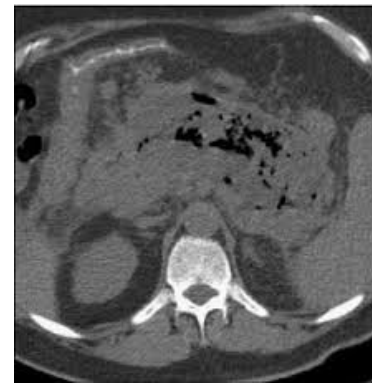


Immediate Management of Acute Severe Pancreatitis



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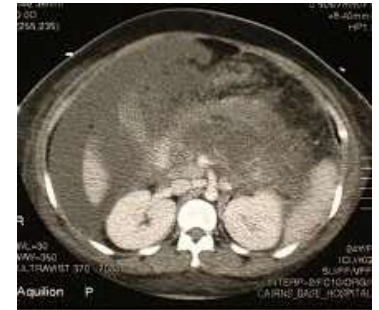
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GAUTENG PROVINCE
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scheme

- Introduction
- Terms & definition/s
- Etiology
- Pathogenesis
- Clinical evaluation/recognition
- Stratification
- Management
- Take Home
- Conclusion



Terms & definitions



- Acute inflammatory process caused by intra-pancreatic enzyme activation with subsequent auto-digestion of surrounding tissue
- Usually rapidly associated with systemic inflammatory response that could either be dampened (mild) or enhanced with all its untoward ramifications
- Parenchymal injury may
 - heal where disease is mild & inflammation quickly abates (with inciting agent removed)
 - persistent/permanent with functional impairment in recurrent acute attacks
- Common medical/surgical emergency
- Major cause of morbidity & mortality
- Significant public health issue



etiology

Usual/common:

- Gallstones
- ETOH
- Hyperlipidemia
- Post ERCP
- ARVs
- Autoimmune (IgG)
- Trauma
- Metabolic:
 - Hypercalcemia
 - hyperparathyroidism

Infrequent/Occasional:

- Hereditary
- Tumors/obstruction
- Infection: mumps
- Steroids:
- Immunosuppressors/chemotherapy
- Congenital anomalies
 - Divisum
 - SOD

Rare:

- Toxins: mushrooms, scorpions
- Vascular/ischemia
- Idiopathic



Cause	Approximate Frequency	Diagnostic Clues	Comments
Gallstones	40%	Gallbladder stones or sludge, abnormal liver-enzyme levels	Endoscopic ultrasonography can reveal very small gallbladder or duct stones.
Alcohol	30%	Acute flares superimposed on underlying chronic pancreatitis	Diagnosis rests on history, obtained with CAGE questions.†
Hypertriglyceridemia	2–5%	Fasting triglycerides >1000 mg/dl (11.3 mmol per liter)	
Genetic causes	Not known	Recurrent acute pancreatitis and chronic pancreatitis	
Drugs	<5%	Other evidence of drug allergy (e.g., rash) only in rare cases	The condition is idiosyncratic and usually mild.
Autoimmune cause	<1%	Type 1: obstructive jaundice, elevated serum IgG4 levels, response to glucocorticoids; type 2: possible presentation as acute pancreatitis; occurrence in younger patients; no IgG4 elevation; response to glucocorticoids	Type 1 is a systemic disease affecting the pancreas, salivary glands, and kidneys; in type 2, only the pancreas is affected.
ERCP	5–10% (among patients undergoing ERCP)		The symptoms can be reduced with rectal NSAIDs (diclofenac or indomethacin) or temporary placement of a stent in the pancreatic duct.
Trauma	<1%	Blunt or penetrating trauma, particularly in midbody of pancreas as it crosses spine	
Infection	<1%	Viruses: CMV, mumps, and EBV most common; parasites: ascaris and clonorchis	
Surgical complication	5–10% (among patients undergoing cardiopulmonary bypass)		The condition is probably due to pancreatic ischemia; pancreatitis may be severe.
Obstruction	Rare	Celiac disease and Crohn's disease, pancreas divisum (controversial), and sphincter of Oddi dysfunction (very controversial)	On rare occasions, malignant pancreatic duct or ampullary obstruction is seen.
Associated conditions	Common	Diabetes, obesity, and smoking	

