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Case report

Severe acute pancreatitis secondary to familial hypertriglyceridemia: case report

Pancreatite aguda grave secundária a hipertrigliceridemia familiar: relato de caso

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Abstract

Objective: to report the clinical case of a patient with severe acute pancreatitis secondary to familial hypertriglyceridemia. **Materials and Methods:** this is a case report conducted through the collection and analysis of data from the patient's medical records. The discussion was based on materials obtained from the following study databases: Pubmed, LILACS, SCOPUS, GOOGLE, Embase, Web of Science, and the OpenGrey gray literature database. The descriptors used were "severe acute pancreatitis" and "hypertriglyceridemia". The inclusion criteria focused specifically on pancreatitis secondary to hypertriglyceridemia, while studies on other etiologies and those published before 2013 were excluded. The last search for study selection was conducted on November 14, 2021. **Case report:** a 19-year-old female patient presented with acute necrotizing pancreatitis affecting over 60% of the organ. The identified trigger factor was hypertriglyceridemia with a probable familial origin. He evolved with organ dysfunction and required intensive care unit management. The therapeutic approach was conservative. **Conclusion:** acute necrotizing pancreatitis is associated with a more severe clinical course. Furthermore, future studies are essential for a better understanding of early diagnosis and treatment of pancreatitis secondary to familial hypertriglyceridemia.

Keywords: Pancreatitis, Acute Necrotizing. Hyperlipoproteinemia Type IV. Acute abdomen.

Resumo

Objetivo: relatar o caso clínico de uma paciente com o quadro de pancreatite aguda grave secundária à hipertrigliceridemia familiar.. **Materiais e Métodos:** trata-se de um relato de caso realizado por meio da coleta e análise de dados contidos em prontuário médico A discussão foi realizada com materiais provenientes das seguintes bases de estudo: Pubmed, LILACS, SCOPUS, GOOGLE, Embase, *Web of Science* e banco de dados da literatura cinza *OpenGrey*. Os descritores utilizados foram "*severe acute pancreatitis*" e "*hypetriglyceridemia*". O critério de inclusão foi abordar de forma mais específica a PA secundária `a hipertrigliceridemia. Estudos com foco em outras etiologias e data anterior ao ano de 2013 foram excluídos. A última pesquisa para a seleção dos estudos ocorreu no dia 14 de novembro de 2021. **Relato de caso:** paciente do sexo feminino,19 anos, apresentou pancreatite aguda necrotizante com acometimento de mais de 60% do órgão. O fator desencadeante identificado foi a hipertrigliceridemia com provável origem familiar. Evoluiu com disfunção orgânica e necessidade de cuidados em unidade de terapia intensiva. A conduta terapêutica instituída foi conservadora. **Conclusão:** a PA necrotizante associa-se com maior gravidade do quadro. Além disso, estudos futuros são importantes para maior esclarecimento acerca do diagnóstico precoce e tratamento da PA secundária a HTG familiar.

Palavras-chave: Pancreatite aguda necrosante. Hipertrigliceridemia familiar. Abdome agudo.

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Introduction

Acute pancreatitis (AP) is an inflammatory disease of the pancreas. This condition is associated with high morbidity and mortality, generally between 2-8%, and in severe cases up to 85%[1-3].

The main etiologies are biliary and alcohol-induced lithiasis, while less prevalent causes include hypertriglyceridemia. It should be noted that HTG above 1000mg/dl is an important cause of acute pancreatitis. Triglyceride metabolites can induce direct and indirect cell damage, causing pancreatitis and, eventually, necrosis. Familial hypertriglyceridemia is a genetic condition associated with mutations in the genes that code for the low-density lipoprotein receptor[2,4,6].

The diagnosis of acute pancreatitis is based on clinical, laboratory or imaging parameters[1,3,5]. Regarding severity, a number of scores are used: Ranson scores classify severe AP with the presence of three or more criteria within 48 hours of admission. APACHE II (Acute Physiology and Chronic Health Evaluation II) has 12 evaluation parameters, plus an extra score based on age and the presence of chronic diseases. Balthazar's classifies AP into five grades, from A to E, and the extent of pancreatic necrosis into zero, 30%, 50% or more than 50%[7].

After the Atlanta symposium in 1992, two well-defined clinical presentations of AP were accepted: the interstitial form and the severe form, also known as necrohemorrhagic or necrotizing AP[7].

The management of patients with AP should be carried out within the first 48–72 hours of admission, since therapeutic decisions during this period can improve the prognosis. Initially, treatment is conservative[1]. The aim of this study was to report a case of severe acute pancreatitis secondary to familial hypertriglyceridemia.

