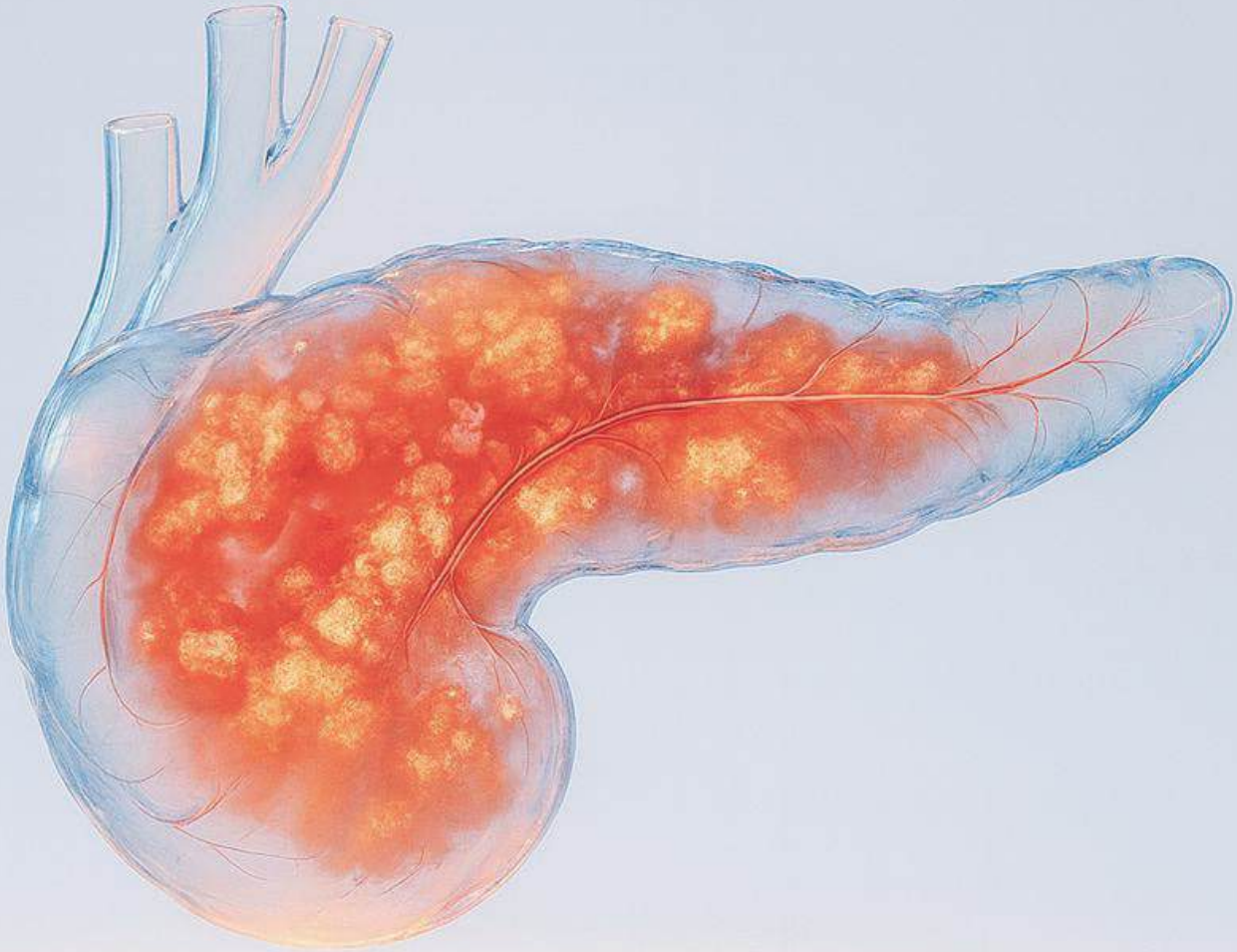
ACUTE PANCREATITIS

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

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Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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ACUTE PANCREATITIS

QUICK REFERENCE GUIDE

Acute pancreatitis is a sudden inflammation of the pancreas caused by premature activation of digestive enzymes within the gland, leading to autodigestion and a systemic inflammatory response. Clinically, it is diagnosed when at least two of three are present: typical acute epigastric pain often radiating to the back, serum lipase or amylase three times the upper limit of normal, or imaging consistent with pancreatitis. The disease spans interstitial-edematous forms that usually resolve with supportive care to necrotizing pancreatitis, which can involve local collections, infection, and organ failure.

Causes, Risk factors & Triggers

- **Common:** gallstones, alcohol.
- **Metabolic:** hypertriglyceridemia, hypercalcemia.
- **Drugs:** azathioprine, valproate, furosemide, didanosine, thiazides, GLP-1 receptor agonists (rare), others.
- **Iatrogenic/trauma:** post-endoscopic retrograde cholangiopancreatography (ERCP), blunt trauma.
- **Infections:** mumps, coxsackie, hepatitis A/E.
- **Genetic/structural:** PRSS1, SPINK1, CFTR, CTRC variants; pancreas divisum.
- **Risks for severe course:** age >60, obesity, systemic inflammatory ... response syndrome (SIRS), persistent organ failure, comorbid cardiopulmonary/renal disease.

Evaluation for Diagnosis

- **Clinical features:** Acute severe epigastric pain radiating to the back, nausea/vomiting, anorexia.
- **Physical examination:** Epigastric tenderness ± guarding/rigidity; tachycardia; hypotension if severe; reduced bowel sounds; pleural effusions in moderate-severe disease.
- **Laboratory investigations**: Serum lipase (preferred) or amylase ≥3× upper limit confirms pancreatic injury. Etiology/severity: complete blood count (CBC), electrolytes, calcium, triglycerides, liver enzymes/bilirubin, blood urea nitrogen (BUN), creatinine, C-reactive protein (CRP); pregnancy test where relevant.

