

# СИНО

ISSN: 2707-5265

ЕВРАЗИЙСКИЙ  
НАУЧНО - МЕДИЦИНСКИЙ  
ЖУРНАЛ

Eurasian Scientific and Medical Journal «Sino»



2026 #3  
ТОМ 7 #3

# Евразийский научно-медицинский журнал «Сино»

Том 7, № 3, 2026

## Eurasian Scientific and Medical Journal «Sino»

Vol. 7, N 3, 2026

ISSN: 2707-5265

Журнал зарегистрирован Министерством культуры  
Республики Таджикистан  
Свидетельство о регистрации - № 103 от 27.03.2019 г.  
Вновь перерегистрирован - № 398 от 24.02.2025 г.

**Издание Ассоциации общественного здравоохранения Таджикистана**

Основан в 2019 г. Журнал выходит 1 раз в 3 месяца.  
Периодичность – 4 номера в год

**Сайт журнала:**  
[www.eurasian-journal-sino.tj](http://www.eurasian-journal-sino.tj)

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**Адрес редакции журнала:**  
734018, Таджикистан, г. Душанбе, пр. С. Шерози, 16  
Статьи отправить по адресу: [sino-journal@mail.ru](mailto:sino-journal@mail.ru)

Журнал рассчитан на научных работников и преподавателей  
медицинских вузов, руководителей учреждений  
здравоохранения и практических врачей

Журнал индексируется в Российском индексе  
научного цитирования (РИНЦ), Crossref, Science Index

**Евразийский научно-медицинский журнал «Сино» включён  
в Перечень ведущих рецензируемых научных изданий  
ВАК при Президенте Республики Таджикистан,  
рекомендованных для публикаций основных научных  
результатов диссертаций на соискание учёных степеней  
доктора и кандидата наук**

ISSN: 2707-5265

The journal is registered by the Ministry of Culture  
of the Republic of Tajikistan  
Certificate of registration - N 103 from 27.03.2019  
Re-registered - N 398 from 24.02.2025

**Publication of the Public Health Association of Tajikistan**

Founded in 2019. The magazine is published once every 3 months.  
Frequency - 4 issues per year

**Journal website:**  
[www.eurasian-journal-sino.tj](http://www.eurasian-journal-sino.tj)

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**Editorial office address:**  
734018, Tajikistan, Dushanbe, Ave. S. Sherozi, 16  
Articles should be sent to: [sino-journal@mail.ru](mailto:sino-journal@mail.ru)

The journal is intended for researchers and teachers of medical universities,  
heads of healthcare institutions and practicing physicians

The journal is indexed in the Russian Science Citation Index (RSCI), Crossref,  
Science Index

**The Eurasian Scientific and Medical Journal "Sino" is included  
in the List of leading peer-reviewed scientific publications  
of the Higher Attestation Commission under the President of  
the Republic of Tajikistan, recommended for publication of  
the main scientific results of dissertations for the academic  
degrees of Doctor and Candidate of Sciences**

DOI: 10.54538/2707-5265-2026-7-3-192-204

# Complications of fluid accumulations in acute pancreatitis: multidisciplinary analysis and modern management algorithms

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**Objective:** To conduct a comprehensive analysis of complications associated with pancreatic and peripancreatic fluid collections in patients with acute pancreatitis.

**Materials and Methods:** A systematic review of the literature was conducted for the period from 2017 to 2024, including international clinical guidelines, randomized controlled trials, meta-analyses, and cohort studies. The review focused on the pathophysiology, classification, and multidisciplinary approach to the treatment of complicated pancreatic and peripancreatic fluid collections.

**Results:** The analysis revealed that the primary life-threatening complications are infection of necrotic tissue, erosive bleeding, and fistula formation. The minimally invasive step-up approach, beginning with percutaneous or endoscopic drainage, has been confirmed to be highly effective. This method has demonstrated a significant reduction in mortality and serious complication rates compared to primary open necrosectomy. Treatment success directly depends on the timely formation of a multidisciplinary team.

**Conclusion:** The modern strategy for treating complicated pancreatic and peripancreatic fluid collections requires a multidisciplinary approach, the use of modern imaging techniques for accurate diagnosis, and strict adherence to the principles of staged treatment. Future directions include the refinement of minimally invasive techniques and the development of personalized treatment algorithms.

