Case Report

Pink Blood

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Abstract

The triad of diabetic ketoacidosis (DKA), hypertriglyceridemia and acute pancreatitis is an unusual presentation of poorly controlled diabetes which occurs in Type 1 and Type 2 diabetes. We report a case of DKA with severe hypertriglyceridemia (17,300 mg/dl). Surprisingly we noticed that the blood was pink in colour while it was drawn for investigations. We also noticed that, the supernatant fluid after centrifugation was milky white in colour. Patient was treated with intravenous insulin therapy as per DKA management protocol, fibrates, niacin and omega-3 fatty acids. The colour of blood, along with the blood sugar level and triglyceride levels returned to normal after initiating the treatment.

Keywords: Diabetic ketoacidosis, Hypertriglyceridemia, Pink colour blood

INTRODUCTION

The traid of diabetic ketoacidosis (DKA), hypertriglyceridemia, and pancreatitis is less common. Hypertriglyceridemia presenting as "pink blood" is unusual.[1] The hypertriglyceridemia of diabetes can be classified into mild to moderate (triglyceride [TG] between 150 and 499 mg/dl) and severe hypertriglyceridemia (TG ≥500 mg/dl).[2] Hypertriglyceridemia also increases the risk of cardiovascular disease.[3] Hypertriglyceridemia can be either primary or secondary. Secondary hypertriglyceridemia is associated with insulin deficiency, insulin resistance, or elevation of counter regulatory hormones seen in diabetes mellitus, obesity, pregnancy, alcohol, and with certain drugs like ostrogen, tamoxifen, thiazides, etc.[4] However, very high serum TG is rare and occurs in <1 in 5000 individuals.[5]

CASE REPORT

A 43-year-old male admitted with his random blood sugar level of 640 mg/dl. Patient had abdominal pain with no other symptoms. Patient is known case of Type 2 diabetes for 5 years on irregular treatment and alcoholic for 6 years. Family history was not contributory. On examination, patient was stable. His general and systemic examinations were normal. There was no evidence of lipemia retinalis and xanthomas.

When blood was drawn for investigations, we noticed that the blood was pink in colour (Figure 1) and the supernatant after centrifugation was milky white in colour (Figure 2). Investigations (Table 1) on the day of admission were total cholesterol - 1530 mg/dl, TG - 17,300 mg/dl, high-density lipoprotein - 356 mg/dl, low density lipoprotein - 198 mg/dl, fasting blood glucose - 449 mg/dl, postprandial blood sugar - 720 mg/dl, HbA1C - 16.7, Haemoglobin - 17 g (falsely elevated haemoglobin), packed cell volume - 27, electrolytes (mEq/L) S.Na - 104 (pseudohyponatremia), serum potassium - 4.0, Cl - 74, HCo_3 - 15, serum amylase and lipase were normal. Arterial blood gas analysis revealed metabolic acidosis with 7.2 pH. Urine ketone was positive. Stool analysis for fat was normal. Computed tomography abdomen showed acute on chronic pancreatitis. All other investigations were normal. Patient was treated with adequate intravenous fluids, insulin therapy, fibrates and niacin.

After 3 days of treatment blood and the supernatant returned to the normal colour (Figures 3 and 4). Improvement in the lipid profile, blood sugar level was noted (Tables 2 and 3). Serum sodium and haemoglobin became normal. Arterial blood gas analysis was normal, and urine ketone was negative.

Family screening was negative for hyperlipoproteinemia.

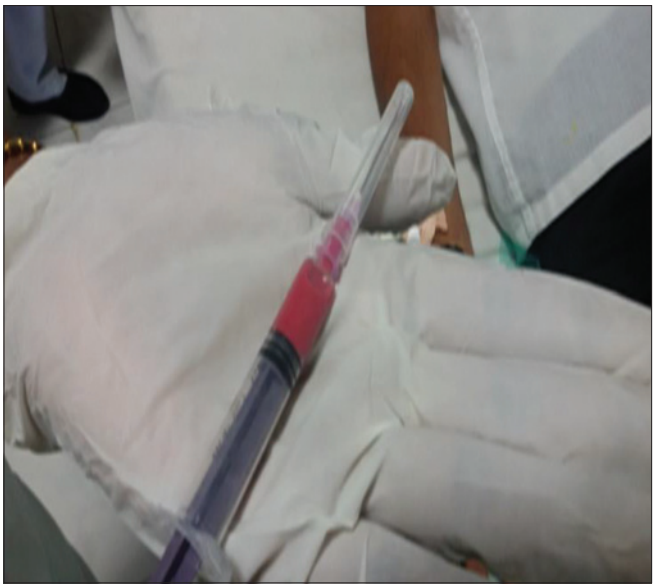

Figure 1: Pink colored blood

Figure 2: Milky white supernatant

Table 1: On admission (mg/dl)

TC	1530
TGL	17,300
HDL	356
LDL	198

VLDL cannot be measured initially due to interferences from high TGL. TC: Total cholesterol, TGL: Triglyceride level, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein

Table 2: After 3 days (mg/dl)

TC	860
TGL	2690
HDL	63
LDL	168

TC: Total cholesterol, TGL: Triglyceride level, HDL: High density lipoprotein, LDL: Low density lipoprotein