Materials and Methods

This is a case report carried out by collecting and analyzing data from the patient's medical records. Clinical and laboratory information was recorded, including demographic data, medical history, diagnostic tests, therapeutic interventions and outcomes. To identify the scientific articles used to discuss the study, a literature search was carried out using the following databases: Pubmed, LILACS, SCOPUS, GOOGLE, Embase, Web of Science and the OpenGrey gray literature database. The descriptors used were "severe acute pancreatitis" and “hypertriglyceridemia”. The inclusion criterion was a more specific approach to acute pancreatitis secondary to hypertriglyceridemia.

Studies focusing on other etiologies and dated before 2013 were excluded. The last search for the selection of studies took place on November 14, 2021.

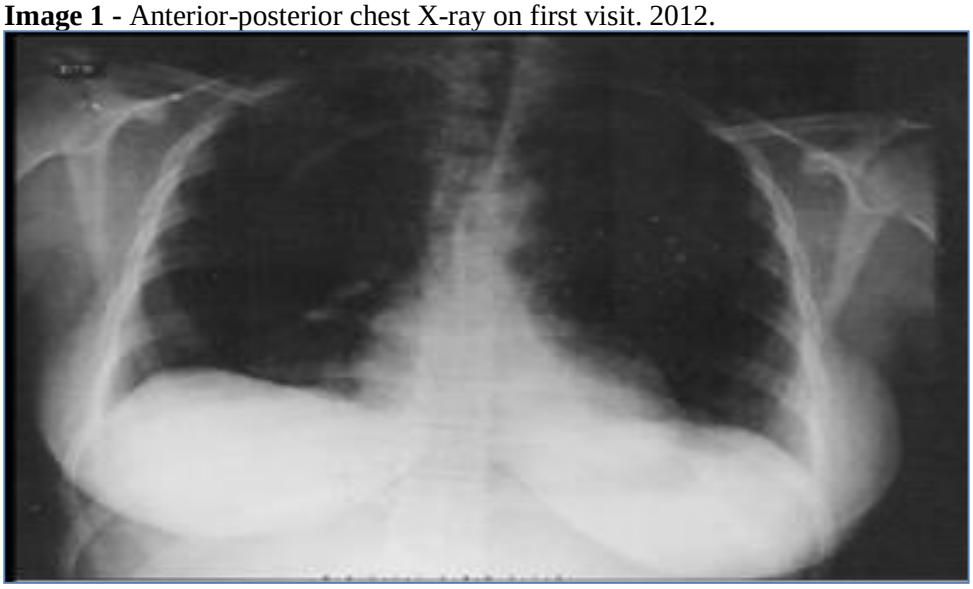
The study was submitted to the Research Ethics Committee of the United Colleges of the North of Minas Gerais with approval, CAAE number 52564721.0.0000.5141.

Case report

Female patient, leukoderma, 19 years old, medical student. She came to the emergency department reporting progressive pain in the lumbar region with an abrupt onset and asthenia. She denied any previous comorbidities or drug allergies. Furthermore, she had been taking oral contraceptives (YASMIN®) for seven months. Family history: sister with a history of hypertriglyceridemia and a father with hypertension and dyslipidemia.

On physical examination, her vital data was within normal parameters. On examination of the digestive system, she had pain on deep palpation in the suprapubic region, tympanic discharge and a negative Giordano's sign. After the clinical examination, laboratory tests and a chest X-ray were requested, as shown in Image 1 and Charts 1 and 2 below.

Image 1 - Anterior-posterior chest X-ray on first visit. 2012.



Legend: radiograph within normal limits.
Source: research participant's own database.

Chart 1 - Erythrogram. 2012.

Erythrogram	1st hospital admission	2nd hospital admission	Discharge from hospital	Reference value
Red blood cells	5,490,000/mm^3	3,310,000/mm^3	3,310000/mm^3	4 a 5,2milhões/mm^3
Hemoglobin	16.1 g/dl	8.6 g/dl	10.2g/dl	12 a 16 g/dl
Hematocrit	44.8%	28.1%	32.4%	36 a 46%
MCV	82 fl	85 fl	82,8 fl	80 a 100 fl
MCH	29 pg	29 pg	28 pg	26 a 34 pg
MCHC	36 g/dl	31 g/dl	31 g/dl	31 a 37 g/dl
Leucogram				
Global leukocytes	**24,910**/mm^3	**65,950**/mm^3	8,520/mm^3	5,000 a 10,000/mm^3
Bastonetes	2,730/mm^3	9,892/mm^3	85/mm^3	0 a 1,000/mm^3
Segmented	**17,437**/mm^3	**48,803**/mm^3	5,879/mm^3	1,800 a 7,000/mm^3
Lymphocytes	4,235/mm^3	3,298/mm^3	1,960mm^3	1,000 a 5,000/mm^3
Monocyte	498/mm^3	298/mm^3	170/mm^3	80 a 1,200/mm^3
Platelets	272,000/mm^3	-	558,000/mm^3	140,000 a 400,000/mm^3

Legend: Hm: red blood cells; Hb: hemoglobin; Ht: hematocrit; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration.
Source: research participant's database.