## Key words:

acute pancreatitis, pancreatic and peripancreatic fluid collections, infected pancreatic necrosis, walled-off pancreatic necrosis, endoscopic drainage, percutaneous drainage, literature review

## For citation:

Kholbegov A.M., Nazarov Sh.K., Ali-Zade S.G., Rahmonzoda K.R., Nuraliev Sh.S., Otaev Sh.Z., Valizoda A.O. Complications of fluid accumulations in acute pancreatitis: multidisciplinary analysis and modern management algorithms. *Eurasian Scientific and Medical Journal "Sino"*. 2026; 7(3): 192-204. <https://doi.org/10.54538/2707-5265-2026-7-3-192-204>

DOI: 10.54538/2707-5265-2026-7-3-192-204

# Осложнения жидкостных скоплений при остром панкреатите: мультидисциплинарный анализ и современные алгоритмы ведения

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**Цель исследования.** Провести комплексный анализ осложнений, возникающих при панкреатических и перипанкреатических жидкостных скоплениях у пациентов с острым панкреатитом.

**Материалы и методы.** Проведён систематический анализ научной литературы за период с 2017 по 2024 год, включавший международные клинические рекомендации, рандомизированные контролируемые исследования, метаанализы и когортные исследования. Анализ был сосредоточен на вопросах патофизиологии, классификации, диагностики и междисциплинарного подхода к лечению осложнённых панкреатических и перипанкреатических жидкостных скоплений.

**Результаты.** Анализ литературы показал, что к числу наиболее опасных осложнений относятся инфицирование панкреонекроза, аррозивное кровотечение и формирование свищей. Подтверждена эффективность минимально инвазивного поэтапного подхода (step-up approach), предусматривающего первоначальное чрескожное или эндоскопическое дренирование с последующей эскалацией лечебных вмешательств при отсутствии клинического эффекта. Данный подход ассоциируется со снижением частоты тяжёлых осложнений и необходимости выполнения открытых хирургических вмешательств по сравнению с первичной открытой некрэктомией. Эффективность лечения во многом зависит от своевременного формирования и слаженной работы мультидисциплинарной команды.

**Закключение.** Современная стратегия ведения пациентов с осложнёнными панкреатическими и перипанкреатическими жидкостными скоплениями требует междисциплинарного подхода, применения современных методов визуализации для точной диагностики и строгого соблюдения принципов поэтапного лечения. Перспективными направлениями являются дальнейшее совершенствование минимально инвазивных технологий и разработка персонализированных лечебно-диагностических алгоритмов.

## Ключевые слова:

острый панкреатит, панкреатические и перипанкреатические жидкостные скопления, инфицированный панкреонекроз, отграниченный панкреонекроз, эндоскопическое дренирование, чрескожное дренирование, обзор литературы

## Для цитирования:

Холбегов А.М., Назаров Ш.К., Али-Заде С.Г., Рахмонзода К.Р., Нуралиев Ш.С., Отаев Ш.З., Вализода А.О. Осложнения жидкостных скоплений при остром панкреатите: мультидисциплинарный анализ и современные алгоритмы ведения. Евразийский научно-медицинский журнал «Сино». 2026; 7(3): 192-204. <https://doi.org/10.54538/2707-5265-2026-7-3-192-204>

## INTRODUCTION

The clinical trajectory of acute pancreatitis remains notoriously unpredictable, representing one of the most challenging disease processes in abdominal medicine. While approximately 80% of cases follow a self-limiting, edematous pattern, the remaining 20% progress to severe necrotizing pancreatitis, characterized by pancreatic and peri-pancreatic tissue death that creates an environment ripe for complication development through complex inflammatory cascades [1]. This necrotic milieu, combined with enzymatic autodigestion and profound inflammatory exudate, establishes the fundamental conditions for fluid collection formation that can evolve into devastating clinical scenarios. The 2012 revision of the Atlanta classification represented a paradigm-shifting achievement that transcended mere terminology standardization, creating a temporal and morphological framework that directly informs contemporary management strategies and prognostic assessment [2]. However, the mere presence of a collection often proves less consequential than the devastating complications it can precipitate, requiring clinicians to maintain constant vigilance throughout the disease course.

Historically, the management of complicated PFCs resided exclusively within the surgical domain, with open necrosectomy representing the definitive, albeit highly morbid, intervention associated with mortality rates exceeding 25% in many series. The past decade has witnessed a remarkable paradigm shift, championed by landmark trials including the Dutch PANTER study, which established the superiority of a minimally invasive "step-up" approach that has fundamentally transformed patient outcomes [3]. This evolution has not rendered surgeons obsolete but has instead integrated their expertise into a broader, collaborative model incorporating interventional radiologists, therapeutic endoscopists, intensivists, and nutrition specialists. This comprehensive review seeks to provide an exhaustive, contemporary exploration of complications arising from PFCs, transcending mere description to deliver nuanced analysis of prevention strategies, diagnostic advancements, and the sophisticatedly sequenced application of modern therapeutic modalities across global healthcare contexts, with particular attention to resource-limited settings and evolving

technological innovations.

