

Interval Open Necrosectomy: Case Report

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ABSTRACT

Background: Acute pancreatitis is caused by intrapancreatic enzymatic activation, this leads to inflammation and destruction of the pancreas, most patients have a mild edematous course with low mortality, infection of the necrotized pancreas is usually associated with a severe course and higher mortality. **Clinical case:** We present the case of a 47-year-old male presenting with acute necrotizing pancreatitis complicated with infection without organ failure. The purpose of this article is to present the role of open necrosectomy in the management and review of literature. **Discussion:** The main indication for drainage and debridement in necrotizing pancreatitis is the presence of infection or clinical deterioration in case sterile necrosis and should be postponed optimally > 4 weeks into the course of the disease for adequate limitation of the necrosis and improved physiological condition. **Conclusion:** While a step-up minimally invasive approach is recommended to open necrosectomy, this is not always possible; open necrosectomy still is a valid alternative for the management of the disease.

Keywords: acute necrotizing pancreatitis, open necrosectomy, case report, interval, covid.

BACKGROUND

Acute pancreatitis is the inflammation of the pancreas, secondary to enzymatic activation within the pancreas leading to destruction of its parenchyma, this results in an important systemic inflammatory response syndrome SIRS; cholelithiasis and alcohol are among its most

common causes.^{1,2,6} The diagnosis is made with 2/3 of the following criteria: characteristic abdominal pain, elevation of amylase or lipase 3 times its normal value or a compatible image^{4,5,6,7} While the majority of the patients have a mild course of the disease (80 %) with a mortality rate of about <1–3% , the other 20% will develop moderate to severe acute pancreatitis, this is associated with a mortality rate up to 35% in some series⁶ Infection of necrotizing pancreatitis occurs in about 40% of patients with severe acute pancreatitis⁶; infection in the presence of organ failure can result in an associated mortality of up to 34.2%, thus making this one of the main indications for drainage and debridement^{6,7}.

We present the case of a patient with a second episode of acute pancreatitis, first episode a few months prior without follow up due to the COVID pandemic.

CLINICAL CASE

J.S.G, 47-year-old male patient born and resident of Mérida, Yucatán, México; married and unemployed; without any relevant family history. He denies any smoking, alcoholic or drug use, up to date with all his vaccines; his personal medical history is positive for bipolar disorder and epilepsy, managed with Fluoxetine, olanzapine and valproate. Hipertension managed with captopril. Previous episode of mild acute pancreatitis in February 2020 released from hospital after pancreatitis remission without follow up and without cholecystectomy, no allergies or other surgical history.

He presents to the emergency department with history of generalized abdominal pain initiated 24h prior, associated with emesis on 2 occasions without fever; on exploration neurologically alert, no jaundice was evident on skin or mucosa's, abdomen with diffuse abdominal pain, predominantly epigastrium, with no rebound, normal capillary filling time; HR 118 RR 19 Temp 36.8° TA 124/80 mmHg TAM 94.667 SAT 91.0%; laboratories: Lipase 1903 Amylase 865.5 Leucocytes 15.86 Neutrophils 85.8, Hb 16.4 Hto 49 Platelets 198 Total bilirubin 1.45 direct bilirubin 0.88 and 0.57 indirect bilirubin, AST 147, ALT 88 , DHL 268 , GGT 285 , alkaline phosphatase 60, creatinine 0.9, normal electrolytes, we determine the presence of acute pancreatitis BISAP score of 2 classification at admission, CT imaging done 2 days after hospitalization with evidence of cholelithiasis , pancreas with associated peripancreatic fluid that reaches pelvis, heterogenous to contrast, pleural effusion compatible with Balthazar C.

On admission with adjust liquids avoiding overload, optimize analgesia and start diet at tolerance, on his first week of hospitalization he remains tachycardic and tachypneic without evidence of organ failure or fever. On the 8th day of hospitalization presents with a hypertensive crisis despite pharmacological management, tachycardia of 170 bpm and fever of 39°C (102.2 F) with leukocytosis $25 \times 10^9/\text{mL}$ and associated neutrophilia compatible with bacteremia, rest of laboratories on normal values.

We decide to restart NPO (nothing by mouth) , ICU determines no need of intensive care management, we draw blood cultures and get new CT scan with evidence of bilateral pleural effusion, acute pancreatitis with evidence of 30% necrosis and associated gas, we decide to start high spectrum antibiotics with generalized improvement shortly after, we restart diet at tolerance and antibiotic therapy continues with imipenem/cilastatine; on the 14th day of hospitalization, painless with adequate food tolerance, he persist with fever and mild tachycardia, negative blood cultures and decreasing leucocytes no organ failure, we decide to

perform a another CT scan with evidence of pancreatitis with > 50% necrosis and gas in parenchyma , severity index 10 points, Balthazar E.

