Unusual presentation of more common disease/injury

Visual hallucination of coloured numbers secondary to hyperglycaemia

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Summary

A 70-year-old Indian woman presented with confusion and visual hallucinations of brightly coloured numbers slowly roving across the right visual field. These hallucinations occurred for a few minutes every few hours. Examination revealed a right homonymous hemianopia. Blood sugar was 37 mmol/l. EEG identified left occipital seizure activity. There was clinical and electrophysiological resolution with normalisation of the hyperglycaemia. There are few cases of hyperglycaemia associated with positive visual phenomena and hemianopia in the literature and this is the first case reported presenting with numerical hallucinations. Hyperglycaemia must be kept on a differential diagnosis list of unusual visual phenomena as it is easily correctable.

BACKGROUND

This case is important as it shows how initially complex seeming neurological symptoms can be secondary to conditions as common as hyperglycaemia, and that simple correction of the underlying biochemical derangement can resolve the clinical problem. When presented with a complex clinical presentation, one must always be vigilant to basic medical management. Further, this case is important as, to our knowledge, it is the first reported case of the condition presenting this way and offers an opportunity to discuss an interesting clinical phenomenon.

CASE PRESENTATION

A 70-year-old Indian woman presented with confusion and visual hallucinations of brightly coloured numbers slowly roving across the right visual field. These hallucinations occurred for a few minutes every few hours.

Her background medical history included ischaemic heart disease and type 2 diabetes. She had recently

returned from a holiday in India. She was noted to be confused during the last week and was treated with antibiotics for presumed urine infection. On the last day of her holiday her confusion worsened and her family, thinking this was secondary to low blood sugar, gave her a large number of sweets. On the return flight her confusion worsened to the point that she was unable to recognise her relatives at the airport. She developed a left sided headache and visual hallucinations and was admitted to hospital.

INVESTIGATIONS

On examination, she had a right homonymous hemianopia. The rest of her physical examination was normal. Blood tests were all normal apart from a serum glucose level of 37 mmol/l. Results of lumbar puncture, including viral PCR, were negative and imaging with MRI was normal.

Fluid resuscitation and insulin were started. On day 2 of her admission she developed episodes of twitching

Figure 1 EEG recording during an episode of visual hallucination. Build up is seen most clearly in P3-O1.

Table 1 Previous cases of hyperglycaemia presenting with homonymous hemianopia and visual hallucinations

Source	Age/sex	Glucose (mmol/l)	Symptoms	Imaging	EEG	Clinical course	Management
Taban <i>et al</i> ¹²	68/M	33.31	Left homonymous hemianopia, episodes of photopsia	MRI: few white matter hyperintensities only, SPECT normal	–	Hallucinations resolved within 1 week, hemianopia within 2 weeks	Correction of glucose (no details given)
Lavin PJ ¹³	39/M	27.92	Left homonymous hemianopia, episodes of photopsias, impaired colour vision, headache. Light stimulation precipitated photopsia, bilateral meiosis, left beating nystagmus and upper limb tremor	MRI: right occipital lobe T2 hypointensity, repeat MRI five months later was normal	Decreased voltage and slowing in right occipital lobe, no ictal discharge (12 h after treatment)	Hemianopia resolved over 16 days	Insulin and fluids
Wang <i>et al</i> ¹⁴	59/F	29.7	Right homonymous hemianopia, episodes of flickering red objects and complex visual phenomena such as flowing water, animals and people with distorted faces	CT: normal, MRI (FLAIR) subcortical hypointensity and gyral hyperintensity in the left occipital lobe. MRS showed decreased N-acetyl aspartate in the left occipital lobe	Spikes in left occipital region during visual hallucinations	Seizures subsided over 11 days	Insulin and fluids. Carbamazepine started on day 10 and continued for 1 month
Freedman <i>et al</i> ¹⁵	72/F	20.26	Headache, dense left homonymous hemianopia, photopsia, fragmented and rolling vision	MRI: normal	–	Visual symptoms resolved over several weeks	Correction of glucose by stopping steroids
Brazis <i>et al</i> ¹⁶	75/M	38.95	Complete left homonymous hemianopia (and right superior field defect), coloured circular whirling objects in both hemifields	CT: normal	Diffuse slowing without epileptiform activity	Resolution of visual phenomena 2 days after presentation, complete resolution of field defect in a week	Insulin and fluids
Harden <i>et al</i> ¹¹	67/F	25.1	Bifrontal headache, left homonymous hemianopia, photopsia	CT: several old lacunar infarcts only	Irregular 10 Hz waves in right occipital and parietal regions	Normal glucose after 5 days, visual hallucinations resolved over next 3 days, homonymous hemianopia several days later	Insulin and phenytoin (but subtherapeutic levels), Phenytoin continued for 3 months. No further seizures
Johnson <i>et al</i> ¹⁷	28/M	20.6 (initially in DKA)	Episodic photopsia (red and green flashing lights) in the left visual field and headache. Left homonymous hemianopia. Eye deviation to left with nystagmus	CT: normal (×2)	9–10 Hz waves ictally	Seizures terminated at 10 days, homonymous hemianopia a few days later	Insulin and phenytoin but persistence of visual phenomena, therefore phenobarbitone used to terminate seizures
	57/M	33.8	Right homonymous hemianopia, photopsias (sparkling red and gold lights), independent episodes of palinopsia with allesthesia followed by left parietal headache and secondary generalisation on one occasion. Few generalised tonic-clonic seizures with left eye deviation	CT: normal	Partial seizure of left occipital lobe	Hallucinations resolved with control of sugar and hemianopia resolved 2 days later	Insulin and fluids
Duncan <i>et al</i> ¹⁸	43/F	27.7	Headache, left homonymous hemianopia, photopsia (flashing lights), then unfamiliar furniture and human faces. Seizures provoked by left lateral gaze	CT and MRI: normal	EEG: right tempo-occipital rhythmic discharges during episodes	Resolution of all visual symptoms after 48 h	Insulin and fluids
Maccario ¹	73/M	41.63	Right homonymous hemianopia, complex visual hallucinations (dead relatives), right hemiparesis	–	Diffuse and continuous delta activity	Resolution of all symptoms over several days	Insulin and fluids

