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Case Report

A rare triad of diabetic ketoacidosis, hypertriglyceridemia and acute pancreatitis: a case report

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ABSTRACT

Diabetic ketoacidosis (DKA), Acute Pancreatitis and Hypertriglyceridemia are an unusual and infrequent presentation of Diabetes. Hypertriglyceridemia leading to acute pancreatitis is most commonly encountered in poorly controlled Diabetics. In this case report we have described a young Type 1 Diabetic female presenting with Diabetic Ketoacidosis and hypertriglyceridemia induced Pancreatitis (HTIP). The proposed mechanism is triglyceride excess is hydrolyzed by pancreatic lipase resulting in the formation of excess free fatty acids. Excess free fatty acids cause acinar cell injury and capillary leakage in pancreatic vascular beds. Hyperlipasemia frequently occurs in DKA without an underlying acute pancreatitis. Half of the patients with HTIP have falsely low or normal serum Amylase. Imaging is required to confirm the diagnosis of pancreatitis. So, high degree of suspicion is required to diagnose this unusual presentation of diabetes.

Keywords: Diabetic ketoacidosis, Hypertriglyceridemia, Pancreatitis

INTRODUCTION

The triad of Diabetic ketoacidosis (DKA), Acute Pancreatitis and Hypertriglyceridemia is an unusual presentation of diabetes. Hypertriglyceridemia leading to acute pancreatitis is most commonly encountered in poorly controlled diabetics. Gallstones and alcohol account for two-thirds of the cases of acute pancreatitis; Hypertriglyceridemia is the third most common cause, and account for 1-4% of acute pancreatitis cases.[1] Diabetic ketoacidosis is a well-known complication of type 1 diabetes and is due to an absolute insulin deficiency but can also occur in type 2 diabetes due to excess of counter-regulatory hormones.[2,3] Diabetes is a risk factor for hypertriglyceridemia and its association with acute pancreatitis is rare. The risk of pancreatitis increases with triglycerides greater than 1000 mg/dL.[4] Several mechanisms have been postulated for Hypertriglyceridemia induced Pancreatitis (HTIP), all leading to common pathway of ischemic injury to acinar cells. Excess triglycerides are hydrolyzed by pancreatic lipase resulting in the formation of excess free fatty acids. Excess free fatty acids cause acinar cell injury and capillary leakage in pancreatic vascular beds.[5] Possible mechanism of pancreatitis in hypertriglyceridemia patients is the damage of acinar cells and microvascular membrane due to excessive free fatty acid and lecithin formation in pancreatic bed from lipoprotein substrates.[6]

The resultant ischemia produces an acidic environment which perpetuates a vicious cycle of subsequent ischemia and necrosis. Likewise, excess chylomicrons cause ischemic injury because the resulting hyper viscous plasma causes sluggish flow through the pancreatic beds. very severe hypertriglyceridemia which can either be of genetic or acquired origin resulting from metabolic disorders (e.g., diabetes), diet (including alcohol and obesity) and/or drugs.[7]

Causes of HTIP can be divided into two main groupswhich were genetic factors includes Familial dyslipidemia and Secondary factors includes Untreated /poorly controlled Diabetes Mellitus (DM), alcohol abuse, pregnancy, and medications.[8]

This case report describes a diabetic patient with diabetic Ketoacidosis, hypertriglyceridemia and acute pancreatitis. Though this association is rare many case reports have been described.

CASE REPORT

A 25-Year-old female k/c/o Type 1 Diabetes mellitus presented with abdominal pain and vomiting for 1 day with RBS - 458 mg/dl, Urinary ketone highly positive (++++) and pH 6.90. On examination, patient had acidotic breathing with features of dehydration. Her systemic examination was normal except mild abdominal tenderness. Patient was admitted as a case of diabetic ketoacidosis and managed with adequate intravenous fluids and insulin.

Investigation on the day of admission showed Total Cholesterol-1445 mg/dl; Triglycerides-7175 mg/dl, WBC-16260/cu.mm, Hemoglobin-17.5 g/dl, Sr.Sodium-126 meq/dl, Sr. Potassium-5.4 meq/dl and Sr. Lipase -882 IU/L. Pseudohyponatremia could be due to hyperglycemia and hyperlipidemia. USG whole Abdomen showed bulky Pancreas. CT Abdomen showed swelling of the Pancreas and peripancreatic fluid collections. Repeat investigations showed a decreasing trend of Lipid profile and Lipase. At discharge triglyceride level was 277mg/dl and lipase level was 32IU/L. Patient was discharged on day 7 of admission without any complications.

DISCUSSION

The triad of DKA, hypertriglyceridemia and pancreatitis is an unusual presentation of poorly controlled diabetes which occurs in Type 1 and Type 2 diabetes.[9] In Diabetic Ketoacidosis, the deficiency of insulin activates lipolysis in adipose tissue releasing increased free fatty acids, which accelerates formation of VLDL in the liver. Reduced activity of lipoprotein lipase in peripheral tissue also decreases clearance of VLDL from the plasma, resulting in hypertriglyceridemia. Mild hypertriglyceridemia is common during episodes of DKA. However, severe hypertriglyceridemia, which is defined as a TG level >2,000 mg/dL, is rare.[10]

"Pink blood" (Figure 1) as aptly described by Sandhya et al.[11] In a case report described the colour of the blood in hypertriglyceridemia. The pink colouration of the patient's blood is due to the intermingling of the opaque white triglyceride (TG) and chylomicrons with the dark red blood cells. Extreme hypertriglyceridemia can result in alteration of the colour of peripheral blood and artificially elevated hemoglobin levels. Automated hemoglobin measurements are typically based on spectrophotometric techniques where other light interfering materials, such as TG containing particles may result in falsely elevated hemoglobin level.[12]

Figure 1: Photograph of patient blood sample (Pink colored blood).

Hyperlipasemia frequently occurs in DKA without an underlying acute pancreatitis. Yadav et al. reported non-specific elevations of amylase and lipase in 16.6% and 24.6%, respectively, in diabetic ketoacidosis. Half of the patients with HTIP have falsely low or normal serum Amylase. This is due to the interference of the plasma lipids with the colorimetric amylase assay. Acute pancreatitis is a close differential of diabetic ketoacidosis, it can initiate DKA, and DKA itself can mask a co-existing acute pancreatitis that occurs in 10-15% of cases. Nonspecific elevations of amylase and/or lipase without clinical evidence of pancreatitis have been reported in 24. 7-79.0% of DKA cases.[13] So, high degree of suspicion is required to diagnose this condition.

CONCLUSION

The triad of Diabetic ketoacidosis, hypertriglyceridemia and acute pancreatitis is a rare presentation of diabetes. This may be called as "pink triad" inferring to the pink colored blood in this condition. Hypertriglyceridemia is a known but rare cause of acute pancreatitis. Presence of hypertriglyceridemia is common in diabetic ketoacidosis, but presence of very high level of triglyceride should prompt clinicians to search for complications like acute pancreatitis. Further study into this triad is required to elucidate its pathophysiology.

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