Chart 2 - Other laboratory tests. 2012.

Other exams	1st hospital admission	2nd hospital admission	Discharge from hospital	Reference value
Creatinine	-	4.2 mg/dl	1.0 mg/dl	0.4 a 1.3 mg/dl
Urea	-	106 mg/dl	18 mg/dl	15 a 40 mg/dl
Calcium	-	8.5 mg/dl	8.5 mg/dl	8.0 a 11 mg/dl
Magnesium	-	2.5 mg/dl	2.5 mg/dl	1.6 a 2.4 mg/dl
Potassium	-	3.4 mEq/l	4.8 mEq/l	3.5 a 5.5 mEq/l
Sodium	-	135 mEq/l	135 mEq/l	130 a 145 mEq/l
Total cholesterol	-	561 mg/dl	211 mg/dl	Desirable < 200 mg/dl
HDL cholesterol	-	12 mg/dl	14 mg/dl	41 a 59 mg/dl
Triglycerides	-	6.978 mg/dl	511 mg/dl	< 160 mg/dl
LDH	-	980 U/L	-	200 a 480 U/L
Amylase	-	471 U/L	-	25 a 125 U/L
Lipase	-	90 U/L	-	< 60 U/L
PCR	-	Positive up to 192 mg/L	-	Negative or < 6,0 mg/dl
TGO	-	23 U/L	-	4 a 36 U/L
TGP	-	33 U/L	-	4 a 32 U/L
GJ	100 mg/dl	-	-	65 a 99 mg/dl
CRP	Positive	-	-	Negative or less than 6.0 mg/dl

Beta HCG	Negative	-	-	-
Urine routine				
Appearance	Cloudy	-	-	-
Color	Yellow	-	-	Yellow
Density	1025	-	-	1.105 a 1.025
PH	6.0	-	-	5.0 a 6.5
Protein	+++	-	-	Absent
Piocytes	8 p/field	-	-	0 to 5 p/field
Red blood cells	2 p/field	-	-	0 to 2 p/field
Microbiota	Increased	-	-	Absent
Epithelial cells	13 p/field	-	-	Absent
Mucus	+	-	-	Absent
Crystals and cylinders	Absent	-	-	Absent

Legend: Ur: urea; Cr: creatinine; FBG: Fasting blood glucose; LDH: Lactate dehydrogenase; TC: Total cholesterol; TG: Triglycerides; CRP: C-reactive protein; SGOT: Oxalacetic transaminase; STGP: Pyruvate transaminase; FGB: Fasting glycemia.
Source: survey participant's database

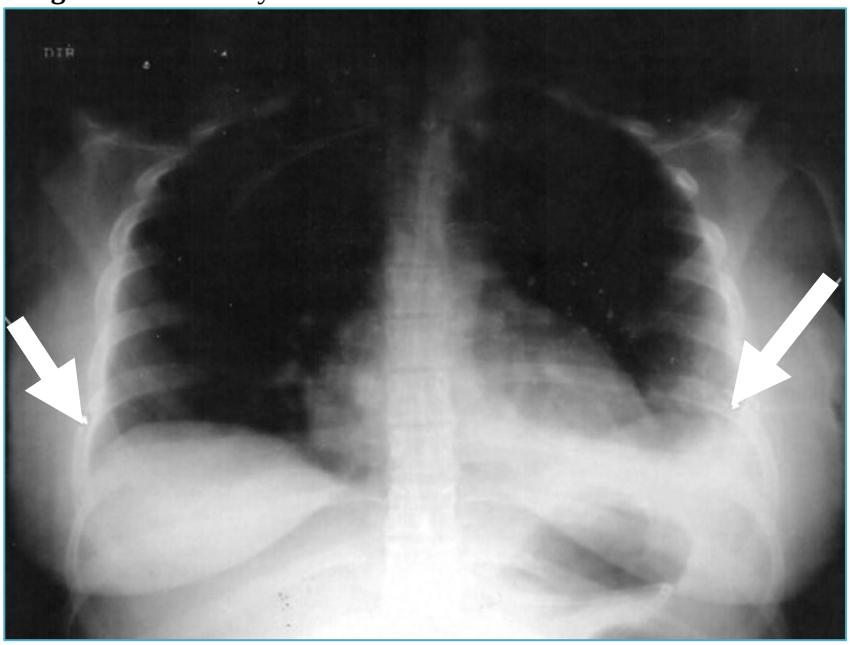
Given her clinical condition and the results of the tests requested, she was initially diagnosed with a urinary tract infection. The approach was based on analgesia (dipyrone and Buscopan®), antibiotic therapy (ciprofloxacin) and guidance. After two days, the patient returned to the emergency department complaining of severe abdominal pain, uncoercible vomiting with a greenish appearance, associated with diarrhea and dyspnea on slight exertion. On physical examination, he presented with intense prostration, tachycardia, tachypnea, diffuse pain on superficial palpation of the abdomen, abdominal distension and a positive Blumberg sign. New laboratory tests were ordered, the most significant alterations of which are shown in Charts 1 and 2, chest X-ray and total abdominal ultrasound.

