Acute upside down reversal of vision in verteobasilar ischaemia

Acute upside down reversal of vision is an uncommon and little known phenomenon consisting of transient complete 180 degree inversion of the visual image. The pathogenesis and the anatomical sites of this dysfunction are unknown. Lesions involving cortical areas, mainly the parieto-occipital region, or the vestibulocerebellar system have occasionally been documented.1,2

We observed two patients who experienced this bizarre visual illusion, both revealing features of verteobasilar ischaemia.

Patient 1, a 69 year old woman, was admitted because two weeks earlier she had experienced sudden malaise, sweating, nausea, vomiting, right occipital headache, followed by a 180 degree vertical inversion of the visual image, lasting about 20 minutes. Two similar episodes had occurred the day before admission. On admission the patient was alert, cooperative and well-oriented. The neurological examination was normal. In particular, no neuroophthalmological abnormalities were found on clinical examination. Blood parameters, urine, chest radiograph and ECG proved normal. Cerebral CT and MRI (figure) showed an ischaemic-like lesion, 2 cm diameter, in the right cerebellar hemisphere in the territory of the medial branch of the posterior inferior cerebellar artery (PICA), without mass effect. Moderate periventricular white matter abnormalities coexisted. Four vessel cerebral angiography revealed a right vertebral artery stenosis (50%) and two small arteriovenous malformations on the course of the right ascending cervical artery; a decreased flow in the basilary artery was noted. Ticlopidine 250mg daily was given and the patient was discharged. No further attacks or other neurological disturbances occurred during the next two years.

Case 2, a 52 year old woman, with a 40 year history of bilateral chronic otitis with residual deafness, had recent recurrent episodes of sudden swelling, pain, nausea, occipital headache, dizziness, sometimes followed by a transient loss of consciousness. The whole episode usually lasted about 30–40 minutes. Frequently, at the height of dizziness, the patient perceived an 180 degree vertical visual inversion of images. These episodes occurred monthly. On admission, the neurological examination was normal. Rare, isolated, left-sided jerks of horizontal nystagmus were revealed by ENG. A 20 mmHg difference between right and left brachial arterial pressure (right > left) was noted. Ultrasound vascular investigations (Doppler cortico-vertebral echomography and cerebral transcranial Doppler) revealed a left subclavian artery stenosis with a steal syndrome. Cerebral SPECT, CT and MRI proved normal. BAER was unavailable due to the previous history of chronic otitis. Cerebral angiography was refused. Fluorarazine 10mg daily was given and the patient was discharged with a warning to avoid strenuous physical activities, especially those involving upper limbs and neck. No further episodes were reported in the subsequent six months.

These two women presented episodes of vertically inverted vision—upside down phenomenon—associated with clinical signs and symptoms of verteobasilar insufficiency. Both reported transient visual inversion of 180 degrees, which was bilateral, of sudden onset and lacking subjective impression of movement (transient visual hallucination). In the first patient, neuroimaging revealed a right hemispheric cerebellar infarction. In the second, a verteobasilar failure due to a left subclavian stenosis was detected. The pathogenetic mechanism underlying upside down visual inversion is unknown. Since the visual images enter the retina inverted, it may be assumed that the upside down phenomenon is associated with the failure of the mechanisms mediating reversion, even though the anatomical structures involved are unknown. In earlier observations,10 parietal and/or occipital lesions were sometimes associated with cortical origins of the dysfunction, probably affecting the integrative control of spatial vision.

More recent cases,11,12 documented with neuroimaging techniques, revealed an association with vestibular/cerebellar lesions, that is, verteobasilar TIAs, Wallenberg's syndrome and also cerebellar infarct in two cases.13 In our patients, the relationship between the vertical visual inversion and the signs and symptoms of verteobasilar insufficiency, without evidence of cerebral damage, supports the idea that a transient inactivation of infratentorial structures may cause this visual phenomenon. Besides the integrity of the visual system, space visual perception needs a flow of extraretinal information, mediated by the vestibular and cerebellar systems.14 It has been suggested that damage to such structures may cause tilt and complete inversion of the visual space.15 The upside down phenomenon may occur following dysfunctions at various levels of the vestibulo-cerebellar-ocular system mediating the stabilization of the visual function so that cortical involvement is not indispensable.

Acute upside down reversal of vision in vertebrobasilar ischaemia.

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