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Rapid Progression of Bilateral Vestibulopathy Due to Metastatic Lung Cancer

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Dear Editor,

The vestibulo-ocular reflex (VOR) ensures that the visual image on the fovea remains steady during head motion, while the vestibulospinal reflex maintains posture during locomotion. These functions indicate that patients with bilateral vestibulopathy (BV) experience unsteadiness and oscillopsia during walking. BV may arise from exposure to ototoxic agents, or in association with autoimmune, genetic, or degenerative diseases.^{1,2} Here we describe a patient who presented with gait unsteadiness as the first manifestation of a hidden lung cancer, and showed rapid progression of unilateral or asymmetric vestibulopathy into a bilateral vestibular failure.

A 75-year-old man suffered from progressive dizziness and unsteadiness during locomotion for about 4 months. The patient showed leftward head tilt and left skew deviation, spontaneous nystagmus beating rightward and clockwise during visual fixation that decreased slightly in darkness, and horizontal direction-changing gaze-evoked nystagmus (GEN). Smooth pursuit was impaired in both horizontal directions, but his saccades were normal. The visually enhanced VOR was impaired bilaterally. The fundus showed leftward ocular torsion, and the subjective visual vertical (SVV) was also tilted to the left. He tended to fall leftward and backward when attempting to stand, and had mild ataxia of bilateral upper extremities. Bedside head impulse tests (HITs) revealed corrected catch-up saccades when stimulating the left horizontal canal (HC). Video HITs also revealed a decreased VOR gain and corrective saccades for left horizontal and both posterior canals (Fig. 1A). However, caloric irrigation elicited bilaterally reduced responses (Fig. 1B). He additionally developed leftside facial palsy 2 weeks later. Bedside HITs were followed-up throughout his admission, which detected catch-up saccades in both directions 1 week later. Follow-up video HITs also revealed decreased gains and corrective saccades for all six semicircular canals (Fig. 1C). A rotatory chair test showed reduced gains of the VOR. Brain MRIs with gadolinium enhancement disclosed nodular enhancements within both internal auditory canals (IACs) along with an increased signal of the cerebellar vermis (Fig. 1D). Whole-body positron-emission tomography revealed a hypermetabolic mass in the hilum of the right lung. A biopsy of the pulmonary lesion confirmed the presence of small-cell lung cancer. The patient refused chemotherapy. His gait unsteadiness worsened to a bedridden status over the following 2 months.

Most IAC lesions are benign tumors, with vestibular schwannoma accounting for 90% of them.³ Metastases to the IAC are rare, constituting 0.3–0.7% of cerebellopontine-angle tumors.⁴ Although uncommon, metastatic carcinoma of the IAC should be considered in the differential diagnosis of cochleovestibular dysfunction due to its potentially devastating outcomes. Differentiating IAC metastases from benign schwannoma is often difficult due to the similarity of their MRI features and manifestations.^{3,4} Severe pain in the mastoid areas has been considered characteristic of IAC metastasis in a few studies, but other studies (including the present one) failed to find any mastoid pain. Instead, rapid progression of the vestibular

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Fig. 1. A: The VOR gain values are decreased for left horizontal (HC, 0.1, normal range ≥ 0.8) and both posterior canals (PCs, 0.3 for the right PC, and 0.1 for the left PC, normal range ≥ 0.7), but normal for right horizontal and both anterior canals (ACs, 1.0 for the right HC, 0.9 for the right AC, and 0.8 for the left AC, normal range ≥ 0.7). Overt saccades are found for these canals with decreased VOR gains, while covert saccades are observed during stimulation of the right PC and left AC. B: Caloric tests show bilaterally reduced responses with the summated SPVs of the induced nystagmus at 14°/s (8°/ s in the right ear, 6°/s in the left ear). C: Video head impulse tests one week later document reduced VOR gains and overt catch-cup saccades for all six semicircular canals (0.2 for the right HC, 0.1 for the left HC, 0.6 for the right AC, 0.4 for the left AC, 0.1 for the right PC, 0.0 for the left PC). D: Brain MRIs reveals nodular gadolinium enhancements within both internal auditory canals (arrows) in addition to increased signals in the cerebellar vermis (arrowheads). AC: anterior canal, HC: horizontal canal, PC: posterior canal, SPV: slow phase velocity, VOR: vestibulo-ocular reflex.

deficits may point to a malignant neoplasm.⁵ Our patient initially showed left-side vestibular hypofunction, as evidenced by spontaneous right-beating nystagmus, ocular tilt reaction, and SVV tilting to the left, and reduced gain of the left horizontal canal. HITs documented symmetric impairments of the VOR in patients with BV due to gentamicin toxicity,⁶ and followup testing documented that vestibular function remained unchanged in those patients.⁷ The asymmetric vestibular dysfunction during the initial evaluation and the rapid deterioration into bilateral failure in our patient can be differentiated from BV due to ototoxicity. Since perioperative, vascular, iatrogenic, traumatic, autoimmune, infective, toxic, and idiopathic etiologies can also progress rapidly,² it is necessary to combine all the clinical, laboratory, and radiological features in order to obtain an accurate diagnosis of BV.

IAC metastasis occurs bilaterally in about half of cases,⁴ and is strongly suggestive of additional metastasis into the brain parenchyma and meninges.⁵ Tumor cells can easily migrate to the adjacent IAC via the CSF within the surrounding cistern due to the contiguity of the meninges.⁵ Differences in the burden of deposited cells between the divisions of the vestibular nerve may have produced the dissociated VOR impairments for the vertical canals during the initial HITs in our patient. The GEN observed in our patient indicates dysfunction of the

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neural integrator and spreading of the tumor cells into the adjacent brainstem. Meanwhile, the lesion involving the cerebellar dorsal vermis appears to have been responsible for bilaterally impaired pursuit.

Our patient presented gait unsteadiness as the first manifestation of a hidden lung cancer, and serial HITs detected rapid progression of mostly left-side vestibulopathy into a bilateral vestibular failure due to metastasis involving both IACs. Metastatic carcinoma of the IACs therefore needs to be considered in BV, particularly when patients show a rapid deterioration or the signs of central vestibulopathy.

Conflicts of Interest

The authors have no financial conflicts of interest.

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