The X-ray showed a bilateral basal infiltrate and veiling of the left costophrenic sinus, as shown in Image 2. The total abdominal ultrasound (Image 3) showed mild steatosis, slight splenomegaly and enlarged kidneys, compatible with acute nephropathy.

The laboratory evaluation, shown in Charts 1 and 2, revealed normocytic and normochromic anemia and significant leukocytosis with a left shift, acute kidney injury, as evidenced by the creatinine and urea levels, hydro electrolytic disorders such as hypokalemia and hypomagnesemia, as well as exuberant hypertriglyceridemia and hypercholesterolemia, which confirmed the patient's dyslipidemic profile. Amylase was more than three times normal and lipase was moderately increased. Markers of acute cellular involvement, such as C-reactive protein and lactate

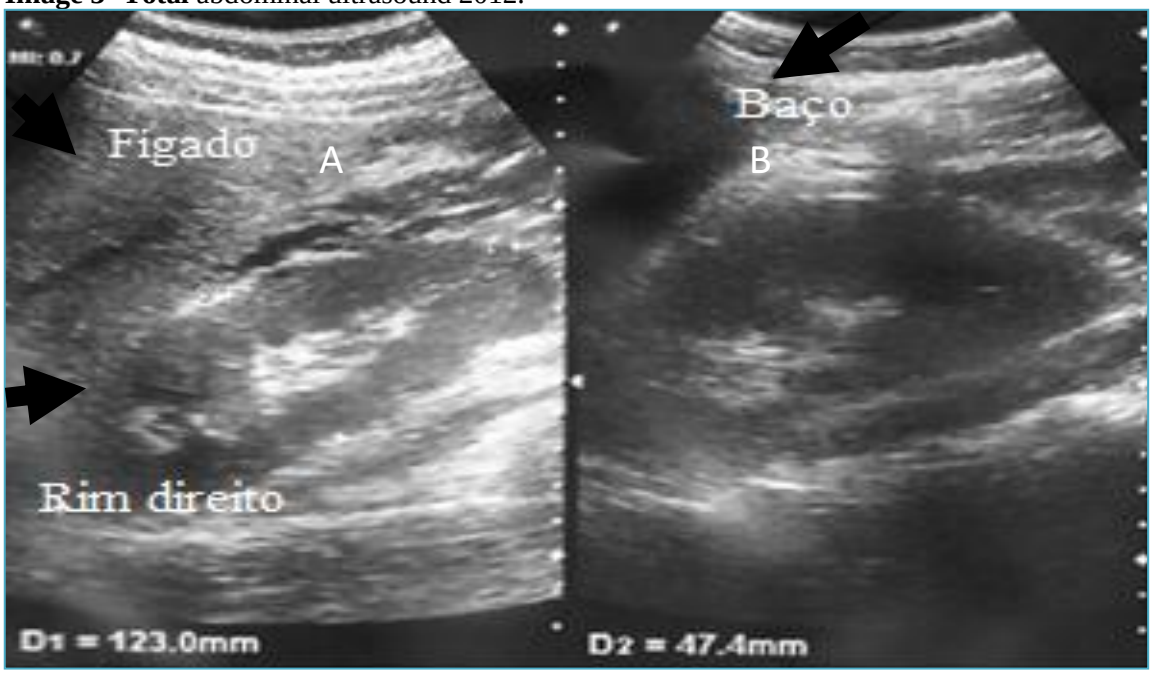
dehydrogenase, which were elevated to high levels, correlate with an intense inflammatory state in progress.

Image 2 – Chest X-ray. 2012.



