HYPERLIPIDEMIA TYPE IIB INDUCED ACUTE RECURRENT PANCREATITIS: A CASE REPORT

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Hypertriglyceridemia is a known but underestimated cause of acute pancreatitis. Although the connection between acute pancreatitis and type I, IV and V hyperlipoproteinemia has been described, using Fredrickson’s classification, the connection between type IIB hyperlipoproteinemia and associated pancreatitis has only been reported in a few more rare cases. That is why we present a female patient with recurrent hyperlipidemic pancreatitis with type IIB hyperlipidemia.

Keywords: HYPERLIPOPROTEINEMIA, FAMILIAL COMBINED HYPERLIPIDAEMIA, PANCREATITIS, DIABETES MELLITUS

INTRODUCTION

Hypertriglyceridemia (HTG) is a known but underestimated cause of acute pancreatitis and acute recurrent pancreatitis. Most frequently, type III hyperlipoproteinemia (HTG pancreatitis) is similar to other causes. A risk factor for acute pancreatitis is a serum triglyceride level of more than 2.5 to 5.18 mmol/L. In a patient with type I, IV or V hyperlipoproteinemia (Fredrickson’s classification) (1), pancreatitis secondary to HTG is typically combined hyperlipidemia (2). Although the connection between acute pancreatitis and type I, IV, and V hyperlipoproteinemia has already been described, connection between type IIB hyperlipoproteinemia and associated pancreatitis has been reported in just a few more rare cases (3, 4). We present a patient with a recurrent episode of pancreatitis associated with type IIB hyperlipoproteinemia.

CASE REPORT

A 60-year-old woman presented in the emergency room of our hospital complaining of epigastric pain and pain under both costal arches with the expansion in the back for 20 days. She had been admitted to our hospital for the second time due to acute pancreatitis with hyperlipidemia. She was already diagnosed with type IIB hyperlipoproteinemia nine years before her present hospitalisation. Also, she was diagnosed with NASH (non-alcoholic steatohepatitis) and cholecystitis. She underwent a hysterectomy due to myoma when she was 45 years old. Seven years before her present hospitalization she was for the first time hospitalised due to acute pancreatitis. Her brother and sister had epilepsy and xanthoma or xanthelasma. She didn’t consume alcohol, and she didn’t have biliary calculus. Her height was 178 cm and her weight was 87 kg, with a BMI 27.5 kg/m² showing obesity. Even though she was advised low-fat diet and prescribed medication for diabetes, she continued smoking and drinking and didn’t take prescribed medication without special reason.

On her physical examination, her abdomen was slightly painful on a deep palpation with hypoactive bowel sounds, and she had direct tenderness on the epigastrium with a rebound phenomenon. On admission, laboratory data showed a blood white cell count of 6.6 x 10⁹/L (3.4-9.7 x 10⁹/L). A blood chemistry test revealed a lactate dehydrogenase level of 278 U/L (25-41 U/L), serum lipase level of 222 U/L (13-60 U/L), serum amylase level of 60 U/L (23-91 U/L). Serum alanine aminotransferase levels of 42 U/L, aspartate aminotransferase levels of 89 U/L and gamma-glutamyltransferase levels of 1002 U/L (8-35 U/L). Serum calcium level was low 1.8 mmol/L (2.44-2.53 mmol/L), and phosphate levels were also low 0.77 (0.79-1.42 mmol/L). Her lipid profiles, such as serum total cholesterol (TC) and triglyceride level (TG), were 22.5 mmol/L (5-5.2 mmol/L) and 24.8 mmol/L (1.7-2.2 mmol/L), respectively, but levels of high-density cholesterol (HDL) and low-density cholesterol (LDL) could not be measured in the laboratory. While taking blood for analysis, it is important to note that the blood was turbid and creamy. It took more than 10 hours to analyse lipid profiles. Dietary restriction and hydration were started immediately after hospitalization. Plasma pharmacists was the first therapy option, but after a blood chemistry test, therapy was started with 24000 units of heparin in 500 ml of 0.9% physiological saline for 24 hours and 8 units of fast-acting insulin in 10% gluco- se solution for 12 hours. The next day lip- id profile levels were measured again. Levels of TC, TG, HDL, LDL were 11.9 mmol/L, 4.7 mmol/L, 1.56 mmol/L, (1.2 mmol/L) and 9.42 mmol/L (<3 mmol/L), respectively, with normalisation of hepatic enzymes. After 3 days, statin therapy was re-introduced. Abdominal CT scan revealed normal density but enlarged li- ver and spleen (17.5 cm and 14 cm respectively). Abdominal MR showed signs of oedema of the pancreatic tail. Post-con- trast injection showed no signs of ne- crosis. Surrounding fatty tissue showed increased signals. The patient diet was restricted to pancreatic fluid, and all in terms of acu- te pancreatitis. Her abdominal pain was resolved within a few days, and she was discharged after 10 days with a complete recovery from acute pancreatitis.