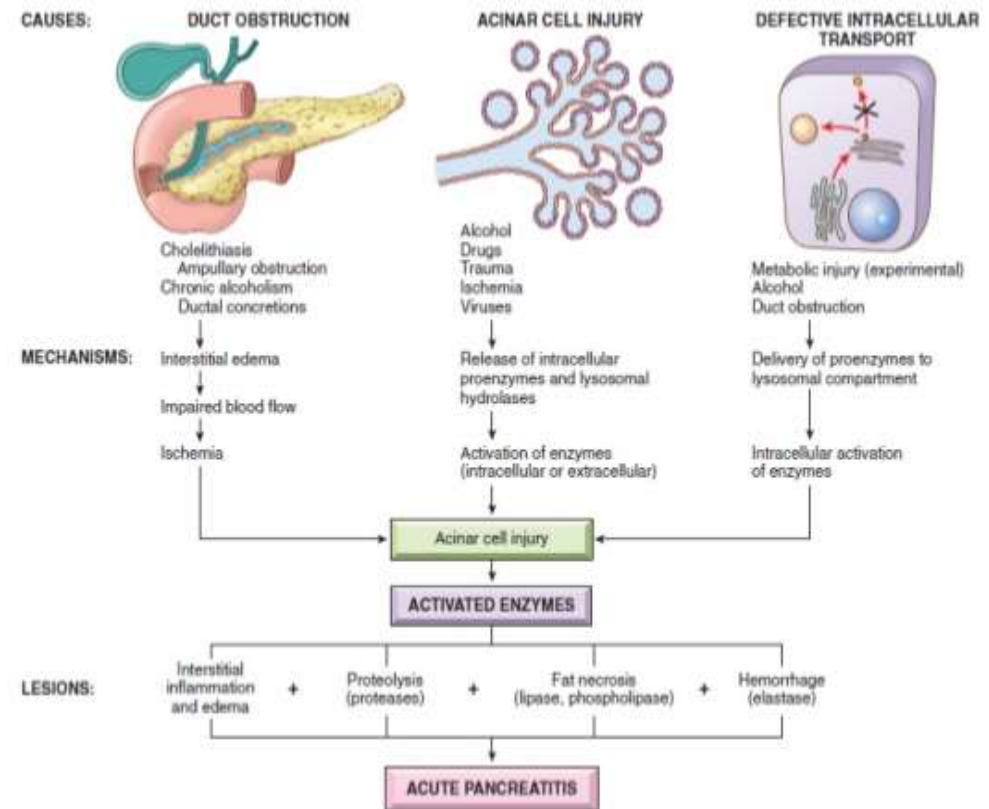
* CMV denotes cytomegalovirus, EBV Epstein–Barr virus, ERCP endoscopic retrograde cholangiopancreatography, and NSAIDs nonsteroidal antiinflammatory drugs.

† CAGE is an acronym for the following questions: Have you ever felt you should cut down on your drinking? Have people annoyed you by criticizing your drinking? Have you ever felt bad or guilty about your drinking? Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (eye opener)?

