CASE REPORT

Stroke as the presenting feature of new onset diabetes in a young man

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SUMMARY

A 34-year-old man presented to a hospital with a 7-day history of nausea, vertigo, ataxia and frontal headache. Examination revealed ipsilateral cerebellar signs. CT of the brain demonstrated left cerebellar hypodensity suggestive of ischaemic stroke or space occupying lesion. Full blood count showed a markedly raised haemoglobin (219 g/L) and haematocrit (0.56). Admission urinalysis was performed but the results not reviewed. Owing to patient deterioration, an arterial blood gas was performed. This showed profound metabolic acidosis. Repeat urinalysis was positive for glucose and ketones. MRI of the brain confirmed ischaemic stroke. The underlying cause of this was hyperviscosity secondary to relative polycythaemia, resulting from undiagnosed diabetic ketoacidosis as a first presentation of diabetes. This case report highlights ischaemic stroke as an unusual presenting feature of diabetic ketoacidosis. Notably, the underlying diagnosis of diabetic ketoacidosis was initially missed, thereby emphasising the importance of performing an admission urinalysis and acting on the results.

BACKGROUND

Diabetes is a common medical condition currently affecting approximately 2.9 million people in the UK. Of these 450 000 have type 1 diabetes with the remaining 2.45 million having type 2 diabetes.¹ Diabetic ketoacidosis (DKA) has long been recognised as a key clinical feature of type 1 diabetes and is the presenting feature in 29% of new cases.² However, DKA has also been identified in people presenting with type 2 diabetes. This subtype is now referred to as ketosis prone type 2 diabetes,³ and is most commonly but not exclusively seen in African-American and other non-white ethnic groups.^{3–5}

This case is important as it highlights ischaemic stroke as an unusual presenting feature of new onset diabetes in a young man. Furthermore, the importance of obtaining and acting on admission serum glucose and urinalysis in acute medical patients is emphasised.

CASE PRESENTATION

A 34-year-old man, web designer, presented to his local district general hospital with a 7-day history of nausea, vertigo, ataxia and frontal headache. Four days prior to this presentation he had been prescribed oral antiemetics by the out of hours general practitioner service for presumed viral labyrinthitis. However, his oral intake continued to decline and this, in conjunction with worsening

frontal headache, precipitated his presentation to hospital. There was no relevant medical history and he took no regular medications. There was a family history of type 1 diabetes in his maternal grandmother. He had never smoked, took alcohol only occasionally and denied recreational drug use. He reported an active lifestyle playing football twice a week.

On general examination he was a tall thin Caucasian man with a body mass index (BMI) of 21. He was noted to be dehydrated and photophobic. He was tachycardic (114/min), tachypnoeic (22/min) and had a mildly raised blood pressure of 143/90 mm Hg. Neurological examination revealed left-sided cerebellar signs with dysidiadochokinesis, nystagmus and past pointing. The remainder of the systemic examination was unremarkable.

INVESTIGATIONS

Routine admission blood tests revealed markedly raised haemoglobin of 219 g/L and haematocrit of 0.56. He had a mildly raised white cell count of $14.9 \times 10^9/L$ and a platelet count of $217 \times 10^9/L$. Acute kidney injury was also identified with creatinine 122 $\mu\text{mol/L}$, urea 10 mmol/L and a low sodium of 122 mmol/L. An ECG showed sinus tachycardia (110 bpm). Urgent non-contrast CT of the brain was performed which showed hypodensity in the left cerebellar hemisphere with associated oedema and obstructing hydrocephalus. The underlying abnormality was felt to be either a space occupying lesion or ischaemic stroke. In view of the obstructing hydrocephalus and need for further neurological imaging, the patient was immediately transferred to the regional neurosurgical centre. On arrival a CT angiogram was performed which showed no evidence of arterial dissection. A subsequent MRI of the brain demonstrated left cerebellar infarction with cortical haemorrhagic transformation and secondary hydrocephalus. The possibility of an underlying space occupying lesion was therefore excluded.

