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Hyperglycaemia as a Stroke Mimic: A Case Report of Homonymous Hemianopia Due to Hyperglycaemia

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Abstract

Focal neurological symptoms, in the context of non-ketotic hyperosomolar hyperglycaemic state (HHS), are a well-documented phenomenon. It is less clear whether focal neurological symptoms can be a manifestation of hyperglycaemia without evidence of ketosis or hyperosmolality. This report describes a case of a 58 year old Caucasian male who presented with a macula-sparing left homonymous hemianopia in the context of hyperglycaemia and a new diagnosis of type 2 diabetes mellitus without any radiological evidence of stroke on Computer Tomography (CT) or Magnetic Resonance Imaging (MRI) of the brain. While chronic hyperglycaemia, in the context of diabetes mellitus, itself is a risk factor for stroke, the aim of this presentation is to increase awareness that hyperglycaemia may present with focal neurological deficits mimicking stroke.

Background

Stroke is one of the most common causes of mortality and morbidity worldwide [1]. The time-critical nature of initiating reperfusion therapy, such as thrombolysis and endovascular clot retrieval, relies on the fast and accurate diagnosis of stroke [1]. The management of stroke is based on an accurate clinical diagnosis supported by imaging, either CT or MRI [1].

Diabetes mellitus is a risk factor for ischaemic stroke [1]. It is a well-documented phenomenon that hyperglycaemia, in the context of hyperosmolar hyperglycaemic state (HHS) can cause focal neurological symptoms which can mimic stroke [2]. Hemiparesis, hemiballismus, choreoathetosis, hemianopia, seizure and coma are commonly recognised presentations of hyperglycaemia [2]. In a recent systematic review of cases of focal neurological deficits mimicking stroke associated with HHS,

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excluding those that are seizure phenomenon, it was found that hemianopia was the most common clinical presentation [2]. The next most common presentation was a combination of symptoms that are consistent with a partial or total anterior circulation syndrome, with the most common symptom being speech disturbance [2].

This report presents a case of a macular-sparing homonymous hemianopia, in the context of hyperglycaemia without HHS, with no abnormalities on EEG or MRI of the brain. The aim is to increase awareness of hyperglycaemia being a possible cause for stroke mimics.

Case presentation

A 58 year old, Caucasian male was referred, by an optometrist, to the Emergency Department (ED) after experiencing five days of visual disturbances. He described seeing "coloured blotches" in the left inferior quadrant of his visual fields. Over this time period, he also noted that he was increasingly bumping into walls and objects on his left more. This was associated with a headache, which fluctuated in intensity. The headache was described as a diffuse pressure-like sensation. There had been no preceding head injury prior to the new headache or visual changes. He had also noticed about 15kg of unintentional weight loss over the previous few months plus polydipsia and polyuria over the previous two weeks. He was a regular smoker, with occasional alcohol use, and was not on any regular medications. On examination, he was hypertensive with a blood pressure of 156/102mmhg. Bed side blood glucose level was 22.4 mmol/L and the bedside ketone level was 1.7 mmol/L. On neurological examination, the visual acuity in the right eye was 6/12 and 6/6 in the left eye. He had a left homonymous heminanopia on confrontation. There were no other neurological deficits noted. In particular, there was no other parietal lobe dysfunction detected, no finger agnosia, no dysgraphia, no left-right disorientation, no alexia, no sensory neglect, no astereognosis. On further review by the ophthalmologist, there were no abnormalities noted in the eyelids, conjunctiva, cornea, anterior chamber, iris, lens or anterior vitreous. On fundoscopy, the vitreous was clear, there was no optic disc swelling, the macula appeared healthy and there were no abnormalities noted with the blood vessels.

Investigations

The haemoglobin A1c was 16.5% with a random serum glucose of 23.0mmol/L on admission. The calculated osmolarity was 295 mmol/L. The total cholesterol was elevated at 6.1 mmol/L, predominantly with elevated LDL cholesterol level of 3.6 mmol/L and non-HDL cholesterol of 5.2mmol/L. The triglycerides were also elevated at 3.6 mmol/L. The HDL cholesterol was 0.88 mmol/L. An atypical optic neuropathy screen was performed and the antinuclear antibodies, extractable nuclear antigen antibody, antineutrophil cytoplasmic antibodies, neuromyelitis optica antibodies, myelin oligodendrocyte glycoprotein antibodies, syphilis antibodies EIA screen and tuberculosis Gamma Interferon Assay were all negative.

Initially, on presentation to the ED, a CT brain, pre and post-contrast, was completed and showed no acute intracranial haemorrhage nor extra-axial collection. There was no enhancing lesion and no evidence of recent territorial infarction. CT angiogram of the head and neck showed normal opacification and no aneurysms of the vertebral arteries, the basilar artery, and all the major vessels of the circle of Willis. The initial MRI brain, performed three days after admission, showed no acute infarct nor focal parenchymal abnormality. The MRI brain was repeated two days later, with gadolinium contrast, and no signs of demyelination were noted.

Electroencephalogram (EEG) showed a symmetrical and reactive 9-10Hz alpha posterior rhythm, with no seizure or epileptiform activity nor focal features recorded. There were normal visual evoked potentials, following left and right eye stimulation.

Optical coherence tomography showed no abnormalities in the macula or optic disc in either eye. A left homonymous hemianopia which was macula sparing was found on Humphrey Visual Field testing.

