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CASE REPORT

Hypertriglyceridemia induced acute pancreatitis in pregnancy☆

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KEYWORDS
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Abstract Hypertriglyceridemia is the third most common cause of acute pancreatitis. The risk of developing acute pancreatitis is 5% in healthy patients and 4% during pregnancy with triglyceride levels >1000 mg/dl. During pregnancy there are changes in the lipid profile that increase between two and four times triglyceride levels. Its increase in excessive form produces an oxidative environment with injury of the endothelium and appearance of complications such as preeclampsia or pancreatitis. We present the case of a pregnant woman with pancreatitis secondary to hypertriglyceridemia.

PALABRAS CLAVE
Pancreatitis;
Hipertrigliceridemia;
Embarazo

Pancreatitis aguda por hipertrigliceridemia durante la gestación

Resumen La hipertrigliceridemia es la tercera causa más frecuente de pancreatitis aguda. El riesgo de desarrollarla es del 5% en pacientes sanos y del 4% en gestantes si las cifras de triglicéridos superan los 1.000 mg/dl. Durante el embarazo se producen cambios en el perfil

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lipídico que elevan entre 2 y 4 veces los niveles de triglicéridos. Su aumento de forma excesiva produce un entorno oxidativo con lesión del endotelio y aparición de complicaciones como la preeclampsia o la pancreatitis. Presentamos el caso de una gestante con pancreatitis secundaria a hipertrigliceridemia.

Introduction

Acute pancreatitis is one of the main reasons for gastroenterology admission. It has a mortality rate that ranges from 5% to 17% in cases of necrotising pancreatitis. The main causes include biliary lithiasis (35–40%), alcohol consumption (30%), hypertriglyceridemia (1–4%) and drugs (0.3–1.4%).[1] Elevation of triglyceride levels is a risk factor for the development of pancreatitis if they rise above 1000 mg/dl. Among others, the disorders that most commonly lead to hypertriglyceridemia are obesity, diabetes mellitus, hypothyroidism, pregnancy, drugs (oestrogens, tamoxifen, beta-blockers, etc.), and nephrotic and Cushing's syndromes.[2–4] The risk of developing pancreatitis with triglyceride levels of 1000 mg/dl is 5% in healthy subjects and, during pregnancy, from 4% to 56% depending on the series consulted.[2] We present the case of a pregnant patient with acute pancreatitis secondary to hypertriglyceridemia.

Case report

This was a 31-year-old woman with no relevant medical history, primipara, in the third trimester of pregnancy (38–39 weeks) who consulted with vomiting and hypoglycaemia. She did not have pyrexia and had no other symptoms. She was assessed by Gynaecology, who detected signs of foetal distress and performed an emergency caesarean section. She was subsequently found to have jaundice, choluria and signs of systemic inflammatory response. Initial blood tests showed: total bilirubin 9.7 mg/dl (0.3–1.2), direct bilirubin 8 mg/dl (0.01–0.2), indirect bilirubin 1.7 mg/dl (0.1–0.8), GOT 137 IU/l (6–31), GPT 104 IU/l (7–40), GGT 670 IU/l (1–30), alkaline phosphatase 255 IU/l (39–118), amylase 175 IU/l (20–110), peak lipase 451 IU/l (5.6–51.3) and peak triglycerides 3130 mg/dl (50–200), cholesterol 519 mg/dl (<200), LDL cholesterol 343 (65–175), HDL cholesterol 58 (40–60), TSH 5.98 mcU/ml (0.3–3 in the third trimester of pregnancy), free T4 1 ng/dl. Abdominal ultrasound and computed tomography scans showed thickened pancreas without necrosis, distended gall bladder with no cholecystitis or evidence of lithiasis, no intrahepatic or extrahepatic bile duct dilatation, and hepatic steatosis. Other causes of cholestasis of pregnancy, such as cholangitis, acute hepatitis, and haemophagocytic syndrome, were ruled out.

