CASE REPORT

Lipemic Serum in a Toddler with New-Onset Diabetes Mellitus Presenting with Diabetic Ketoacidosis

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ABSTRACT

Context Significant hyperlipidemia causing lipemic serum in patients with poorly controlled diabetes is under-reported in children. The recognition of the severe hyperlipidemia is important for proper management and to prevent associated morbidities. Severe hyperlipidemia in patients with diabetic ketoacidosis should be considered. **Case report** In this case we report a 2-year-old girl with new onset type 1 diabetes mellitus, who presented with severe diabetic ketoacidosis and hyperlipidemia. Hyperlipidemia was resolved with hydration and insulin therapy. **Conclusion** It is important to diagnose hyperlipidemia by checking serum lipid profile for all pediatric patients presenting with hyperglycemic crisis to prevent morbidities.

INTRODUCTION

Diabetic ketoacidosis is an acute life-threatening complication of diabetes mellitus. Up to 30% of patients, with newly diagnosed diabetes, present with diabetic ketoacidosis at the onset [1, 2, 3]. Diabetic ketoacidosis is defined by the triad of hyperglycemia (blood glucose greater than 13.9 mmol/L/ (i.e., 250 mg/dL), ketonemia/ketonuria, and acidemia (pH equal to, or less than, 7.3 and serum bicarbonate equal to, or less than, 15 mmol/L) [4]. A mild increase in serum lipid concentrations is a common feature of uncontrolled, untreated diabetes but significant hyper-lipidemia is under-reported in children. It is often overlooked in diabetic ketoacidosis. We report a 2-year-old girl with new onset type 1 diabetes mellitus, who presented with severe diabetic ketoacidosis and severe hyperlipidemia.

CASE REPORT

A previously healthy 2-year-old African-American girl presented with vomiting and abdominal pain for 2 days. She had no fever, cough or diarrhea. Her parents also described frequent urination and increased thirst associated with weight loss, despite good appetite, during the past three months. Past medical history was unremarkable and family history was negative for diabetes.

On arrival in the emergency department, she was vomiting, severely dehydrated and had rapid, but shallow breathing (hyperventilating with Kussmaul breathing) with acetone-smell. Her physical examination included a heart rate of 146 beats/min, blood pressure of 100/66 mmHg, respiratory rate of 26 breaths/min, and temperature of 37.7 °C (99.8 °F). Her growth parameters (Figure 1) were as follows: weight 11.8 kg (10-25th percentile) and length 86 cm (5th percentile). Her mucous membranes were dry and the capillary refill was more than 5 seconds. Her hair was pale and sparse. Chest was clear to auscultation with bilateral breath sounds. Her abdomen was soft but tender. There was no hepatosplenomegaly or palpable masses. The remainder of her examination was unremarkable.

Blood drawn for work up showed milky (lipemic) appearance (Figure 2). Serum chemistry showed abnormal values of blood glucose (22.4 mmol/L, reference range 3.8-6.5 mmol/L; 403 mg/dL, reference range 70-118 mg/dL), sodium (108 mEq/L, reference range 137-147 mEq/L), potassium (2.8 mEq/L, reference range 3.6-5.2 mEq/L), chloride (85 mEq/L, reference range 99-112 mEq/L), bicarbonate (3 mEq/L, reference range 23-32 mEq/L), and serum creatinine (760.2 µmol/L, reference range 53.0-114.9 µmol/L; 8.6 mg/dL, reference range 0.6-1.3 mg/dL). On the other hand, blood urea nitrogen was normal (4.3 mmol/L, reference range 2.4-6.4 mmol/L; 12 mg/dL, reference range 7-18 mg/dL). The initial blood gas showed pH 6.89 (reference range 7.32-7.42) and a base deficit of

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Table 1. Laboratory results during hospitalization.

