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RESEARCH ARTICLE

EXTREME LIPAEMIC DIABETIC KETOACIDOSIS IN AN UNDIAGNOSED TYPE 1 DIABETES MELLITUS TEENAGER

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ABSTRACT

This case report describes the presentation of a 17 year old female teenager who presented with a three day history of colicky central abdominal pain, nausea and vomiting. She denied any fever and had no significant past medical history. She is known to have a strong family history of Type 2 diabetes mellitus. Physical examination revealed severe dehydration. She was tachycardic with a feeble pulse. Her bedside capillary blood sugar was 21mmol/L with an arterial blood gas showing severe uncompensated metabolic acidosis with respiratory compensation. Her dipstick urine ketones was 3+. The blood samples taken were grossly lipaemic and turbid which were consistent with severe lipaemic diabetic ketoacidosis. She did not exhibit any stigmata of chronic hyperlipidemia. The fasting triglyceride and low density lipoprotein (LDL) levels were moderately elevated. With aggressive fluid resuscitation and continous intravenous insulin infusion replacement, her lipaemia and ketoacidosis subsequently resolved within 48 hours resulting in euglycemia. She was then switched successfully to basal bolus insulin regimen in which her blood sugars remained well controlled during subsequent follow ups at the specialist clinic.

INTRODUCTION

Diabetic lipaemia associated with diabetic ketoacidosis is a well recognized but a rare condition (McLean *et al.,* 2009). It is a life threatening condition. Why this occurs to some patients and not others is unknown (McLean *et al.,* 2009). Lipaemia is a medical term often used to describe the turbidity of a blood sample due to lipid presence. Those with severe diabetes ketoacidosis have a much higher mortality and high risk of complications (Diabetes Ketoacidosis treatment protocol, Barbara Davis Center for Childhood Diabetes, 2013). Severe diabetic ketoacidosis usually results from defective metabolism of dietary lipids and increased lipolysis which causes accumulation of abundant free fatty acids. Due to increased lipolysis and decreased lipogenesis, abundant free fatty acids are converted to ketone bodies: acetoacetate, β-hydroxybutyrate (β-OHB), and acetone (Kitabchi and Gosmanov, 2012). Lipaemic diabetic ketoacidosis can be effectively treated with aggressive fluid resuscitation and continous intravenous insulin infusion therapy.

Case Presentation

A 17 year old female teenager presented with symptoms of colicky central abdominal pain, diarrhea and vomiting for a duration of three days. She reported about three to four episodes of vomiting and diarrhea daily for the past three days. The stool was described as watery with no slimy discharge or blood seen. The colicky abdominal pain was relieved partially with defecation. She was well prior to this. She denied taking any outside food. Other family members who lived with her did not have any of these symptoms. There was no fever. She did not complain of any osmotic symptoms such as polyuria, polydipsia or polyphagia. However, she admitted to having unexplained loss of weight for the past month with generalized lethargy. She denied any usage of traditional/herbal or over the counter medications. No recent travel. There was no symptoms of thyroid over or underactivity. She had a strong family history of Type 2 Diabetes Mellitus. She was a teetotaler and a non smoker. Physical examination revealed severe dehydration with lethargy. She had a thin habitus with a BMI of 18 kg/m^2.

She was tachycardic with a heart rate of 120 beats per minute, feeble in character. Her blood pressure was maintained at 100/62 mmHg. She was alert and afebrile with Kussmaul respiration of 28 breaths per minute. Her breath smelt of ketones. She had obvious lipiduria. There was no stigmata of chronic hyperlipidemia. Her lungs were clear with equal breath sounds. She had normal heart sounds. On abdominal examination, there was vague tenderness over the periumbilical area with no palpable masses. The bowel sounds were hyperactive in nature. Fundoscopy showed normal retinal vessels and did not reveal any lipaemia retinalis. Venous and arterial blood were grossly lipaemic and turbid (Figure 1). Bedside capillary glucose was 21 mmol/L. The Arterial blood gas revealed severe uncompensated metabolic acidosis with a

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pH of 7.11, PaO_2 of 117mmHg, $PaCO_2$ of 15 mmHg, bicarbonate of 10 mmol/L and base excess of -25 mmol/L. Bedside urine ketones stood at 3+.Her hemoglobin count was at 12.7 g/dL, total white cell count of 9.0 x 10^9/L and platelet count of 160000/uL. Her renal profile was deranged with the serum creatinine at 202umol/L and urea at 3.5 mmol/l. Electrolytes were within the normal limits. The liver function tests parameters, serum amylase and lipase were all within normal ranges.. The fasted lipid profile showed elevated triglycerides of 9.8mmol/L and LDL of 4mmol/L. Her fasted c-peptide level was low with a HbA1c of 11%. Lipoprotein lipase genetic testing was not done as it was not available nationwide. Her autoantibodies mainly the islet cell and glutamic acid decarboxylase (GAD) autoantibodies were detected.

