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Case Report

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Acute Pancreatitis as First Manifestation of Severe Hypothyroidism: A Rare Case Report

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1. Abstract

We described the case of a 34-year-old Bangladeshi man with an unremarkable personal and family history who presented to the emergency department with severe upper abdominal pain of one day's duration and with elevated serum amylase and lipase. Further work-up revealed a serum triglyceride level of 39.8 mmol/L and his thyroid function test was consistent with primary hypothyroidism. The patient received intravenous fluids, and analgesia, and was placed on a fast. Thyroxine 125 µg and intravenous insulin were also started. The patient gradually improved over the next few days. On the fifth day of his hospital stay, he was pain-free, and fenofibrate and omega-3 were introduced after intravenous insulin was stopped. The patient was discharged the following day with no sign of pain and a triglyceride level of 6.3 mmol/L. A month after discharge, the patient was seen in the outpatient clinic. He was completely stable with no pain. His triglyceride level was 4.2 mmol/l, FT4 was 12 pmol/L, and TSH> 100,000 mlU/L, and he was advised to increase the dose of thyroxine to 150 µg daily.

2. Introduction

Thyroid hormones play an important role in the body and affect every organ system. The release of thyroid hormone can regulate the sugar, protein, and fat metabolism in the human body; affect the growth and development of the body; and, to some extent, affect the functioning of the nervous, endocrine, cardiovascular, and reproductive systems in the body. Hypothyroidism is a common endocrine disorder resulting from thyroid hormone deficiency; It is distributed worldwide and affects multiple organ systems with a variety of clinical symptoms and signs [1,2]. The most common clinical presentations in adults are fatigue, lethargy, cold intolerance, weight gain, constipation, voice changes, and dry skin [2]. However, the literature review revealed several case reports describing the unusual presentation of hypothyroidism such as pericardial effusion and cardiac tamponade [3], periodic paralysis [4], and elevated serum pancreatic amylase and trypsin without acute pancreatitis [5]. Acute pancreatitis, presenting as the first manifestation of hypothyroidism, is rarely described beforehand. Here we present a case report of a middle-aged male patient with hypertriglyceridemia-induced acute pancreatitis as the first manifestation of primary hypothyroidism.

3. Case Report

A 34-year-old Bangladeshi man with an unremarkable personal and family history presented to the emergency department complaining of severe epigastric pain that had persisted for a day and was relieved by bending forward. He denied fever, palpitations, nausea, vomiting, changes in bowel habits, or urinary symptoms. Clinical examination showed a conscious and well-oriented patient with a good build. His oral temperature was 37.3 °C, blood pressure 133/86 mm Hg, pulse 109/min, and respiratory rate 17/min. Abdominal examination showed a soft abdomen with slight epigastric tenderness. The rest of his examination was unremarkable. Initial laboratory tests showed a white blood cell count of 11100/µL, hemoglobin 12.6 g/dl, and platelets 234,000/µl. Blood chemistry showed serum creatinine of 79 µmol/L; sodium 124 mmol/L; potassium 3.8/mmol/l; chloride 91 mmol/L; adjusted calcium 2.29 mmol/L; cholesterol 15.8 mmol/L; triglyceride 39.8 mmol/L; high-density lipoprotein choles-

terol (HDL-C) 0.3 mmol/L; and low-density lipoprotein cholesterol (LDL-C) of 1.2 mmol/L. The pancreatic enzymes study showed a pancreatic amylase level of 171 U/L (normal: 13-53) and lipase of 268 U/L (normal: 13-60), random glucose of 9.8 mmol/L and C-reactive protein of 7 mg/L.

Abdominal ultrasound showed no evidence of cholelithiasis or dilated bile ducts, while a computed axial tomography (CT) scan of the abdomen showed heterogeneous parenchymal enhancement of the head of the pancreas with associated fluid accumulation around the head of the pancreas, and the periduodenal region. This picture was consistent with acute pancreatitis. Pericardial effusion has also been noted with a maximum thickness of 2.0 cm. Echocardiography showed pericardial effusion without clear echocardiographic evidence of tamponade. Thyroid function tests showed severe hypothyroidism (free thyroxine (FT4) 2 pmol/L, TSH > 100,000 mIU/L). He was diagnosed with hypertriglyceridemia-induced acute pancreatitis and his high triglyceride levels were attributed to severe hypothyroidism.

