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LETTER TO THE EDITORS

Cerebellar arteriovenous malformation presenting with recurrent positional vertigo

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Dear Sirs,

Posterior fossa arteriovenous malformations (AVMs) are rare neurovascular lesions which are incidentally diagnosed or present with headaches, seizures or neurological deficits due to intracranial haemorrhage or mass effect [1–3]. Vertigo is an uncommon primary manifestation. We describe a patient who presented with episodic positional vertigo and nystagmus attributable to a cerebellar AVM.

A 43-year-old female presented with a 15-year history of brief episodes of spinning vertigo brought on by turning over in bed. She had intermittent right-sided tinnitus and throbbing headaches unassociated with the vertigo. On initial interictal assessment, she had no spontaneous, gaze-evoked or head-shaking nystagmus, and a negative head impulse test. Very low amplitude (< 2°/s) persistent geotropic torsional nystagmus was seen on both Dix–Hallpike tests. A differential diagnosis of benign positional vertigo (BPV) or vestibular migraine was considered. Audiovestibular testing and imaging were planned, however, the patient did not attend her investigations.

Five months later, the patient returned with a recurrence of positional vertigo and was assessed on the Epley Omniax rotator (Vesticon, Portland, Oregon, USA). She had no spontaneous nystagmus but persistent downbeat nystagmus lasting more than 2 min in both Dix–Hallpike positions (Online Resource 1, Fig. 1a). Nystagmus onset was rapid (Right: 0 s, Left: 1.5 s); with the right ear down, the slow phase velocity (SPV) rose to 33°/s after 12 s and was enhanced with leftward gaze to 55°/s at 38 s; with the left ear down, a SPV of 97°/s was reached at 9 s. On either side, nystagmus decayed to 10°/s at 90 s but did not cease. Resuming the upright position did not reverse nystagmus direction. Magnetic Resonance Imaging and CT angiography revealed a right superior cerebellar AVM measuring 2.2 × 1.8 × 2.8 cm (Fig. 1b, c). After surgical excision of the AVM, no further positional vertigo was reported.

Positional nystagmus of central origin may be persistent or paroxysmal, often has no latency, its direction may be atypical for the stimulated canal plane, it may not fatigue with repeat positioning and is refractory to repositioning manoeuvres [4]. In contrast, benign positional nystagmus (BPN) is most often paroxysmal with a crescendo-decrescendo velocity profile and its axis is orthogonal to the stimulated canal plane [5]. Reversal of the provocative position often results in reversal of nystagmus direction. BPN is fatigable and responsive to appropriate repositioning.

Positional downbeat nystagmus may be observed in anterior canal BPV and in central pathologies affecting the cerebellum and dorsolateral medulla [6–8]. Lesions of the nodulus and uvula are thought to cause positional nystagmus by disturbing the transduction and processing of gravitational signals from the otolith-organs [9]. Anterior canal BPV can exhibit downbeat nystagmus without a clear torsional component on both Dix–Hallpike tests, due to the proximity of the anterior canals to the sagittal plane [5]. The nystagmus usually has an immediate or brief onset similar to that of our patient, but a briefer duration of less than 1 min [10]. Persistent positional downbeat or torsional nystagmus is seen in vestibular migraine and tends to be of a low velocity (2–7°/s) [11].

The symptoms of AVMs have been attributed to mass effect and hemodynamic disturbance arising from arterial hypotension and venous hypertension [12]. Oedema, necrosis and gliosis may occur in surrounding tissues and