Legend: the white arrows show bilateral basal infiltrates and veiling of the left costophrenic sinus.
Source: research participant's database.

Image 3- Total abdominal ultrasound 2012.



Legend: black arrow in image 3A indicates mild steatosis, evidenced by increased liver echogenicity in relation to the spleen and kidneys in image 3B.
Source: research participant's database.

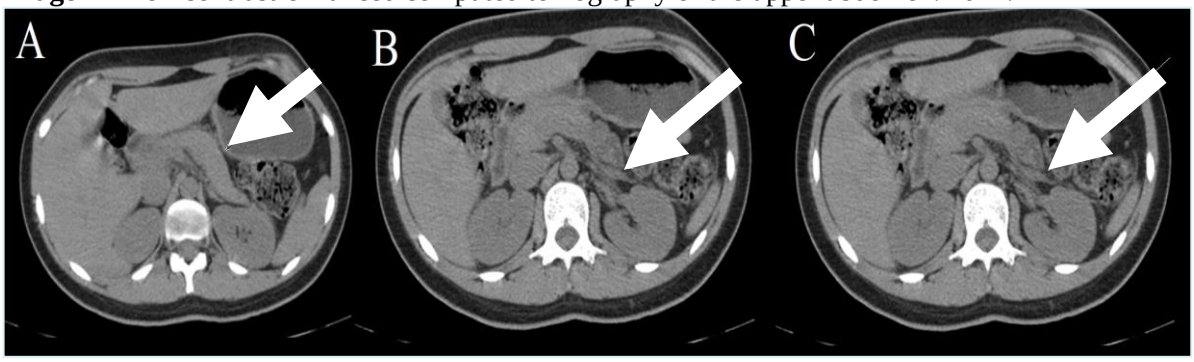
He subsequently underwent a computed tomography scan of the upper abdomen, which showed bilateral pleural effusion, hepatosplenomegaly, an enlarged and heterogeneous pancreas, as shown in Chart 3 and Image 4.

Chart 3- Upper abdominal computed tomography reports. 2012.

CT of upper abdomen without initial contrast	Bilateral pleural effusion; enlarged liver and spleen; enlarged pancreas, with heterogeneous texture and imprecise limits, associated with extensive peripancreatic collections and in the right parietocolic gutter.
Contrast-enhanced upper abdominal CT after worsening general condition	The findings were compatible with acute pancreatitis, with the presence of a large hypointense collection (necrosis) inside the pancreas, affecting more than 60% of the organ; a liquid collection in the right iliac fossa; free liquid in the pelvis and bilateral pleural effusion.
Contrast-enhanced upper abdominal CT after hospital discharge	Pancreatic pseudocyst.

Source: research participant's database.

Image 4 - Non-contrast-enhanced computed tomography of the upper abdomen. 2012.



Legend: **A)** White arrow shows a slightly enlarged pancreas with a loss of the "cerebriform" pattern. **B)** White arrow shows densification of the fat planes / thin peri-pancreatic liquid sheets. **C)** White arrow shows densification of the fat planes / thin liquid sheets in the left parietocolic gutter.
Source: survey participant's database.

In view of the clinical and laboratory progression and the alterations shown by imaging tests, the patient's diagnostic hypotheses were acute pancreatitis, familial hypertriglyceridemia, pyelonephritis and non-dialytic acute renal failure.

The procedure adopted was hospitalization and was prescribed an oral hypolipid diet according to her tolerance, oxygen therapy through a nasal catheter at 3 L/min, staggered analgesia, antiemetics, ciprofibrate 100 mg orally once a day, ciprofloxacin 400 mg 12/12 hours intravenously, intravenous hydration with a solution consisting of 0.9% saline solution, 5% isotonic glucose solution, 50% hypertonic glucose solution and 10% potassium chloride. A total of 3,600 ml of this

solution was administered over a 24-hour period. She was also monitored by general surgery and nephrology.

After these assessments, he underwent a new contrast-enhanced CT scan of the upper abdomen, shown in Chart 3, with findings compatible with acute necrotizing pancreatitis, affecting more than 60% of the pancreas.

The patient's general condition progressively worsened, with accentuated anemia, intense and persistent pain in the upper abdomen, associated with nausea, vomiting and persistent dyspnea on slight exertion. Renal function showed slight improvement, as described in Chart 2, but renal slags remained above normal values. The patient was transferred to the Intensive Care Unit (ICU) at the hospital where she had been admitted, and was prescribed enteral nutritional support, escalation of antibiotic therapy, with suspension of ciprofloxacin and initiation of meropenem, and administration of concentrated red blood cells.