**Objective:** To conduct a comprehensive analysis of complications associated with pancreatic and peripancreatic fluid collections in patients with acute pancreatitis.

**Materials and Methods:** A systematic review of the literature was conducted for the period from 2017 to 2024, including international clinical guidelines, randomized controlled trials, meta-analyses, and cohort studies. The review focused on the pathophysiology, classification, and multidisciplinary approach to the treatment of complicated pancreatic and peripancreatic fluid collections.

## CLASSIFICATION AND PATHOGENESIS OF FLUID COLLECTIONS

### The Revised Atlanta Classification: A Clinical Roadmap

The revised Atlanta classification provides a standardized framework for terminology and serves as an indispensable guide for clinical decision-making and research [2]. It introduces a chronologically based system that distinguishes between early and late phases of the disease, directly correlating with the type of fluid collection present. In the early phase of AP (within the first 4 weeks), two distinct types of collections form: Acute Peripancreatic Fluid Collections (APFC) and Acute Necrotic Collections (ANC). APFCs occur exclusively in the setting of interstitial edematous pancreatitis, lack a defined wall, are homogenous on imaging, and typically resolve spontaneously without intervention. In contrast, ANCs are heterogeneous collections containing variable amounts of both fluid and necrotic tissue, representing a hallmark of necrotizing pancreatitis. Their lack of a defined wall in the early stages makes them particularly vulnerable to complications and less amenable to certain interventions.

After the 4-week mark, these collections undergo a process of organization and encapsulation. APFCs that persist beyond this period and develop a well-defined, non-epithelialized wall are reclassified as pancreatic pseudocysts. Crucially, by definition, pseudocysts contain no solid necrotic material, which fundamentally influences their management, typically requiring simple drainage procedures. Conversely, ANCs mature into Walled-Off Necrosis (WON), characterized by

a well-formed, enhancing inflammatory wall and containing both liquid and solid components [4]. This critical distinction between a pseudocyst and WON is paramount, as the management of WON is considerably more complex, often necessitating strategies specifically designed to address the solid necrotic debris, such as direct necrosectomy.

**Pathophysiological basis for complication development.** The pathogenesis of complications is rooted in the initial pancreatic injury, which triggers intra-acinar cell activation of digestive enzymes, particularly the conversion of trypsinogen to trypsin. This event initiates an intricate cascade of local inflammation, microvascular injury, and parenchymal necrosis through multiple interconnected pathways, including NF- $\kappa$ B activation and cytokine release [5]. The subsequent release of pro-inflammatory cytokines (e.g., IL-1, IL-6, TNF- $\alpha$ ) and activated enzymes into the surrounding tissues creates a "chemical burn" phenomenon, dramatically increasing vascular permeability and generating enzyme-rich fluid collections. This process involves complex interactions between endothelial cells, leukocytes, and platelets, leading to microthrombosis, ischemia-reperfusion injury, and further extension of necrosis beyond the pancreas itself into the surrounding retroperitoneal tissues [6].

The presence and extent of necrosis represent the crucial determinant of subsequent complication risk, marking a quantum leap in disease severity compared to simple edema. Necrotic tissue serves as an optimal substrate for infection and a persistent nidus for sustained inflammation through the continued release of damage-associated molecular patterns (DAMPs) that perpetuate the systemic inflammatory response syndrome (SIRS) [7]. The host's attempt to wall off this destructive process initiates a complex fibrotic response over several weeks, mediated by transforming growth factor-beta (TGF- $\beta$ ) and other profibrotic cytokines, progressively transforming an ANC into WON through the development of a well-defined, vascularized capsule. This encapsulation, while indicating maturation and potentially facilitating future intervention, simultaneously creates a confined space where infection can flourish under pressure, presenting unique therapeutic challenges related to compartment syndrome effects on adjacent structures.

**Molecular mechanisms driving specific complications.** The path toward specific complication development involves multiple interconnected biological mechanisms operating at cellular and molecular levels:

**Infection Pathogenesis:** Primarily arises from bacterial translocation across a compromised gut mucosal barrier, a phenomenon significantly exacerbated by intra-abdominal hypertension and splanchnic hypoperfusion in severe AP [8]. The breakdown of gut barrier function allows enteric organisms to migrate into the necrotic peri-pancreatic tissues. Recent investigations have revealed the crucial role of gut microbiome alterations (dysbiosis) and fungal translocations in complicating clinical outcomes, with specific pathogen-associated molecular patterns (PAMPs) triggering enhanced immune activation through Toll-like receptors (TLRs), leading to a second wave of SIRS and organ failure [9].