Confirmation of diagnosis (need 2 of 3)

1)Typical pain; **2)** Lipase/amylase ≥3× upper limit; **3)** Imaging consistent with AP.

Imaging

- **Ultrasound (≤24 h):** gallstones/ductal dilatation.
- **Contrast-enhanced computed tomography (CECT) or magnetic resonance imaging (MRI):** if diagnostic uncertainty or clinical deterioration; best window 72-96 h from onset to stage necrosis.

- **CT angiography:** suspected mesenteric ischemia or hemorrhage.
- **Endoscopic ultrasound (EUS)/magnetic resonance cholangiopancreatography (MRCP):** evaluate microlithiasis/ductal issues when stable.

Classification / severity assessment criteria

- **Mild AP:** no organ failure, no local/systemic complications.
- **Moderately severe:** transient organ failure (<48 h) and/or local/systemic complications.
- **Severe:** persistent organ failure (≥48 h).
- **Scores:** BISAP (Bedside Index for Severity in Acute Pancreatitis) day-1; SIRS at 0 and 48 h; modified CT Severity Index (mCTSI) after imaging; Marshall for organ failure.

Differential Diagnosis

- Biliary colic/cholecystitis/cholangitis; peptic ulcer disease or perforation; bowel obstruction/perforation; mesenteric ischemia; acute hepatitis; diabetic ketoacidosis; myocardial infarction; aortic dissection; basilar pneumonia; renal colic; gastroduodenitis.

Management Goals & principles

- Resuscitate early, relieve pain, initiate early enteral nutrition, prevent/treat complications, and treat the cause (biliary, alcohol, triglycerides).
- No routine prophylactic antibiotics.
- Prefer minimally invasive, step-up strategies for infected necrosis.

Step-wise management of acute pancreatitis

0. Triage and confirm

- **Recognize:** acute, severe epigastric pain ± back radiation; nausea/vomiting.
- **Confirm (need 2 of 3)**: typical pain, lipase/amylase ≥3× ULN, or imaging consistent with pancreatitis.
- **Red flags for ICU/transfer now:** shock, hypoxia, altered mentation, persistent organ failure, lactate rising.

1. First hour (ABCs and stabilization)

- **Airway/Breathing:** OXYGEN to keep SpO_2 94-98%; escalate if distress.
- **Circulation (fluids):** start isotonic crystalloids (prefer Lactated Ringer's). If hypovolemic, give bolus 10-20 mL/kg, then goal-directed infusion (targets: MAP ≥65 mmHg, urine output ≥0.5 mL/kg/h, falling Hct/Cr).
- **Analgesia/antiemetic:** opioid (e.g., IV morphine/fentanyl) + ondansetron/metoclopramide.
- **NPO initially,** insert NG tube only if persistent vomiting/ileus.
- **Baseline labs:** CBC, CMP, Ca, triglycerides, CRP, BUN/Cr; pregnancy test if applicable.
- **Etiology screen:** RUQ ultrasound for gallstones/duct dilation.

- **Antibiotics:** do not start routinely (only if infection suspected such as cholangitis, pneumonia, infected necrosis).

2. First 24 hours (risk stratify and set the course)

- **Monitor closely:** vitals, urine output, pain score, Hct/Cr (q6-8h initially). Avoid over-resuscitation (rales, edema).
- **Severity scoring:** SIRS/BISAP at admission and ~24 h; use for level of care decisions.
- **Nutrition early:** if pain improving and no ileus, start oral/enteral feeding within 24 h (clear/soft low-fat). If not tolerated by 48-72 h, start NG/NJ enteral feeds.
- **If diagnostic uncertainty or deterioration:** defer contrast CT/MRI to 72-96 h from onset unless another diagnosis must be ruled out now.
- **Gallstone pancreatitis:** if cholangitis or ongoing obstruction - urgent ERCP. Otherwise plan index-admission cholecystectomy for mild cases.

3. 24-48 hours (adjust and prevent complications)

- **Titrate fluids to endpoints**; reassess need if edema/respiratory compromise.
- **Glycemic/electrolyte control:** correct K/Mg/Ca; manage hyperglycemia.
- **Analgesia plan:** regular dosing; consider patient-controlled analgesia if needed.
- **Disposition:** Disposition: ward vs HDU/ICU by scores and organ support needs.

4. Etiology-directed measures (in parallel)

- **Alcohol-related:** brief intervention, thiamine if risk, link to de-addiction.
- **Hypertriglyceridemia:** insulin infusion to lower TG; consider plasmapheresis for severe/refractory with organ failure (per local availability).
- **Drug-induced:** stop culprit (e.g., azathioprine, valproate, furosemide).
- **Idiopathic/recurrent/familial:** consider EUS/MRCP after recovery; genetics if strong family history.

5. Complications - step-up approach

- **Suspected infected necrosis:** fever/leukocytosis with necrosis on imaging; start targeted antibiotics; step-up drainage (percutaneous/endoscopic) - minimally invasive necrosectomy if no improvement.
- **Empiric antibiotics options:** Meropenem 1 g IV q8h or Cefepime 2 g IV q8-12h + metronidazole 500 mg IV q8h or Ciprofloxacin 400 mg IV q12h + metronidazole 500 mg IV q8h (use fluoroquinolones cautiously). Duration: Usually 4-6 weeks in stable patients while source control and wall maturation proceed; de-escalate to cultures.
- **Pancreatic/extra-pancreatic collections:** drain only if infected or causing symptoms/obstruction.