The patient's clinical and laboratory condition gradually improved after 31 days in hospital, three days of which were spent in the ICU, and conservative management was maintained without the need for surgery. After stabilization and consequent discharge criteria, she was prescribed ciprofibrate 100 mg 1x a day and referred for outpatient control of hypertriglyceridemia, the probable etiology of acute pancreatitis. A CT scan of the upper abdomen, as shown in Chart 3, revealed a pancreatic pseudocyst, indicating conservative management and outpatient follow-up.

Discussion

The case report describes severe acute pancreatitis secondary to hypertriglyceridemia in a young patient. In 90% of cases, acute pancreatitis (AP) is mild or edematous. However, 10% of cases are severe and can progress to pancreatic necrosis, local infection, distributive shock and multiple organ failure[8,9]. In the case described, the patient developed a severe form of the disease, with areas of pancreatic necrosis.

Hypertriglyceridemia is causally related in up to 2-5% of cases of AP[13]. The likelihood of hypertriglyceridemia triggering AP correlates with serum triglyceride levels, so that the risk increases as triglycerides rise[10].

Triglyceride levels above 1000 mg/dL are enough to cause AP, with a potential risk of severity and complications[11]. The patient in question had hypertriglyceridemia with values above 6000 mg/dl and severe AP with organic complications, facts that corroborate the evidence of AP severity with high triglyceride levels.

Hypertriglyceridemia associated with AP is correlated with uncontrolled diabetes, alcoholism, dyslipidemia, the use of drugs that affect sugar and lipid metabolism, lipoprotein lipase deficiency, apoprotein C-II deficiency and familial hypertriglyceridemia with high plasma triglyceride levels (>1000 mg/dL)[10].

The patient has a family history of isolated hypertriglyceridemia. The genetic factor leads to the hypothesis of familial hypertriglyceridemia, the primary cause of hypertriglyceridemia. In addition, she had been taking the oral contraceptive YASMIN® for seven months. The use of contraceptives, a medication that alters the course of lipoprotein metabolism, may be related to hypertriglyceridemia as a precipitating factor. However, drug-related causes occur in less than 5% of all cases[12].

Symptoms of AP include intense abdominal pain in the upper abdomen and periumbilical region, radiating in a band to the back, associated with nausea, vomiting and abdominal distension8,9. The patient evolved with the typical symptoms of AP. The clinical presentation of AP secondary to hypertriglyceridemia does not differ from the course of other etiologies, in which the serum triglyceride level is a decisive factor in distinguishing it from other causes[10].

The diagnostic definition of AP is based on the presence of two of the following three criteria: clinical (upper abdominal pain), laboratory (serum amylase or lipase > 3x the upper limit of normal) and/or imaging criteria[13]. The patient in this study had all the diagnostic criteria.

In patients considered to have AP with no known etiology, after routine investigation negative for biliary etiology, abdominal ultrasound is recommended as the first step to evaluate occult microlithiasis, neoplasms and chronic pancreatitis. After a negative ultrasound for the aforementioned etiologies, it is recommended to request an abdominal CT scan[11].

The most common CT findings in necrotizing AP are heterogeneity and hypodensity typical of ischemic and necrotic tissue. In the case described, the findings were compatible with necrotizing AP with the presence of a large hypointense collection inside the pancreas affecting more than 60%. Magnetic resonance imaging should be reserved for diagnostic doubts or contraindications to the iodinated contrast used in computed tomography, and was therefore not necessary in the case described[9,14].

The main differential diagnoses of AP are complications of peptic ulcer, acute myocardial infarction, cholecystitis, rupture and/or dissection of aortic aneurysm, diabetic ketoacidosis, cholangitis, gastric outlet obstruction, hepatitis, intestinal volvulus, pancreatic neoplasm, intestinal

infarction and thrompo-ovarian abscess, since the manifestations of these pathologies can be similar[14].

Chart 4 below discusses the main etiologies with their respective prevalence and peculiarities, which help in the differential diagnosis of AP.

Chart 4 - The main etiologies of acute pancreatitis.

Etiology	Frequency	Clinical and laboratory findings	Imaging test findings
Gallstone disease	40%	History of biliary colic, elevated liver enzymes	Calculi or sludge in the gallbladder
Alcoholism	30%	Diagnosis is based on data from the patient's history.	New inflammatory focus superimposed on areas of chronic pancreatitis
Hypertriglyceridemia	2-5%	Fasting triglycerides > 1000 mg/dl	Nothing noteworthy
Post-ERCP	4%	-	-
Drugs	< 5%	Association of medication as a causal factor. Other evidence of drug allergy, such as skin rash.	Nothing noteworthy
Autoimmune	< 1%	Type 1: obstructive jaundice, high IgG4 levels	Type 1: This is a systemic disease affecting the pancreas, salivary glands and kidneys. Type 2: Only the pancreas is affected
Trauma	< 1%	Type 2: younger patients, no IgG4 elevation	Liquid in cavity at FAST
Infections	< 1%	Blunt or penetrating trauma, most commonly to the body of the pancreas	-

Source: Campion, 2016[12].

