Reversible blindness secondary to severe diabetic ketoacidosis

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Abstract

We present a case of sudden-onset reversible blindness secondary to severe diabetic ketoacidosis. The exact mechanism is not fully understood but is postulated to be due to uncoupling of retinal electrical transmission in a severe acidic environment. This report adds to the small number of reported cases of reversible blindness secondary to severe diabetic ketoacidosis and serves to raise awareness of this unusual diagnosis. It should be considered in patients who present with acute-onset reversible bilateral blindness only after other causes are excluded via detailed retinal examination and cranial imaging.

Keywords: diabetic ketoacidosis, reversible blindness

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Case presentation

A 45-year-old female with type 1 diabetes mellitus, on a basal bolus insulin regimen, presented to accident and emergency with a 2-day history of sudden-onset bilateral blindness. She had been suffering from diarrhoea and vomiting, and had missed numerous doses of her insulin over the preceding 2 weeks. She smoked five cigarettes per day but did not drink any alcohol.

On examination blood pressure 112/76 mmHg, heart rate 57 bpm, SpO_2 100% on air and temperature 36.0 °C. The cardiovascular, respiratory and abdominal examinations were normal. There were roving eyes movements and a complete loss of perception to light bilaterally. Pupils were dilated pharmacologically prior to consultant review making pupillary response assessment unreliable. Urgent retinal examination performed by a consultant ophthalmologist was completely normal. The remaining neurological examination was normal.

Laboratory investigations confirmed a severe diabetic ketoacidosis (DKA): plasma glucose 34.9 mmol/l; pH 6.8; serum bicarbonate 3 mmol/l; and capillary ketones 4.9 mmol/l (Table 1). An emergency brain CT was normal.

She was commenced on treatment following the Scottish DKA Care Pathway comprising intravenous insulin, potassium and crystalloid fluids. Over the course of 3 days her acidosis and eyesight gradually and concurrently returned to normal.

A brain MRI performed 3 days later demonstrated a small T2-weighted hyperintensity in the right parietal lobe in keeping with small vessel disease. There was no corresponding diffusion restriction to suggest this was an acute ischaemic lesion.

Visual acuity measured by Snellen chart prior to discharge was 6/7.5 in the right eye and 6/4.5 in the left eye.

Discussion

Acute-onset blindness in DKA has well-recognised causes, including bilateral posterior circulation stroke, posterior reversible encephalopathy syndrome (PRES), nonarteritic anterior ischaemic optic neuropathy (NAAION) and posterior ischaemic optic neuropathy (PION).[1–4]

These alternative causes of blindness were excluded in our patient with cranial imaging and detailed retinal examination. A normal brain CT excluded PRES and a follow-up brain MRI excluded acute cerebral ischaemia. The combination of normal optic nerves on fundoscopy with full rapid recovery of vision excluded NAAION and PION.

Reversible blindness in severe DKA without the above aetiologies is rare. Searching PubMed using the string [reversible AND blindness AND acidosis] and examining references, yields only four reported cases of reversible blindness in patients with severe DKA.[5–8] There have been a small number of other reports of reversible blindness with alcoholic ketoacidosis and also lactic acidosis.[9–11] The common factor in all of these cases, along with our case, was a pH <7.0 and the full recovery of vision with normalisation of pH.

Electrical transmission of information from the photosensitive rods and cones in the retina is mediated by horizontal cells. Experiments in mammalian retinae have found this

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Laboratory test	Day 1	Day 2	Day 3
pH	6.8	7.0	–
Bicarbonate (mmol/l)	3	14	21
Glucose (mmol/l)	34	9.5	10

Table 1 Biochemistry results

transmission to be pH dependent, with uncoupling of transmission occurring at or below pH 7.0.[12]

Conclusion

Sudden-onset bilateral reversible blindness is a rare presentation of severe acidosis and is related to altered, pH-dependant, retinal electrical transmission. It should be considered in patients with acute-onset bilateral blindness, but should not be diagnosed without full ophthalmic examination and cranial imaging to exclude other aetiologies.

Informed consent

Written informed consent for the paper to be published (including images, case history and data) was obtained from the patient for publication of this paper, including accompanying images.

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