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Prognosis of Acute Pancreatitis: It's a Challenge

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Abstract: Introduction: acute pancreatitis is a mild to very severe inflammation of the pancreas, presenting a broad clinical spectrum and significant risk of morbidity and mortality. Objective: to determine whether base and lactate deficiency are predictive factors in patients with severe acute pancreatitis. Method: retrospective, longitudinal, observational and descriptive study in patients with acute pancreatitis in the Surgery Department of a second-level care hospital. Results: 65 patients with acute pancreatitis: 39 women 60%, 26 men 40%. Higher incidence of age from 20 to 29 years. Of which with severe pancreatitis: 23 (35%) and mild: 42 (65). The excess of base predicting severity with BISAP scale/risk of organ failure with Marshall scale yields sensitivity of 44.4% and a specificity of 75.9% and 38.9% and 75.9% respectively, so 55.6% and 61.1% of patients with acute pancreatitis. Discussion: currently, there are multiple scoring systems to assess the severity of acute pancreatitis. The revised Atlanta classification is a clinically proven, widely used, and universally accepted classification system. To identify the severity of acute pancreatitis early, with stratification of the patient's risk on admission, distinguishing between mild and potentially fatal forms. Allowing rapid intervention with aggressive therapies in severe cases, reducing complications and mortality. Conclusions: markers such as baseline deficit and lactate are predictive factors in patients with acute

pancreatitis, they have been shown to be non-specific, but with a certain degree of sensitivity determining the severity of the patient.

Keywords: Acute pancreatitis, Base deficit, Lactate, Predictive markers, BISAP scale, Marshall scale, Pancreas.

INTRODUCTION

The human pancreas is a lobed, pale yellow retroperitoneal organ, weighing between 150 and 200 g in adults, with a horizontal length of 12 to 15 cm. Its anteroposterior diameter varies from 1 to 3 cm, and its height varies from 4 to 8 cm, gradually tapering towards the tail. **[1]** Physiologically the pancreas is an exocrine and endocrine gland, which develops embryologically from the fusion of two anterior bowel vaginations; this organ is fixed in position within the abdominal cavity, supported by the posterior wall of the abdomen by its connections with the duodenum and excretory ducts. **[2]** As a result, pancreatic surgery presents serious challenges in terms of the risk of intraoperative hemorrhage due to the complex anatomy and dense vascularization, a common difficulty in both open and minimally invasive procedures. **[3]**

As far as history is concerned, around 320 B.C., Herophilus of Chalcedon, a Greek anatomist, dissected more than 600 corpses, where he discovered and gave the name to the duodenum, described the pancreas but did not identify it as a glandular organ. **[4]** Its anatomical location behind the stomach, colon and mesentery, small size and soft yellowish consistency were sufficient reasons for its late discovery as an independent, glandular organ. **[5]** In the sixteenth and seventeenth centuries the first information on the anatomy and physiology of the pancreas is documented, Johann Georg Wirsung in 1642 describes the pancreatic duct that bears his name. The intervention of the pancreas in digestion began to be protocolized in the eighteenth and nineteenth centuries, with the discovery of lipase in 1815 by the Englishman Alexander Marcet (1770-1822), in addition, Claude Bernard described between 1849-1856 the pancreatic exocrine function, which is why he was considered the father of pancreatic physiology. **[6]** In 1901 Opie proposed his theory of the common canal by discovering the sphincteric muscle of the duct, suggesting that a stone can produce obstruction of the ampulla of Vater. **[7, 8]**

In the nineteenth century it is pointed out that the pancreas plays a role in carbohydrate metabolism, but it was not until 1921 that Frederick Banting, Charles Best, managed to isolate insulin. In addition, acute pancreatitis was identified as a clinical-pathological entity in the nineteenth century, but the diagnosis and treatment even today is uncertain due to certain roughness. **[9]**

