ROUX-EN-Y BILIARY BY-PASS – A NEW APPROACH IN THE TREATMENT OF HYPERTRIGLYCERIDEMIA INDUCED RECURRENT ACUTE PANCREATITIS. CLINICAL CASE STUDY

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Acute pancreatitis is a disease with significant mortality. Hypertriglyceridemia (HTG) is the third most common etiological factor of this disorder after alcohol and gall-stones. The authors presented a case of 42-years old caucasian female who was hospitalized due to recurrence of acute pancreatitis. She had been diagnosed with HTG. She had earlier seven episodes of acute pancreatitis. Endoscopic papillotomy and conservatory treatment didn’t change her complaints and she was consented for surgery. Exclusion of distal part of bile duct was performed. The common bile duct was anastomosed side-to-side to the 70 cm long Roux loop of the jejunum with the ligation of the distal part of the common bile duct. Following the surgery authors observed normalization of amylase, lipase, leukocytosis and CRP levels. During six months after procedure patient didn’t have any new episode of pancreatitis. Exclusion of distal part of bile duct may be a useful tool in surgical treatment of recurrent acute hypertriglyceridemia-induced pancreatitis.

Key words: acute pancreatitis, hypertriglyceridemia, surgical treatment
relative insufficiency of insulin decreases the ability of lipoprotein lipase in adipocytes to reduces TG into fatty acids. In type 2 diabetes insulin resistance enhances production and reduced clearance of TG. All these mechanisms result in elevation of TG concentration and increased liver production of very low-density lipoproteins (VLDL) (5). There are many other documented factors leading to HTG such as some medications (protease inhibitors, estrogen, tamoxifen, clomiphene, olanzapine, propofol) and hypothyroidism (6).

HTG is often accompanied by cholelithiasis. Biliary microlithiasis – difficult to diagnose – are one of the etiological factors of acute pancreatitis, whereas symptomatic gall-stone disease is met in about 16% of patients with HTG (7). Changes in gall-bladder motility and bile acids metabolism, crucial in the pathogenesis of gall-stones, are linked to changes in triglyceride metabolism. Bile overloaded with cholesterol in patients with HTG facilitates processes of its nucleation and crystallization.

Insulin and heparin, which enhance lipoprotein lipase activity, play the essential role in the treatment of HTG (8). Other available modalities – apheresis and plasma exchange – allow to decrease the serum concentration of TG. Majority of investigations on apheresis associated with HTG reports that plasmapheresis lowers TG levels by 60-70% during one session (9). Well known side effect of plasmapheresis is loss of immunoglobulins and coagulation factors along with TG. Selective lipoprotein apheresis is devoid of this disadvantage, but also less effective in lowering TG (10). Oral pharmacologic therapy of HTG contains usage of fibrates, which significantly (by 40-60%) reduce TG levels and raise HDL cholesterol levels. Less effective is niacin – lowering TG by 30-50%. Some patients, especially those with familial hyperlipidemia, can benefit from combination of simvastatin and ciprofibrate (11). Obviously lifestyle changes and treatment of comorbidities can’t be omitted. Diet low in fat, carbohydrates and alcohol should be followed along with Ω-3 fatty acid rich fish oil supplementation.

**CASE REPORT**

42-years old caucasian female was admitted to the hospital due to recurrence of acute pancreatitis. Diagnosis was based on typical clinical symptoms confirmed by laboratory and radiological findings. The examination revealed distended, painful abdomen, diminished bowel sounds and tachycardia. Blood pressure was normal. Half year earlier for similar reasons she was submitted for ERCP with papillotomy. This procedure showed presence of biliary sludge. Since then she was put on oral ursodeoxycholic acid and – due to diagnosed HTG – hypolipidemic agents (atorvastatin). Before papillotomy she had four episodes of acute pancreatitis, which were treated conservatively. In the period between papillotomy and the last hospital admission there were next three more episodes of pancreatitis. Twelve years ago she did have cholecystectomy because of acute gall-stone cholecystitis. She was diagnosed then with type 2 diabetes, liver steatosis and hypertension.