Due to the Kussmaul respiration and severe metabolic acidosis, she was referred to the anaesthesiology team for respiratory support. Initially she was supported with non invasive ventilation which subsequently resulted in intubation in view of impending respiratory collapse. She was nursed and monitored in the intensive care unit. This patient was aggresively hydrated with intravenous fluids using central venous pressure monitoring to guide fluid balance. She was on continous intravenous insulin infusion for a few days until the ketoacidosis fully resolved before being switched to basal bolus insulin. Her condition improved over the next few days. The lipaemia and lipiduria subsequently resolved within 48 hours of admission and so with her renal function. Subsequently as her condition improved, she was started on basal bolus insulin-subcutaneous aspart 16 units thrice daily and glargine 32 units prebed. She was counseled by the dietitian and referred to the ophthalmologist for formal eye screening. She was seen on follow up at the specialist clinic. Her blood sugars were optimally controlled and the dosages of the insulin were adjusted accordingly.

Figure 1.

DISCUSSION

Diabetic lipaemia as illustrated in this case is rare and seldom present as a complication of diabetic ketoacidosis. It can result in significant mortality if treatment is not instituted immediately. In many earlier reports, lipid profile actually improved with standard diabetic ketoacidosis treatment alone. This enhanced lipid metabolism is a result of probable absolute insulin deficiency commonly seen in Type 1 diabetes in which it becomes an important source of energy and contributes to the serum lipaemia. The exact mechanism is unclear but transient insulin deficiency may cause a decrease in the activity of lipoprotein lipase accounting for the much more common moderate hyperlipidemia (Howard, 1987). The hyperglycemia, ketosis and dehydration result in production of counter-regulatory hormones which causes further hyperglycemia and lipolysis. Therefore, the emphasis on appropriate hydration and continous intravenous insulin infusion replacement for the first few days are vital to reduce the intensity of the lipolysis. In this patient, her blood samples were extremely lipaemic which caused significant difficulty in analysis using standard centrifugation measures. Initially she was fasted for more than 12 hours before her blood samples were drawn. Despite being fasted, the samples were still grossly lipaemic. Lipaemic samples are cause of analytical errors and present challenges for laboratories, particularly for those without ultracentrifuges (Dimenski *et al.,* 2011). The lipaemia could affect the accuracy of many biochemical test results. The procedure utilized using a high speed micro-centrifuge showed it is effective in reducing lipid levels and provides a suitable alternative to ultra centrifuged samples to provide accurate results (Dimenski *et al.,* 2011).

There are other factors that might contribute to lipaemic serum. Hence the differential diagnosis for lipaemia. Certain proportion of the lipemic samples in the laboratory originates from various pathophysiological conditions such as multiple myeloma, acute pancreatitis, kidney failure or hypothyreosis, however some preanalytical factors significantly contribute to lipemia (Nora Nikolac, 2014). The most common preanalytical cause of lipemia is inadequate time of blood sampling after the meal (Nora Nikolac, 2014). In this patient, her fundus did not reveal any lipaemia retinalis. Lipemia retinalis is a rare and asymptomatic condition which occurs when high levels of triglycerides and chylomicrons are present in the blood (Fred *et al.,* 1999). Creamy white appearance of retinal vessels occurs when triglyceride value reaches more than 2000 mg per deciliter (Cypel and Manzano, 2008).

Conclusion

In conclusion, diabetic lipaemia should be considered in a patient who presents with severe diabetic ketoacidosis. It carries a high mortality if not swiftly treated. Aggressive fluid resuscitation and adequate insulin replacement remain the mainstay of treatment in this condition. Careful examination of other organ systems are of paramount importance to avoid chronic complications of the disease.

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