It is defined as "acute pancreatitis is a sudden inflammation of the pancreas that can range from mild cases to more severe forms with necrosis and systemic complications, presenting a broad clinical spectrum with a significant risk of morbidity and mortality." **[10]** And it is a major cause of morbidity and health resource utilization, with hospitalization costs exceeding $30,000 per patient in the United States. **[11]**

Most patients develop mild and self-limiting pancreatitis of the disease, between 15% and 25%, if they progress to moderately severe or severe acute pancreatitis. These advanced forms are associated with systemic complications, persistent organ failure, and

have mortality rates that have been documented to reach 30%. **[12, 13]** Advances in understanding the pathophysiology, diagnosis, and treatment of acute pancreatitis have improved outcomes; however, the heterogeneity of the disease and the expansion of therapeutic options has become a complex/confusing skein, which fortunately are more successful that allow for better treatment and consequently a better prognosis. **[14]**

In acute pancreatitis, gallstones and alcohol consumption are the main causes, and a smaller, but significant, number of cases are due to metabolic problems such as hypertriglyceridemia and hypercalcemia, among many other causes. **[15]** In the East, alcohol is the main etiology 45-55%, followed by idiopathic pancreatitis 20-30%, and gallstone-related disease 15%-20%. The mean age of presentation is typically younger: 35-45 years. compared to Western data, with a marked male predominance (male/female ratio of 4:1 to 7:1. **[16]** In addition, other causes of pancreatitis include autoimmune pancreatitis, which is a distinct form of chronic pancreatitis stigmatized by specific imaging features, including diffuse or focal pancreatic enlargement and irregular narrowing of the main pancreatic duct. **[17]** Acute pancreatitis following endoscopic retrograde cholangiopancreatography is the complication with the highest incidence and clinically relevant to the potential risk of morbidity of this procedure. While mechanical, hydrostatic, and chemical lesions have traditionally been linked to its pathogenesis, recent studies highlight additional mechanisms, such as microvascular dysfunction, pancreatic steatosis, and calcium and calcineurin-mediated acinar injury. **[18]** In addition, immune checkpoint inhibitor-related pancreatitis has been reported, with an incidence ranging from 0.5% to 5.7% due to irreversible pancreatic toxicity and damage, so risk-benefit should be considered. **[19]** It is also important to detect eosinophilic pancreatitis, which accounts for <1% of pancreatitis cases, poses significant diagnostic challenges due to its heterogeneous manifestations ranging from mild to life-threatening, with only >10/field eosinophils per biopsy being the only form of diagnosis. **[20]** However, data for alcohol-related pancreatitis predominated and were associated with a higher likelihood of the composite endpoint of in-hospital complications than biliary pancreatitis. These findings suggest a region-specific etiological pattern and indicate that alcohol-related disease may contribute substantially to the caseload. **[21]**

Regarding the pathophysiology of acute pancreatitis, stress hyperglycemia is a well-established phenomenon in acute diseases. Hyperglycemia >140 mg/dL has been reported in 33%–73% of patients in an intensive care unit and 60% of patients with acute myocardial infarction. Very limited data are available, but it is estimated that 60% of the incidence is at 60%. **[22]**

OBJECTIVE

To determine whether laboratory reports, specifically baseline deficiency and lactate, are predictive predictors in patients with acute pancreatitis.

METHOD

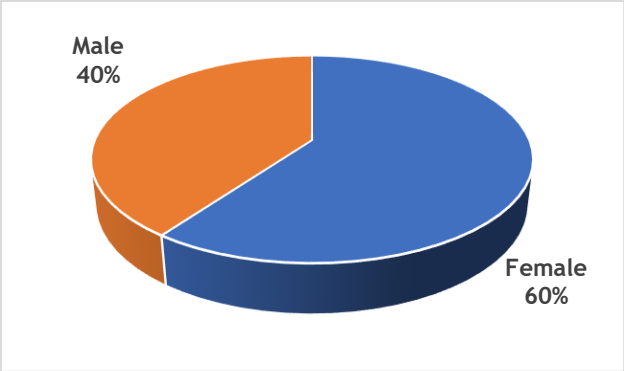
This is a retrospective, longitudinal, observational and descriptive study of the Surgery Service of the La Villa General Hospital in Mexico City, a second-level health care hospital. In a study period that spanned from January 2024 to January 2025. With the use of the

