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Case Report

Severe hypertriglyceridemia presenting with abdominal pain and peripheral neuropathy in a patient with uncontrolled diabetes and chronic alcohol use: a case report

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ABSTRACT

Severe hypertriglyceridemia (triglyceride levels >1000 mg/dl) is a recognized high risk metabolic condition, particularly when associated with pancreatitis and the third most common cause of acute pancreatitis. However, its presentation without pancreatic involvement poses substantial diagnostic challenges and may delay appropriate management. We report a case of a 42-year-old male with chronic alcohol consumption (approximately 180 ml whiskey daily) and poorly controlled type 2 diabetes mellitus who presented with abdominal pain, right scapular pain, and a six-month history of peripheral neuropathy. Laboratory evaluation revealed markedly elevated triglycerides (>2000 mg/dl), preserved pancreatic enzymes, and imaging demonstrating a normal pancreas with grade 1 fatty liver on CT scan. Upper gastrointestinal endoscopy revealed mild antral gastritis. The patient was managed with lipid-lowering therapy (fenofibrate-statin combination), aggressive glycemic control with insulin, injectable thiamine, alcohol cessation, and dietary modification, resulting in significant biochemical improvement with triglycerides declining from 2158 to 1054 mg/dL within one week. This case illustrates the synergistic role of insulin resistance and alcohol-induced dyslipidemia in precipitating extreme triglyceride elevation and underscores the importance of early recognition and intervention to prevent catastrophic complications such as acute pancreatitis and chylomicronemia syndrome.

Keywords: Hypertriglyceridemia, Diabetes mellitus, Alcohol, Peripheral neuropathy, MAFLD, MetALD, Pancreatitis, Chylomicronemia, Insulin resistance

INTRODUCTION

Hypertriglyceridemia is an increasingly prevalent metabolic disorder, affecting an estimated 25-30% of adults in developed nations.[1] While mild-to-moderate elevations (150-499 mg/dl) are predominantly managed through lifestyle interventions, severe hypertriglyceridemia (≥1000 mg/dl) constitutes a distinct clinical entity associated with markedly increased risk of acute pancreatitis, chylomicronemia syndrome, and end-organ damage. Triglyceride-induced pancreatitis accounts for approximately 1-4% of all pancreatitis cases and is the third most common cause after gallstones and alcohol.[1,4] The risk escalates substantially at levels exceeding 2000 mg/dl, with some series reporting pancreatitis in up to 20-30% of such patients.[4]

The proposed mechanisms of triglyceride-induced pancreatitis include chylomicronemia-related hyperviscosity causing ischemia to pancreatic acini, along with pancreatic lipase–mediated hydrolysis of circulating triglycerides into toxic free fatty acids (FFAs). These FFAs directly injure acinar cells, activate inflammatory cascades, and promote oxidative stress, ultimately triggering the pancreatitis cascade.[1,4]

Hypertriglyceridemia may be primary (genetic)-as in familial hypertriglyceridemia or lipoprotein lipase (LPL)

deficiency-or secondary, with the latter accounting for the vast majority of adult cases. Key secondary causes include uncontrolled diabetes mellitus (particularly type 2), obesity, metabolic syndrome, hypothyroidism, renal disease, certain medications (corticosteroids, oral estrogens, beta-blockers, antiretrovirals), and excessive alcohol consumption.[2] When multiple secondary factors co-exist, as in the present case, triglyceride levels can rise precipitously and unpredictably.

Insulin resistance, a hallmark of type 2 diabetes, drives hypertriglyceridemia through two major mechanisms: (1) increased hepatic synthesis of very low-density lipoprotein (VLDL) particles, and (2) impaired activity of LPL, the enzyme responsible for peripheral triglyceride clearance. The net result is an overproduction-underclearance state that sustains triglyceride accumulation in the circulation.[2] Chronic alcohol consumption amplifies this process by stimulating hepatic de novo lipogenesis, increasing NADH/NAD^+ ratio (thereby diverting metabolic flux toward fatty acid synthesis), and generating acetaldehyde-a direct inhibitor of mitochondrial fatty acid β-oxidation.[6] Together, these mechanisms render the combination of uncontrolled diabetes and heavy alcohol use one of the most potent precipitants of severe, potentially pancreatitis-inducing hypertriglyceridemia.

