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Contemporary Strategies in the Management of Patients with Acute Pancreatitis

Ali A. Ghweil, Ibrahim Gamal Eldin Ibrahim, Shamardan Bazeed

[1]Professor of tropical medicine and gastroenterology, Department of Tropical Medicine and Gastroenterology, Faculty of Medicine, South Valley University, Qena, Egypt
[2]Assistant lecturer of tropical medicine and gastroenterology, Department of Tropical Medicine and Gastroenterology, Faculty of Medicine, South Valley University, Qena, Egypt
[3]Professor of tropical medicine and gastroenterology, Department of Tropical Medicine and Gastroenterology, Faculty of Medicine, South Valley University, Qena, Egypt

Corresponding Author
Ibrahim Gamal Eldin Ibrahim
Email: ibrahim@med.svu.edu.eg

ABSTRACT

Acute pancreatitis (AP) is a serious gastrointestinal disorder with rising global incidence, ranging from 4.9 to 73.4 per 100,000 population. Biliary stones are the leading cause, accounting for up to 70% of cases, while alcohol, hypertriglyceridemia, drugs, genetics, and autoimmune conditions contribute variably. Although most cases are mild and self-limiting, 20% progress to severe disease with mortality reaching 30%. Diagnosis requires a combination of abdominal pain, elevated enzymes, and imaging, though biomarkers and clinical scores aid severity prediction. Management focuses on early supportive measures including oxygen therapy, fluid resuscitation, analgesia, and nutritional support. Ringer's lactate is preferred for resuscitation, while strong opioids remain the mainstay for pain control. Enteral feeding is recommended within 1–2 days, with parenteral nutrition reserved for intolerance. Severe disease may require ICU support, with interventions for infected necrosis delayed until walled-off collections form. Prophylactic antibiotics are not indicated, but targeted therapy is crucial for confirmed infections. Prevention strategies include index-admission cholecystectomy for biliary pancreatitis, alcohol and smoking cessation, lipid-lowering therapies, and drug withdrawal when relevant. ERCP plays a central but selective role: indicated in patients with cholangitis or persistent obstruction, while conservative management is preferable in uncomplicated cases. Despite a 5–15% risk of complications, timely ERCP can prevent progression in high-risk patients. Future research should refine predictive tools, optimize fluid strategies, and clarify the timing of endoscopic interventions.

KEYWORDS: Acute pancreatitis, Biliary pancreatitis, ERCP, Complication management.

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INTRODUCTION

Acute pancreatitis **(AP)** is a severe gastrointestinal disorder that imposes emotional, physical, and financial burdens **(Omar et al., 2023).** Its incidence has increased worldwide, ranging between 4.9 and 73.4 cases per 100,000 population. Common etiologies include biliary stones, alcohol intake, iatrogenic injury, metabolic disturbances, autoimmune disease, infections, and pancreatic tumors, though a considerable number of cases remain idiopathic **(Goljan et al., 2023)**.

Acute biliary pancreatitis **(ABP)** is the most frequent subtype, usually triggered by transient obstruction of the bile and pancreatic ducts, leading to bile reflux or elevated ductal pressure. A biliary origin can be suspected when alanine transaminase **(ALT)** levels rise more than threefold, showing a 95% positive predictive value. Pathogenesis is explained mainly by two mechanisms: reflux of bile into the pancreatic duct or temporary ampullary blockage by sludge or impacted stones **(Ikoma et al., 2023)**.

Clinically, about 80% of gallstone pancreatitis cases are mild and self-limiting, whereas 20% progress to severe disease with mortality reaching 30%. Spontaneous passage of common bile duct stones into the duodenum occurs in nearly 50% of cases **(Taha et al., 2025).** Although conservative treatment is generally effective, biliary complications occur in up to 20% of patients **(Chan et al., 2022) (Fig 1)**.

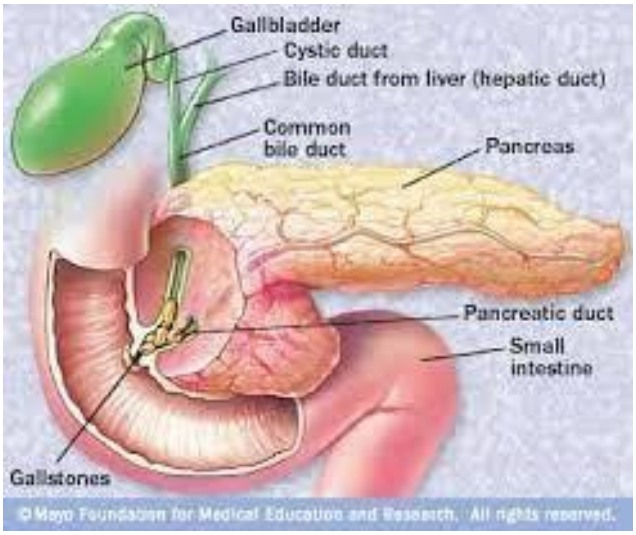

Fig (1): Pancreas and biliary system anatomy (Ilhan et al., 2012).