Epidat program, patients who meet criteria for the diagnosis of acute pancreatitis according to Atlanta 2018. The statistical analysis is carried out with the Statistical Package for the Social Sciences system. In addition, multivariate logistic regression is performed to analyze the factors that influence death, and odds ratios (ORs) and 95% confidence intervals (95% CI) are shown. The value of P < 0:05 will be considered significant. While continuing to carry out the operational principles of the Declaration of Helsinki for the development of medical research.

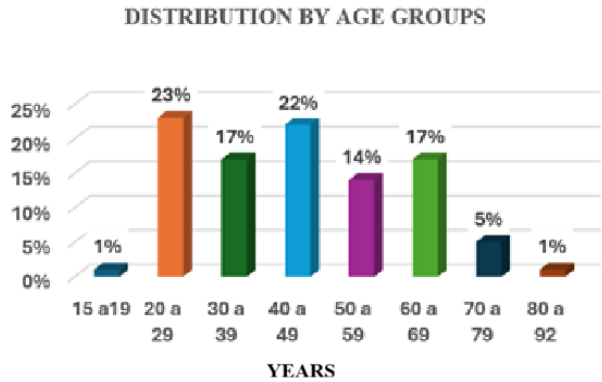
RESULTS

The clinical records of a total of 95 patients were reviewed, of which only 65 met inclusion criteria. In relation to gender, there was a higher frequency of acute pancreatitis in female patients in a total of 39 patients (60%), compared to the male gender with 26 patients (40%), **see graph 1**, without division according to severity or presentation of any organ failure. According to age, the range of presentations of this pathology corresponded to 20-29 years (23%), followed by the age group between 40-49 years (22%), with less frequency of presentation in the age group of 15-19 years and 80-92 years (1%). **See graph 2.**

Distribution By Sex



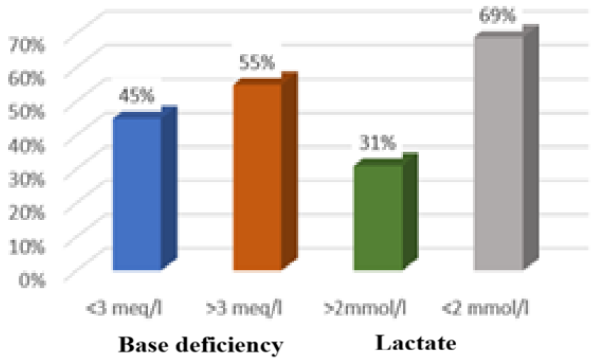
Graph 1: Gender Frequency in patients with acute pancreatitis



Graph 2: Age frequency in patients with acute pancreatitis

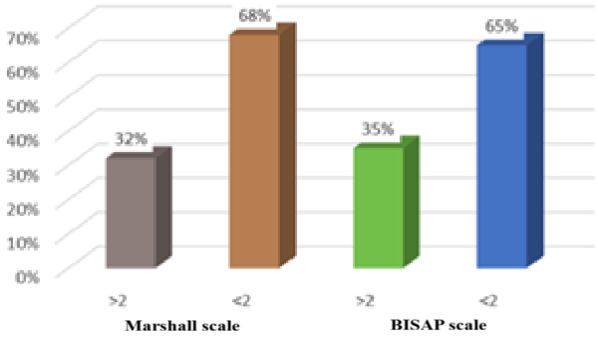
A division was made into groups with baseline deficits <3 and >3; the first group included 29 patients (45%) and the second 46 patients (45%), in the same way patients with

lactate levels >2mmol/l and <2mmol/l were organized into groups, including within the first group 20 patients (31%) and in the second 45 patients (69%). **See graph 3.**



Graph 3: Frequency of patients with acute pancreatitis with lactate levels <2 or >2 mmol/L and base deficit <3 or >3 meq/L

