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Management of Hemorrhagic Pancreatitis: Review of the Current Literature

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ABSTRACT

Pancreatitis is the disease that affects the organ of the pancreas and should be given early attention, since its complications are quite lethal and can involve other organs. This research work provides a compilation of important information that expresses, through its development, the most common complications associated with pancreatitis, especially in its hemorrhagic presentation, as well as the necessary treatments used in this condition. In the course of pancreatitis, the first two to four days of symptoms are the most important because this is the period during which 15% to 25% of patients progress to its severe form.

According to clinical and experimental data, this period is characterized by an initial state of hypovolemia. The morbidity of severe acute pancreatitis is known to occur in two stages. The first two weeks are characterized by a systemic inflammatory response syndrome (SIRS), which results from the release of inflammatory mediators. Organ failure is common and usually occurs even in the absence of infection. The early mortality rate is 42 to 60%. The second stage begins approximately two weeks after symptom onset and is characterized by sepsis-related complications resulting from infection of pancreatic necrosis. Therefore, there is an association with systemic complications, such as pulmonary failure, renal failure, and cardiovascular failure, known as multiple organ failure syndrome (MOFS).

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INTRODUCTION

Acute pancreatitis (AP) can be defined as the set of disorders that appear after inflammation of the pancreas. It is a consequence of the intracellular activation of the enzymes that it produces and secretes and that cause cellular self-digestion of the gland and adjacent tissues due to coagulative necrosis. It is an infrequent disease, which obeys to different etiologies, with its two most frequent etiological forms being that coexisting with gallstones and that associated with chronic alcohol intake. Other studies have concluded that 20% of cases are idiopathic, 39% are associated with gallstones and 10% are associated with excessive alcohol consumption, the remaining 10% comprising other causes such as post-endoscopic retrograde cholangiopancreatography pancreatitis, pharmacological, traumatic and acute papillitis in 1%. The reason why the natural means of self-protection fail is unknown, since this does not occur under physiological conditions.[1,2]

Neither is the mechanism known by which, once this process has started, it sometimes progresses to cause necrosis of the pancreas and adjacent tissues, but in most cases only a mild, transient and self-limited inflammatory reaction occurs with appropriate treatment. digestive rest being essential for 24 hours. Patients who survive the acute process usually have pancreatic abscesses and pseudocysts as sequelae, they can suffer recurrences over time or also a chronification of the disease. Inflammation is rarely irreversible and lethal. When the course of the disease is fatal, a clinical autopsy is rarely performed to confirm the diagnosis, which is initially clinical, with analytical determinations and radiodiagnostic tests being useful.[3,4]

Such as computed tomography (CT) or endoscopic retrograde cholangiopancreatography (ERCP). Exceptionally, necrotic-hemorrhagic pancreatitis is a cause of death diagnosed in the forensic autopsy room.[4]

PATHOPHYSIOLOGY

In acute necrotizing-hemorrhagic pancreatitis, there is a large effusion of ascitic fluid and intense retroperitoneal edema, due to increased capillary permeability in the pancreas and surrounding tissues as a consequence of the release of vasoactive substances (kinins, histamine) by the injured pancreas , a situation that also leads to hypovolemic shock. The immediate cause of death in most cases is considered to

be the onset of hypovolemic shock due to Acute Necrotic-Hemorrhagic Pancreatitis, although as in other described cases of acute hemorrhagic pancreatitis, pulmonary edema was found during autopsy.[5]

Other relevant autopsy findings were esophageal and gastric erosions, hepatomegaly with hepatic congestion, hepatic fatty degeneration, and dilated intestinal loops. Acute pancreatitis is not a frequent disease in medical practice, and when it does occur it is usually mild and self-limiting, therefore it is not usual to proceed to the anatomopathological study of the pancreas. Although some studies suggest that if pancreatitis is mild, it often manifests with a complication, which can even be fatal. Exceptional cases of sudden death due to a hemorrhagic complication of asymptomatic pancreatitis, consisting of portal vein rupture, have been described.[5]

The compilation of the medical and clinical history prior to death is essential to guide the autopsy. The diagnosis of pancreatitis must be considered and ruled out in patients who report radiating abdominal pain in a "belt" and is accompanied by nausea, vomiting, fever, and upper abdominal distension or intestinal obstruction. In most cases, the macroscopic findings of the study of the pancreas were conclusive, finding extensive necrosis of the pancreatic tissue accompanied by diffuse hemorrhage of the gland, that is, findings of the most serious anatomopathological form of acute pancreatitis. However, one must not confuse the processes of cell necrosis that occur during pancreatic autolysis and that entail a series of changes in the organs and tissues with the appearance of consistent changes in reddish coloration, friability and softening, which when it begins in small areas of the pancreas can present gray-white areas and be confused with necrosis of the pancreas of vital origin.[6]

CLINICAL MANIFESTATIONS

Depending on the severity of the clinical picture, pancreatitis can be mild or severe. Two anatomopathological forms of acute pancreatitis have been described, the edematous form and the necrotic-hemorrhagic form. Mild pancreatitis is generally edematous and corresponds to 80-90% of cases, while severe pancreatitis is caused by pancreatic necrosis and occurs in the remaining 10-20%. Its mortality ranges from 40 to 70% and is related to etiology and age, being higher in people over 50 years of age 5% of patients with acute pancreatitis die from shock during the first week of the disease. [7]

