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Clinical Review

ACUTE PANCREATITIS: UPDATES FOR EMERGENCY CLINICIANS

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☐ **Abstract—Background: Acute pancreatitis is a frequent reason for patient presentation to the emergency department (ED) and the most common gastrointestinal disease resulting in admission. Emergency clinicians are often responsible for the diagnosis and initial management of acute pancreatitis. Objective: This review article provides emergency clinicians with a focused overview of the diagnosis and management of pancreatitis. Discussion: Pancreatitis is an inflammatory process within the pancreas. While the disease is often mild, severe forms can have a mortality rate of up to 30%. The diagnosis of pancreatitis requires two of the following three criteria: epigastric abdominal pain, an elevated lipase, and imaging findings of pancreatic inflammation. The most common etiologies include gallbladder disease and alcohol use. After the diagnosis has been made, it is important to identify underlying etiologies requiring specific intervention, as well as obtain a right upper quadrant ultrasound. The initial management of choice is fluid resuscitation and pain control. Recent data have suggested that more cautious fluid resuscitation in the first 24 h might be more appropriate for some patients. Intravenous opiates are generally safe if used judiciously. Appropriate disposition is a multifactorial decision, which can be facilitated by using Ranson criteria or the Bedside Index of Severity in Acute Pancreatitis score. Complications, though rare, can be severe. Conclusions: Pancreatitis is a potentially deadly disease that commonly presents to most emergency departments. It is important for clinicians to be aware of the current evidence regarding the diagnosis, treatment, and disposition of these patients. Published by Elsevier Inc.**

☐ **Keywords—pancreatitis; lipase; imaging; gallstones; alcohol; fluids; necrosis**

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INTRODUCTION

Pancreatitis is the result of an inflammatory process within the pancreas, which is typically associated with abdominal pain, nausea, and vomiting (1,2). Most patients do not present with severe illness, though a small percentage of cases result in significant morbidity and mortality (3). Emergency department (ED) evaluation typically includes laboratory and ultrasound assessment, with management classically focusing on analgesia and resuscitation with i.v. fluids. This review provides an evidence-based investigation of the etiologies, pathophysiology, and ED evaluation and management of acute pancreatitis.

METHODS

We searched PubMed and Google Scholar for articles using a combination of the keyword *pancreatitis* and Medical Subject Heading "pancreatitis." The literature search was restricted to studies published in English. We decided

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which studies to include for the review by consensus. A total of 137 articles were selected for inclusion in this review.

DISCUSSION

Epidemiology

The incidence of pancreatitis has been increasing worldwide over the past few decades. It affects between 14 and 45 per 100,000 people and is the 21st most common cause of hospital admissions (4). Pancreatitis is also the most common admission diagnosis among gastroenterological complaints (5). Approximately 1% of all patients admitted to the hospital with the diagnosis of acute pancreatitis die during the admission (6). However, in patients who develop pancreatic necrosis, the mortality rate increases to 15%, which approaches 30% with infected pancreatic necrosis (6). The ability to predict those who will develop pancreatic necrosis remains elusive, but up to 15% of patients with pancreatitis may develop severe disease or pancreatic necrosis (5–7).

Pathophysiology and Etiology

Pancreatitis is an inflammatory disease of the pancreas wherein the pancreatic tissue autodigests itself. Cell damage initially occurs, resulting in trypsinogen activation and recruitment of macrophages and neutrophils. The activation of trypsin from trypsinogen leads to further cell injury, as well as activation of other digestive enzymes, ultimately resulting in massive destruction of pancreatic tissue. In most individuals who develop acute pancreatitis, this positive feedback loop stops spontaneously. However, in a select number of patients, the disease progresses and leads to a more serious systemic illness, resulting in diffuse pancreatic necrosis and potentially infection (6,8).

