



## Case Report

### Bruns-Cushing nystagmus due to hypertensive unilateral paramedian pontine base infarction

#### Abstract

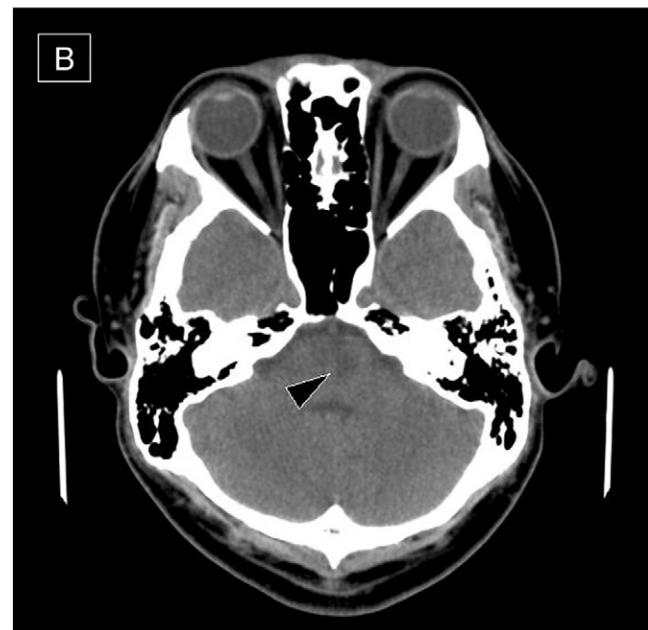
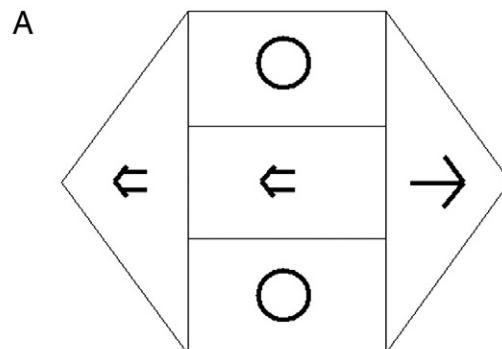
Bruns-Cushing nystagmus is unusually rare and is known to be related with cerebellopontine angle tumor. A 32-year-old male patient came to our emergency department 3 times because of dizziness, right upper limb ataxia, hypertension, and Bruns-Cushing nystagmus. Magnetic resonance imaging demonstrated left paramedian pontine infarction. In conclusion, Bruns-Cushing nystagmus not only indicates a cerebellopontine angle tumor but may also be associated with pontine infarction.

Bruns-Cushing nystagmus, also called *Bruns bidirectional nystagmus* or *Bruns nystagmus* [1,2], is unusual and is associated with a cerebellopontine angle tumor with brain stem distortion, flocculus compression, or vestibulocerebellum compression [1,3]. It occurs in 11% of vestibular schwannoma sufferers, in 67% of whom it is more than 3.5 cm in diameter [2]. It is composed of large nystagmus with low oscillating frequency (gaze paretic nystagmus) when looking toward the lesion side and small nystagmus with high oscillating frequency (vestibular nystagmus) when looking toward the other side.

A 32-year-old man had been healthy until that day, when he had experienced sudden dizziness combined with right upper limb ataxia. There was no headache, tinnitus, auditory disturbance, paresthesia, phonophobia, photophobia, or other neurologic focal signs. His blood pressure was 172/104 mm Hg, with a heart rate of 92/min. His muscle power was grade 3 (Medical Research Council Scale) in the right upper limb and grade 4 in the other 3 limbs. The deep tendon reflexes in all 4 limbs were symmetrically normal. Both sides of his body, including the limbs and trunk, were sensitive to pain on a pinprick test (10/10). There was Bruns-Cushing nystagmus, looking toward the right, with a frequency of 2/s and an amplitude of 0.5 mm by a stationary ruler (Fig. 1A, ←), leftward nystagmus with a frequency of 1/s and an amplitude of 1.0 mm, looking toward the left (Fig. 1A, →). Nifedipine given sublingually lowered the blood pressure to 125/73 mm Hg, with a heart rate of 75/min. He was relieved of his dizziness, but the right hemiparesis persisted. Computed

tomography demonstrated a hypodense lesion at left pontine base (Fig. 1B, ▶). The patient was admitted for further study of his condition.

