

Severe Hypertriglyceridemia in Pregnancy: Clinical Approach and Management Strategies

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Abstract

Severe hypertriglyceridemia (HTG) during pregnancy is a rare but serious condition associated with an increased risk of maternal and fetal complications. We report the case of a 36-year-old woman diagnosed with severe HTG at 28 weeks of gestation, with triglyceride (TG) levels of 2184 mg/dL (SI: 24.68 mmol/L) (reference range, 26.50–150 mg/dL [SI: 0.30–1.69 mmol/L]). Despite dietary intervention and ω -3 supplementation, levels continued to rise, requiring hospitalization and treatment with intravenous insulin. Due to refractory HTG, plasmapheresis was initiated at 33 weeks, with significant lipid reduction. At 34 weeks, the patient underwent an emergency cesarean delivery due to preterm labor. This case underscores the challenges of managing severe HTG in pregnancy and highlights the importance of early, multidisciplinary intervention. Further research is needed to refine treatment strategies and establish standardized guidelines for this high-risk population.

Key Words: hypertriglyceridemia, pregnancy, plasmapheresis, intravenous insulin, triglycerides, gestational complications

Abbreviations: ω -3, omega-3; Apo, apolipoprotein; BMI, body mass index; FCS, familial chylomicronemia syndrome; HDL-C, high-density lipoprotein cholesterol; hPL, human placental lactogen; HTG, hypertriglyceridemia; LPL, lipoprotein lipase; TC, total cholesterol; TG, triglycerides; VLDL, very low-density lipoprotein.

Introduction

During pregnancy, physiological alterations in lipid metabolism occur to support fetal development. These adaptations are primarily driven by hormonal changes, leading to mildly higher triglyceride (TG) levels [1, 2].

Clinically significant gestational hypertriglyceridemia (HTG) is defined as fasting TGs greater than or equal to 500 mg/dL (SI: 5.6 mmol/L) (reference range, 26.50–150 mg/dL [SI: 0.30–1.69 mmol/L]), with levels greater than or equal to 1000 mg/dL (SI: 11.3 mmol/L) indicating high risk for pancreatitis and requiring urgent management [3]. It is most commonly secondary to conditions such as diabetes, obesity, and medication use such as antipsychotics, β -blockers, corticosteroids, thiazide diuretics, and oral estrogen therapy [4, 5]. Genetic factors affecting lipoprotein lipase (LPL), apolipoprotein (Apo)C-II, ApoA-V, ApoE, or glycosylphosphatidylinositol-anchored high-density lipoprotein-binding protein 1 (GPIHBP1) may also be involved [6, 7].

Early diagnosis is crucial to prevent maternal complications such as preeclampsia, hyperviscosity syndrome, gestational diabetes, or acute pancreatitis, a potentially life-threatening condition. Fetal risks include preterm delivery, large-for-gestational-age, and in severe cases fetal demise [8–11].

Severe HTG in pregnancy requires individualized, multidisciplinary management due to limited therapies.

Case Presentation

A 36-year-old White woman, at 28 weeks of gestation in her third pregnancy (G6 P2 A3), was referred to the endocrinology department at Germans Trias i Pujol Hospital in Badalona, Spain, due to an incidental finding of severe HTG.

Diagnostic Assessment

The patient's TG levels were elevated at 2184 mg/dL (SI: 24.7 mmol/L), with a total cholesterol (TC) of 504 mg/dL (SI: 13 mmol/L) (reference range 69–200 mg/dL [SI: 1.8–5.2 mmol/L]), HDL cholesterol (HDL-C) of 52.10 mg/dL (SI: 1.35 mmol/L), and non-HDL-C of 452 mg/dL (SI: 11.69 mmol/L). No visible chylomicrons were detected after refrigeration during 24 hours at 4 °C [12]. The 1-hour 50-g glucose challenge test was 121 mg/dL (SI: 6.72 mmol/L), ruling out gestational diabetes. The TG/HDL-C ratio was 41.92, significantly higher than values reported in the literature, suggesting insulin resistance and profound dyslipidemia [13]. The

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patient was asymptomatic, with serum amylase and lipase levels within normal ranges.

