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Case Report

CASE REPORT

Severe dyslipidemia associated with diabetic ketoacidosis in newly diagnosed female of type 1 diabetes mellitus

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Abstract

Diabetic ketoacidosis (DKA) is considered as a serious complication of type 1 diabetes mellitus in pediatrics. Severe dyslipidemia in DKA is a rare eventuality. We report on a 10-year-old female presented with severe DKA. The serum was lipemic with severe hypertriglyceridemia and hypercholesterolemia. Laboratory workup: the values of glycemia, sodium and HbA1c were misleading; a method of dilution was used to obtain the correct values. Triglyceride and cholesterol returned gradually to normal levels only with the management of DKA without any complication. Mild dyslipidemia is a common feature in DKA, but severe dyslipidemia is a very rare event whose pathophysiology is not completely elucidated. It needs close surveillance because it might be responsible for acute pancreatitis and lipidemia retinalis.

INTRODUCTION

Type 1 Diabetes mellitus (T1DM) is considered as the first metabolic disorder caused by an insulin deficiency resulting from loss of pancreatic beta cells, with epidemic features especially in industrialized countries [1].

The incidence of T1DM has increased steadily over the last 25 years with a prevalence of almost 2 per 1000. Diabetic ketoacidosis (DKA) is the first manifestation of T1DM in 30% of patients [1]. Severe hypertriglyceridemia has been reported in limited cases with DKA. We report a case of a female diagnosed recently with T1DM by exhibiting DKA with severe dyslipidemia.

CASE REPORT

A 10-year-old Syrian female of non-consanguineous parents was admitted into the pediatric intensive unit care for the first presentation of severe DKA. She had polyuria and polydipsia for 2 months. There was no family history of DM or dyslipidemia. Physical examination: her weight was 22 kg (−2.3 standard deviation [SD]) and height 135 cm (M). Her vital signs were: temperature 37°C, blood pressure 100/70 mm Hg, heart rate140 bpm and respiratory rate 40 breaths per minute. The girl was pale with the presence of loss of consciousness, kussmaul respiration and severe dehydration. The first laboratory workup was misleading because of the lipemic serum. Methods of dilutions with saline solution were used to eliminate interference [2, 3]. Based on the laboratory tests (Table 1), she was diagnosed with severe DKA with hyperglycemia, severe metabolic acidosis and ketonuria. She had severe elevated levels of TG and TC. Computed tomography (CT) of the brain was normal. Normal levels of amylase, lipase and normal CT scan of abdomen excluded acute pancreatitis.

Management of DKA associated with hypertriglyceridemia was initiated with intravenous fluids and continuous insulin infusion at 0.1 unit/kg/h (ISPAD guidelines 2018, 3], in addition to biantibiotic therapy (cefotaxime 150 mg/kg/day, Ampicillin 200 mg/kg/day). Her DKA resolved within 72 hours of treatment, and then we resumed subcutaneous insulin treatment with oral nutrition. TG and TC levels were returned gradually to normal

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Table 1: Lab test on admission

Test	Result	Test	Result
Complete blood count	18×10^3/μl	*Blood chemistry*	580 mg/dL
White blood cells	78%	Glucose	30 mg/dL
Neutrophils	20%	Urea	0.5 mg/dL
Lymphocytes	7.8 mg/dL	Creatinine	135 mmol/l
Hemoglobin	626×10^3/μl	Sodium	2.5 mmol/l
Platelets	6.99	Potassium	2.5 g/dL
Blood gas	3.8 mEq/L	Albumin	65 U/L
PH	15.7 mm Hg	Amylase	55 U/L
HCO3	144 mm Hg	Lipase	5000 mg/dL
PCO2	96%	Triglyceride (TG)	1500 mg/dL
PO2		Cholesterol (TC)	158 mg/dL
SO2		C-reactive protein (CRP)	9.7%
		Glycosylated hemoglobin C (HbA1C)	Ketones +3
		Urine analysis	

Table 2: Case reports of hypertriglyceridemia in a new onset of DM associated with DKA

Author	Year	Age (years)	Sex	TG (mg/dL)	TC(mg/dL)	Management
Lutfi *et al.* [5]	2012	10	Female	16 334	—	Plasmapharesis
Kin *et al.* [6]	2012	2	Female	1721	1001	Rehydration—insulin
Lee *et al.* [7]	2019	14	Female	14 820	1004	Plasmapharesis
Walsh *et al.* [8]	2020	13	Female	3540	—	Fenofibrate—omega3

after 14 days of hospitalization. Ophthalmological consultation was normal. The results of: Adrenocorticotropic hormone, Cortisol, anti-TTG IgA were normal. Titration of Insulin, peptide-C and islet-specific pancreatic autoantibodies were unavailable.

At the age of 11 year, her height was: 142 cm (−0.5 SD), weight: 31 kg (−1 SD), tanner stage 3, HbA1c: 6.4%, TC: 157 mg/dL, TG: 125 mg/dL, and TSH: 4.62 mIU/L. She is actually on the regimen of (lispro: three daily injections, lantus: one daily injection) and on diet.

DISCUSSION

Severe dyslipidemia is a very rare event of ketoacidosis which increases the risk of many complications. Deficiency of insulin activates lipolysis in adipose tissue releasing increased free fatty acid, which in turn accelerates the formation of very low density lipoprotein (VLDL) in the liver. In addition, reduced activity of lipoprotein lipase (LPL) in peripheral tissue decreases removal of VLDL from plasma resulting in hypertriglyceridemia in DKA. Coexisting genetic predisposition might be an aggravated factor of the pathogenesis in severe cases of hypertriglyceridemia especially in the presence of mutations in the gene coding for LPL, which is located in chromosome 8; more than 100 mutations of that gene were reported [4]. Detecting the underlying genetic defect is essential for proper management. We could not perform the genetic analysis in our setting. Some cases of severe hypertriglyceridemia associated with DKA were reviewed in Table 2.

There is no guideline concerning the management of severe dyslipidemia associated with DKA. Continuous insulin infusion has been shown to stimulate adipocyte LPL activity, restore impaired LPL activity in both adipocytes and skeletal muscle leading to reduction in serum triglycerides [9]. Fibrates and omega-3 fatty acids can be considered in severe hypertriglyceridemia, Fibrates are not beneficial for patients with LPL deficiency [8]. Plasmapharesis is preserved for patients with hypertriglyceridemic pancreatitis. However, some patients may require plasmapharesis, especially those with multi-organ failure [5]. LPL gene therapy (Alipogene Tiparvovec) might be potential treatment in patients with LPL deficiency [10].

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CONFLICT OF INTEREST STATEMENT

None declared.

ETHICAL APPROVAL

No ethical approval is required.

INFORMED CONSENT

The patient parents have given written consent for publication of this case report.

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