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Acute Pancreatitis: A Short Review

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Mini Review

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Keywords: Acute Pancreatitis; Epigastric; Alcohol

Abbreviations: GBD: Global Burden of Disease; ERCP: Endoscopic Retrograde Cholangiopancreatography; AST: An aspartate Transaminase; WSES: World Society of Emergency Surgery; NSAID: Non-Steroidal Anti-Inflammatory Drugs.

Mini Review

To expose the global importance of this disease, in 2012, an American study determined an estimated and annual cost of 2,6 billion dollars for inpatient costs as well as being the most common gastrointestinal cause for hospitalization [1]. In addition, based on the Global Burden of Disease Study 2019 (GBD 2019), there was an incidence of 2,814,972 cases globally and 115,053 deaths due to acute pancreatitis [2]. Although, in 1990 compared with 2019 there was a global decrease of 8,4% in incidence and 17,2% in associated mortality [2].

Acute pancreatitis characteristic pain has been described as localized in the epigastric, irradiated as a belt with preference to the left side, extremely painful, and worsens with alcohol and food intake [3]. Even more, the pain is described as sudden and an extremely violent onset, continuous, excruciating, and often collapsing, sharing with myocardial infarction the annihilating sensation or imminent death that usually accompanies it [3]. Furthermore, it can present with facial blush (not presented in other abdominal pains) and pleural effusion especially left-sided [3].

Since the 2012 revision of the Atlanta classification and definitions, acute pancreatitis diagnosis is established when 2 of 3 features are present: characteristic abdominal pain, elevated serum lipase o amylase activity ≥ 3 times the normal upper limit, and characteristic findings on contrast-enhanced computed tomography, magnetic resonance or ultrasonography [4]. Other relevant definitions exposed are types: interstitial oedematous and necrotizing, organ failure: a complication of 2 of 3 systems; cardiovascular, renal, or respiratory (calculated by the Marshall score), that can be transitory if it is less than 48 hours or persistent if it is more than 48 hours, severity: mild (no complications), moderately severe (transitory organ failure or local complication) and severe (persistent organ failure), phases: early (1 week) in which local complication can be: acute peripancreatic fluid collection or the acute necrotic fluid collection and late (> 1 week) with local complications such as pseudocyst, wall-of necrosis or infected wall-off necrosis [4].

Regarding the etiology, gallstones are the paramount cause and must be ruled-out before assuming other causes [5], representing about 40-50% of cases, and alcohol-related represent 20-30% [6]. Although, a mnemonic to recall other etiologies is: "IGETSMASHED": Idiopathic, Gallstones, Ethanol, Trauma, Steroids, Mumps (and other microorganisms: Mycoplasma spp., Cytomegalovirus, Epstein-Barr, Mycobacterium avium complex, Human Immunodeficiency Virus, Legionella spp. or Ascaris), Autoinmune, Scorpion and Snake venom, Hypertriglyceridemia and Hypercalcemia, Endoscopic retrograde cholangiopancreatography (ERCP) and Drugs (more than 500) [7,8].

To establish the etiologic cause, clinical context, anamnesis, and physical examination must be the priority [8,9]. Even though, as previously mentioned, the biliary cause must be excluded. An aspartate transaminase (AST) elevated more than 3 times the normal upper limit or greater than 150 UI/L has a positive predictive value of 95% for biliary disease [10]. An abdominal ultrasound must be performed to look for cholelithiasis, microlithiasis, or dilatation of the biliary tree, other possible finding could be a tumor [6,9,11]. Later on, a history of alcohol consumption could be associated with and a measurement of triglycerides. If it is less than 1.000 mg/

dL could be pancreatic inflammation, and above 1.000 mg/dL (or more strictly 2.000 mg/dL) can be considered as the etiologic cause [9].

Furthermore, have been linked factors to mortality and severity, such as, older than 59 years [12] and more than 2 comorbidities, especially congestive heart failure, peripheral vascular disease, cerebrovascular disease, moderate or severe renal disease, and metastatic tumor ($p < 0,05$) [13]. Obesity with a body mass index (BMI) ≥ 30 kg/m2 in non-Asian countries and ≥ 25 kg/m2 in Asian countries had RR of 2,20 (CI: 1,82-2,66) for mortality and 2,168 (CI: 2,09-3,43) for incidence of local complications [14].

Treatment

After the Revised Atlanta classification of 2012, there are 4 guidelines to highlight. The most recent one is by the French Society of Anaesthesia and Intensive Care Medicine (Société française d'anesthésie et de réanimation: SFAR) with 24 authors and 24 recommendations [6]. Secondly, the World Society of Emergency Surgery (WSES) in 2019 includes 19 specialists (mostly surgical) from Finland, Italy, Paraguay, Canada, The Netherlands, the USA, the UK, and Israel, and addressed 54 recommendations [11]. Thirdly, in 2018 American Gastroenterological Association Institute was constituted by 5 gastroenterologists and wrote 9 recommendations [15]. Fourth and finally, in 2015 by the Japanese Society of Hepato-Biliary-Pancreatic Surgery, which included medical and surgical specialists and external review by an internist, general surgeons, and the public, and has 43 recommendations [9].

