

Lipemic serum in a 4 year-old girl with new-onset diabetes mellitus

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Abstract

Diabetic ketoacidosis usually occurs in up to 30% of newly diagnosed patients with type 1 diabetes. Diabetic ketoacidosis associated lipemia is a rare and under-reported entity in children. It is suspected by the presence of eruptive xanthomas and lactescent serum. Hypertriglyceridemia may induce acute pancreatitis, thus increasing mortality.

The patient reported is a 4 year -old girl who presented to the pediatric emergency room for 10 days of polyuria and polydipsia. She was diagnosed with diabetes mellitus type 1. A lactescent serum was noticed. Lipid profile was drawn and revealed hypertriglyceridemia. We started treatment by Insulin infusion according to standard protocol. On day 6 of hospitalization, glycemic and lipid profile were normalized.

Keywords

diabetic ketoacidosis; pediatric emergency; diabetes mellitus

Introduction

Diabetic lipemia, a form of acquired fat-induced lipemia or also called milky plasma, was first used in the nineteenth century to describe the combination of uncontrolled diabetes mellitus and dyslipidemia found in severe diabetic patients. Hyperlipidemia, due to insulin deficiency and lipoprotein lipase dysfunction, should be diagnosed in order to prevent co-morbidities [2]. In this paper, we present the case of a 4 year-old girl who presented with polyuria, polydipsia and a lipemic serum.

Case Presentation

The patient reported is a 4-year old girl, born full-term by uncomplicated vaginal delivery from a non-consanguineous marriage and had an uneventful nursery course. Her past medical and surgical histories are negative. She has no family history of diabetes, dyslipidemia or autoimmune diseases. The child

was at 50th percentile for weight and height (Figure 1). She was brought to the pediatric emergency room for 10 days of polyuria and polydipsia. She denied having any fever, hematuria, dysuria, abdominal pain, vomiting, or change in appetite. The parents denied any weight change, witnessed cranial trauma or smelling any fruity odor from her mouth.

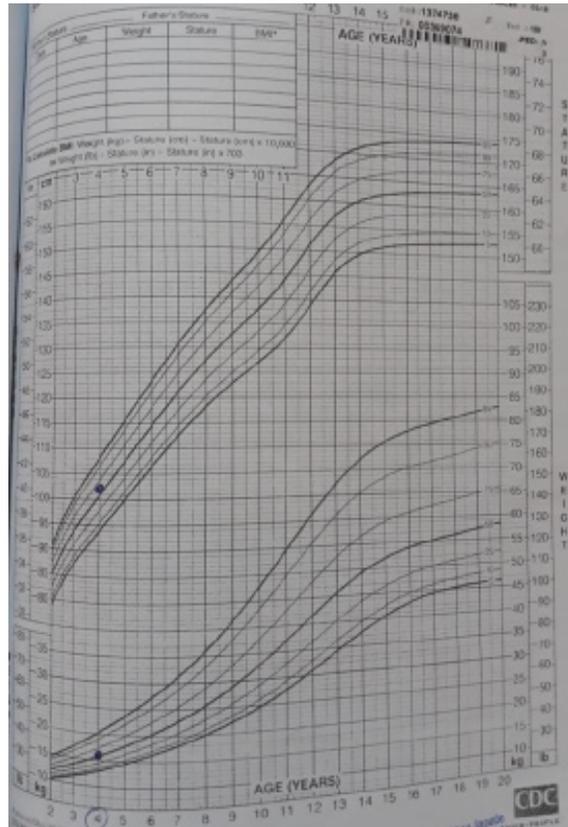


Figure 1: Height and weight of a 4 year- old girl presenting with diabetic lipemia

Vital signs: O_2 Saturation: 96% Blood Pressure: 100/60 mmHg; Pulse: 90 bpm; temperature: 37° C; Blood glucose level: 498 mg/dl.

On physical exam, the girl was tonic and conscious. She cooperated with the attending physician by replying to his questions. The physical exam was normal expect for a dry tongue. Neither shallow breathing nor acetone-smell was noticed. The evaluation for the presence of xanthomas and lipemia retinalis appeared to be normal.

A bolus of 200 ml of normal saline was administrated over 1 hour followed by 500 ml of normal saline over 3 hours in addition to 20 mEq of potassium.

A complete blood count with differential was normal (Table 1). Urinalysis showed glycosuria and ketonuria (Table 2). While sending the venous blood to be analyzed, the attending physician noticed a grossly lipemic serum (Figure 2) and a lipid panel was ordered (Table 3). She was admitted and treated with intravenous fluid and insulin infusion according to the protocol.

On the next day of hospitalization, her glucose level was 591 mg/dl. On day 6 of hospitalization, her glucose level dropped to 75 mg/dl with a level of triglycerides of 116 mg/dl (Figure 3). To notice that lipemic samples causes analytical errors in the laboratory and poses challenges to fluid management and

electrolytes replacement in treatment of diabetic ketoacidosis. One limitation was the non-availability of screening for lipoprotein lipase.