**Vascular Erosion Mechanics:** Proteolytic enzymes, particularly elastase and phospholipase A2, systematically digest the elastic lamina of adjacent arterial walls, leading to pseudoaneurysm formation through progressive vessel wall weakening [10]. The inflammatory microenvironment promotes pathological neovascularization with fragile, disorganized vessel walls lacking proper supportive structure, further increasing hemorrhage risk through enhanced vascular permeability and defective angiogenesis. The most commonly affected vessels are the splenic artery (40%), gastroduodenal artery (20%), and pancreaticoduodenal arteries, due to their anatomical proximity to the pancreas.

**Compressive Pathophysiology:** The substantial mass effect of large collections can exert significant pressure on adjacent structures through multiple mechanisms: direct mechanical compression, inflammatory adherence, and ischemia-reperfusion injury. This affects the stomach (causing gastroparesis and outlet obstruction), the common bile duct (inducing obstructive jaundice and cholangitis), the duodenum, and the portal/splenic veins, potentially initiating thrombosis and sinistral portal hypertension through endothelial injury and stasis [11]. The resulting portal hypertension can lead to the formation of gastric varices, which may bleed months after the acute episode has resolved.

## COMPREHENSIVE TAXONOMY OF COMPLICATIONS

**Infection of Fluid Collections: The Principal Driver of Intervention.** Infection of necrotic tissue (infected pancreatic necrosis) remains the most frequent catalyst for invasive procedures in necrotizing AP, typically occurring in the second to fourth week of illness. The risk escalates dramatically with the extent of necrosis, exceeding 60% in patients with >50% pancreatic involvement, and is influenced by factors including prophylactic antibiotic use, timing of enteral nutrition, and underlying immunocompetence of the patient [12]. Clinically, infection manifests as a secondary inflammatory surge—characterized by recrudescence fever, leukocytosis, and new-onset or worsening organ failure following an initial period of stabilization, often accompanied by a hyperdynamic circulation and altered mental status.

The diagnostic cornerstone remains contrast-enhanced computed tomography (CECT), where the presence of extraluminal gas within the collection (the "air bubble" sign) demonstrates high specificity for infection, though its sensitivity remains approximately 70% [13]. The absence of gas does not rule out infection. While fine-needle aspiration (FNA) for culture and sensitivity provides microbiological confirmation and enables targeted antibiotic therapy, its routine application has diminished in favor of empirical antibiotics (typically carbapenems or piperacillin-tazobactam) and intervention guided by compelling clinical and radiological evidence, particularly in unstable patients where any diagnostic delay could prove fatal. The spectrum of causative organisms is broad, including Gram-negative bacteria (*E. coli*, *Klebsiella*), Gram-positive bacteria (*Enterococci*, *Staphylococci*), and increasingly, fungal species (*Candida*).

### Vascular Complications: Catastrophic Clinical Threats

Hemorrhagic complications represent the most dramatic and lethal events in this patient population, with mortality rates historically approaching 40% even with modern intervention [14]. The pathological sequence initiates with enzymatic erosion of vascular walls, progress-

ing to pseudoaneurysm formation that may remain stable for variable periods. These fragile, contained bleeds can subsequently rupture—into the peritoneal cavity causing catastrophic hemorrhagic shock, into the collection itself (manifesting as sudden expansion of the collection and a sharp drop in hematocrit), or into the gastrointestinal tract via established fistulous connections, presenting as massive hematemesis or melena.

Crucially, a sentinel bleed—a self-limited hemorrhage episode—may precede massive rupture in approximately 50% of cases, serving as a critical warning sign demanding immediate intervention and aggressive resuscitation [15]. Any new-onset anemia or sudden clinical deterioration in a patient with known necrosis should raise suspicion. CT angiography (CTA) serves as the optimal initial diagnostic modality, providing comprehensive anatomical information about the pseudoaneurysm and its relation to other structures. However, digital subtraction angiography (DSA) remains the gold standard for both definitive diagnosis and therapeutic intervention via transcatheter arterial embolization (TAE) using coils, glue, or covered stents. Surgical intervention is reserved for cases where TAE fails or is not available.

