Current Knowledge of the Hypertriglycerideremic Pancreatitis

Mini Review

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Hypertriglyceridemia is a well-known cause of acute pancreatitis. The triglyceride level of more than 1000 to 2000 mg/dl is a dangerous risk factor [1-9]. On the other hand severe hypertriglyceridemia with concentrations > 1000 mg/dl can also interfere with clinical laboratory test, making patient diagnosis and management more difficult. Hypertriglyceridemia can be primary in the 5% of the cases and more often secondary to other causes such as diabetes, obesity, excess carbohydrate intake, hypothyroidism, alcohol, pregnancy, hepatitis, sepsis, Kidney failure and drugs like cyclosporine, thiazide, glucocorticoids, B-blocker, estrogen and so on [1,3,6].

Chylomicrons are triglyceride-rich lipoprotein particles. They are present in the circulation when triglycerides are more than 900 mg/dl. These are large enough to occlude the pancreatic capillaries leading to ischemic and subsequent acinar structural alteration. Chylomicrons are the product of dietary fat absorption. Therefore, abstinence from eating after pancreatitis may allow rapid metabolism of the triglyceride-rich chylomicrons. Other measures, non-pharmacological include loss of weight, exercise, reduced calories, fat and refined carbohydrate intake and control of potential concomitant endocrinopathy (e.g. diabetes mellitus and hypothyroidism are imperative measure in the management of hypertriglyceridemia) [4-6].

Fibrates are the mainstay of therapy, but when the acute pancreatitis is severe, this treatment is slow. The fibrates reduce plasma triglyceride levels by to 45 to 50% and raise the high-density lipoprotein (HDL) cholesterol by 15 to 20%. Besides, fibrates modulate peroxisome proliferator activate receptors alfa in the liver with decreased secretion of VLDL and the other hand increased lipolysis of the triglycerides in plasma. Currently the literature talk about the role of omega-3 associated with fibrates [1,4,7].

In the acute pancreatitis, have been used in the reduction of recurrent episodes antioxidant therapies such as selenium, B carotene, vitamin C, alfa-tocopherol [6].

Other novel therapies include the use of insulin and heparin, lipoprotein lipase gene therapy and plasmapheresis [5]. Insulin treatment is considered helpful in lowering triglyceride, because insulin activates LPL leading to an acceleration of the chylomicron degradation. Heparin stimulates the release of endothelial LPL into the circulation, but this treatment is subject to controversy due to the only transient rise in LPL followed by increased degradation and depletion of plasma store resulting in LPL deficiency. Gene therapy is potentially useful for causes of severe hypertriglyceridemia with documented LPL deficiencies [1,5,7]. The treatment consists of local intramuscular application of a viral vector containing a LPL gene.

In case of acute pancreatitis with severe hypertriglyceridemia (triglyceride level in serum > 1000 mg/dl) rapid reduction of levels to well below 1000 mg/dl can improve outcome and prevent further episodes of pancreatitis. Plasmapheresis is a therapeutic option in such medical emergencies because conventional management of hypertriglyceridemia dietary fat restriction and pharmacotherapy is time consuming [5-9].

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The indications of plasmapheresis [5-7] in such cases are: 1) Serum triglyceride exceed 1000 mg/dl. 2) Patient refractory to nutritional and pharmacological approaches. 3) Serum lipase 3 times the upper limit of normal. 4) Severe hypocalcemia. 5) Lactic acids and 6) Worsening inflammation and organ dysfunction.

The beneficial effect of plasmapheresis is believed to be because of rapid decrease on triglyceride levels [6-8], however removal of excessive proteases from the plasma which are key enzymes in inflammation and replacement of consumed protease inhibitors might be and additionally benefit. According to the data of Lennertz, et al [8] and Yet., et al [9] a single session of plasmapheresis proofs capable of dramatically lowering triglyceride levels by up to 70% producing clear clinical and laboratory improvement of acute pancreatitis.

The timing of initiation of plasmapheresis might be crucial. There are reports showing that reduction in morbidity and mortality with plasmapheresis can be achieved when is used early. Patient may undergo plasmapheresis with 48 hours of diagnosis [1,6,8]. A study showed a significant improvement in their general condition and a regression of pain.

Technical details concerning apheretic treatment are also under investigation. Plasma exchange therapy seemed slightly more efficient than double membrane filtration [1,8].

Bibliography