Family history revealed HTG in her father before age 55 years, and acute myocardial infarction before age 65 years in her paternal aunt, as well as a stroke in a paternal uncle before age 55 years, with unknown lipid levels.

The patient's obstetric history included 2 previous cesarean deliveries (one at 34 weeks due to suspected chorioamnionitis and another at term due to failure to progress) and 3 pregnancy losses (1 spontaneous abortion, 1 induced abortion, and 1 missed abortion requiring uterine evacuation). There was no history of severe HTG or gestational diabetes in previous pregnancies.

The patient had a history of stable ulcerative colitis. Just 1 elevated TG measurement of 289 mg/dL (SI: 3.27 mmol/L) was recorded 8 years prior to this pregnancy, with normal TC levels. The patient denied consumption of alcohol and tobacco, and was not taking any medications, aside from pregnancy supplementation (iodine and folic acid). Her pregestational body mass index (BMI) was 24.3. Physical examination revealed no xanthomas, xanthelasmas, corneal arcus, nor acanthosis nigricans.

Treatment

At the initial consultation, the patient started a low-fat, controlled-carbohydrate diet. At the follow-up visit at 29 weeks of gestation, TG levels had increased to 3068 mg/dL (SI: 34.67 mmol/L) with TC levels of 566 mg/dL (SI: 14.64 mmol/L) and non-HDL-C levels of 516 mg/dL (SI: 13.34 mmol/L), prompting hospital admission due to the high risk of acute pancreatitis. No chylomicrons were visually detected after refrigerating the blood sample for 24 hours at 4 °C [12]. The TG/HDL-C ratio was high at 61.4 as well as the TG-glucose index, which was 6.2, suggesting insulin resistance [13-15].

The patient was maintained on a strict low-fat (<20% of total caloric intake), high-protein, and controlled-carbohydrate diet, supplemented with ω -3 fatty acids (2 g twice daily) and 20 mg/day of medium-chain TG oil to compensate for caloric restriction.

At 29 weeks and 6 days of gestation, TG levels were 2619 mg/dL (SI: 29.59 mmol/L), with TC levels at 614 mg/dL (SI: 15.88 mmol/L) and non-HDL-C levels at 564 mg/dL (SI: 14.59 mmol/L). There were visible chylomicrons at 4 °C, reflecting a transient surpassing of the chylomicronemia threshold due to hormonal changes and reduced lipolysis in late pregnancy. However, their presence in a single determination suggests a nonmonogenic, transient etiology [16]. Apolipoprotein B (ApoB) was at 1.96 g/L (SI: 3.82 μ mol/L) (reference range, 0.55-1.25 g/L [SI: 1.07-2.44 μ mol/L]).

The TG/ApoB ratio was 1337 mg/dL:g/L (SI: 15.1 mmol/L:g/L), exceeding the cutoff of greater than 880 mg/dL:g/L (SI: >10 mmol/L:g/L), while the TG/TC ratio was 4.3 mg/dL:mg/dL (SI: 1.86 mmol/L:mmol/L), below the threshold of greater than 5 mg/dL (SI: >2.2 mmol/L); although this ratio alone does not strongly indicate lipoprotein excess, the elevated ApoB (1.96 g/L [SI: 3.82 μ mol/L]) and TG/ApoB ratio support a diagnosis of severe polygenic HTG [17, 18].

Considering the TG level trend over time and the absence of chylomicrons in previous samples, the most likely diagnosis was stated to be an underlying polygenic HTG. This is further supported by the elevated TG/ApoB and TG/cholesterol ratios, upper-limit ApoB, and absence of persistent

chylomicronemia, along with familial history, findings consistent with very low-density lipoprotein (VLDL) overproduction rather than familial chylomicronemia syndrome (FCS). Nonetheless, partial LPL deficiency or common variants in genes such as *ApoA-V*, *LPL*, and *ApoE* cannot be excluded. Pregnancy likely acted as a metabolic stressor, unmasking this polygenic predisposition [19].

