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Severe Hypertriglyceridemic Acute Pancreatitis with Normal Lipase: A Case Report

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Abstract **Case Report**

Introduction. Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis (AP). Serum lipase may remain below three times the upper limit of normal (ULN), which delays diagnosis. ***Case presentation.*** A 29-year-old obese man (BMI 30 kg/m²) was admitted to the intensive care unit 72 h after the onset of epigastric pain radiating to the back. He had respiratory distress (PaO2/FiO2 120), KDIGO stage 3 acute kidney injury, thrombocytopenia (120,000/mm³) and hypocalcemia. The serum was lactescent, triglycerides were 9.57 g/L and lipase was 66 IU/L, below 3 × ULN. Computed tomography showed Balthazar grade E AP with pancreatic necrosis of less than 30% (CTSI 6). The modified Marshall score was 4 and three SIRS criteria were met. Other causes of AP were excluded, including biliary lithiasis by magnetic resonance cholangiopancreatography (MRCP). Organ failures were corrected with echocardiography-guided fluid resuscitation, norepinephrine, renal protective measures, oxygen therapy and non-invasive ventilation. HTG was treated with insulin, unfractionated heparin, fenofibrate and lipid-free parenteral nutrition; triglycerides decreased by 93% in 4 days without plasmapheresis. The patient was transferred to the gastroenterology department on day 5. ***Conclusion.*** Lipase below 3 × ULN does not rule out hypertriglyceridemic AP, including severe forms. Diagnosis then relies on clinical presentation, imaging and exclusion of other causes.

Keywords: acute pancreatitis; hypertriglyceridemia; lipase; insulin; heparin; fibrates.

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INTRODUCTION

HTG accounted for 7.7% of AP cases in a prospective multicenter cohort of 716 patients [1]. The risk of local complications and organ failure increases with triglyceride levels [1, 2], and the risk of recurrence is higher than in AP of other etiologies [3]. The diagnosis of AP requires two of the following three criteria: typical abdominal pain, lipase above 3 × ULN and characteristic imaging findings [4]. In hypertriglyceridemic AP, lipase may remain below this threshold [5]. We report a case of severe hypertriglyceridemic AP with lipase below 3 × ULN and discuss the mechanisms of this discrepancy, the etiological work-up, the mechanisms of organ failure, and the role of insulin, plasmapheresis, heparin and fibrates.

CASE PRESENTATION

A 29-year-old man (95 kg, BMI 30 kg/m²), a former smoker (10 pack-years, quit), with no alcohol consumption and no long-term medication, whose mother has dyslipidemia, presented with epigastric pain radiating to the back associated with vomiting of food and then bile. He was transferred to the intensive care unit 72 h after symptom onset.

On admission, the patient was conscious and afebrile. Blood pressure was 102/61 mmHg and heart rate 113/min. He had respiratory distress with a respiratory rate of 40/min, signs of increased work of breathing and a PaO2/FiO2 ratio of 120. The abdomen was distended and tender in the epigastrium and right upper quadrant. The serum was lactescent. Triglycerides were 9.57 g/L (10.8 mmol/L) and HDL cholesterol 0.08 g/L. Lipase was 66 IU/L, below 3 × ULN. CRP was 450 mg/L, procalcitonin 3.8 ng/mL and white blood cell count 16,000/mm³. Platelets were 120,000/mm³. Serum creatinine was 16 mg/L, compared with 6 mg/L three months earlier, and the patient was oliguric.

Hypocalcemia was present. Liver tests were normal, blood glucose was normal and HbA1c was 5.6%.

Abdominal computed tomography showed Balthazar grade E AP with pancreatic necrosis of less than 30% and peripancreatic collections in the anterior pararenal spaces, without signs of infection (CTSI 6). The gallbladder contained no stones and the bile ducts were not dilated. MRCP showed no common bile duct stones and no biliary or pancreatic duct abnormality (Figure 1). Other causes of AP were excluded: no alcohol consumption or medication use, no biliary lithiasis on computed tomography or MRCP, no hypercalcemia and normal liver tests. HTG was considered the cause of AP.

