

Critical Shock-Related Acute Pancreatitis

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Abstract

The critical acute pancreatitis has recently emerged as the most severe acute pancreatitis. The shock is a complex pathophysiological process that often results in multiple organ dysfunction syndrome (MODS) and death. The MODS, though newly described, was observed in intensive care unit (ICU) patients for several decades. However, the cardiogenic shock-related acute pancreatitis is a rare event.

A 53-year-old Caucasian man with a critical shock-related acute pancreatitis (distributive and hypovolemic) was admitted in our ICU. He had a chronic pancreatitis medical history. He had alcohol drinking and cigarette smoking habits and a chronic HCV.

The patient developed a cardiovascular, a renal, respiratory MODS and a compartmental syndrome. The patient recovered over a few weeks with invasive support by trans-pulmonary thermo-dilution, with a cardiogenic/ hypovolemic shock; norepinephrine support; invasive ventilation support and dialysis (CRRT). Emergency operations were undertaken: a total colectomy (sigmoid ischemia), an ileostomy and a cholecystectomy. After 5-months internment in the hospital, with some nosocomial infections, the clinic evolution was good enough for the patient to leave the hospital with hemodynamic stability.

Patients with severe acute pancreatitis require intensive care. Within hours to days, a number of complications (e.g. shock, pulmonary failure, renal failure, gastrointestinal bleeding, or multi-organ system failure) may develop. The goals of medical management are to provide intensive supportive care, to limit infection and to identify and treat complications whenever appropriate.

Keywords: Critical Acute Pancreatitis; Multiple Organ Dysfunction Syndrome; Intensive Care Unit; Emergency Operations; Complications

Introduction

Recent U.S. estimates from the National Inpatient Sample report that acute pancreatitis is the most common inpatient principal gastrointestinal diagnosis [1]. The diagnosis is established by two of the following criteria: typical abdominal pain in the epigastrium that may radiate to the back, threefold or greater elevation in serum lipase and/or amylase, and confirmatory findings on cross-sectional abdominal imaging [1]. The critical acute pancreatitis (CAP) has recently emerged as the most severe acute pancreatitis. The shock is a complex pathophysiological process that often results in multiple organ dysfunction syndrome (MODS) and death. The MODS was observed in intensive care unit (ICU) patients for several decades. However, the cardiogenic shock-related acute pancreatitis is a rare event.

Case Report

A 53-year-old Caucasian man was presented to the emergency department of Cascais Hospital in Portugal with 2-day history of constant and umbilical pain accompanied by non-bloody diarrhea. He took paracetamol, without results. He hadn't any vomiting, nausea, anorexia, jaundice, choluria or acholia. On admission, the patient reported alcohol abuse and a past diagnosis of chronic pancreatitis,

chronic HCV and a history of a past HBV infection (serum *anti-Hbs* and Hbc antibodies were positive and *Ag Hbs* was negative). He had no relevant surgical, social or family history. His current medications were esomeprazole and oxazepam and no medications allergies were reported. During the physical examination, the patient was afebrile and with normal blood pressure. His gastrointestinal examination showed hypogastrium tenderness and his liver was enlarged, exceeding costal margin by 2 cm. His initial white blood count was 12400×10^6 cells/L, a C-reactive protein level of 0.20 mg/dL, a serum amylase of 555 IU/L, an aspartate aminotransferase (AST) of 88 IU/L, an alanine aminotransferase (ALT) of 56 IU/L and a LDH of 296 IU/L. The initial diagnosis was unclear, and a Computed tomography (CT) scan of the abdomen was performed, which revealed (Figure 1) acute pancreatitis with parenchymal necrosis and a peripancreatic fluid collection. The modified CT score was D (with an estimated mortality rate of 6% and an estimated rate of complications of 35%), but his bedside index of severity in acute pancreatitis (BISAP) was 0.