At 31 weeks and 1 day of gestation, the patient's TG levels remained elevated at 1772 mg/dL (SI: 20.02 mmol/L), with pancreatic amylase and lipase within normal limits. An intravenous insulin infusion was initiated at 1 IU/h for 6 to 8 hours combined with 10% dextrose, to stimulate LPL activity and promote TG clearance. Glycemic control remained stable. Serial TG measurements showed a progressive decline, reaching 1245 mg/dL (SI: 14.07 mmol/L) after 12 hours, allowing for hospital discharge with ongoing dietary recommendations and ω -3 supplementation.

At 33 weeks of gestation, the patient's TG levels rose again to 2139 mg/dL (SI: 24.17 mmol/L), despite adherence to dietary modifications and intravenous insulin infusion, leading to the decision to perform plasmapheresis (Fig. 1). The procedure lowered her TG levels to 1400 mg/dL (SI: 15.82 mmol/L) at 72 hours post treatment and was well tolerated. Weekly plasmapheresis sessions were planned, with the second session performed 6 days later, at 34 weeks of gestation. The procedure was uneventful, and her TG levels, which had risen to 2229 mg/dL (SI: 25.19 mmol/L), decreased to 861 mg/dL (SI: 22.27 mmol/L) (Fig. 2).

Outcome and Follow-up

One day later, at 34 weeks and 1 day, the patient developed premature rupture of membranes, uterine activity, and self-



Figure 1. Triglyceride-rich plasma removed during plasmapheresis performed in the third trimester for severe hypertriglyceridemia.

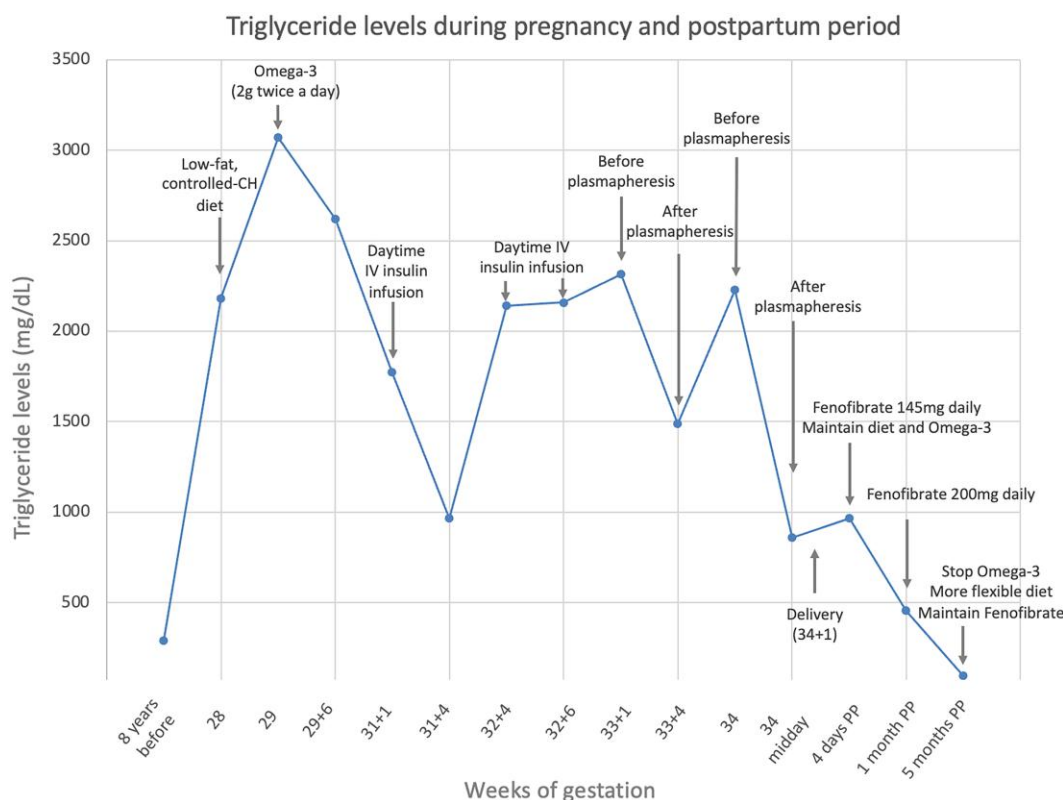


Figure 2. Triglyceride levels during pregnancy and postpartum (PP) period.

limited third-trimester bleeding, necessitating an emergency cesarean delivery. A healthy male infant was delivered, weighing 2020 g (percentile 37), with Apgar scores of 9/10/10, and artificial breastfeeding was initiated.