- **Respiratory failure, AKI, shock:** move to ICU; provide organ support.
- **Abdominal compartment syndrome/ hemorrhage/pseudoaneurysm:** urgent surgical/IR involvement.

6. Imaging strategy (summarized)

- **Ultrasound (≤24 h)**: gallstones/ choledocholithiasis screen.
- **CECT/MRI (best at 72-96 h):** define necrosis/collections and guide intervention.
- **CT angiography:** if concern for mesenteric ischemia or vascular complication.

7. Nutrition escalation

- **Enteral preferred** (oral - NG - NJ).
- **Parenteral nutrition** only if enteral goals cannot be met.

8. Discharge and prevention

- **Criteria**: pain controlled on oral meds, tolerating diet, vitals stable, no new organ failure, clear outpatient plan.
- **Gallstone disease**: ensure cholecystectomy before discharge (mild cases) or scheduled when recovered (severe/necrotizing).

- **Education**: alcohol/tobacco cessation, triglyceride control, medication review, return precautions.
- **Follow-up:** surgery/GI appointments; repeat imaging only if clinically indicated.

Assessment of response, Review; follow-up and adjustment

- **Monitor domains**: pain, hemodynamics (MAP/HR/lactate), respiratory status (SpO_2/OXYGEN need), urine output/creatinine, hematocrit, electrolytes/glucose, CRP (~48 h), nutrition tolerance.
- **Improving**: falling pain and CRP, stable MAP, UO ≥0.5 mL/kg/h, advancing diet.
- **If plateau/worsening**: reassess fluids (avoid overload), check for infection/ necrosis; obtain CECT (72-96 h) if not done; escalate level of care.
- **Nutrition adjustment**: move oral - NG/ NJ if intake inadequate by 48-72 h; reserve TPN for intolerance/ contraindication to enteral.
- **Before step-up/step-down**: verify adherence, fluid balance, analgesia adequacy, and etiology control (ERCP/ cholecystectomy, alcohol cessation, triglyceride control).

Referral for specialist consultation or tertiary care (tiered)

- **Immediate transfer/ICU:** shock, hypoxemia, persistent organ failure, rising lactate/creatinine, severe SIRS.
- **Gastroenterology / advanced endoscopy:** cholangitis or persistent biliary obstruction (urgent ERCP), symptomatic pseudocyst/WON, suspected infected necrosis.

- **Interventional radiology / surgery - step-up strategy for infected necrosis:**
  1. **Percutaneous catheter drainage** + antibiotics
  2. **Endoscopic transluminal drainage ± direct endoscopic necrosectomy (DEN)** if no improvement in ~72 h if endoscopic access not feasible, Open necrosectomy only if minimally invasive options fail or instability persists.
- **Low-resource:** early phone consult/ transfer pathways; stabilize with fluids, oxygen, analgesia, and NG/NJ feeds.

Complications

- **Local:** pseudocyst, WON, sterile/ infected necrosis, hemorrhage/ pseudoaneurysm, gastric outlet/ biliary obstruction, fistulae.
- **Systemic:** acute respiratory distress syndrome (ARDS)/effusions, acute kidney injury (AKI), shock, sepsis, hyperglycemia.
- **Timing: early (<4 weeks**) collections vs late (≥4 weeks) pseudocyst/WON.

Objectives of Patient education & Instructions to the patient/ caregiver

- **Know the cause** (stones/alcohol/ triglycerides) and the plan to address it (ERCP/cholecystectomy, abstinence, lipid control).
- **Diet & fluids**: small, low-fat meals; hydrate; avoid alcohol; limit sugary/ fizzy drinks.
- **Medicines**: take analgesics/enzyme supplements exactly as prescribed; avoid unapproved OTCs.
- **Activity**: walk as tolerated; avoid heavy lifting until cleared.
- **Red flags** (seek care now): worsening epigastric pain, persistent vomiting, fever ≥38 °C, jaundice, dizziness/ syncope, breathlessness.
- **Follow-up**: attend lab/imaging visits; confirm gallbladder surgery timeline if biliary; review triglycerides and diabetes screening; discuss smoking/ alcohol cessation support.

INTRODUCTION

Acute pancreatitis is a sudden inflammation of the pancreas caused by premature enzyme activation, leading to autodigestion and a systemic inflammatory response. Most cases are due to gallstones, alcohol use, or severe hypertriglyceridemia. Illness ranges from mild, self-limited pain to necrotizing disease with organ failure. Globally, incidence is about 35 per 100,000 per year; roughly 80% are mild, while 20% become severe. Mortality is ~3% in interstitial edematous pancreatitis and can reach ~20% with necrosis. Risk rises with age and varies by region based on alcohol use and biliary stone prevalence.

SCOPE OF THIS GUIDELINE

These recommendations apply to adults presenting with acute pancreatitis, covers triage and early risk stratification; covering initial assessment, diagnosis, management, follow-up, and referral criteria and complications with a step-up drainage/debridement approach; plus disposition criteria, referral pathways, and audit indicators for both low-resource and tertiary settings. Also, detailed surgical protocols (laparoscopic cholecystectomy timing, necrosectomy approaches) are not included in the guidelines.

Intended users

It is intended for emergency physicians, hospitalists, surgeons, gastroenterologists, intensivists, anesthetists, radiologists, nurses, dietitians, and pharmacists, as well as primary-care clinicians coordinating follow-up and administrators implementing quality and referral systems

- **Primary (PHC, GP, emergency first-contact):**Recognize and stabilize suspected acute pancreatitis.
- **Secondary (Atoll/Regional hospital/General hospital) -** Diagnose, risk-stratify, and manage mild-moderate disease; organize definitive care for biliary etiologies.
- **Tertiary (Teaching hospital/Referral center with ICU, GI endoscopy, IR, HPB surgery -** Comprehensive care for severe/complicated pancreatitis and advanced etiological therapy.

