Simultaneous bilateral cerebral infarct: a case report

Bilateral stroke is an extremely rare event. In a study of 1,838 patients, bilateral anterior infarction accounted for only 0.27% of stroke patients. We report the case of an 84-year-old woman who presented with bifrontal haemorrhagically transformed infarcts in the anterior cerebral arteries.

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A 84-year-old white woman presented to the emergency department with a Glasgow coma score of 5/15 (E1, V1, M3) after collapsing at home. Other vital observations were stable; capillary blood glucose was 6.4 mmol/l. Her husband revealed that she had been sitting watching television when her left leg made a sudden jerking movement. She then made an incomprehensible sound, became unresponsive, and was incontinent of urine. No myoclonic movement or tongue-biting was witnessed.

Other than essential hypertension, which was well controlled with nifedipine, she had been fit and well. Cardiovascular, respiratory, and abdominal examinations were unremarkable. There was no obvious facial droop. Her pupils were equal and reactive to light with no deviation of gaze, and fundoscopy revealed no papilloedema. Hypertonia and hyper-reflexia were seen in all four limbs, along with decorticate posturing. The patient also had bilateral positive Babinski reflexes. All her blood results were normal apart from a raised cholesterol of 5.8 mmol/l. She had a normal chest radiograph but electrocardiogram showed sinus rhythm with first degree heart block. An urgent CT of the head was done. Small basal ganglia lacunar infarcts were noted, which were thought to represent non-specific post-ischaemic changes (figure 1).

The patient was managed conservatively with a nasogastric tube inserted for feeding. Once-daily aspirin 75 mg and simvastatin 10 mg were given via nasogastric tube. We then did a repeat head CT 3 days later, which showed evidence of large space-occupying lesions seen bifrontally with moderate perifocal oedema, and compression of the frontal horns of both lateral ventricles (figure 2).

Brain MRI and MRA scans were then done to further characterise these lesions (figure 3). They showed bilateral infarcts with haemorrhagic transformation (ie, bleeding from primary cerebral pathology) around the anterior cerebral arteries, both of which showed multi-segmental narrowing. We consulted a neurosurgeon, but surgery was not indicated. We decided to give the patient dexamethasone 8 mg twice daily because of the extensive oedema around the infarct. Throughout treatment, the patient was unrousable, except for a brief few hours 3 days after initial presentation, when she opened her eyes but remained unresponsive. Unfortunately, the patient did not improve, and she died 16 days after presentation with severe cerebrovascular event from aspiration pneumonia. A post-mortem examination was not done.

Case discussion

Bilateral stroke is extremely rare. Bilateral internal carotid artery occlusion was first described by Fisher1 in 1954, and few cases have been reported since. In a study of 1,838 patients with stroke at the Department of Neurology, Asan Medical Centre, Seoul, South Korea, bilateral anterior circulation infarction accounted for only 0.27% of patients.2 All patients who had bilateral anterior circulation infarcts presented invariably with sudden coma, quadriplegia, and decerebrate or decorticate rigidity. Causes were mainly artherosclerosis, cardiogenic embolism from atrial fibrillation, or both. Additionally, because of poor Glasgow coma scores on admission and large-volume infarcts, all cases were associated with high mortality—all patients studied died within 3 days of presentation.

Management of our case was extremely difficult. The patient was given aspirin, since it has been proven to be beneficial in the management of cerebrovascular disease. In the past 30 years, the anti-platelet effect of aspirin has revolutionised the management
of cerebrovascular disease. A combined meta-analysis of 40,000 acute stroke patients from two large randomised trials revealed significant reduction in recurrent ischaemic strokes and deaths in hospital with aspirin. We continued to treat our patient with aspirin, even after the diagnosis of haemorrhagically transformed infarct, to prevent recurrent ischaemic events. In the meta-analysis mentioned above, 800 patients (2%) were inadvertently randomised after a primary haemorrhagic stroke. No evidence that aspirin had an adverse effect on haemorrhagic stroke was recorded; rather, fewer patients on aspirin had further stroke or death (63) than did control patients (67), although this difference was not significant.

Treating our patient with dexamethasone, conversely, was a somewhat more controversial management decision. Although steroids have proven benefits in reduction of cerebral oedema caused by brain tumours, their value in stroke remains debatable. Numerous animal studies have reported benefit of steroids in cerebral infarction, but the few small clinical trials conducted have shown otherwise. Despite these results, 20% of physicians in the USA and in China report routine use of steroids for stroke. The lack of evidence for such treatments suggests a need for further clinical trials.

This case had poor prognosis from the start. The patient was elderly, had a low Glasgow coma score at admission, and had a large-volume infarct. Sadly, she died 16 days after presentation without ever regaining consciousness. It is impossible to comment on whether steroid treatment made a difference, although we believed that this treatment was worth trying. Given the poor prognosis, moreover, would any other management strategy have made a difference? Nevertheless, this case is worth reporting. It illustrates an extremely rare event, and allows us to reflect on the limitations of existing strategies to combat such a poor prognosis.

We have no conflict of interest.

Permission to publish this case report was obtained from the patient’s next of kin.

References