Endoscopic retrograde cholangiopancreatography **(ERCP)** remains debated in ABP regarding its timing, safety, and indications. The procedure carries a 5–15% complication risk, including pancreatitis, cholangitis, bleeding, perforation, recurrent bile duct stones, cholangiocarcinoma, and anesthesia-related events **(Albana et al., 2025)**.

DIAGNOSIS

Clinical Features and Criteria

AP typically presents with epigastric or diffuse abdominal pain **(80–95%)**, nausea and vomiting **(40–80%)**, abdominal distension, fever, or breathlessness. On examination, pyrexia, tachycardia, hypotension, guarding, ileus, or oliguria may occur. Risk factors include gallstones, obesity, alcohol, smoking, hyperlipidemia, and drugs. Alcohol excess is best assessed using the 10-item Alcohol-Use Disorders Identification Test **(AUDIT) (Kim and Hendershot, 2020)**. Diagnosis requires two of three: **(1)** characteristic abdominal pain, **(2)** amylase or lipase ≥3× normal, and **(3)** imaging consistent with pancreatitis **(CT, MRI, or TUS in children)**. Using only the first two may miss 25% of cases, while falsely diagnosing 10% **(Kim and Hendershot, 2020)**.

Aetiology

Gallstones

Gallstones are the leading cause, responsible for 20–70% of cases in Western populations, with higher prevalence in women **(~two-thirds of cases)** and increasing incidence with age **(Hussien et al., 2023)**. Ultrasound is recommended to detect gallstones and duct dilatation, indicating possible cholangitis **(AlHussaini, 2025)**.

Alcohol and Smoking

Alcohol is the second most common cause, contributing up to one-third of cases in North America and Europe, but is the leading cause in Eastern Europe. Reported prevalence varies geographically, from 2% in Latin America and <10% in China, to 46% in Japan and 70% in Finland. Binge drinking [≥80 mL ethanol/session in UK men, ~70 g in US men] increases risk, especially with chronic heavy intake **(>210 g/week in men, >112 g/week in women)**. Only a minority of heavy drinkers develop AP, suggesting co-factors like genetics, hypertriglyceridemia, and smoking. Smoking independently increases risk of acute, recurrent, and chronic pancreatitis, while low alcohol intake may reduce first-episode risk in non-smokers **(Hegab et al., 2025)**.

Hypertriglyceridemia

Hypertriglyceridemia accounts for ~9% of cases globally, reaching 33% in Chinese cohorts, making it the third most common cause. Risk rises 4% for every 100 mg/dL increase above 1000 mg/dL, though 500 mg/dL is now often considered the diagnostic threshold. It is crucial to measure levels on admission, as hypertriglyceridemia-associated AP is more severe **(Ismaeil et al., 2024)**.

Drugs

Drug-induced AP accounts for up to 5% of cases. Evidence is strongest when recurrence occurs after rechallenge with the same drug. Drugs are classified from Class I **(strongest evidence, e.g., antiretrovirals, thiopurines, valproic acid, enalapril)** to Class IV **(single reports)**. Mechanisms may be intrinsic **(dose-related toxicity)** or idiosyncratic **(host–drug interaction)**. Onset ranges from days to months, requiring careful drug history **(Nabil et al., 2024).**

Endoscopic Retrograde Cholangiopancreatography (ERCP)

ERCP induces AP in ~9% of patients, rising to 14% in high-risk groups **(Schepers et al., 2020)**. Risk is greatest in young women with small bile ducts or sphincter dysfunction. Complications increase with repeated cannulation or prolonged procedures. Preventive strategies exist, including exploiting accessory pancreatic ducts to reduce pressure **(Hussien et al., 2023)**.

Genetic Factors

Hereditary pancreatitis, usually presenting in childhood or adolescence, arises from PRSS1 mutations **(e.g., R122H, N29I)**. Other mutations in CTRC, CPA1, PNLIP, CEL, SPINK1, CFTR, and CLDN2 also predispose by enhancing enzyme activation or impairing secretion **(Yu et al., 2023).** Patients often present with recurrent AP before chronic pancreatitis is evident on imaging. Genetic variants also contribute to secondary causes, such as hypertriglyceridemia from LPL mutations **(Li et al., 2020) (Fig 2)**.