The concept of metabolic dysfunction-associated fatty liver disease (MAFLD) has recently replaced the older NAFLD terminology, emphasizing the central role of metabolic dysfunction-including insulin resistance, obesity, and dyslipidemia-in the pathogenesis of hepatic steatosis.[3] In patients with concomitant alcohol use and metabolic risk factors, the term MetALD (metabolic and alcohol-related liver disease) has been proposed, recognizing the additive hepatotoxic effects. Such patients demonstrate an atherogenic lipid profile characterized by elevated triglycerides, low HDL-cholesterol, and increased small dense LDL particles, conferring both hepatic and cardiovascular risk.[3]

Peripheral neuropathy in patients with combined diabetes and alcohol use is typically multifactorial in origin. Chronic hyperglycemia causes neuropathy through several interconnected pathways: activation of the polyol pathway, accumulation of advanced glycation end-products (AGEs), oxidative stress, and microvascular injury leading to endoneurial ischemia.[5] Alcohol independently induces axonal degeneration and demyelination through direct neurotoxic effects of acetaldehyde and nutritional deficiencies, particularly thiamine (vitamin B1), whose deficiency can also precipitate Wernicke's encephalopathy if untreated.[5,9]

Although severe hypertriglyceridemia is classically associated with pancreatitis, a clinically significant subset of patients-as illustrated in this case-may present with abdominal pain, neuropathy, and metabolic derangements in the absence of pancreatitis. Recognition of this presentation is critical, as delayed diagnosis may result in progression to life-threatening acute pancreatitis, which carries a mortality of 5-10% in severe cases.[1,4] This report aims to highlight the clinical complexity, diagnostic considerations, and management principles in such a patient.

CASE REPORT

History

A 42-year-old male presented to the medicine department with a 3-4 day history of abdominal pain and right scapular pain, along with tingling and pain in all four limbs persisting for 6 months. He is a known chronic alcohol consumer, consuming approximately 180 mL of whiskey daily for several years. There was no prior history of diagnosed dyslipidemia, pancreatitis, or cardiovascular disease.

Clinical examination

Clinical exam included-hemodynamically stable at presentation, no icterus, ascites, or peripheral stigmata of chronic liver disease, neurological examination revealed features consistent with peripheral neuropathy in all four limbs, autonomic dysfunction evidenced by orthostatic hypotension on standing and abdomen: mild diffuse tenderness without guarding or rebound; no organomegaly on initial examination.

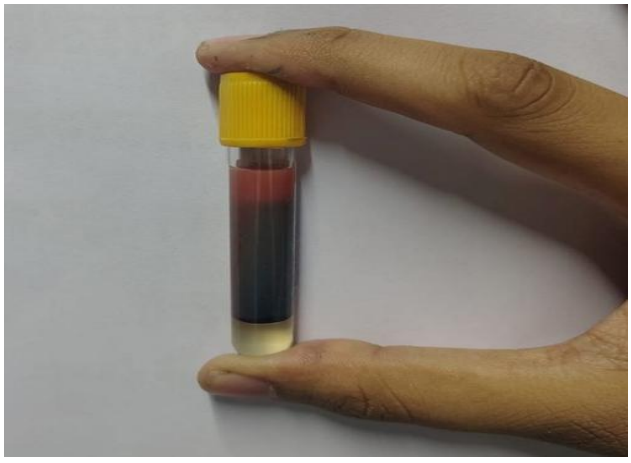

Figure 1: Fat layer/lipemia in serum sample.

Table 1: Hematological and biochemical profile.

Parameters	Value	Reference range
Hemoglobin	16 g/dl	13-17 g/dl
WBC count	9,310/mm³	4,000-11,000/mm³
Platelets	2.28 lakh/mm³	1.5-4.0 lakh/mm³

Continued.

Parameters	Value	Reference range
Blood urea	27 mg/dl	15-45 mg/dl
Serum creatinine	1.0 mg/dl	0.7-1.2 mg/dl
Sodium	131 mEq/l ↓	135-145 mEq/l
HbA1c	10.8% ↑↑	<5.7%
Urine sugar	++++	Negative
Fasting insulin	13.8 µIU/ml	2-25 µIU/ml
C-peptide	4.7 ng/ml	0.8-3.1 ng/ml ↑
Serum lipase	Normal	Normal
Urine ketone	Negative	Negative

Table 2: Liver function tests-serial monitoring.

Parameters	Admission (Day 1)	Follow-up (Day 3)
Total bilirubin	1.4 mg/dl	1.2
Direct bilirubin	0.6 mg/dl	0.6
AST (SGOT)	30 IU/l	129 IU/L ↑
ALT (SGPT)	40 IU/l	149 IU/L ↑
ALP	178 IU/l	170 IU/L

Table 3: Serial lipid profile-response to treatment.