Gallstones may be the most common cause of pancreatitis, with studies estimating that gallstones are the culprit in 40–70% of cases (9,10). The underlying etiology is that pancreatic duct obstruction from gallstones increases the intrapancreatic duct pressure, which causes acid to reflux into the pancreas and activate trypsinogen inside the pancreas, leading to subsequent cell damage (11). Other causes of mechanical obstruction (eg, pancreatic cancer, sphincter of Oddi dysfunction, post-endoscopic retrograde cholangiopancreatography [ERCP]) likely have a similar etiology. Risk factors for developing post-ERCP pancreatitis include history of sphincter of Oddi dysfunction, female sex, younger age, and repeated attempts to cannulate the papilla (12). While less common than gallstones, it is also important to consider pancreatic cancer in patients with dilated biliary ducts without the presence of gallstones on imaging.

Alcohol ingestion is the second most common etiology, occurring in 25–41% of patients (2,13,14). Binge drinking is usually associated with pancreatitis 12–24 h after the cessation of alcohol intake (15). These patients often have a history of chronic alcohol abuse, defined as more than 5 years of heavy alcohol consumption of > 50 g (3.5 drinks)/day (2,16,17). Although the alcohol-naïve patient may have alcohol-induced pancreatitis, it is much less common in this group (18). The underlying mechanism for alcohol-induced pancreatitis is less understood and may have both genetic and environmental components (19). One proposed mechanism is that ethanol sensitizes pancreatic cells to cholecystokinin, thereby increasing trypsin production in the pancreas (20). There has not been an established threshold in which alcohol use will cause pancreatitis, and < 5% of alcohol-dependent persons will develop pancreatitis (15,21).

Hypertriglyceridemia is the third most common cause of pancreatitis worldwide, accounting for up to 10% of all cases and up to 50% of cases in pregnancy (22). Hypertriglyceridemia typically causes pancreatitis when triglyceride levels exceed 1000 mg/dL (23). Triglycerides themselves do not appear to be dangerous. Instead, it is the free fatty acids, created when pancreatic lipase breaks down triglycerides, which induce inflammatory changes in the pancreas (23).

Many medications have been hypothesized to cause pancreatitis. However, Table 1 includes only Class I medications that have been confirmed to be associated with pancreatitis (52,53). Additional causes of pancreatitis include hypercalcemia, fibrosis, autoimmune etiologies, toxins, scorpion stings, and congenital etiologies. No definitive cause is identified in up to 25% of cases (54).

History and Physical Examination

The classic presentation of pancreatitis is persistent upper abdominal pain (55). The pain may radiate to the back and is associated with nausea and vomiting in up to 90% of cases (55). Pain is often exacerbated by oral intake, so patients may present with anorexia (56). While only a small proportion of patients present without abdominal pain, the exact proportion that this comprises is not currently known (57,58). Most patients present with severe pain. However, the level of pain has not been found to correlate with the degree of clinical severity (2). When obtaining the history, it is also important to assess for risk factors, including both medications and family history.

Patients may present with abnormal vital signs. One study found that 41% of patients admitted to the hospital for acute pancreatitis met two or more systemic inflammatory response syndrome (SIRS) criteria within the first 24 h (59). Physical examination findings can include epigastric or upper abdominal tenderness. Late-stage

Table 1. Class I Medications Causing Pancreatitis (24–51)

Class I Medications
α-Methyldopa
All-transretinoic acid
Amiodarone
Azathioprine
Bezafibrate
Cannabis
Clomiphene
Codeine
Cytosine
Dapsone
Enalapril
Furosemide
Isoniazid
Ifosfamide
Lamivudine
Losartan
Pravastatin
Simvastatin
Hydrocortisone
Sulfamethoxazole
Sulindac
Tetracycline
Valproic acid
6-Mercaptopurine
Trimethoprim-sulfamethoxazole
Methimazole
Nelfinavir
Norethindrone/mestranol
Omeprazole

pancreatitis can result in skin discoloration around the umbilicus (Cullen's sign) or flank (Grey Turner's sign) from retroperitoneal hemorrhage (60). However, these findings are rare, with Grey Turner's sign found in 0.96% of patients and Cullen's sign present in 0.77% of patients (61).

