Pontine infarction responsible for wall-eyed bilateral internuclear ophthalmoplegia syndrome

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Video

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A 68-year-old man with hypertension, cardiac arrhythmia, and a mechanical heart valve replacement treated with vitamin K antagonists was admitted for the acute onset of diplopia. Neuro-ophthalmologic examination showed a bilateral exotropia in primary gaze position (wall-eyed) with bilateral adduction deficit that did not improve by convergence (figure, video at Neurology.org/cp). In lateral gaze, a horizontal nystagmus of the abducting eyes appeared. Vertical saccades were normal (video). Pupils were symmetric and reactive to light. Brain MRI with diffusion-weighted images showed an acute pontine ischemic lesion (figure). Echocardiography revealed a thrombus in left auricle and anticoagulants were optimized. Neuro-ophthalmologic symptoms progressively improved over 3 months (video).

These clinical findings suggest the diagnosis of wall-eyed bilateral internuclear ophthalmoplegia (WEBINO) syndrome.¹ WEBINO is a rare manifestation due to pontine or midbrain lesions leading to a particular and severe form of bilateral INO (bINO).¹ Anatomically, bINO results from the disruption of both medial longitudinal fasciculi (MLF). Bilateral exotropia with impaired convergence, in midbrain forms of WEBINO, may be explained by the involvement of medial rectus subnuclei. In the pontine forms of WEBINO, the association of a paramedian pontine reticular formation lesion and contralateral vestibular imbalance resulting from MLF lesions have been postulated, but the underlying pathophysiology remains unclear and disputed.^{1,2}

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Figure Neuro-ophthalmologic examination

(A) Ocular motility. (B) Brain MRI: diffusion-weighted images (left panel) and apparent diffusion coefficient images (right panel) reveal a focal ischemic stroke located in the dorsal pons (arrows).

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