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Case Report

Acute Pancreatitis Due to Hypertriglyceridaemia in Pregnancy

Funda Gök, Selçuk Köker, Alper Kılıçaslan, Gamze Sarkılar, Alper Yosunkaya, Şeref Otelcioğlu
Department of Anaesthiology and Reanimation, Necmettin Erbakan University Meram Faculty of Medicine, Konya, Turkey

Abstract

Acute pancreatitis due to hypertriglyceridaemia during pregnancy is a rare but severe clinical condition that may cause fatal results for both the mother and the foetus. Acute pancreatitis developed in a 37-year-old pregnant woman with familial hypertriglyceridaemia and diabetes mellitus in the 31st week of pregnancy. As intrauterine foetal death developed, the pregnancy of the patient was terminated. Additionally, insulin, octreotide and plasmapheresis with "double membrane filtration" were applied, and triglycerides rapidly decreased. After 24 hours, the level of triglycerides decreased from 9742 mg dL^{-1} to 432 mg dL^{-1}. The patient was discharged from the intensive care unit at the end of 5 days and was discharged from the hospital after 32 days. The current article presents the successful treatment of severe hypertriglyceridaemia in a pregnant case.

Keywords: Acute pancreatitis, pregnancy, hypertriglyceridaemia, plasmapheresis

Introduction

Hypertriglyceridaemia (HTG) is responsible for an important part of acute pancreatitis that develops during pregnancy. In addition to familial factors, which mean that the disease is caused by genetic factors, only pregnancy may be an aetiological factor in the development of HTG (1). It was reported that severe pancreatitis is mostly seen in the third trimester, and the cause is HTG (2). If the level of triglycerides (TG) increases more than 1000 mg dL^{-1}, it is defined as severe acute pancreatitis (1).

No guideline has been defined for the management of HTG in acute pancreatitis that develops during pregnancy. Case reports and series were published, including treatment methods, such as intravenous insulin and glucose, heparin and apheresis, together with restriction of feeding (3-6). Successful management of HTG affects both maternal and foetal mortality. In the present case, severe HTG caused intrauterine foetal death. Together with the applied treatment, TG level was successfully decreased within the first 24 hours. The present article aimed to present a case with acute pancreatitis that developed due to familial HTG and to discuss the treatment modalities concerning the case.

Case Presentation

A 37-year-old patient in the 31st week of pregnancy was admitted to the Obstetrics and Gynaecology Department with complaints of severe abdominal pain radiating to the back and vomiting. The patient had a medical history of diabetes mellitus (DM) and familial HTG. As the foetus died, the patient was taken in for an emergency caesarean operation at the Obstetrics and Gynaecology Clinic and was then admitted to the intensive care unit during the postoperative period. The patient was conscious, oriented and cooperative, and there were contractions in both hands due to hypocalcaemia. Upon admission to the intensive care unit, the patient's blood pressure was 85/52 mmHg, the heart rate was 138 min^{-1}, the number of breaths per minute was 28 min^{-1}, the body temperature was 37.6°C, the acute physiology and chronic health evaluation (APACHE) II score was 15 and Ranson's score was 4. The serum taken from the patient and the bloody fluid from drainage tubes had a milky appearance. She had severe metabolic acidosis (pH: 7.12, PO_2: 132 mmHg, PCO_2: 27 mmHg, HCO_3: 8.6 m Eq L^{-1}, lactate: 4.13 mmol L^{-1}). Abnormal laboratory findings included CRP: 160 mg dL^{-1}, WBC: 11,500 mm^{-3}, Na: 129 mmol L^{-1}, calcium: 4.54 mg dL^{-1}, ionised Ca: 0.58 mmol L^{-1}, amylase: 570 U L^{-1} (normal range 25-125 U L^{-1}), lipase: 319 U L^{-1} (normal range 8-78 U L^{-1}), triglyceride: 9742 mg dL^{-1}, total cholesterol: 705 mg dL^{-1}, LDH: 788 U L^{-1}, ALT: 60 U L^{-1} and GGT: 42 U L^{-1}. Abdominal ultrasonography revealed no cholelithiasis or perihepatic fluid collection. Computed tomography scan

Address for Correspondence: Dr. Funda Gök, Necmettin Erbakan Üniversitesi Meram Tıp Fakültesi, Anesteziyoloji ve Reanimasyon Anabilim Dalı, Konya, Türkiye Phone: +90 332 223 70 30 E-mail: fundagok@gmail.com

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findings were consistent with AP. These findings were consistent with a diagnosis of AP due to hypertriglyceridaemia.

Dextrose-insulin, octreotide infusions and one session of double-filtration plasmapheresis (DMF) were performed for treatment. DMF was performed for 8 hours at a blood flow rate of 200 mL min^{-1} and plasma change of 2.5 L h^{-1}. The patient's metabolic acidosis was corrected after 6 hours, and the triglyceride (TG) levels decreased to 432 mg dL^{-1} after 24 hours. Calcium infusion (Calcium Picken 10%), at approximately 10 mL h^{-1}, was administered for the first 48 hours, achieving an ionised calcium level of 0.9 $mmolL^{-1}$. Dextrose-insulin infusion (5% dextrose 1000 mL plus 10 IU regular insulin day^{-1}) was continued for 3 days, achieving a blood glucose level of 120-180 mg dL^{-1}. Octreotide therapy, at a dose of 400 µg day^{-1}, was continued for 7 days. On the second day of her admission to the intensive care unit, the symptoms improved, and oral feeding began on the third day. On the fourth day of her admission, amylase was 107 U L^{-1}, lipase was 77 U L^{-1} and WBC was 7500 mm^{-3}, all at normal levels. On the fifth day of admission to the intensive care unit, the patient was discharged to the gastroenterology service, while the TG level was 556 mg dL^{-1}. The CRP level, being above 160 mg dL^{-1} in the first 6 days, decreased later (it was 134 mg dL^{-1} on the 15th day and 29 mg dL^{-1} on discharge from the hospital). The patient was discharged from the intensive care unit 5 days later and was discharged from the hospital 32 days later.