Diagnosis

When considering the diagnosis of acute pancreatitis, clinicians should obtain a complete blood count, basic metabolic panel, liver function studies, triglyceride level, and lactate dehydrogenase level. In order to make a diagnosis of acute pancreatitis, clinicians should follow the Atlanta Criteria (1,62,63). This requires at least two of the following three conditions to be met: upper abdominal pain suggestive of pancreatitis (clinical), serum lipase or amylase more than three times the upper limit of normal (laboratory), or imaging findings of pancreatic inflammation (1,62,63).

Lipase levels more than three times the upper limit of normal have been previously found to be 100% sensitive and 99% specific for pancreatitis (64). However, a 2017 Cochrane Review challenged the notion that elevated lipase more than three times the upper limit of normal was infallible (65). The review found that up to 10% of patients may be wrongly diagnosed with acute pancreatitis (65). Moreover, up to 4.4% of patients with inflammatory bowel disease may have elevated lipase more than two times the upper limit of normal (66). Diabetic ketoacidosis, bowel obstruction, traumatic brain injury, and renal insufficiency also contribute to elevated lipase levels, but rarely reach levels meeting diagnostic criteria (67,68).

Although amylase is also commonly elevated in acute pancreatitis, it is less sensitive and specific than lipase (64,65). Moreover, amylase can confuse the clinical picture, as it may be falsely negative in pancreatitis induced by alcohol or hypertriglyceridemia (69,70). It is also secreted by the salivary glands, small intestine, ovaries, adipose tissue, and skeletal muscles. In isolation, amylase demonstrates a sensitivity of only 81%, and levels decrease more quickly than lipase, rising within 24 h and then returning to normal levels within 5 days (65,70,71).

Urinary trypsinogen-2 level is another possible diagnostic test that can be used to assess for acute pancreatitis, but it is not widely available (72). One study found that it is 72% sensitive and 90% specific for pancreatitis when values are > 50 ng/mL (65).

Imaging

Although computed tomography (CT) of the abdomen can identify pancreatic inflammation early in the disease course, it does not add to sensitivity of diagnosis and can be negative if performed too early (73–75). CT is not sensitive for diagnosing early pancreatitis, as patients with mild disease may not display imaging findings. A false-negative rate has been reported in up to 27% of cases (76,77). CT is useful in identifying pancreatic and extrapancreatic complications, such as peripancreatic fluid collections, estimating the degree of pancreatic necrosis, identifying pleural effusions, and assessing for the presence of organ infarction (78). Magnetic resonance imaging (MRI) is superior at differentiating soft tissue pathology and results in better evaluation of biliary and pancreatic ductal obstruction, though this does not need to be obtained in the ED (75,76). Direct pancreatic ultrasound has limited diagnostic or prognostic value in acute pancreatitis, as the study is frequently inadequate and, given the availability of MRI and CT, it does not add additional information (77,79). CT is more useful to detect complications of pancreatitis if the patient deteriorates or is not recovering at the expected rate, usually 72 h after symptom onset (62).

All patients without a clear etiology for their pancreatitis should receive a right upper quadrant ultrasound in the ED. If the patient has cholangitis or choledocholithiasis with common bile duct dilatation found on ultrasound, ERCP should be performed within 72 h to prevent further

clinical deterioration (2,80). The diagnosis of obstructing stone should be certain prior to ERCP, as complications from the procedure occur in 5–10% of patients (10,81). Magnetic resonance cholangiopancreatography (MRCP) and endoscopic ultrasound (EUS) are useful if there is uncertainty regarding the presence of an obstructing stone (82,83).