The severity of the condition can be predicted by clinical factors such as advanced age, previous comorbidities, obesity and chronic alcoholism. Laboratory data associated with severity are persistent high azotemia and hematocrit after fluid therapy. Amylase and lipase levels do not correlate with AP prognosis, unlike this case, which had none of the clinical factors related to severity[12].

According to the revised Atlanta classification, the severe form is defined by the presence of systemic or local complications, or both. Pancreatic and peripancreatic necrosis, sterile or infected, is

a local complication. Persistent failure of an organ system for more than 48 hours is the main predictor of a more serious outcome[12]. The patient in question had persistent systemic and local complications, constituting severe AP. The management of severe cases has the following objectives: clinical support, monitoring, treatment of complications and prevention of a new episode of AP. Intensive care unit care is indicated for all critically ill patients, as defined by the revised Atlanta classification[11].

The administration of fluids in the first 24 hours is the most important initial course of action for the best prognosis, as the loss of volume to the third space is the genesis of several complications in AP. Fluid therapy should be carried out with crystalloid solution, preferably ringer's lactate, at 2500-4000 ml in 24 hours. Volume overload can be an adverse effect of intense volume replacement, so it should be guided by some non-invasive parameters, such as heart rate <120 bpm, mean arterial pressure between 65-85 mmHg, urine output >0.5 and 1ml/kg/hour and hematocrit between 35-44%[11,13]. In the case in question, the patient received intravenous hydration with a solution consisting of 0.9% saline solution, 5% isotonic glucose solution, 50% hypertonic glucose solution and 10% potassium chloride, totaling 3600 ml of this solution over a 24-hour period. This was significant to maintain blood volume.

Enteral tube feeding should be the first line of choice for patients with severe AP. Total parenteral nutrition is the second line of choice. The oral diet can be restarted depending on clinical improvement and patient tolerance[11,12]. In the case described, enteral nutritional support was chosen.

Prophylactic antibiotic therapy is not recommended and antibiotics are only indicated in cases of suspected or confirmed infection[11,12]. In this report, the initial use of antibiotics was due to a urinary tract infection and later to treat the complication of AP.

Necrotizing AP initially consists of semi-solid and solid tissue. Over a period of at least four weeks, the necrotic collection becomes encapsulated, and the predominant content is liquid. At this point, the necrosis is classified as sterile and does not require specific therapy. The development of infected necrosis is rare in the first two weeks of the disease, and should be suspected if there is fever, leukocytosis and worsening of the clinical picture[11,12].

Surgical intervention is indicated if there is clinical suspicion or evidence of infected necrotic tissue. The main options are image-guided percutaneous drainage, catheter drainage or transluminal drainage. Drainage can be followed by necrosectomy, either endoscopic or surgical, if symptoms persist or worsen. The patient in the case described, however, did not require surgery, as her symptoms were controlled with conservative treatment.

In current practice, efforts are being made to avoid invasive interventions within the first four weeks because after this period, the necrosis evolves into a delimited process with liquid content that is better handled surgically. Therefore, respecting this time frame reduces the chances of adverse events[11]. The main indications for invasive intervention in sterile necrotizing AP are: obstruction due to the mass effect of necrosis, persistent organ failure without signs of infection and complete transection of the pancreatic duct with symptomatic pancreatic collection[11].

Around 60% of patients with necrotizing AP can be treated conservatively by administering antibiotics, percutaneous drainage as necessary and, after a delay of several weeks, debridement of the necrotic lesion by minimally invasive methods. This approach is superior to traditional open necrosectomy and has a lower complication rate[15]. The case described here presented the formation of a pancreatic pserudocyst as a complication of conservative treatment.

In addition to standard AP management, specific treatments for hypertriglyceridemia are used, such as insulin, heparin, fibrates and plasmapheresis[15]. Heparin and insulin act to increase the degradation of chylomicrons, due to increased lipoprotein activity, and thus reduce the toxic effect of free fatty acids on pancreatic acinar cells[15].

Evidence in the current literature suggests that a combination of heparin and insulin may be an effective first-line therapy for hypertriglyceridemia in the context of AP. However, the efficacy of this approach has not been well described and there is no consensus on the best heparin to use, time of use or the ideal route of administration[16].