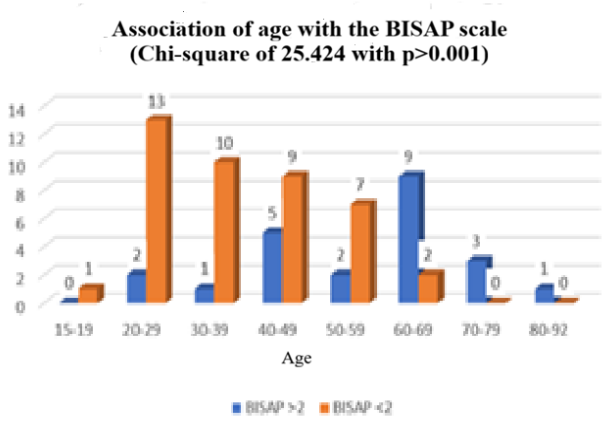
On the other hand, of the total number of patients who presented a score greater than two points within the BISAP scale, which indicates severe pancreatitis, a total of 23 (35%) were among those patients with mild pancreatitis in a total of 42 (65); among the main alterations were elevated urea levels and the presence of data of systemic inflammatory response. likewise, those patients who presented a score of >2 within the Marshall scale; which is cataloged as patients with some organ failure, with a total of 21 patients and 44 patients who had a presentation of acute pancreatitis without organ failure. **See graph 4**. Among the main organ failures is respiratory, with a Kirby index (PaO2/FiO2) between 201-300 mm Hg in a total of 9 patients, as well as acute renal failure in a total of 9 patients with a range between 1.9-3.6 mg/dL in creatinine levels.



Graph 4: Frequency of patients with scores >2 and <2 on the Marshall and BISAP scales

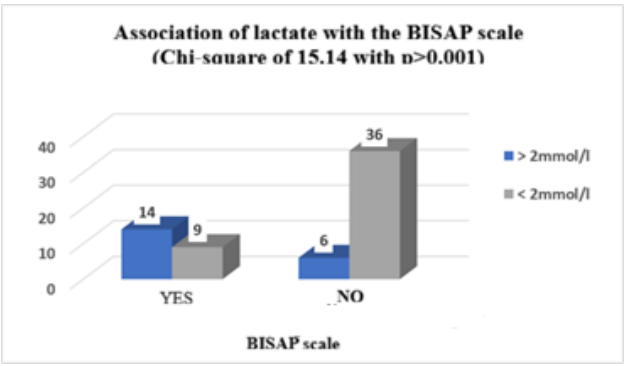
Associating factors such as age and sex, with respect to the BISAP and Marshall scale, it was possible to determine that these factors with a Chi-square of 7.799 (age) and .047 (sex) with a p of 0.351 and 0.829 respectively, were not statistically significant, therefore, it does not show an association with the Marshall scale. indicating that they are not decisive to indicate the risk that a patient with acute pancreatitis has of suffering from some organ failure during the time of its evolution. However, when determining the association between age and the BISAP scale, a Chi-square of 25.424 with a p<0.001 was obtained, being statistically significant, to determine that, according to the age of presentation of acute

pancreatitis, there is a greater risk in terms of its incidence, within the study, in the age range of 60-69 years. followed by 40-49 years old. **See graph 5**. With respect to sex, no association was demonstrated, between this and the risk of developing severe acute pancreatitis, with a chi-square .908, with a p of 0.341, which is not statistically significant.



Graph 5: Association of age with the BISAP scale with a value of p <0.05.

The BISAP scale and the levels of base excess >3 meq/l, a chi-square of 2.89 was obtained with a p of 0.89, which is not statistically significant according to the values determined for p, on the other hand according to the levels of base excess with the Marshall scale a chi-square of 1.59 with a p of 0.20 was obtained. which was not significant, determining that the levels of base excess have no association with the BISAP and Marshall scales, as well as the risk of presenting severe acute pancreatitis or some organ failure. On the other hand, within the lactate levels >2 or <2 mmol/l and its association with the BISAP and Marshall scale, a chi-square of 15.14 was obtained with a p of <0.001, which was statistically significant, having an association between levels >2 with the BISAP scale and the risk of severe acute pancreatitis. **See graph 6**.