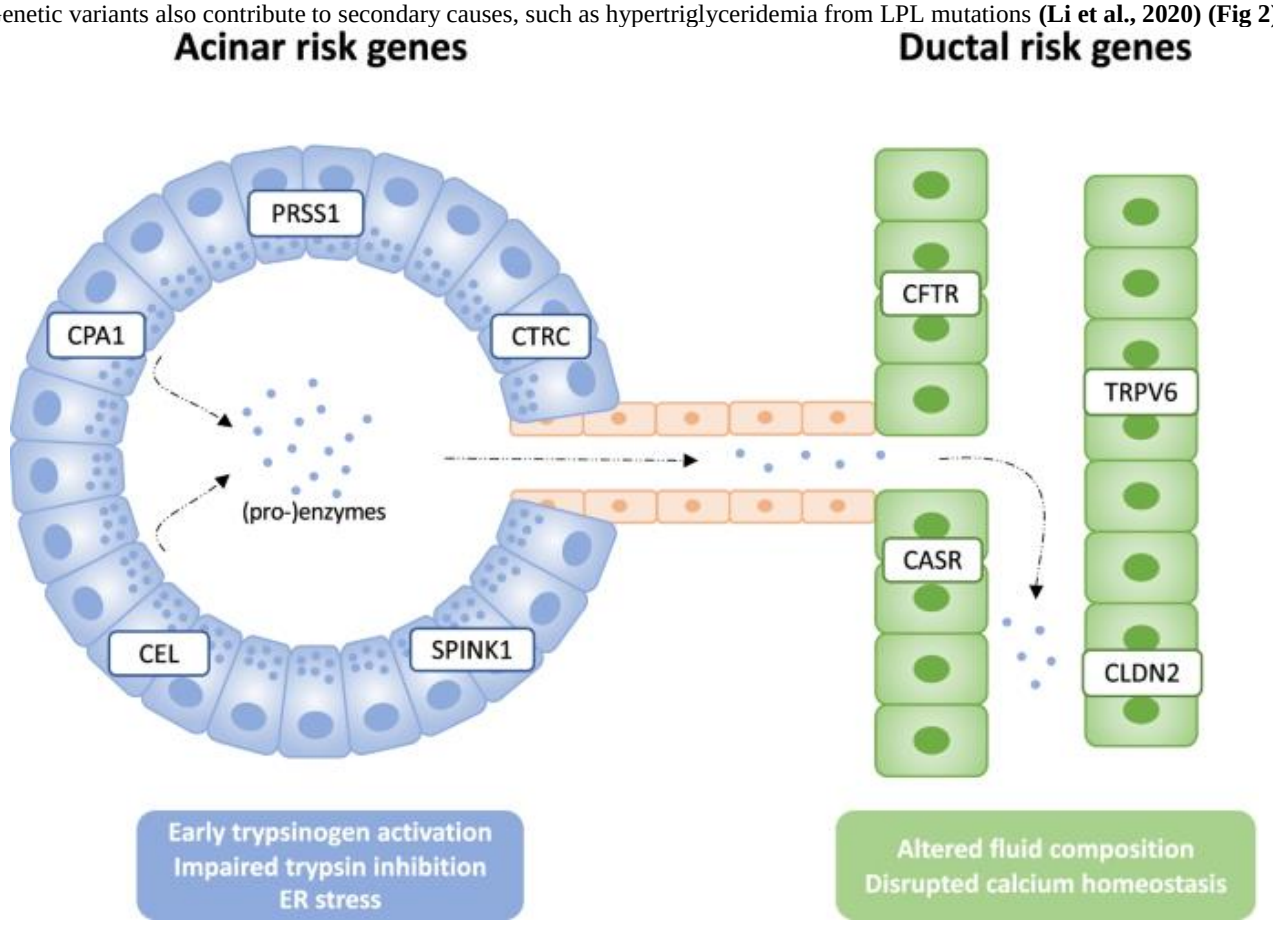

Fig 2: Overview of acinar and ductal pancreatitis risk genes and their underlying disease-causing mechanisms (Piseddu et al., 2022).