Death usually occurs from neurological shock due to pain caused by glandular inflammation and/or hypovolemic shock resulting from vascular injury, vomiting, fluid retention in intestinal loops due to paralytic ileus, and increased capillary permeability. There is also release of toxins, related to the by-products derived from the action of activated pancreatic enzymes on the blood and tissues. Other causes of death are multiple organ failure, acute respiratory distress syndrome, acute renal failure, and disseminated intravascular coagulation.[7]

Necrotic-hemorrhagic pancreatitis is an unusual cause of sudden death, or rather unassisted death due to ignorance of the process. In mild cases of pancreatic inflammation, the patient may be asymptomatic or if he presents symptoms, usually by treating the cause that produces it, the inflammation subsides and it is not necessary to extract a sample for histopathological study. In cases in which subsequent complications such as abscesses or pancreatic pseudocysts appear, the process becomes chronic or a neoplasia is diagnosed, which requires a resection of the gland, this is partial and therefore there is no study of the gland in its whole.[8]

DIAGNOSIS

Taking an accurate clinical history and physical examination of the patient constitute the first essential link for the diagnosis of acute pancreatitis. Currently the diagnosis of acute pancreatitis is established with the presence of at least two of the following criteria:[9,10]

1. Intense epigastric abdominal pain suggestive of acute pancreatitis.
2. Serum lipase and/or amylase levels above 3 times their normal value.
3. Characteristic findings in imaging tests: CT, ultrasound or Magnetic Resonance. Initially, imaging tests are not indicated if the pain and lipase and/or amylase levels are elevated at the time of the diagnosis. In patients with typical pain but without this increase of lipase and/or amylase in blood, the diagnosis should be confirmed with imaging tests.[11,12]

In cases of severe pancreatitis, physical examination may detect signs of peritoneal irritation (Blumberg's sign, Murphy's sign, muscular defense, peritoneal reaction). Sometimes we can see cyanotic spots on the abdominal wall (Halsted sign), periumbilical ecchymosis (Cullen's sign) or on the flanks (Cullen's sign). Grey-Turner), by subcutaneous infiltration of exudates from the retroperitoneum. Less frequently they can be seen as tender subcutaneous nodules as a consequence of digestion of subcutaneous fat induced by circulating lipase. Sometimes we observe the loss of vision due to the so-called retinopathy. Purtscher syndrome, characterized by ischemia of the posterior pole, with whitish exudates and hemorrhages around the papilla.[13,14,15]

TREATMENT

At present, it is recommended to avoid any intervention in the first two weeks of severe acute pancreatitis due to high mortality in these patients. Pancreatic intervention should be delayed until septate necrosis develops (usually 3-5 weeks after symptom onset). Some indications for intervention include confirmed infection of the necrosis and persistent organ failure for several weeks with septate necrosis.[16]

Interventionism is indicated when there is suspicion or confirmation of a necrosis infection.[17]

The guideline of the International Association of Pancreatology indicates the need for intervention on pancreatic necrosis in cases following:

– Documented infection or clinical suspicion of infection of the necrosis with clinical deterioration. In these cases, infection by the presence of gas in the collections, by CT or NMR, or in obtaining positive cultures by puncture-aspiration with a fine needle (FNA). As we have already indicated, the germs involved are usually enterobacteria, mainly Escherichia coli, fungi, gram positive organisms such as staphylococci and anaerobes. Although empirically the most appropriate antibiotics are carbapenems, quinolones, metronidazole and cephalosporins, antibiograms in blood cultures or cultures of the liquid will indicate the precise antibiotic in each case.[18,19]

– In undocumented infection, when there is sustained organic failure or persistence of symptoms beyond 6-8 weeks. Clinical suspicion, even without microbiological confirmation, is established in situations in which there are clinical parameters (increased abdominal pain, temperature > 38.5 oC) and analytical parameters (markers of inflammation), or in cases of persistence of sepsis despite of a maximum and correct intensive treatment, after having ruled out other possible sources of infection and with the appearance of a new failure organic or maintenance/worsening of the already established one.[20,21]

Previously, the management of infected necrosis was surgical debridement where an open necrosectomy was performed. However, this approach began to be challenged and it was shown that when less invasive techniques are used, morbidity and complications decrease. Currently, a multidisciplinary approach known as "step-up" is recommended, in which one starts with minimally invasive techniques (such as percutaneous drainage, transgastric endoscopic drainage, and minimally invasive retroperitoneal necrosectomy) and escalates to more invasive interventions only if necessary. [22,23]

CONCLUSIONS

In conclusion, acute pancreatitis is an aggressive inflammatory process that can have regional and variable distance organ involvement. The local complications of pancreatitis are many and can result in a greater or lesser degree of morbidity and mortality or in the appearance of complications that can occur at any time during the course of the acute disease. Careful monitoring of patients taken to quickly recognize complications can ultimately change the course of the disease. The management of complications is guided by the identification and understanding of the specific complications and also on the knowledge and availability of specialists who treat patients with this disease.[24,25]

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