Acute stroke presenting as syncope: Wallenberg syndrome

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DESCRIPTION

A man in his 50s presented to the emergency department with two episodes of syncope when standing up. They were not preceded by prodromal symptoms, chest pain, dyspnoea or palpitations. On arrival, the patient mentioned vertigo and instability when standing. Previous medical history was relevant for obesity, hypertension and liver transplant 3 years prior due to alcoholic cirrhosis. Physical examination showed multidirectional, non-suppressible, nystagmus, right-eye ptosis and myosis, loss of pain and temperature over the left side of the body (sparing face) and right-sided cerebellar hemiataxia. Romberg sign was absent due to severe unsteadiness when standing. Vital signs were normal and there was no difference between right-arm and left-arm pulse or blood pressure. Orthostatic vitals were not recorded. Blood glucose on admission was normal, as was a 12-lead ECG. CT of the head and neck with angiogram showed a hypoplastic left vertebral artery. Cranial MRI showed a T2 and fluid-attenuated inversion recovery hyperintense lesion on the inferior lateral right bulbar region (figures 1 and 2), with restricted diffusion pattern (figure 3). Blood tests, including Rapid Plasma Reagin (RPR) and haemoglobin A1c levels, 24-hour Holter and transthoracic echocardiogram were normal. Doppler ultrasound of the cervical vessels was negative for subclavian steal syndrome or arterial dissection. Acute stroke from occlusion of the right posterior inferior cerebellar artery (PICA) was diagnosed. Clinical presentation was compatible with Wallenberg syndrome (WS). The patient showed almost complete resolution of his neurologic symptoms during admission.

Syncope is a form of transient and self-limited loss-of-consciousness caused by abrupt drop in cerebral blood flow, more specifically to the brainstem's reticular activating system. Major causes of syncope are reflex-mediated, such as vasovagal or carotid sinus hypersensitivity, orthostatic hypotension and cardiogenic[1] . Although it results from brain hypoperfusion, stroke is only rarely the true cause of syncope, as the brain has a very redundant blood supply. One notable exception is the subclavian steal syndrome, which results in vertebrobasilar ischaemia during arm exercise due to subclavian artery stenosis. Stroke should be considered a cause of syncope when persistent and significant neurological changes occur before the episode or during the recovery period.

WS, also known as lateral medullary syndrome, is the most prevalent posterior ischaemic stroke syndrome. It commonly results from occlusion of the PICA or one of its branches. It is characterised by crossed thermoalgic hypoesthesia, ipsilateral cerebellar ataxia, vertigo, bulbar palsy and Horner's syndrome. Clinical presentation varies, often resulting in any combination of these symptoms. A wide range of unusual presentations of WS,

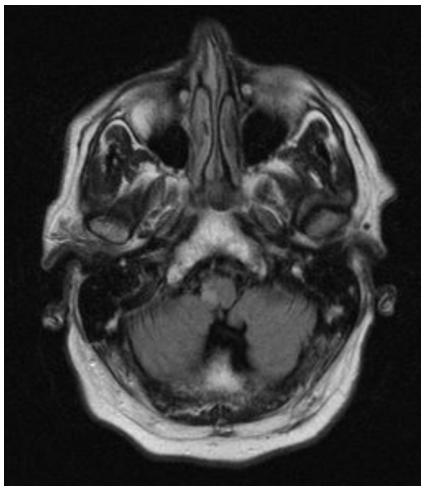

Figure 2 Fluid-attenuated inversion recovery (FLAIR)-hyperintense lesion on the inferior lateral right medulla oblongata.

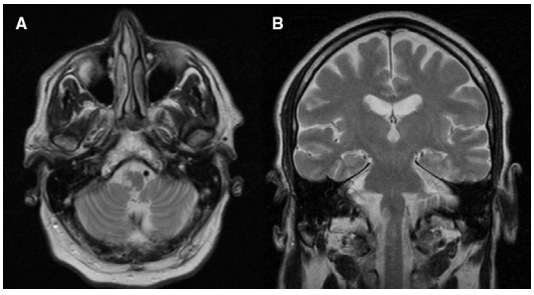

Figure 1 T2-hyperintense lesion on the inferior lateral right medulla oblongata, axial (A) and coronal (B) views.

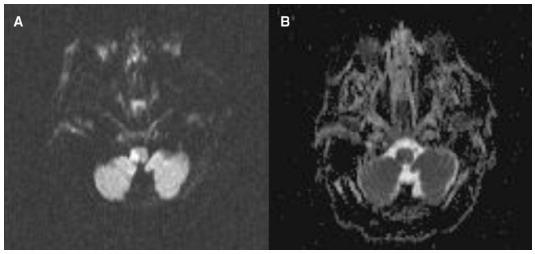

Figure 3 Diffusion-weighed imaging with restricted diffusion (A) and low apparent diffusion coefficient (ADC) values (B) on the right lateral medulla oblongata.

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Table 1 Case reports of lateral medullary infarction with associated autonomic dysfunction

Case report	Manifestation of autonomic dysfunction
Huynh *et al*[4]	Pre-syncope and orthostatic hypotension
Altavilla *et al*[5]	Orthostatic hypotension and trigeminal-autonomic cephalalgia
Pascual *et al*[6]	Retro-ocular headache with autonomic features resembling "continuous" cluster headache
Lee *et al*[7]	Sick sinus syndrome
Takawaza *et al*[8]	Sinus arrest and post-hiccup *cough syncope*
Alsaad *et al*[9]	Sinus arrest and *syncope*, requiring permanent pacemaker placement

from trigeminal neuralgia to hiccups, has been reported in the literature.[2 3] Autonomic dysfunction has been described in case reports, although rarely (table 1). Huynh *et al* reported a case of lateral medullary infarction presenting with presyncope in which autonomic testing showed impaired sympathetic and parasympathetic cardiovascular reflexes.[4] Altavilla *et al* described a case of dorsolateral medullary infarction associated with orthostatic hypotension and trigeminal autonomic cephalalgia.[5] Pascual and colleagues described another case presenting with cluster-like headache.[6] In two separate case reports, sinus node dysfunction was diagnosed, with prolonged sinus pauses recorded on electrocardiography monitor.[7 8] In another report, a patient presented with sinus arrest, requiring permanent pacemaker placement.[9] To the best of our knowledge, however, only two cases of syncope associated with lateral medullary infarction have been reported, by Takawaza *et al* and Alsaad *et al*..[8 9] These manifestations seem to be secondary to the involvement of sympathetic fibres and the *nucleus tractus solitarii* in the medulla, affecting the baroreceptor regulatory centre. In fact, Hong's study of autonomic function in patients with lateral medullary infarction suggested susceptibility to cardiac parasympathetic dysfunction, especially in the ventral medulla.[10]

Our patient's syncope was compatible with orthostatic hypotension, which can be seen in autonomic failure. Although cardiac dysfunction following brainstem stroke is rare, this case highlights the importance of recognising syncope as a possible presenting symptom of medullary infarction and WS.

Learning points

- Stroke is a rare cause of syncope.
- Persistent neurological deficits should prompt consideration of stroke as a cause of syncope.
- Syncope can be the presenting feature of Wallenberg syndrome, or lateral medullary infarction, secondary to autonomic dysfunction.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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