms operationalize best practice—prioritizing buffered crystalloids, dynamic hemodynamic and urine output targets, and vigilant avoidance of fluid overload—thereby reducing SIRS, organ failure, and downstream ICU utilization. Laboratory stewardship anchors this pathway: lipase remains the diagnostic gatekeeper, whereas CRP serial trends help flag patients likely to develop severe disease, particularly when interpreted alongside evolving physiology and imaging. When this triad is embedded within multidisciplinary pathways that also attend to early enteral nutrition, analgesia, and etiologic control (e.g., biliary decompression, triglyceride lowering, medication withdrawal), hospitals can shorten stays and improve patient-centered outcomes. Future advances will refine this framework with biomarker panels and precision-risk tools, but current evidence already supports a pragmatic standard: use CTSI to stage local disease, empower nurses with protocolized fluids and monitoring, and leverage lipase/CRP appropriately—confirm to diagnose, trend CRP to anticipate severity. Such integration converts complex pathobiology into reliable bedside actions and delivers safer, more efficient care for patients with acute pancreatitis.

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