Pathogenesis & sequelae



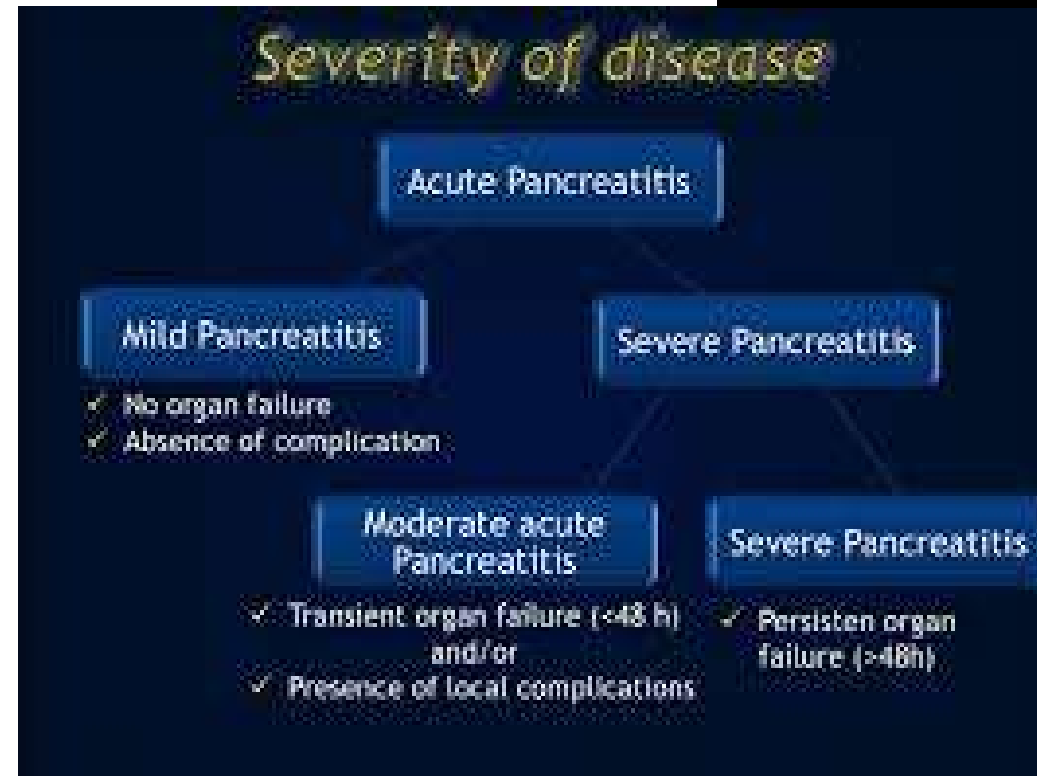
- Release of kallikrein and chymotrypsin results in increased capillary membrane permeability, leading to leakage of fluid into the interstitium and development of edema and relative hypovolemia
- Elastase is the most harmful in terms of direct cell damage, it causes dissolution of the elastic fibers of blood vessels and cuts, leading to hemorrhage
- Phospholipase A destroys phospholipids of cell membranes causing severe pancreatic and adipose tissue necrosis
- Lipase flows into damaged tissue and is absorbed into systemic circulation, resulting in fat necrosis of the pancreas and surrounding tissues

