

# **World Journal of Medicine and Health Care**

# Acute Fulminant Pancreatitis in a Patient with Excessive Erythrocytosis

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Submitted: 20 February 2024 Accepted: 26 February 2024 Published: 04 March 2024

Citation: Arturo Zárate Curi, Luis Enrique Núñez Moscoso, Brenda Caira, Karoline Zarate Pareja, Alejandro Ramos Paredes, et al. (2024) Acute Fulminant Pancreatitis in a Patient with Excessive Erythrocytosis. Wor Jour of Medic and Heal Care 2(1), 01-04.

#### Abstract

Introduction: Severe acute pancreatitis is associated with a high morbidity and mortality due to the development of pancreatic and extrapancreatic necrosis, and subsequently necrotic tissue infection resulting in multisystem organ failure. Excessive erythrocytosis and hyperviscosis could promote hypoperfusion and ischemia of pancreatic tissue, favorecing the systemic inflamatory response (SIRS) and worsening the curse of the acute pancreatitis.

Case Presentation: We describe the case of a 71-year-old male patient from Arequipa, who worked in La Riconada, Puno, the place with the highest altitude above sea level (5300 msnm) in Peru. The patient presented to the emergency department with several abdominal pain and dyspnea, the physical examination revealed peritonial irritation and cyanosis in the extremities, and análisis revealed a posible coagulation disorder. The patient presented with severe acute pancreatitis along with acute respiratory failure requiring an intensive care unit for management. Extreme polycythemia and insufficient management quickly led to necrotizing pancreatitis that led to multisystem failure and death of the patient.

**Conclusion:** In conclusion, we report a case of severe acute pancreatitis in a patient with extreme polycythemia that rapidly led to necrotizing pancreatitis, multiorganic failure and patient death, which is not commonly reported in the literature in the management of patients diagnosed with severe pancreatitis.

Keywords: Accute Severe Pancreatitis, Polycythemia, Peru

#### Introduction

Globally, it is estimated that more than 140 million people live in localities above 2500 meters above sea level (masl), of these, 80 million people live in Asia and 35 million in the Andes, in the later the highest population density was found above 3500 meters above sea level [1, 2]. The population at this altitude lives in an environment of hypobaric hypoxia and consequently low partial pressure of inspired oxygen. As a result, he develops chronic alveolar hypoxia, hypoxemia, and polycythemia [3].

According to the International Consensus con Chronic Diseases published in 2005, Chronic Mounatin Sickness is characterized by excessive erythrocitosis (women Hb  $\geq$ 19 g/dL; men Hb  $\geq$  21 gr dL), severe hipoxemia and in some cases, moderate or severe pulmonary hypertension which can progress to pulmonary heart and heart failure, due to maladaption to altitude, in habitants at more than 2500 meters above the sea level [4].

Mosts patients with acute pancreatitis develop a mild course, but about 15-20% progress to severe acute pancreatitis, defined as the presence of organ failure or local complications such as necrosis, abscess an pseudo-cyst which is associated with a mortality rate of 3-5% del 3-5% despite advances in its management [5].

In many cases, inadequate diagnostic approach in patients with a significant epidemiological history may led to inadequate clinical mangement and thus probably increase morbidity and mortality and the use of unnecesary medical resources.

The objective of this report is to present a case of severe acute pancreatitis with fatal outcome in a male patient with concomitant excessive erythrocytosis, inhabitant of one of the highest villages in Peru and the world (La Rinconada, Puno at 5300 m.a.sl.); to subseuently carry out a review of the literatura pertinente to the case.

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#### **Case Presentation**

Male patient, 71 years old. No history of chronica diseases. He worked in an informal mine located in La Rinconada, district of Ananea in the province of Puno, which is 5300 meters above the sea level (masl) and therefore considered the highest town in the world, aproximatelly every 6 months he descended to a lower altitude, like Arequipa. Likewise, he denied undergoing periodic medical evaluations.

He went to the Emergency Department of the Carlos Alberto Seguín Escobedo National Hospital in the city of Arequipa, reporting an apparently 12-hours sick time, whose main symptom was acute abdominal pain after ingestions of fatty foods, located in the epigastrium and radiated to the dorsum, of great intensity (Analog Pain Escale 10/10) opressive type, accompanied by nausea, vomiting at various times and sweating. He denied having previously self-medication. He denied the intake of alcoholic beverages chronically and prior to the onset of the disease.

We were struck by the poor general condition with respiratory, neurological and hemodinamic compromise with which he was admitted.