Date	Cholesterol (mg/dl)	Triglycerides (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)
Day 1	269	2158 ↑↑↑	21 ↓	-	-
Day 3	223	1952 ↑↑↑	19 ↓	390 ↑↑	-111*
~1 week later	215	1054 ↑↑	16 ↓	210 ↑	-11.8*

*Negative LDL values reflect the limitations of the Friedewald equation at extreme triglyceride levels.

Imaging and endoscopic findings

Ultrasound abdomen showed grade 1 fatty liver and no biliary pathology.

CECT abdomen showed mild hepatomegaly; normal pancreas-no peripancreatic fat stranding, necrosis, or fluid collection.

Upper GI endoscopy (OGD scopy) showed mild antral gastritis-no peptic ulcer, varices, or active bleeding.

Final diagnosis

Severe hypertriglyceridemia (multifactorial: uncontrolled T2DM + chronic alcohol use), uncontrolled type 2 diabetes mellitus (HbA1c 10.8%), MetALD-metabolic+alcohol-associated fatty liver disease with transaminitis, peripheral neuropathy (combined diabetic + alcoholic etiology), autonomic dysfunction (orthostatic hypotension), mild antral gastritis (alcohol-related mucosal injury) and possible high risk for hypertriglyceridemia-induced pancreatitis (TG >2000 mg/dL).

Management

Acute lipid-lowering measures

Given triglyceride levels exceeding 2000 mg/dL, pancreatitis was considered a significant risk.

Plasmapheresis was considered as a therapeutic option to rapidly reduce triglyceride levels, but was deferred after imaging excluded active pancreatitis and the patient remained hemodynamically stable.

Pharmacological lipid reduction was initiated with a fenofibrate-statin combination (Atorvastatin+fenofibrate 160/10 mg). Fenofibrate acts primarily by activating PPARα, thereby upregulating LPL activity and reducing hepatic VLDL synthesis.[2,7]

Glycemic control

Aggressive glycemic correction was initiated with IV insulin-based therapy to restore LPL activity and reduce endogenous VLDL overproduction-a critical step in managing secondary hypertriglyceridemia in the context of poorly controlled diabetes.[2]

Nutritional and supportive measures

Injectable thiamine 600 mg/day-to prevent Wernicke's encephalopathy and treat alcoholic peripheral neuropathy, gabapentin + lignocaine ointment for peripheral neuropathy symptomatic relief, multivitamin supplementation, proton pump inhibitor for the gastritis and upper gastro intestinal protection and the benzodiazepines for the alcohol withdrawal symptom management.

Lifestyle modification

Strict alcohol cessation-alcohol removal dramatically reduces hepatic lipogenesis and low-fat diet (<10-15% of total calories from fat)-reduces dietary chylomicron production as well as structured diabetic diet education and glycemic monitoring.

Outcome

With the above management, triglyceride levels declined from 2158 mg/dL to 1054 mg/dL within approximately one week-a reduction of over 50%. The patient did not develop acute pancreatitis throughout the admission. Clinical stabilization was achieved with improvement in abdominal pain. The patient was discharged with ongoing outpatient follow-up for lipid monitoring, glycemic control, and neuropathy rehabilitation.

DISCUSSION

The coexistence of severe hypertriglyceridemia, autonomic neuropathy, and MetALD in the absence of pancreatitis remains sparsely described. This case presents a paradigmatic example of extreme secondary hypertriglyceridemia driven by the convergence of two potent metabolic stressors: uncontrolled type 2 diabetes mellitus and chronic heavy alcohol consumption. The interplay of these two conditions creates a biochemical milieu of VLDL overproduction, reduced LPL mediated clearance, and accelerated hepatic lipogenesis that can rapidly push triglyceride levels into the pancreatitis risk zone.[2,6]

Why no pancreatitis despite TG >2000 mg/dL?

Despite triglyceride levels far exceeding the commonly cited 1000 mg/dL threshold for pancreatitis risk, this patient did not develop acute pancreatitis. This observation is consistent with emerging literature suggesting, the relationship between triglyceride levels and pancreatitis is non-linear and influenced by additional cofactors such as the duration of elevation, the rate of rise, genetic susceptibility, concurrent alcohol-related ductal injury, and microcirculatory factors.[1,4] Early recognition and aggressive triglyceride reduction both pharmacological and through glycemic correction possibly interrupted the pathophysiological cascade before acinar cell injury could develop. This case reinforces the concept that early treatment appears to play a crucial role in the outcome in severe hypertriglyceridemia. The abdominal pain can be attributed to alcoholic gastritis.