Figure 3: Normal colour blood after treatment

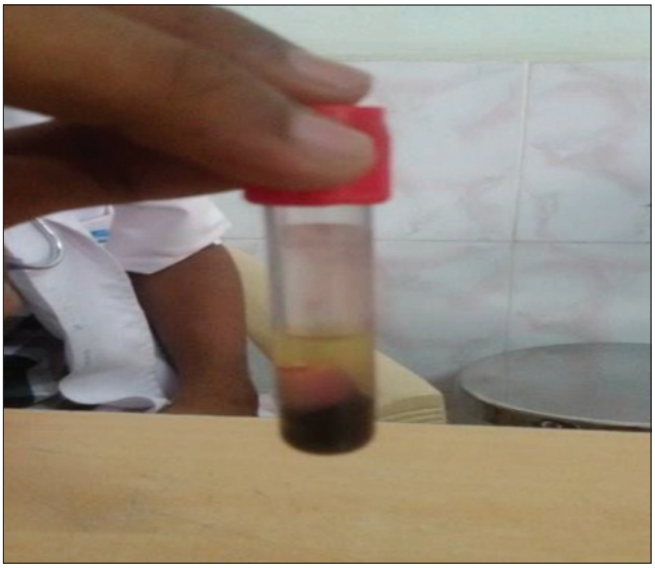

Figure 4: Normal colour serum after treatment

Table 3: At discharge

TC	135
TGL	73
HDL	40
LDL	62
VLDL	15

VLDL cannot be measured initially due to interferences from high TGL. TC: Total cholesterol, TGL: Triglyceride level, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein

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.[6] The pink

colouration of the patient's blood can be attributed to the intermingling of the opaque white TG containing very low density lipoprotein (VLDL) and chylomicrons with the dark red blood corpuscles.[7] The milky white appearance of serum was mainly due to the high level of VLDL or chylomicrons.[5] Extreme hypertriglyceridemia can result in alteration of the colour of peripheral blood and artificially elevated haemoglobin levels.[7] Automated haemoglobin measurements are typically based on spectrophotometric techniques where other light interfering materials, such as TG containing particles may result in falsely elevated haemoglobin level.[7]

Pseudohyponatremia is a false result of the tests (flame emission spectrophotometry and indirect potentiometry I-ISE) that measure sodium levels in the whole serum. When the volume of the non-aqueous phase of the serum increases due to severely increased lipid or protein levels or radio contrast substances or dextran, these test show hyponatremia, which is only pseudohyponatremia, because the sodium level in the aqueous phase of the serum remain unchanged. Pseudohyponatremia can be confirmed by calculating the corrected sodium using the formula; correction formula for sodium in hypertriglyceridemia and hyperlipidemia: Plasma TG (mg/L) × 0.002 = mEq/L decrease in Na.[8] (17300 × 0.002 = 34.6, 104 + 34.6 = 138.6).

In uncontrolled diabetes, hypertriglyceridemia occurs due to the decrease activity of lipoprotein lipase enzyme (LPL) which hydrolyses the TGs into fatty acids that enters muscle cells to be utilized as a source of energy and in fat cells to get converted into TGs and get stored.[9] The hypertriglyceride itself was attributed to DKA.[9] Hypertriglyceridemic pancreatitis is due to direct damage to the pancreatic tissue by high levels of free fatty acids. High concentration of free fatty acids reduces the pH, which may activate the trypsinogen. The chylomicrons may damage the distal pancreatic blood circulation, thus inducing ischemia. This change alters the acinar function and exposes the pancreatic tissue to the TGs. This activates the pancreatic lipase, which in turn induce inflammation and a sustained pancreatic enzyme activity. The study made by Chag *et al.*, has identified the specific genes which are associated with hypertriglyceridaemic pancreatitis. The cystic fibrosis transmembrane conductance regulator mutation/variant/haplotype and the tumour necrosis factor promotor polymorphism were both found to be an independent risk factors.[4] A serum triglyceride level (TGL) above 11.3 mmol/l indicates an increased risk of developing acute pancreatitis with incidence of up to 21%.[10] Serum amylase level is less useful in diagnostic because substantial hyperamylasemia might not be seen in nearly half of the patients with hypertriglyceridemia induced pancreatitis.[11] The underlying reason of low amylase level was unknown but could be related to the suppression of enzyme activity by a circulating inhibitor.[12]

Treatment with fenofibrate and niacin/omega 3 fatty acids helps to decreases TGL by increasing the activity of LPL and reducing the hepatic TG synthesis, insulin/heparin helps to stimulate LPL and decrease the TGL[13] and finally plasmapheresis for rapid removal of chylomicrons.[14]

CONCLUSION

According to world literature <10 cases of DKA with severe hypertriglyceridemia were reported. Highest levels of TGL so far reported were 16,334 mg/dl[14] in a 10 years old Type 1 diabetic girl and 15,240 mg/dl[15] in a 20 year old Type 1 diabetic woman but in our case the TGL was more than the reported level (17,300 mg/dl). Other peculiarities in our case were pink coloured blood, milky white supernatant, falsely elevated haemoglobin and pseudohyponatremia, which were attributed to severe hypertriglyceridemia. Patients with DKA should be evaluated for hypertriglyceridemia and pancreatitis. Early treatment will reduce the complications and improves the outcome of the patient. Hypertriglyceridemia should be ruled out if we come across pink discolouration of blood.

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