Autoimmune Pancreatitis

Autoimmune pancreatitis is a rare form, classified as type 1 **(lymphoplasmacytic sclerosing pancreatitis, linked to IgG4 disease)** and type 2 **(idiopathic duct-centric disease)**. Type 1 often involves systemic manifestations such as sclerosing cholangitis, thyroiditis, or nephritis, with elevated serum IgG4. Type 2 is more often associated with inflammatory bowel disease and carries a less favorable prognosis **(Notohara et al., 2021)**.

Other Causes

Less common causes include trauma, hypercalcemia, viral infections **(mumps, CMV, coxsackie B, SARS-CoV-2)**, tumors, congenital variants **(pancreas divisum)**, surgery, toxins **(organophosphates)**, and scorpion bites **(*Tityus trinitatis*)**. SARS-CoV-2 uses ACE2 receptors on acinar and islet cells, potentially worsening AP and complicating it with diabetes and metabolic dysfunction (**Memon et al., 2021)**.

Pathophysiology

Understanding the mechanisms of acute pancreatitis **(AP)** is crucial for developing targeted therapies to reduce pancreatic and systemic injury **(Eid et al., 2022).** At the molecular level, AP begins with injury to pancreatic acinar and ductal cells due to disruption of intracellular calcium signaling that normally regulates stimulus–secretion coupling **(Wiley et al., 2023)**.

Calcium Dysregulation

In acute biliary pancreatitis, gallstone obstruction of the ampulla of Vater increases ductal pressure and/or reflux of bile into the pancreatic duct. High pressure triggers abnormal calcium influx into acinar cells, normally maintained at nanomolar concentrations, through Piezo 1 and TRPV4 channels. Bile acids further activate G protein-coupled bile acid receptor 1, leading to calcium release from the endoplasmic reticulum **(ER)** via IP3R and RyR channels **(Sutton, 2020)**. Depletion of ER calcium stores stimulates extracellular calcium entry via STIM1, ORAI, and TRPC channels. Normally, calcium influx is regulated by SARAF and microRNA-26a, but both are downregulated, enhancing calcium entry. Oxidative stress adds to the process by opening TRPM2 channels **(Fanczal et al., 2020)**.

Persistent calcium influx overloads mitochondria, opening the mitochondrial permeability transition pore, collapsing membrane

potential, and impairing ATP production **(Bruce et al., 2021)**. While insulin can partly preserve glycolytic ATP, this is less efficient than oxidative phosphorylation. ATP depletion weakens SERCA and PMCA calcium pumps, worsening calcium toxicity. Similar pathways occur in alcohol-associated AP, where fatty acid ethyl esters **(FAEEs)** and fatty acids from FAEEs or triglyceride lipolysis inhibit mitochondrial ATP production **(Bruce et al., 2020)**. Severity correlates with toxin exposure, as seen with rising triglyceride levels and AP severity **(Lu et al., 2020)**.

Cellular Injury and Inflammation

ATP loss impairs autophagy, disrupts zymogen secretion, promotes co-localisation of zymogen granules and lysosomes, and activates inflammasomes, leading to necrosis. Calcium overload also activates NFAT and NF-κB pathways, increasing cytokine release and NLRP3 inflammasome activation **(Wen et al., 2020; Gao et al., 2021)**. Multiple pancreatic cell types—including ductal, stellate, macrophages, and neutrophils—further amplify injury. Damage-associated molecular patterns **(ATP, nucleic acids, cytokines)** stimulate innate immune responses **(Karki and Kanneganti, 2021)**.

Cytokines such as TNF-α and interleukins 1α, 1β, 6, and 18 drive systemic inflammation, leading to systemic inflammatory response syndrome **(SIRS)**, multi-organ dysfunction, or cytokine storm syndrome **(de Oliveira et al., 2020)**. Anti-inflammatory responses, such as regulatory T cell activation, may reflect dysregulation, predisposing to persistent inflammation, immunosuppression, and catabolism. Gastrointestinal barrier damage leads to bacterial translocation, endotoxemia, and infection of pancreatic necrosis, further driving systemic complications and mortality. Notably, Enterococcidae in the gut microbiome strongly correlates with severe disease **(Yu et al., 2020)**. Obesity worsens outcomes through adipocyte lipolysis, raising triglyceride levels hydrolyzed by pancreatic and adipocyte lipases **(Yang et al., 2022)**.

ASSESSMENT OF SEVERITY

Predicted Severity

Severity prediction aims to identify patients at risk of complications early after admission. While actual severity requires time for confirmation, prediction guides intensive management. Clinical scoring systems, such as the Original Atlanta Classification, show ~70% accuracy on admission, improving to ~80% on day two **(van den Berg et al., 2020)**. Machine-learning tools, including EASY-APP developed by the Hungarian Pancreatic Study Group and validated in >3000 patients, achieve 89% accuracy and are available online for clinical use. Omics technologies and point-of-care tools show promise but need wider evaluation **(Kui et al., 2022)**.

