

LETTERS TO THE EDITOR

"Alice in Wonderland" syndrome and infectious mononucleosis in children

Visual illusions (metamorphopsia) characterised by distortion of form, size, movement or colour was initially labelled the Alice in Wonderland Syndrome (AWS).¹ It has rarely been reported in children.^{2,3} In 1977 Copperman² reported three patients with metamorphopsia as the initial symptom of infectious mononucleosis (IM). We reported a similar observation.³ We now describe a child with AWS with no obvious history nor relevant clinical signs that suggested active IM but followed a mild clinical, but serologically, proven course of IM.

A six year old girl with an uneventful medical history and no history of migraine, epilepsy or behavioural abnormalities, was referred to us because of metamorphopsia associated with mild headache and anxiety. These episodes recurred several times a day lasting a few minutes each time. Detailed history revealed that two weeks before these symptoms the patient had a "throat infection" with high fever and enlarged cervical lymph nodes. Throat cultures were sterile and the symptoms disappeared gradually without treatment. Physical examination was unremarkable except for the spleen which was palpable 3cm below the costal margin. Neurological examination showed an alert intelligent child who was able to describe her symptoms accurately.

Laboratory findings revealed a white cell blood count of 15000/mm³ with 16% large atypical lymphocytes (Downey's cells). The initial viral capsid antibody titre for Epstein-Barr virus (EBV) was 1:80 and two weeks later 1:20. Epstein-Barr nuclear antibody titres were 1:10 and 1:20 four and six months respectively after the initial episode. Computerised tomography of the brain was normal. An EEG performed a week after the beginning of her complaints was abnormal showing normal background activity of alpha rhythm with a few generalised series of sharp high voltage waves mostly in the parieto-occipital region. Repeat EEG two weeks later was normal. There was complete resolution of the visual symptoms after four weeks. During 12 months of follow up there were no visual complaints nor any other neurological symptoms.

Various neurological and psychiatric symptoms have been described in association with infectious mononucleosis.^{2,4,5} Metamorphopsia, a rarely reported symptom, may appear before the onset^{2,3} or after the resolution of all clinical symptoms, as described in our patient. In all the previously reported patients no neurological deficits were observed. Electroencephalograms were either normal or showed left temporal slow waves. The diagnosis of infectious mononucleosis was clearly established in all patients by the strongly positive haematological and serological findings.^{2,3} The duration of the visual illusions ranged between two weeks and seven months, and all patients recovered completely.

Metamorphopsia has also been related to lesions of the occipital, occipitotemporal or occipitoparietal areas of the brain.⁶ In our patient no structural brain abnormalities were observed, however, the EEG suggested an electrical dysfunction in the parieto-occipital region.

Few patients with AWS secondary to IM have been reported, but this combination may not be that rare. The question of whether these visual illusions of AWS are specific to IM or might be seen in association with other viral diseases, remains unsolved.

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Infarct in the territory of the medial branch of the PICA

Many cases of small cerebellar infarcts mimicking labyrinthine dysfunction have been reported, but clinico-anatomic correlations were often imprecise.¹⁻⁷ We report a case in which magnetic resonance imaging (MRI) localised the lesion to the territory of the medial branch of the posterior inferior cerebellar artery (PICA).

A 62 year old female was admitted because of a sudden rotatory vertigo accompanied by nausea and vomiting. She was a heavy smoker who had hyperlipidaemia and paroxysmal atrial fibrillation. In April 1987, a right carotid endarterectomy had been performed for a tight stenosis. There was also a tight stenosis of the prevertebral subclavian artery. She was taking aspirin and disopyramide.

On examination, the patient had persistent vertigo, nausea and vomiting. She was unable to walk or stand. There was no spontaneous or initiated nystagmus. Caloric irrigation produced symmetrical responses and there was no directional preponderance. The audiogram was normal. The remainder of the neurological examination was normal and in particular there was no dysmetria or sensory disturbance. CT, with and without contrast, performed on 21 April 1989 was normal. Three days later, the MRI on T1 and T2 weighted sequences revealed a left sided hyperintensity of the nodulus and of the postero-inferior part of the cerebellar hemisphere (fig), consistent with a haemorrhagic infarct in the cerebellar territory of the medial branch of the PICA. The patient's clinical

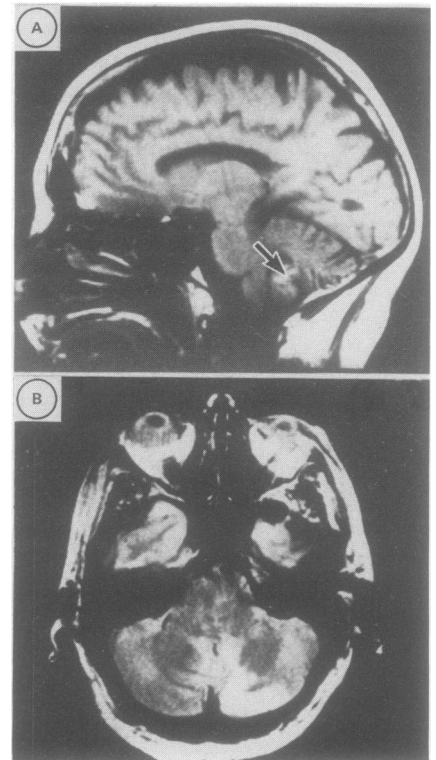


Figure (a) T1 weighted MRI: hyperintensity of the nodulus. (b) T2 weighted MRI: hyperintensity of the nodulus and of the postero-inferior part of the cerebellar hemisphere.

condition improved spontaneously over 36 hours.

The PICA contributes to the supply of the medulla and cerebellum. In the medulla, it always supplies the dorsal territory, but its contribution to the supply of the lateral part is extremely variable. In the cerebellum, the PICA ends in two branches: a lateral one, which supplies most of the posterior and inferior part of the cerebellar hemisphere and a medial one which supplies the dorsal part of the medulla, the inferior part of the vermis including the nodulus, and the adjacent cerebellar hemisphere.⁸ Infarcts may be limited to the cerebellar territory of this branch as demonstrated by MRI in our case and pathologically in the case of Amarenco *et al.*¹

In both these cases, acute vertigo was the only symptom, neurological examination was normal and there was no noticeable dysmetria. The electronystagmogram performed in our case was normal and caloric irrigation produced symmetrical responses. This syndrome may be explained by the involvement of the nodulus, part of the flocculo-nodular complex, that has primary vestibular connections.

In a comparable case of Duncan *et al.*² the nodulus was intact but the flocculus, usually supplied by the anterior inferior cerebellar artery (AICA), was involved. Small cerebellar infarcts in which acute vertigo is the only presenting symptom may closely mimic an acute peripheral labyrinthine disorder. Our case supports the view of Guiang *et al.*³ that normal caloric responses suggest a cerebellar lesion.

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