



Diabetes as the Cause of a Stroke Mimic

Lucinda Tran¹; Roy G Beran^{1,2,3,4,5,6,7*}

¹Department of Neurology and Neurophysiology, Liverpool Hospital, Liverpool, South Western Sydney clinical School, University of New South Wales, Sydney, NSW, Australia.

²Neurology Department, Liverpool Hospital, Liverpool 2170, NSW, Australia.

³Conjoint Professor, South Western Sydney Clinical School, University of NSW, Sydney, NSW, Australia.

⁴Ingham Institute for Medical Research, South Western Sydney Area Health Service, NSW, Australia.

⁵Professor, School of medicine, Griffith University, Southport, Qld, Australia.

⁶Professor, Chair, Sechenov Moscow First State University, Moscow, Russia.

⁷Conjoint Professor, School of Medicine, Western Sydney University Sydney, NSW, Australia.

***Corresponding Author(s): Roy G Beran**

Ingham Institute for Medical Research, South Western
Sydney Area Health Service, NSW, Australia.

Email: roy.beran@unsw.edu.au

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Editorial

The case of a 58 year old, Caucasian man, presenting with homonymous hemianopia, in conjunction with previously undiagnosed diabetes, who attend the Emergency Department with an elevated haemoglobin A1c of 16.5% and a random serum glucose of 23.0 mmol/L, was published in your journal [1]. This case was identified as the first report of a 'stroke mimic', presenting with hemianopia in conjunction with diabetes, confirmed following full investigation, including repeated Magnetic Resonance Imaging (MRI) of his brain, investigation for vasculitis, exclusion of occipital seizure or migraine and detailed ophthalmological evaluation, without there being any evidence of a focal lesion being demonstrated[1]. The patient's symptoms of hemianopia resolved with appropriate management of his

diabetes. As was stated in the previous report, "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" [1].

Within the previous report, there was a concerted effort to try to explain how the hyperglycaemia evoked the hemianopia, in the absence of any discernible focal pathology being identified, despite in depth investigation [1]. Proffered explanations included: possible hyperglycaemia induces seizures, causing depression of activity in the Krebs's cycle, resulting in reduced γ -aminobutyric acid (GABA) and lowered seizure threshold [1-2] but evaluation for seizure, including electroencephalography, including sleep deprivation provocation, was negative; the potential for focal ischaemia, secondary to the hypergly-



caemia, resulting in the hemianopia but there was an absence of the typical MRI findings [1,-3]; and possible sympathetic dysregulation, based on the hypothesis that there is less sympathetic innervation to the posterior circulation driving such posterior circulatory conditions, typified by reversible posterior leukoencephalopathy syndrome [4]. Despite the attempt to explain the phenomenon, none of these hypotheses were convincing and there remained the conundrum as to why uncontrolled diabetes, in the absence of any other contributing factors, could evoke the homonymous hemianopia in the absence of any definable lesion on cerebral imaging.

The patient was subsequently seen, in the neurology outpatient clinic, 2 months after his discharge from hospital, at which time the only acceptable explanation, for his new onset diabetes, thought to produce a 'stroke mimic', was that of a metabolic disturbance, associated with the significant hyperglycaemia, consequent to the diabetes. Since discharge from hospital, he underwent a further MRI which confirmed an absence of any identifiable lesion, an echocardiogram which was unremarkable and he was reviewed by the ophthalmologists who confirmed normal and full visual fields. He was significantly overweight, at 109 kgs, for a height of 168 cms, placing him in the order of 40 kgs more than his ideal body weight for height, representing morbid obesity, with a calculated body mass index (BMI) of 38.6 which raised concern regarding the potential for obstructive sleep apnoea (OSA). In the absence of a sleeping partner, he could neither confirm, nor deny, whether he was, or was not, a snorer, accepting that he did acknowledge some increased fatigue. Physical examination, including fundoscopy revealed beautiful venous pulsations, mitigating against raised intracranial pressure. Clinically his visual field were full, to confrontation, with no evidence of hemianopia. Visual acuity, as tested with Snellen chart, was 6/12-1 in the left eye and 6/9-3 or 6/12 in the right eye, without errors. Remainder of cranial and peripheral neurological examination was normal. Cardiac, carotid, orbital and respiratory auscultation were normal. Blood pressure was 130/90 mmHg, taken in the right arm in sitting position, with pulse rate of 90 b/m.

To further ensure that there was not a subclinical lesion in the optic pathways, potentially too subtle to be identified on MRI, that might account for his transient hemianopia, he underwent visual evoked response testing that was completely normal, following both left and right eye stimulation. On the basis of his morbid obesity, he was referred for an all-night, diagnostic Polysomnograph (PSG) which confirmed the presence of very serious OSA. The PSG revealed an Apnoea Hypopnoea Index (AHI) of 113 (normal being ≤ 5) with oxygen desaturation from 94% to 80% which indicated a need for urgent intervention. He has since been referred for a PSG monitored, Continuous Positive Air Pressure (CPAP) titration study in which he showed a short sleep latency with the sleep architecture severely fragmented and a low sleep efficiency of 52%. He had difficulty transitioning to consolidated sleep with an absence of slow wave sleep and no Rapid Eye Movement (REM) sleep being observed during the study. Even with CPAP titration, he continued to have respiratory evoked arousals (~12/hour) and spontaneous event (~23/hour). He had recurrent central apnoea events at all pressures tested, from 6 to 18 cmH₂O, and, in the absence of REM sleep, during the study, it was impossible to adequately titrate CPAP to compensate for the previously observed events that occurred in REM sleep. He is still to undergo an adequate trial of CPAP, to be complemented with the addition of acetazolamide.

The purpose of reporting this further development, in this case of a 'stroke mimic' in a patient with previously undiagnosed diabetes, is that the patient has been demonstrated to have a serious concomitant contributing factor which may have sufficiently exacerbated the impact of the diabetes to evoke the hemianopia. The fact that the hemianopia resolved, with appropriate management of the diabetes, with both oral hypoglycaemic agents plus insulin, despite the ongoing evidence of severe OSA, indicates that the OSA was not the primary cause of the hemianopia but it may well have been the contributing factor which compounded the effects of the diabetes, on already ischemic brain tissue, caused by previously unsuspected OSA. Reverting to the various hypotheses, proffered in the earlier description of this case, the imposition of severe cerebral hypoxia, associated with the OSA in conjunction with the significant diabetes, would lend further credence to the hypothesis advocating sympathetic dysregulation in the vulnerable posterior cerebral circulation [4].

This case highlights a number of issues that deserve consideration. In the absence of a viable hypothesis, when confronted with a case that remains an enigma, it is imperative to widen the net to continue to explore all possible avenues. Accepting that it was the diabetes which was the primary cause of the identified 'stroke mimic', in the absence of any alternative lesion being found, despite detailed and thorough investigation, clinicians should consider the presence of diabetes when confronted with a 'stroke mimic'. Diabetes, alone, may be insufficient to be accepted as an isolated cause for a 'stroke mimic' and, as demonstrated in this case, it amplifies the need for ongoing scepticism when faced with such a scenario and the need to continue the search for confounding variables that may compound the impact of the diabetes. This explanation does not detract from the previous publication [1] which remains the first report of diabetes presenting as a potential 'stroke mimic' in which hemianopia is expected to be accompanied by a lesion within the visual cortex but no such lesion was identified.

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