

A CASE OF SEVERE HYPERTRIGLYCERIDAEMIA COMPLICATING NEW-ONSET TYPE 1 DIABETES MELLITUS

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INTRODUCTION

Extreme hypertriglyceridaemia is uncommon in the paediatric population but can have devastating consequences^{1,2}. It can lead to acute pancreatitis, cutaneous eruptive xanthomas and lipaemia retinalis³. Severe hypertriglyceridemia likely accounts for 1-10% of acute pancreatitis cases in adults but there is very limited data in children^{1,4}.

Hypertriglyceridaemia can be primary (genetic) or secondary in origin. Severe hypertriglyceridemia due to excessive lipolysis from lack of insulin action during diabetic ketoacidosis can provoke acute pancreatitis⁵.

AIM

- To highlight a clinical case of new-onset type 1 diabetes mellitus (T1DM) presenting in diabetic ketoacidosis (DKA) with acute pancreatitis owing to severe hypertriglyceridemia.

CASE HISTORY

- A previously healthy 13-year-old boy (34 kg, -1.67 SDS) presented to his local hospital with a short history of lethargy and abdominal pain. He was found to be in severe DKA (pH 6.88, glucose 21.5 mmol/L, bicarbonate 5.3 mmol/L, ketones 5 mmol/L). He received intravenous insulin and fluids. He was later transferred to our institution owing to reduced consciousness and agitation.
- A CT head ruled out cerebral oedema. His blood samples were lipaemic with a maximum triglyceride level of 53 mmol/L (normal <1.7mmol/L) and cholesterol 16.1 mmol/L. His amylase peaked at 989 IU/L (normal 25-125 IU/L), but CT abdomen was relatively unremarkable. His mild pancreatitis was managed conservatively.
- On day 2 of admission, he was transitioned to subcutaneous insulin with initiation of strict low-fat oral diet (fat <10 g/day). Serum triglycerides and amylase levels decreased rapidly to 4.21 mmol/L and 282 IU/L respectively by discharge.
- T1DM was confirmed by positive GAD and IA2 antibodies. He received intensive dietetic input after discharge and lipid profiles showed gradual improvement. With cautious dietary fat liberalisation and adequate insulin treatment, triglycerides (0.81 mmol/L) and cholesterol (3.2 mmol/L) completely normalized by three months (Figure 1) at which point he returned to full fat intake of 70-80 g/day
- Owing to a strong family history of T1DM and heart disease, he had a familial chylomicronaemia syndrome gene panel which was negative.

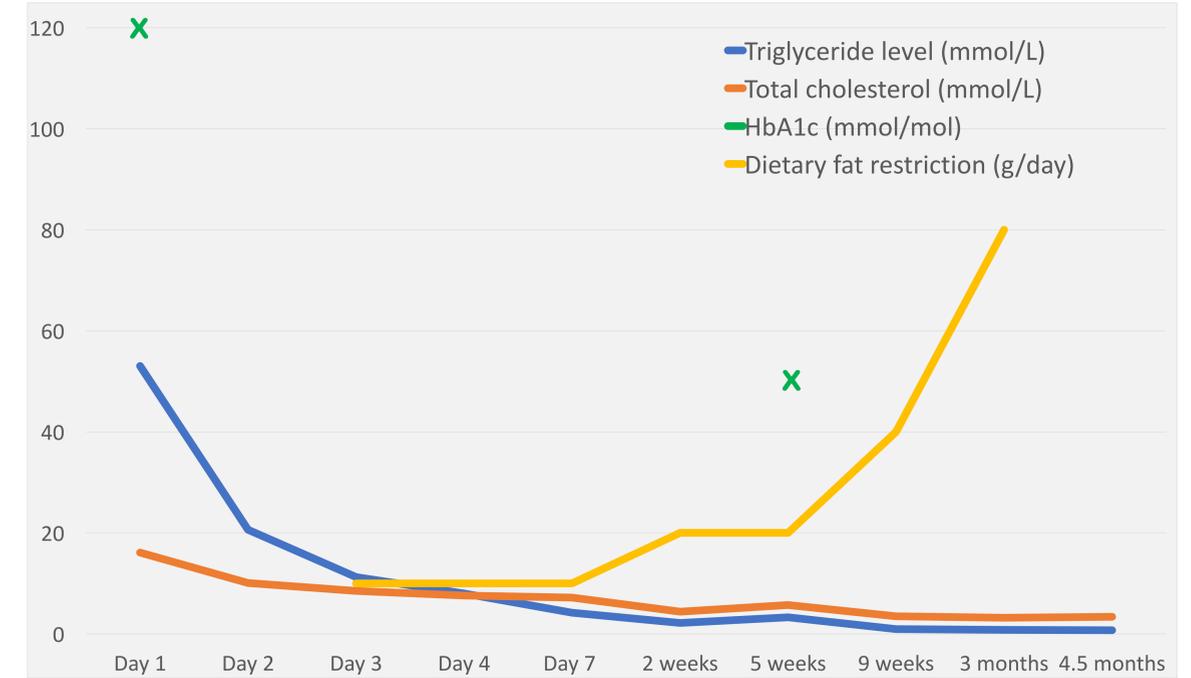


Figure 1: Lipids, HbA1c and dietary fat restriction over time

- This patient is under regular follow up by the paediatric diabetes team. He had good recovery from the acute episode and his current weight is 40.6 kg (-0.9 SDS).
- He is currently on multiple daily injections (Lantus, Novorapid with carbohydrate counting). His latest lipid profile was completely normal, even following an unrestricted diet.

CONCLUSIONS

- Although very rare in children, hypertriglyceridaemia can occur in T1DM owing to metabolic derangements and severe insulin deficiency.
- Increased hepatic free fatty acid uptake produces high very low density lipoprotein which causes hypertriglyceridaemia. It can lead to acute pancreatitis, especially with levels >10 mmol/L.
- Insulin treatment to facilitate lipoprotein lipase mediated metabolism and careful dietary fat restriction are the mainstay of management.
- Refractory cases warrant urgent plasmapheresis.
- Our case illustrates that early recognition and timely intervention can lead to a satisfactory outcome with conservative management, which may avoid plasmapheresis.

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