From admission, the patient had respiratory failure, acute kidney injury, thrombocytopenia and hypocalcemia. Three systemic inflammatory response syndrome (SIRS) criteria were met (heart rate, respiratory rate and white blood cell count). The modified Marshall score was 4 (respiratory 3, renal 1). Acute kidney injury was classified as KDIGO stage 3 on the urine output criterion (urine output below 0.3 mL/kg/h for 24 h), with serum creatinine at 2.7 times baseline. Respiratory failure persisted for 3 days, which classifies the AP as severe according to the revised Atlanta classification [6].

Respiratory management included oxygen therapy via nasal cannula and face mask, non-invasive ventilation sessions for alveolar recruitment, semi-recumbent positioning, chest physiotherapy with incentive spirometry, analgesia and fluid balance monitoring; intubation was not required. Renal management included echocardiography-guided fluid resuscitation with Ringer's lactate, a mean arterial pressure target of 80 to 85 mmHg to improve renal perfusion, with norepinephrine titrated to continuous arterial pressure monitoring through an arterial catheter, a urinary catheter with hourly urine output monitoring, avoidance of nephrotoxic agents, dose adjustment to renal function and correction of hypokalemia and hypocalcemia. Abdominal compartment syndrome was sought by intravesical pressure measurement, which remained below 12 mmHg. Measures to prevent intensive care complications were applied.

HTG treatment comprised an insulin infusion at 1.6 IU/h (0.017 IU/kg/h) with glucose supplementation, unfractionated heparin at 4,800 IU/24 h under close clinical and laboratory monitoring for bleeding, and fenofibrate 160 mg/day, whose tolerance was monitored by measurement of transaminases, serum creatinine and, because of renal failure, creatine kinase, without treatment-related abnormalities. Enteral feeding was stopped on day 1 because of digestive intolerance, and lipid-free parenteral nutrition was started. Oral feeding was gradually resumed from day 4 and parenteral nutrition was stopped on day 6. No plasmapheresis was performed. Between days 1 and 3, a right pleural effusion, ileus and fever spikes up to 39 °C occurred. No antibiotics were prescribed: the CRP elevation was attributed to the inflammatory response of AP, blood and urine cultures were negative and the rest of the infectious work-up was unremarkable.

The outcome was favorable. Respiratory distress resolved progressively over 3 days. Urine output recovered to above 1 mL/kg/h, and serum creatinine was 12 mg/L on day 2 and 9 mg/L on day 5. Platelets were 135,000/mm³ on day 2 and 150,000/mm³ on day 5. Triglycerides decreased to 6.23 g/L on day 2 and to 0.66 g/L on day 4, a 93% reduction (Table 1). Lipase was 31 IU/L on day 2 and 25 IU/L on day 4. The patient was transferred to the gastroenterology department on day 5, after correction of organ failures.

Table 1: Course of laboratory parameters

Day	Triglycerides (g/L)	Lipase (IU/L)	CRP (mg/L)	Platelets (/mm³)	Creatinine (mg/L)
Admission	—	—	450	120,000	16
D1	9.57	66	305	—	—
D2	6.23	31	279	135,000	12
D4	0.66	25	254	—	—
D5	—	—	—	150,000	9

