Early Plasmapheresis for Treatment of Acute Pancreatitis Related to Hypertriglyceridemia

Hipertrigliseridemiye Bağlı Akut Pankreatit Vakasında Erken Plazmaferez Uygulaması

Hypertriglyceridemic Acute Pancreatitis

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Özet

Akut pankreatit pankreasın ani inflamasyonu ile karakterizedir. Hipertrigliseridemi akut non-bilier pankreatitin önemli fakat genellikle atlanan, göreceli olarak nadir rastlanan bir nedenidir. Aferez çoğunlukla konzervatif tedaviye yanıt vermeyen hiperlipidemik olgularda uygulanır. Bu olgu sunumunda tip I hiperlipidemi ve tekrarlayan pankreatit atakları olan bir hastada erken plazmaferez uygulaması örneği verilmektedir.

Anahtar Kelimeler

Akut Nekrotizan Pankreatit; Hipertrigliseridemi; Plazmaferez

Abstract

Acute pancreatitis is characterized by a sudden inflammation of the pancreas. Hypertriglyceridemia is an important but usually unnoticed, relatively rare cause of acute non-biliary pancreatitis. Apheresis is mostly an option for hyperlipidemic cases failing to respond to conservative therapy. This study reports a case of acute necrotizing pancreatitis with a medical history of type I hyperlipidemia and recurrent attacks of pancreatitis for which early plasmapheresis was performed.

Keywords

Acute Necrotizing Pacreatitis; Hypertriglyceridemia; Plasmapheresis.

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Introduction

Acute pancreatitis (AP) is characterized by s sudden inflammation of the pancreas in which tissue architecture may be conserved (except interstitial edema and inflammatory cells in acute edematous pancreatitis) or destroyed by hemorrhagic and necrotic areas (in acute hemorrhagic/necrotizing pancreatitis). Inappropriate activation of pancreatic enzymes and then the autodigestion of pancreatic tisssue form the basis of the pathophysiology of AP. Focal or extensive fat necrosis can be revealed under a microscope in necrotizing form. Severity of the disease is based on clinical symptoms and laboratory and radiological findings. Mild disease cases usually recover uneventfully. However, local or systemic complications with organ dysfunction are inevitable if the disease is severe. Most cases are related to alcohol use or to biliary stones.

Severe hypertriglyceridemia is the most common cause of acute pancreatitis after biliary stones and alcohol use, according to the literature [1,2]. Hypertriglyceridemia is an important but unnoticed cause of acute non-biliary pancreatitis, responsible for approximately 3% of all acute pancreatitis cases [3]. On the other hand, acute pancreatitis occurs in 12-38% of hyperlipidemic patients [2,4]. Local excessive release of free fatty acids and lysolecithin from lipoprotein substrates exceeding the transport capacity of albumin, and therefore the development of injury in acinar cells and microvascular membranes, is the probable cause of acute pancreatitis in hyperlipidemic patients [5,6]. The main target of treatment is to lower serum triglyceride level and to repress the systemic inflammatory response. Patients with excessively elevated triglyceride levels need a fast and effective lowering of these levels in order to prevent a severe pancreatitis episode, so immediate apheretic treatment may be an option for a rapid lowering of these levels. Recently, the use of plasmapheresis for treating patients with severe hypertriglyceridemia has not only been confirmed in a number of studies, but has also been suggested by the American Society for Apheresis (ASFA) Committee on Clinical Applications [7].

Case Report

A 22-year-old male patient presented to the emergency room with abdominal pain and vomiting. The pain, which was obscure and localized from the upper part of the umbilicus to the lumbar region, had started early in the morning. Intermittent exacerbations, relieved by bending forward, and nausea and vomiting were other characteristics of the pain. His medical history was positive for continuous drug use to treat type I hyperlipidemia and past hospitalizations due to acute pancreatitis episodes; he had no related family history. Physical examination was unremarkable other than tenderness in the periumbilical region on superficial and deep palpation. There was no pathological finding on arterial blood gas analysis, postero-anterior chest X-ray, or electrocardiogram. Serum triglyceride and amylase levels were found to be high (2142 mg.dL-1 and 268 IU.L-1 respectively) in biochemical investigation. Minimal pleural effusion in scanned sections of the thorax, minimal ascites, diffuse enlargement of pancreatic size, lack of contrast enhancing on pancreatic head and body, and peripancreatic fluid collection consistent with acute necrotizing pancreatitis were determined on CT scan. The patient was taken into the intensive care unit (ICU).