Four days post partum, the patient's TG levels remained at 965 mg/dL (SI: 10.90 mmol/L), and fenofibrate therapy (145 mg/day) was initiated, along with continued ω -3 supplementation (2 g twice a day) and a low-fat diet.

At the 1-month follow-up, her TG levels were 457 mg/dL (SI: 5.16 mmol/L), with the patient having abandoned ω 3 supplementation. ω -3 was reintroduced and fenofibrate was increased to 200 mg per day. At the 5-month follow-up, her TG levels had normalized to 96 mg/dL (SI: 1.08 mmol/L), with a TC level of 173 mg/dL (SI: 4.47 mmol/L) and LDL cholesterol at 107 mg/dL (SI: 2.76 mmol/L) with pharmacological treatment. These findings suggest an underlying polygenic form of HTG, secondarily exacerbated by pregnancy, and responsive to conventional therapy, which makes monogenic disorders such as FCS, typically unresponsive to standard interventions, less likely [19]. Lipoprotein A was 22 mg/dL (SI: 0.22 g/L) (reference range, 0-30 mg/dL [SI: 0-0.3 g/L]). At that point, dietary measures were liberalized, ω -3 was suspended, and fenofibrate therapy was maintained (see Fig. 2). The patient did not attend subsequent visits, resulting in loss of follow-up.

Discussion

During pregnancy, hormonal and metabolic changes gradually increase TG levels to support fetal development. As pregnancy evolves, there is a rise in estrogen and human placental lactogen (hPL) levels, which contribute to

progressive insulin resistance. This results in reduced LPL activity in maternal adipose tissue and increased hepatic VLDL synthesis, leading to higher circulating TG levels [2]. Consequently, TGs rise 2- to 4-fold, peaking in the third trimester, but usually remaining below 250 to 300 mg/dL (SI: 2.83-3.39 mmol/L) [8, 18]. Gestational HTG can be associated with pathogenic variants in genes that reduce the function or levels of *LPL*, *ApoC-II*, *ApoA-V*, *ApoE*, and *GPIHBP1*, thereby impairing TG transport and clearance mechanisms [1, 6, 7].

Secondary factors such as uncontrolled diabetes mellitus, obesity, or hypothyroidism may exacerbate TG elevation [8, 20].

HTG is associated with maternal complications such as preeclampsia, hyperviscosity syndrome, gestational diabetes, and acute pancreatitis [10, 11, 19, 21]. The fetus is at risk of preterm delivery, intrauterine growth restriction, and stillbirth [10, 11].

HTG-induced acute pancreatitis typically occurs when TG levels exceed 1000 mg/dL (SI: 11.3 mmol/L) [8, 19]. It is hypothesized that excessive pancreatic lipase-mediated TG hydrolysis generates cytotoxic free fatty acids that damage pancreatic capillaries, leading to an inflammatory cascade [8, 9, 20].

Pregnant women should be assessed for HTG risk factors, including family history, obesity, and diabetes mellitus. Early optimization of glycemic control may help prevent severe cases. Medications such as antipsychotics, β -blockers, corticosteroids, thiazide diuretics, and oral estrogen therapy can influence lipid metabolism and should be reviewed, along with lifestyle factors such as alcohol consumption and diet [5]. A physical evaluation should include assessment of BMI,

Suggested management of hypertriglyceridemia during pregnancy	
TG > 250 mg/dL	<ul style="list-style-type: none"> • Start on a low-fat, controlled-carbohydrate diet • Advise physical activity and alcohol avoidance • Screen for secondary factors* and genetic causes* • Assess current medications* • Close surveillance
TG > 500 mg/dL	<ul style="list-style-type: none"> • Reassess previous steps • Consider starting Omega-3 fatty acids
TG > 1000 mg/dL	<ul style="list-style-type: none"> • Reassess previous steps • Consider starting iv insulin infusion or plasmapheresis • Assess for potential maternal and fetal complications*