Magnetic resonance imaging (Fig. 2A, ▶) demonstrated that the lesion had a hypointense signal in T1 fluid-attenuated inversion recovery (FLAIR), a hyperintense signal in T2

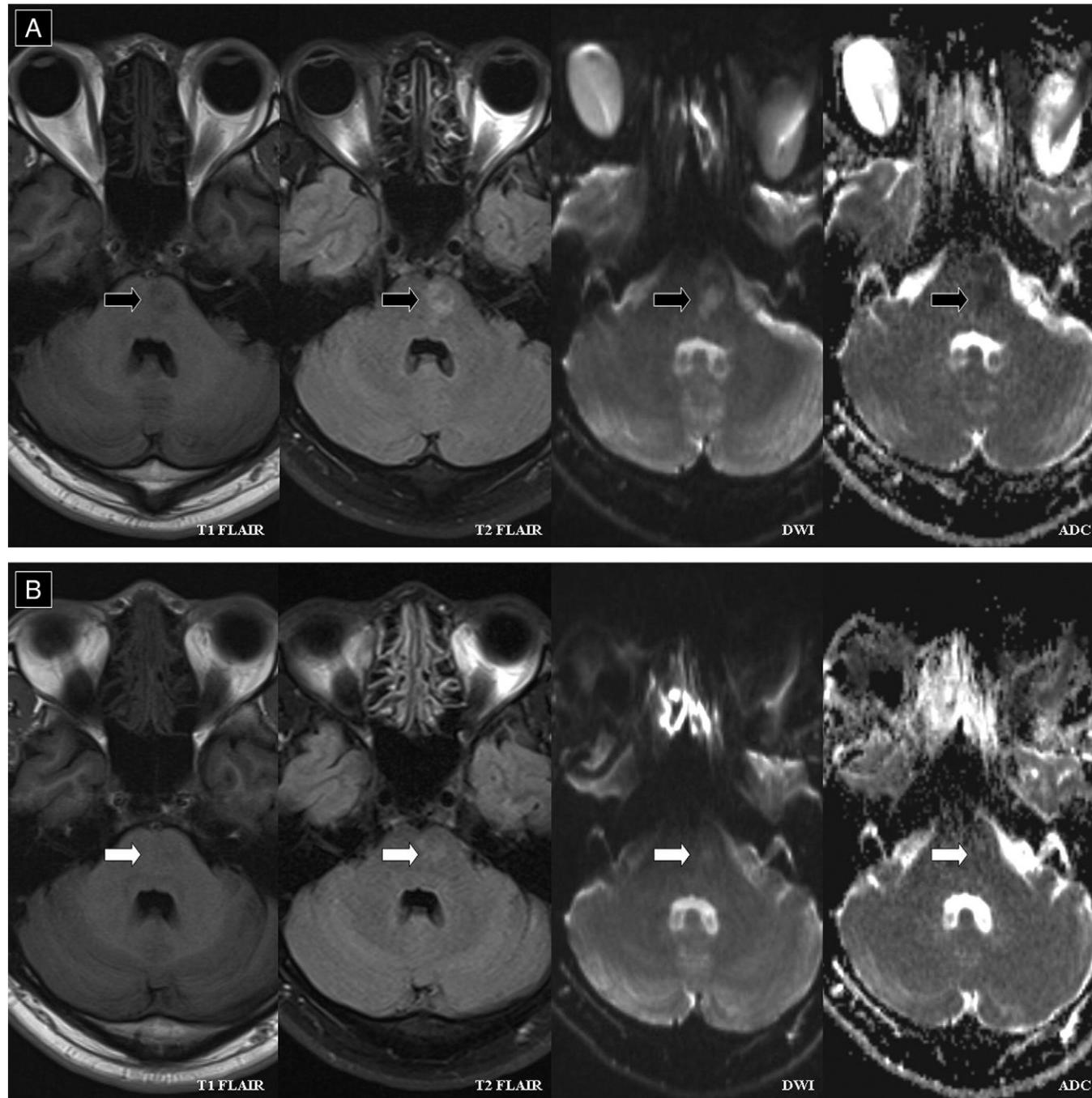


**Fig. 1** Symptomatic gaze nystagmus test (A) and brain computed tomography (B).

FLAIR, a hyperintense signal in diffusion weighted imaging (DWI), and a hypointense signal in an apparent diffusion coefficient map. Result of the 3-dimensional time-of-flight angiogram was unremarkable. Left paramedian pontine base infarction was confirmed. Conservative treatment was recommended, and his condition improved. Magnetic resonance imaging was repeated before discharge from hospital and demonstrated improvement in the lesion

(Fig. 2B,  $\Rightarrow$ ). Over the following 2 months, dizziness with right upper limb ataxia recurred twice and was associated with Bruns-Cushing nystagmus and hypertension.

Hypertensive patients suffered dizziness more frequently than those in a control group, possibly due to cochlear dysfunction related with hypertensive vascular disease [4] or to brain stem hypertensive encephalopathy related with hypertensive crisis [5,6]. Our patient's dizziness was



**Fig. 2** Symptomatic (A) and asymptomatic follow-up (B) magnetic resonance imaging.

accompanied by high blood pressure and left upper limb ataxia, indicating motor system impairment from hypertensive encephalopathy. Results of the pinprick test and deep tendon reflex test were normal. Both T2 FLAIR and DWI revealed a hyperintense lesion at the left paramedian pontine base, indicating an acute edematous change; and apparent diffusion coefficient map revealed it was hypointense, excluding a hemangioma or arteriovenous malformation (Fig. 2A). The result: left paramedian pontine base infarction was confirmed.

