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A REVIEW ARTICLE ON ACUTE AND CHRONIC PANCREATITIS: A COMPREHENSIVE REVIEW OF MECHANISMS AND THERAPEUTICS

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ABSTRACT

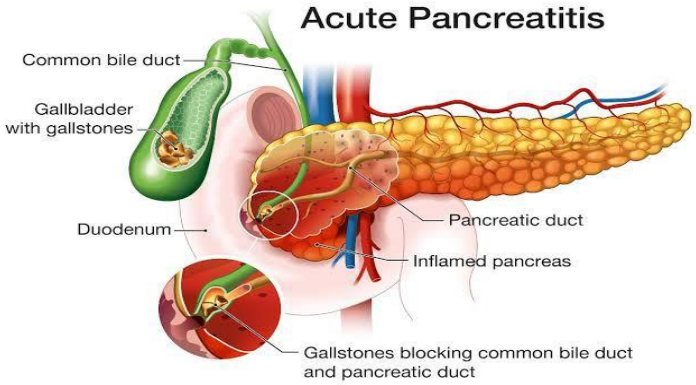
Pancreatitis is an inflammatory condition of the pancreas presenting as acute or chronic disease, both associated with significant morbidity and healthcare burden. Acute pancreatitis (AP) is characterized by sudden inflammation, while chronic pancreatitis (CP) involves progressive, irreversible damage leading to fibrosis and loss of function. This review summarizes the etiology, pathophysiology, clinical manifestations, diagnostic approaches, and current therapeutic strategies for both conditions, highlighting recent advances and future directions.

KEYWORDS: Acute pancreatitis, Chronic pancreatitis, Pancreatic enzymes, Trypsin, Fibrosis, Pancreatic insufficiency, Therapeutics, Inflammation.

1. INTRODUCTION

Pancreatitis encompasses a spectrum of inflammatory disorders of the pancreas. Acute pancreatitis is typically reversible, whereas chronic pancreatitis leads to permanent structural damage and functional impairment. The global incidence of AP ranges from 13–45 cases per 100,000 population, while CP is less common but associated with long-term complications such as diabetes mellitus and pancreatic insufficiency.

The most common etiologies include gallstones, alcohol abuse, metabolic disorders, genetic predisposition, and certain drugs. Understanding the underlying mechanisms is essential for effective management and prevention.



2. Etiology and Risk Factors
Acute Pancreatitis
Gallstones (most common)

Alcohol consumption
Hypertriglyceridemia (>1000 mg/dL)
Drugs (e.g., azathioprine, valproate)
Post-ERCP (endoscopic retrograde cholangiopancreatography)
Trauma and infections
Chronic Pancreatitis
Chronic alcohol abuse
Genetic mutations (PRSS1, SPINK1, CFTR)
Autoimmune pancreatitis
Obstructive causes (tumors, strictures)
Idiopathic

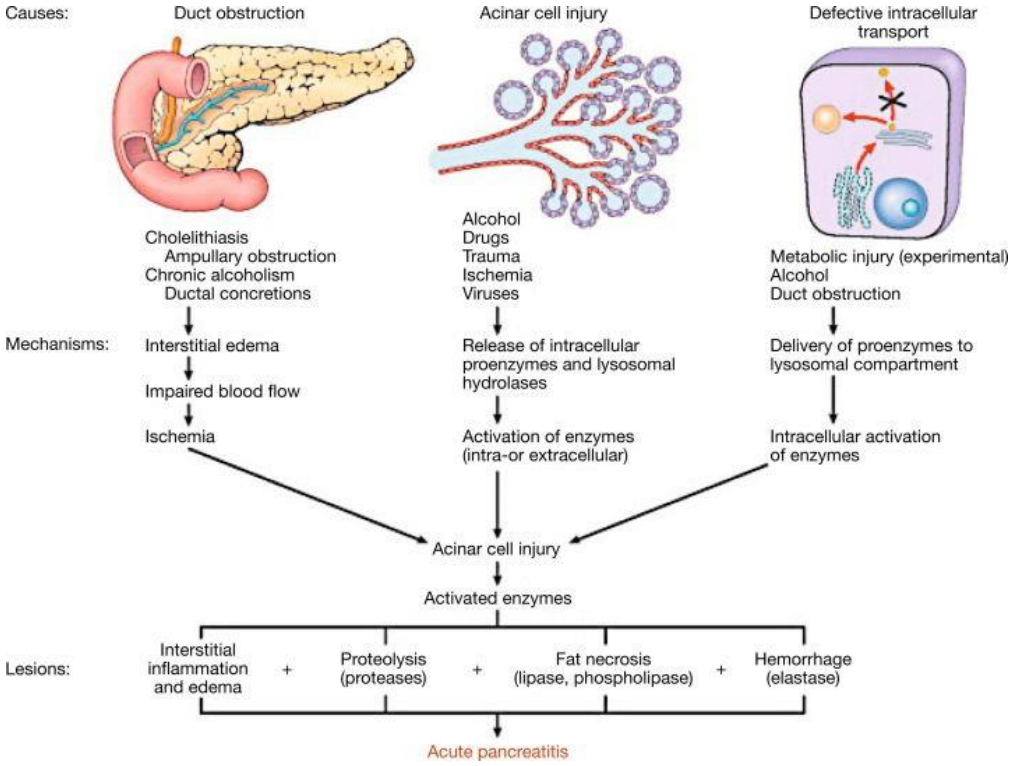
3. PATHOPHYSIOLOGY AND MECHANISMS
3.1 Acute Pancreatitis Mechanism
Acute pancreatitis results from premature activation of pancreatic enzymes within acinar cells, leading to autodigestion.