Laboratory test	Reference range	Hospitalization day		
		1	2	3
Sodium (mEq/L)	137-147	108	127	142
Potassium (mEq/L)	3.6-5.2	2.8	2.1	3.4
Chloride (mEq/L)	99-112	85	106	117
Bicarbonate (mEq/L)	23-32	3	12	17
Glucose (mg/dL)	70-118	403	178	105

29 mmol/L (reference range -3.3-1.2 mmol/L). Serum acetones were moderate and measured serum osmolality was 287 mosm/kg (reference range 277-302 mosm/kg). Serum triglyceride level was 19.4 mmol/L (reference range 0.39-1.24 mmol/L; 1,721 mg/dL, reference range 35-110 mg/dL) and total cholesterol was 25.8 mmol/L (reference range 0-5.1 mmol/L; 1,001 mg/dL, reference range 0-200 mg/dL). Her urinalysis showed specific gravity of 1.027 (reference range 1.005-1.035), +3 ketones and glucose. Serum amylase and lipase were normal. The initial glycosylated hemoglobin was 24.9% (reference range 0-5.7%). Anti-glutamic acid decarboxylase (GAD65) antibody was positive but the islet cell and insulin autoantibodies were negative. Thyroid function tests were normal.

She was evaluated for the presence of xanthomas and lipemia retinalis, both of which were absent. She was admitted to the intensive care unit and treated with intravenous fluid (volume resuscitation) and insulin infusion according to the standard diabetic ketoacidosis protocol. The hyperlipidemia improved with the resolution of diabetic ketoacidosis and serum electrolytes returned to normal. The laboratory values, during hospital course are in Tables 1 and 2. She was discharged 9 days after hospitalization. She has been followed in our endocrine clinic and her lipid profile remained normal.

DISCUSSION

Diabetic ketoacidosis usually occurs in non-compliant patients with diabetes. It can also occur in patients with new onset diabetes who have an absolute insulin deficiency. Insulin not only affects glucose metabolism, but also protein and lipid metabolism. The derangement of lipid metabolism in diabetes mellitus has been known for many years [5, 6]. Insulin inhibits lipolysis, decreasing the supply of free fatty acids to the liver for ketogenesis. It also increases the clearance of triglyceride-rich chylomicrons from the circulation via stimulation of lipoprotein lipase, an extracellular enzyme present in the capillary wall of most tissue predominantly adipose tissue, cardiac and skeletal

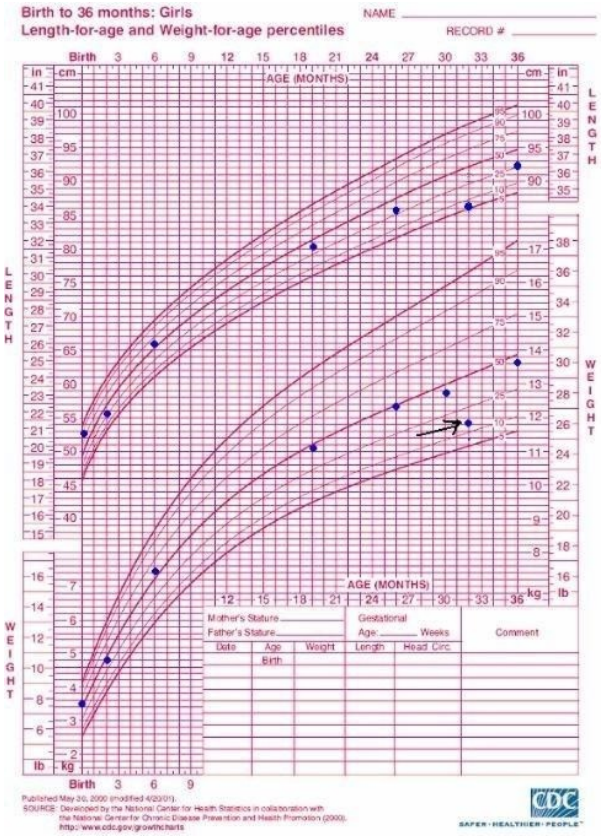

Figure 1. Growth chart showing a failure to gain weight and improvement after the management.

