LETTERS

Acute rotatory vertigo caused by a small haemorrhage of the vestibular cortex

Central rotatory vertigo is in most cases caused by a lesion of the cerebellum or brain stem. We describe a patient with acute rotatory vertigo following a small haemorrhage in the left medial temporal gyrus, which probably injured the vestibular cortex.

Case history

A 53 year old woman suddenly experienced leftwards directed rotatory vertigo in the yaw plane and nausea without vomiting. She felt unsteady and had short lasting slurring of her speech. She had no hearing loss or tinnitus. On examination, she could stand unaided but tended to fall after a short while, without a directional preponderance. Gait was severely unsteady and she could not walk unaided. The rotatory vertigo was worse when she was sitting upright than when lying down in bed. Vertigo was also increased by head movements.

Examination of the cranial nerves showed no abnormalities; specifically there was no nystagmus or hearing loss and the eye movements were normal. Neurological examination of the limbs (motor and sensory function, coordination, and reflexes) was normal. Electroencephalography showed no abnormalities, supporting a non-epileptic cause of the vertigo.

Magnetic resonance imaging (MRI) on sagittal T1 weighted and transverse T2 weighted spin echo and FLAIR images showed a small (2.0 × 1.5 cm) haemorrhage in the left medial temporal gyrus, adjacent to the superior temporal sulcus (fig 1). There were no lesions of the brain stem or cerebellum. The appearance of the temporal lesion was consistent with haemorrhage from a small cavernous haemangioma.

Vestibular function was evaluated by electronystagmography (gaze, saccade, smooth pursuit, optokinetic, torsion swing, velocity step, and caloric tests; search for spontaneous nystagmus), and by video-oculography (ocular counter rolling induced by lateroflexion and eccentric rotation). No abnormalities were found. Additional testing included the Romberg test, galvanically induced body sway, and the subjective visual vertical. On Romberg testing there was abnormally increased body sway (especially with the eyes closed). The patient could stand long enough with the eyes closed to measure the galvanically induced body sway, which had normal excitability. The subjective visual vertical showed a 6° rightwards (clockwise) tilt and a reduced accuracy (SD 4.7°). The neurological symptoms and signs gradually disappeared over a few weeks.

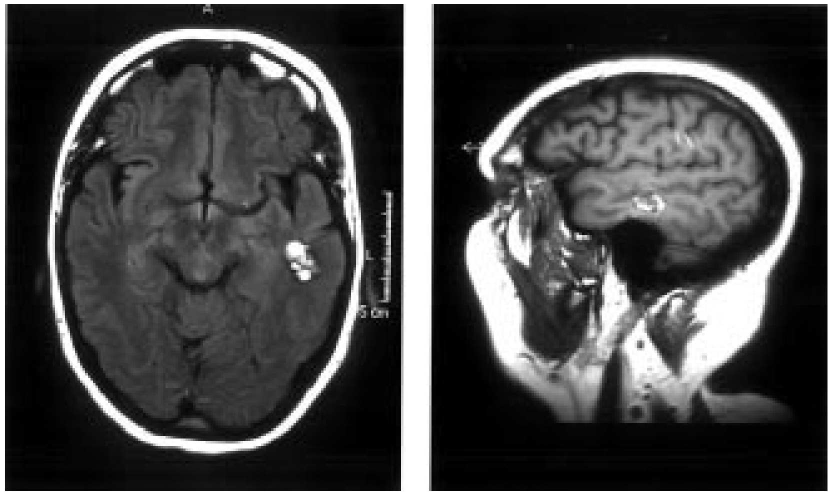

Figure 1 Transverse T2 weighted, fluid attenuated inversion recovery (FLAIR) image (left) and sagittal T1 weighted spin echo image (right). Both show a small popcorn shaped area of increased signal intensity demarcated by a rim of decreased signal intensity (haemosiderin), located in the left medial temporal gyrus. The combination of recent blood products and older haemorrhagic residues is consistent with the diagnosis of a cavernous haemangioma.

Comment

Our patient is of interest for two reasons. First, she demonstrates that acute rotatory vertigo may be caused by a lesion of the cerebral cortex, supporting the existence of a cortical area in humans with a substantial vestibular input. Second, she could be considered as an "experiment of nature": a small lesion confined to a particular brain structure, enabling precise localisation of an area in the cortex that seems to be very much engaged with the vestibular system.

The exact location of the vestibular cortex in humans has not yet been established. Primate studies have shown a well defined vestibular cortical system.[1] In all likelihood, a similar system probably also exists in humans, including, as in primates, several cortical areas.[1,2] However, one has to be careful in extrapolating results from primates to humans,[2] so human studies are important to further elucidate the existence and location of the human vestibular cortical system.

The vestibular cortical system seems to be distributed among several multisensory areas in the parietal and temporal cortex, and is integrated in a larger network for spatial attention and sensory-motor control. The parieto-insular cortex is postulated to be the core region within the vestibular cortical system; representation is bilateral, with a right hemispheric dominance.[3] Recent research seems to indicate that there might be no specific vestibular cortex, contrary to the visual and auditory systems. Electrophysiological recordings of vestibular cortical neurones, positron emission tomography, and fMRI brain activation studies during caloric and galvanic stimulation all confirm the multisensory character of cortical areas that receive a substantial vestibular input.[4–6] One can understand this when one realises that during motion not only the labyrinths but also the visual and proprioceptive systems will be stimulated. This could make a unimodal vestibular cortex unnecessary.[3]

We are aware of one other reported patient with rotatory vertigo and a cortical lesion on MRI.[7] That patient, however, had two cortical lesions: the main lesion was an infarct located in the right posterior insula involving the long insular and transverse temporal gyrus; the other lesion was in the right parietal cortex. We believe that our patient is the first reported case of rotatory vertigo resulting from a lesion (haemorrhage) of the medial temporal gyrus, adjacent to the superior temporal sulcus. Functional brain studies have shown that the human vestibular cortical system may be located in the superior temporal region posterior to the auditory area, probably in the superior temporal gyrus.[8,9] The results of functional brain studies, the previously reported patient,[7] and our own patient indicate that the human vestibular cortical system is located in several adjacent cortical areas: the superior temporal gyrus, the long insular and transverse temporal gyrus, and the medial temporal gyrus.

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