Among biomarkers, C-reactive protein ≥150 mg/L on day two indicates severe systemic inflammation and can prompt CT imaging for complications. The Pancreatitis Activity Scoring System **(PASS)** is also valuable, especially when pain medication scoring is excluded. Frequent monitoring of respiratory, cardiac, and renal function is essential for timely organ support **(Paragomi et al., 2022)**.

Actual Severity

The Revised Atlanta Classification **(RAC)** divides AP into: **(1)** mild—no complications or organ failure; **(2)** moderately severe—transient organ failure **(<48 h)**, local complications, or comorbidities; and **(3)** severe—persistent organ failure **(≥48 h)**, most often respiratory. Around 65–70% of cases are mild, resolving within days, with patients requiring 3–6 weeks off work. Moderate disease affects 20–25%, with fluid collections or necrosis, hospital stays >2 weeks, and 6–12 weeks off work. Severe AP occurs in ~10%, with multi-organ failure and hospital stays >4 weeks; mortality reaches 50% in this group, giving an overall mortality rate of 1–5% **(Garg and Sing, 2019)**.

The Determinants-Based Classification **(DBC)** offers four categories: mild **(no necrosis/organ failure)**, moderate **(sterile necrosis or transient organ failure)**, severe **(infected necrosis or persistent failure)**, and critical **(infected necrosis with persistent failure)**. Recent evidence suggests infected necrosis contributes less to mortality than previously thought. Modified DBC schemes, such as those developed by the Epidemiología de la Pancreatitis Aguda en Medicina Intensiva group, which separate transient vs. persistent failure and necrosis status, correlate more closely with outcomes than RAC or DBC. Another variant further subdivides severe DBC into two groups for greater prognostic precision **(Zubia-Olaskoaga et al., 2021)**.

Acute Pancreatitis in Special Populations

Children

Paediatric acute pancreatitis shares many features with adults, though incidence is lower at 10–15 per 100,000 annually and continues to rise. Lower rates were reported by the British Paediatric Surveillance Unit due to incomplete ascertainment. Diagnosis follows the same criteria as adults, with ultrasound preferred for initial imaging. In infants and toddlers, irritability, vomiting, and abdominal distension may indicate disease. CECT is recommended 5–7 days after onset to detect necrosis, while MRI/MRCP helps delineate ductal anomalies with less radiation exposure **(Iannuzzi et al., 2022).**

Attempts to establish severity criteria in children include parameters such as age, WBC, albumin, calcium, urea, LDH, and fluid collections, but no consensus exists **(Walkowska et al., 2022)**. The lower incidence of severe disease necessitates multicentre collaboration for research and management.

Aetiologies differ from adults, with lifestyle playing a minor role. Drug-induced pancreatitis is frequent, linked to asparaginase, azathioprine, 6-mercaptopurine, sodium valproate, tetracyclines, aminosalicylic acid, steroids, sulfasalazine, and NSAIDs. Other causes include gallstones, anatomic anomalies **(pancreas divisum, long common channel, duodenal diverticulum, biliary obstruction)**, metabolic disorders **(hypertriglyceridaemia, hypercalcaemia, methylmalonic acidaemia)**, trauma **(accidental,**

abuse, ERCP-related), infection, hereditary, autoimmune, and idiopathic origins **(Walkowska et al., 2022)**.

Pregnancy

Acute pancreatitis occurs in 1 in 500–5000 pregnancies, higher than in the general population **(Yang et al., 2020)**. Hormonal changes induce a pro-lithogenic state, making gallstones the leading cause, followed by alcohol and hypertriglyceridaemia. In some Chinese series, hypertriglyceridaemia accounted for up to one-third of cases. Estrogen alters lipid metabolism, and preventive measures are recommended in women with high triglycerides **(Gupta et al., 2022)**.

Hypertriglyceridaemia-related acute pancreatitis is often severe and worsens maternal and fetal outcomes. It has been linked to fertility treatment, oral contraceptives, and HRT, with one cohort of 31,494 women showing a relative risk of 1.57 for pancreatitis in HRT users. Caesarean section or pregnancy termination lacks clear guidelines, but preliminary data suggest early intervention improves outcomes **(Oskarsson et al., 2014; Liu et al., 2024).**

Elderly

Incidence of acute pancreatitis rises with age, with gallstones as the predominant cause. Cholecystectomy is the preferred preventive treatment, though endoscopic sphincterotomy is suitable for frail patients. Frailty and comorbidity worsen outcomes **(Baeza-Zapata et al., 2021)**. Idiopathic pancreatitis remains common, reported in 30–40% of elderly patients even with advanced imaging. Polypharmacy may contribute through overlooked drug-induced cases **(Baeza-Zapata et al., 2021)**.