Limitations at primary and secondary care:

- Imaging options may be restricted to ultrasound; CT and MRCP are generally unavailable.
- Laboratory support may not extend beyond basic biochemistry and hematology.
- Advanced interventions (enteral tube feeding, ERCP, ICU-level monitoring) are seldom feasible.

In these settings, the focus should be on early recognition, fluid resuscitation, pain control, basic laboratory evaluation, and timely referral to higher-level facilities when severity or complications exceed local capacity.

DEFINITION

According to the Revised Atlanta Classification (2012), a diagnosis of acute pancreatitis requires at least two of the following three criteria:

1. Characteristic abdominal pain consistent with pancreatitis (acute onset, severe, persistent, epigastric, often radiating to the back).
2. Serum amylase and/or lipase levels at least three times the upper limit of normal.
3. Imaging findings (ultrasound, CT, or MRI) characteristic of acute pancreatitis with or without necrosis.

If the first two criteria are met, imaging is not always required for confirmation but may be done to assess severity or rule out complications.

The Revised Atlanta Classification divides acute pancreatitis into two forms:

- **Interstitial edematous pancreatitis:** inflammation of pancreatic parenchyma and surrounding fat.
- **Necrotizing pancreatitis:** cell death within the pancreas and nearby tissues.

Severity is further stratified into three categories:

- **Mild:** no organ failure or local/systemic complications.
- **Moderately severe:** transient organ failure (< 48 hours) and/or local complications.
- **Severe:** persistent organ failure (> 48 hours) affecting one or more organ systems.

RISK FACTORS & TRIGGERS

Gallstones and alcohol account for approximately 80% of cases. Other triggers include hypertriglyceridemia, hypercalcemia, medications (e.g., azathioprine, furosemide), post-ERCP procedures, abdominal trauma, infections (mumps, coxsackie, acute HAV/HEV), and genetic predisposition (trypsinogen mutations).

EVALUATION FOR DIAGNOSIS

Domain	What to look for	Key findings	Why it matters / Next step
Symptoms	Character and radiation of pain; associated GI symptoms	Sudden, severe epigastric pain radiating to the back with nausea, vomiting, anorexia. Biliary: sharp, acute, shooting posteriorly. Alcohol/metabolic: dull, diffuse ache.	Classic presentation; raises suspicion for acute pancreatitis.
Focused history	Etiology clues and risks	Gallstone risk factors; years of heavy alcohol use; culprit medications; smoking; consider rare familial forms if common causes absent.	Guides targeted workup and early management pathway.
Physical exam	Severity markers and peritonism	Epigastric tenderness with guarding/ rigidity; tachycardia; hypotension (severe cases); reduced bowel sounds.	Identifies patients needing urgent stabilization/transfer.
Complication signs	Hemorrhagic features	Flank ecchymosis (Grey Turner's), periumbilical bruising (Cullen's).	Suggests hemorrhagic pancreatitis; escalate care and imaging.
Initial labs	Diagnose, assess severity, seek cause	Serum amylase and lipase; CBC; liver enzymes; electrolytes; calcium; BUN; creatinine; C-reactive protein.	Confirms diagnosis, stratifies risk, and screens for biliary/metabolic triggers.

CONFIRMATION OF DIAGNOSIS

- **History (find the cause):** biliary symptoms or ultrasound gallstones; unexplained weight loss or new-onset diabetes; years of heavy alcohol use; culprit drugs; recent trauma; recent endoscopic retrograde cholangiopancreatography; high triglycerides or calcium; autoimmune disease; family history of recurrent pancreatitis.
- **Baseline labs:** serum triglycerides, calcium, liver enzymes, complete blood count, renal function (blood urea nitrogen, creatinine), C-reactive protein.
- **Genetics (when to consider):** strong family history or recurrent/idiopathic episodes - test PRSS1/SPINK1/CFTR/CTRC per local protocol.
- **Early imaging (first 24 h):** abdominal ultrasound to detect gallstones or bile-duct dilation.
- **Chest X-ray (moderate-severe cases)**: look for pleural effusions as a severity marker.
- **Contrast-enhanced computed tomography (CECT)**
  - **Indication:** diagnostic uncertainty, poor response to conservative care, or clinical deterioration.
  - **Best timing:** 72-96 hours after symptom onset to assess severity/necrosis (earlier scans can miss necrosis).

- **Follow-up CECT:** if clinical course worsens or when planning invasive intervention for local complications (e.g., drainage/necrosectomy).

Further Investigations in Idiopathic AP

- After the first or second episode of idiopathic acute pancreatitis, a repeat transabdominal ultrasound should be performed after discharge.
- If no cause is identified, proceed with endoscopic ultrasonography (EUS).
- If EUS findings are negative, perform magnetic resonance imaging (MRI) with MR cholangiopancreatography (MRCP).
- Genetic testing should be considered when the etiology remains unidentified, particularly after recurrent episodes.