Admitted in poor general condition, sporous, Glasgow Scale 10/15, dry oral mucose, fill capillary in more than 3 seconds, blood pressure 100/60mmHg, HR 98/min, SO2 75% with 0.21 and 80% with FiO2 90 % (mask with reservoir), RR 40/min, with poor ventilatory pattern, tachypneic, drowsy, cold skin and mild cyanosis in fingernails and toenails, the abdomen was distended, painful to superficial and deep palpation, with signs of peritoneal irritation (Blumberg and Rovsing positive) and intestinal ileus.

Regarding laboratory tests, the antigenic and molecular test for COVID-19 were negative, leukocytosis 12,370/mm3, Hemoglobin 27.4mg/dl, Hematocrit 81.7%, Platetes 120000/uL, glucosa 298mg/dl, total bilirrubin 3.4, direct bilirrubin 1.8, prothrombin time 26.7 seconds, INR 2.52, TPT 86.6 segundos, TGO 234U/L, TGP 148U/L, amylase 1455U/L, lipase de 1420U/L, CRP 19.7mg/dl, Urea 54.6mg/dl, creatinine 2.07mg/dl, Ph 7.25, PCO2 38, PO2 70mmHg, HCO3 17 mmHg, lactate 6.7mmol/L, Na 133mmol/L, K 4.3mmol/L, Ca 1.11mmol/L, Cl 112 mmol/L, D-dimer 1.35ugr/ml, troponin T 0.003ng/ml, CPK 68U/L, proB-NP 3263pg/ml Apache II in 22 points with a calculated mortality of 54.9%, BISAP of 04 points and calculated mortality higher than 15%.

The chest tomography (TEM) showed condensation-type hiperdensities in both bases and pleural effusion and the abdominal TEM showed enlarged pancreas with acute inflamatory signs and smalls collections and gallbladder with absence of litiasis, concluding: Acute necro-hemorragic pancreatitis.

Echo cardiography: severely depressed left ventricular (LV) systolic function, LEVF 20%, basal and medial LV akinesia.

He was intubated and connected to invasive mechanical ventilation, received support with active vessel amines and others for a critically ill patient. A detail observed at the time of performing the hemoglucotest was that the blood coagulated quickly, making it difficult to register it.

16 hours after being admitted to the Emergency Department, he was transferred to the ICU. At no time was hemodynamic stabilization achieved, but rather greater Multiorgan Dysfunction (MOD), requiring higher doses of amines (Norepinephrine, Vasopressin, Dobutamine) with Acute Renal Failure, Acute Respiratory Failure with Acute Respiratory, Hepatic, Gastrointestinal and Cardiovascular Distress with acute stress cardiomyopathy with Takotsubo syndrome and therefore unfavorable prognosis in the short term, dying 30 hours after admission.

#### Discussion

The patient came from La Rinconada, located in the department of Puno, in the south-eastern region of Peru, considered to be the highest town in the world with more than 17000 inhabitants, located at an altitude of up to 5300 meters above sea level who developed severe and fulminant acute pancreatitis, it was not possible to demonstrate the two main causes of it such as gall-stones and alcohol, but rather we were very surprised to find one of the highest values of Hemoglobin (Hb) and Hematocrit (Hto) [6, 7].

In relation to the abnormal elevation of Hb and Hto, there is terminology that needs to be clarified. High-altitude erythrocytosis differs from terms such as secondary polycythemia and polycythemia vera, which would be related to chronic diseases such as COPD, pulmonary fibrosis, etc. or an onco-haematological disease in which there is an alteration not only in the concentration of haemoglobin but also of the three hematopoietic series, respectively [8].

In relation to the term acute fulminant pancreatitis, the following was found in the literature: the criteria for defining early severe AP (ESAP) was severe organ failure (OF) within 07 days after pancreatitis. Patients with ESAP were subdivided into fulminant and subfulminant AP according to the time of OF, i.e., <72 h and between 4 and 7 days of pancreatitis, respectively, it is also described that the greater the organ failure, the higher the mortality [9].

The patient developed severe acute pancreatitis according to the Atlanta classification, and the Apache II and BISAP scores, the systemic inflammatory response was aggressive and progressive towards multiple organ failure, requiring ventilatory support and hemodynamic monitoring [10-13].