The Friedewald equation paradox: negative LDL values

A notable laboratory finding was the calculated negative LDL value (-111 mg/dL), which is a well-recognized artifact in extreme hypertriglyceridemia. The Friedewald equation-LDL=Total cholesterol-HDL-(TG/5)-was derived and validated for triglyceride levels below 400 mg/dL.[7] At triglyceride concentrations exceeding 1000 mg/dL, the VLDL-cholesterol fraction (estimated as TG/5) becomes a gross underestimate, and the formula systematically yields implausibly low or negative LDL values. In such scenarios, direct LDL measurement, ultracentrifugation-based methods, or the Martin-Hopkins equation (which adjusts the VLDL divisor based on non-HDL cholesterol) may provide more reliable estimates.[7,10]

MetALD and the transaminitis

The rise in transaminases on follow-up (AST 129, ALT 149 IU/L) with ALT predominance is characteristic of metabolic steatohepatitis rather than alcoholic hepatitis, where AST:ALT ratios typically exceed 2:1.[3] This pattern is consistent with a MetALD flare-a term encompassing patients with both metabolic risk factors and significant alcohol intake-where hepatic lipid accumulation (compounded by both insulin resistance and alcohol-induced lipogenesis) precipitates steatohepatitis. Ultrasound demonstrating grade 1 fatty liver and CECT showing mild hepatomegaly corroborate this diagnosis. Notably, the normal pancreas on imaging reinforced the non-pancreatic origin of abdominal pain in this case.

Peripheral neuropathy: a multifactorial syndrome

The six-month history of tingling and pain in all four limbs, combined with autonomic dysfunction (orthostatic hypotension), is consistent with a combined distal symmetrical sensorimotor and autonomic neuropathy. In this patient, the neuropathy is best understood as having at least three concurrent contributors: (1) Diabetic neuropathy mediated by polyol pathway overactivation, AGE accumulation, and microvascular ischemia in the context of HbA1c 10.8%; (2) Alcoholic neuropathy due to direct axonal toxicity of acetaldehyde and reduced nerve trophic support; and (3) Thiamine deficiency resulting from reduced absorption, altered storage, and impaired utilization in the setting of chronic alcohol use.[5,9]

The administration of high-dose injectable thiamine was therefore both therapeutic and prophylactic against Wernicke's encephalopathy. Patient was advised nerve conduction velocity studies on follow up due to resource limitation

Role of plasmapheresis

Therapeutic plasma exchange (plasmapheresis) is an established rescue therapy for severe hypertriglyceridemia-induced pancreatitis, capable of reducing triglyceride levels by 50-80% within a single session.[8] In this case, it was appropriately considered but deferred given clinical stability and imaging exclusion of pancreatitis.

Current evidence supports reserving plasmapheresis for cases with documented pancreatitis, clinical deterioration, or failure of the pharmacological triglyceride reduction.[8]

The importance of comprehensive metabolic management

This case illustrates that severe hypertriglyceridemia rarely exists in isolation-it is invariably embedded within a network of metabolic comorbidities (diabetes, fatty liver disease, neuropathy, potential cardiovascular risk) that require simultaneous attention.

Treating the triglycerides alone, without correcting glycemia, addressing alcohol dependence, and providing nutritional rehabilitation, would yield transient biochemical improvement at best. The 2018 ACC/AHA lipid management guidelines emphasize a comprehensive cardiometabolic risk reduction framework, particularly for patients with combined metabolic risk factors.[7]

CONCLUSION

Severe hypertriglyceridemia is a recognized high risk metabolic condition, that can manifest with abdominal pain, neuropathy, and systemic metabolic derangements even in the absence of acute pancreatitis. This case powerfully demonstrates how the combination of uncontrolled type 2 diabetes mellitus and chronic alcohol use creates a synergistic storm of VLDL overproduction and impaired triglyceride clearance, resulting in extreme lipid elevation.

Early recognition, prompt pharmacological triglyceride reduction, aggressive glycemic correction, thiamine replacement, and strict alcohol cessation can prevent progression to life-threatening pancreatitis. Clinicians should maintain a high index of suspicion for severe hypertriglyceridemia in patients with the described metabolic risk profile, even when presenting atypically. A multidisciplinary approach addressing the metabolic, hepatic, neurological, and gastrointestinal dimensions of this condition is essential for optimal patient outcomes and prevention of recurrence.

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