

Co-existence of new onset diabetic ketoacidosis with severe hypertriglyceridemia in a 9 year old girl

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INTRODUCTION

Insulin regulates the activity of lipoprotein lipase, the enzyme responsible for triglyceride metabolism.

Although mild hypertriglyceridemia is seen in diabetic patients, severe hypertriglyceridemia (>1000 mg/dL) is extremely rare in the pediatric population.

We present a case of co-existence of new onset diabetic ketoacidosis (DKA) with severe hypertriglyceridemia, together with therapeutic outcome.

CASE REPORT

A 9-year-old, previously healthy girl presented with a 1-month history of epigastric pain, 3 kg weight loss and a 10-day history of polydipsia, polyuria, and vomiting. On initial examination, she had dry mucous membranes while other system examinations were normal. Capillary glucose was 440 mg/dL, capillary beta-hydroxybutyrate 6 mmol/L, pH was 7.18 and bicarbonate was 8.7 mmol/L, indicating DKA. It was noted that the blood specimen was lipemic (Figure 1). Due to this lipemia, serum glucose and electrolyte levels were not accurately measured. Fluid replacement and insulin infusion were started.

Analysis of the sample after ultracentrifugation showed glucose was 410 mg/dL, sodium was 123 mmol/L and triglyceride was 15495 mg/dL. Amylase and lipase were normal. There were no findings associated with hyperlipidemia, such as xanthelasma, eruptive xanthoma, and lipemia retinalis, on examination. The patient did not have a family history of remarkable cardiovascular disease and/or hyperlipidemia. Parental lipid levels were normal. Although DKA resolved at the 24th hour of treatment, insulin infusion was continued. Triglyceride levels decreased rapidly. At the 72nd hour of treatment, she was allowed to start a low-fat diet and started subcutaneous insulin, omega 3 and gemfibrozil therapy. On the 5th day of hospitalization, triglyceride levels returned to normal. Pancreatitis did not develop on follow-up. Gemfibrozil was stopped after three weeks. We did not perform genetic analysis for disorders of lipid metabolism because of the rapid improvement in hyperlipidemia with insulin therapy and the absence of a family history of hyperlipidemia and early cardiovascular events. In subsequent follow-up, there was no increase in triglyceride levels and the last triglyceride measurement was 50 mg/dL.

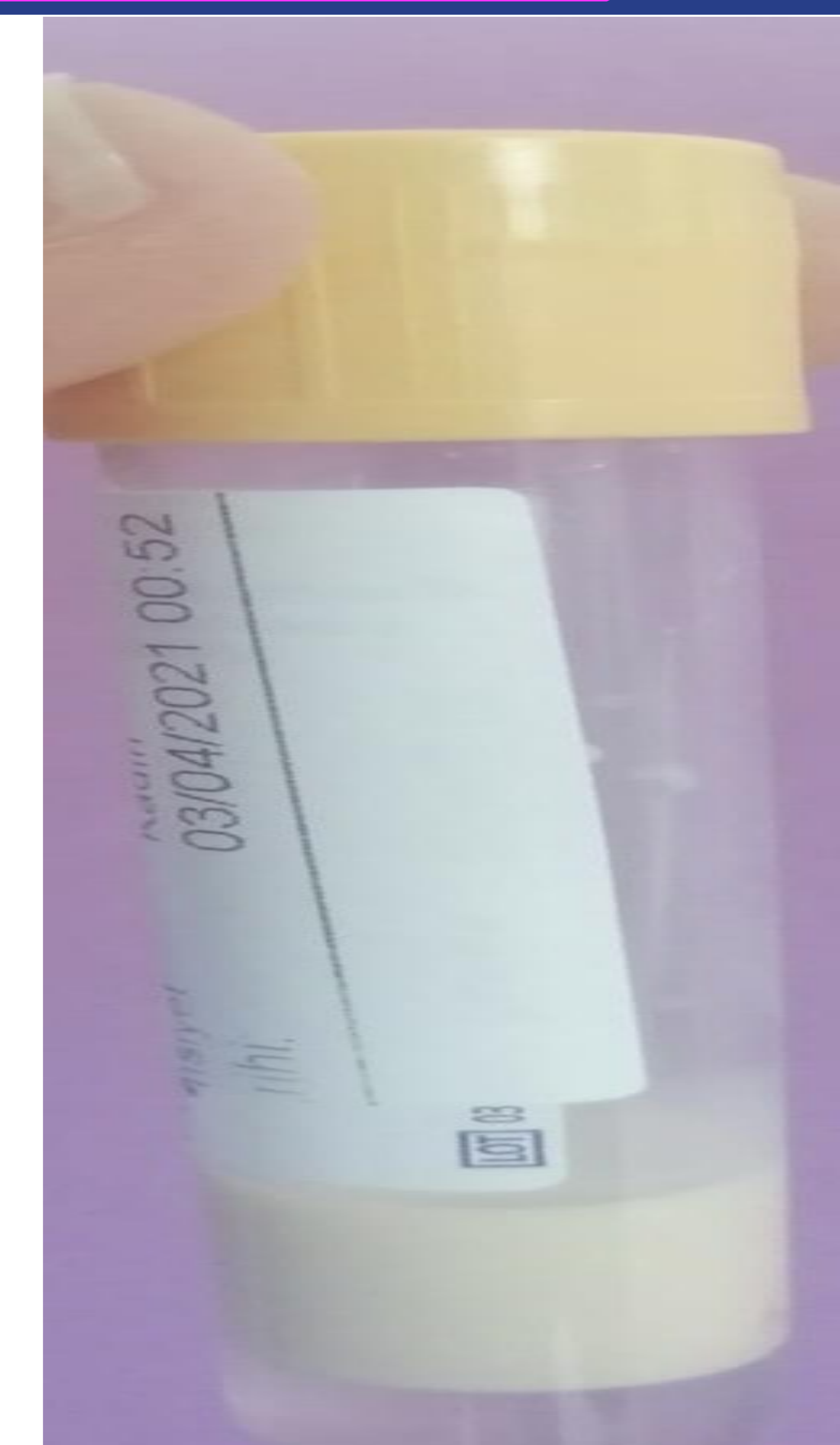


Figure 1: Figure shows the lipemic blood specimen.

CONCLUSIONS

To the best of our knowledge this case is one of the most extreme elevations of hypertriglyceridemia reported in a pediatric patient with DKA. Triglyceride elevation was thought to be secondary to DKA, due to the absence of family history and hyperlipidemia phenotype coupled with rapid recovery with insulin therapy.

We suggest that insulin infusion should be continued in children with DKA and severe hypertriglyceridemia, even after resolution of the DKA.