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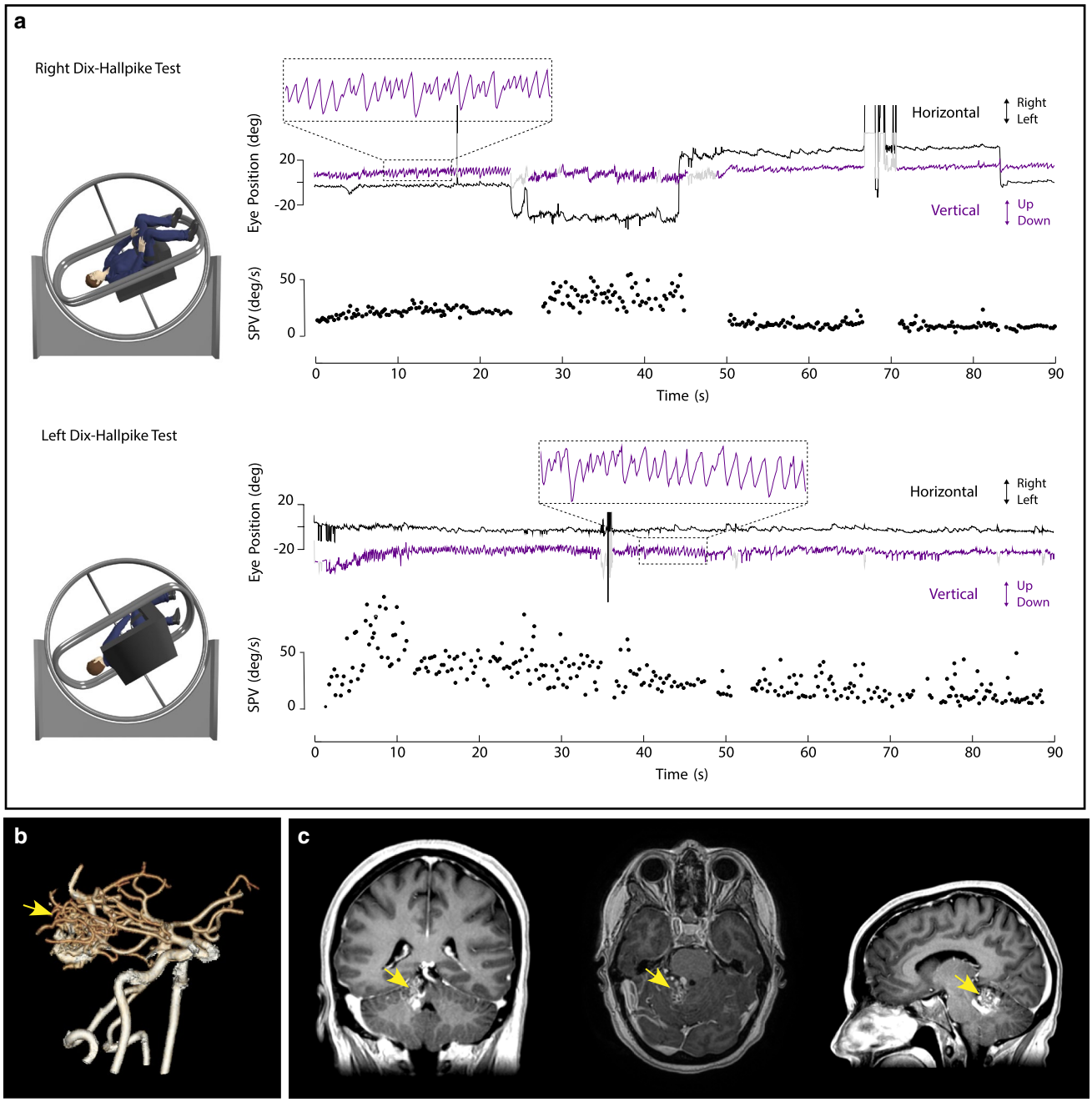

Fig. 1 **a** The upper right panel illustrates vertical and horizontal eye position and vertical slow phase velocity (SPV) as a function of time during Dix–Hallpike tests on the Epley Omniax rotator. Testing the patient in the right Dix–Hallpike position revealed persistent downbeat nystagmus with no latency. The peak SPV was 33°/s at 12 s with centre gaze and reached 55°/s with leftward gaze. In the left Dix–Hallpike position, downbeat nystagmus with minimal latency and a higher peak SPV (97°/s at 9 s) was seen. Following the initial peak, the nystagmus SPV slowly decayed in an exponential fashion. **b** CT angiogram reconstruction showing AVM within the right superior cerebellum (yellow arrow). **c** Coronal, axial and sagittal T1-weighted brain MRI with contrast showing cerebellar AVM (yellow arrows)

hydrocephalus is a frequent complication [2, 13]. These were not present in our patient. Fluctuating symptoms may be accounted for by non-infarctional ischaemia in areas adjacent to the AVM [12]. There are few reports documenting positional vertigo and nystagmus in patients with posterior fossa AVMs. Kikuchi et al. [14] reported a case of a cerebellar AVM and varix on the pons demonstrating downbeat nystagmus on anteflexion and retroflexion of the neck (attributed to altered vascular flow in the varix), with no symptoms of vertigo. More recently, Komiyama et al. [15] described episodic vertigo lasting hours and

direction-fixed horizontal nystagmus arising from an AVM in the cerebellar vermis.

This patient's history illustrates how an unruptured posterior fossa AVM can present with longstanding, seemingly innocuous symptoms compatible with BPV and completely reversible physical signs. While the presence of unilateral tinnitus and throbbing headaches merited imaging, it was her compelling central positional nystagmus observed during the ictus that eventually led to the diagnosis.

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Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflict of interest.

Ethical standards This case study has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Informed consent was obtained from the patient.

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