Initial laboratory findings revealed: white blood cells (WBC) 20270/cm³, c-reactive protein (CRP) level 6.17 mg/dl, serum amylase 374 IU/l, serum lipase 1657 IU/l, urine amylase 7078 IU/l. Other results were as following: alkaline phosphatase (ALP) 89 IU/l, total bilirubin 0.19 mg/dl, gamma-glutamyl transpeptidase (GGTP) 134 IU/l, total cholesterol 256 mg/dl, low-density lipoprotein (LDL) cholesterol 153 mg/dl, high-density lipoprotein (HDL) cholesterol 36 mg/dl, TG 993 mg/dl. Renal function tests and coagulogram were normal.

Ultrasound scans of the abdomen demonstrated enlarged (10 cm below of costal margin), not homogenous liver, enlarged (up to 48 mm) pancreatic head with ill-defined margins and normal main pancreatic duct. There was no evidence of fluid in peritoneal cavity. Magnetic resonance cholangiopancreatography (MRCP) showed not dilated intra- and extrahepatic bile ducts, slightly narrowing of hepatic ducts in the region of the confluence – probably post inflammatory changes, common bile duct of the diameter of 11 mm. The contents of bile and pancreatic ducts have homogenous, correct signal of fluid. She was initially treated conservatively with intravenous fluids, antibiotics, painkillers and subcutaneously administered low molecular weight heparin and insulin.

Because of maintaining symptoms of pancreatitis the patient was consented for surgery. Our intention was to exclude distal part of bile duct to prevent bile from contact with pancre-
Roux-en-y biliary by-pass in the treatment of hypertriglyceridemia induced acute pancreatitis

The common bile duct was anastomosed side-to-side to the 70 cm long Roux loop of the jejunum (this loop was made after transection of proximal jejunum about 20 cm. from the ligament of Treitz) and the distal part of the common bile duct was ligated below of anastomosis with use of unabsorbable suture. Following the surgery we observed normalization of amylase, lipase, leukocytosis and CRP levels. Postoperative course was uneventful and she was discharged after 15 days with antibiotics, antiphlogistics, analgesics, ursodeoxycholic acid and simvastatin prescribed. Regular follow-up for six months in our out-patients clinic didn't reveal any complication, no episode of abdominal pain was noted. Current laboratory findings are as following: TG – 245 mg/dl, total cholesterol – 164 mg/dl, LDL cholesterol – 65 mg/dl, HDL cholesterol – 50 mg/dl, WBC – 8.77 G/l, ALP – 102 IU/l, GGTP – 22 IU/l, total bilirubin – 0.44 mg/dl, alanine aminotransferase – 16 IU/l.

DISCUSSION

The association between acute pancreatitis and hyperlipidemia is well established. To our knowledge, this is the first case which describes surgical treatment of acute recurrent HTG induced pancreatitis. As a result of this procedure the bile goes directly to the jejunum with omitting of the ampulla of Vater. The results of Roux-en-Y biliary bypass on the cholesterol metabolism is still not well recognized. Several studies referring to bariatric surgery suggest a decrease of 15-20% in serum total and LDL cholesterol, 40-60% decrease in serum TG and 20-40% increase in HDL cholesterol following Roux-en-Y gastric bypass (12, 13).

As the high levels of TG are the major risk factor for acute pancreatitis, effect of this procedure should reduce the risk of recurrence of pancreatitis in our patient. Mechanism of absorption of cholesterol and TG after this procedure seems to be impaired because of reduced intestinal absorption area and alterations in circulation of bile acids and sterols.

HTG is well known independent risk factor and is involved in the atherogenic process. Via this mechanism, it may contribute to prethrombotic state and thrombogenesis resulting in development of cardiovascular diseases (14). Similarly it may lead to thrombosis in pancreatic vessels and then to the pancreatitis. Procoagulation state may be the result of the abnormal plasma lipoprotein oxidative modifications. Studies of Huai Bai et al. found that decreased HDL and elevated LDL and VLDL in HTG subjects can accelerate activation of factor VII, factor X and prothrombin (15). During follow-up we observed improvement of lipid profile especially with decrease of TG. Up till now patient didn't have any new episode of pancreatitis. It may be the result of improvement of lipid profile leading to the diminished risk of thrombotic processes in pancreatic vessels and exclusion of bile ducts leading to lack of contact between bile and pancreatic duct.

REFERENCES


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