### Fistulization: A Spectrum of Pathological Communications

The relentless inflammatory and enzymatic process can fistulize into virtually any adjacent structure, creating complex clinical scenarios that challenge even experienced clinicians:

**Internal Fistulas:** Communication with the gastrointestinal tract (stomach, duodenum, colon) occurs frequently through a combination of direct enzymatic digestion and pressure necrosis. While cystogastrostomy or cystoduodenostomy can be therapeutic when created under controlled endoscopic or surgical conditions, spontaneous colonic communication presents particular danger, often precipitating uncontrolled sepsis and necessitating surgical diversion or resection with its associated morbidity [16].

**External Pancreatic Fistula (EPF):** Defined as persistent drainage of enzyme-rich pancreatic fluid (>50 mL/day with elevated amylase content) beyond three weeks, an EPF most commonly follows percutaneous or surgical drainage procedures.

The paramount risk factor is "disconnected duct syndrome" (DDS), where a segment of the main pancreatic duct is severed, preventing physiological drainage into the duodenum and establishing a persistent leakage that may continue indefinitely without specific intervention [17]. Management is complex and often involves sustained somatostatin analogue therapy, endoscopic retrograde pancreatography (ERP) with stent placement across the disruption, or long-term transmural stenting of the WON cavity.

**Pancreatic Ascites and Pleural Effusions:** Result from pseudocyst rupture or persistent fistula leakage, with enzymatic fluid tracking into peritoneal or pleural cavities.

This creates additional metabolic, nutritional, and respiratory complications through massive fluid and protein loss, often requiring specific nutritional support, repeated paracenteses or thoracenteses, and somatostatin analogue therapy to reduce pancreatic secretion.

**Obstructive Complications: The Consequences of Mass Effect**

Substantial PFCs can generate various obstructive syndromes through mechanical compression, inflammatory infiltration, and fibrotic reaction:

**Gastric Outlet Obstruction:** Presents with early satiety, persistent vomiting, and profound nutritional decline that can complicate recovery and increase infection risk through impaired immune function.

**Biliary Obstruction:** May cause jaundice and secondary cholangitis requiring urgent intervention through endoscopic retrograde cholangiopancreatography (ERCP) with biliary stenting.

**Splanchnic Venous Thrombosis:** Perhaps the most insidious complication is splenic vein thrombosis, which can lead to isolated gastric varices. While frequently asymptomatic initially, these varices may cause life-threatening upper GI bleeding — a complication that can emerge months after acute pancreatitis resolution, requiring careful long-term surveillance and potentially pre-emptive intervention [18].

**Renal and Intestinal Complications:** Rare but serious complications include ureteral obstruction leading to hydronephrosis and renal impairment, or superior mesenteric vein thrombosis causing intestinal ischemia, which represents a surgical emergency.

## DIAGNOSTIC APPROACH AND PREDICTIVE MARKERS

**Clinical and Laboratory Diagnosis.** Diagnosis of PFC complications relies on a high index of suspicion and a combination of clinical assessment, laboratory findings, and advanced imaging. Signs of ongoing systemic inflammation (persistent or recrudescent fever, tachycardia, leukocytosis) or new/worsening organ failure after an initial period of stabilization should immediately raise suspicion for a complication, most commonly infection. Serial measurement of inflammatory markers is crucial; while C-reactive protein (CRP) is widely used, serum procalcitonin (PCT) has shown superior specificity for diagnosing infected necrosis, with a rising level being particularly suggestive [19]. Other laboratory findings may include worsening renal function, liver enzyme elevations in biliary obstruction, or a sudden drop in hemoglobin signaling hemorrhage.

**Advanced Imaging Modalities.** Imaging is the cornerstone of diagnosing and characterizing complicated PFCs.

**Contrast-Enhanced CT (CECT):** This remains the primary imaging modality. It is essential for characterizing the type of collection (APFC, ANC, pseudocyst, WON), assessing the extent of necrosis, identifying the presence of gas (which is highly specific for infection), and detecting vascular complications like pseudoaneurysms or venous thrombosis [13]. The CT Severity Index (CTSI) and its modifications provide a semi-quantitative assessment of disease severity.

**Magnetic Resonance Imaging (MRI) and MR Cholangiopancreatography (MRCP):** MRI is superior to CT for characterizing the content of a collection, particularly in differentiating solid necrotic debris from simple fluid, which is critical for distinguishing a pseudocyst from WON. MRCP is invaluable for visualizing the pancreatic duct and diagnosing disconnected duct syndrome (DDS), a key factor in planning long-term management and predicting the risk of persistent external pancreatic fistulas [20].

