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Research on Comprehensive First Aid Treatment of Severe Acute Pancreatitis

Haowei Ti[1.a.*]. Reda Barakat[2.b]. Yu Ti[3] and Xijie Zhao[4]
[1]Doctor of Clinical Medicine, Rey Juan Carlos University, Madrid, 28028, Spain
[2]Resident Physician,St.Martinus Hospital, Langenfeld, 40764, Germany
[3]Qiaotou Middle School, Dongguan, 523000, China
[4]Dalang Middle School, Dongguan, 523000, China
Email: [a*]cancer.drug@yahoo.com; reda.[b]barakat@stmartinus-langenfeld.de

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Abstract

The core of comprehensive emergency care for acute pancreatitis is early fluid resuscitation, organ-protective measures, and nutritional support. The primary task is to provide aggressive fluid resuscitation in the emergency department within 72 hours of onset (especially the first 12-24 hours), combined with analgesia, inhibition of pancreatic enzyme secretion, monitoring for complications, and early transfer of high-risk patients (severe SAP) to the ICU, following a treatment strategy that emphasizes both systemic and local aspects.

Keywords

ICU; Acute Pancreatitis; Emergency Treatment; Anti-Shock Treatment; Analgesia

1. Introduction

Acute pancreatitis is a sudden and severe inflammation of the pancreas. This inflammation occurs within pancreatic cells because the pancreatic digestive juices cannot flow through the pancreatic ducts and instead digest the pancreatic tissue itself, leading to inflammation.

Acute pancreatitis is a relatively common acute abdominal condition, ranking 3rd to 5th in incidence. More than 80% of patients have mild cases, namely acute edematous pancreatitis, which can be cured non-surgically. Approximately 10% of patients have severe pancreatitis, namely acute hemorrhagic necrotizing pancreatitis, where the inflammation of the pancreas is irreversible or self-limiting, often requiring surgical treatment. Furthermore, the mortality rate in these cases remains high, ranging from 30% to 60%.The main symptoms of the disease include abdominal pain, nausea and vomiting, and systemic symptoms (including fever and jaundice). A small number of patients experience a very rapid onset, and may have

no obvious symptoms or develop symptoms shortly before going into shock or dying; this is called sudden death pancreatitis or fulminant pancreatitis.

2. Severe Acute Pancreatitis

Severe acute pancreatitis is a dangerous acute abdominal condition with a mortality rate as high as 20% to 30%. Its main characteristics are pancreatic tissue hemorrhage, necrosis, and organ failure, often accompanied by systemic infection [1]. Within hours of the onset of acute pancreatitis, there is a large amount of inflammatory exudate around the pancreas (within the lesser omentum) and in the abdominal cavity, resulting in significant fluid loss, especially due to the "chemical burns" to the retroperitoneum caused by pancreatitis.Therefore, in a severe case of pancreatitis, the amount of fluid lost per 24 hours due to exudation around the pancreas, in the abdominal cavity, and in the retroperitoneum can reach 5-6 liters. Furthermore, due to paralytic ileus, vomiting, and the accumulation of intestinal contents caused by peritonitis, the daily loss will far exceed 5-6 liters [2]. In addition to fluid loss, a large amount of electrolytes is also lost, leading to an acid-base imbalance. Approximately 5-6 liters of fluid and a large amount of electrolytes need to be infused within 24 hours; if the infusion rate is too rapid, pulmonary edema will occur.Therefore, for large-volume intravenous infusions, while minimizing complications, comprehensive emergency measures should be taken. This includes monitoring central venous pressure (CVP) and urine output, and administering intravenous fluids based on changes in central venous pressure, urine volume, and specific gravity. Administering vasodilators such as 654-2, along with albumin to expand blood volume and reduce inflammatory exudation, is also recommended [3].