Figure 2: picture showing the grossly lipemic serum while sending the venous blood to be analyzed.

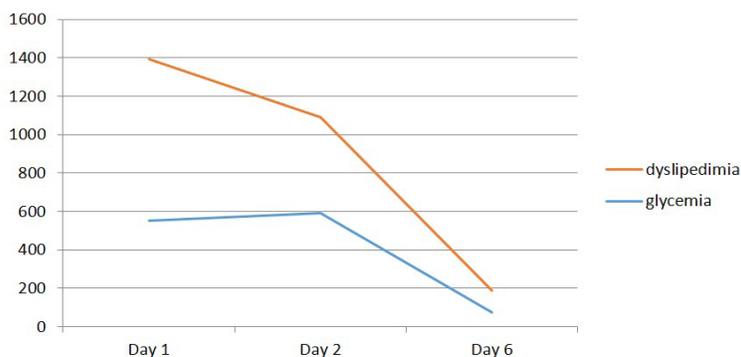


Figure 3: Drop of dyslipidemia from 843mg/dl to 116mg/dl and glycemia from 591 to 75mg/dl during hospitalization.

Table 1: The complete blood test results.

Anti ICA	Positive	Anti-insulin	2.7UI/ml
Anti GAD	1700UI/ml	Anti IA2	10UI/ml
IgA anti endomysium	Negative	HbA1c	9.9%
IgA anti-transglutaminase	Negative	Peptide C	0.4ng/ml
IgG anti-transglutaminase	Negative	Insulin	2.9microU/ml
TSH	2.32mIU/l	Glucose	552mg/dl
T4	15pmol/l	Na	131mmol/l
SGOT	5U/l	Cl	91mmol/l
SGPT	12 U/l	CO2	20 mmol/l
ALP	325U/L		

Table 2: The lipid panel results

Triglycerides	843mg/dl
Total cholesterol	201mg/dl
HDL	28 mg/dl
LDL	111mg/dl

Discussion

Diabetic ketoacidosis occurs in children with insulin deficiency presenting with polyuria and polydipsia. Diabetic lipemia occurs during insulin deficiency and uncontrolled hyperglycemia. It is usually suspected by a lactescent serum, eruptive xanthomas, fatty liver, lipemia retinalis and acute pancreatitis [3-4].

The role of insulin is anabolic; it is used to store not only glucose but also proteins and lipids [1-2]. Indeed, the insulin role is to decrease the storage of free fatty acids in the liver in order to store them in the muscles and adipose tissue. It also stimulates lipoprotein lipase, a triglyceride hydrolase enzyme found in adipose tissue, cardiac and skeletal muscle that catabolizes triglycerides into monoglycerides, fatty acids and glycerol. The fatty acids generated are then taken up by muscle and adipose tissue via triglyceride-rich chylomicrons. Chylomicron remnants are taken up by the liver where lysosomes degrade them. In turn, the liver produces Very Low Density Lipoproteins (VLDL) composed of triglyceride and carries them from the liver to peripheral tissues where triglycerides are degraded by lipoprotein lipase.

Under low insulin level conditions, lipolysis increases, and triglycerides are not stored in peripheral tissues but in the liver in the form of free fatty acids. VLDL increases rapidly. However, lipoprotein lipase becomes non-functional which results in hypertriglyceridemia [5-7].

While rare, clinicians should always be aware of hypertriglyceridemia. Indeed, it can induce lipemia retinalis and acute pancreatitis [5-7]. In addition, the treatment of diabetic lipemia is the same as diabetic ketoacidosis.

Previous findings reported that plasma, glucose and lipid levels dramatically decreased after insulin infusion and fluid administration [8-10]. Although, if the level of triglyceride did not decrease, clinicians should suspect genetic mutations such as lipoprotein lipase deficiency [8]. Another reason to suspect genetic mutations is that high levels of triglyceride is rare in a type 1 diabetes mellitus presenting patient [11].

Finally, diabetic lipemia may present with pseudo-hyponatremia and other electrolyte disturbances. The only treatment of this abnormality is to correct the glycemia level and avoid hypertonic saline solutions [8].

Conclusion

Diabetic ketoacidosis usually occurs in up to 30% of newly diagnosed patients with diabetes type 1.

Associated lipemia is a rare and under-reported entity in children. It is suspected by the presence of eruptive xanthomas, lactescent serum and lipemia retinalis. Hypertriglyceridemia may induce acute pancreatitis, thus increase mortality.

Finally, clinicians must be aware of diabetic lipemia and its complications. Our recommendation is to obtain a lipid profile in every diabetic ketoacidotic children.

All cases of lipemic serum with diabetic ketoacidosis should be followed up because they may develop later on in life an association due to low lipoprotein lipase.

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