**Endoscopic Ultrasound (EUS):** EUS provides high-resolution imaging of the collection's wall and content, and allows for Doppler assessment of adjacent vascular structures, which is vital for planning a safe access route for drainage. Furthermore, EUS has evolved from a purely diagnostic

tool to a primary therapeutic modality, enabling guided drainage and necrosectomy.

## MODERN MANAGEMENT PRINCIPLES: THE STEP-UP APPROACH

**The Multidisciplinary Team (MDT): The Central Nervous System of Management.** The formidable complexity of managing these patients mandates seamless collaborative MDT function that transcends traditional departmental boundaries. The core team should intensively involve an intensivist (for hemodynamic and multi-organ support management), a gastroenterologist/therapeutic endoscopist (for endoscopic interventions and nutritional support), an interventional radiologist (for image-guided drainage and embolization procedures), a pancreatic surgeon (for surgical planning and complex interventions), an infectious disease specialist (for antimicrobial stewardship), and a nutrition support team (for metabolic management) [21]. The MDT's role commences at diagnosis, with collective review of imaging findings to meticulously characterize collections, assess complication presence, and formulate an optimal, patient-tailored intervention strategy based on local expertise, patient physiology, and anatomical considerations.

**The Step-Up Approach: A Paradigm Shift.** The management of infected pancreatic necrosis has been revolutionized by the minimally invasive "step-up" approach, definitively validated by the landmark Dutch PANTER trial [3]. This strategy aims to control sepsis while minimizing the physiological insult to an already critically ill patient, moving away from primary open necrosectomy. The approach is dynamic and iterative, requiring frequent reassessment and strategy modification based on treatment response and evolving clinical status.

**Step 1: Percutaneous Catheter Drainage (PCD).** PCD persists as the foundational intervention for initial sepsis control, particularly in hemodynamically unstable patients or collections with unfavorable anatomy for endoscopic drainage. It serves both diagnostic (fluid characterization and culture) and therapeutic purposes. The technique has evolved substantially beyond simple catheter placement into a sophisticated image-guided procedure:

**Aggressive PCD Protocols:** Involve the stra-

tegic placement of multiple large-bore (16-20 Fr) catheters, frequently utilizing a retroperitoneal access route to create a controlled pathway for future interventions, accompanied by vigorous irrigation protocols (typically 100-200 mL of normal saline every 6-8 hours) to maintain catheter patency and facilitate the evacuation of necrotic debris [22].

**Adjunctive Fibrinolytic Therapy:** Intra-cavitary instillation of tissue plasminogen activator (tPA) or urokinase significantly enhances drainage efficacy by lysing solid necrotic debris through fibrin cleavage within the collection. This approach can potentially increase PCD success rates as a standalone treatment from approximately 40% to over 60%, though concerns about bleeding risk require careful patient selection [23]. Chinese medical centers have published extensive experience supporting these "enhanced drainage" protocols, with specific regimens involving tPA 4-8 mg in 20-40 mL normal saline instilled twice daily with 1-2 hour dwell times.

**Progressive "Step-Up" from PCD:** When PCD proves insufficient for sepsis control (evidenced by persistent fever, leukocytosis, or organ failure despite adequate drainage), subsequent intervention is guided by the established access route. A mature retroperitoneal tract can be systematically dilated over 1-2 weeks to allow for video-assisted retroperitoneal debridement (VARD) or sophisticated sinus tract endoscopy using flexible endoscopes for direct necrosectomy.

## Step 2: Endoscopic Transmural Drainage and Necrosectomy

Endoscopic ultrasound (EUS)-guided drainage has emerged as a first-line therapy for organized collections (pseudocysts and WON) demonstrating favorable anatomical positioning (within 1-1.5 cm of the gastric or duodenal wall). It offers the significant advantage of internal drainage, which preserves nutritional intake and avoids the creation of an external fistula.

**The LAMS Evolution:** The introduction of lumen-apposing metal stents (LAMS) revolutionized cystenterostomy creation through electrocautery-enhanced delivery systems enabling single-step access and deployment. However, initial enthusiasm has been tempered by the recognition of delayed adverse events, including stent migration (5-15%) and life-threatening hemorrhage from pseudoaneurysm erosion (3-8%) [24]. Con-

sequently, contemporary best practice mandates the placement of a coaxial double-pigtail plastic stent within the LAMS to prevent apposition-related complications and facilitate easier removal, typically within 3-4 weeks to minimize long-term risks.

