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Elevation of Serum Amylase and Lipase Levels in Diabetic Ketoacidosis without Acute Pancreatitis: A Case Report and Literature Review

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Abstract **Case Report**

Serum amylase and lipase levels are commonly used as diagnostic markers for acute pancreatitis, but elevated levels of these enzymes can also occur in other conditions. We report a case of a 45-year-old male with a known history of type 2 diabetes who presented with nausea, vomiting, and abdominal pain. Laboratory investigations revealed hyperglycemia, ketonemia, metabolic acidosis, and elevated levels of serum amylase and lipase. However, imaging studies did not reveal any evidence of acute pancreatitis or other abdominal pathology. The patient was diagnosed with diabetic ketoacidosis and managed with fluid resuscitation, insulin therapy, and correction of metabolic acidosis. The serum amylase and lipase levels gradually normalized, supporting the diagnosis of diabetic ketoacidosis without complicated with acute pancreatitis. This case highlights the importance of considering alternative diagnoses in the setting of elevated serum amylase and lipase and appropriate management of diabetic complications to prevent the development of more severe conditions.

Keywords: Amylase; Lipase; Diabetes ketoacidosis; Acute pancreatitis.

I. INTRODUCTION

Diabetic ketoacidosis (DKA) is a life-threatening complication of diabetes mellitus (DM) characterized by hyperglycemia, metabolic acidosis, and ketonemia/ketonuria. DKA can occur in people with type I or type II diabetes, although it is more commonly seen in type I diabetes. Type II diabetes mellitus (DM) is a metabolic disorder characterized by high blood glucose levels due to insulin resistance and/or inadequate insulin production. In people with type II diabetes, DKA typically occurs when there is a significant decrease in insulin levels, often due to an illness or infection that causes increased insulin resistance. The insulin requirements in patients with COVID-19 and severe insulin resistance are much higher than those generally reported in studies of critical illness patients [1]. It occurs when the body starts breaking down fat at a rate that is much too fast. This can lead to an accumulation of ketone in the blood, which can cause a range of symptoms such as nausea, vomiting, abdominal pain, conscious disturbance, and difficulty breathing. On the other hand, acute pancreatitis is a condition characterized by inflammation of the pancreas, which can be caused by various factors including alcohol consumption, gallstones, and some medications, with a clinical course that ranges from mild to severe, and can even result in multiple organ dysfunction and death. Individuals with type II DM may be at an increased risk of developing acute pancreatitis [2]. In particular, poorly controlled blood glucose levels and obesity, which are common features of type II DM, are associated with an increased risk of pancreatitis. Serum amylase and lipase levels are commonly used to diagnose acute pancreatitis. However, the elevation of amylase and lipase levels can be nonspecific, and an elevation of either one >3 times the normal level is usually indicative of acute pancreatitis. In this report, we describe a patient with diabetic ketoacidosis who presented with elevated levels of serum amylase and lipase, but without evidence of acute pancreatitis.

II. CASE REPORT

We present a case of a 25-year-old male with a past history of type II DM without regular control, hypertension, and paranoid schizophrenia. He presented with a change in consciousness, accompanied by dyspnea, vomiting, and chest pain in recent days, and was brought to the emergency department. On examination, he had conscious disturbance (GCS: E3V2

M4); T/P/R: 36.7/118/26; BP: 117/53 mmHg, and his blood sugar test showed high levels (647 mg/dl). Physical examination showed no abdominal tenderness, and he had full four-limb muscle power. Laboratory data revealed leukocytosis (WBC: 24.7 x103 /uL), hyponatremia (Na: 125 mmol/L), acute kidney injury (creatinine: 2.04 mg/dl), elevated HbA1C (13%), elevated CRP (44.2 mg/L), high blood osmolality (318 mOsm/Kg), high serum ketone body (6.3 mmol/L), elevated amylase (357 IU/L >3 times normal), and lipase (961 U/L >3 times normal). Arterial blood gas showed severe metabolic acidosis (pH:6.88, Act cHCO3: 5.3 mmol/L, and Base Excess:-26.9 mmol/L). Chest X-ray showed increased infiltration of both lower lungs (Fig 1). Due to dyspnea with acute respiratory failure, intubation was performed with ventilator support. DKA and pneumonia were suspected, and the patient was admitted to the Intensive Care Unit (ICU) for further treatment. At the ICU, sputum culture showed no growth, but blood culture revealed Staphylococcus epidermis. Antibiotics (Piperacillin/tazobactam and Minocin) were initiated. Adequate fluid replacement and insulin sliding scale were implemented for sugar control. The patient's amylase and lipase levels fluctuated, so an abdomen computed tomography (CT) scan was performed to rule out acute pancreatitis. However, the CT scan revealed no evidence of acute pancreatitis (Fig 2). Renal function deteriorated, and acute pulmonary edema was noted, so hemodialysis was initiated. The patient's blood sugar was gradually under control, and the chest X-ray showed improvement of lung infiltrates. His amylase and lipase levels returned to near normal, he was successfully weaned from mechanical ventilator, and his renal function improved after a two-week admission to the ICU. Finally, he was transferred to a general ward, discharged, snd followed up at OPD.

