Lipemic serum with Dyslipidemia in a 9-year-old girl with new-onset Type-1 DM presenting as Diabetic ketoacidosis: A Case report

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ABSTRACT:

Diabetic ketoacidosis associated lipemia is a rare and under-reported entity in children. Here, we discuss a young girl who presented with DKA and had a 'milky white' plasma appearance. Due to severe hyperlipidemia, both serum & blood investigations were not processed in initial few days. The treating team had to rely upon serial arterial blood gas and blood sugar for management. Later on, on day 5 of admission lipid profile showed hyperlipidemia. We emphasize that clinicians should remain aware of dyslipidemia with diabetic ketoacidosis and diabetic children with poor glucose control. It also should be kept in mind that blood measurements can be inaccurate in hyperlipidemic conditions; thus, stimulating the laboratory in the presence of hyperlipidemia may require a second measurement.

KEYWORDS:

Lipemic serum, DKA, diabetes, dyslipidemia

INTRODUCTION:

The most common endocrine metabolism disorder of the childhood is Type 1 diabetes which leads to impaired energy homeostasis due to insulin deficiency, and Diabetic ketoacidosis (DKA) is known to be the most common acute complication of this[1]. Rarely due to insulin deficiency hyperlipidemia or milky plasma can occur which can lead to complications like acute pancreatitis and lipemia retinalis[2]. The majority of cases of hyperlipidemia are secondary to disease and medication of which poorly controlled diabetes is most common[2]. Diabetic lipemia term was first used in nineteenth century to describe the combination of uncontrolled diabetes mellitus and dyslipidemia found in severe diabetic patients. It a form of acquired fat-induced lipemia or also called milky plasma[2]. Mild hyperlipidemia is common in diabetic ketoacidosis but, severe hyperlipidemia and milky appearance of plasma are extremely rare[3]. There is a limited number of cases with an association of severe hyperlipidemia and DKA in children[3].

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CASE PRESENTATION:

A 9-year-old girl was brought to the emergency with complaints of polyuria and abdominal discomfort for 10 days and fast breathing for one day. She did not have fever, cough, hematuria, dysuria, vomiting, or altered sensorium. The parents denied any weight change or change in appetite. She has no significant past medical and personal history. She has no family history of diabetes, dyslipidemia, or autoimmune diseases.

Vitals at admission: Temperature -98.7 °F, Pulse-

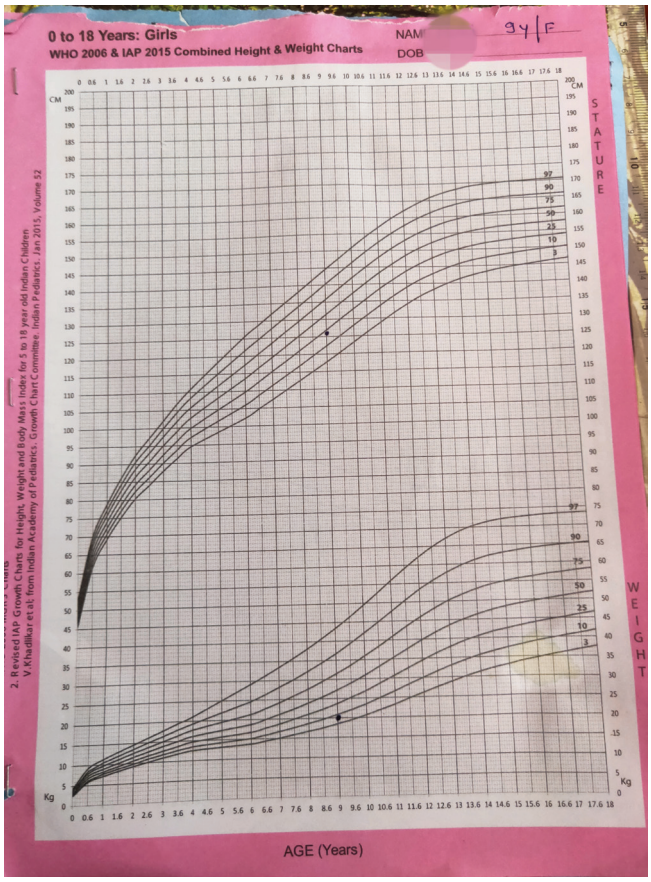

Figure 1: Growth Chart (Height b/w 10th to 25th centile, weight b/w 3rd to 10th centile)

