



Isolated tonsilar infarction presenting with positional vertigo and nystagmus

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Dear Sirs,

The flocculus and paraflocculus (or tonsil) play a pivotal role in control of eye movements [1]. Based on animal experiment, it is suggested that the flocculus is more integral in governing vestibulo-ocular reflexes (VORs), whereas the tonsil mainly contributes to smooth pursuit (SP) [2, 3]. In humans, however, an isolated lesion of the tonsil is sparse [4, 5], and oculomotor findings of the tonsil lesion have been explored only in one patient [4]. We describe oculomotor abnormalities in a patient with positional vertigo as a result of unilateral infarction of the tonsil, and compare results with a previously reported patient [4].

A 71-year-old man with hypertension and diabetes mellitus developed positional vertigo. The vertigo was induced when his head was turned to either side in supine position. He also reported nausea and vomiting, but denied other neurological symptoms. Eye movement recording using video-oculography showed no spontaneous and gaze-evoked nystagmus. In supine position, rightward head turning induced right-beating horizontal nystagmus with no latency, lasting more than 1 min (Fig. 1a). Leftward head turning did not elicit nystagmus. Horizontal SP was abnormal in both directions, but strikingly more to the left side (Fig. 1b). Horizontal saccades and bithermal caloric tests were normal. Rotatory chair tests showed mildly decreased gain (0.37, normal range 0.5–0.7) during sinusoidal rotation at 0.16 Hz, but had normal gains at other frequencies (0.01, 0.04, and 0.64 Hz).

Video head impulse tests in all canal planes were normal. Testing of subjective visual vertical revealed no tilt. MRI of the brain exhibited an acute infarction, restricted to the left cerebellar tonsil (Fig. 1c). Due to the presence of atrial fibrillation on an electrocardiogram, the patient was treated with an oral anticoagulant. Positional vertigo resolved after several days, and follow-up evaluation 2 months later showed resolution of the positional nystagmus, and impaired horizontal SP.

Findings regarding our patient extend knowledge about oculomotor abnormalities of the human tonsilar lesion. Similar to previous report, our patient showed impaired horizontal SP, especially ipsilaterally [4]. VOR responses to low- and high-frequency stimuli were relatively preserved in both cases, while patients with isolated floccular infarction had decrease of VOR gain at high frequency [6, 7]. These findings support the current hypothesis that the flocculus is more concerned with vestibular response and the tonsil, more with the SP function [1–4]. The tonsil is known as a part of a descending SP pathway from posterior cortical areas and the dorsolateral pontine nuclei [1]. Responses of Purkinje cell activity also favor an integral role for the dorsal paraflocculus and lobulus petrosus (together presumably correspond to the human tonsil) in SP [8].

Intriguingly, our patient presented with positional vertigo and nystagmus induced by head turning in supine position. Positional vertigo and nystagmus are mostly observed in peripheral vestibular disorders such as benign paroxysmal positional vertigo (BPPV), but can be also encountered in central lesions [9–11]. Common presentations of central positional nystagmus (CPN) are apogeotropic nystagmus upon supine roll and downbeat nystagmus during straight head-hanging position in lesions involving the nodulus and uvula [9]. These structures are known as candidates for generation of CPN by impaired central processing of semicircular canal and otolith cues [9]. A mathematical model proposed that loss of information about the estimated gravity to the rotational feedback loop, may cause apogeotropic CPN