DIFFERENTIAL DIAGNOSIS

Abdominal pain can stem from many sources, and pinpointing acute pancreatitis starts with a focused history and exam. Common alternatives to consider include:

Condition	Differentiation from acute pancreatitis	Key bedside clues	Best initial test(s)	Quick action / pitfall
Peptic ulcer disease / perforation	Burning epigastric pain; perforation - sudden severe pain with peritonitis	Guarding, board-like abdomen; history of NSAIDs	Upright CXR for free air; CT abdomen if uncertain	Surgical consult if perforation; don't delay imaging
Cholangitis	Biliary sepsis from duct obstruction	Fever, jaundice, RUQ pain (Charcot triad) ± hypotension/ confusion	LFTs (cholestatic), RUQ ultrasound; blood cultures	Urgent antibiotics and biliary drainage (ERCP)
Cholecystitis	Gallbladder inflammation	RUQ pain after fatty meals; positive Murphy sign	RUQ ultrasound	Early surgery; distinguish from biliary pancreatitis
Bowel obstruction	Crampy pain, distension, obstipation	High-pitched/absent bowel sounds, vomiting of feculent material	Abdominal X-ray/CT	NG decompression, fluids; find cause
Bowel perforation (non-ulcer)	Sudden generalized pain	Peritonitis, sepsis	Upright CXR/CT for free air	Immediate surgical evaluation
Mesenteric ischemia	Ischemic gut pain	"Pain out of proportion," AFib, severe vascular disease	CT angiography	Time-critical revascularization; high mortality if missed
Acute hepatitis	Hepatic inflammation	Malaise, jaundice, RUQ tenderness	AST/ALT markedly elevated; hepatitis panel	Supportive care; rule out acetaminophen toxicity

Diabetic ketoacidosis (DKA)	Metabolic emergency mimicking abdominal pain	Polyuria, polydipsia, Kussmaul breathing	Glucose, venous blood gas pH, bicarbonate, ketones; anion gap	Insulin + fluids + electrolytes; pancreatitis can coexist
Basilar pneumonia	Referred upper-abdominal pain	Cough, fever, pleuritic pain, basilar crackles	Chest X-ray	Start antibiotics if bacterial
Myocardial infarction (inferior/atypical ACS)	Cardiac ischemia presenting as epigastric pain	Diaphoresis, dyspnea, cardiac risk factors	ECG + troponin	Treat as ACS until excluded; don't anchor on GI
Aortic dissection	Catastrophic vascular tear	Tearing pain to back, pulse/BP differentials, neuro deficits	CT angiography (or TOE if unstable)	Immediate vascular/ surgical team; do not delay
Renal colic	Ureteric stone pain	Flank - groin colicky pain, hematuria	Urinalysis; non-contrast CT KUB or renal ultrasound	Analgesia, urology if obstructed/infected

MANAGEMENT GOALS

In 85-90% of cases, the disease is self-limiting and subsides in 3-7 days with moderate fluid resuscitation, management of pain and nausea, and early oral feeding. There is no proven treatment of acute pancreatitis. Treatment is mainly supportive.

Immediate goals are:

- Relieve symptoms, especially pain
- Correct fluid and electrolyte imbalances
- Prevent local and systemic complications
- Identify and treat the underlying cause
- Reduce risk of recurrence

MANAGEMENT PRINCIPLES

- Fluids: Early resuscitation in the first 24-48 h (prefer Lactated Ringer's), titrated to perfusion and urine output; avoid fluid overload.
- Analgesia: Adequate pain control using appropriate agents (WHO ladder as guide).

- Nutrition: Start early enteral feeding within 24-48 h if tolerated. Advance to soft, low-fat diet as symptoms improve. If oral intake is inadequate by 72 h, use nasogastric or nasojejunal feeding. Use parenteral nutrition only when enteral goals cannot be met.
- Etiology-directed care: Alcohol cessation counseling; manage hypertriglyceridemia; for biliary disease, arrange timely ERCP when indicated and index-admission cholecystectomy for mild cases.
- Antibiotics: Do not give prophylactically; reserve for proven/suspected infection (e.g., infected necrosis).
- Imaging/intervention: Use imaging judiciously; intervene for complications (necrosis, obstruction) when indicated.
- Monitoring: Close watch of vitals, labs (hematocrit, creatinine, CRP), and organ function to detect deterioration or fluid overload.
- Escalation: Prompt ICU transfer or specialist input for persistent organ failure, rising severity scores, or clinical worsening.

PHARMACOLOGICAL THERAPY

First hour (ABCs and stabilization)

1. **Airway/Breathing:** Oxygen to keep SpO_2 94-98%; escalate if distress.
2. **All confirmed/suspected cases, first 24-48 h - Restore and maintain intravascular volume:** A moderate fluid infusion rate of 1.5 ml/kg/h is recommended. Bolus is given if signs of hypovolemia are present 10 ml/kg bolus. No bolus if normovolemia; 1.5 ml/kg/hour maintenance for all patients. Lactated Ringer's is the preferred fluid. Additional fluids may be given depending on hematocrit and clinical signs of hypovolemia. Reassess q4-6h; avoid overload (rales, edema, rising oxygen need) then consider ICU. **Caution:** In rare patients with acute pancreatitis due to hypercalcaemia, lactated Ringer's is contraindicated because it contains 3 mEq/L calcium. In these patients, normal saline should be used for volume resuscitation. Avoid hydroxyethyl starch-containing fluids as there is no demonstrable mortality benefit and possible risk of multiple organ failure.

   ### Hemodynamic and renal targets:

   - Maintain mean arterial pressure (MAP) between 65-85 mmHg.
   - Ensure urine output ≥ 0.5 mL/kg/hour.
   - Maintain blood urea nitrogen (BUN) <20 mg/dL (or blood urea <40 mg/dL).
   - Maintain hematocrit <44%.

   Note: Invasive monitoring should be reserved for patients in an intensive care unit setting.