Fluid resuscitation has been one of the most important therapeutics in pancreatitis and is recommended to all patients [6,9,11]. SFAR recommend 3 to 5 mL/kg/h in the first 24 hours [6]. Japones guideline recommends if signs of shock to administer 150 to 600 mL/h and if it has no signs of dehydration 130 to 150 mL/h and then as needs to maintain a mean arterial pressure more than 65 mmHg and a diuresis more than 0,5 mL/kg/h [9]. Finally WSES exposes that it must be guided by its hemodynamic profile and must be frequently evaluated [11]. In 2022 the waterfall trial was performed, and it compared two groups in the first 24 hours: In 2022 the waterfall trial was performed, and it compared two groups in the first 24 hours: aggressive hydration (bolus of 20 mL/kg followed by 3 mL/kg/h and moderate hydration (if hypovolemia a bolus of 10 mL/kg if no hypovolemia no bolus, followed by 1,5 mL/kg/h), which as primary outcome being the development of severe or moderately severe pancreatitis, and showed no difference between the groups (RR: 1,28, 95% CI: 0,77-2,12), but it did show fluid overload associated in the aggressive group (adjusted RR: 2,85, 95% CI: 1,36-5,94) [16].

Nutrition must be one of the therapeutic pillars because of the risk of malnutrition that accompanies it and the association with morbidity and mortality [17]. The patient must initiate oral nutrition as soon as clinically tolerated [6,15] specially after abdominal pain control [9], nausea and vomiting [15]. If not clinically tolerated, enteral nutrition must be started with a nasogastric tube in the first 24 to 72 hours, which is not inferior to nasojejunal tube [6,15,17]. Nutrition is a key component whereas enteral nutrition is preferred over total parenteral nutrition, as confirmed in a meta-analysis that identified a 50% reduction in overall mortality (CI 0,28 - 0,91) and 82% in the severe pancreatitis group (CI 0,23-0,65) 61% in infection rate (CI 0,23-0,65), 45% in multiorgan failure (CI 0,37 - 0,81) and 66% in need of surgery (CI 0,29-0,67) [19].

As for pain management, no guideline established a recommendation with a superior intervention [9,11], whereas it may be related to food intake and lessening in suffering. A meta-analysis with the need for rescue analgesics as their primary outcome, demonstrated that opioids and non-steroidal anti-inflammatory drugs (NSAID) are equally effective (OR 0.56, 95% CI 0.24 to 1.32, $p = 0.18$) [18].

Antibiotics must be initiated only in infected pancreatic infection, established by a positive culture, or suspected by radiologic findings such as pancreatic or peripancreatic gas or a positive procalcitonin (especially in a deteriorating patient) [6,9,11]. There is no place for prophylactic antimicrobial therapy [9,11,15]. The most common microorganisms are *Escherichia coli*, *Klebsiella* spp, *Pseudomonas* spp, *Proteus* spp, *Acynetobacter* spp, and not uncommonly gram positive and fungi (*Candida* spp.) [6,11]. This last microorganism, in a meta-analysis showed a relative risk of 3,59 (CI: 2,36-5,46) for mortality [19]. Even more, in any guideline is a recommendation of a specific antibiotic above another [6,9,11], the most used because of their coverage on gram negative and some on anaerobes are carbapenems, piperacillin/tazobactam, quinolones: moxifloxacin and ciprofloxacin and metronidazole [11].

Finally, in regard to surgical intervention, in the presence of a colelithiasis as the etiologic cause, a cholecystectomy must be performed before discharge [6,9]. ERCP has utility only in cholangitis scenery [6,9,11]. Additional pertinent surgical interventions could be performed in the presence of abdominal hypertension, hemorrhage (if there is no endovascular management option), intestine ischemia, and fistule drainage in an intrapancreatic collection [11].

To conclude, acute pancreatitis may present with an open range of severity and has no specific treatment. In every situation, clinical history must insist on searching for the probable etiologic cause. Using the Atlanta classification

on type, phase, severity, organ failure (Marshall score), local/systemic complications and prognostic scale can determine: mortality, morbidity, chronological state of the disease, presentation, image requirement, need for intensive care unit, and additional management. There is outstanding significance to uncover how much endovenous hydration the patient needs, start oral or enteral nutrition at least 72 hours, if there is pain, always treat it, and the specific indications for antibiotic treatment.

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