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Severe Acute Pancreatitis with Normal Amylase and Lipase Levels, A Case Report

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1. Abstract

Acute pancreatitis is a reversible inflammatory process that, depending on local or systemic involvement, can be mild or severe, with vesicular lithiasis and alcoholism being the 2 main causes, which together cause pancreatitis in 80% of cases. Some reviews attribute hypertriglyceridemia as the third cause of this entity (1.3 to 38%) of the cases reported. There is also a case report of acute pancreatitis with serum values of normal pancreatic enzymes.

1.1. Objective: To discuss and analyze the etiology of severe acute pancreatitis, without elevation of pancreatic enzymes.

1.2. Case Report: We present the case of a male adult, obese with hypertriglyceridemia (509 mg / dl), recent and chronic alcohol intake and high-fat diet prior to the development of severe acute pancreatitis without elevation of pancreatic enzymes; During his stay in the Hospital, Diabetes Mellitus type 2, Arterial Hypertension and episodes of fat dyspepsia were diagnosed (without being of lithiasic origin).

1.3. Conclusion: Acute Pancreatitis became severe, without the enzymatic elevation necessary for laboratory diagnosis, probably due to the interaction of multiple previous organic factors, such as: hypertriglyceridemia, chronic and recent alcoholism, type 2 diabetes mellitus y overweight.

2. Introduction

Acute pancreatitis is a reversible inflammatory process, which can be limited to organ involvement or produce multi-system involvement; its severity ranges from mild to severe and the latter has a mortality rate of 10-30%, depending on the presence of sterile or infected necrosis [1].

Its etiology is very varied, but among the main causes, cholelithiasis and choledocholithiasis and alcohol consumption stand out [2]. Hypertriglyceridemia is the third cause of acute pancreatitis in the western population and is the cause of 1-8% of acute pancreatitis. Hypertriglyceridemia that does not exceed the cut-off point of 1000 mg/dl has a 1.5% risk of developing pancreatitis and when these levels exceed 1000mg/dl, the percentage rises to 20.2% (2,3). There are less frequent causes such as toxins, other metabolic etiologies, vascular, mechanical, infectious, and idiopathic origin, among others.

For the development of acute pancreatitis induced by hypertriglyceridemia, it has been recognized that patients with type I-IV and V hyperlipidemia according to the Fredrickson classification have a higher risk of presenting it when their triglyceride levels are not controlled [3].

3. Description of the Case

This is a 50-year-old male patient, without comorbidity and with a history of having undergone surgery for a pulmonary hydatid cyst 26 years ago on 02 occasions. In addition, having a maternal aunt with high blood pressure and type 2 diabetes mellitus.

One week before admission, he went on a trip during which he ate foods high in fat, carbohydrates, and alcoholic beverages; Three