3. **Optimize nutrition:** Start oral intake as soon as pain, nausea, vomiting, and ileus resolve (see below)
4. **Control pain effectively**
    - Initiate with NSAIDs, following the WHO analgesic ladder: progress to opioids (e.g., fentanyl 1-2 µg/kg or morphine 0.1 mg/kg every 4 h, titrated to effect, or meperidine), and consider regional blocks or epidurals as needed. Reassess pain regularly and tailor type, dose, route, and frequency to patient response.
5. **Prevent and manage infection**
    - Avoid routine prophylactic antibiotics.
    - Antibiotics should be used if there is proven extrapancreatic infection or strong suspicion of infected necrotizing pancreatitis.
    - Start empiric therapy and then narrow down therapy once cultures results are available. Positive microbiologic cultures from body fluids, e.g., blood, sputum, bile, urine, and drain fluid, are definite indications for antibiotic therapy.

Note: The presence of gas bubbles within the pancreatic/peripancreatic necrotic collection on a CT scan suggests infected pancreatic necrosis and is an indication for antibiotic therapy. Elevated levels of C-reactive protein (CRP), white blood cell (WBC) count, or procalcitonin (PCT) alone should not be used as biomarkers to start antibiotic therapy.

6. **Antiemetics:** Ondansetron 4 mg IV every 8 h as needed.
7. **Perform nasogastric tube aspiration** if there is evidence of paralytic ileus, abdominal distension and vomiting.
8. **Monitor for complications (see below)**
    a. Watch for signs of fluid overload during volume resuscitation.
    b. Track organ function, inflammatory markers, and hemodynamics to catch evolving multiorgan dysfunction syndrome (MODS) early.

MANAGEMENT OF COMPLICATIONS

- **Early (<4 weeks):** acute peripancreatic fluid collections, acute necrotic collections.
- **Late (≥4 weeks):** pseudocyst, walled-off necrosis (WON).
- **Local:** necrosis/WON, pseudocyst, abscess.
- **Systemic:** respiratory failure, renal impairment, cardiovascular instability, sepsis.

Core principles

- Prefer minimally invasive, step-up strategies (antibiotics, drainage, minimally invasive necrosectomy; open surgery last).
- No prophylactic antibiotics. Start antibiotics for suspected/confirmed infection or extrapancreatic infection.
- Maintain early enteral nutrition; optimize fluids, electrolytes, glycemia; ICU-level monitoring when organ failure is present.

Local complications

Lesion	When to just observe	When to intervene	First-line intervention	Notes
Acute peripancreatic fluid collection (early)	Hemodynamically stable, painless or improving, no infection	Worsening pain, early sepsis, compartment concerns	Image-guided percutaneous drainage (if infected/tense)	Many resolve spontaneously
Pseudocyst (late, encapsulated fluid, no solid debris)	Asymptomatic, stable size	Rapid enlargement, infection, pain, gastric outlet/biliary obstruction	Endoscopic transmural drainage (LAMS or plastic stents); percutaneous if endoscopy unavailable	<10% progress to pseudocyst; delay intervention until mature wall unless unstable
Sterile necrosis / WON	Mild symptoms, improving clinically	Persistent pain, vomiting/obstruction, disconnected duct, failure to thrive	Endoscopic step-up: transmural drainage ± DEN (direct endoscopic necrosectomy) after maturation	Time CT/MRI at 72-96 h initially; intervene after wall maturation if stable
Infected necrosis / pancreatic abscess	-	Fever, sepsis, gas in collection on CT, positive culture, clinical deterioration	Antibiotics + drainage (percutaneous or endoscopic); escalate to minimally invasive necrosectomy if no improvement	Start antibiotics promptly; tailor to cultures; open surgery if step-up fails

Antibiotics for suspected/confirmed infected necrosis

- Start immediately if infection is suspected:
- **Empiric antibiotics options:** Meropenem 1 g IV q8h or Cefepime 2 g IV q8-12h + metronidazole 500 mg IV q8h or Ciprofloxacin 400 mg IV q12h + metronidazole 500 mg IV q8h (use fluoroquinolones cautiously).
- Confirm with CT gas in collection and/or CT-guided aspiration for Gram stain/culture.
- Duration: Usually, 4-6 weeks in stable patients while source control and wall maturation proceed; de-escalate to cultures.

Systemic complications (organ failure)

Complication	Immediate actions	Ongoing targets / escalation
Respiratory failure (ARDS/effusions)	Oxygen to keep SpO_2 94-98%; low threshold for ICU; lung-protective ventilation if intubated; drain large symptomatic pleural effusions	Daily fluid reassessment; manage pain to reduce splinting; consider HFNC/NIV if appropriate
Acute kidney injury	Goal-directed crystalloids (avoid overload), correct electrolytes; hold nephrotoxins; monitor urine output	Early nephrology input if oliguria or rising creatinine; consider RRT if refractory
Cardiovascular instability/shock	Rapid fluid resuscitation; vasopressors (norepinephrine) if hypotension persists after fluids; lactate-guided resuscitation	Invasive monitoring in ICU; evaluate for infected necrosis or hemorrhage
Sepsis	Broad-spectrum antibiotics after cultures; look for sources (infected necrosis, cholangitis, pneumonia, line sepsis)	Early source control (drainage/ERCP); de-escalate antibiotics to culture results

Biliary complications and obstruction

- Cholangitis or persistent biliary obstruction: Urgent ERCP with sphincterotomy/stone extraction within 24 hours of presentation
- Mild gallstone pancreatitis: Index-admission cholecystectomy once stabilized to prevent recurrence (defer in necrotizing/severe disease until recovery window).

Monitoring, imaging, and timing

- **Imaging strategy:** ultrasound ≤24 h for gallstones; CECT/MRI at 72-96 h if severity assessment needed or course worsens; repeat imaging when planning interventions or with deterioration.

Monitor trends in vitals, urine output, hematocrit, creatinine, CRP; watch for fluid overload.