Crash fluid rescucitation, pain relief, cessation of oral intake, antihyperlipidemic agents, insulin, and low molecular weight heparin were the early approaches for treatment. Plasmapheresis with fresh frozen plasma (FFP) was performed at the second hour of hospitalization. Serum triglyceride level was reduced to 270 mg.dL-1 after the procedure. A single application was enough for clinical and laboratory recovery in this case. Medical treatment was then continued. A control tomography on the 36th hour of hospitalization revealed decreased amount of peripancreatic fluid. The patient was transferred to service on the sixth day of ICU follow up.

Discussion

Necrotizing pancreatitis is the most serious form among acute pancreatitis (AP) types. Because of its high morbidity and mortality, clinicians become alert in cases of diagnosis or even suspicion of this clinical scenario. Sepsis, acute respiratory distress sydrome, and multiple organ failures are the most common causes of mortality in such cases. Hypertriglyceridemia is responsible for approximately 3% of all AP cases. Serum triglyceride level over 500 mg.dL-1 can induce an attack [3]. Necrosis tissue forms in 9-20% of pancreatitis attacks [7]. Treatment of necrotizing pancreatitis has been a popular topic for the last 10 years. As a result of studies performed, investigators propound that there are two stages in the progress. In the first stage, which is 14 days long, there is a systemic inflammatory response caused by inflammatory mediators released from necrotic tissue [9]. There is no need to expect an accompanying infection causing organ failure or mortality in this phase. The second stage starts on the 15th day and is characterized by septic process due to infection of necrotic tissue [9]. Necrosis has been found to be accompanied by an infection in 40-70% of cases and the mortality risk is higher in such cases [10].

Hyperlipidemia, which can be an etiologic factor or the result of acute pancreatitis, should be kept in mind in the differential diagnosis of acute non-biliary pancreatitis. Different studies report different ratios of hyperlipidemia in AP. However, similar rates of it are detected in both edematous and necrotizing pancreatitis [11]. Limited data about the pathogenesis of hyperlipidemic pancreatitis is currently available. Nevertheless, it is believed that acinar cells and the capillary endothelium are damaged by free fatty acids. The clinical scenario is not different in hyperlipidemic pancreatitis; abdominal pain, nausea, and vomiting are the most common presenting complaints. As a clinical distinction, serum amylase level may be normal in these cases because hyperlipemic plasma interferes with the determination of actual serum amylase level [12]. Hyperlipidemic pancreatitis is more severe than other pancreatitis forms in terms of its clinical course and complication rate, but mortality rates are the same [13]. Treatment is controversial and there is no published guideline for this topic. Insulin and/or heparin, which increase lipoprotein lipase activity, or apheresis, which removes triglycerides, are treatment options in the literature mentioned above [14]. However, there is not yet adequate clinical experience for the routine use of these therapies. Apheresis is mostly an option for the cases failing to response to conservative therapy [14]. The main target of treatment is to lower

the serum triglyceride level and to repress the systemic inflammatory response. Heparin and insulin accelerate the degradation of chylomicrons by increasing lipoprotein lipase activity in addition to enhancing microcirculation and preventing neutrophil activation. Plasmapheresis was used as a lipid-lowering therapy in a study of serial hyperlipidemic AP cases and provided complete recovery in 75% of the patients [15]. Short term veno-venous hemofiltration is another effective treatment modality in hyperlidemic AP that decreases circulating TNFα while increasing IL-10 level [14]. Mao et al. published their five-step systematic therapy, which they call 'penta association therapy,' in the literature after using it in 32 hypertriglyceridemia-related severe AP cases [16]. There are five items in this modality: purification of the blood (triglyceride absorbtion and hemofiltration), antihyperlipidemic agents (fluvastatin or lipanthyl), low molecular weight heparin (fragmin), insulin, and the topical application of Pixiao (a traditional Chinese drug). The success rate of this therapy is reported to be 80% in the early phase of the disease [16]. In addition, the American Society for Apheresis (ASFA) Committee on Clinical Applications suggested plasmapheresis for treating severe hypertriglyceridemia [7]. In light of this information, we also started conservative therapy quickly for our AP case who had a history of type I familial hyperlipidemia and who had had recurrent AP episodes, but we did not wait for response to the conservative therapy before beginning plasmapheresis. The main concerns about apheresis are cost and availability. After one plasmapheresis session, serum triglyceride level was less than 500 mg/dL, so apheresis was stopped. A single application was enough for clinical and laboratory recovery in this case.

In conclusion, early use of plasmapheresis, together with conservative therapy, is a hopeful, and probably better treatment option in hypertriglyceridemia-related acute pancreatitis cases, as a single session was enough for recovery in our case.

Competing interests

The authors declare that they have no competing interests.

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