Treatment

Fluid resuscitation is a mainstay of therapy in acute pancreatitis. Patients often present after multiple episodes of nausea and vomiting and are volume-depleted. Release of inflammatory mediators also contributes to increased vascular permeability and third spacing of fluid (19). Inadequate fluid resuscitation after 24 h of hospital care has been associated with increased rates of pancreatic necrosis (84). Consequently, large volume fluid resuscitation is often performed in these patients.

However, large volume fluid resuscitation may not be necessary in many patients and has also been associated with an increased risk of clinical deterioration. A randomized controlled trial evaluated rapid (10–15 mL/kg/h) or controlled (5–10 mL/kg/h) fluid expansion after the diagnosis of severe acute pancreatitis (85). Although the rapid volume expansion group achieved their hemodynamic and laboratory parameters more quickly (13.5 h vs. 24 h), the rate of mechanical ventilation (94.4% vs. 65%), incidence of abdominal compartment syndrome (72.2% vs 32.5%), sepsis within 2 weeks (63.9% vs 37.5%), and survival rate (69.4% vs. 90%) were significantly worse in the rapid volume expansion group compared to the controlled fluid replacement group (85). The authors performed a similar study in 2010 that demonstrated worsened liver function and creatinine clearance in the group that received aggressive fluid resuscitation. Those patients also developed sepsis earlier and more frequently and were much less likely to survive to hospital discharge (66.1% vs. 84.7% survival) (86).

A 2011 prospective cohort study demonstrated that receiving < 3.1 L of fluid in the first 24 h was not associated with a poor prognosis, but receiving > 4.1 L of fluid volume during the initial 24 h was associated with the development of persistent organ failure compared to a smaller volume (87). In 2017, Singh et al. found a statistically significant increase in local complications and need for invasive interventions in patients who received > 4.3 L fluid in the first 24 h of hospitalization (88). The current data suggest a trend towards more moderate fluid resuscitation, which has been recognized by the American Gastroenterological Association Institute's 2018 guidelines that recommend "judicious goal-directed fluid therapy versus other methods," as goal-directed fluid resuscitation has not been shown to improve outcomes (89).

Antibiotics

Routine administration of antibiotics in patients with acute pancreatitis is not currently recommended (62). A Cochrane Review did not demonstrate a statistically significant mortality benefit with antibiotic prophylaxis, although imipenem/cilastatin was associated with a decrease in the development of infected pancreatic necrosis (90). At this time, routine prophylaxis is not indicated. However, patients presenting with cholangitis or another co-existent infection should receive appropriate antibiotics. Because severe acute pancreatitis may be indistinguishable from sepsis, empiric antibiotic treatment is recommended in toxic-appearing patients with a mixed presentation until infected pancreatic necrosis can be excluded (55).

Pain Control

Abdominal pain is often severe and should be treated in the ED. As pain is often reproduced early in the disease with oral intake, patients should be kept nil per os while pain is severe (91). Patients with mild disease who are likely to be discharged may be started on oral acetaminophen or opioids if pain is improved with these in the ED (92). For patients with more significant pain, starting with i.v. acetaminophen is reasonable, as i.v. acetaminophen was found to provide equivalent pain relief compared to i.v. tramadol (93). Other nonopioid analgesics, such as procaine and metamizole, have not been proven effective in a recent review (94). A ketamine drip at a rate of 3 μg/kg/min was used successfully to treat refractory pain in post-ERCP pancreatitis in one study, but further studies are needed to determine its efficacy (95). Thoracic epidural analgesia is effective in severe, intractable pain from acute pancreatitis, but is generally not performed in the ED (96). If non-opioid agents fail to provide significant relief, i.v. opioids such as fentanyl, morphine, or hydromorphone are effective and safe if used judiciously (55,94,97).

Nutrition

Bowel rest is no longer the standard of care, and early enteral nutrition is associated with decreased rates of infection and lower risk of complications compared to parenteral nutrition (98,99). Choice of nutrition is unlikely to affect the patient in the ED, but recent studies have shown benefit within the first 24–48 h of hospital course (100). In patients with mild disease in which discharge directly from the ED is being

considered, providing oral fluids in the ED is imperative to ensure patients will be able to tolerate liquids at home (101).