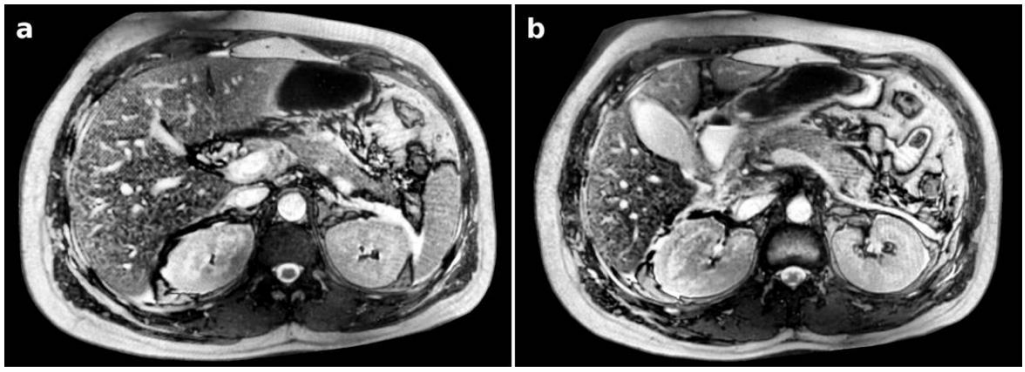




Figure 1: MRCP, axial slices. (a) Slice through the pancreas and both kidneys. (b) Slice through the gallbladder and pancreas. (c) Fat-saturated slice. (d) Slice through the pancreas and renal hilum

DISCUSSION

This case combines two features: severe AP of hypertriglyceridemic origin and lipase below 3 × ULN from the first measurement. According to the American College of Gastroenterology guidelines, AP is diagnosed when two of the three diagnostic criteria are present [4]. Our patient had typical pain and characteristic computed tomography findings; the diagnosis was therefore established independently of lipase. When lipase is below the diagnostic threshold in a patient with HTG, diagnosis relies on contrast-enhanced computed tomography [7].

The hypertriglyceridemic origin was established by exclusion of other causes. Guidelines recommend investigating biliary lithiasis and alcohol consumption, then measuring triglycerides when neither is present [4]. Attribution of AP to HTG requires that no other cause has been identified [7]. In our patient, the absence of alcohol consumption and long-term medication, the absence of stones on computed tomography and MRCP, the absence of ductal abnormality, the absence of hypercalcemia and normal liver tests ruled out the main other etiologies. Positive arguments supported HTG: lactescent serum, very low HDL cholesterol, obesity and a family history of dyslipidemia.

The triglyceride level of 9.57 g/L (957 mg/dL) is below the 1,000 mg/dL threshold usually used to attribute AP to HTG [4, 7]. However, it was measured 72 h after symptom onset, after several days of vomiting. Triglycerides decrease rapidly after AP onset [8], with an estimated half-life of 30.6 h during fasting [9]. The initial value therefore very likely exceeded this threshold.

Two mechanisms explain lipase below 3 × ULN in our patient. The first is analytical interference: lipid particles scatter and absorb light and distort spectrophotometric assays [10], and falsely normal amylase and, to a lesser extent, lipase values have been reported in HTG [7]. The lactescent serum and a triglyceride level close to 10 g/L at the time of measurement make this interference the main explanation. Sample dilution or ultracentrifugation can limit it [10]. The second is the delay between symptom onset and sampling: lipase peaks at around 24 h and then decreases until it normalizes within 8 to 14 days [5]. At 72 h, lipase was in its declining phase; this delay may have contributed to a lower value but cannot by itself explain a value below 3 × ULN.

Comparable cases have been published. Okerberg and Lee reported AP with triglycerides of 9,990 mg/dL and normal amylase, whose elevation was revealed only after serum dilution [11]. Ibrahim *et al.,*described AP with normal amylase and lipase and lipemic serum, without being able to rule out analytical interference [5].

The severity of this episode is consistent with the dose-dependent association between HTG and organ failure [1, 2]. Hydrolysis of triglycerides by pancreatic lipase releases free fatty acids, which at high concentrations inhibit mitochondrial complexes, cause intracellular calcium overload and cytokine release, and contribute to respiratory, renal and cardiovascular failure [12]. Obesity aggravates this lipotoxicity: in obese patients, unsaturated fatty acids released by lipolysis of peripancreatic adipose tissue are associated with necrosis and organ failure, and their infusion causes lung and kidney injury in animals [13]. Hypocalcemia is one of the Glasgow-Imrie severity criteria [14]. In hypertriglyceridemic AP, low serum calcium on admission is independently associated with moderately severe and severe forms [15].