Local complications include pancreatic abscess and pancreatic pseudocyst; both require surgical treatment if necessary. During pancreatitis, systemic organ failure can occur, including circulatory failure, arrhythmias and heart failure, acute respiratory failure or acute respiratory distress syndrome, acute renal failure, gastrointestinal bleeding, disseminated intravascular coagulation, and pancreatic encephalopathy [4].

The most common symptom of acute pancreatitis is severe upper abdominal pain, a feeling of tightness in the abdomen, often accompanied by unconscious bending over to relieve the pain. The pain is often persistent and worsens after eating. Some patients experience nausea and vomiting, and some even vomit repeatedly to the point of collapse.

An acute attack of pancreatitis causes persistent, severe upper abdominal pain, often so severe that opioid injections are required. Approximately 50% of patients experience radiating pain down the back. The pain of cholelithiasis-related pancreatitis often has a sudden onset; in alcoholic pancreatitis, the pain develops gradually over several days. Abdominal pain usually lasts for several days. Sitting and leaning forward can relieve the pain, but coughing, strenuous activity, and deep

breathing may worsen it. Nausea and vomiting are common [5].

Patients with acute pancreatitis present with an acutely ill appearance and sweating. The pulse rate is usually elevated (e.g., 100 to 140 beats per minute). Breathing is shallow and rapid. Blood pressure may rise or fall transiently, accompanied by marked orthostatic hypotension. Initially, body temperature may be normal or even below normal, but can rise to 37.7 to 38.3°C within hours. Consciousness may be impaired or even dulled. Scleral icterus may occasionally occur due to inflammation and edema caused by gallstones obstructing the bile duct or the head of the pancreas. Lung examination may reveal signs of restricted diaphragmatic movement and atelectasis.Patients with moderate pancreatitis may experience intestinal obstruction, resulting in decreased bowel sounds and abdominal distension. Significant abdominal tenderness is present, almost exclusively in the upper abdomen. In rare cases, severe peritoneal irritation can cause a rigid, board-like abdomen. Rupture of the pancreatic duct may lead to ascites (pancreatic ascites). Grey Turner's sign and Cullen's sign indicate extravasation of bloody exudate. These two signs occur in <1% of cases and suggest a poor prognosis [6].

3. Comprehensive Emergency Treatment Plan

3.1. Fluid Resuscitation (Core)

Fluid Resuscitation (Core): Upon diagnosis, rapid intravenous fluid administration (balanced salt solution or lactated Ringer's solution is preferred) is necessary to correct hypovolemic shock and local inadequate perfusion. Early fluid allocation requires close nursing care, including monitoring urine output and blood pressure.

Fluid therapy is indicated for patients who present with severe hypovolemia within 72 hours of symptom onset and meet three or more of the following criteria:

1. Heart rate ≥120 beats/minute;
2. Mean arterial pressure ≥85 mmHg or ≤60 mmHg;
3.Blood lactate concentration ≥2 mmol/L, urine output ≤0.5 ml/(kg·h), hematocrit ≥44%;

Crystalloid solutions, primarily including normal saline and lactated Ringer's solution, act as buffers and diuretics. However, they have poor plasma volume expansion capabilities, requiring large volumes during resuscitation. Rapid infusion of large doses of normal saline carries the risk of hyperchloremic acidosis. Lactated Ringer's solution, with its ionic composition closely resembling that of the human body, is relatively safer for resuscitation.

Colloid solutions mainly consist of plasma, human serum albumin, whole blood, and plasma substitutes. The demand for colloid solutions in fluid resuscitation is relatively low. They are primarily used to rapidly restore effective circulating blood volume and propel fluid from the interstitial and third spaces into the

bloodstream. However, they may increase cardiac load, impair kidney function, and carry a risk of allergic reactions.

Human serum albumin should be prioritized in comprehensive emergency care to avoid affecting the kidney and coagulation functions. During fluid resuscitation, norepinephrine should be administered based on the patient's blood pressure, the resuscitation process, and post-resuscitation. Appropriate supplementation of vitamins and trace elements should also be considered to ensure fluid balance in patients with acute pancreatitis and improve the effectiveness of fluid resuscitation.

