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LETTER TO THE EDITOR

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Severe dyslipidaemia in diabetic ketoacidosis in 7-year female child: a rare presentation

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Sir,

Type 1 diabetes mellitus (DM) is an autoimmune disease caused due to insulin deficiency which is prone to develop diabetic ketoacidosis (DKA) [1]. Various factors precipitate DKA like skipping doses of insulin, infection, trauma, use of steroids. The diagnostic criteria for DKA are blood glucose > 200 mg/dl, ketosis and metabolic acidosis [2]. Insulin is required for lipoprotein lipase (LPL) activity. It hydrolysis triglycerides carried by chylomicrons and very low-density lipids. So, insulin deficiency results in hypertriglyceridaemia known in diabetes but severe hypertriglyceridaemia giving a milky look-like appearance to plasma, especially in the paediatric population, is rare [3]. The pathogenesis cannot be only explained by insulin deficiency, but may be aggravated by a coexisting genetic predisposition to hypertriglyceridemia, especially mutations in the gene coding for LPL which is located in chromosome 8 [4]. We present a case of a 7-year-old female child, 2nd in birth order product of non-consanguinous marriage full-term delivered by normal vaginal delivery. The patient is a known case of type 1 DM diagnosed in 2016 and was on our regular follow-up (historically optimally controlled blood glucose, with initial presentation as DKA) in paediatric OPD. She was on insulin Lispro 8–8-7 and Glargine 8 units SC bedtime. No family history of DM or dyslipidaemia. The patient presented with severe DKA due to missing multiple doses of insulin with an initial blood glucose of 850 mg/dl and ketonuria with venous blood gas showing severe acidosis (Table 1). There was no history suggestive of any cough, loose motions, fever, or any drug intake (steroids). Her plasma was lipemic as shown in Fig. 1. Her weight and height were 18 kg (25th percentile) and 115 cm (25th–50th percentile) respectively, body mass index = 13.84 kg/m^2. Clinical exam revealed irritability, no signs of insulin resistance, BP = 100/60, PR = 92b/pmnt, Spo2 = 95% on room air, and rest exam was unremarkable. SMR (Sexual maturity rating) as: A1B2P1. Patient was managed as per 2018 guidelines [5] with IV fluids (normal saline @ 60 ml/h and insulin(R) 0.1U/kg which was continued for 3 days. Her DKA resolved (acidosis resolved, she started taking orals, bicarbonate increased to18) and later she was shifted to multiple subcutaneous insulin (MSI) regimen as per total calculated dose (she required 34 units of insulin).

During hospitalization, she was found to have severe dyslipidaemia (Table 2) on multiple readings. There were no features like xanthelasma, tendon xanthoma, premature cardiac death in the family and any syndromic association. The lipid profile of his parents, brothers, and sisters was normal. Rest systematic examination was unremarkable.

Her liver function test and thyroid profile were normal as shown in Table 1. Her dyslipidaemia started settling during hospitalization and on discharge her lipid profile is shown in Table 2. We did not give any hypolipidemic drug for dyslipidaemia. Her plasma cleared after

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Table 1 Laboratory parameters in our patient

Haemoglobin (gm/dl)	TLC × 10^3	DLC	PLT × 10^5	MCV (FL)	MCH (pg/ml)
10.7	10.9	55/46	1.50	88	32
Urea (mg/dl)	Creatinine (mg/dl)	Bilirubin (mg/dl)	ALT (U/L)	AST (U/L)	ALB (g/dl)
20	0.3	0.65	26	25	3.85
TSH u 1U/ml	T4 (ug/dl)	T3(ng/ml)			
2.62	10.2	1.54			
PH	Na	K	HCO3		
7.0	133	3.36	2.0		
7.43	136	3.5	24.3		

TLC Total leucocyte count, *DLC* Differential leucocyte count, *PLT* Platelets, *MCV* Mean corpuscular count, *ALT* Alanine transaminase, *AST* Aspartate transaminase

Fig. 1 Lipemic plasma on admission

Table 2 Lipid profile of our patient

	TGs (mg/dl)	LDL (mg/dl)	HDL (mg/dl)	Cholesterol (mg/dL)
D1	1509	212	35	567
D3	1155	182	34	465
D5	850	150	37	300
D7	500	130	40	250
Discharge	300	110	42	200
After 1 month	168	82	45	150

TGs Triglycerides, *LDL* Low-density lipid, *HDL* High-density lipid

resolution of DKA as shown in Fig. 2. After 1 month of discharge her lipid profile normalized and she is in our regular follow-up doing well.

Severe dyslipidaemia which is defined as TGs level more than 1000 mg/dl is rarely associated with DKA in paediatric population [6]. Prevalence of severe hypertriglyceridemia is about 1–8% in adults but few data have been reported in children with severity ranging from asymptomatic to severe acute pancreatitis [7]. As such there are no guidelines about management of hypertriglyceridemia in DKA in acute setting. Usually, continuing insulin infusion for days help in clearing it but in refractory case plasmapheresis use, as has been reported [8].

In our case report dyslipidaemia improved during hospitalization due to intravenous fluids and insulin. This is due to the fact that major mechanism of hypertriglyceridemia is insulin deficiency. To our best of knowledge, this is perhaps first case reported from our state with severe dyslipidaemia associated with DKA in paediatric population.

However, there were limitations in our case, we could not do genetic testing to rule out other cause of dyslipidaemia. GAD-65 or ICA (Islet cell antibody) antibodies not done in this patient because of affordability issue.

Fig. 2 Clear plasma of the same patient after resolution of DKA

Authors' contributions
Dr. Ajaz Qadir prepared the manuscript. Dr. Shariq Rashid Masoodi finalized the manuscript. Dr. Abid Rasool collected the data.

Declarations

Ethics approval and consent to participate
Not required. It does not contain any studies with human or animal subjects performed by any of the author.

Consent for publication
Consent taken from patient for publishing: yes.

Competing interests
The authors declare that they have no competing interests.

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