Pitfalls in hypertriglyceridemia induced acute pancreatitis

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Acute pancreatitis one of the most common cause of hospitalization in gastroenterology clinics (1). Acute pancreatitis has high morbidity and mortality rates unrelated to its etiology (1,2). The most common etiologies are gallstone and alcohol abuse (2). However, many developments have occurred in diagnostic tools like increased availability of endoscopic ultrasound in gastroenterology clinics, it is still impossible to find out any etiologic factor in some cases with acute pancreatitis which is called idiopathic acute pancreatitis. Hypertriglyceridemia has been reported as an etiologic factor in 3-5% of cases with acute pancreatitis (1). This etiology of acute pancreatitis has some specific features and pitfalls in practice.

In the article by Sezgin O et al, the authors evaluated their cases with acute pancreatitis induced by hypertriglyceridemia and compared their demographic and clinical features, severity, local and systemic complications, morbidity and mortality rates with acute pancreatitis cases caused by other etiologies (1). The most common hypertriglyceridemia occurs due to familial dyslipidemia type 1, 4 and 5. Even it represents the experience of single center, this article is relevant to our practice, because it gives information about a rare hereditary cause of acute pancreatitis. If the index disease is caused by a familial disorder then local data gets great value.

Hypertriglyceridemia induced acute pancreatitis has some different aspects from acute pancreatitis caused by other causes.

Hypertriglyceridemia induced acute pancreatitis is hard to diagnose.

Acute pancreatitis is diagnosed as having 2 out of 3 factors; characteristic abdominal pain, elevated amylase and lipase serum levels more than 3 times of normal upper limits and radiologic signs of acute pancreatitis (3). The initial symptoms may not be typical, pain pattern and severity can be in very hard to characterize. Amylase level may be normal due to high triglyceride concentration in the serum which may make the clinician rule out the diagnosis of acute pancreatitis (3). And patients with hypertriglyceridemia induced acute pancreatitis are mostly obese, so ultrasonography has some limitations to show acute pancreatitis. Also, it is not common approach for clinicians to order serum triglyceride level measurement in patients with abdominal pain in emergency room. When the clinician decides to make differential diagnosis a few days after hospitalization of the patient, the triglyceride level may return to normal values.

Its treatment strategy has not been well established.

Treatment of patients with hypertriglyceridemia-induced pancreatitis is management of acute pancreatitis and lowering serum triglyceride levels with the aim of preventing local - systemic complications (3). Management of a patient with acute pancreatitis includes supportive care with fluid resuscitation, pain control, and nutritional support. Still there is no well-established treatment strategies or published guidelines for the treatment of hypertriglyceridemia induced acute pancreatitis (3). Even insulin therapy is a recommended treatment of the disease, still there has been no comparative study demonstration its superiority against supportive management (2,3). The role of heparin in the management of hypertriglyceridemia-induced pancreatitis is controversial (1,3). Heparin has a transient effect in lowering triglyceride levels, it may have potential risk of lipotoxicity from free fatty acids and increased incidence of bleeding. Plasmapheresis removes triglycerides and chylomicron from the circulation very rapidly. But studies comparing plasmapheresis and conservative management have failed to show any differences in clinical outcomes (3). Some expert centers recommend plasmapheresis in patients with worrisome clinical features with citrate as an anticoagulant. So, there is great need for more comparative, prospective randomized high-quality studies in the treat-

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ment of hypertriglyceridemia induced acute pancreatitis. A simple treatment algorithm for hypertriglyceridemia induced acute pancreatitis which is modified from the article by Garg and Rustagi is given in Figure 1.

Hypertriglyceridemia induced acute pancreatitis is a more common and more important problem in pregnant women.

As the authors of the study mentioned more than half of acute pancreatitis diagnosed in pregnant women is due to hypertriglyceridemia (1,4). Hormonal changes in pregnancy can increase otherwise controlled lipid levels in women with familial hypertriglyceridemia (3). Acute pancreatitis in a pregnant woman may cause increased morbidity and mortality for mother and fetus. Pancreatitis should be kept in mind in a pregnant woman with abdominal pain and uterine contractions in the emergency room. The diagnose may be more difficult in this specific population because of the limited diagnostic role of physical examination and ultrasound due to pregnancy. The computerized tomography scan cannot be a choice

