Case Report

Severe Hypertriglyceridemia Causing Pancreatitis in a Child with New-onset Type-I Diabetes Mellitus Presenting with Diabetic Ketoacidosis

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Abstract

The triad of pancreatitis, hypertriglyceridemia, and diabetic ketoacidosis and its treatment has not been extensively discussed in the pediatric literature. We report a 4-year-old child with severe hypertriglyceridemia, pancreatitis, and diabetic ketoacidosis. Hypertriglyceridemia and pancreatitis with diabetic ketoacidosis can be successfully managed with insulin and hydration therapy in children. Early recognition of this triad is important as insulin requirements, recovery duration, and prognosis can be altered.

Keywords: Diabetic ketoacidosis, pancreatitis, severe hypertriglyceridemia

Introduction

Around 25% of newly diagnosed diabetic children present with diabetic ketoacidosis at the onset.[1] Severe hypertriglyceridemia (triglyceride [TG] >1000 mg/dL) is another rare complication of ketoacidosis which increases the risk of acute pancreatitis. Pancreatitis attributable to hypertriglyceridemia was seen in 4% of diabetic ketoacidosis episodes in adults.[2] However, the triad of acute pancreatitis caused by hypertriglyceridemia, along with diabetic ketoacidosis and its treatment, has not been extensively discussed in the pediatric literature. Only eight cases have been reported previously. We report the youngest child with the triad of severe hypertriglyceridemia, acute pancreatitis, and diabetic ketoacidosis.

Case Report

A 4-year-old female presented with vomiting, abdominal pain for 2 days, and progressive breathing difficulty and lethargy since last night. She had no fever, cough, or diarrhea. She also had polyuria, polydipsia, and 3 kg weight loss, despite good appetite during last 1 month. Past medical history was unremarkable and family history was unremarkable. On arrival, the child had Glasgow Coma Scale score of 11/15 (E3V3M5). She was severely dehydrated and had rapid breathing (72/min), pulse rate was 152/min, and peripheral pulses were weak with cold peripheries. Capillary refill time was more than 5 s. Her blood pressure was 76/40 mmHg and temperature was 37.1°C. Pupils were normal size, normally reacting, and meningeal signs were absent. Her abdomen was soft but tender. The rest of examination was normal. Her blood sugar was 404 mg/dl. Blood gas revealed pH 6.85, HCO_3 3 mmol/l, and pCO_2 10 mmHg. Urine revealed glycosuria 4+ and ketonuria (large). The child received normal saline bolus of 20 ml/kg and started on injection insulin and hydration therapy in the Pediatric Intensive Care Unit. Blood drawn for analysis was highly milky (lipemic) in appearance [Figure 1]. Laboratory values revealed hemoglobin 24.7 g%, total leukocyte counts 33.3×10^9/L, neutrophils 57.6%, lymphocytes 31.2%, platelet count $288/10^3/mm^3$, urea 18 mg/dl, creatinine 0.6 mg/dl, sodium 133 meq/L, potassium 3.5 meq/L, and initial glycated hemoglobin (HbA1c) 7.8%. Serum TG was 13846 mg/dl, total cholesterol 1267 mg/dl, amylase 150 U/L, and lipase 230 U/L. Sensorium, general condition, and metabolic acidosis

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gradually improved and diabetic ketoacidosis resolved over 36 h. However, pain abdomen persisted. Ultrasound abdomen revealed bulky pancreas. The child was continued on injection insulin, intravenous fluids for further 48 h. Pain abdomen improved and serum TG and cholesterol showed a decreasing trend [Table 1]. Repeat HbA1c at discharge was 14.2%. Serum C-peptide was low <0.3 ng/ml (0.81–3.85) and insulin antibodies were positive by enzyme immunoassay. The child was started on oral feeding and subcutaneous insulin. She was discharged 8 days after hospitalization. During follow-up, her lipid profile remained normal.

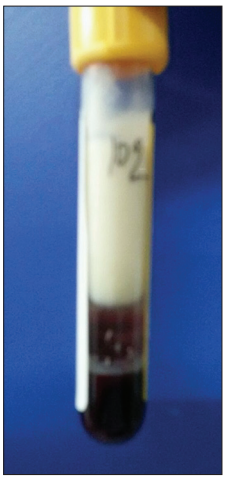

Figure 1: Lipemic appearance of the blood sample.