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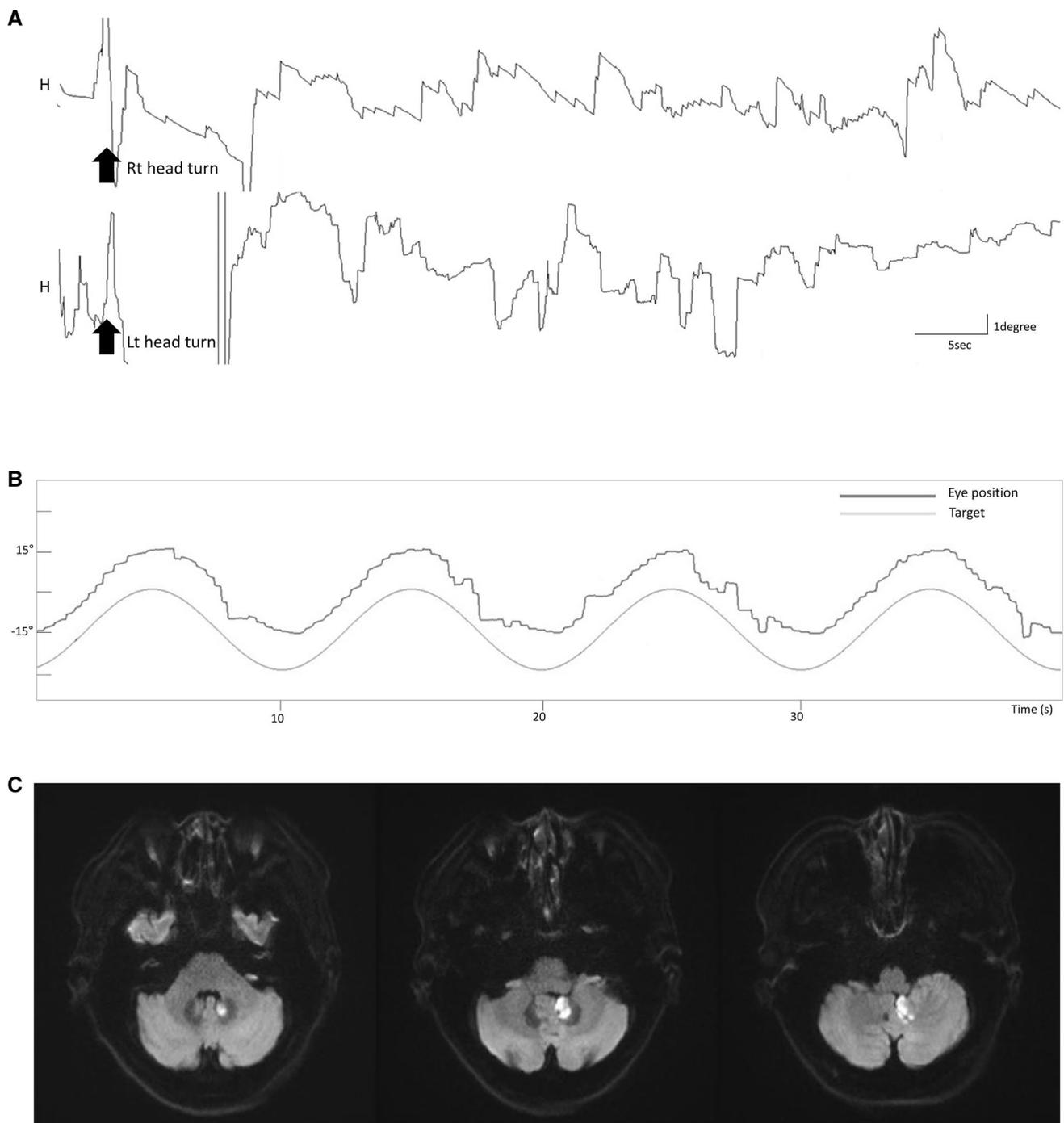


Fig. 1 **a** Horizontal eye movement recording using video-oculography shows right-beating nystagmus during rightward head turning. Leftward head turning does not induce any positional nystagmus. **b** Horizontal smooth pursuit at a peak target velocity of 15°/s was

impaired in both directions, more severe to the left side. **c** Diffusion-weighted MRIs show an acute infarction restricted to the left tonsil. *H* horizontal position of the left eye

[10]. However, our patient had an isolated tonsillar lesion without involvement of the nodulus/uvula, and showed positional horizontal nystagmus when the head was turned to just one side while supine. A few reports have described this type of CPN, considered atypical of horizontal canal BPPV

[9, 11]. Since tonsil and nodulus/uvula are mainly supplied by the medial posterior inferior cerebellar artery, there is a possibility that concurrent involvement of adjacent nodulus/uvula by abnormal perfusion resulted in positional vertigo and nystagmus in our patient. However, the tonsil was

also supposed as the structure for generation of CPN, with the nodulus and uvula [9–11]. Recent study revealed that the tonsil was the common area of injury, causing central geotropic positional nystagmus [12]. There is experimental evidence that otolith projections reach the vestibulocerebellum, including the tonsil [13]. Thus, positional vertigo and nystagmus in our patient may be ascribed to dysfunction of the generation and transfer of estimated gravity by involvement of the tonsil itself.

Compliance with ethical standards

Conflicts of interest We have no disclosure of any competing interest.

Ethical standards All the experiments followed the tenets of the Declaration of Helsinki and this study was approved by Institutional Review Board.

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