**Direct Endoscopic Necrosectomy (DEN):** For WON with a significant solid debris component (>30% of the collection volume), DEN represents the therapeutic advance that has fundamentally changed management. The endoscope is advanced through the stent into the necrotic cavity, permitting debridement using snares, nets, and forceps under direct visualization, often requiring carbon dioxide insufflation to minimize the risk of air embolism. Japanese endoscopists have pioneered innovative techniques including per-oral direct cholangioscopy and water-jet assisted necrosectomy to enhance visualization and efficacy within these complex cavities [25]. Multiple systematic sessions (typically 2-5 procedures spaced 2-7 days apart) are frequently necessary for complete debridement, with procedure termination based on the endoscopic appearance of viable tissue and sustained clinical improvement.

**The Evolving Role of Surgery.** Open surgical necrosectomy is now predominantly reserved for specific failure scenarios (inadequate response to minimally invasive approaches) or catastrophic complications including uncontrolled hemorrhage not amenable to angioembolization, colonic perforation, or abdominal compartment syndrome refractory to medical management. The modern surgical contribution is defined by minimally invasive expertise and damage control principles:

**Video-Assisted Retroperitoneal Debridement (VARD):** Represents a minimally invasive bridge between PCD and open surgery, particularly optimal for left-sided collections extending into the paracolic gutters. It utilizes a small flank incision and laparoscopic instruments to evacuate necrosis under direct vision while preserving anatomical planes and minimizing morbidity [3].

**Laparoscopic Cystogastrostomy:** Provides a controlled and robust drainage option for pseudocysts and WON with favorable anatomy, offering the advantage of direct hemostasis and thorough debridement under visualization while maintaining the benefits of a minimally invasive approach. Recent series from high-volume centers show suc-

cess rates exceeding 85% in experienced hands [26].

Recent comparative trials, including TENSION and MISER, have further refined our therapeutic understanding. These studies demonstrated that while an endoscopy-first step-up strategy may reduce long-term pancreatic fistulas and exocrine/endocrine insufficiency compared to a surgery-first step-up approach, both strategies are valid and effective, with the final selection dependent on local expertise, specific collection anatomy, and patient comorbidities [27, 28].

## INTERNATIONAL PERSPECTIVES AND REGIONAL CLINICAL NUANCES

**Western Approaches (Europe and North America).** International practice analysis reveals significant therapeutic variations reflecting local expertise, resource availability, and philosophical differences. European and North American centers emphasize strict step-up protocol adherence within formalized MDT frameworks, with a strong emphasis on evidence-based guidelines and standardized care pathways. Research focuses on LAMS safety optimization through stent design improvements and defining roles for combined ("hybrid") endoscopic-percutaneous approaches that leverage the strengths of both modalities [29]. There is growing interest in protocol-based PCD with objective criteria for escalation to minimize unnecessary procedures while ensuring timely intervention.

**Asian Innovations (China and Japan).** Chinese literature presents extensive experience with aggressive PCD incorporating fibrinolytics and novel endoscopic necrosectomy devices, with particular expertise in managing massive necrosis extending throughout the retroperitoneum. Large-volume centers report success rates exceeding 70% with PCD alone when combined with vigorous irrigation and protocolized fibrinolytic therapy [30]. Japanese centers, with exceptional endoscopic expertise and early adoption of advanced technologies, frequently prefer primary EUS-guided interventions and have refined DEN to exceptional technical levels, utilizing specialized accessories and techniques that maximize debridement efficacy while minimizing complication risks [31]. The Japanese approach often incorporates nasocystic irrigation tubes for continuous la-

vage of the necrotic cavity.

**CIS Country Adaptations (Russia and Tajikistan).** Russian surgical schools maintain strong traditions of surgical innovation, reporting extensive experience with modified retroperitoneal and laparoscopic techniques adapted to local anatomical considerations and resource availability. Recent publications emphasize the importance of staged approaches with careful timing of interventions to maximize effectiveness while minimizing morbidity [32]. Research from Tajikistan highlights successful step-up implementation in resource-conscious environments, confirming the philosophy's universal applicability while demonstrating necessary adaptations in settings with limited advanced endoscopic availability, such as greater reliance on PCD and minimally invasive surgical techniques [33]. These regional experiences underscore that the core principles of the step-up approach are universally beneficial, even if the specific tools vary.

### LONG-TERM SEQUELAE AND COMPREHENSIVE FOLLOW-UP STRATEGIES

**Pancreatic Insufficiency and Metabolic Consequences.** Survivors of severe acute pancreatitis confront substantial long-term health challenges requiring dedicated management protocols extending far beyond the acute hospitalization:

**Endocrine Insufficiency:** New-onset diabetes (Type 3c) develops in 15-40% of patients following necrotizing pancreatitis, with risk strongly correlating with the extent of pancreatic necrosis and any resection procedures. This form of diabetes presents unique management challenges due to concurrent exocrine insufficiency and variable counter-regulatory hormone responses, requiring specialized endocrine follow-up and patient education [34].