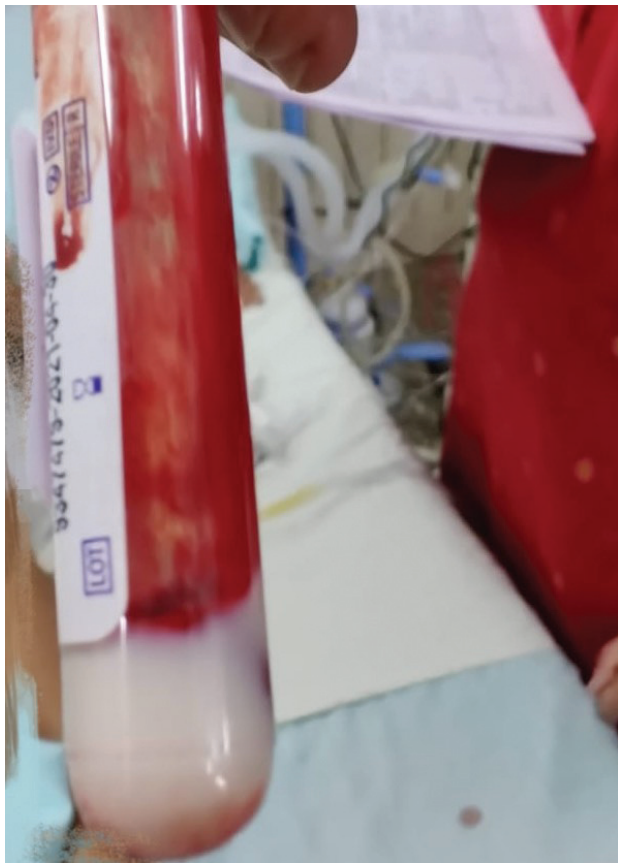

Figure 2: Lipemic Serum (Milky Plasma)

140/min (sinus tachycardia), RR- 38/min (deep and laboured breathing), BP- 100/76 mmHg (in-between 50th to 90th percentile), Saturation- 96%

Physical examination revealed no abnormality apart from dry mucosa and delayed capillary refill time. Her anthropometry was normal (Figure 1).

Her initial investigations suggested hyperglycemia, severe metabolic acidosis, ketonemia, ketonuria, and high HbA1c. The clinical picture was consistent with DKA. Intravenous fluids and insulin were initiated as per the management protocol of DKA (BSPED)[4]. The blood sample withdrawn for laboratory analysis was milky white in appearance (Figure 2) and because of lipemic serum many samples could not be processed, and we had only an arterial blood gas report, so we managed the case initially on the findings of arterial blood gas only. Assessment was done regarding the presence of xanthomas and lipemia retinalis which was normal. S. Amylase and S. lipase were normal. Intravenous insulin was replaced by subcutaneous insulin and oral nutrition started. The child remained clinically stable afterward. On 3rd day of admission, blood sample for histogram was processed, whereas serum investigations were reported on 5th day. Lipid profile on day 5 showed significant hyperlipidemia. The details of the investigations are mentioned in Table 1. No treatment for dyslipidemia was given during this period. The screening for lipoprotein lipase was not available.