Discussion

High TG levels in this patient with acute pancreatitis due to familial HTG, in which intrauterine foetal death developed, was successfully treated with dextrose 5%, insulin, octreotide, and double membrane filtration.

According to the Atlanta criteria (7), acute pancreatitis is defined as the presence of abdominal pain with serum lipase levels 3-fold higher than the normal upper limit. Safi et al. (4) reported that 8 of 9 acute pancreatitis attacks that previously developed in a 28-week pregnant woman with familial HTG developed during pregnancy, and intrauterine foetal death occurred in her 2 previous gestations. Geng et al. (5) reported that 83.3% of the cases in which foetal death developed were acute pancreatitis due to HTG, and the mean TG concentration was 2.760±896 mg dL^{-1}. In the present case, the reason for the higher TG levels when compared with the levels mentioned might be familial HTG and type II DM. This is due to the fact that insulin resistance in type II DM increases TG production and decreases clearance (8). Additionally, when the TG level is greater than 500 mg dL^{-1}, amylase levels can be falsely normal, and sodium levels can be lower than normal (9).

Severe acute pancreatitis causes a decrease in placental perfusion, foetal distress and abnormal contractions. If there is severe maternal morbidity and if gestation is close to term, termination of the pregnancy should be considered. Preterm delivery prevents the development of an inflammatory reaction. In addition, delivery decreases intra-abdominal pressure, and thus, the treatment of the mother becomes easier (drainage and enteral nutrition), and lipid-lowering drugs can be used safely (10). Insulin, heparin, plasmapheresis, octreotide, oral antihyperlipidemic agents and omega-3 fatty acids are among the treatment modalities of acute pancreatitis (1, 3-6). The target is a TG value lower than 500 mg dL^{-1} with these treatments (1, 11). Insulin increases lipoprotein lipase activity and is especially beneficial in patients who have uncontrolled DM, together with severe HTG (1, 11). Heparin stimulates endothelial lipoprotein lipase secretion. Octreotide is a long-acting somatostatin analogue, and in addition to inhibiting proinflammatory cytokine secretion, it also has inhibitory activity against exocrine pancreatic secretions (12). In this case, the researchers used insulin infusion for 3 days and octreotide treatment for 7 days.

Due to limited data and conflicting reports in acute pancreatitis caused by HTG, the American Society for Apheresis Guidelines in 2010 presented one category with three recommendations (13). Many studies have demonstrated that treatment with apheresis decreased TG levels in a short period (14-16). However, data regarding when to start apheresis treatment are insufficient, and it is suggested that starting treatment in the first 48 hours is beneficial (14). Chen et al. (15) reported that apheresis in acute pancreatitis caused by HTG does not change mortality or complications. Similarly, apheresis treatment did not improve mortality and complications in pancreatitis caused by severe HTG (15).

In addition, there is no consensus on the plasmapheresis modality to be performed. This could be therapeutic plasma exchange (TPE), double-membrane filtration (DMF) or selective lipoprotein apheresis (3, 17). Basar et al. (3) found that the plasmapheresis modality is more effective than DMF. If fresh-frozen plasma is used during plasmapheresis, allergy- and transfusion-related complications are more frequently observed (16). It is supported that using fresh-frozen plasma provides additional benefits for lipoprotein lipase and apolipoprotein. During DMF procedures, the loss of low-molecular-weight plasma proteins, such as albumin and immunoglobulins, is lower than in plasmapheresis. Yeh et al. (16) performed TPE in 12 patients with severe HTG and DMF in 6 patients with severe HTG. While triglyceride levels decreased at a rate of 65%-70% with TPE in the first session, it decreased at a rate of 57.5% with DMF. In the present case, the decrease in TG level with one session of DMF was approximately 95%. Termination of pregnancy may also have contributed to this high ratio. Lipid levels decrease at a rate of 15%-20% in the first 24 hours following pregnancy and reach non-pregnant levels after 6 weeks (5). However, cases that differed from this situation were also reported. Basar et al. (3) reported a TG level of 12,000 mg dL^{-1} in a pregnant woman with familial hypertriglyceridaemia. As the TG level was 4614 mg dL^{-1} 1 month after delivery in a patient who underwent 27 sessions of apheresis during pregnancy, apheresis was performed again. Hovland et al. (17) performed selective lipid apheresis for resistant hypertriglyceridaemia. They em-

phasised that selective modalities are superior for the prevention of adverse effects of basic plasmapheresis, which would be effective in reducing cytokines and inflammatory mediators in acute pancreatitis and provide some protection from an attack of pancreatitis. However, this modality is quite costly, despite being more selective than other modalities.

Conclusion

In the present case, the presence of familial HTG, together with DM and pregnancy, caused a severe increase in triglycerides and caused acute pancreatitis and intrauterine foetal death. Together with dextrose 5%-insulin, octreotide and double-membrane filtration, TG levels decreased from 9742 mg dL^{-1} to 500 mg dL^{-1} at the end of the first day, and significant improvement was achieved in the clinical parameters of the mother.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

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