For patients with acute pancreatitis accompanied by shock or dehydration, administer intravenous fluids at a rate of 20 ml/kg over the initial 30–45 minutes after admission, followed by 5–10 ml/(kg·h) over the next 24 hours. For patients without dehydration, provide appropriate fluids. If the mean arterial pressure remains <60 mmHg, administer rapid intravenous fluids over 30 minutes while simultaneously using vasopressors to restore it to above 60 mmHg, subsequently maintaining the infusion rate at 5–10 ml/(kg·h). Assess the patient's fluid resuscitation needs every 6 hours during resuscitation; if blood urea nitrogen shows a decreasing trend or decreases, the resuscitation rate can be halved.

The following criteria indicate successful resuscitation: urine output > 0.5–1 ml/(kg·h), mean arterial pressure > 65 mmHg, central venous pressure 8–12 mmHg, central venous oxygen saturation ≥ 70%, heart rate < 120 beats/min, blood urea nitrogen < 7.14 mmol/L (if blood urea nitrogen > 7.14 mmol/L, it must decrease by at least 1.79 mmol/L within 24 hours), and hematocrit between 35% and 44%.

3.2. Inhibition of pancreatic secretion and analgesia

Somatostatin and its analogues are used to reduce pancreatic juice secretion, proton pump inhibitors are used to suppress gastric acid, and analgesics are administered.Most patients experience significant relief of abdominal pain after intravenous infusion of somatostatin or octreotide. For severe abdominal pain, meperidine can be administered intramuscularly at a dose of 50-100 mg. Morphine is contraindicated due to its potential to increase Oddi sphincter pressure.

Since organ ischemia due to insufficient blood volume is a significant factor in the development and progression of acute pancreatitis, abdominal pain and elevated amylase levels should strongly suggest severe pancreatitis. Patients should be closely monitored and receive intravenous fluid therapy according to the following protocol:

1. Administer sodium chloride and lactated Ringer's solution at a rate of 1–2 L/h to maintain urine output at 100–200 ml/h.
2. If urine output remains low after infusion of 2–4 L, insert a urinary catheter as soon as possible.

3. If urine output remains low, insert a central venous catheter for intravenous fluid administration and central venous pressure monitoring.
4. Depending on urine output and central venous pressure, the infusion volume should be 6–10 L/day, or even faster.
5. During the first 3 days, the patient can be given a cumulative total of 6-12 L of fluids and 600-1200 mmol of sodium chloride.

During subsequent treatment, changes in body fluids and sodium levels can be frequently observed, along with improvements in clinical symptoms (such as restoration of bowel motility, and a decrease in CRP and amylase levels).

During an acute attack of pancreatitis, strict fasting is required. Once abdominal pain subsides, bowel sounds return, and serum amylase levels return to normal, a pure sugar-free, fat-free liquid diet can be introduced, including fruit juice, rice water, lotus root powder, almond cream, vegetable juice, fruit juice, and jelly. After the patient adapts, vegetable puree and fruit puree, such as apple puree, banana puree, potato puree, and carrot puree, can be gradually added. Then, a very low-fat semi-liquid diet can be given, such as rice porridge, plain noodles, dried noodles, steamed buns, egg whites, soft vegetables, and fruits. For patients who cannot eat for an extended period, enteral nutrition should be initiated via nasogastric tube or nasojejunal tube, gradually transitioning from amino acid or short-peptide low-fat elemental diets to whole protein diets. Foods that stimulate gastric and pancreatic juice secretion, such as meat broth, chicken broth, fish broth, milk, soy milk, and egg yolks, should be avoided.

This process progresses from fat-free liquid diet → low-fat liquid diet → low-fat, low-residue semi-liquid diet → low-fat, low-residue soft diet → low-fat soft diet → low-fat diet → regular diet.