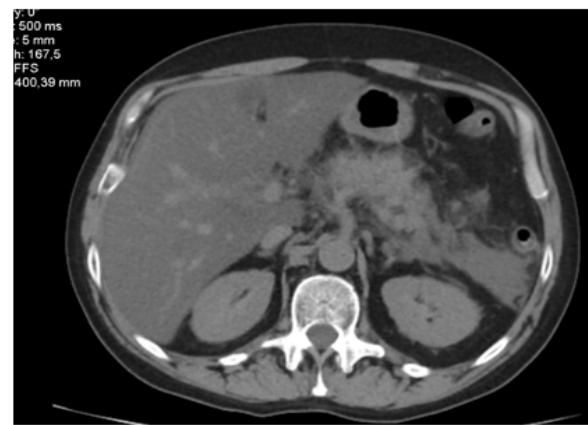


Figure 1: CT scan of the abdomen on admission.

Vigorous fluid resuscitation was started immediately, including pain control and bowel rest. However, 48 to 54 hours later, the patient rapidly evolves to shock requiring intensive care unit support.

After 48 hours, he was sub-febrile (37.8°C), the pulse rate was 118 per minute and a blood pressure of 146/108 mm Hg. The Glasgow coma scale was 13. Laboratory data showed high C-reactive protein, high amylase and lipase levels and renal dysfunction: white blood count 5500×10^6 cells/L, 50000 platelets, c-reactive protein 32.74 mg/dL; serum amylase 742 IU/L, lipase 6225 IU/L, a BUN of 53 mg/dL, an AST of 502 IU/L, a serum calcium of 8.4 mg/dL. The fluid sequestration was superior to 6 liters. The blood gases revealed metabolic acidosis with a pH of 7.29, pCO_2 32 mm Hg, pO_2 55 mm Hg, lactate 2.7 mmol/l, bicarbonate level of 18 mmol/L and SpO_2 of 84%.

After 54 hours, a critical shock-related acute alcohol pancreatitis was developed, a distributive and hypovolemic/cardiogenic shock, with cardiovascular, a renal, a respiratory MODS. At physical examination, he was unconscious, hemodynamically unstable (i.e. hypotensive [systolic blood pressure 60 mm Hg] and tachycardic [heart rate 130 beats per minute]) and with a central venous pressure (CVP) of 10 mm Hg. He had cool and cyanosis extremities and the skin was ashen. He was oliguric (urinary output 0.3 mL/kg/h), with an acute renal failure (cr 1.53 mg/dL) and a global respiratory insufficiency (pCO_2 55 mmHg; PO_2 60 mmHg), including high lactate levels (3 mmol/L) associated with neurological dysfunction, not controlled pain and hypoglycemia (30 - 50 mg/dL).

A PiCCO system was introduced with hemodynamic points that suggesting cardiogenic shock (See table 1). Consequently, several tests were performed. Laboratory data showed a troponin I of 0.51 ug/L, NT-pro-BNP (N-terminal pro b-type natriuretic peptide) was 13350 pg/mL and, creatinine kinase (CK) was 2118 UL/L. The electrocardiogram showed sinus tachycardia, with a HR of 150 bpm, PR interval of 120 ms, QRS duration of 80 ms. Chest radiography showed cardiomegaly and clear pulmonary fields. The transthoracic echocardiography was unclear. Therefrom, the cardiogenic shock etiology was a stress cardiomyopathy in which intensive stress caused rapid and severe heart muscle weakness.

	Day 0	Day 1	Day 2	Day 3	Day 4
Cardiac Index (CI) [N: 2.5 - 4 L/min]	1.79	2.4	2.79	4.83	3.18
Systolic Index (SI) [N: 41 - 51 mL/m ²]		19.1			
Sistemic vascular resistance index (SVRI) [N: 1200 - 1.800 dyn*seg*m ² /cm ⁵]	2858	1763	2178	530	2416
Stroke volume variation (SVV)	15		7	26	5
Extra-vascular lung water (EVLW) [N: 3 - 7 mL/kg]	8.8	7.9	8.3	11.9	
Intrathoracic blood volume (ITBVI) [N: 850 - 1000 mL/m ²]				866	712
Central venous pressure (CVP) [N: 12 - 15 mmHg, if ventilatory support]	11	10	11	16	
MAP (mean arterial pressure) [N > 70 mmHg]	60	70	75	60	96
GEDI (Global end-diastolic index) [N: 680 - 800 mL/m ²]		483	773		570
ScvO ₂ (central venous oxygen saturation) [N: 70 - 90%]		60			70
GapCO ₂ [N < 6%]		10			5

Table 1: Hemodynamic points obtained by PiCCO (Pulse Induced Continuous Cardiac Output) during 4 days at UCI.