DISCUSSION

The mechanism by which hypertri- glyceridemia leads to pancreatitis was suggested by Havel et al (5). Hypertri- glyceridemia can be a consequence of increased VLDL (very low-density li- poprotein) production, decreased VLDL and/or chylomicron catabolism, or most likely both these mechanisms. As a re- sult, hypertriglyceridaemia results in an increase in high triglyceride TG level in the following types of lipoproteins: 1) VLDL (familial hypertriglyceridaemia, known as Fredrickson type IV hyperli- poproteinaemia (HLP), or familial combi- ned hyperlipidaemia (Fredrickson III); and LDL cholesterol level is also increased); 2) VLDL and chylomicrons (HLP type IV); 3) VLDL remnants and chylomicron remnants (dysbeta lipoproteinaemia, also known as remnant disease or HLP type III); and 4) chylomicrons only (HLP type I) (6). The most frequent is fami- liar hypertriglyceridaemia (5-10% of the population), which is related to genetic factors and secondary factors superimposed on genetic susceptibility, including environmental factors, leading to an elevation of TG level is usually in the range of 5.18-12.95 mmol/l (Less frequent polygenic hyper- triglyceridaemia (1-2% of the population) include familial combined hyperlipopeno- teinaemia which results from an increa- sed hepatic synthesis of apolipoprotein B (apo B), leading to an increase in VLDL production (7). An even less frequent condition is dysbetalipoproteinaemia (0.01% of the population) (7). IFG level exceeds 5.6 mmol/L, and in particular 11.3 mmol/L, lipoprotein lipase (LPL) becomes saturated by VLDL triglyceri- de, and chylomicrons appear in plasma in fasting conditions. By this mechanism, fasting chylomicrons may de- velop in familial hypertriglyceridaemia, familial combined hyperlipidaemia, and dysbetalipoproteinaemia, leading to HLP type IV.

The most complicated scenario of severe hypertriglyceridaemia (SHTG) with fasting chylomicronaemia is acute pancreatitis. SHTG is the third-common cause of acute pancreatitis after alcohol abuse and cholelithiasis, and it is respon- sible for up to 10% of all episodes of acu- te pancreatitis (9). It is interesting that the rates of SHTG among patients with acute pancreatitis in 2006 studied were 12%, 21%, and 22% (10). Altho- ugh the threshold TG level for an increa- sed risk of pancreatitis has not been defined, it is often arbitrarily set at >11.3 mmol/L. The exact mechanism for acute pancreatitis due to SHTG is not known. It has been assumed that high low-density-lipoprotein (LDL) cholesterol levels (0.01% of the population) (7). If TG level excesses 5.6 mmol/L, and in particular 11.3 mmol/L, lipoprotein lipase (LPL) becomes saturated by VLDL triglycerides, and chylomicrons appear in plasma in fasting conditions. By this mechanism, fasting chylomicrons may develop in familial hypertriglyceridaemia, familial combined hyperlipidaemia, and dysbetalipoproteinaemia, leading to HLP type IV.

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Familial combined hyperlipidaemia is one of the most common hereditary lipoprotein disorders that affects at least two family members. Despite its prevalence and potential health consequences, it is often underdiagnosed and undertreated (15). Although acute pancreatitis is a rarely described complication, earlydi- agnosis, and treatment of this hyperlipo- proteinaemia are necessary, assis- tance of two family members.
For sure, accurate and fast diagnosis with diet, lifestyle changes, and medications is important for the treatment and prevention of recurrent HTG pancreatitis and the long-term management of TG levels.

**LITERATURE**


**Sažetak**

**AKUTNI REKURENTNI PANKREATITIS IZAZVAN HIPERLIPIDEMIJOM TIP IIb: PRIKAZ SLUČAJA**

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Hipertrigliceridemija je poznat, ali podcijenjen uzrok akutnog pankreatitisa. Iako je već opisana veza između akutnog pankreatitisa i hiperlipoproteinemije tipa I, IV i V, korištenjem Fredricksonove klasifikacije, veza između hiperlipoproteinemije tipa IIb i pridruženog pankreatitisa zabilježena je samo u još nekoliko rijetkih slučajeva. Upravo zbog toga prikazujemo pacijentu s rekurentnim hiperlipidemijskim pankreatitismom s hiperlipidemijom tipa IIb.

Ključne riječi: HIPERLIPROTEINEMIJA, OBITELSKA MJEŠOVITA HIPERLIPIDEMIJA, PANKREATITIS, DIJABETES MELITUS

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