Pontine infarcts account for approximately 15% of acute vertebrobasilar ischemic strokes [7]. Unilateral pontine base ischemia seems to be usually caused by small arterial occlusion of paramedian or circumferential branches rather than large-artery atherothrombosis or cardiogenic embolism [8,9]. In our patient, sudden onset of hypertension was presumed locally to induce fibrinoid necrosis, fibrohyalinosis, lipohyalinosis, microatheroma, or other endothelial inflammatory changes of the left penetrating small arterial branches of basilar artery and their interconnections with the penetrating small arterial branches of the left anterior inferior cerebellar artery [10,11], resulting in left paramedian pontine base infarction.

The clinical presentations of pontine base infarction included pure motor hemiparesis, sensorimotor stroke, ataxic hemiparesis, and dysarthria—clumsy hand syndrome [9]. Our patient presented with dizziness and ensuring ipsilateral upper limb ataxia. The left paramedian pontine base infarction influenced the left abducent nuclei, the left paramedian pontine reticular formation, and the medial longitudinal fasciculus, leading to leftward gaze paretic nystagmus during a leftward gaze test [12,13]. This influenced the left proximal vestibular nerve and left superior vestibular nucleus and led to left vestibulopathy that presented with rightward gaze vestibular nystagmus during a rightward and forward gaze test (Fig. 1A).

The hyperintense signal at the left paramedian pontine base was reduced in the symptomatic follow-up T2 FLAIR and DWI (Fig. 2B), proving that the patient's symptoms were related to the lesion. Because the symptoms recurred 3 times and high blood pressure was found at each symptomatic time, the patient was diagnosed with hypertensive left paramedian pontine base infarction. There is no effective strategy for treatment other than the control of hypertension. Because our patient's high blood pressure occurred occasionally, a short-onset antihypertensive, such as sublingual nifedipine, was recommended to prevent further cerebrovascular complications. In conclusion, Bruns-Cushing nystagmus, although unusual, indicates not only a cerebellopontine angle tumor but also pontine infarction.

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## References

- Croxson GR, Moffat DA, Baguley D. Bruns bidirectional nystagmus in cerebellopontine angle tumours. *Clin Otolaryngol Allied Sci* 1988; 13(2):153-7.
- Lloyd SK, Baguley DM, Butler K, et al. Bruns' nystagmus in patients with vestibular schwannoma. *Otol Neurotol* 2009;30(5):625-8.
- Nedzelski JM. Cerebellopontine angle tumors: bilateral flocculus compression as cause of associated oculomotor abnormalities. *Laryngoscope* 1983;93(10):1251-60.
- Esparza CM, Jáuregui-Renaud K, Morelos CM, et al. Systemic high blood pressure and inner ear dysfunction: a preliminary study. *Clin Otolaryngol* 2007;32(3):173-8.
- Shintani S, Hino T, Ishihara S, et al. Reversible brainstem hypertensive encephalopathy (RBHE): clinicoradiologic dissociation. *Clin Neurol Neurosurg* 2008;110(10):1047-53.
- Uchino M, Haga D, Nomoto J, et al. Brainstem involvement in hypertensive encephalopathy: a report of two cases and literature review. *Eur Neurol* 2007;57(4):223-6.
- Bassetti C, Bogousslavsky J, Barth A, et al. Isolated infarcts of the pons. *Neurology* 1996;46(1):165-75.
- Caplan LR. Intracranial branch atheromatous disease: a neglected, understudied, and underused concept [published correction appears in *Neurology* 1990;40:725]. *Neurology* 1989;39(9):1246-50.
- Kim JS, Lee JH, Im JH, et al. Syndromes of pontine base infarction. A clinical-radiological correlation study. *Stroke* 1995;26(6):950-5.
- Ogata J, Yamanishi H, Ishibashi-Ueda H. Review: role of cerebral vessels in ischaemic injury of the brain. *Neuropathol Appl Neurobiol* 2011;37(1):40-55.
- Rosenblum WI. Fibrinoid necrosis of small brain arteries and arterioles and miliary aneurysms as causes of hypertensive hemorrhage: a critical reappraisal. *Acta Neuropathol* 2008;116(4):361-9.
- Tan E, Kansu T. Bilateral horizontal gaze palsy in multiple sclerosis. *J Clin Neuroophthalmol* 1990;10(2):124-6.
- Alemdar M, Kamaci S, Budak F. Unilateral midbrain infarction causing upward and downward gaze palsy. *J Neuroophthalmol* 2006;26(3):173-6.