The patient had to remain in resuscitation with intensive care monitoring and symptomatic treatment. Subsequently, she was moved to the Internal Medicine ward, where progressive improvement of the analytical and radiological data was observed with symptomatic treatment.

The patient had no relevant personal or family medical history. There were no lipid or thyroid abnormalities in her pre-pregnancy analyses. Outpatient follow-up showed normalisation of all parameters without specific treatment for dyslipidemia; at her last check-up, the patient's body mass index was 27.3, and blood tests showed: cholesterol 159 mg/dl (LDL 78, HDL 71), triglycerides 52 mg/dl and TSH 1.9 mcU/ml.

Discussion

The clinical presentation of acute pancreatitis due to hypertriglyceridemia is similar to that of other causes. On physical examination, some physical findings such as the presence of xanthomas, lipaemia retinalis or hepatosplenomegaly due to fatty infiltration may help point to hypertriglyceridemia as the cause. The diagnosis is made through analytical parameters and imaging techniques (abdominal ultrasound, computerised axial tomography, magnetic resonance imaging or echo-endoscopy) that rule out other aetiologies such as gallstones.[2,3,5]

In terms of treatment, there is debate about whether hypertriglyceridemia should be treated in the acute phase. In the first 24–48 h of the condition, triglyceride levels decrease as a result of fasting, and parenteral nutrition decreases VLDL secretion from the liver which reduces the blood levels of chylomicrons. If we consider it necessary to treat hypertriglyceridemia, plasmapheresis may be used in patients with triglyceride levels greater than 1000 mg/dl who have elevated lipase 3 times above normal and signs of hypocalcaemia, lactic acidosis or organ dysfunction. If plasmapheresis is not available, is not tolerated, or plasma glucose levels exceed 500 mg/dl, regular intravenous insulin infusion may be initiated. Heparin has been used in some studies, subcutaneously or intravenously, either in isolation or in combination with insulin; its indication is unclear.[2–4]

In pregnancy, changes occur in the metabolism of lipoproteins with alterations in the lipid profile at the beginning of the second trimester. In a healthy pregnant woman, triglyceride levels multiply between 2 and 4 times due to the influence of oestrogens, progestogens and placental lactogen. The mother's triglycerides do not cross the placental barrier; the action of the lipoprotein lipase is required to hydrolyse them in order to allow the passage

of essential fatty acids to the foetus.[6,7] The problem arises when they increase to levels that can cause complications such as acute pancreatitis; one case is recorded for every 1500–4500 pregnancies.[5] The main aetiology of acute pancreatitis in pregnancy is lithiasis (70%), followed by hypertriglyceridemia (4%) and other less common causes, such as hyperparathyroidism and toxicity.[3,5] Elevation of triglycerides in pregnant women has been associated with complications such as preeclampsia, gestational diabetes and preterm labour.[7,8] The significant increase in triglyceride levels produces an oxidative environment with injury of the endothelium, which can predispose to preeclampsia. Some authors have linked maternal hyperlipidaemia during pregnancy to the increase of precursor lesions of atherosclerosis in foetuses.[7] All of the above leads to an increase in morbidity and mortality rates for both the mother and the foetus. The use of lipid-lowering agents is contraindicated during pregnancy; there are published cases of plasmapheresis causing no harm to the mother or the foetus.[9] In our case, after the emergency caesarean, both the mother and the child made good progress with conservative treatment.

Pregnant women are a risk group for the development of acute pancreatitis due to hypertriglyceridemia, since pregnancy itself favours the at times extreme increase in triglyceride levels. In view of the risks in terms of morbidity and mortality for both the mother and the foetus, pregnant women should be closely monitored for this condition.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this research.

Confidentiality of data. The authors declare that they have followed the protocols implemented in their place of work regarding the publication of patient data.

Right to privacy and informed consent. The authors have obtained the informed consent of the patients and/or subjects referred to in the article. This document is in the possession of the corresponding author.

Conflicts of interest

There are no conflicts of interest.

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