4. Treatment of Complications and Organ Support

Infection Control: Rational use of antibiotics that can penetrate the pancreatic barrier (e.g., carbapenems, quinolones combined with metronidazole).

Respiratory Function: Oxygen therapy or mechanical ventilation.

Renal Function: Continuous renal replacement therapy (CRRT) to remove inflammatory mediators.

Complications of acute pancreatitis can be classified as.

Local: Pancreatic and peripancreatic effusion, splenic vein thrombosis, pseudoaneurysm formation, and gastric outlet dysfunction.

Systemic: Shock, organ failure.

Enzyme-rich pancreatic juice may appear in the early stages of the disease, forming fluid accumulations in the pancreas and peripancreatic cavity. These accumulations may contain only fluid or both fluid and necrotic material. Most of these accumulations resolve spontaneously, but if they persist after about four weeks, a fibrous cyst may form, visible in cross-sectional imaging. This encapsulated fluid

accumulation is called a pancreatic pseudocyst or encapsulated necrosis, depending on its contents; a pseudocyst contains only fluid, while an encapsulated necrosis contains both fluid and solid necrotic material. Approximately one-third of pseudocysts resolve spontaneously. About one-third of patients with pancreatic necrosis experience infection of their fluid accumulations by intestinal bacteria, leading to extremely high morbidity and mortality rates.

Systemic complications of acute pancreatitis include acute single or multiple organ failure (e.g., cardiovascular and/or respiratory failure, acute kidney injury) and shock. This is at increased risk in patients with underlying comorbidities and/or persistent systemic inflammatory response syndrome.

In an appropriate clinical setting, SIRS is defined as the presence of two or more of the following :

Body temperature >38.3°C or <36°C

Heart rate >90 bpm

Respiratory rate >20 breaths/min or $PaCO_2$ <32 mmHg

White blood cell count >12,000/μL ($12*10^9$/L), <4,000/μL ($4*10^9$/L) or band neutrophils >10%

Organ failure is defined using the modified Marshall scoring system (see Table Modified Marshall Organ Failure Scoring) (2), which is based on laboratory and vital signs indicators of respiratory, renal, and cardiovascular impairment.Treatment of severe acute pancreatitis and its complications includes:

1. Intensive Care Unit (ICU) care
2. Enteral nutrition is superior to parenteral nutrition
3. Antibiotic treatment for extrapancreatic infections and infectious necrosis
4. Necrotization (removal of necrotic tissue) for the treatment of infectious necrosis
5. Endoscopic retrograde cholangiopancreatography (ERCP) for the treatment of acute pancreatitis and concurrent acute cholangitis. Patients with severe acute pancreatitis should be closely monitored in the ICU for the first 24–48 hours. Patients with worsening conditions or extensive local complications requiring interventional treatment should, if possible, be transferred to a pancreatic disease-focused treatment center.Patients with severe acute pancreatitis may require artificial nutritional support, but the optimal timing and duration of initiation remain uncertain. The 2013 guidelines from the American Gastroenterological Association recommend enteral nutrition, with parenteral nutrition only administered when enteral access is unavailable, intolerable, or fails to meet caloric requirements. Enteral access is preferred because it helps maintain the intestinal mucosal barrier, prevents intestinal atrophy that may result from prolonged fasting (and helps prevent bacterial translocation that can lead to pancreatic necrosis), and avoids the risk of central venous catheter infection.According to the 2013 ACG guidelines and the 2018 American Gastroenterological Association guidelines for the initial management of acute

