



Use of Plasmapheresis: Acute Pancreatitis Due to Hypertriglyceridemia: Case Report

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Abstract

Hypertriglyceridemic pancreatitis (PATG) is described as an uncommon cause with an incidence that ranges from 2 to 4% of cases, generates significant morbidity and mortality between 40 per 100,000 inhabitants in a western population [1,2]. There are several pathophysiological mechanisms that explain the appearance of pancreatitis caused by hypertriglyceridemia, one of them is the direct toxic role of free fatty acids on pancreatic tissue and another widely accepted mechanism is chylomicron-mediated vascular obstruction, given the decrease in the gene expression of lipoprotein lipase (LPL) [3]. The American Society for Apheresis (ASFA) indicates the use of plasmapheresis when there is severe pancreatitis, when triglyceride levels exceed more than 2000 mg/dl and when there is no satisfactory response to first-line treatment; the success of the treatment is based on a decrease in triglycerides below 500 mg/dl to achieve the goal of using plasmapheresis [4]. In this case, it was demonstrated that the timely assessment of the patient was essential to make treatment decisions in the appropriate time with a favorable response to it.

Keywords: *Acute Pancreatitis, Hypertriglyceridemia, Plasmapheresis, Triglycerides*

Introduction

Pancreatitis is described as an inflammatory process that affects the gland; Among the most common causes are those induced by gallstones as the main cause and alcohol abuse, leaving hypertriglyceridemic pancreatitis as an uncommon cause with an incidence that ranges from 2 to 4% of cases, generating significant morbidity and mortality. between 40 per 100,000 in the western population [1,2]. Studies have been carried out to establish the cut-off of triglycerides that can cause acute pancreatitis, although it is still unknown exactly, levels of greater than 1000 mg/dl have been established.

Although the pathophysiology is not clearly established, it is indicated that it is the result of several interactions with multiple factors, the accepted theory being excessive metabolism by pancreatic lipase and free fatty acids together with hyperviscosity due to excess triglycerides in the pancreatic capillaries that can cause ischemia [3,5]. It is associated with genetic disorders such as familial hypertriglyceridemia due to elevated ApoB and non-HDL cholesterol [6] due to lipoprotein lipase, which is an enzyme that hydrolyzes triglycerides into lipoproteins and plays an important role in triglyceride synthesis [7].

There are local complications such as necrosis, abscesses, pseudocyst and ascites, as well as systemic complications that start from

gastrointestinal bleeding, hyperglycemia, hypercalcemia, diabetic ketoacidosis and acute respiratory distress syndrome, multiorgan dysfunction, disseminated intravascular coagulation and compartment syndrome [4].

Plasmapheresis is the method in which the plasma is disintegrated from the blood to purify it and, after the procedure, infuse the cleared plasma into the patient [8]. Plasmapheresis is used as a treatment option since it rapidly eliminates triglycerides and chylomicrons from the circulation, stopping the factor that triggers the clinical picture, the additional inflammation and pancreatic damage that it can produce compared to conservative treatment that takes days for its action. this treatment has been proposed especially in critically ill patients [1].

Currently the guidelines do not determine first-line therapies to act against this disease whose cause to be treated is hypertriglyceridemia, among the available options we have oral lipid-lowering drugs, insulin therapy, heparin and plasmapheresis [3]. The American Society for Apheresis (ASFA) indicates the use of plasmapheresis when there is severe pancreatitis when triglyceride levels exceed more than 2000 mg/dl and when there is no satisfactory response to first-line treatment; It has been evidenced between 46 to 80% reduction of symptoms with 2 to 3 sessions of plasmapheresis [9,10]. There are contraindications for the use of plasmapheresis such as mental disorders, circulatory failure, unstable cardiac or cerebral infarction, intracranial hemorrhage or severe cerebral edema and more than 72h from the beginning of PATG.