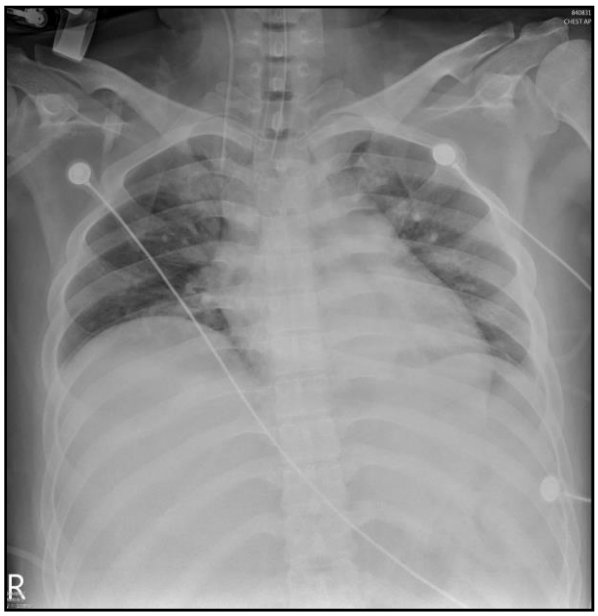

Figure 1: Endotracheal tube confirmed in situ. Bilateral lung infiltrates present

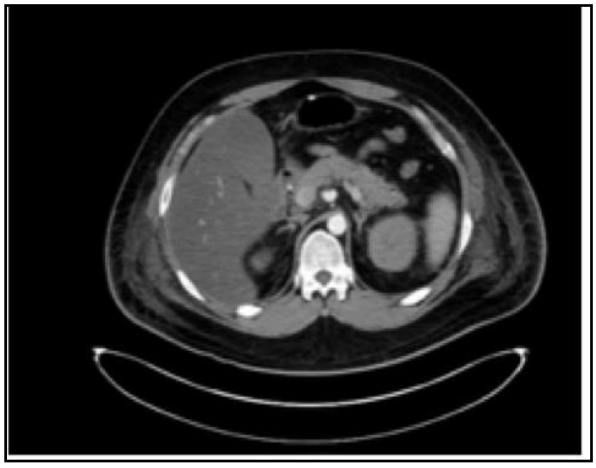

Figure 2: Based on the results of the abdominal computed tomography (CT) scan, there were no signs or evidence of acute pancreatitis

III. DISCUSSION

Diabetic ketoacidosis (DKA) is a common acute hyperglycemic emergency in people with diabetes mellitus, particularly in young patients (<65 years). DKA is confirmed when all three criteria are present: elevated blood glucose levels, the presence of high urinary or blood ketoacids, and a high anion gap metabolic acidosis [3]. Typically, the serum glucose concentration in DKA is less than 800 mg/dL (44 mmol/L) and often in the 350 to 500 mg/dL (19.4 to 27.8 mmol/L) range. Hyperglycemic crises can induce oxidative stress and lead to the generation of reactive oxygen species, resulting in elevated pro-inflammatory cytokines such as tumor necrosis factor-alpha, interleukin (IL)-1B, and IL-6. Lipid peroxidation markers and C-reactive protein (CRP) are also increased [4]. In patients with DKA, the elevated anion gap metabolic acidosis is caused by the accumulation of beta-hydroxybutyric and acetoacetic acids, resulting in a reduction of sodium bicarbonate concentration. The serum bicarbonate concentration in DKA is usually moderately to markedly reduced.

This Type II DM patient meets the definition of severe DKA proposed by the American Diabetes Association [5]. DKA occurred in this patient due to extreme stress of pulmonary infection. Treatment of DKA involved correction of the fluid and electrolyte abnormalities that are typically present (hyperosmolality, hypovolemia, metabolic acidosis, and potassium depletion), the administration of insulin and addressing the underling cause.

Guidelines for the diagnosis of acute pancreatitis commonly require the presence of two of the three following criteria: typical abdominal pain; characteristic computed tomography or ultrasonography findings, and/or amylase and/or lipase levels of three or more times the upper limit of normal [6]. The main sources of amylase in humans are the pancreas and salivary glands. Lipase is synthesized and stored mainly as granules in the pancreatic acinar cells. There are several sources of lipase in the human body, including lingual, pancreatic, gastrointestinal tract, and hepatic lipase. Serum amylase and lipase are common tests obtained as biochemical markers for diagnosing acute pancreatitis in patients presenting with upper abdominal pain. However, several non-pancreatic conditions can present with abnormal serum amylase and lipase levels [7]. Most extra-pancreatic causes of amylase and lipase elevations are not associated with a greater than three times normal has been reported in patients with renal insufficiency, malignant tumors, and esophagitis [8].

Acute pancreatitis may precipitate or complicate DKA and it can present or coexist with DKA and deteriorate its severity [9]. Acute pancreatitis can also be induced by DKA, complicated by hypertriglyceridemia [10]. In this DKA patient, even though amylase and lipase levels are high (both >3 times normal), there is no evidence of coexisting acute pancreases. Elevated serum amylase and lipase in DKA patients may be due to metabolic derangements or decreased clearance of enzymes, and not due to acute pancreatitis attack [11]. Therefore, the diagnosis of acute pancreatitis in patients with DKA should be based on clinical findings and imaging studies.

IV. CONCLUSION

This case highlights the importance of a thorough differential diagnosis and appropriate management of underlying conditions when evaluating patients with elevated serum amylase and lipase levels. It underscores the importance of early intervention and monitoring to prevent the progression of diabetic complications and associated pancreatic conditions. Increased amylase and lipase levels have been reported in cases of diabetic ketoacidosis. However, the diagnosis of acute pancreatitis based solely on elevated amylase and/or lipase, even > 3 times normal, is not justifiable. Clinician should arrange for abdominal computed tomography or ultrasonography to confirm whether DKA coexists with acute pancreatitis.

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