Early neurological deterioration after ischaemic stroke due to cardiac arrhythmia and intracranial stenosis

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An 83-year-old lady presented with right hemiparesis and aphasia with unknown time of onset. Admission National Institutes of Health Stroke Scale (NIHSS) was 7. Her electrocardiogram showed first-degree heart block and left bundle branch block. Carotid Doppler and computed tomography (CT) brain were unremarkable. She was diagnosed with a left partial anterior circulation stroke and started on aspirin and atorvastatin.

She had an episode of acute worsening of aphasia and hemiparesis the next day with NIHSS rising to 11, lasting less than an hour. Repeat CT brain was normal and she was recommenced on telemetry. A similar episode recurred the following day during which telemetry demonstrated Mobitz II block (Figure 1A) followed by six seconds of ventricular standstill (Figure 1B). Metoprolol was discontinued. Magnetic resonance angiography of the brain demonstrated areas of restricted diffusion within the left middle cerebral artery territory (Figure 2) and critical stenosis/occlusion of the left intracranial carotid artery (Figure 3). Permanent pacemaker was inserted and she made an uneventful recovery.

Early neurological deterioration after an ischaemic stroke may be due to non-haemodynamic (eg, sepsis, metabolic disorders, haemorrhagic transformation, etc) and haemodynamic factors (eg, cardiac arrhythmias, arterial re-occlusion, etc). Though extracranial carotid stenosis is a more common cause of stroke in our population, intracranial stenosis should be considered if carotid Doppler imaging is unremarkable and no alternative cause of stroke is discovered, particularly in patients with recurrent events or neurological symptoms in association with haemodynamic stressors. In patients with a symptomatic intracranial stenosis of 70–99%, treatment includes intensive vascular risk factor modification and dual antiplatelet therapy for 90 days. In this case, the patient's neurological deterioration was likely due to transient cerebral hypoperfusion during the arrhythmia, aggravated by intracranial carotid stenosis.
**Figure 1:** Telemetry strip (A) during an episode of acute neurological deterioration showing Mobitz type II. (B) Followed by six-seconds of ventricular standstill that resolved spontaneously.

**Figure 2:** Magnetic resonance imaging (MRI) showing high signal on B1000 (left image) and low ADC signal (right image) representing restricted diffusion typical of an acute infarct in the left thalamic and internal capsule area.
CLINICAL CORRESPONDENCE

Figure 3: Magnetic resonance angiography (MRA) demonstrating critical stenosis of the left intracranial carotid artery.

Competing interests:
Nil.

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