Despite the evidence of persistent inflammatory response syndrome, the patient presented with adequate pain and oral tolerance and no evidence of organ failure was found, so we continued medical management and antibiotic therapy, liquids, and oral diet according to literature.

On 33rd day of hospitalization after 3 full antibiotic schemes, liquids, analgesia, and diet, with remission of leukocytosis, fever, normal vital sings no evidence of inflammatory response syndrome (SIRS) or organ failure, we discharged J.S.G with scheduled cholecystectomy and open necrosectomy after 4 weeks of pancreatitis remission.

On the day of readmission laboratories showed evidence of mild leukocytosis , grade II anemia with plaquetosis and normal lipase and amylase, no evidence of organ failure or new onset diabetes, control CT scan without changes from prior to discharge; we performed an open cholecystectomy and necrosectomy via an abdominal chevron incision with the following findings: atrophic gall bladder with multiple stones inside, thin wall, pancreas with purulent necrotic discharge, necrosis of approximately 50% of its body with firm stomach- pancreas adhesions ; we left 2 penrose drainages in the paracolic recesses and a Blake drainage at the level of Winslow foramen, after adequate postsurgical evolution and normal laboratory values we removed the drains and discharged him with an outpatient appointment for reassessment at 10 days postop , 1 month and 3 months postop, with evidence of normal clinical evolution and laboratory values.

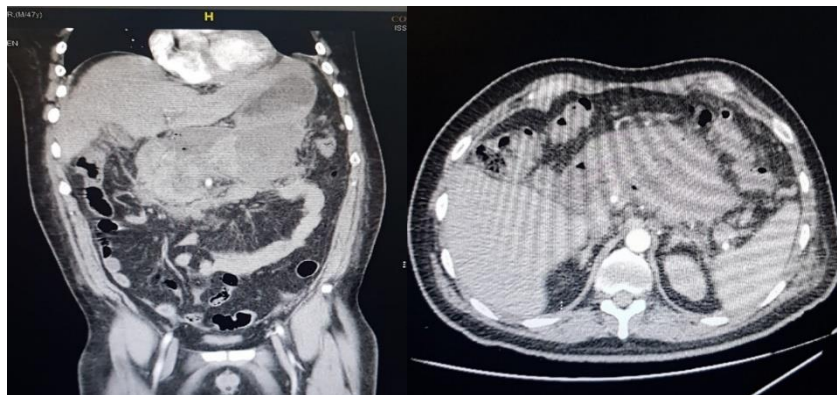


Figure 1 and 2, Necrotizing pancreatitis with > 50% necrosis, Balthazar E.



Fig 3, Chevron incision



Figure 4, Product of necrosectomy



Figure 5, Gallbladder with gallstones

DISCUSSION

Acute pancreatitis is the inflammation of the pancreas secondary to intrapancreatic enzymatic activation, this leads to acinar destruction and parenchymal inflammation, when this activation persists this produces autodigestion of the pancreatic parenchyma, as well as the surrounding fat and vascular structures, and is accompanied by an important systemic inflammatory response; among its most common causes are cholelithiasis and alcohol.^{1,2,6} Other less common causes are hypertriglyceridemia, hypercalcemia, trauma, ERCP, genetic and idiopathic, it's important to know the etiology to prevent further episodes^{1,2,6}

Global incidence increases as time passes, as seen in a recent metanalysis, in most occidentalized countries there was an annual increase of up to 3.07%.³ The most accepted classification is the modified Atlanta classification, created in 1992 and modified in 2012, classifies severity as mild, moderately severe and severe acute pancreatitis, depending on the presence of organ failure and local complications or worsening or previous systemic conditions, and it identifies two phases of the disease, early and late.⁴ Histologically, we subclassify acute pancreatitis in acute interstitial (edematous) pancreatitis and acute necrotizing pancreatitis^{1,4}; interstitial AP is not associated with organ failure or local complications and it usually resolves during the first week (early phase); the moderately – severe form of the disease usually presents with organ failure that doesn't last >48 hours, local complications or exacerbation of comorbid disease^{4,6}