Description of the Clinical Case

A 32-year-old female patient with a personal medical history: diabetes mellitus II, treated with Glimpiride/Metformin 2/850 mg per day. Hypertension diagnosed in February 2018, uncontrolled. Patient is admitted with clinical symptoms of approximately 8 hours of evolution, prior to admission, characterized by abdominal pain in the left upper quadrant with dorsal radiation in the hemicbelt, of great intensity 8/10, which does not subside with analgesia and is accompanied by nausea that they progress to vomiting on 3 occasions of nutritional content preceded by excessive intake of copious and high-fat food. On physical examination, dry mucous membranes, depressible soft abdomen, painful on deep palpation, hemicbelt pain, increased air-fluid noises, a nasogastric tube is placed where bilious discharge is evidenced. Hemodynamically sta-

ble with vital signs BP 134/74 mmHg, HR 89 bpm, RR 19 rpm, Sat 98% room air, glycemia 333 mg/dl. According to the clinical picture and complementary tests (Table 1), acute pancreatitis induced by Hypertriglyceridemia is determined as a diagnostic impression and is staged with the following scores: APACHE score 15 MORTALITY 30.4%, MARSHALL: 1, BISAP: 1, RAMSAY: 5, RAMSON: 8 and by Tomographic study Baltasar: B.

Blood profile	Leukocytes 12.88 x103ul granulocytes 76% lymphocytes 16% hemoglobin 28.1 g/dl Hcto. 32.4% platelets 361x103Ul. Evidence shows lipemic.
Liver profile	TGO 60.0 U/l TGP 45.0 U/L, Bilirubin total 19.30 mg/dl, direct bilirubin 15.60 mg/dl Amylase 890 U/l Lipase 1080 U/l.
Renal profile	Urea 18.60 mg/dl. Creatinine 0,60 mg/dl.
	PCR 198,10 mg/l Dehydrogenase Lactate 1.560,0 u/l
Electrolytes	Na 142.7 mmol/l K 5.40 mmol/l Cl. 116.9 mmol/L Ca. 6.90 mmol/l.
Arterial blood gases	Ph 7,0 Pco ₂ 11.7 Po ₂ 154.5 HCO ₃ 5.9 mmol/l BE -24,5 mmol/L sat 97.7%
Lipid Profile	Cholesterol Total 1.270,0 mg/dl. HDL 285.0 mg/dl. LDL 431 mg/dl. Triglycerides 7,080.0 mg/dl.
Total Abdomen Ultrasound	Moderate fatty liver disease, acute pancreatitis, peripancreatic fluid, splenomegaly, aerocolia.
CT scan of the abdomen and simple pelvis	Acute pancreatitis, splenomegaly, fatty liver disease, follicles in ovaries.

Table 1: Entrance exams.

The critical medicine service is consulted because the patient, during the first hours of hospitalization, shows great intensity pain in the abdominal region 10/10, generalized paleness, tachycardia, heart rate 140, with neurological deterioration, evidence in gasometric examination metabolic acidosis, we proceed to orotracheal intubation, with the following ventilatory parameters: Ventilatory mechanical ventilation: VCV, Total volume: 500 cc, PEEP: 6, FIO₂: 60%, RF: 18.

Extensive hydration, insulin infusion, and analgesia are started are obvious results. Plasmapheresis is indicated as adjunctive therapy to the clinical picture using a high-flow vascular catheter. Duration of the procedure: 3 hours; 3,600 ml of milky plasma are extracted and the procedure is repeated after 24 hours. Duration 3 hours, obtaining 4000 ml of lipemic plasma. It was decided to perform plasmapheresis where a lipemic sample was extracted (Illustration 1) with triglycerides of 7080 mg/dl, achieving a 50% decrease in the first session and 2 more sessions were carried out, achieving a final result of triglycerides of 406 mg/dl (Table 2). According to clinical evolutionary phase and laboratory plasmapheresis in this patient were effective, reaching triglyceride values at the desired objectives, an appropriate neurological window was performed with spontaneous eye opening without being able to progress from mechanical ventilation due to high use of vasopressor, tachycardia, bilateral pulmonary infiltrates, metabolic acidosis, having to maintain mechanical ventilation, restart sedation, evolving unfavorably because he presented septic shock of pulmonary origin, bacteria were isolated in tracheal aspirate (Table 3), it was accompanied by hemorrhage and cerebral edema, leading to fatal death on the ninth day of hospitalization.