In AP, the reduction in intravascular volume can be detected as an increase in the serum hematocrit level, which can lead to decreased perfusion of the microcirculation of the pancreas and result in pancreatic necrosis. In a study conducted by Banks on "Hemoconcentration as an Early Predictor of Organ Failure and Pancreatic Necrosis" they concluded that a hematocrit greater than or equal to 44 at admission or during the first 24 hours had an increased risk of pancreatic necrosis and that a decrease in hematocrit during the first 24 hours had a lower risk of pancreatic necrosis however, most studies show that this is not a reliable predictor of severity [14-17].

Excessive high altitude erythrocytosis would be an important factor in the development of severe acute pancreatitis pathologically speaking. Blood concentration and blood hyperviscosity is a risk factor for the severity of acute pancreatitis as described by multiple studies such as severity prognostic factors in patients with acute pancreatitis in hospital nacional sergio e. vernales [18]. In its conclusions, it was found that patients with a hematocrit >40% were 4.17 times more likely to develop moderately severe acute pancreatitis and severe acute pancreatitis, as opposed to those who obtained a hematocrit <40% (OR: 4,17; IC 95%: 2,07-8,39; value p: <0,001)

Hemoconcentration would be due to multiple causes, such as the implicit state of volume depletion due to lack of intake, vomiting, abnormal distribution of water in the third space, etc. Hematocrit values above 40% have been described related to severity and higher mortality from this disease, this in turn would condition less perfusion at the level of the microcirculation of the pancreatic tissue, with a risk of ischemia and necrosis, clinically expressed in severe presentation of acute pancreatitis and increased risk of mortality [19].

The presence of SIRS and its duration over time would be related to a greater severity of acute pancreatitis.

In the study by Hai-Hong Zhu et al, called The relationship between systemic inflammatory response syndrome and severity of acute pancreatitis combined with plateau erythrocythemia, they found that there was a significant difference between the erythrocythemia group and the non-erythrocythemia group, not only in the incidence of patients who patients who developed SIRS, but also in two elements of patients who did or did not meet the diagnostic criteria for SIRS (P < 0.05). The more severe the acute pancreatitis combined with erythrocythemia, the longer the duration of SIRS [20-22].

More recently (2022), the same researcher in his publication, Association of High Altitude Polycythemia with an Increased Risk of Systemic Inflammatory, Response Syndrome in Acute Pancreatitis describes a retrospective study evaluating the relationship between acute pancreatitis and high-altitude polycythemia in 100 patients admitted to the People's Hospital of Qinghai Province during 2006-2016. Patients were divided into two groups: one with acute pancreatitis and high-altitude polycythemia (PA+HAPC) and one with acute pancreatitis without high altitude polycythemia (AP). The results showed that high-altitude polycythemia was associated with an increased risk of SIRS in patients with acute pancreatitis. In addition, the greater the severity of acute pancreatitis combined with high-altitude polycythemia, the longer the duration of SIRS. The development of SIRS and the severity of AP+HAPC are closely related. Patients with HAPC are more vulnerable to the effects of AP with concomitant SIRS [23].

Multiorgan dysfunction in severe pancreatitis has been reported to be greater in patients with hypoxia due to high-altitude erythrocytosis. In the studio The changes and significance of multiple organ functions in acute pancreatitis patients under hypoxic condition on plateau, Hai-hong ZhuL, describes that alanine aminotransferase (ALT) and creatinine (Cr) were significantly higher

in patients with AP who complicated plateau erythrocythemia compared with uncomplicated AP patients with plateau erythrocythemia. Arterial partial pressure of oxygen (PaO2) and arterial oxygen saturation (SaO(2) were significantly lower in AP with plateau erythrocythemia complication than AP without complication of plateau erythrocytemia. There was no difference in aspartate aminotransferase (AST), blood urea nitrogen (BUN) and partial blood pressure carbon dioxide (PaCO2); however, their levels were higher in cases of plateau erythrocythemia than in those without plateau erythrocythemia [24].

#### **Conclusions**

In conclusion, we report the case of a 71-year-old male patient with excessive erythrocytosis who developed severe acute pancreatitis. Erythrocytosis possibly accelerated the picture rapidly leading to necrotizing pancreatitis, multi-organ failure and death of the patient, this is not commonly reported in the literature in the management of patients diagnosed with severe pancreatitis. Clinical research in patients with chronic conditions such as erythrocytosis is essential to establish the clinical approach and management of these patients in urgent or emergency settings such as acute pancreatitis or others. In addition to recognizing that timely diagnosis and treatment is essential to avoid severe complications that quickly progress.

# **Contribution to Authorship**

All authors writing of the manuscript, and all participated in the review and approval of the final manuscript.

### **Funding Statement**

Self-funded

#### **Disclosure**

The authors declare no competing conflicts of interests.

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