The patient was kept fasting and was given intravenous fluids and analgesia. Intravenous insulin and oral thyroxine 125 μg were also initiated. In the following days, the patient gradually improved. Intravenous insulin was discontinued and therefore fenofibrate and omega 3 started. On the 5th day of hospitalization, the patient was pain-free, and his triglyceride level dropped to 6.3 mmol/L. He was allowed to eat and was discharged the next day. A month after discharge, the patient was examined in an outpatient clinic. He was completely stable with no pain. His triglycerides were 4.2 mmol/l, FT4 12 pmol/l, and TSH > 100,000 mIU/l, and he was advised to increase the dose of thyroxine to 150 μg daily.

4. Discussion

Two of the following three criteria, including abdominal pain specific to acute pancreatitis, serum amylase and/or lipase levels three times above normal, and imaging findings specific to acute pancreatitis, particularly computed tomography (CT) scans required for the diagnosis of acute pancreatitis. Our case met the three criteria [6]. Acute pancreatitis is largely associated with gallstones and alcohol abuse, accounting for 80% of cases. Less commonly, acute pancreatitis may be associated with drugs and toxins, metabolic disorders (hypertriglyceridemia and hypercalcemia), connective tissue disease, infection, and others such as gastric sleeve resection [7]. Although accounting for up to 7% of cases, hypertriglyceridemia is the third most common cause of acute pancreatitis [8]. Hypertriglyceridemia can be divided into primary and secondary types [9]. Although secondary causes of hypertriglyceridemia are estimated to account for more than 75% of pancreatitis caused by hypertriglyceridemia, these secondary causes are often insufficient to raise triglyceride levels to the point where pancreatitis develops, indicating the presence of a pre-existing primary lipid disorder. Because clinical indicators such as eruptive xanthomas or retinal lipemia can serve as clues to the diagnosis of the primary cause of hypertriglyceridemia, these patients should be properly evaluated through history and physical examination, and a family history of lipid disorders such as dyslipidemia and cardiovascular disease also being evaluated. [9,10,11]. In our case, the primary causes are considered unlikely due to the unremarkable personal and family history of dyslipidemia and cardiovascular disease. Secondary causes such as diabetes mellitus, drugs, obesity, and alcohol use were also excluded. Therefore, we believe that the hypertriglyceridemia-induced pancreatitis in our case is most likely related to untreated primary hypothyroidism.

In its early stages, hypothyroidism may not show any obvious symptoms. Untreated hypothyroidism can lead to a variety of health problems over time, including obesity, heart disease, and hypertriglyceridemia-induced acute pancreatitis. The risk of hypertriglyceridemia-induced pancreatitis increases with increasing triglyceride levels, particularly above 500 mg/dL (5.6 mmol/L), with the risk of acute pancreatitis increasing by 4% for each 100 mg/dL increase (1.13 mmol/l) increases in triglycerides [12].

There are two main aspects to the treatment of hypertriglyceridemia-induced acute pancreatitis. Similar to other types of acute pancreatitis, one focuses on managing pancreatic inflammation through aggressive fluid resuscitation, bowel rest, analgesia, and thromboprophylaxis. Triglyceride reduction is the other component. For this clinical entity, there is no evidence-based treatment. Various treatment options have been tried and reported with variable success rates. These include therapeutic plasmapheresis, hemoperfusion, hemofiltration, heparin or insulin infusion, and fasting [12]. In our case, fasting and the initiation of intravenous insulin and levothyroxine resulted in significantly reduced triglyceride levels. Although high levels of chylomicrons are necessary to trigger an attack of hypertriglyceridemia-induced acute pancreatitis, the exact mechanism of this condition is not clearly understood. In the proposed pathogenesis, acinar cells and capillary endothelium may be harmed toxically. Free radical damage is produced when pancreatic lipase hydrolyzes triglycerides and releases free fatty acids, which can directly damage cell membranes [9-12].

5. Conclusion

Acute pancreatitis as the first manifestation of hypothyroidism is a rare clinical entity. Hypothyroidism can cause severe hypertriglyceridemia, which can trigger acute pancreatitis. Fasting and the initiation of intravenous heparin and levothyroxine result in significantly reduced triglyceride levels.

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