Case report

Not all raised blood sugars are diabetes!

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Introduction

Hyperglycaemia in hospitalized non-diabetic patients with acute medical or surgical conditions is common. This can be due to stress hyperglycaemia where the raised plasma glucose will resolve spontaneously after the resolution of acute illness, while in other cases, this represents undiagnosed diabetes which has a high prevalence (~20%) among hospitalised patients.¹ Early identification of diabetes is important to improve the long-term outcome.² In clinical practice, however, differentiating between undiagnosed diabetes and stress hyperglycaemia among non-diabetic subjects can be challenging as illustrated in this case report.

Case 1

A 20-year-old asthmatic female was admitted following a significant paracetamol overdose. She ingested a total of 25 g of paracetamol with the level of 265 mg/l at 8 h and was given parvolex infusion. Salicylate level was normal. The admission plasma glucose was 17.9 mmol/l with heavy ketonuria. The arterial blood gas showed pH 7.26 and HCO₃⁻ 14.2 mmol/l. Glycosylated haemoglobin, HbA1c, was not checked on admission. She was treated as newly diagnosed type 1 diabetic presenting with diabetic ketoacidosis. Her clinical progress was uncomplicated. The metabolic acidosis resolved with insulin infusion and she was subsequently commenced on basal bolus insulin injections. After 1 week, she was discharged from hospital. Her diabetes was monitored in the diabetes clinic and over the next 18 months, she experienced repeated episodes of hypoglycaemia resulting in her insulin dose being substantially reduced and ultimately stopped. HbA1c level was between 5.0% and 5.5% during this period. The anti-GAD and islet cell antibodies were negative. The diagnosis of diabetes was questioned and oral glucose tolerance test (OGTT) was performed on two separate occasions, both of which were normal excluding this diagnosis. The patient was relieved not to continue with life-long insulin injections and home blood glucose monitoring.

Case 2

A 38-year-old morbidly obese (BMI 41) male was admitted with severe sepsis secondary to peritonsillar abscess and acute renal failure. He had no significant past medical history. As he was extremely unwell with reduced conscious level, he was transferred to intensive care unit. On admission, the plasma glucose was raised at 47 mmol/l with pH 6.9 and HCO₃⁻ of 7 mmol/l. Anti-GAD and islet cell antibodies were negative. Nevertheless, he was treated as a newly diagnosed type 1 diabetic presenting with diabetic ketoacidosis. He also developed abdominal distention and CT scan showed multiple abscess collections with the possibility of perforated appendix. Emergency laparotomy showed a collection in the right iliac fossa consisting of ischaemic necrotic liquefied bowel. His subsequent clinical progress was complicated by pneumonia and intra-abdominal abscess which required drainage and repeated courses of
intravenous antibiotics. After the hyperglycaemic episode in the initial stages of this admission which responded well to insulin infusion, the blood glucose settled spontaneously and remained between 5 and 8 mmol/l. He did not require further treatment with insulin. HbA1c performed 2 months after admission was normal at 5.6%. After 4 months, he was discharged from the hospital. A year after the initial admission, HbA1c was 5.9% and the OGGT showed a normal 2 h post-challenge glucose level of 7.3 mmol/l, thus excluded diabetes.

Discussion

Our case report highlights the difficulty in differentiating between diabetes and stress hyperglycaemia in clinical practice. The clinical dilemma was reflected by the presence of acute metabolic acidosis with hyperglycaemia in both cases and in Case 2, severe hyperglycaemia to the level not commonly associated with the stress of acute illness. In retrospect, the metabolic acidosis in these cases could be attributed to paracetamol overdose and ischaemic necrotic bowel. Furthermore, hyperglycaemia has been observed in paracetamol overdose. The true diagnosis only became clear after a protracted time of observation with repeated investigations to monitor for diabetes, particularly after the resolution of the acute illness episode. Case 2 was not started on diabetes treatment as his hospital admission was prolonged which gave the opportunity to closely monitor the glycaemic profile. For Case 1, performing glycosylated haemoglobin, HbA1c which is a measure of the glycaemic exposure in the preceding 8–10 weeks, may have helped to exclude diabetes during her hospital admission. Misdiagnosis of diabetes has undesirable clinical ramifications, namely, inappropriate anti-hyperglycaemic treatment with associated hypoglycaemic complications, implications for lifestyle issues such as driving and the psychological repercussion of living with a chronic disease condition.

Some studies reported increased mortality associated with stress hyperglycaemia among non-diabetic subjects in intensive care units or those who had acute myocardial infarction, acute coronary syndrome and cerebrovascular disease. However, the prognostic implication of stress hyperglycaemia for our subjects is unclear since they were clearly a different cohort from these study populations in terms of age and comorbidity. A recent study showed that stress hyperglycaemia may be a predictor of future diabetes. This is plausible given the underlying pathophysiology of stress hyperglycaemia that involves production of counter-regulatory hormones and inflammatory cytokines leading to increased hepatic glucose production, insulin resistance, lipotoxicity and glucotoxicity which may unmask the underlying beta cell dysfunction. The marked hyperglycaemia in Case 2 could be due to the exaggerated inflammatory and neuroendocrine responses triggered by the severe acute illness. In the longer term, he may be at risk of developing type 2 diabetes due to the presence of obesity. Long-term follow-up with annual blood glucose monitoring may need to be considered.

In conclusion, the possibility of stress hyperglycaemia has to be considered even though the picture of clinical presentation is suggestive of diabetes. In our experience, the guideline for the management of inpatient hyperglycaemia does not differentiate between stress hyperglycaemia and diabetes. For the undiscerning clinician who may not appreciate the transient and dynamic nature of stress hyperglycaemia that responds to the changes in disease course, there is a tendency to diagnose hyperglycaemic patients as diabetics. From the patient perspective, making the correct diagnosis is imperative as it can have a significant impact on their quality of life.

Conflict of interest: None declared.

References