Table 1: Sequential laboratory results

Laboratory test	Reference range	Days					
		1	3	5	8	14	28
TG (mg/dl)	0-200	13,846	5609	2215	675	490	90
Cholesterol (mg/dl)	0-200	1267	929	710	555	395	110
Amylase (U/L)	25-125	150	90	40	100	135	60
Lipase (U/L)	13-60	230	240	110	442	133	45
pH	7.35-7.45	6.85	7.36				
PCO_2 (mmHg)	35-45	10	28				
HCO_3 (mmol/L)	22-26	3	15.8				
RBS (mg/dl)	60-140	404	222	102	242		

TG: Triglyceride; RBS: Random blood sugar

Discussion

This case highlights the challenges faced during management of severe diabetic ketoacidosis complicated by severe hypertriglyceridemia and resulting in acute pancreatitis. Our case presented with encephalopathy and possibility of cerebral edema, shock encephalopathy, or electrolyte disturbances was considered. Rapid improvement in Glasgow Coma Scale with volume resuscitation made strong possibility of shock encephalopathy as likely etiology. An important problem faced was an interpretation of laboratory measurements due to lipemic sample. Severe lipemia interferes with clinical laboratory testing through three mechanisms: (1) turbidity resulting in light scattering, (2) an increase in the nonaqueous phase of the sample, and (3) partitioning between the polar and nonpolar phases. Laboratory results were ambiguous in the case such as hemoglobin 24.7 mg% and HbA1c value of just 7.8%. Serum electrolytes could be ascertained after 12 h which has an important bearing as serum corrected sodium needs to be monitored to prevent cerebral edema. Even after resolution of diabetic ketoacidosis, the child continued to have pain in the abdomen which necessitated further evaluation. This rare complication of acute pancreatitis was considered, and intravenous fluid and insulin were continued for prolonged duration.

Diabetic ketoacidosis is an absolute insulin deficiency state leading to increased fat mobilization by activating lipolysis in adipose tissue and releasing increased free fatty acids. This accelerates the formation of very low-density lipoprotein (VLDL) in the liver. Reduced activity of lipoprotein lipase in peripheral tissue decreases the removal of VLDL from the plasma, resulting in hypertriglyceridemia. Moderate hypertriglyceridemia is common during episodes of diabetic ketoacidosis. However, severe hypertriglyceridemia is rare, and clinicians should be aware that devastating consequences such as acute pancreatitis or lipidemia retinalis can occur.[3] Review of available literature has shown 12 previous children with severe hyperlipidemia with diabetic ketoacidosis, and acute pancreatitis was present in only eight cases [Table 2].[3,4] Our case is the youngest case to present with the triad of severe hyperlipidemia, acute pancreatitis, and diabetic ketoacidosis. The risk of mortality in severe diabetic ketoacidosis is

Table 2: Reported cases of pediatric DKA with hypertriglyceridemia and acute pancreatitis

Patients	Age	Peak TG levels (mg/dl)	Management	Days to normalized of TG level
Cywinski *et al*., 1965[5]	12.5	>1000	Hydration and insulin	7
Slyper *et al*., 1994[6]	14	3119	Hydration and insulin	NA
Kadota-Shinozaki *et al*., 1997[7]	19	3386	Hydration and insulin	2
Hahn *et al*., 2010[8]	20	15,000	Hydration and insulin	3
Lutfi *et al*., 2012[4]	10	16,334	Plasmapheresis	2
Kota *et al*., 2012[9]	12	1020	Hydration and insulin	3
Aboulhosn and Arnason, 2013[10]	18	1724	Hydration and insulin	3
Wolfgram and Macdonald, 2013[11]	10	8300	Hydration and insulin	7
Our case	4	13,846	Hydration and insulin	8

TG: Triglyceride

much higher in children below 5 years and association with hyperlipidemia, and acute pancreatitis can further increase this risk. Appropriate management includes intravenous fluid and insulin administration according to diabetic ketoacidosis guideline; however, some may require plasmapheresis, especially if having organ failure.[4] A greater degree of clinical monitoring is required as laboratory results are usually delayed and unreliable.

Conclusion

Severe hypertriglyceridemia and acute pancreatitis can be seen with diabetic ketoacidosis in children below 5 years. Early recognition of this triad has important implications in the management of the patient as insulin requirements, recovery duration, and prognosis can be altered.

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Conflicts of interest

There are no conflicts of interest.

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