Hypertriglyceridemia

For patients with pancreatitis caused by severely elevated triglyceride levels, rapid reduction of elevated triglycerides to below 1000 mg/dL has been shown to improve recovery (102–104). A case series of 6 patients with pancreatitis and serum triglyceride level averaging > 3000 mg/dL suggested that a combination of insulin, heparin, and gemfibrozil may be effective (23). Insulin is hypothesized to stimulate the production of lipoprotein lipase, which breaks down lipids, while heparin mobilizes lipoprotein lipase that is bound in tissues to facilitate a rapid decrease in triglycerides (105,106). Both unfractionated heparin and low-molecular-weight heparin have been used effectively for this purpose (102). Gemfibrozil, niacin, and omega fatty acids are often given early in the course, but their importance in the acute lowering of triglycerides is not well-established (102,103). Plasmapheresis is another route of rapid clearance of triglycerides, but is more invasive and may have a comparable rate of clearance to insulin and heparin infusions (103,107). The data suggest that this is a safe and effective treatment for rapid clearance of hypertriglyceridemia-induced pancreatitis, but as there is no established protocol for this approach, early consultation with gastroenterology for optimal management is indicated in this population (108,109).

Fluid Choice

There remains controversy over which type of fluids should be utilized for resuscitation. Normal saline can lead to a hyperchloremic metabolic acidosis in large volume fluid resuscitation (110–112). This acidemic environment can increase the activation of trypsinogen, potentially exacerbating the disease (113). Therefore, balanced solutions (eg, lactated ringer's) may be preferable in this population (114–116). A small randomized controlled trial from 2011 demonstrated that the use of lactated ringer's solution compared to normal saline reduces systemic inflammation based on a decrease in the number of patients meeting SIRS criteria and decreased serum C-reactive protein (CRP) levels at 24 h (110). Colloids have not been shown to improve outcomes compared to crystalloids, and the American Gastroenterological Association cautions against overuse of colloids. Currently, packed red blood cells are recommended when the hematocrit falls below 25% and albumin if the serum albumin level drops below 2 g/dL (117).

Risk Stratification

Patients with pancreatitis often present early in the course of illness due to pain and can quickly deteriorate if they develop necrotizing pancreatitis. With a mortality rate for severe acute pancreatitis up to 30%, it is important to identify which patients will need closer observation and intensive care unit (ICU) admission (19). Many laboratory and vital sign parameters have been found to be correlated with poor outcome, including patients meeting SIRS criteria and those with persistently elevated CRP levels (118,119). Consequently, several risk stratification tools have been created in an effort to increase the accuracy for identifying which patients will have a poor prognosis. The three most commonly used risk stratification tools in the ED setting include Ranson criteria, Bedside Index of Severity in Acute Pancreatitis (BISAP) score, and Acute Physiologic Assessment and Chronic Health Evaluation (APACHE) II Scoring System. CT-based scoring systems are usually not relevant from the ED, as they are not routinely performed and have similar sensitivity for prognosis as clinical scoring systems (120–122). They may also underestimate severity in patients who present early in the course of disease, given the time interval required to develop significant findings on imaging (1,123).

Ranson criteria were originally developed in 1974 and were the mainstay in mortality prediction for several decades (Table 2) (124). There are five laboratory values designed to be evaluated in the ED, followed by 6 more that are to be assessed at 48 h after admission to predict mortality. A meta-analysis of 26 trials found a pooled sensitivity of 90% and specificity of 67% with a score > 2 at 48 h (125). There are few trials evaluating the sensitivity of the score upon admission, but an initial value of > 2 strongly warrants admission (124,125).