Graph 6: Association of lactate levels with BISAP scale with a value of p<0.05

When making an association between lactate levels with the Marshall scale, a chi-square of 2.12 with a p of 0.15 was obtained, which is not statistically significant, and there is no association between lactate levels and the presentation of organ failure in patients with acute pancreatitis. **See graph 7**.

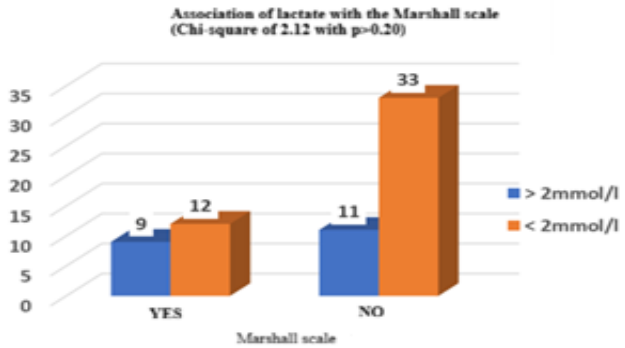


Chart 7: Association of lactate levels with the Marshall scale with a p value <0.05

Multivariate analysis with logistic regression: the predictive parameters of pancreatitis severity determined by BISAP scale and lactate levels had a significance of p > 0.15, which is not statistically significant, however, an OR of 7.6 was obtained; that is, with lactate levels > 2mmol/l there is a 7.6 times greater probability of presenting severe pancreatitis. with a score >2 on the BISAP scale, which coincides with the data demonstrated with the chi-square. On the other hand, when determining the baseline deficit as a predictor of severity, a significance of p < 0.08 was obtained, which is statistically significant, with an OR of 0.231 and an inverse of 4.32, so that an increase in the base excess >3 meq/l is 4.3 times more likely to present a score >2 on the BISAP scale. of developing severe pancreatitis.

The association of the Marshall scale with lactate levels had a p significance of 0.387 which is not statistically significant, with an OR of 0.484 with an inverse of 2.066, that is, with lactate levels > 2mmol/l there is a 2 times greater probability of presenting severe pancreatitis, with a score >2 on the BISAP scale, while the levels of base excess with the Marshall scale a significance of p 0.660 was obtained, which is not statistically significant, with an OR of 1.074, which has no association between the levels of base excess and the risk of presenting a score >2 on the Marshall scale or of presenting organ failure.

The sensitivity and specificity obtained (70% and 80% respectively) with lactate levels and the BISAP scale tell us that it is a study that can be used as a method of identification of patients who have a high probability of presenting severe pancreatitis during their evolution, however, 30% of patients who presented moderately severe or severe acute pancreatitis had normal lactate levels. However, with respect to the Marshall scale, lactate levels show a sensitivity of 45% and a specificity of 73.3%, which is why it is not a factor that helps determine the risk of organ failure in patients with acute pancreatitis. According to the excess base as a predictor of severity with the BISAP scale or the risk of presenting organ failure with the Marshall scale, a sensitivity of 44.4% and a specificity of 75.9% and 38.9% and 75.9% respectively were obtained, so that 55.6% and 61.1% of patients with acute pancreatitis with a score >2 on the BISAP or Marshall scale, They will have a normal result in the levels of base excess, clearly indicating the need to use other more sensitive markers, in order to establish the risk of severe pancreatitis or organ failure in these patients.