Malignancy is a critical concern. Obstructive pancreatitis from benign or malignant tumours often presents mildly and recurrently; management includes surgical resection or palliative stenting. Autoimmune pancreatitis, more common in older men, requires IgG4 testing. Up to 50% of autoimmune pancreatitis cases are associated with malignancy, often gastric, lung, or prostate cancer, supporting its role as a paraneoplastic syndrome. Other cancers—myeloma, parathyroid carcinoma, leukaemia, and small-cell lung cancer—can induce pancreatitis via hypercalcaemia **(Freitas et al., 2022)**.

MANAGEMENT OF ACUTE PANCREATITIS

Inpatient Management

Investigations on Admission

On admission, vital signs and oxygen saturation should be assessed; arterial blood gases may be measured, but oxygen saturation is quicker in the emergency setting. SARS-CoV-2 testing is performed. Before diagnosis, patients usually require oxygen, IV fluids, and analgesia, which does not hinder accurate diagnosis of abdominal pain.

Initial investigations include serum amylase/lipase, triglycerides, lipid panel, full blood count, renal/liver function tests, glucose, HbA1c, calcium, and transabdominal ultrasound **(TUS) (Yang et al., 2021)**. Chest X-ray or ultrasound can detect pleural effusion, a marker of severe disease. Amylase and lipase lack specificity, as both may rise in peptic ulceration, cholecystitis, mesenteric ischaemia, or macroamylasemia. When uncertainty persists, MRI or CT can confirm pancreatitis **(swollen pancreas, fat stranding, peri-pancreatic collections)** or alternative diagnoses **(AK et al., 2024).**

Gallstone pancreatitis is common, with most stones passing into the duodenum early. Transient ALT/AST elevation is typical. If fever, jaundice, and bile duct dilation suggest cholangitis, ERCP **(avoiding pancreatic duct cannulation)**, sphincterotomy, and stone extraction are appropriate; EUS before ERCP helps confirm the need. Without cholangitis, conservative treatment is preferred **(Manes et al., 2020)**.

Initial Therapy

Core management includes oxygen, IV fluid resuscitation, analgesia, and nutrition. Nil-by-mouth regimens, prophylactic antibiotics, and avoidance of early opiates have been disproven in trials and excluded from guidelines. Antivirals may be needed for SARS-CoV-2 infection, and antivenom for scorpion stings in endemic regions. Severe cases may require ICU support **(Iacopetta et al., 2022)**.

Oxygen

A target SpO_2 of 94–98% is appropriate; 88–92% is advised for patients at risk of hypercapnic failure **(COPD, obesity)**. Nasal cannulae or simple masks deliver lower oxygen, while Venturi and reservoir masks provide higher concentrations. If SpO_2 < 85%, 15 L/min via a reservoir mask is indicated, then reduced as stable. Oxygen should not be withdrawn for room-air testing; ABG is required if deterioration occurs despite higher oxygen delivery, with critical care input if needed **(Gehdoo et al., 2021)**.

Intravenous Fluid Resuscitation

Prompt IV fluid therapy prevents hypoperfusion and organ failure **(Machicado et al., 2020)**. Early resuscitation **(within 24 h)** lowers risk of systemic inflammatory response and organ failure, with rates of 5–10 mL/kg/h recommended. Targets include HR < 120/min, urine output > 0.5 mL/kg/h, MAP 65–85 mm Hg, and haematocrit 35–44%. Invasive monitoring may be used in critically ill patients.

Mild and moderate disease usually resolve with fluids; however, excess resuscitation increases mortality from multi-organ effects **(Malbrain et al., 2020)**. Fluid responsiveness must be assessed, with passive leg raising a potential method. Ringer's lactate is preferred over saline, supported by small trials and systematic reviews **(Malbrain et al., 2020)**.

Pain Management

Pain is often severe; strong opioids are first-line and reduce need for additional analgesia **(Cai et al., 2021)**. NSAIDs are alternatives in mild disease but may cause renal injury in severe cases. Despite experimental concerns about sphincter of Oddi contraction, trials show opioids are safe and effective.

