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LETTER TO THE EDITORS

Cerebellar tuberculous granuloma mimicking benign paroxysmal positional vertigo: progression after initial misdiagnosis

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Dear Sirs,

Positional vertigo and nystagmus may occur in association with central as well as peripheral vestibular lesions. Differentiation of central from benign paroxysmal positional vertigo (BPPV) is important due to its potentially grave underlying pathology in central positional nystagmus (CPN) [1, 2]. Herein, we report a patient with apogeotropic CPN due to tuberculous granuloma involving the cerebellar nodulus who had been misdiagnosed as having BPPV initially even with brain MRIs twice and showed a progression into severe imbalance over the following years. This report emphasizes the importance of scrutinized evaluation of the brain using gadolinium-enhanced MRIs in patients with a suspicion of CPN.

A 72-year-old man had worsening vertigo and unsteadiness for about 2 years. Initially, the patient was diagnosed as apogeotropic horizontal canal BPPV (HC-BPPV) based on the observed positional nystagmus. Since the positional nystagmus did not respond to repeated canalith-repositioning maneuvers (CRMs), he took brain MRIs without gadolinium-enhancement twice, both reported as normal (Fig. 1a).

At presentation to our dizziness center, the patient showed left beating spontaneous nystagmus only without visual fixation (Fig. 1d). During straight head hanging, he showed transient downbeat nystagmus along with the preexisting left beating nystagmus. He showed perverted head-shaking-nystagmus (pHSN). Apogeotropic nystagmus was induced during supine roll test, and the left beating nystagmus in the rightward head-turned position was greater than the right beating apogeotropic nystagmus (Fig. 1e). The apogeotropic nystagmus had no latency, peaked initially, and lasted more than one minute. Repeated CRMs for left apogeotropic HC-BPPV were in vain without fatigability of the nystagmus. He could not walk without a cane. Findings of other neurological examination were normal.

With a suspicion of CPN, we reviewed his previous MRIs and found a suspicious lesion involving the area of nodulus. Repeated brain MRIs with gadolinium-enhancement confirmed a ring-enhancing lesion involving the nodulus with an increased cerebral blood volume (Fig. 1b, c). Whole body and brain positron emission tomography (PET) revealed multiple hypermetabolic nodules in the right lung. Chest CT and bronchoscopy finally confirmed pulmonary tuberculosis. With a suspicion of tuberculous granuloma involving the lung and brain, the patient was placed on anti-tuberculous medications for 1 year. Follow-up MRIs documented a gradual resolution of the brain lesions. He also showed a marked improvement of gait ataxia, and became able to walk unaided.

Even with the initial suspicion of CPN based on refractoriness to repeated CRMs, improper protocols of repeated brain MRIs without a gadolinium-enhancement delayed the diagnosis in our patient.

Central positional nystagmus may be paroxysmal or persistent. Paroxysmal forms of CPN are ascribed to enhanced responses of the vestibular afferents during positioning due to lesions involving the nodulus and uvula [3]. In