DISCUSSION

Four phases of the pathophysiology of acute pancreatitis have been detected: intracellular, intra-acinary, pancreatic and their systemic effects. **[23]** Severe acute pancreatitis involves

dynamic interactions between immune dysregulation and inflammatory infiltration, both of which are still unclear; leading to a life-threatening gastrointestinal emergency characterized by progressive pancreatic necrosis and intra-abdominal collections, a high-intensity systemic inflammatory response without any regulation. **[24, 25]** Acute pancreatitis initially behaves sterilely and may mimic a diagnosis of "sterile abdominal sepsis," which results in a large systemic inflammatory response, multiorgan failure, and death, resulting from aseptic chemical peritonitis, from irritants that are distributed in the abdominal cavity. Inflammation of the peritoneal serosa at its beginning sterile and fatal. **[26].**

The diagnosis of acute pancreatitis was confirmed with three parameters: clinically, elevation of pancreatic enzymes to more than 3 times baseline (serum lipase/amylase) and by imaging with computed tomography. According to the revised Atlanta classification, the severity of acute pancreatitis is divided into three grades: mild, moderately severe, and severe, which is a result of organ failure and local and systemic complications that the patient presents. **[13, 27, 28]** Radiologic or imaging methods, such as detailed parameters and optimal times for dynamic contrast-enhanced computed tomography or magnetic resonance imaging, as these criteria can vary from site to site and their homogenization is difficult in a matter of acute pancreatitis imaging. **[29]**

Although most cases are mild and self-limiting, approximately 20 to 30% progress to severe acute pancreatitis, where the complications of acute pancreatitis are diverse, complex in their diagnosis, in their treatment, evoking a delayed response and/or with permanent sequelae: divided into local and systemic; **[30]** Because infected pancreatic necrosis is fatal and requires aggressive surgical management or transabdominal percutaneous drainage represents a promising alternative to conventional drainage, **[31]** however, aseptic pancreatic necrosis resolves spontaneously with supportive care, intraperitoneal rupture and hemorrhage represent rare but devastating complications. **[32, 33]**

Pancreatic pseudocysts are encapsulated accumulations of fluid with a well-defined inflammatory wall, which usually develops between four and six weeks and have an incidence of 5% to 16%.**[34]**

Patients with acute pancreatitis, treated with opioids for pain, most develop physiological dependence on these drugs. Have opioid withdrawal syndrome with similar symptoms. It will be important to differentiate the benefits related to opioid withdrawal treatment from those related to pain management. It will also be important to understand whether buprenorphine treatment allows for greater patient self-care and decreases hospitalization rates. **[35]**

Exocrine pancreatic insufficiency, on the other hand, can occur due to acute pancreatitis, affecting one in five patients in the long term. Alcoholic etiology and necrosectomy are two causes of risk for induction. **[36]** Another observation is chronic pancreatitis as a repetitive result of a causal etiologic factor or by misdiagnosis or poorly defined diagnosis, estimating that 50% to 90% of patients with chronic pancreatitis may develop it, so lateral pancreatojejunostomy, or the surgery known as the Frey procedure, is the gold standard as its treatment. **[37]**

Currently, there are multiple scoring systems to assess the severity of acute pancreatitis. The revised Atlanta classification is a clinically proven, widely used, and

universally accepted classification system. **[38, 39, 40]** This is indisputable with the goal of early identification of the severity of acute pancreatitis, allowing stratification of the patient's risk upon admission, distinguishing between mild and potentially fatal forms. Allowing rapid intervention with aggressive therapies in severe cases, reducing complications and mortality and ensuring management in intensive care or specialized centers, without delay in treatment. **[41, 42]** Estimating disease severity, including the Acute Physiology and Chronic Health II (APACHE II) Assessment, the Simplified Acute Physiology Score (SAPS II), the Sequential Organ Failure Assessment (SOFA), and the Bedside Acute Pancreatitis Severity Index (BISAP) score. APACHE II is widely used in intensive care, but it requires multiple physiological variables, which limits its rapid application. In contrast, BISAP is a simple tool based on five variables collected in the first 24 hours and has gained popularity for its simplicity. **[43, 44, 45]**