Figure 3. Suggested management of hypertriglyceridemia (HTG) during pregnancy. *Secondary causes of HTG: uncontrolled diabetes mellitus, metabolic syndrome, obesity, hypothyroidism, renal disease. *Pathogenic variants associated with gestational HTG: genes encoding *LPL*, *Apo C-II*, *Apo A-V*, *Apo E*, and *GPIIIBP1*. *Medications that can affect triglyceride (TG) levels: antipsychotics, β -blockers, corticosteroids, thiazide diuretics, oral estrogen therapy. *Maternal complications: preeclampsia, hyperviscosity syndrome, gestational diabetes, acute pancreatitis. Fetal complications: preterm delivery, intrauterine growth restriction, stillbirth.

blood pressure, and a skin evaluation for the presence of xanthomas, xanthelasma, corneal arcus, and acanthosis nigricans. Laboratory tests should encompass a complete lipid profile, thyroid and liver function tests, fasting glucose, and glycated hemoglobin A1c [5, 22]. At postpartum, women with a history of gestational hypertension, preeclampsia, gestational diabetes, or HTG should be monitored for long-term cardiometabolic risk [8].

Once HTG is diagnosed during pregnancy, dietary intervention should be initiated immediately (Fig. 3). A very low-fat diet (<20% of total calories) helps reduce chylomicron production and TG levels [2, 5, 8].

Pharmacological therapy should be considered when TG levels remain elevated at greater than 500 mg/dL (SI: 5.65 mmol/L) despite dietary intervention (see Fig. 3). ω -3 fatty acids can lower TG levels by 25% to 30% at doses of 3 to 4 g/day [2, 19]. The use of fibrates remains controversial due to potential teratogenic effects seen in animal models (US Food and Drug Administration pregnancy category C) [23, 24].

Insulin infusion has been shown to enhance LPL activity and facilitate TG clearance, as was shown in our patient [1, 19, 25, 26].

Plasmapheresis should be considered as a therapeutic option in cases of refractory HTG with TG levels greater than 1000 mg/dL (SI: 11.3 mmol/L), to prevent HTG-induced acute pancreatitis [8, 19, 25, 27] (see Fig. 3). Current evidence suggests that plasmapheresis is not associated with adverse obstetric outcomes nor with an increased risk of preterm birth, although close monitoring is recommended [28-30].

Following delivery, TG levels typically decrease as estrogen and hPL levels decline. However, they may remain elevated in patients with underlying dyslipidemias [6, 7]. Postpartum follow-up should involve lipid profiling and family history assessment to identify familial dyslipidemias. When necessary, treatment options may include fibrates, statins (if not breastfeeding), and ω -3 supplementation [8, 19, 23].

Regarding our case, the patient's HTG during pregnancy was likely driven by insulin resistance, given an elevation both of the TG/HDL-C ratio and TG-glucose index [13-15]. Additionally, a potential polygenic familial component may have contributed to the HTG, given the elevated TG/ApoB

ratio and familial history [17, 18]. The loss of follow-up prevents a complete assessment of the progression.

Learning Points

- Severe HTG in pregnancy requires early recognition and prompt management to prevent maternal and fetal complications.
- Dietary and lifestyle modifications should be the first-line approach, with therapies like ω -3 fatty acids, insulin infusion, and plasmapheresis reserved for refractory cases.
- A multidisciplinary approach is essential to optimize maternal and fetal outcomes.
- The current limited evidence highlights the need for further studies to establish optimal treatment protocols in this high-risk population.

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Contributors

All authors made individual contributions to authorship. B.S. and I.V. were involved in the clinical diagnosis, management, and follow-up of the patient. A.Rav. and B.S. contributed to data interpretation, study design, and manuscript preparation. A.Ram., N.A., and M.P.D. critically revised the manuscript and contributed to the discussions and interpretation of the outcomes. All authors reviewed and approved the final draft.

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Disclosures

The authors declare that they have no known competing financial interest or personal relationship that could have appeared to influence the work reported in this paper.

Informed Patient Consent for Publication

Signed informed consent could not be obtained from the patient or a proxy but has been approved by the treating institution.

Data Availability Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed.

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