Respiratory failure (respiratory Marshall score of 3) was present from admission. Free fatty acids damage the endothelium and are involved in lung injury in hypertriglyceridemic AP [13, 16]. Non-invasive ventilation combined with oxygen therapy avoided

intubation, with resolution within 3 days. Acute kidney injury was classified as KDIGO stage 3 on the urine output criterion. Its rapid correction with echocardiography-guided fluid resuscitation and renal protective measures, with recovery of urine output and a decrease in serum creatinine from 16 to 9 mg/L within 5 days, suggests a predominantly functional mechanism related to hypovolemia from vomiting and fluid sequestration. Intravesical pressure measurement ruled out abdominal compartment syndrome, a cause of kidney injury to be sought in severe AP.

Insulin activates lipoprotein lipase and reduces adipocyte lipolysis [7, 17]. Its clinical benefit has not been demonstrated. In a cohort of 106 patients, the decrease in triglycerides at day 4 was 85% with insulin and 79% with conservative treatment, without significant difference [18]. Fasting and hydration alone reduce triglycerides by 69.8% within 48 h [9]. A faster decrease in triglycerides is not associated with a reduction in organ failure [19]. A meta-analysis published in 2026 showed no superiority of any strategy [20]. In our patient, the respective contributions of fasting, hydration and insulin to the 93% decrease in triglycerides over 4 days cannot be determined.

Plasma exchange lowers triglycerides slightly faster than insulin, without difference in clinical outcome [21]. The American Society for Apheresis classifies hypertriglyceridemic AP as a category III indication, for which the optimal role of apheresis is not established and decisions should be individualized, and the Endocrine Society guidelines do not recommend plasmapheresis in this setting [16]. It has been proposed when triglycerides decrease insufficiently after 12 to 24 h of conservative treatment [16]. In our patient, the rapid decrease in triglycerides under medical treatment did not warrant its use.

Heparin releases lipoprotein lipase from the endothelium, but prolonged administration depletes enzyme stores and may cause a rebound in triglycerides [7, 17]. A randomized trial published in 2025 showed no benefit of adding low-molecular-weight heparin to insulin [20, 22]. Anticoagulation in AP is associated with a moderate increase in the risk of gastrointestinal and pancreatic bleeding [20]. In our patient, heparin was administered in the presence of pancreatic necrosis, under close monitoring for bleeding. Given the lack of demonstrated efficacy on triglycerides and the bleeding risk, its use cannot be recommended in this indication.

Nutritional management was consistent with the ESPEN guidelines, which favor enteral nutrition and reserve parenteral nutrition for intolerance or contraindication of the enteral route [23]. The absence of lipid emulsion avoided exogenous triglyceride intake during the acute phase. Withholding antibiotics was consistent with guidelines, which advise against antibiotic prophylaxis in severe AP and reserve antibiotics for documented infection [4].

Fibrates are the first-line treatment for severe HTG in patients at risk of pancreatitis, combined with restriction of fat, simple sugars and alcohol, and weight loss [7, 17]. Triglyceride-lowering therapy is indicated above 500 mg/dL [17]. No data show that initiation during the acute phase changes the course of the episode; their benefit lies in preventing recurrence. Fasting triglycerides should be checked one month after discharge [4]. The family history warrants lipid screening of relatives, as genetic forms of HTG have been described [7]. Apolipoprotein C-III inhibitors are a recent option: in the CORE and CORE2 trials (1,063 patients), olezarsen reduced the incidence of AP by 85% (1.1 vs 6.2 episodes per 100 patient-years) [24].

This case has limitations. Triglycerides and lipase were not measured in the first hours, and lipase was not measured after serum dilution.

CONCLUSION

Lipase below 3 × ULN does not rule out hypertriglyceridemic AP, including severe forms. Interference of lipemia with the assay is the most likely explanation, and sampling delay may contribute. Diagnosis then relies on clinical presentation, imaging and exclusion of other causes. Triglycerides should be measured on admission. The severity of hypertriglyceridemic AP is related to free fatty acid lipotoxicity. Triglycerides decrease rapidly with fasting and hydration; the clinical benefit of insulin, plasmapheresis and heparin has not been demonstrated, and heparin carries a bleeding risk. Prevention of recurrence relies on fibrates and lifestyle measures.

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