Recurrent Acute Pancreatitis Due to Hypertriglyceridemia and Acute Lung Injury

Hipertrigliseridemi Nedeniyle Tekrarlayan Akut Pankreatit ve Akut Akciğer Hasarı

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Hypertriglyceridemia is a common etiological factor in acute pancreatitis. In addition, acute lung injury is a component of multiple organ dysfunction syndrome and may cause acute pancreatitis. Recurrent acute pancreatitis due to hypertriglyceridemia and acute lung injury are serious conditions with high morbidity and mortality. Here we present a case report of a 44-year-old male patient with recurrent acute pancreatitis due to hypertriglyceridemia and acute lung injury.

Key Words: Recurrent acute pancreatitis, hypertriglyceridemia, acute lung injury

Introduction

Acute pancreatitis is diagnosed by abdominal pain in the upper abdomen and is associated with elevated levels of serum amylase and lipase. Acute pancreatitis is also a complication of hypertriglyceridemia; however, the mechanisms leading to hypertriglyceridemia-induced pancreatitis are not fully understood (1, 2).

Acute pancreatitis is also associated with increased risk of acute lung injury, which is characterised by acute inflammation of the lung. There are few studies describing the pathogenesis of acute lung injury associated with pancreatitis and the clinical spectrum of acute lung injury may vary depending on the degree of hypoxia (3, 4).

The diagnosis and management of acute pancreatitis with acute lung injury requires a multidisciplinary approach.

Case Report

A 44-year-old male patient was admitted to our clinic with nausea, bilious vomiting and pain in the upper abdomen. The patient had a history of 2 episodes of acute recurrent pancreatitis. He also smoked for 75 pack years, consumed 35 cc of alcohol 3-4 days per week and suffered from type 2 diabetes mellitus. In addition, he had a history of pantoprozol, fenofibrate, and metformin use. He also presented with acute pancreatitis due to hypertriglyceridemia 2 months prior to admittance to the clinic.

The general health of the patient was poor with arterial blood pressure of 95/60 mmHg, temperature of 37.4°C, pulse rate of 94 beats/minute and respiratory rate of 26 breaths/minute. Examination of the gastrointestinal system showed evidence of abdominal distension and rebound tenderness with normoactive bowel sounds. A digital rectal examination was normal. A pulmonary examination revealed bilateral inspiratory crackles at the base of the lungs.

The laboratory findings displayed hemoglobin levels of 13.4 g/dL, hematocrit of 39%, and white blood cell counts of 9.84x10³ mcg/L. The biochemical measurements consisted of 368 mg/dL glucose, 483 U/dL amylase (normal: 25-125 U/L), 1797 U/L lipase (normal: 8-78 U/L), 358 mg/dL total cholesterol, 1653 mg/dL triglycerides, 31 U/L ALT, 68 U/L GGT, 0.4 mg/dL total bilirubin, 608 U/L LDH and 299.51 mg/L CRP. The levels of urea, creatinine, sodium, and potassium were within the normal range. The prothrombin time was also within the normal range. Analysis of the arterial blood gas parameters revealed a pH level of 7.30, 30 mmHg CO₂, 62 mmHg PO₂, 19.5 mEq/L HCO₃⁻.
and 1.3 mmol/L lactate. Abdominal spiral computed tomography was performed and widespread peripancreatic fluid collections were observed to be compatible with pancreatitis. The patient was admitted to the intensive care unit with a score of Ranson of 4 and APACHE II of 12.

Acute recurrent pancreatitis due to hypertriglyceridemia was diagnosed after excluding any other etiological factors known to cause pancreatitis. Supportive therapy was planned and included treatment with proton-pump inhibitors, analgesics and antibiotics. Plasmapheresis was also started following catheterisation of the right internal jugular vein, resulting in a decrease in the serum triglyceride levels to 305 mg/dL. On follow up, hemodynamic instability, dyspnea and tachypnea developed. Continuous venovenous haemodiafiltration was also planned because of the arterial blood gas analysis. The data obtained was as follows: 73 mg/dL Urea, 2.16 mg/dL creatinine, pH 6.9, 3.3 mEq/L HCO₃, 28.3 mmHg PO₂, 48.4 mmHg pCO₂ and 10.22 mmol/L lactate. Bilateral pulmonary infiltrates were also observed on lung X ray and were consistent with acute respiratory distress syndrome. The patient died in the intensive care unit on the seventh day despite supportive mechanical ventilation and hemodialfiltration.