DISCUSSION:

Children with insulin deficiency presenting with polyuria and polydipsia are more prone to develop Diabetic Ketoacidosis. About 30% of children with new-onset type 1 diabetes present with Diabetic ketoacidosis. Insulin being an anabolic hormone plays an important part in storage of not only glucose but also proteins and lipids[2]. In Diabetes mellitus, impaired lipid metabolism and its effects are known since long[5]. Catabolism of triglycerides into monoglycerides, glycerol and fatty acids is carried via lipoprotein lipase which is mainly segregated in adipose tissue, cardiac and skeletal muscle. This lipoprotein lipase which is a triglyceride hydrolase enzyme is stimulated by insulin secretion. The triglyceride rich chylomicrons help in uptake of fatty acids by muscle and adipose tissue. Chylomicron residues are taken by the liver, where they are converted to apolipoprotein, cholesterol esters, cholesterol, fatty acids, and amino acids. Very low-density lipoproteins (VLDLs) synthesized from triglycerides are transported to

Table 1: List of Investigation

Investigations	Day 1	Day 2	Day 3	Day 4	Day 5	Follow-Up
Hb			10.2		9.1	
TLC			6600		11100	
DLC	64/29/1/6		60/32/2/6			
Platelets			194000		350000	
RBS	550	478	489	310	237	
HBA1c	14.4					
Na	135	137	135	132	135	
K	3.4	4.3	2.9	2.8	3.1	
Ca(Ionized)	1.34	1.42	1.52	1.25	1.18	
Urine Ketone	+3				+1	
Urine Glucose	+3	+3			+2	
S. Acetone	Positive			Positive		
SGPT					19	
Total Protein			5.3		6	
S. Albumin			2.1		2.2	
BUN			54		40	
S.Creatinine			1.37		0.92	
S. Amylase			131			
S. Lipase			166			
S. Cholesterol					455	113
Triglyceride					391	96
H D L					22	58
L D L					276	36
V L D L					78.2	19.2
TC/HDL					20.68	1.95
LDL/HDL					12.55	0.62
SerumTSH			1.78			
pH	6.94	7.13	7.29	7.29	7.35	
pCO2	12	21	20	25	33	
TTG-IgA						0.59 (Negative)

peripheral tissues and degraded by lipoprotein lipase. Lipolysis increases when insulin levels are low which leads to triglycerides being stored in liver in the form of free fatty acids which ultimately leads to fatty liver and rapid increase in VLDL levels[1]. As in our case, the child showed severe hyperlipidemia with severe diabetic ketoacidosis.

Awareness regarding hypertriglyceridemia should be there for children's as well as clinicians as it can lead to lipemia retinalis and acute pancreatitis[2]. Besides, the treatment of diabetic lipemia is the same as DKA[6]. Some of the previous reports suggests role

of intravenous fluids and insulin infusion in rapidly decreasing plasma glucose as well as lipid levels[1,7]. Our patient was also managed as DKA and after resolution of DKA, lipid profile returns to normal on follow-up. Clinicians should also suspect some genetic mutations like lipoprotein lipase deficiency if the level of triglyceride does not decrease and remains persistently high.[1]. Severe lipemia impedes accurate measurements in laboratory tests by 3 mechanisms: 1) dispersion of light due to turbidity; 2) decrease in sample viscosity and; 3) division between polar and non-polar phases[8]. This particularly altered the value of biochemical parameters termed biochemical interference. Waseem *et al*[6] in a case report concluded that serum sodium, potassium, chloride, and bicarbonate values were low, whereas serum creatinine, triglyceride, total cholesterol were high amongst the children. In our case, we are unable to process blood investigations due to milky plasma except for arterial blood gas results.

Lessons learnt:

- The association of severe hyperlipidemia and diabetic ketoacidosis in children manifested as lipemic serum has been a rare and under-reported entity and patient might require repeated investigations as samples cannot be processed due to lipemic serum.
- Pediatricians must be aware of diabetic lipemia and its complications like acute pancreatitis, thus increase mortality.
- We recommend measurements of serum lipid levels in children with diabetic ketoacidosis or poorly controlled DM.

CONFLICT OF INTEREST: None
SOURCE(S) OF SUPPORT: None

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