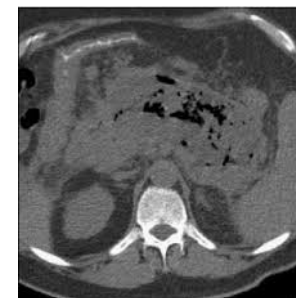
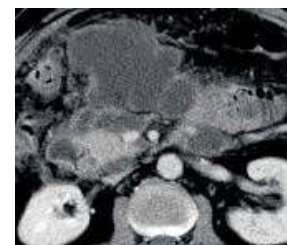
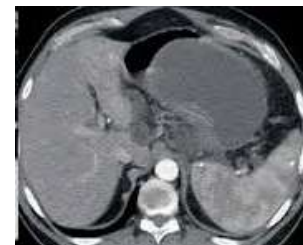
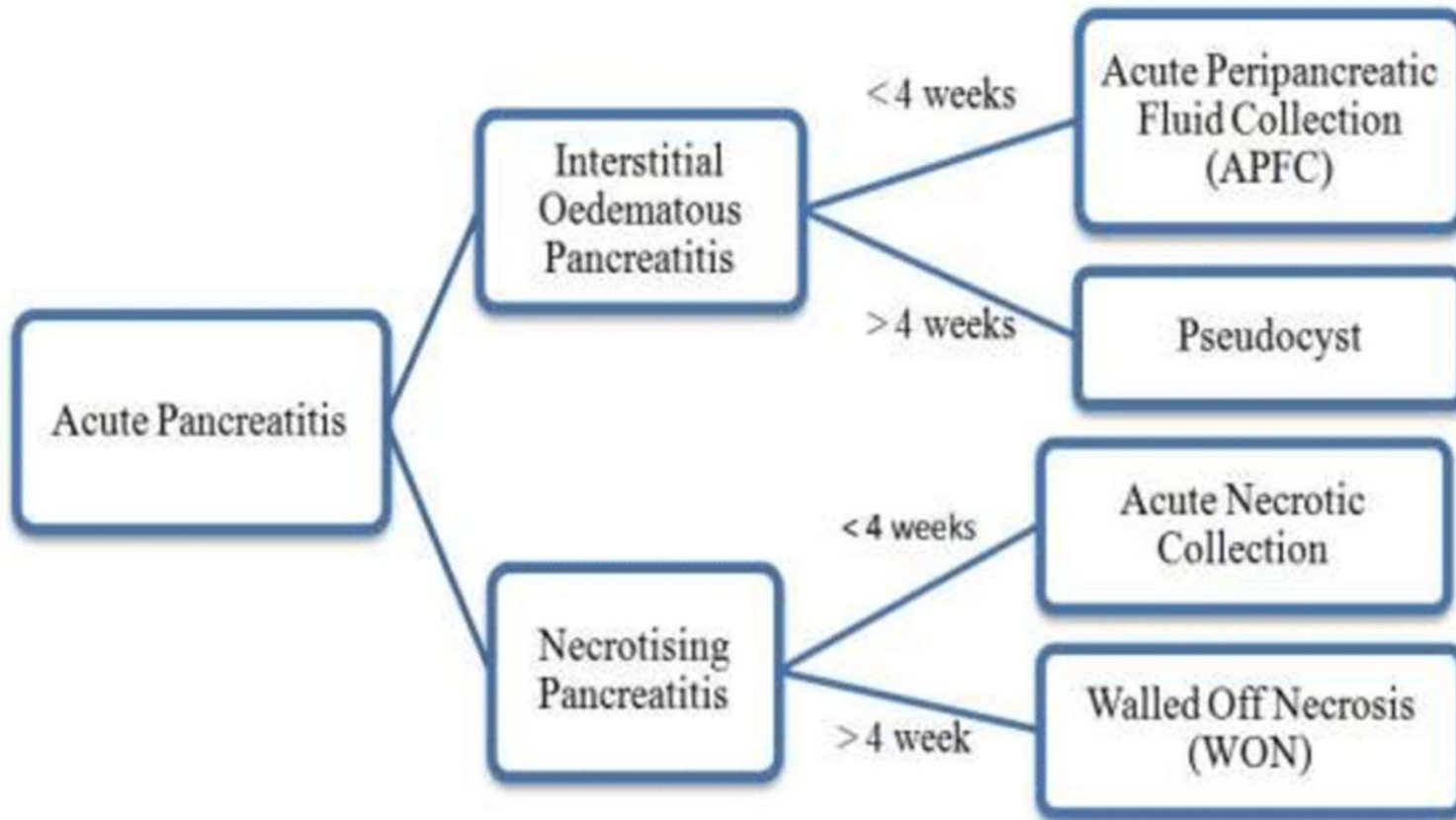


Clinical presentation/Recognition



- Acute abdominal pain following exposure to or contact with an inciting agent
 - Pancreatic pain:
 - upper or central abdomen radiating or penetrating to back
 - Associated with nausea/vomiting, or sudden collapse
 - Elevated serum pancreatic enzymes >3
 - lipase or amylase
 - Imaging: US, CT, MR