First session of Plasmapheresis	Cholesterol total 620.0 mg/dl. HDL 37.7 mg/dl. LDL 26.0 mg/dl. Triglycerides 3.040.0 mg/dl. Amylase 577.0 U/l Lipase 388 U/l.
Second session of Plasmapheresis	Total cholesterol 569 mg/dl. Triglycerides 1700 mg/dl. Amylase 101.0 U/l Lipase 80 U/l.
Third session of Plasmapheresis	Total cholesterol 217.0 mg/dl Triglycerides: 406.0 mg/dl. Amylase: 54 u/l, Lipase 48 u/l

Table 2: Lipid profile after plasmapheresis session.

Gram stain	Coconut bacillus gram negative polymorphonuclear4 - 6 x field
Counting	X108 cfu/ml
Isolated Germ Interpretation	<i>Acinetobacter baumannii</i> multiresistant
Mayor = 105 ufc/ml	Infection
Minor = 104 cfu/ml	Colonization

Table 3: Tracheal aspirate skin report.

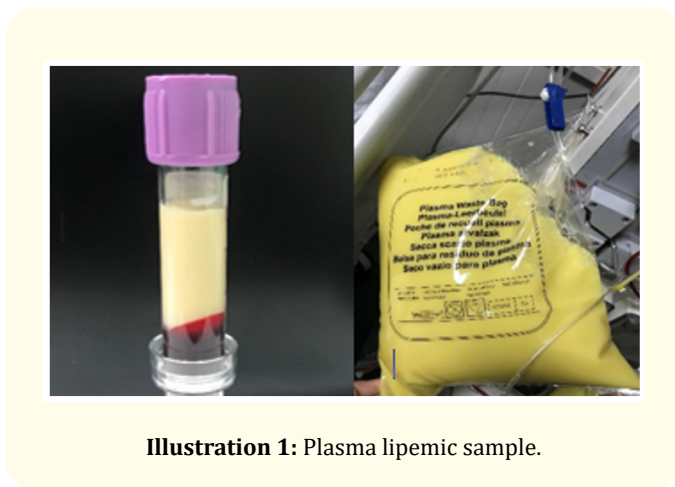


Illustration 1: Plasma lipemic sample.

Discussion

Mortality in Pancreatitis secondary to Hypertriglyceridemia varies from 2 to 4% of the cases registered in the literature, higher than other etiologies of acute pancreatitis: triglycerides can be elevated in mild or moderate pancreatitis of any etiology, however, its elevation it is < 500 mg/dl not comparable with hypertriglyceride-

mic pancreatitis in which the level will be > 1000 mg/dl. Plasmapheresis has shown a reduction in mortality when used between the first 24 to 48 hours, lowering triglyceride values in the first session to normal levels or close to ranges between 50 and 80% of cases. According to the Apheresis Society and the American Medical Association, it is an indication III for therapeutic plasma exchange; Plasmapheresis therapy was used in the case described, determining very effective due to the decrease in triglycerides in the least possible time to correct the pathophysiological cascade. The results obtained in the case described with this technique were positive and its effectiveness was determined by the evident clinical improvement and decrease in triglyceride levels. It should be mentioned that this type of procedure is often not feasible due to the cost or because the hospital center does not have this procedure, however, when it is available, it is advisable to use it to lower triglyceride values, improve the patient's symptoms and thereby reducing mortality, which despite being low constitutes a serious complication.

Conclusion

It can be concluded that plasmapheresis in these patients with severe hypertriglyceridemia is an effective treatment to reduce the risk of developing complications and mortality, taking into account the indications of the procedure and time of the therapy that should be performed at intervals of 24 to 48 hours, evaluating from upon admission, the possibility of performing the technique without delay, with this, it is intended to reduce complications and thus mortality.

Recommendations

The exhaustive evaluation of the patient with a history or with a clinical picture compatible with the disease is the fundamental pillar for making treatment decisions that have a special influence on these critical patients, thus reducing complications, morbidity and mortality.

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