days before, he presented hyporexia and dyspepsia, which he attributed to alcoholic intake, hoping that it would subside spontaneously as on other occasions; The following day, abdominal pain was added in the epigastrium and mesogastrium of an intense oppressive type, constant, not irradiated, without nausea or vomiting, he went to the medical center of his jurisdiction and received analgesic treatment, the pain partially subsided. That same day in the afternoon, he went to the Police Hospital in Arequipa due to a recurrence of symptoms with similar characteristics but of greater intensity, associated with general malaise, hyporexia, nausea, and vomiting with bilious content. Analgesic treatment was indicated. antispasmodic and proton pump inhibitor, remaining under observation by the emergency service. The abdominal ultrasound showed "Hepatic steatosis II-III degree, increased abdominal meteorism and free fluid in the cavity with an amount of 31 cc. Laboratory tests: normal pancreatic enzymes (amylase 46 U/L; lipase 63 U/L), leukocytes 17,600, cell count 13%, hematocrit: 45%, hemoglobin: 15 g%, platelets: 270,000, INR 1.10, TPT 33 seconds, VDRL (-), HBsAg (-), HCAc (-), blood glucose: 256 mg/dl, creatinine: 0.72 mg/dl. With these analyzes, emergency surgical intervention was decided: "Exploratory laparotomy", finding: pancreatic area covered by foci of steatonecrosis, with increased volume and citrine fluid in the cavity, dense, malodorous, with a volume of approximately 1500 cc. He entered the Intermediate Care Unit - NICU and found a patient in fair general condition, poor hydration status, Glasgow scale 15, Blood Pressure: 148/92, FC: 88x', FR: 22x', SatO2: 95 %, FiO2: 28%, Diuresis: 900cc (24h), Weight: 84 K, Height: 1.70 m, non-jaundiced sclerae, dry oral mucosa, with nasogastric tube containing mucobilious fluid, with supplemental oxygen by binasal cannula, Jugular engorgement (-), Hepatojugular Reflux (-), ventilating spontaneously, lungs without sounds added to auscultation, hemodynamically stable without amine support and regular heart sounds, no heart murmurs were heard, globular abdomen, with tubular drains on both flanks with content serohematic, distant hydro-air sounds, tense on palpation, tympanic in the upper hemiabdomen and matte in areas of decline, genitourinary with bladder catheter and transparent urine content. The analyzes requested in the NICU showed the following:

PCR: 354 mg/L, TGO: 16, TGP: 15 GGTP: 34.7, serum calcium: 6.82 mg/dl, triglycerides:

509 mg/dl, total cholesterol: 167 mg/dl, HDL-C: 30 mg/dl, LDL-C: 115 mg/dl,

Procalcitonin: 2.63 ng/ml, D-dimer: 0.18 mg/L, ESR: 52 mm/1h, Arterial Gases: pH: 7.12, PO2: 67, PCO2: 26.9, HCO3: 8.8, Contrast-enhanced abdominal tomography (1st day of diagnosis): Hepatomegaly with moderate to severe diffuse steatosis (Figure 1 A, B). Pancreas with blurred contours, heterogeneous fat and peripancreatic collections that extend to the mesogastrium and right parietocolic slide, air bubbles at the level of the peritoneal cavity with heterogeneous and irregular peritoneum, concluding as the Balthazar E Score (Figure 2 A-D). The biopsy of the tissue obtained from the soaps was reported as steatonecrosis (Figure 3). It was calculated: APACHE II: 11 points, RAMSON: 5 points, BISAP: 3.

The evolution was favorable, and enteral nutrition was started through a nasojejunal tube on the 4th day after the diagnosis of acute pancreatitis, with good tolerance. During his hospitalization, it was determined that the hyperglycemia shown was not only due to the acute inflammatory process, but rather that the patient was a first-time diabetic (glycosylated Hb: 8.3 g), in addition to being hypertensive, for which the respective treatment was included.

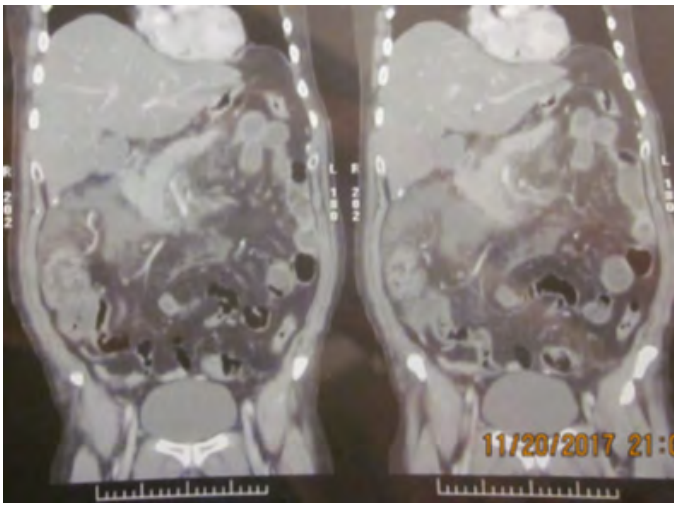

Figure 1A, 1B: Contrast-enhanced Multislice Helical Tomography, showing liver of increased dimensions and diffusely decreased density, without focal lesions. No bile duct dilation. Gallbladder adequately distended and thin-walled.