**Exocrine Insufficiency:** Clinically significant pancreatic exocrine insufficiency (PEI) affects 30-60% of survivors, manifesting as steatorrhea, weight loss, and fat-soluble vitamin deficiencies (A, D, E, K) that may be overlooked without specific questioning and testing. Pancreatic enzyme replacement therapy (PERT) requires careful dosing and administration timing to optimize nutritional absorption and quality of life, with monitoring of clinical response and nutritional parameters [35].

**Chronic Pain and Quality of Life Consid-**

**erations.** A significant patient subset (20-40%) develops chronic abdominal pain following severe pancreatitis, with mechanisms including persistent strictures, disconnected duct syndrome, central sensitization, and pancreatic duct hypertension. Management requires comprehensive pain assessment and multidisciplinary approaches combining medications (avoiding narcotics where possible), endoscopic therapy when indicated (e.g., stent placement for strictures), and psychological support [36]. Quality of life studies consistently show substantial long-term impairments in physical and mental health domains following severe pancreatitis, highlighting the importance of structured rehabilitation programs and continued specialist follow-up beyond the immediate recovery period. Factors such as persistent fistulas, the need for repeated interventions, and the development of incisional hernias after open surgery further contribute to long-term morbidity.

### FUTURE DIRECTIONS AND EMERGING TECHNOLOGIES

**Advanced Imaging and Predictive Analytics.** The management of pancreatic fluid collection complications continues to evolve with emerging technologies that promise further improvements in outcomes. Artificial intelligence (AI) and machine learning applications in radiology are showing promise in the early prediction of complications through automated analysis of CT imaging features, potentially identifying high-risk collections before clinical deterioration occurs [37]. Advanced MRI techniques, including diffusion-weighted imaging (DWI) and secretin-enhanced MRCP, provide superior characterization of collection composition and ductal anatomy, guiding more precise intervention selection and timing. The development of validated clinical prediction rules that incorporate biochemical, clinical, and radiological parameters may enable truly personalized complication risk assessment and preemptive management strategies.

**Novel Therapeutic Approaches and Technical Innovations.** Technical innovations continue to expand the therapeutic armamentarium. Robotic-assisted endoscopy is being explored to enhance visualization, instrument control, and stability during complex necrosectomy procedures. Endoscopic vacuum-assisted closure (EVAC) sys-

tems, adapted from wound care applications, show promise in managing complex fistulas and contaminated cavities through continuous drainage and promotion of tissue granulation and remodeling from the inside out [38]. Biological therapies targeting specific inflammatory pathways (such as IL-1 and TNF- $\alpha$  inhibition) are under investigation for modulating the excessive inflammatory response and potentially reducing the incidence or severity of complications, though their role remains experimental. Nanotechnology approaches for targeted drug delivery to necrotic tissues and innovative stent designs with drug-eluting or biodegradable capabilities represent frontier areas of research that may transform future management.

### CONCLUSION

The management of pancreatic fluid collection complications exemplifies the progress of modern medicine – transitioning from high-morbidity, one-size-fits-all open surgery toward nuanced, patient-centered, team-based approaches that prioritize physiological preservation, organ function, and long-term quality of life. The "step-up" philosophy, supported by an array of minimally invasive techniques and reinforced by multidisciplinary collaboration, has unequivocally saved lives and improved long-term outcomes for patients with severe pancreatitis. Future directions include further technical refinements, the development of sophisticated predictive biomarkers for complications, and the personalization of treatment algorithms based on precise anatomical and patient-specific factors. As technology advances with robotic-assisted endoscopy, advanced imaging analytics, and targeted biological therapies, the medical community continues to advance toward confronting these formidable complications with ever-greater precision and efficacy. The ongoing challenge remains the global dissemination of these advanced approaches while maintaining flexibility for adaptation to local resources and expertise, ultimately aiming to improve outcomes for all patients affected by this devastating disease.

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#### FINANCING

There was no financial support.

#### CONFLICT OF INTEREST

The authors declare no conflict of interest.

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#### ФИНАНСИРОВАНИЕ

Финансовой поддержки не было.

#### КОНФЛИКТ ИНТЕРЕСОВ

Авторы заявляют об отсутствии конфликта интересов.

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