pancreatitis, prophylactic antibiotics are not recommended for patients regardless of the type or severity of acute pancreatitis. However, antibiotics should be initiated if the patient develops an extrapancreatic infection (e.g., cholangitis, pneumonia, bloodstream infection, urinary tract infection) or infectious pancreatic necrosis.Patients with acute pancreatitis who show signs of worsening (such as fever, hypotension, tachycardia, altered mental status, and elevated white blood cell count) or who do not improve after 7 to 10 days of hospitalization should be suspected of having an infection (pancreatic or extrapancreatic). Most infections of pancreatic necrosis are caused by a single bacterium from the gut. The most common organisms are Gram-negative bacteria; Gram-positive bacteria and fungi are rare. In patients with infected necrosis, antibiotics known to penetrate necrotic pancreatic tissue, such as carbapenems, fluoroquinolones, and metronidazole, are recommended.For necrosis resection (removal of infected tissue), minimally invasive approaches are superior to open surgery and should be the first-line treatment. The 2013 guidelines from the American Gastroenterological Association recommend that, for patients with stable conditions, drainage of infected necrosis (radiological, endoscopic, or surgical methods) should be delayed, ideally for >4 weeks, to allow for liquefaction of the contents and the formation of a fibrous capsule around the necrotic material (encapsulated necrosis).In over 80% of patients with cholelithiasis-related pancreatitis, the stones pass spontaneously without the need for endoscopic retrograde cholangiopancreatography (ERCP). Patients with acute pancreatitis complicated by acute cholangitis should undergo ERCP early. Patients with mild biliary pancreatitis who resolve spontaneously should undergo cholecystectomy before discharge to prevent recurrence.

5. Anti-infective Treatment

For pancreatitis with no signs of infection, prophylactic antibiotics are not recommended. When signs of infection appear, antibiotics with a broad antibacterial spectrum and good pancreatic penetration are recommended.

Imipenem or meropenem is the first choice. Anti-infective treatment generally lasts about 2 weeks. During the course of treatment, cephalosporins can be used in combination with metronidazole or quinolones for anti-anaerobic purposes, in a step-down manner. If fungal infection is suspected, empirical antifungal drugs can be used.In cases of secondary infection in severe pancreatitis, broad-spectrum antibiotics such as injectable cefoperazone sodium and sulbactam sodium are required. For concurrent biliary tract infection, injectable imipenem and cilastatin sodium can be used. Anti-infective treatment needs to be adjusted according to drug sensitivity results, and the course of treatment usually lasts 7-14 days.

Somatostatin (SST) is mainly released by gastrointestinal endocrine cells and nerve endings, and is a multifunctional immuno-neuro-humoral regulatory peptide. In the absence of real-time monitoring of circulating SST levels, for MAP patients, an

intravenous infusion of SST 250 μg/h and octreotide 25 μg for 3 days should be administered immediately after onset. For MSAP and SAP, an intravenous infusion of SST 500 μg/h and octreotide 50 μg for 3 days should be given at the onset of the disease, followed by an intravenous infusion of SST 250 μg/h and octreotide 25 μg for 3 days; octreotide 100 μg should be injected subcutaneously three times daily for 3 days. This dose reduction regimen can bring serum SST levels closer to normal, helping to balance the body's inflammation and anti-inflammatory response. Randomized controlled clinical studies have shown that early, adequate replacement supplementation of this anti-inflammatory peptide can reduce the incidence of SAP by 23% and local pancreatic complications by 15%, thus significantly benefiting SAP patients.H2 receptor antagonists or proton pump inhibitors: These can indirectly inhibit pancreatic secretion by suppressing gastric acid secretion, and can also prevent the occurrence of stress ulcers. Famotidine 20-40 mg or omeprazole 40-80 mg can be added to the fluid for intravenous drip or intravenous injection, 1-2 times/day.

6. Conclusion

Early and thorough evaluation of patients with acute pancreatitis in the emergency department is crucial to assess the severity of local and systemic pancreatic reactions. Comprehensive emergency measures include determining the presence of concurrent infections and early complications, inferring the likelihood of progression to acute pancreatitis (SAP), and reducing the chance of disease deterioration through measures such as oxygen therapy, analgesia, fluid support, suppression of pancreatic secretion, control of inflammatory response, and appropriate anti-infective treatment. These comprehensive measures aim to prevent organ dysfunction and reduce the mortality rate of pancreatitis.

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