Differential diagnoses

On initial presentation, given the findings of a left homonymous hemianopia, with the risk factors of untreated diabetes mellitus, current smoker and obesity, the provisional diagnosis was a right occipital stroke. The MRI brain showed no evidence of acute or chronic infarction in the occipital lobe or anywhere else along the optic tract and further investigations were organised to explore other differential disorders. The patient reported photopsias and an EEG was performed and excluded occipital seizures, as a cause for the left homonymous hemianopia. The patient has been reviewed extensively by ophthalmology and an optic neuropathy screen was performed which found no serological evidence for systemic lupus erythematosus, ANCA-associated vasculitis, neuromyelitis optic-spectrum disorder, tuberculosis infection or syphilis infection. Repeated ophthalmological examination and optic coherence tomography have shown no abnormalities in the macula or optic disc of either eye, to account for the visual field loss. It was concluded that the homonymous hemianopia was a phenomenon of a highly localised neurological sign caused by hyperglycaemia, secondary to previously undiagnosed diabetes mellitus.

Treatment

He was initially commenced on aspirin 100mg daily and clopidogrel 75mg daily for stroke prevention. Clopidogrel was ceased given there were no findings for stroke, on repeated MRI brain imaging. He was commenced on atorvastatin for hypercholesterolaemia. For the management of diabetes mellitus, he was commenced on metformin, sitaglipin, Novorapid and Optisulin.

Outcome and follow up

On review at two and three months after the initial presentation, the left homonymous hemianopia has resolved based on clinical examination. Progress MRI brain and orbits with gadolinium contrast at two months after the initial presentation showed no brain parenchymal signal abnormality; and the appearance of the globes, optic nerve sheaths and chiasms remained within normal limits.

Discussion

This case is similar to previous case reports in terms of the clinical presentation, in addition to the homonymous hemianopia, he also experienced visual hallucinations and headache, both of which are common features with previously reported case reports [3]. While the patient suffered from hyperglycaemia, in a similar range to the other case reports, he did not meet the criteria for HHS. While most cases of homonymous heminanopia, secondary to HHS, have previously shown an occipital

lesion on MRI, with the most common finding of decreased T2 signal of the white matter, then restricted diffusion, gyral hyperintensity and gyral swelling [2,3], the present case did not have any occipital lesions on MRI, even on repeated scans. He also did not have any changes on EEG, while many previously reported cases did have abnormalities noted, the most common being epileptiform discharges, followed by slowing activity without epileptiform discharges [3]. Consistent with similar previous case reports, this patient's visual fields recovered after treatment for hyperglycaemia was initiated. In terms of the clinical features, the present case is consistent with previous case reports of homonymous hemianopia. Unlike previously reported cases, this case showed no abnormalities on either imaging or EEG. This may be because this case presented with severe hyperglycaemia, but, was not yet in a hyperosmolar state, like many of the previously reported case studies. This suggests that severe hyperglycaemia itself can cause focal neurological signs without hyperosmolarity, and that in cases of severe hyperglycaemia with hyperosmolarity, physiological disturbances can become detectable on MRI and EEG.

The pathophysiology underlying how a non-ketotic hyperglycaemic state causes localising neurological symptoms, such as homonymous hemianopia, remains under debate [3]. Most cases of homonymous hemianopia from hyperglycaemia are associated with visual hallucinations, ranging from flashes of coloured spots to complex images [3]. This has led to the hypothesis that the phenomenon is based on seizure activity in the occipital lobe, causing the positive visual symptoms, and the homonymous hemianopia representing a post-ictal phenomenon, analogous to a Todd's paresis [3]. Evidence supporting this hypothesis includes the common finding of epileptiform discharges in the occipital region of patients presenting with homonymous hemianopia and hyperglycaemia [3]. There have also been a small number of case reports looking at FDG-PET scanning in these cases which show hypermetabolism in the occipital regions, again supporting the possibility that the neurological deficit is from an ictal phenomenon, as opposed to an ischaemic cause [4]. A possible explanation for the underlying pathophysiology, of how hyperglycaemia induces seizures, involves the depression of activity in the Kreb's cycle which affects the equilibrium in the metabolism of γ-aminobutyric acid (GABA), resulting in reduced GABA and consequently decreased seizure thresholds [3, 4].

The other main competing hypothesis, on how non-ketotic hyperglycaemia causes homonymous hemianopia and other localising neurological symptoms, relates to focal ischaemia, secondary to hyperglycaemia. Supporting the hypothesis that focal ischaemia drives the hemianopia is the common imaging finding, on MRI, of increased signal on diffusion weighted imaging (DWI) and decreased signal on T2 weighted imaging [3,5]. Increased signal on DWI can be seen in cytotoxic oedema secondary to ischaemic and osmotic dysfunction, and seizures. However, early cortical ischaemia is associated with T2 hypointensity, whereas, focal seizures are usually associated with T2 hyperintensity [5]. Sympathetic dysregulation is an additional mechanism by which vascular dysfunction could contribute to the phenomenon of homonymous hemianopia. This is based on the hypothesis that there is less sympathetic innervation to the posterior circulation and that this drives the posterior predominance of conditions, such as reversible posterior leukoencephalopathy syndrome and the homonymous hemianopia seen in hyperglycaemia [5]. Similarly, supporting this hypothesis, in the study by Rossi et al. of case reports of focal neurological symptoms secondary to hyperglycaemia, the most common neurological symptom was homonymous hemianopia accounting for almost three quarters of the cases, then partial or total anterior circulation stroke syndrome.

There is little evidence to guide the use of antiseizure medications as most patients with hemianopia from hyperglycaemia have resolved symptoms after the management of the hyperglycaemia, with insulin and rehydration [3].

Learning points

While diabetes mellitus is traditionally considered as a risk factor for stroke, it is also important to keep in mind that severe hyperglycaemia can also be a stroke mimic and can present with focal neurological symptoms. A prompt evaluation of the blood glucose level, at the time of presentation, is an important piece of information, in terms of aiding the clinical diagnosis of stroke and differentiating from stroke mimics.

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