NON-PHARMACOLOGICAL INTERVENTIONS

Nutrition:

- Start oral intake within the first 24-48 hours as soon as pain, nausea, vomiting, and ileus resolve. Offer a soft, low-residue, low-fat diet first, then advance to normal consistency as tolerated.
- For severe cases or persistent intolerance, place a nasojejunal tube placed beyond the ligament of Treitz placed endoscopically or radiologically should be provided rather than initiating parenteral nutrition. Deliver continuous feeds at a low rate, then gradually increase to goal calories. This approach maintains mucosal integrity and stimulates pancreatic rest, without the risks associated with parenteral nutrition.
- If the target rate is not achieved within 48-72 hours and if severe acute pancreatitis is not resolved, supplemental parenteral nutrition should be provided.

Assessment of response

Domain	What to monitor	Target / improving trend	Concerning trend	Action if concerning
Pain & nausea	Pain score, antiemetic use, ability to mobilize	Pain falling; longer dosing intervals; ambulating	Escalating pain, persistent vomiting	Reassess fluids/analgesia plan; check for complications; consider NG decompression
Hemodynamics	HR, BP/MAP, capillary refill, lactate	HR <100, MAP ≥65, lactate normalizing	Hypotension, rising lactate, cool extremities	Fluid reassessment; start vasopressors if needed; move to HDU/ICU
Respiratory	SpO_2, work of breathing, CXR if indicated	SpO_2 94-98% on room air; comfortable breathing	Rising O_2 needs, tachypnea, new effusions/infiltrates	Escalate oxygen; evaluate for ARDS/ effusion; consider ICU
Urine output / renal	mL/kg/h, Cr, BUN	UO ≥0.5 mL/kg/h; Cr/ BUN falling	Oliguria, rising Cr/BUN	Reassess fluids; stop nephrotoxins; nephrology input
Volume status	JVP, edema, lung exam, daily weight	Euvolemia; no new rales/edema	Fluid overload signs	Slow/stop fluids; diurese if needed; ICU if hypoxemic
Inflammation/ organ failure	SIRS/BISAP, Marshall score	Scores stable or improving at 24-48 h	Worsening score or new organ failure	Escalate level of care; search for infection/ necrosis
Hematocrit	6-8 hourly early, then daily	Falling to normal with resuscitation	Persistent >44% or rising	Inadequate fluids or hemoconcentration; reassess strategy
Creatinine	6-8 hourly early, then daily	Down-trend	Rising	AKI -optimize hemodynamics, consider ICU
Electrolytes & Ca/G1C	K, Mg, Ca, glucose	Within range; stable glucose	Hypocalcemia, dysglycemia	Correct electrolytes; manage hyper/ hypoglycemia
CRP (or procalcitonin)	At ~48 h then as needed	CRP plateau/fall	CRP >150 mg/L at 48 h or rising	Higher severity; evaluate for necrosis/infection
GI function / nutrition	Nausea, ileus, diet tolerance, NG/NJ needs	Tolerates oral/enteral within 24-48 h	Persistent ileus, feeding intolerance	Start/continue NG/NJ feeds; evaluate obstruction/collection
Etiology control	Biliary clearance, TG levels, alcohol abstinence	ERCP/ cholecystectomy completed when due; TG falling; counseling done	Ongoing obstruction; uncontrolled TG; ongoing alcohol	Urgent ERCP; lipid-lowering measures; addiction services
Infection surveillance	Fever, WBC, cultures, CT signs (gas in collections)	Afebrile or defervescing; negative cultures	Persistent fever/ leukocytosis, gas in necrosis	Start/adjust antibiotics; plan drainage (step-up)
Imaging	US ≤24 h; CECT/MRI at 72-96 h if needed	No new complications	New/worsening collections/necrosis	Multidisciplinary plan: drain vs observe; schedule follow-up imaging
Mobility & VTE risk	Ambulation, calf tenderness, SpO_2 with activity	Early mobilization; no DVT signs	Immobility, DVT signs	Mechanical ± pharmacologic prophylaxis per bleeding risk

REVIEW, FOLLOW-UP & ADJUSTMENT

Before discharge

- **Clinical:** pain controlled on oral meds; tolerating diet; afebrile; stable vitals; no new organ failure.
- **Etiology addressed:** ERCP done if indicated; cholecystectomy completed or scheduled; alcohol cessation plan; triglycerides falling; culprit drugs stopped.
- **Education:** return precautions (fever, worsening pain, vomiting, dyspnea), diet, alcohol/tobacco cessation, meds.
- **Plan:** written follow-up dates and contacts.

Outpatient follow-up timeline

- **7-14 days:** review symptoms, weight, oral intake, analgesia needs; repeat BMP (creatinine/electrolytes), glucose, ± CRP if severe course.
- **2-6 weeks:** surgical follow-up for cholecystectomy (if deferred); lipid panel for hypertriglyceridemia; alcohol-use counseling check-in.
- **4-8 weeks:** assess for late complications (pseudocyst, walled-off necrosis) if persistent pain, early satiety, vomiting, or palpable mass; order contrast CT/MRI or EUS if symptomatic/collections suspected.
- **8-12 weeks:** screen for exocrine insufficiency (weight loss, steatorrhea) and consider pancreatic enzyme replacement; screen for post-pancreatitis diabetes (fasting glucose/HbA1c).
- **As indicated:** genetics/EUS/MRCP for recurrent or idiopathic cases.

Triggers for earlier re-evaluation or escalation

- New/worsening pain, fever, or sepsis signs; rising creatinine, persistent ileus or feeding intolerance; jaundice; GI bleed; respiratory decline; inability to maintain hydration/nutrition.