Early oral feeding in mild disease reduces pain and opioid use. Severe cases may delay solid intake due to pain. Pain severity correlates with disease severity and total opioid requirement. Continuous IV opioids may be used, with patient-controlled methods preferable post-surgery. Epidurals show no clear advantage, though larger trials are awaited **(Thavanesan et al., 2022)**

Pain and anxiety are interlinked; patients need clear explanations, counselling, and support. Analgesia should not substitute for communication. Psychological support is especially important in adolescents and young adults. Most patients should not be discharged on strong opioids **(Gehdoo et al., 2021)**.

Nutrition

Acute pancreatitis induces hypermetabolism with catabolism, insulin resistance, and weight loss, worse in severe disease. Early oral feeding is encouraged; if not tolerated, liquid supplements or enteral feeding within 1–2 days is recommended **(Yao et al., 2022)**. NG feeding is easier, though NJ may be needed for intolerance.

Enteral feeding reduces inflammatory response and bacterial translocation compared to parenteral nutrition, which carries infection risk **(Lakananurak et al., 2020)**. Enteral feeding may be limited in unstable patients, or when interrupted for procedures. Combined enteral–parenteral nutrition has not shown clear benefit **(Alsharif et al., 2020)**.

For hypertriglyceridaemia-induced pancreatitis, treatment focuses on lowering triglycerides with IV insulin, fluids, or plasmapheresis in severe cases, guided by endocrine teams. Oral fibrates are introduced once feeding resumes. Parenteral nutrition with minimal lipid is used if oral/enteral intake is not possible. Long-term management includes maintenance lipid-lowering therapies **(Simha, 2020)**.

MANAGEMENT OF COMPLICATIONS IN ACUTE PANCREATITIS

Critical Care

Severe acute pancreatitis is life-threatening, with persistent organ failure **(≥48 h)** involving respiratory, cardiac, or renal dysfunction. Supportive measures include endotracheal intubation with mechanical ventilation, inotropes for hypotension, and renal replacement therapy **(haemofiltration/haemodialysis)** for oliguria or anuria. These treatments carry risks such as ventilator-induced lung injury, ischaemia from inotropes, and haemodynamic instability during dialysis. Complications may be reduced by careful sedation, early weaning, and conservative fluid management **(Weinberger et al., 2021)**. Ongoing care requires haemodynamic stability, avoidance of nephrotoxic agents, balanced fluid and electrolytes, adequate analgesia, nutrition, and monitoring of organ function. Contrast-enhanced CT **(CECT)** is essential for early detection of intra-abdominal complications. Most critically ill patients present with both persistent organ failure and infected necrosis, underscoring the need for targeted therapies **(Gad et al., 2025; Reddy et al., 2020)**.

Treatment of Infection

Prophylactic antibiotics have not shown benefit in trials, yet surveys across 22 countries reveal frequent overuse. Patients with moderately severe or severe pancreatitis are at high risk of infection due to bacterial translocation across a compromised gut barrier. Infected necrosis is a major concern, with early infection increasing mortality **(Moran et al., 2022)**. Diagnosis may be aided by gas bubbles on CECT. Management includes source control **(percutaneous drainage if needed)**, targeted antibiotics, and physiological stabilization. In the first 4 weeks, diffuse inflammation makes major interventions risky, so antibiotics such as piperacillin-tazobactam, tigecycline, or cephalosporin with metronidazole are standard, guided by microbiology. Severe disease may involve fungal infection, often secondary to broad-spectrum antibiotics, requiring early antifungal therapy **(Singh et al., 2021)**.

Management of Necrosis

Local complications must be promptly identified to prevent readmission in worse condition. Persistent pain, inability to eat for ≥3 days, CRP ≥150 mg/L, or scoring systems suggesting more than mild disease warrant CECT around day 7 **(Baron et al., 2020)**. Patients with necrosis or collections should be referred to specialist centres for selective, step-up management. Endoscopic or percutaneous drainage is preferred before minimally invasive or open surgery, which is reserved for exceptional cases such as adjacent organ necrosis. Interventions are best delayed until collections become walled off **(>4 weeks)**. Infection or critical illness may necessitate earlier drainage, though randomized evidence supports delayed procedures. Endoscopic necrosectomy usually requires multiple sessions but allows earlier discharge, whereas surgical necrosectomy demands prolonged external irrigation and hospital stay **(Boxhoorn et al., 2021)**.