Due to the patient's clinical instability, invasive support by trans-pulmonary thermodilution was required, as well as, volume expansion with 0.9% saline solution, nor-epinephrine support (vasoactive drugs) to elevate blood pressure, mechanical ventilation and dialysis (CRRT). He recovered within seven days and after that, his diet was slowly advanced.

During the hospital stay, some operations were undertaken. On the first days, the patient had had a compartment syndrome with an intra-abdominal pressure > 20 mm Hg, which determined a laparostomy with lavage and necrosectomy of pancreatic and intestinal segments. Two weeks later, the patient had developed gangrenous cholecystitis, mesenteric and hepatic ischemia and so, a percutaneous cholecystectomy and a sub-total colectomy were done. In consequence of sigmoid ischemia, a total colectomy was done three days later that. The peripancreatic fluid drainage was done, one month later.

On the 2nd admission month, the ascitic fluid cultures were positive for *Escherichia coli*, an *Enterococcus faecalis* and a *Candida albicans* and an intravenous piperacillin/tazobactam therapy was started plus Fluconazole, during 21 days.

Four months later, he had had an intra-abdominal collection that resolved with antibiotic therapy and with percutaneous drainage and also an *ESBL* bacteremia was treated with Meropenem and digital peripheral ischemia, result at distal phalanges (1st, 2nd, 3rd and 4th) amputation.

After five months, the clinic evolution was good enough for the patient to leave the hospital with hemodynamic stability. Probably because of the fragility of surgeries and the intensive care that were undertaken, the patient died a few months at home.

Discussion

Acute pancreatitis is a potentially severe disease (as, after all, around 75% of the cases are mild), however if deterioration is suspected, early ICU admission cannot be encouraged enough. The mortality rate is nearly 10%, if severe disease is not diagnosed [2]. In patients with more severe pancreatitis, based on the severity of their comorbid conditions, early organ system failure or substantial third space

fluid losses, admission to an intensive care unit (ICU) is appropriate [3]. Within hours to days, a number of complications may develop [2]. In these cases, mortality can approach 30% in patients with more severe comorbid conditions/with pancreatic necrosis, infection, or organ system failure. Necrotizing critical acute pancreatitis, as was seen in this patient, is often associated with transient or ongoing single or multi-organ failure, potentially requiring admission to the intensive care unit (ICU); repeated imaging, particularly when necrotic tissue becomes infected and repeated percutaneous, endoscopic or surgical drainage or debridement [4].

Appropriate fluid resuscitation is the most important treatment [3] and can be gauged by serial measurements of BUN, hematocrit and by urine output. Careful monitoring of progressive organ system failure and metabolic complications is critical in these patients.

Apart from the peripheral shock is more common in the acute pancreatitis course, the patient developed a cardiogenic/hypovolemic shock. The cardiogenic shock is a rare complication of critical pancreatitis. In these patients, the nor-adrenalin should be the first choice. Despite beneficial effects on hemodynamics, there is none benefit on prognosis. As a consequence of enlarging myocardial oxygen consumption and the fact of vasoconstrictors may impair microcirculation, as well as, tissue perfusion by catecholamines, their use should be restricted to the shortest possible duration and the lowest possible dose. Then, critical pancreatitis treatment can be controversial and we should rationally choose the best one. As well, we should pay more attention to clinical instability.

Despite several cases of acute pancreatitis cause multiple organ failures, this case report is an advice for the possibility of cardiogenic shock-related acute critical pancreatitis. As well, the availability of imaging exams to detect very critical illness. Initially, the patient didn't demonstrate a typical presentation of acute pancreatitis.

Conclusion

The diagnosis of pancreatitis is largely a clinical one based on physical signs and symptoms as well as serum levels of pancreatic enzymes. However, radiology plays important role in confirming diagnoses, evaluating the severity and identifying and managing complications of acute pancreatitis.

To sum up, the goals of medical management are to provide intensive supportive care, to limit infection and to identify and treat complications whenever appropriate. Critical pancreatitis remains a challenge for clinicians.

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