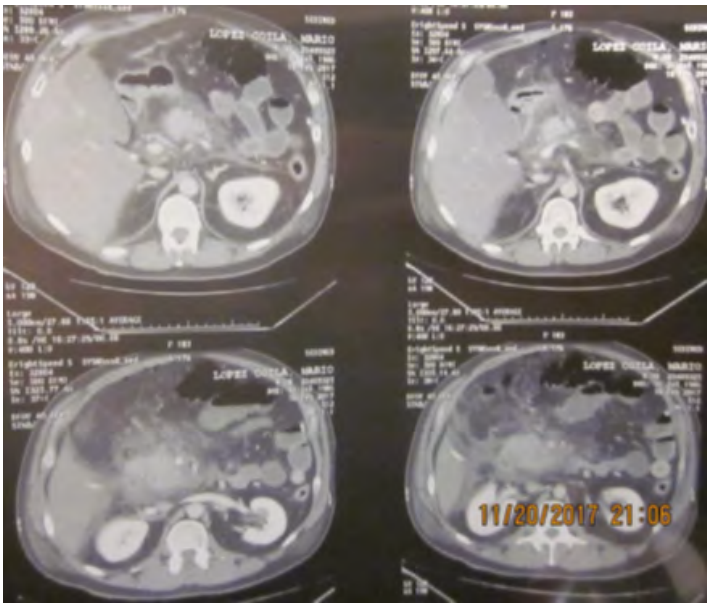

Figure 2A, B, C, D: Contrast-enhanced abdominal tomography showing heterogeneous pancreas with blurred contours, its margins are lost, heterogeneous peripancreatic fat with the presence of peripancreatic collections that extend through the mesogastrium and right parietocolic groove. (Acute pancreatitis with necrosis Balthazar E). Normal kidneys, heterogeneous perirenal fat with an inflammatory appearance secondary to pancreatic involvement.

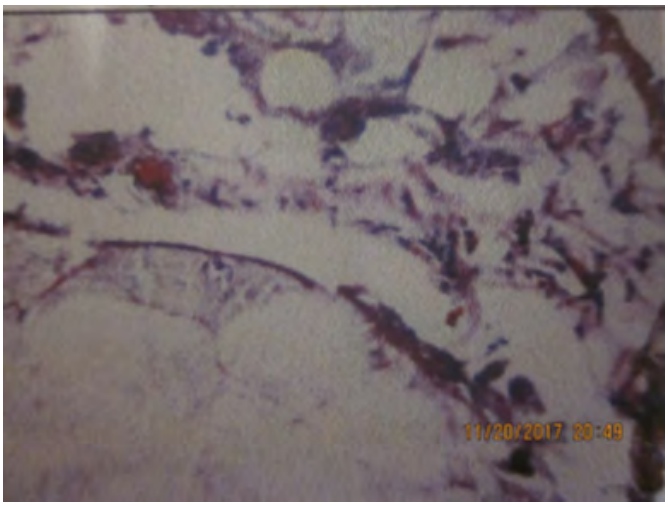

Figure 3: Pathology: shows necrosis and steatonecrosis (arrow)

4. Discussion

Acute pancreatitis is a pathology on the rise. The American College of Gastroenterology establishes the diagnosis by the presence of 2 of the following three criteria:

1) Compatible clinical picture

2) Elevation of pancreatic enzymes 3 times above the normal upper limit.