Discussion

Hypertriglyceridemia may cause acute, recurrent or chronic pancreatitis. Hypertriglyceridemia was reported as an etiological factor in acute pancreatitis in 1.3-3.8% of patients and triglyceride levels > 500 mg/dL may cause pancreatitis (1, 5). Hypertriglyceridermia-induced pancreatitis has similar complications to those associated with other forms of acute pancreatitis and may lead to fatal complications such as acute lung injury. The rate of mortality may be as high as 20%. Recurrent acute pancreatitis due to hypertriglyceridemia and acute lung injury are serious conditions with high morbidity and mortality (4, 6).

The mechanisms underlying hypertriglyceridermia-induced acute pancreatitis are not clearly defined. Elevated serum triglyceride levels increase the risk for acute pancreatitis. Free fatty acids may induce capillary injury leading to ischemia as a result of modifications in the hyperviscosity of blood (7).

Treatment of hypertriglyceridemia consists of weight reduction, restriction of alcohol intake, increased physical activity and adoption of a low fat diet. Fibric acid derivatives or nicotinic acid are effective for patients who do not respond to non-pharmacologic methods (7, 8). Higher triglyceride levels >500 mg/dL may cause pancreatitis. In this study, the triglyceride level of the patient was 1653 mg/dL. Treatment options for pancreatitis are similar, irrespective of the underlying cause. Treatment consists of transportation of the patient to the intensive care unit followed by nutritional support, anti-inflammatory agents, hemodynamic management, analgesics, and cessation of oral fluid intake in favour of intravenous hydration. Lipopheresis is a safe and effective method to reduce triglyceride levels in patients with acute pancreatitis. Additional options to treat hypertriglyceridemia include therapies such insulin and heparin application (7-9).

Acute respiratory distress syndrome is a major component of multiple organ dysfunction syndrome and may accompany acute pancreatitis. Multiple organ dysfunction syndrome is a response to inflammation and may be a result of acute pancreatitis. Acute respiratory failure within the first few days of acute pancreatitis has been reported to be associated with higher mortality. Diffuse pulmonary infiltrates, progressive hypoxemia, acute lung injury and respiratory distress may develop in patients with acute pancreatitis (3, 4, 10).

Acute respiratory distress syndrome may be caused by oxygen radicals that are released by activated neutrophils and proinflammatory cytokines, which may affect the lung endothelium. Elevated concentrations of pancreatic enzymes might be associated with lung injury, perhaps by promoting a pro-inflammatory state (11). Acute lung injury may occur during the early stages of acute pancreatitis. The incidence of pulmonary complications in acute pancreatitis was reported to be between 15% and 55%. Severe pancreatitis is associated with lung injury and with higher mortality rates.

Dendritic cells (DC) are antigen-presenting cells and, together with monocytes and macrophages, are part of the custocyte system. DC play a critical role in the induction of naive CD4 and CD8 T cells. Wang et al. have reported the association of endothelial permeability with the pulmonary custocyte system in acute pancreatitis (12).

Conclusion

Pulmonary hemodynamics should be closely monitored to detect the presence of systemic complications and hypertriglyceridemia should be considered as an etiological factor in pancreatitis and acute respiratory failure.

Conflict of Interest

No conflict of interest was declared by the authors.

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Author Contributions


Çıkar Çatışması

Yazarlar herhangi bir çikar çatışması bildirmemİŞlerdir.

Hakem değerlendirme: Diş bağımsız.

Yazar Katkıları


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