Fibrates can be used for triglyceride levels above 500 mg/dL and are the first class of oral medication indicated for the management of hypertriglyceridemia. Statins, nicotinic acid and omega-3 fatty acids can be added to the treatment with caution, in terms of adverse effects, if the hypertriglyceridemia is resistant to initial treatment[15]. The case described received fibrate during hospitalization and was instructed to continue using it after discharge.

Plasmapheresis is an effective and safe method capable of reducing hypertriglyceridemia by 60-85% in a two-hour session, sensitizing the peripheral action of insulin and controlling the ongoing inflammatory process[10,15]. The efficiency of plasmapheresis in hypertriglyceridemia-induced AP is promising, however, the difficulty of accessibility in many centers, its high cost and the lack of more elaborate studies limit the use of this therapy.

Identifying the etiology of AP is important to recognize probable triggers for a new episode of AP. Hypertriglyceridemia as a probable etiology promotes control of triglyceride levels, preventing recurrence of the condition. The patient should continue with strict outpatient follow-up and family

members should be screened for familial hypertriglyceridemia. If hypertriglyceridemia is not clear as the cause of AP, repeated measurements of serum triglyceride levels are recommended after hospital discharge[10,12].

One of the limitations of this study was the low number of studies specifically addressing AP secondary to hypertriglyceridemia. In addition, there are no guidelines for the management of hypertriglyceridemia in the context of AP. The majority of scientific articles address only three etiologies in a more defined way (biliary, alcoholic and idiopathic origin), with the others being addressed superficially.

Chart 5 below shows some studies that specifically address the management of hypertriglyceridemia in the context of AP.

Conclusion

This case report highlights the importance of recognizing hypertriglyceridemia as a potentially serious cause of acute pancreatitis. An early and appropriate therapeutic approach, with monitoring of risk factors, is fundamental to improving clinical outcomes and reducing the morbidity and mortality associated with this condition. There is no consensus on specific therapeutic measures to control hypertriglyceridemia in this situation, so future studies are always welcome to enrich the possibility of early diagnosis and appropriate treatment of these patients.

Authors' Contribution

The authors have approved the final version of the manuscript and declare themselves responsible for all aspects of the work, including ensuring its accuracy and integrity.

Conflict of interest

The authors declare no conflicts of interest.

Chart 5 - Management of hypertriglyceridemia in the context of acute pancreatitis. Three case series studies.

Authors	Country	Cases	Treatment	Results
Uyar *et al.*, 2017[15]	Belgium	9	a) Standard management with volume resuscitation and pain control; b) Insulin, low molecular weight Heparin and fibrate; c) Plasmapheresis after failure with the initial treatment proposed; d) Outpatient follow-up after discharge, fibrate, statin, diet and control after 4–6 weeks.	Of the patients, 7 responded well to the initial treatment. However, 2 underwent plasmapheresis on the fourth day of hospitalization due to a decline in general condition, an increase in pancreatic enzymes and triglycerides. After plasmapheresis, TG levels fell by more than 50% of the initial value and the clinical condition improved rapidly.
Kuchay *et al.*, 2017[16]	India	4	a) Suspension of oral diet and fluid replacement; b) Regular insulin infusion of 2–5 units/hour, gradually increasing to 8–12 u/hour; c) The average duration of insulin infusion was 72 hours; d) Heparin was started simultaneously with insulin infusion on the first day of hospitalization; e) All had a coagulogram on admission.	All patients made a full recovery from acute pancreatitis with control of their blood glucose and triglyceride levels. None of the patients developed hypoglycemia during treatment.
Kandemir *et al.*, 2018[18]	Turkey	33	a) Restriction of oral intake, intravenous hydration and analgesics; b) Immediate therapeutic plasmapheresis with the aim of reducing triglyceride levels, carried out daily until reduction < 1000 mg/dl; c) Low molecular weight heparin was used only for thrombosis prophylaxis; d) Insulin was administered when the glucose level was > 150 mg/dL.	Plasmapheresis was well tolerated. Vomiting was observed in 5 patients, palpitations and tachycardia in 4 patients and asymptomatic hypotension in 3 patients; 2 patients had hypervolemia. Catheter occlusion was observed in 1 patient. Plasmapheresis did not have to be discontinued in any patient. Triglyceride levels fell below 1000 mg/dL with a single session of therapeutic plasmapheresis in 27 patients (81.8%). Acute necrotizing pancreatitis occurred in 2 patients; one underwent endoscopic necrosectomy, while the other died of pulmonary insufficiency, cardiovascular insufficiency and sepsis. The total mortality rate was 3%.

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