The APACHE II score was developed in 1985 in order to predict mortality among critically ill patients (Table 3) (126). In the validation study, it was found to have a sensitivity of 89% and specificity of 69% (127). Additional limitations include the large number of variables, need for a score calculator, and the use of arterial blood gas

Table 2. Ranson Criteria

Time of Admission	48 h after Admission
WBC > 16,000	Hematocrit drop > 10% from admission
Age > 55 years	BUN increase > 5 mg/dL from admission
Glucose > 200 mg/dL	Ca < 8 mg/dL within 48 h
AST > 250 U/L	Arterial pO_2 < 60 mm Hg within 48 h
LDH > 350 IU/L	Fluid needs > 6 L within 48 h

AST = aspartate aminotransferase; BUN = blood urea nitrogen; Ca = calcium; LDH = lactate dehydrogenase; pO_2 = partial pressure oxygen; WBC = white blood cells.

Table 3. Acute Physiologic Assessment and Chronic Health Evaluation II Score

History of severe organ failure or immunocompromise
Acute renal failure
Age
Temperature
Mean arterial pressure
pH
Heart rate
Respiratory rate
Sodium
Potassium
Creatinine
Hematocrit
WBC count
GCS
A-a gradient or PaO_2

A-a = alveolar-arterial oxygen gradient; GCS = Glasgow Coma Scale; PaO_2 = arterial partial pressure of oxygen; WBC = white blood cells.

(ABG) measurement. The ABG is needed to determine the PaO_2 or alveolar-arterial oxygen gradient (126).

The BISAP score was developed in 2008 and has several advantages when compared with Ranson criteria and the APACHE II score (Table 4) (118,128). These factors include simpler variables that are either present or absent, the ability to determine the score using data collected on initial presentation, and the requirement for only one laboratory value (blood urea nitrogen), which is frequently obtained early in the course. When compared with the APACHE II Scoring System, it performed similarly in predicting mortality, each having an area under the curve of 0.83, but in a meta-analysis, the BISAP score was found to be 76% sensitive and 87% specific (125,128). A score of > 2 correlates with a mortality of > 1.9% (128).

A cohort study of 87 patients in an Australian ED suggested that the BISAP score was better than Ranson criteria and APACHE II at predicting mortality in the ICU; in the ED, the BISAP score is non-inferior to APACHE II and may be helpful to determine whether a patient requires ICU care (129). Most of the prediction models were developed for use in the hospital floor or ICU setting, and there is a paucity of data evaluating these clinical prediction scores in the ED.

Table 4. Bedside Index of Severity in Acute Pancreatitis Score

BUN > 25 mg/dL
Altered mental status
> 2 SIRS criteria
Age > 60 years
Pleural effusion present

BUN = blood urea nitrogen; SIRS = systemic inflammatory response syndrome.

Age, comorbidities, and vital sign abnormalities are not present in all the scoring systems, but should also be considered as part of ED risk stratification. In a study evaluating SIRS criteria in patients with acute pancreatitis, nearly all patients who developed persistent organ failure, pancreatic necrosis, need for the ICU, and mortality had > 2 SIRS criteria on the first day of hospitalization (118).

Complications

Infected pancreatic necrosis can develop within a few days of symptom onset but is more common later in the course (130). This is a devastating complication, as it is associated with a 30% mortality rate (6). These patients will often present with a combination of tachycardia, hypotension, fever, and leukocytosis (55,131). Empiric antibiotics and ICU admission are essential in this patient group (6). In severe cases, surgical consultation should be obtained for surgical source control. This may include drainage of fluid collections that are thought to be a source of infection or necrosectomy, although this is less commonly performed as a first-line intervention (6,132). Hemorrhage can occur rarely when pancreatic enzymes are released, leading to damage to the surrounding blood vessels and resulting in life-threatening intra-abdominal bleeding.