The following day the patient deteriorated and became drowsy with an increasing respiratory rate. An arterial blood gas was therefore performed on room air which demonstrated a metabolic acidosis with partial respiratory compensation: H^+ 60 nmol/L, partial carbon dioxide tension 2.25 kPa, arterial oxygen tension 16.4 kPa, HCO_3^- (std) 10.3 mmol/L, HCO_3^- (act) 6.8 mmol/L and BE -17.7 mmol/L. The anion gap was calculated and found to be raised at 22 mmol/L. To elucidate the underlying cause of this, further investigations revealed elevated serum glucose of 16.9 mmol/L

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with glucose and ketones detected on urinalysis. On review of the case notes from the admitting hospital the admission serum glucose was also raised at 17.2 mmol/L with a positive urinalysis for glucose and ketones. Unfortunately, neither of these results had been noted, acted on or communicated to the receiving neurosurgical centre. A diagnosis of DKA as a first presentation of diabetes was made and subsequently confirmed by markedly raised glycated haemoglobin of 93 mmol/mol (10.7%).

Further investigations were performed to exclude any underlying causes for stroke in a young patient. These included a full vasculitis screen, anticardiolipin antibody, homocysteine level, lipid profile, HIV test and syphilis serology all of which were within normal limits. An echocardiogram (non-bubble) was also performed which demonstrated normal left ventricular function, with no evidence of endocarditis and no patent foramen ovale. Additional outpatient investigations were normal including a 24 h tape and full thrombophilia screen.

DIFFERENTIAL DIAGNOSIS

On initial presentation to hospital the most likely diagnosis was felt to be atypical migraine given the patient's age and lack of comorbidities. However, the presence of ipsilateral cerebellar signs necessitated the exclusion of stroke or space occupying lesion.

With regard to the markedly raised haemoglobin of 219 g/L and haematocrit of 0.56, the most likely explanation was felt to be relative polycythaemia from profound dehydration. Primary and secondary polycythaemia were also considered. However, these were thought to be much less likely and therefore screening was not immediately performed.

The unexpected finding of profound metabolic acidosis with a raised anion gap changed the diagnostic focus. The potential underlying causes included lactic acidosis, renal failure, salicylate overdose, methanol/ethylene glycol consumption or DKA. However, in the context of a raised serum glucose and urinalysis positive for glucose and ketones, a diagnosis of DKA as a first presentation of diabetes was made.

The differential for the patient's new onset diabetes was either type 1 diabetes or ketosis prone type 2 diabetes. The diagnosis was, however, felt to strongly favour type 1 diabetes, supported by his relatively young age of 34, active lifestyle, BMI of 21, Caucasian ethnicity and family history of type 1 diabetes. As the diagnosis was not felt to be in doubt, autoantibodies were not performed. However, the author recognises that these may have provided further useful information.

With the benefit of further neuroimaging, the possibility of a space-occupying lesion was excluded and a left-sided ischaemic cerebellar stroke was confirmed. Risk factors for stroke were examined but few could be identified. He was a non-smoker, took alcohol infrequently and had no family history of stroke. He had a normal BMI, normal lipid profile and exercised twice a week. The only positive risk factor identified was the newly diagnosed diabetes. Causes of ischaemic stroke in a young person were considered including: atrial fibrillation, arterial dissection, vasculitis and thrombophilia but were ruled out with appropriate investigations. In conclusion, the ischaemic stroke was attributed to hyperviscosity secondary to relative polycythaemia, resulting from undiagnosed DKA as a first presentation of diabetes.

TREATMENT

Initial treatment was centered around the finding of cerebral oedema and obstructing hydrocephalus. The patient was therefore started on intravenous dexamethasone 8 mg twice daily. Slow intravenous fluid therapy in the form of six hourly 0.9%

saline was initiated. Owing to the hyponatraemia noted on admission (122 mmol/L), more aggressive fluid resuscitation was not instituted due to concerns surrounding rapid correction of serum sodium.

Once the diagnosis of DKA was performed, appropriate intravenous fluid resuscitation was started along with an insulin sliding scale as per the hospital guideline. After 3 days the sliding scale was converted to a basal bolus insulin regime of lantus and novorapid.

When subsequent neuroimaging confirmed ischaemic stroke, the patient was given aspirin 300 mg daily, which was changed to clopidogrel 75 mg daily on discharge. Antiembolism compression stockings were applied in conjunction with intermittent pneumatic compression devices until the patient was mobile.

OUTCOME AND FOLLOW-UP

The patient's glycaemic control rapidly improved following institution of insulin sliding scale. Haemoglobin and haematocrit normalised following appropriate fluid resuscitation to 145 g/L and 0.40, respectively. Furthermore, renal function rapidly returned to baseline. From a neurological perspective, the patient made an excellent recovery with only minimal input required from the inpatient physiotherapy team.