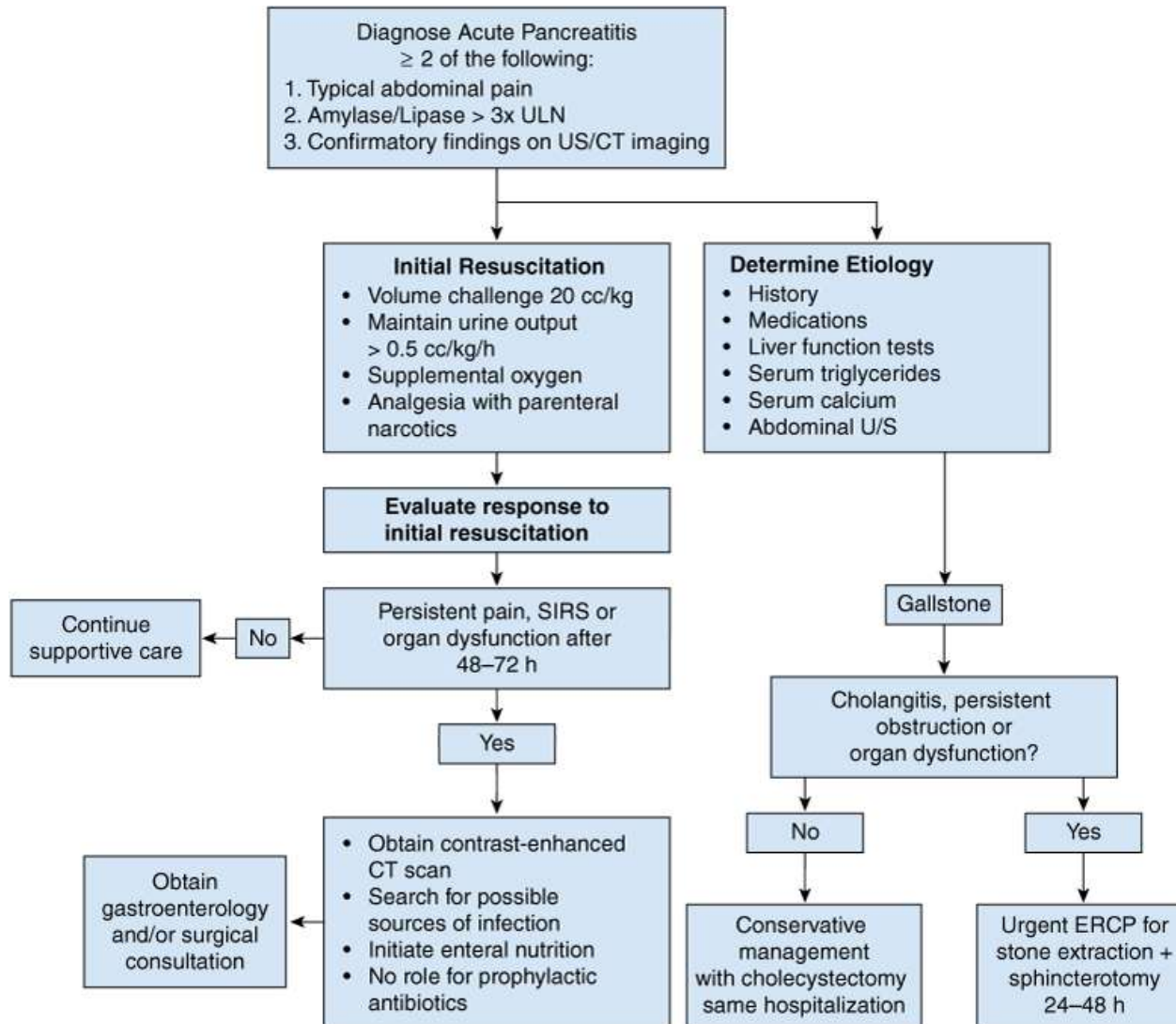






Immediate management

- Clinical recognition: pain/inciting agent, enzyme, imagin
- Stratify illness: presence or absence of organ dysfunction
- Resuscitation/stabilization
 - Governed by disease severity & patient's condition
 - Fluids: ringers or balsol best
- Analgesia as necessary: do not withhold morphine
- Ensure adequate oxygenation: supplemental oxygen often necessary
- Obtain necessary imaging or refer as appropriate



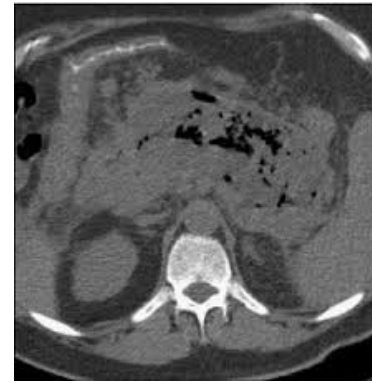
Source: McKean S, Ross JJ, Dressler DD, Brotman DJ, Ginsberg JS: *Principles and Practice of Hospital Medicine*: www.accessmedicine.com

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Take Home

- Acute pancreatitis a common acute medical/surgical emergency
- Most (70%) will be mild. 30% severe illness – 10% Critically severe
- Early recognition & appropriate stratification best strategy
- Mild, uncomplicated illness resolves within 7 – 10 days.
- ASP serious emergency: immediate intensive care admission + judicious resus
- Adequate, yet judicious fluids therapy with analgesia main stay
- Antibiotics for infected complications. Otherwise unnecessary
- Timely referral to appropriate expert saves lives.

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