3) Imaging study compatible with acute pancreatitis [16]

Serum lipase is a very sensitive marker for acute pancreatitis (85-100%); normal values for this enzyme are rare. Serum lipase increases within 4-8 hours after the onset of acute pancreatitis, peaking at 24 hours, and begins to decrease within 8-14 days. Amylase sensitivity is reported between 81%-95% in various studies. In acute pancreatitis, amylase levels often increase for 6 to 24 hours with a peak at 48 hours and normalize over the next 5 to 7 days [16]. The better sensitivity of lipase may be due to the fact that amylase is not elevated as often in cases of alcoholic BP and hypertriglyceridemia. These findings reinforce the idea that in all patients with abdominal pain compatible with acute pancreatitis, the diagnosis should be considered even with normal enzymes. This consideration must take into account subsequent imaging studies such as ultrasound, tomography or magnetic resonance imaging of the abdomen to confirm the diagnosis. Factors that can lead to normal amylase and lipase values are hypertriglyceridemia, extensive pancreatic necrosis (fulminant acute pancreatitis), exacerbated chronic pancreatitis, or very early stage of pancreatitis when it has not led to extensive acinar cell destruction.

Normal lipase levels have been reported in acute pancreatitis in about 19% of cases and normal amylase up to 32%. A normal level of serum amylase and lipase does not exclude pancreatitis, at the same time it is good to remember that these enzymes may be elevated in other conditions. Multiple factors may be associated with the absence of elevated enzyme levels on admission. This may include the inability of the inflamed pancreas to produce amylase or the return of enzyme levels to normal before hospitalization. In the present case, we describe a patient who is overweight, diabetic, dyslipidemic (hypertriglyceridemia >500 mg/dl), with a diet rich in fat and carbohydrates frequently, and it is likely that the patient has had previous episodes of subclinical pancreatitis, with the consequent, pancreatic insufficiency.

It is estimated that 10% of chronic alcoholics suffer from acute pancreatitis. Over time, a significant proportion of patients with acute alcoholic pancreatitis who continue to consume alcohol develop chronic pancreatitis. The pathogenesis of alcoholic pancreatitis is not clearly known.

Hypertriglyceridemic acute pancreatitis is defined by the presence of high levels of Triglycerides (TG), in the absence of other etiological factors of pancreatitis [1,2]. Hypertriglyceridemia is the 3rd cause of acute pancreatitis in the western population and is the cause of 1-38% of acute pancreatitis. Hypertriglyceridemia that does not exceed the cut-off point of 1000mg/dl has a 1.5% risk of developing pancreatitis and when these levels exceed 1000mg/dl, the percentage rises to 20.2% [2,3]. It is postulated that there is an underlying alteration of lipid metabolism, on which a secondary factor acts [4]. The clinical picture is similar to that of acute pancreatitis of other etiologies, although its course seems to be more torpid and recurrent. For its diagnosis it is necessary to know that some parameters of the analysis can be confounding factors which can lead to a failure in the diagnosis. Such is the case of amylase, which can be falsely low.

The exact mechanism by which hypertriglyceridemia causes acute pancreatitis remains unknown. Some authors have even questioned its etiological role, remarking that we are not sure that hypertriglyceridemia is the cause, consequence, or underlying condition in terms of the development of acute pancreatitis [7].

There are several works, mainly on animal models, that help to clarify this point. The most widespread theory holds that the action of pancreatic lipase on a plasma with excess TG causes the accumulation of fatty acids in the pancreatic tissue, which, through the production of free radicals, damage the pancreas both at the acinar and vascular level [8-10]. Some authors speak of the secondary role of TG excess in exacerbating pre-existing damage. However, various studies find it possible that excess TG is a sufficient cause for the development of pancreatitis [11].

When we talk about acute pancreatitis and dyslipidemia, we can find 4 main scenarios: poorly controlled diabetic with or without a history of hypertriglyceridemia; alcoholic in which it is found with hypertriglyceridemia; non-obese, non-diabetic and non-alcoholic patient with hypertriglyceridemia secondary to drugs or diet; or a patient with familial hyperlipidemia without a secondary factor [2].