He was discharged home from hospital 11 days after admission with minimal residual neurological deficit. On review in neurology clinic 6 months later he was back playing sport, with only mild dizziness when changing direction quickly. His clopidogrel was therefore stopped and he was discharged from follow-up at the regional neurosurgical centre. With regard to his diabetes care, this is followed up at his local district general hospital. His diabetes remains well controlled 10 months after initial presentation and he continues on a basal bolus insulin regime of lantus and novorapid. He was questioned as to whether he had suffered from symptoms of diabetes prior to his acute presentation to hospital. In hindsight he admitted to polyuria, polydipsia and weight loss of one stone in 3 months preceding his presentation.

DISCUSSION

Diabetes and DKA are associated with a prothrombotic state through a number of mechanisms including abnormalities in coagulation factors and platelet activation along with changes in blood volume.^{6,7} However, DKA associated with stroke in adults remains rare.⁸ On review of the literature, there are only a small number of case reports regarding the development of ischaemic stroke in adult patients with DKA.⁸⁻¹⁰ Of note, all of these patients had established type 1 diabetes and therefore stroke was not the presenting feature as in our case.

This case report is important as it highlights ischaemic stroke as an unusual presenting feature of new onset diabetes in a young man. The diagnosis of DKA was initially overlooked due to the distraction created by the nature and severity of the presenting

Learning points

- ▶ Stroke can be a rare presenting feature of diabetic ketoacidosis (DKA).
- ▶ Perform and act on urinalysis and serum glucose on every acute medical patient.
- ▶ When considering common causes of metabolic acidosis, always calculate the anion gap.
- ▶ Type 1 diabetes and ketosis prone type 2 diabetes can present with DKA.

neurological symptoms. Appropriate tests in the form of serum glucose and admission urinalysis were performed but the results not reviewed. This led to a delay in diagnosis of the underlying diabetes. This highlights the importance of obtaining and acting on admission serum glucose and urinalysis in acute medical patients.

Contributors RJ had the idea for the article and performed the literature search. RJ and OR wrote the article. RJ is the guarantor. OR was involved in the clinical management of the case. EM has provided expert endocrinology advice.

Competing interests None.

Patient consent Obtained.

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REFERENCES

- 1 Diabetes UK. *Diabetes in the UK 2012: key statistics on diabetes*. <https://www.diabetes.org.uk/Documents/Reports/Diabetes-in-the-UK-2012.pdf> (accessed 15 Dec 2013).
- 2 Elsevier. *Clinical key: diabetic ketoacidosis*. <https://www.clinicalkey.com/topics/endocrinology/diabetic-ketoacidosis.html> (accessed 16 Dec 2013).
- 3 Umpierrez GE, Smiley D, Kitabchi AE. Narrative review: ketosis prone type 2 diabetes mellitus. *Ann Intern Med* 2006;144:350–7.
- 4 Umpierrez GE. Ketosis-prone type 2 diabetes: time to revise the classification of diabetes. *Diabetes Care* 2006;29:2755–7.
- 5 Umpierrez GE, Casals MM, Gebhart SP, et al. Diabetic ketoacidosis in obese African-Americans. *Diabetes* 1995;44:790–5.
- 6 Foster JR, Morrison G, Fraser DD. Diabetic ketoacidosis-associated stroke in children and youth. *Stroke Res Treat* 2011;2011:219706.
- 7 Carr ME. Diabetes mellitus. A hypercoagulable state. *J Diabetes Complications* 2001;15:44–54.
- 8 Dixon AN, Jude EB, Banerjee AK, et al. Simultaneous pulmonary and cerebral oedema and multiple CNS infarctions as complications of diabetic ketoacidosis: a case report. *Diabet Med* 2006;23:571–3.
- 9 Chaudhary M, Kamalaksha S, Trainer VJ, et al. Acute cerebellar infarction in a young patient presenting with diabetic ketoacidosis. *Pract Diabetes* 2012;29:377–9a.
- 10 Okamura M, Suzuki K, Takekawa H, et al. Cerebral infarction associated with diabetic ketoacidosis in an elderly patient. *Neurol Clinl Neurosci* 2013;1:44.

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