muscle. Lipoprotein lipase breaks down triglycerides into monoglycerides, fatty acid and glycerol. The fatty acids generated are then taken up by muscle and adipose tissue, in which they are oxidized or stored. Chylomicron remnants are taken up by the liver where lysosomes degrade apolipoprotein and cholesterol ester into cholesterol, fatty acid and amino acid. The liver in turn, produces very low density lipoproteins (VLDL)

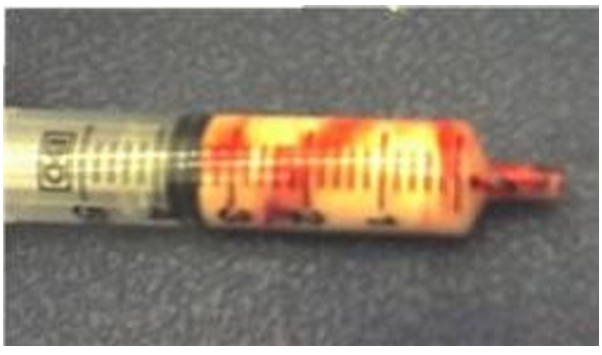

Figure 2. Lipemic appearance of blood sample.

Table 2. Lipid profile during hospitalization.

Laboratory test	Reference range	Hospitalization day				
		1	3	4	7	14
Triglycerides (mg/dL)	35-110	1,721	756	1,113	409	113
Total cholesterol (mg/dL)	0-200	1,001	1,007	734	447	173
LDL cholesterol (mg/dL)	0-100	628	591	474	329	112
HDL cholesterol (mg/dL)	36-73	31	65	37	36	38

composed of triglyceride and carries them from the liver to peripheral tissues where triglycerides are degraded by lipoprotein lipase. This tissue-specific effect of insulin on lipoprotein lipase results in diversion of triglycerides to adipose tissue for storage [7].

Under hypoinsulinemic conditions, such as uncontrolled diabetes mellitus, fat mobilization is greatly increased and its clearance decreased resulting in an oversupply of free fatty acids to the liver. This increase in lipolysis and decrease in utilization and excretion results in hyperlipidemia in patients with insulin deficiency. Newly diagnosed patients with type 1 diabetes mellitus, presenting with diabetic ketoacidosis have an absolute insulin deficiency that can result in severe hyperlipidemia as seen in our patient. The recognition of this association is important as severe hypertriglyceridemia can complicate diabetic ketoacidosis by the development of pancreatitis, which may increase the morbidity and mortality [8, 9, 10, 11]. As mortality rate is higher in children under the age of 5 years presenting with diabetic ketoacidosis, it may be prudent to screen them for hyperlipidemia and if present, for pancreatitis.

The extremely abnormal triglyceride level may suggest the presence of LPL deficiency (type 1 hyperlipidemia) or deficiency of apolipoprotein C II, especially if lipid abnormalities persist after the resolution of diabetic ketoacidosis and control of diabetes, and genetic studies may be necessary to confirm or exclude the diagnosis [12].

Similarly in our patient serum creatinine was significantly elevated at presentation but returned to normal after correction of dehydration and acidosis. We assume that severe dehydration might have been the cause for acute renal decompensation.

CONCLUSION

Severe hyperlipidemia causing lipemic serum in patients with diabetic ketoacidosis rarely reported in children less than 3 years of age. Risk of mortality in severe diabetic ketoacidosis is much higher in very young children. Diabetic ketoacidosis associated with hypertriglyceridemia, as observed in our patient, can increase this risk further. Early identification of type 1 diabetes mellitus by recognizing symptoms and signs, such as polyuria, polydipsia, failure to gain weight despite of increase in appetite, can prevent severe morbidity and mortality. We suggest that the lipid level should be monitored in children regardless of age, when presented with poorly controlled diabetes or diabetic ketoacidosis.

Conflict of interest The authors have no potential conflict of interest

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