DKA, diabetic ketoacidosis; F, female; FLAIR, fluid-attenuated inversion recovery; M, male; MRS, magnetic resonance spectroscopy; SPECT, single photon emission CT.

of the right eyelid and eye deviation to the right which occurred in conjunction with her visual hallucinations and an EEG was performed. This showed a build up of sharp wave activity occurring over the posterior part of the left hemisphere, spreading to the right hemisphere a few minutes before her eyes deviated to the right and she became confused (figure 1).

TREATMENT

The patient was treated with carbamazepine and clobazam and her glucose level was gently lowered over the next few days.

OUTCOME AND FOLLOW-UP

Her visual hallucinations and confusion resolved within 2 days. Her homonymous hemianopia resolved over the next week. She has been completely well in the following year.

DISCUSSION

An association between seizures and hyperglycaemia has been described since 1968.¹ Overall, 25% of patients with non-ketotic hyperglycaemia (NKH) have been reported to have seizures, which were the presenting symptom in 6% of cases.^{2 3}

The underlying mechanism is not clear. In rats, hyperglycaemia has been shown to lower seizure threshold and experimentally induced hyperosmolality has been shown to precipitate seizures in animals with focal cortical lesions.^{4 5} It is postulated that patients with diabetes may be particularly susceptible to the cellular insults of hyperosmolality due to underlying vascular lesions.³ However, many of the clinical cases described had only mild hyperosmolality, and hyperosmolality may occur in NKH without the precipitation of seizures, and so the correlation is not straightforward. NKH results in inhibition of Krebs's cycle with decreased cerebral glucose levels, and γ -aminobutyric acid (GABA) metabolism is increased as an alternative energy source. Loss of this inhibitory neurotransmitter may lower seizure threshold.⁶

Seizures are much rarer in diabetic ketoacidosis (DKA) than NKH even at higher glycaemic levels.⁷ In an experimental model of ketoacidosis, brain GABA levels were not shown to be diminished,⁸ partially because the majority of the brain's energy requirements in DKA are met by the metabolism of acetoacetate.⁷ Further, intracellular acidosis has been shown to increase glutamic acid decarboxylase activity leading to increased levels of GABA,⁹ and increased levels of this inhibitory neurotransmitter may offer neuroprotection.

Grant and Warlow showed that the majority of seizures in hyperglycaemia are partial motor seizures,¹⁰ with or without secondary generalisation. Harden *et al* reported that in a review of 80 patients with NKH and seizures, 69 (86.2%) had partial motor seizures, none (11.3%) had generalised seizures and two (2.5%) had visual seizures.¹¹

The visual phenomena reported most commonly are photopsias, but well formed complex visual hallucinations of moving objects, transpositions and perseverance are also reported. Negative symptoms, such as hemianopias as reported here, also occur (table 1).

Ten other cases of hemianopia with positive visual hallucinations secondary to hyperglycaemia have been reported. Their key features and management are summarised in table 1. Ours is the only reported case of numerical hallucinations. In all subjects the symptoms resolved with resolution of the metabolic imbalance and the patients experienced no long-term deficits.

With hyperglycaemic seizures, neuroimaging is usually normal, but a range of abnormalities including transient cortical, and occasionally subcortical, hyperintensities and hypointensities on MRI T2 and fluid-attenuated inversion recovery, and restricted diffusion on diffusion weighted imaging, have been reported.¹⁴ These imaging findings are non-specific, so imaging at the moment is primarily to exclude other pathology.

In all cases described in the literature, the resolution of symptoms lagged behind the achievement of glycaemic control by approximately 24 h to several days, and hallucinations improved before the hemianopia.

The role of anticonvulsants is not clear. Seizures self-terminate in many cases and patients remain seizure free with normal glucose levels. Further, seizures recur with loss of glycaemic control, despite the use of anticonvulsants. Phenytoin was used in two of the cases reported by Harden *et al*,¹¹ and in one case the seizure frequency increased despite the improving metabolic abnormalities. (Phenytoin has been shown to inhibit insulin secretion and increase insulin resistance, and may worsen glycaemic control and therefore increase seizure frequency.¹⁹) On the 10th day they added phenobarbitone which terminated the seizures. It was several more days before the homonymous hemianopia resolved and it is therefore difficult to be certain as to the natural history had antiepileptic medication not been used. In their case, as in ours, the seizures were distressing and were an indication for the use of antiepileptic medication, but the optimum duration of use is unclear. Harden *et al* weaned their patient off antiepileptic medication at 4 months, and with good glycaemic control there were no further seizures.

Learning points

- ▶ Hyperglycaemia can cause an array of visual phenomena secondary to occipital lobe seizure activity.
- ▶ These hallucinations can be well formed and complex.
- ▶ Hyperglycaemia should be considered in all patients presenting with visual hallucinations.
- ▶ Simple correction of electrolyte imbalances can correct the clinical abnormalities.

Competing interests None.

Patient consent Obtained.

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