Management of Diabetes Mellitus

Hyperglycaemia indicates severe pancreatitis. Impaired glucose tolerance occurs in up to 60% of patients within 5 years of a first attack, with risk highest in necrosis. Clinical diabetes develops in ~15% after mild and ~40% after severe pancreatitis. Reduced pancreatic islets after necrosectomy often necessitate multiple daily insulin injections. For patients with recurrent hypoglycaemia, continuous subcutaneous insulin pumps with or without glucose monitoring may be used under specialist guidance **(Dovc and Battelino, 2020)**.

Pancreatic Enzyme Replacement Therapy

Exocrine insufficiency is present in >50% of inpatients with acute pancreatitis, persisting in >50% with necrosis. The adult pancreas normally secretes 1–2 million lipase units daily, but secretion is impaired during pancreatitis. Replacement therapy is indicated in moderate-to-severe disease until faecal elastase-1 normalizes **(≥200 µg/g)**, and long-term in >50% necrosis. Steatorrhoea also justifies lifelong therapy if faecal elastase-1 remains <100 µg/g. Standard adult dosing is 50,000 lipase units per meal, half with snacks, adjusted to symptoms. Children's doses are weight-based but should not exceed 10,000 IU/kg/day to ensure growth and vitamin absorption **(Donadello et al., 2024).**

PREVENTION OF RECURRENCE

Biliary Pancreatitis

Cholecystectomy is the most effective prevention method. The PONCHO trial confirmed its cost-effectiveness when performed during index admission. with sphincterotomy halves recurrence in non-surgical patients but increases risk of cholecystitis. Even in idiopathic cases, cholecystectomy reduces recurrence compared to conservative management **(Umans et al., 2020)**.

Alcohol and Smoking

After a first attack, ≥20% of patients relapse, with ~50% progressing to chronic pancreatitis, especially in men who smoke or drink. Abstinence **(<24 g alcohol/2 months)** prevents recurrence. In Iceland, one-third of persistent drinkers relapsed within 5 years, while none of the abstainers did. Counselling and specialist support increase abstinence success **(Sorrento et al., 2022)**. Smoking cessation is equally important, as it increases risks of recurrence, chronic pancreatitis, and pancreatic cancer **(Klein, 2021)**.

Hypertriglyceridaemia

Recurrent hypertriglyceridaemia-related pancreatitis requires triglyceride levels <1000 mg/dL **(11.3 mmol/L)**, ideally <500 mg/dL **(5.65 mmol/L)**. Management includes weight loss, exercise, low-fat diet, alcohol cessation, omega-3 fatty acids, fibrates, niacin, and statins. Plasmapheresis may be used in resistant cases. Novel therapies include alipogene tiparvovec, mipomersen, evinacumab, lomitapide, volanesorsen, and pradigastat **(Packard et al., 2023)**.

Drug-Induced Pancreatitis

If a drug is implicated, cessation is required. Resumption depends on alternatives, indication, and severity. Asparaginase-induced necrotising pancreatitis should prevent re-use, while mild interferon alpha-related cases may allow re-trial. biomarkers lack specificity, and better tools are needed **(Shyam Sunder et al., 2023).**

Cystic Fibrosis and Genetic Causes

Pancreas-sufficient cystic fibrosis patients benefit from CFTR modulators **(ivacaftor, tezacaftor)**, which reduce pancreatitis risk, though in pancreas-insufficient CF these drugs may paradoxically increase risk disabling idiopathic recurrent pancreatitis, ductal stenting, surgery, or total pancreatectomy with islet autotransplantation may be considered in selected patients **(Gould et al., 2022)**.

Autoimmune Pancreatitis

First-line therapy is oral prednisolone **(2 mg/kg, max 60 mg daily)**, tapered by 5–10 mg to a maintenance dose of 5–7.5 mg/day for ~6 months, with close monitoring for disease progression and side effects such as hypertension or diabetes. Steroid-resistant or relapsing cases may require azathioprine, 6-mercaptopurine, or rituximab **(Bischoff et al., 2020)**.

CONCLUSION

Acute biliary pancreatitis represents the most common form of acute pancreatitis, with outcomes determined largely by disease severity and timely intervention. Supportive management—including oxygen, fluid resuscitation, pain control, and nutrition—remains the cornerstone of therapy. ERCP is invaluable in patients with cholangitis or obstructive jaundice but should be reserved for these indications, as conservative management is sufficient in most other cases. Early cholecystectomy during the index admission is critical to prevent recurrence. Advances in severity prediction, fluid management, and endoscopic techniques continue to refine care, yet individualized patient selection remains essential. Future trials should focus on determining optimal timing of ERCP, evaluating novel pharmacologic and nutritional strategies, and integrating genetic and microbiome insights to improve outcomes and reduce mortality in acute biliary pancreatitis.

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