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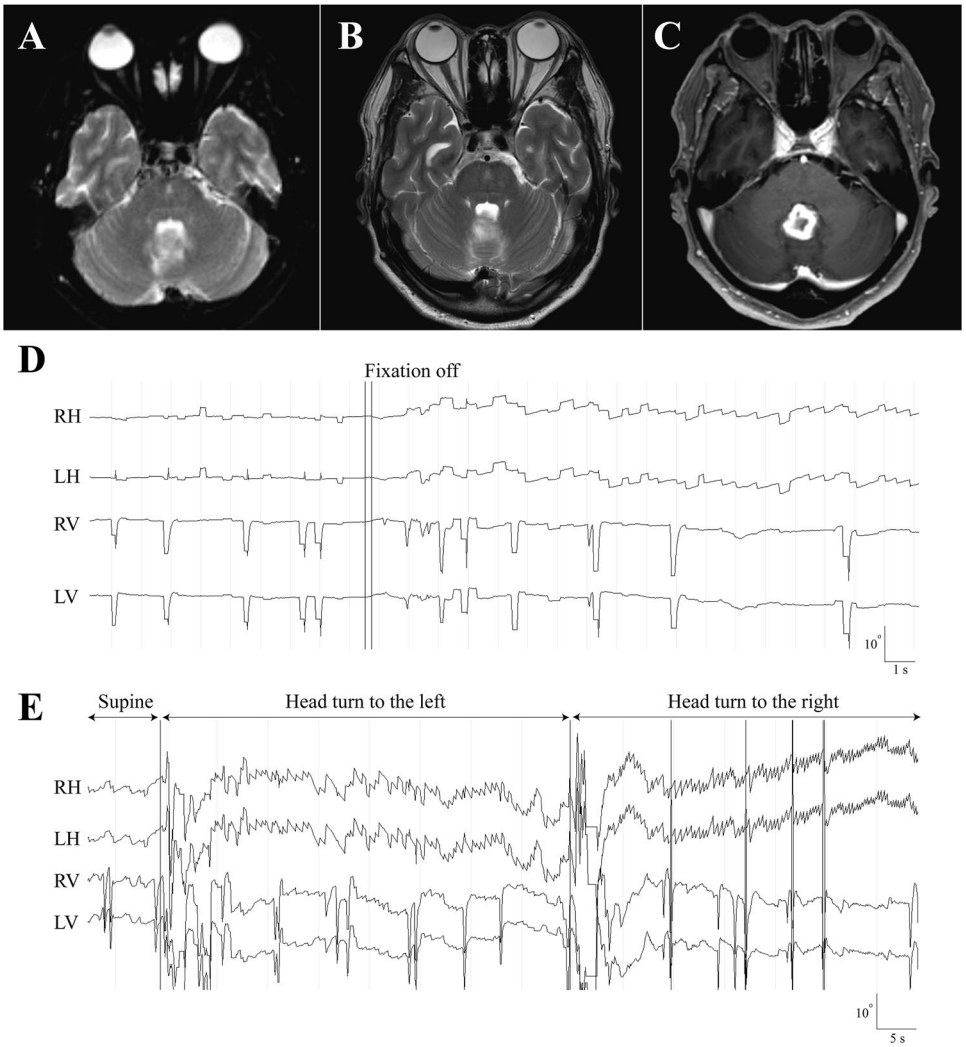

Fig. 1 **a** T2-weighted axial MRI 3 months before the presentation showed an increased signal intensity in the midline cerebellum. **b** Follow-up T2-weighted axial MRI confirmed an increase in the signal intensity in the area of cerebellar nodulus. **c** Gadolinium-enhanced T1-weighted MRI disclosed a ring-enhancing lesion in the corresponding area. **d** Video-oculography shows left beating spontaneous nystagmus only without visual fixation in darkness. **e** Video-oculography demonstrates apogeotropic nystagmus during head turning while supine, which is greater during rightward head turning left beating nystagmus. Upward deflection indicates rightward and upward eye motion in **d** and **e**

apogeotropic CPN, the lesions are also mostly overlapped in the vestibulocerebellum [4]. Due to the overlap of the lesion location, both paroxysmal and persistent type of CPN frequently coexist [3].

Nystagmus in apogeotropic HC-BPPV and apogeotropic CPN shares some common characteristics in addition to triggering positions and nystagmus direction. Both nystagmus develops without a latency and usually persist during head turning to either side while supine. Induced apogeotropic nystagmus is also stronger when it is in the direction of spontaneous nystagmus (ipsiversive) while supine in both cases. However, CPN shows the features distinct from those of HC-BPPV. First, the paroxysmal nystagmus commonly coexists with persistent nystagmus in CPN [3]. Second, the nystagmus gradually increases and then decreases in BPPV, but it usually peaks initially in CPN [3].

Besides apogeotropic CPN, our patient also showed pHSN and severe imbalance. Indeed, most (81.5%) patients with CPN show additional central signals [3]. The pHSN is frequently observed in lesions involving the vestibulocerebellum [5, 6]. Truncal ataxia without appendicular dysmetria usually indicates a midline cerebellar lesion [7].

Author contributions KK analyzed and interpreted the data and wrote the manuscript. HJK, J-YC, ZL, and XY analyzed and interpreted the data, and revised the manuscript. JSK designed and conceptualized the study, interpreted the data, and revised the manuscript.

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Compliance with ethical standards

Conflicts of interest Drs. K. Kim, H.J. Kim, Choi, Liqun, and Yang reports no disclosures relevant to the manuscript. Dr. J.S. Kim serves as an associate editor of Frontiers in Neuro-otology and on the editorial boards of Journal of Clinical Neurology, Frontiers in Neuro-oph-

thalmology, Journal of Neuro-ophthalmology, Journal of Vestibular Research, Journal of Neurology, and Medicine.

Ethical standard This study followed the tenets of the Declaration of Helsinki, and was performed according to the guidelines of Institutional Review Board of Seoul National University Bundang Hospital (B-1907–552-702).

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