Management of severe hypertriglyceridemia in pregnancy: case report and therapeutic strategies

7 ABSTRACT

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10 Severe hypertriglyceridemia (HTG) during pregnancy is a rare but serious complication, especially 11 in patients with pre-existing dyslipidemia. This condition carries a high risk of acute pancreatitis, 12 obstetric complications, and increased maternal-fetal morbidity. We report the case of a pregnant 13 woman with familial hypertriglyceridemia who developed acute pancreatitis during pregnancy, 14 requiring multidisciplinary management. The therapeutic approach included strict dietary 15 modification, high-dose omega-3 supplementation, and plasmapheresis, which led to a rapid 16 reduction in trialyceride levels and clinical improvement. This case highlights the importance of 17 close monitoring and early intervention in at-risk patients to prevent severe maternal and fetal 18 complications. Personalized management combining dietary measures, appropriate 19 pharmacological therapy, and extracorporeal techniques remains the cornerstone of treatment for 20 severe HTG during pregnancy.

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Keywords: Severe hypertriglyceridemia, pregnancy, pancreatitis, plasmapheresis, omega-3 fatty
 acids, dietary management, maternal-fetal outcome.

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25 INTRODUCTION

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27 Severe hypertriglyceridemia (HTGS) during pregnancy is a rare but potentially serious 28 condition, which can be life-threatening for both mother and child. While pregnancy is 29 physiologically accompanied by an increase in triglycerides, this phenomenon can become 30 pathological in women with pre-existing hypertriglyceridemia, either genetic in origin or 31 secondary to other risk factors. In these patients, pregnancy acts as a decompensating factor, 32 exposing them to an increased risk of complications such as acute pancreatitis, pre-eclampsia, 33 hyperviscosity syndrome and adverse obstetric outcomes [1,2,3,4]. Early recognition of this 34 situation and multidisciplinary management are essential to prevent maternal-fetal 35 complications.

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39 CASE REPORT:

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- 41 42 A 38-year-old woman who had been treated for dyslipidemia for 22 years, and who had 43 stopped taking cholestyramine and polyunsaturated fatty acids a year earlier, presented with 44 abdominal pain associated with vomiting for two months. MRI revealed Balthazar stage C 45 pancreatitis, simple hepatic and renal cysts, and discrete bilateral obstructive uropathy. 46 Initially hospitalized in gynecology, the patient was transferred to the maternity ward after the 47 discovery of an alarming lipid profile, revealing total cholesterol at 8.6 g/L and triglycerides 48 at 98 g/L. On admission, laboratory tests showed moderate leukopenia, anemia and normal 49 platelets. Total bilirubin was elevated, mainly in the unconjugated fraction, while liver 50 enzymes remained undisturbed. Fasting blood glucose is normal, renal function is preserved, 51 and glomerular filtration rate is increased. 52 53 The patient's blood was milky in appearance, indicating significant lipemia, which was 54 confirmed by the cloudy appearance of the plasma after centrifugation. Obstetrical ultrasound 55 revealed a progressive pregnancy with a single fetus in cephalic presentation, a fundal 56 placenta and normal amniotic fluid. 57 58 After an initial plasmapheresis session, triglycerides fell to 38.6 g/L, total cholesterol to 7.15 59 g/L, and HDL to 0.29 g/L. Lipase remained moderately elevated. Transthoracic 60 echocardiography revealed concentric left ventricular hypertrophy, a 60% ejection fraction 61 and moderate mitral insufficiency. Fundus examination revealed bilateral retinal lipemia. 62 Renal function tests were unremarkable. 63





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Figure 2 : plasma extracted from patient

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A second plasmapheresis lowered triglycerides to 21 g/L and total cholesterol to 3.3 g/L, with HDL at 0.31 g/L. Management includes prescription of high-dose omega-369, proton pump inhibitors, corticosteroid injection for fetal lung maturation, preventive anticoagulation with low-molecular-weight heparin, and close monitoring of maternal-fetal cardiac and renal functions.

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80 **DISCUSSION**

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82 In women with pre-existing hypertriglyceridemia, pregnancy is a major aggravating factor in 83 lipid imbalance. Physiological hormonal changes, notably the increase in estrogen, 84 progesterone and placental lactogenic hormone, stimulate hepatic triglyceride synthesis while 85 reducing the activity of lipoprotein lipase, the key enzyme in chylomicron catabolism. This 86 situation particularly exposes these patients to extreme triglyceride elevations, which can 87 exceed 50 mmol/L, and favours the occurrence of severe complications such as acute 88 pancreatitis, hyperviscosity syndrome, preeclampsia, and even adverse obstetric outcomes 89 such as prematurity or fetal death in utero [1,2,3,4]. 90

91 Therapeutic management of severe hypertriglyceridemia during pregnancy must be

92 multidisciplinary and rapid. It is based first and foremost on a strict dietary adjustment,

93 favoring a low-fat diet (<20% of total caloric intake) but sufficiently balanced to avoid a

94 deficiency in essential fatty acids, essential for fetal neurological and visual development

95 [1,5]. High-dose omega-3 intake has also been shown to reduce hepatic triglyceride synthesis

and promote triglyceride oxidation, with a satisfactory safety profile in pregnant women [6,7].

In the most severe forms, particularly in cases of acute pancreatitis or resistance to dietary and
 drug treatment, plasmapheresis is an effective therapeutic option, enabling a rapid and

100 significant reduction in circulating triglycerides, and an improvement in the mother's clinical

101 condition [6,8,9]. However, this technique requires specialized expertise and close

102 monitoring, particularly in terms of hemodynamics and obstetrics.

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The use of fibrates, niacin or statins remains controversial during pregnancy due to their
teratogenic potential, although there are case reports of the cautious use of gemfibrozil in
exceptional situations [1,10]. Finally, surveillance must be continuous, with close monitoring

- 107 of triglyceride levels, pancreatic function and fetal growth, in order to adapt management
- 108 according to clinical evolution.
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This comprehensive, personalized approach, combining lifestyle modification, appropriate
drug therapy, plasmapheresis if necessary and multidisciplinary follow-up, significantly
reduces maternal-fetal morbidity and optimizes the prognosis of these high-risk patients

- 113 [1,2,3,4,6,7,8,9].
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115 CONCLUSION

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- 117 Severe hypertriglyceridemia in pregnant women with pre-existing dyslipidemia represents a
- 118 therapeutic emergency requiring individualized, multidisciplinary management. A
- 119 combination of diet modification, omega-3 and plasmapheresis can rapidly reduce
- 120 triglycerides and improve maternal-fetal prognosis. Close monitoring and joint involvement
- 121 of specialized teams are essential to optimize management.
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