Key mechanisms
Trypsinogen → Trypsin activation inside pancreas
Acinar cell injury
Inflammatory cascade activation (TNF-α, IL-1, IL-6)
Microcirculatory impairment

Phases
Early phase (first week): Systemic inflammatory response syndrome (SIRS)
Late phase: Local complications (necrosis, pseudocysts)

3.2 Chronic Pancreatitis Mechanism
Chronic pancreatitis involves repeated inflammation leading to fibrosis.

Key features
Stellate cell activation → fibrosis
Ductal obstruction
Progressive acinar cell destruction
Loss of endocrine and exocrine function



4. Clinical Features

Acute Pancreatitis

Sudden severe epigastric pain radiating to back
Nausea and vomiting
Fever
Abdominal tenderness
Severe cases: shock, organ failure

Chronic Pancreatitis
Chronic abdominal pain
Steatorrhea (fat malabsorption)
Weight loss
Diabetes mellitus (late stage)

5. Diagnosis

5.1 Laboratory Findings

Serum amylase and lipase (lipase more specific)
Elevated CRP (severity marker)
Liver function tests (gallstone etiology)
Blood glucose (CP)

5.2 Imaging

Ultrasound: gallstones
CT scan: pancreatic inflammation, necrosis
MRI/MRCP: ductal abnormalities
Endoscopic ultrasound (EUS): early CP detection
Diagnostic Criteria (Acute Pancreatitis)
Diagnosis requires ≥2 of:
Characteristic abdominal pain
Serum lipase/amylase ≥3× normal
Imaging findings

6. Severity Assessment (Acute Pancreatitis)

Ranson's criteria
APACHE II score
BISAP score

Severity classification

Mild
Moderately severe
Severe (persistent organ failure)

7. Complications

Acute Pancreatitis

Pancreatic necrosis
Pseudocyst
Acute respiratory distress syndrome (ARDS)
Renal failure

Chronic Pancreatitis

Pancreatic insufficiency
Diabetes mellitus
Pancreatic cancer (increased risk)
Bile duct obstruction

8. Therapeutic Management

8.1 Acute Pancreatitis Management

Initial Management

Aggressive IV fluid resuscitation (Ringer's lactate)
Pain control (opioids)
NPO (nil per oral) initially
Oxygen therapy if needed
Nutritional Support
Early enteral feeding (preferred over parenteral)

Specific Management

ERCP for gallstone pancreatitis with obstruction
Antibiotics only in infected necrosis (not routine)
Management of complications (drainage, surgery if needed)

8.2 Chronic Pancreatitis Management
Lifestyle Modifications
Alcohol cessation
Smoking cessation
Dietary fat restriction
Medical Therapy
Analgesics (stepwise approach)
Pancreatic enzyme replacement therapy (PERT)
Insulin for diabetes
Endoscopic Treatment
Ductal decompression
Stone removal
Stenting
Surgical Management
Indicated for severe pain or complications
Procedures
Pancreaticojejunostomy (Puestow procedure)
Whipple procedure (in selected cases)

9. Emerging Therapies and Research
Anti-inflammatory agents targeting cytokines
Antioxidant therapy
Stem cell therapy (experimental)
Genetic-targeted treatments

10. Prevention Strategies
Early cholecystectomy in gallstone pancreatitis
Control of triglyceride levels
Avoidance of alcohol
Drug monitoring

11. Prognosis
Acute pancreatitis: Mostly self-limiting; severe cases have high mortality (~15–30%) Chronic pancreatitis: Progressive disease with reduced quality of life and increased risk of malignancy.

12. CONCLUSION
Acute and chronic pancreatitis represent distinct yet interconnected disease processes involving inflammation and pancreatic injury. Early diagnosis, understanding of underlying mechanisms, and timely therapeutic interventions are critical to improving outcomes. Advances in molecular research may offer targeted therapies in the future, potentially transforming management strategies.

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