Patients with 2 or fewer points had a mortality < 1%. Those with BISAP > = 3 had mortality between 5-20%, which the system can predict early mortality in those patients who do not have early organ failure. **[46]** And it consists of: *"1) Urea Nitrogen > 25 mg/dl; 2) alteration of mental status evidenced by disorientation; 3) presence of systemic inflammatory response (2 or more of the following variables: heart rate > 90 beats/min, respiratory rate > 20 per min, or PaCo2 < 32 mmHg, temperature > 38 or < 36°C, and leukocytes > 12,000 or < 4,000 cells per mm3 or > 10% bands); 4) pleural effusion in chest X-ray or tomographic study, and 5) age > 60 years"* **[47]**

The Marshall score assesses organ failure in acute pancreatitis, including the respiratory, cardiovascular, and renal systems; two or more points out of 12 are defined as organ failure, and APACHE II is a system that classifies disease severity using 12 different items related to age, previous functional status, and physiological parameters; a score ≥ 8 distinguishes mild acute pancreatitis from severe acute pancreatitis. **[48, 49]** "a*) the respiratory system (PO_2/FIO_2 ratio); b) the renal system (serum creatinine concentration); c) the hepatic system (serum bilirubin concentration); d) the hematological system (platelet count); and e) the central nervous system (Glasgow Coma Scale)."* **[50]**

The two scales should be analyzed and combined since they determine the severity and prognosis of patients with acute pancreatitis, however there are more scales that adhere to a meta-analysis where the authors are supported by the Newcastle-Ottawa scale, where elevated systemic inflammatory response syndrome on admission is associated with an increased risk of severe acute pancreatitis. higher values were observed in patients with severe disease compared to those with mild pancreatitis. **[51]**

In a type I experimental study, measuring serum concentrations of creatinine, total bilirubin, and alkaline phosphatase at presentation may help identify cases of acute pancreatitis with an increased risk of death. Although creatinine and bilirubin with the clinical outcome had already been described, identifying alkaline phosphatase as another prognostic marker could represent a novel finding to refine. **[52]** In the search for oxidative stress that is implicated in acute pancreatitis), but its diagnostic/prognostic value at presentation is still uncertain, with the study of oxidative stress signatures covarying with inflammation and apoptosis with the strong GSH-P-carb coupling and moderate correlations with caspase-3 with CRP and P-carb. However, further investigation of the antioxidant-protein-oxidation and apoptosis-inflammation axes is required. **[53]** Other biomarkers of mortality in acute pancreatitis that have been studied, such as vascular endothelial growth

factor, von Willebrand factor, endothelin, E-selectin, and inflammation, such as tumor necrosis factor-alpha (TNF-α), C-reactive protein, procalcitonin; at day 0 at admission, 1, and 7 days. Where a robust interaction between endothelial dysfunction and systemic inflammation was demonstrated in acute pancreatitis, they provide superior prognostic accuracy for mortality. **[54]** On the other hand, serum lipase levels, using a cut-off value, have been estimated to have shown a statistically significant, albeit limited, association with clinical risk stratification in cases with acute pancreatitis; but it should not be considered an independent predictor of mortality in acute pancreatitis. **[54]**

CONCLUSIONS

Predicting severe outcomes in patients with acute pancreatitis remains a challenge today. Being able to recognize high-risk patients early can facilitate clinical decision-making, guide the intensity of follow-up, and improve prognosis.

Various methods, scales, markers and several more have been used to predict the severity of acute pancreatitis and its evolution; clinical evaluation, imaging and the determination of various biochemical markers. However, this pathology is a very complex disease and, despite the existence of several clinical, biochemical and imaging criteria to assess its severity, predicting its prognosis is not an easy task. The markers of this protocol, such as baseline and lactate deficiency, are predictive factors in patients with severe acute pancreatitis, they have shown to be non-specific, but with a certain degree of sensitivity to determine severity in patients, however, with a weak statistical association and a poor outcome, however, they are still a reference guideline for new research.

Conflict of Interest

The authors stated that they had no potential conflicts of interest regarding the research, authorship, and/or publication of this article.

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