For diagnosis, acute pancreatitis requires 2/3 of the following criteria: characteristic abdominal pain, an elevation of amylase or lipase 3 times its normal value and a compatible image^{5,6,7} Abdominal CT is the diagnostic imaging procedure of choice, particularly when evaluating the severity of the condition, it can detail the presence of local complications, necrosis and signs of infection, it's habitually performed around 72h from beginning of symptoms when it can show clinical predictors of severity or in case of clinical decline; earlier than 72h should only be performed in case of diagnostic uncertainty; subsequently in case of absence of clinical improvement or in case of upcoming intervention^{5,6,7}

Most patients will have a mild course (80-85%) that self resolves, with an associated mortality of < 3 %; however around 20% will develop a moderate to severe condition with a mortality rate of 13-35%.⁶ Local complications of acute pancreatitis can be subclassified in early (< 4 weeks): Acute peripancreatic collections for interstitial acute pancreatitis and acute necrotic collection for acute necrotizing pancreatitis; late (>4 weeks) pseudocyst in interstitial and walled-off necrosis in necrotizing.^{4,5} The severity of the condition attempts to be predicted with severity scores like Ranson, Apache II, Bisap, APACHE, Balthazar; APACHE II score on admission and 48 hours later help distinguish mild from severe pancreatitis and to predict death, could help selecting the patients that need early resuscitation and prompt referral in some cases^{5,6}.

Acute necrotizing pancreatitis is associated with a substantial mortality; SIRS produces hypovolemia-hypoperfusion that precedes organ failure, early resuscitation based on dynamic parameter with intravenous crystalloid solutions, like lactated ringers, are often recommended in a goal directed resuscitation, so intravenous fluid should be enough to normalize clinical and biochemical parameters of hypoperfusion : urinary output, lactate, hematocrit, etc.^{6,7,8} Among other recommendations enteral diet should be started as soon as it is tolerated, as this prevents bacterial translocation and infection of pancreatic necrosis , if necessary nasogastric or nasojejunal feeding tube should be installed and consider parenteral nutrition only if no other enteral route is achieved^{6,7}; prophylactic antibiotic therapy in presence of sterile necrosis is not recommended and should only be started in the presence of suspected or confirmed infection with a positive culture or gram test with a sample taken with guided fine needle aspiration or in presence of indirect signs of infection like gas in pancreatic parenchyma on CT imaging, specially in the presence of clinical evidence of sepsis; choice antibiotics are carbapenems , quinolones, and metronidazole as it has good pancreatic necrosis penetration^{6,7}

Patients with evidence of severe pancreatitis or persistent organ failure (> 48 h) should be treated in the intensive care unit preferably^{2, 6,7} Indication for surgical drainage and debridement is the presence of pancreatic necrosis infection or clinical deterioration in case of sterile necrosis^{6,7} other indications are symptomatic walled-off pancreatic necrosis, intractable abdominal pain, bowel obstruction; however, this should be done preferably after 4 weeks of remission, for this allows adequate limitation of the necrosis and necrotic collections.^{2, 6,7,8}

Currently minimally invasive measures like percutaneous drainage guided by CT or ultrasonography, endoscopic drainage with transmural debridement and placement of self expandible stents with irrigation, VARD or video assisted retroperitoneal drainage, and laparoscopic transgastric debridement are part of the so called "Step-up approach"^{7,8,9} that consists of escalating minimally invasive procedures if the initial intervention is unsuccessful, and are gradually becoming the standard of care but could take multiple sessions to achieve complete debridement^{2,7,8,9} Open necrosectomy involves exploratory laparotomy with blunt debridement of all visible necrotic pancreatic tissue, it can achieve a complete debridement in one procedure, but due to its associated morbimortality , around 22%¹⁰ in a recent cohort of 109 postop open necrosectomy patients over a period of 12 years , its considered by some as obsolete; from this study they observed that certain conditions prior to the procedure increased mortality, such as age > 60 y/old , preexisting conditions, prolong organ failure prior to the surgery , < 4 weeks of remission, organ failure with important leukocytosis and organ

failure as indication for surgery; in presence of multiple factors, mortality could reach up to 50% after open necrosectomy and in absence of them it could be as low as 0%¹⁰, so the patients general condition prior to surgery determines outcome more than the procedure itself and it highlights the importance of adequate medical management prior to surgery.¹⁰

CONCLUSION

Up to 40% of patients with necrotizing pancreatitis will present infection of the necrotic tissue, in absence of organ failure or clinical worsening, an optimized medical approach followed by interval surgical resolution is recommended, this could be done with an open approach without added morbi-mortality.

Disclaimers: The views expressed in the submitted article are my own and not an official position of the institution.

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