Disposition

There is a paucity of data regarding discharge from the ED for patients with pancreatitis. A score developed by Whitlock et al. revealed increased risk of readmission within 30 days if any of the following are present at time of hospital discharge: unable to tolerate solid diet, gastrointestinal symptoms (defined by nausea, vomiting, or diarrhea), pancreatic necrosis, antibiotic use, or pain at discharge (133). Patients with the presence of 0–1 criteria in the validation cohort had a readmission rate of 5%, 2–3 criteria had a rate of 18%, and those with > 4 criteria had a readmission rate of 68% (126). Although used primarily for discharge after inpatient admission, the presence of oral fluid intolerance, severe gastrointestinal symptoms, and persistent pain is associated with worse outcomes and should prompt re-evaluation if planning to discharge (122,134).

While prognostic guidelines may be used to screen for patients who must be admitted, they are not useful for determining whether a patient is appropriate for discharge. Patients who are older with comorbidities, unable to take in food or liquids orally, have intractable pain, or have gallstones will likely require admission. Those who have vital sign abnormalities or are at increased risk of worse outcomes should be admitted to the ICU

for close observation and judicious fluid resuscitation. Close follow-up with a gastroenterologist is especially important for those patients who are discharged without an identified cause of their disease.

Consultations

Surgical consultation should be obtained from the ED for those patients who have gallstone pancreatitis, as early cholecystectomy is associated with decreased recurrence of symptoms (4,135,136). Gastroenterology consultation should be obtained in any patient who has liver dysfunction consistent with cholangitis or with a dilated common bile duct for possible EUS, MRCP, or ERCP (10,80). In patients whose symptoms resolve spontaneously, transient obstruction may have occurred, and ERCP may not be required (10). However, gastroenterology consultation is still recommended in this setting. If there is concern for alcohol abuse or dependence, discussion of cessation and referral to a counseling program is recommended (137).

CONCLUSIONS

The emergency clinician has an important role in caring for patients with pancreatitis. The diagnosis can usually be made by history, physical examination, and laboratory assessment with lipase elevation. While some patients may require a CT scan to evaluate for pancreatic inflammation, CT is often not helpful until later in the disease process, or if the patient presents critically ill. Patients should receive an abdominal ultrasound to evaluate for biliary etiology and to determine if there is common bile duct dilatation, which necessitates gastroenterology consultation for ERCP or MRCP/EUS. Treatment with fluids should be judicious, and careful reassessment is crucial, especially in patients who have the potential to become critically ill, as determined by scoring systems or clinical assessment. Pain control with i.v. opioids is likely safe in the short term. Risk stratification tools can assist with determining which patients can be discharged, hospitalized, or admitted to the ICU. Tolerance of oral feeding should be assessed prior to discharging patients with mild disease. Knowledge of pancreatitis is essential to ensure appropriate patient evaluation and management.

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ARTICLE SUMMARY

1. Why is this topic important?

Acute pancreatitis is a common gastrointestinal disease resulting in admission. Emergency clinicians are often responsible for the diagnosis and initial management of acute pancreatitis.

2. What does this review attempt to show?

This narrative review evaluates the emergency medicine diagnosis and management of pancreatitis, based on current literature.

3. What are the key findings?

Pancreatitis is an inflammatory process within the pancreas. The most common etiologies include gallbladder disease and alcohol use. Severe forms can have a mortality rate of up to 30%, though most cases are mild. Diagnosis requires two of the following criteria: epigastric abdominal pain, elevated lipase, or imaging findings of pancreatic inflammation. Once diagnosed, identification of an underlying etiology and obtaining a right upper quadrant ultrasound are key components. The initial management of choice includes fluid resuscitation and analgesia. Appropriate disposition is a multifactorial decision, which can be facilitated by the Ranson criteria or Bedside Index of Severity in Acute Pancreatitis score. Complications are typically rare, though they are severe if they occur.

4. How is patient care impacted?

Pancreatitis is a potentially deadly disease. Emergency clinicians must be aware of the current evidence regarding the diagnosis, treatment, and disposition of these patients.