The slight elevation of TG can be present in many cases of acute pancreatitis of other etiologies, especially those of alcohol origin. However, it is very rare for this hypertriglyceridemia secondary to

acute pancreatitis to exceed values of 1,000mg/dl. This should not be confused with severe hypertriglyceridemia causing pancreatitis. From this level, chylomicrons begin to form and the plasma may take on a milky appearance. Amylase can show falsely low values, unlike lipase, which appears to be unaffected. This is true for both blood and urine measurements. The existence of an inhibitor of unknown origin has been postulated, leaving a minimal role for TGs per se. To obtain a true result, one can resort to diluting the serum with saline, or calculating the urinary amylase/creatinine clearance ratio.

In conclusion, we can postulate in the present case that the acute pancreatitis became severe, without the enzyme elevation necessary for laboratory diagnosis, due to the interaction of multiple previous factors such as hypertriglyceridemia and probably the moment in which the samples were taken for measure pancreatic enzymes.

References

1. Athyros VG, Giouleme OI, Nikolaidis NL, Vasiliadis TV, Bouloukos VI, Kontopoulos AG. Long-term follow-up of patients with acute hypertriglyceridemia-induced pancreatitis. J Clin Gastroenterol. 2002; 34: 472-475.
2. Fortson MR, Freedman SN, Webster PD. Clinical assessment of hyperlipemic pancreatitis. Am J Gastroenterol. 1995; 90: 2134-2139.
3. Linares CL, Pelletier LA, Czernichow S, Vergnaud AC, Bonnefont-Rousselot D, Levy P, et al. Acute pancreatitis in a cohort of 129 patients referred for severe hypertriglyceridemia Pancreas. 2008; 37: 13-22.
4. Yadav D, Pitchumoni CS. Issues in hyperlipidemic pancreatitis. J Clin a Gastroenterol. 2003; 36: 54-62.
5. Rader DJ, Hobbs HH. Trastorno del metabolismo de las lipoproteína. Harrison: A Principios de medicina interna. 16: 2522-2523.
6. Kyriakidis AV, Raitsiou B, Sakagianni A, Harisopoulou V, Pyrgioti M, Panagopoulou A, et al. Management of acute severe hyperlipidemic a pancreatitis. Digestion. 2006; 73: 259-264.
7. Domínguez-Muñoz JE, Jünemann F, Malfertheiner P. Hyperlipidemia in acute pancreatitis. Cause or epiphenomenon?. Int J Pancreatol. 1995; 18: 101-106.
8. Havel RJ. Pathogenesis, differentiation and management of a hypertriglyceridemia. Adv Intern Med. 1969; 15: 117-154.
9. Kimura W, Mossner J. Role of hypertriglyceridemia in the pathogenesis of experimental acute pancreatitis in rats. Int J Pancreatol. 1996; 20: 177
10. Hofbauer B, Friess H, Weber A, Baczako K, Kisling P, Schilling M, et al. Hyperlipaemia intensifies the course of acute oedematous and acute necrotising pancreatitis in the rat. Gut. 1996; 38: 753-758.
11. Saharia P, Margolis S, Zuidema GD, Cameron JL. Acute pancreatitis with hyperlipidemia: studies with an isolated perfused canine pancreas. Surgery. 1977; 82: 60-67.
12. Rosenson RS, Shott S, Lu L, Tangney CC. Hypertriglyceridemia and other factors associated with plasma viscosity. Am J Med. 2001; 110: 488-492.
13. Leaf DA. Chylomicronemia and the chylomicronemia syndrome: A practical approach to management. Am J Med. 2008; 121: 10-12.
14. Steinberg W, Tenner S. Acute pancreatitis. N Engl J Med. 1994; 330: 1198-210.
15. Warshaw AL, Bellini CA, Lesser PB. Inhibition of serum and urine amylase activity in pancreatitis with hyperlipemia, Ann Surg. 1975; 182: 72-75.
16. Agrawal A, Parikh M, Thella K. Case Report. Acute pancreatitis with normal lipase and amylase: an dilemma. American Journal of Emergency Medicine. 2016; 34: 2254.e3–2254.