Documentation checklist (each review)

- Severity score (SIRS/BISAP) trend; fluid balance; organ supports; nutrition route and adequacy; etiology actions taken; imaging results and planned interventions; discharge readiness or reasons to defer

PROGNOSIS

- Overall mortality is low (~1-2%) but rises sharply in severe acute pancreatitis.
- Early risk stratification is essential; traditional scores often need 48-hour data.
- SIRS at admission, especially persistent SIRS at 48 h, predicts worse outcomes: ~25% mortality vs ~8% with transient SIRS.
- Adding high C-reactive protein (CRP) or interleukin-6 (IL-6) improves prediction (sensitivity ~77-89%, specificity ~79-86%).
- BISAP (day-1 bedside score): 0-2 - <2% mortality; 3-5 - >15% risk. More specific than Ranson/APACHE II, but somewhat less sensitive.
- Modified CT Severity Index (mCTSI) (0-10): 0-2 mild, 4-6 moderate, 8-10 severe; higher scores correlate with necrosis, complications, and mortality, guiding intervention timing.

Referral for specialist consultation or Step-up interventions

Indication	Who to involve	First step	Reassess / next step (~72 h)	Key notes
Persistent organ failure (respiratory, renal, cardiovascular)	Gastroenterology, surgery, ICU	Stabilize; search for source (infected necrosis, cholangitis)	If source identified - proceed per below; escalate organ support	Early multidisciplinary huddle; consider transfer to tertiary center
Suspected / confirmed infected necrosis (fever, sepsis, gas in collection on CT or positive culture)	Gastroenterology, interventional radiology (IR), surgery	Image-guided percutaneous catheter drainage (PCD) + broad-spectrum antibiotics	If febrile/no clinical improvement after ~72 h - endoscopic transluminal drainage ± direct endoscopic necrosectomy (DEN)	Step-up limits tissue trauma, controls sepsis, reduces complications; delay debridement until wall maturation when stable
Cholangitis or persistent biliary obstruction in gallstone pancreatitis	Gastroenterology, IR, surgery	Endoscopic transmural drainage (lumen-apposing metal stent or plastic)	Add DEN if symptoms persist or sepsis develops	Avoid early intervention; prefer minimally invasive routes
Symptomatic sterile necrosis / walled-off necrosis (pain, vomiting/obstruction, disconnected duct)	Gastroenterology, IR, surgery	Endoscopic transmural drainage (lumen-apposing metal stent or plastic)	Add DEN if symptoms persist or sepsis develops	Avoid early intervention; prefer minimally invasive routes
Hemorrhage/ pseudoaneurysm from pancreatitis	IR, surgery	CT angiography for IR embolization	If bleeding persists - surgical hemostasis	Maintain high suspicion in sudden shock or falling Hb
Large, symptomatic pseudocyst	Gastroenterology	Endoscopic cystogastrostomy/ cystoduodenostomy	If not resolving - exchange/add stents or percutaneous drain	Most collections resolve; intervene only if infected, enlarging, or obstructive

Antibiotics for infected necrosis: start immediately (e.g., carbapenem or cefepime/ceftazidime + metronidazole or fluoroquinolone + metronidazole), tailor to cultures, continue ~4-6 weeks while source control and wall maturation proceed.

PATIENT EDUCATION

- Explain to the patient about preventing another attack: what to do after acute pancreatitis
- Know your trigger: gallstones, alcohol, or high triglycerides cause most cases.
- If alcohol was the cause: complete abstinence. Even “social” drinking raises relapse risk.
- Quit smoking as it fuels inflammation and recurrence.

Do’s

- Hydrate: aim for ~8 glasses of water daily; sip regularly.
- Eat small, frequent, low-fat meals (5-6/day). Start with bland diet: broths, plain rice, boiled veg, lean proteins (chicken/fish/pulses). Favor complex carbs.
- Take medicines exactly as prescribed, including pancreatic enzymes (with meals/snacks) if advised, and pain medicines.
- Walk daily as tolerated; increase activity gradually.
- Keep all follow-ups for blood tests, imaging, and medication review.
- Plan gallstone care: if stones caused your attack, schedule cholecystectomy (gallbladder removal) within weeks once you’re stable.
- Manage risks long term: target triglycerides, diabetes, weight, and blood pressure with your clinician.

Don’ts

- No alcohol (any amount) if alcohol-related pancreatitis.
- Don’t smoke or vape.
- Don’t eat fried foods, creamy sauces, rich desserts, or heavy late meals.
- Don’t use carbonated drinks or sugary beverages while recovering.
- Don’t skip enzyme doses if prescribed; don’t self-start OTC antacids without approval.
- Don’t lift heavy or do strenuous exercise until your doctor clears you.
- Diet at a glance

- Choose: soups, khichdi/plain rice, idli/poha, boiled veg, curd, dal, grilled/steamed fish or chicken, oats, fruits (non-fried).
- Limit/Avoid: deep-fried foods, biryanis with rich gravies, butter/cream, bakery sweets, red meat, alcohol, fizzy drinks.

Red flags - seek care now

- Sudden worsening epigastric pain
- Persistent vomiting or inability to keep fluids down
- Fever ≥38 °C (100.4 °F)
- Abdominal swelling or severe tenderness
- Jaundice (yellow eyes/skin)
- Fast heartbeat, fainting, or dizziness

FOLLOW-UP PLAN

- 1-2 weeks: clinic review (symptoms, weight, labs).
- Within admission or soon after: plan cholecystectomy for gallstone pancreatitis.
- 2-6 weeks: triglycerides and diabetes review; adjust diet/meds.
- As advised: imaging if symptoms persist (to check for pseudocyst/walled-off necrosis).
- Bring your medication list to visits and note any foods or activities that trigger symptoms.

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