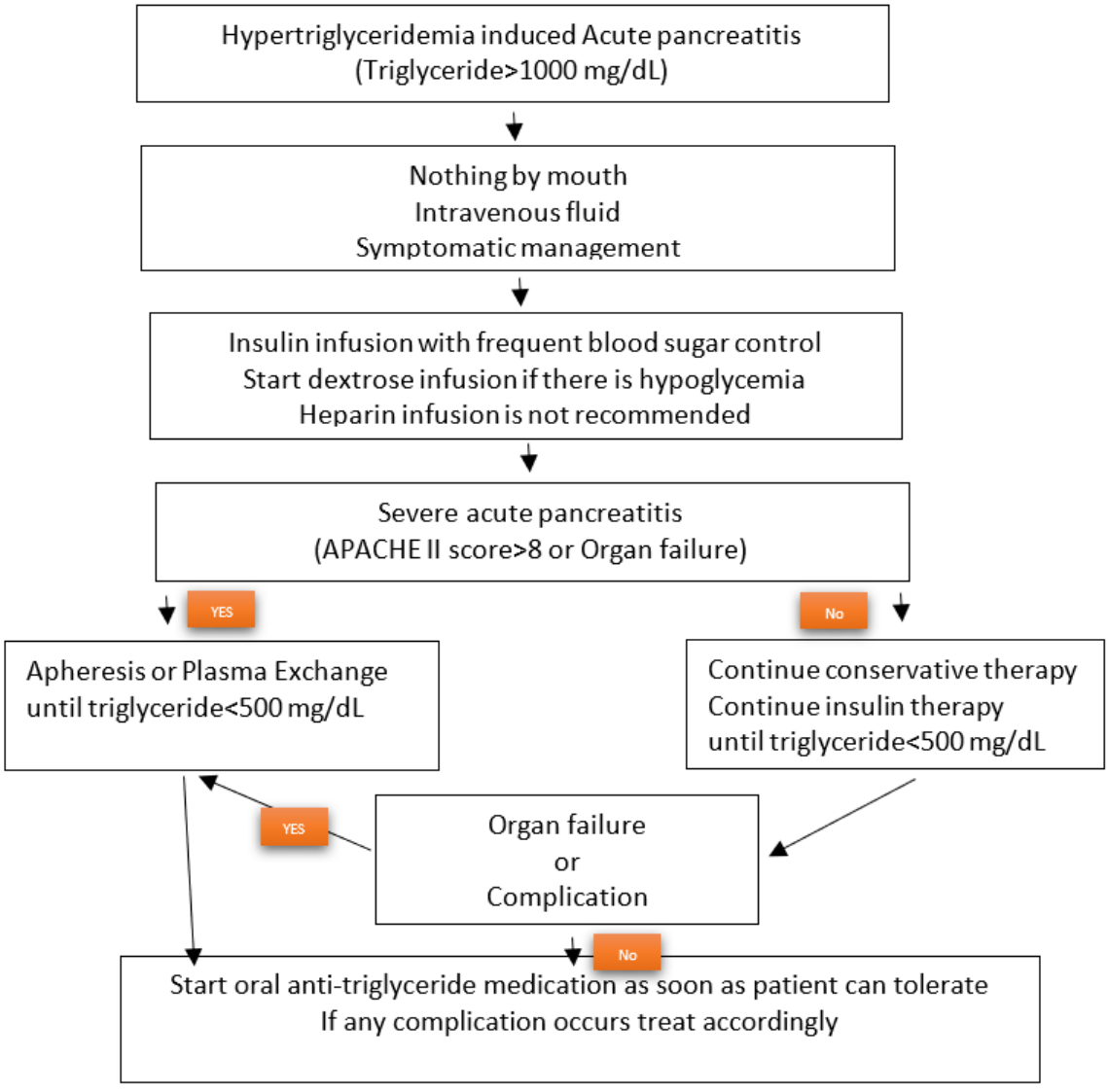

Figure 1. Simple treatment algorithm for hypertriglyceridemia induced acute pancreatitis (3)

for the imaging of pancreas in these patients. There are some case reports about the fetal and maternal complications during hypertriglyceridemia induced acute pancreatitis in the literature (4,5). But there are no evidence showing that the complication and mortality rates are greater in pregnant woman. The treatment is same as nonpregnant cases and mostly conservative (5).

Due to its nature, hypertriglyceridemia induced acute pancreatitis has tendency to be cause of recurrent acute pancreatitis.

The underlying cause of hypertriglyceridemia induced acute pancreatitis cannot be cured permanently. So, recurrences are common, when there is hormonal and metabolic changes in patient with previous history of acute pancreatitis due to hypertriglyceridemia (3). So, patients should have an educational program about the diet, importance of continuous use of medications and regular monitoring of serum triglyceride levels.

Even this study is very much relevant for our colleagues and exciting to read all these data brought together by the authors. The study has some important limitations too. It is a retrospective study reported from a single center with cases collected in ten years. Many changes and development happened during this time period in the classification, diagnosis and treatment modalities of acute pancreatitis. Many improvements have done in the guidelines. So, the first and last case could not be evaluated in same manner. The number of patients with hypertriglyceridemia induced acute pancreatitis group is relatively small in the study. They were not randomly selected or classified according to the severity, there is no previously defined objective criteria for admission to hospital -normal ward or intensive care unit- or for the decision for treatment modality. These were caused most probably due to retrospective nature of the study. But it supports the gastroenterologist for very important data about this specific group of acute pancreatitis etiology and compares it with other etiologies. The gastroenterologist with specific interest of pancreatic diseases should design multicenter, randomized and prospective studies to establish the most appropriate diagnostic and management modalities in this very special group of acute pancreatitis.

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