Altitudinal hemianopia in multiple sclerosis

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ABSTRACT. A patient with remittent multiple sclerosis presented subacutely macular visual dysfunction and superior altitudinal hemianopia documented with static and kinetic perimetry. MRI showed extensive demyelination in both temporo-occipital optic radiations. This is the first reported case of altitudinal hemianopia due to multiple sclerosis.

Key words: multiple sclerosis; altitudinal hemianopia; occipital lobe syndrome; retrochiasmal visual pathways

INTRODUCTION

Perimetric deficits in relation to demyelination in the retrochiasmal visual pathways are very uncommon in multiple sclerosis (MS) (see review). Since the CT scan era, there exist only a limited number of appropriately documented cases showing homonymous hemianopia or quadrantanopia. MRI correlations were available in only one of these publications. We document with MRI the first case of MS-related altitudinal hemianopia.

CASE REPORT

A man, born in 1936, working as a schoolteacher, had several episodes of diffuse paresthesiae, often with a Lhermitte's sign or diplopia, since 1958. Cerebrospinal fluid, examined in 1968 after a right hemiparetic attack, was normal, including electrographic profile. A diagnosis of remittent MS was made. CT scan was done in 1984 following a second episode of right hemiparesis and showed bilateral small enhancing foci in the hemispheres, which were consistent with MS plaques. At that time, he began to complain of fluctuating disturbed vision. Corrected VA was 8/10 RE and 7/10 LE. Goldmann perimetry was normal. Visual evoked potentials were pathologically delayed for either eye. Since then, he experienced no further attack, but he felt that his vision got progressively worse and that the upward field of vision was restricted. VA was 8/10 RE and 5/10 LE, but dropped to 3/10 RE and 2/10 LE a few months later. Ocular examination showed only a mild optic disk pallor. Severe acquired dyschromatopsia was demonstrated with the Farnsworth D15 Panel. The contrast sensitivity function was depressed for the whole spatial frequencies range. Goldmann perimetry showed a superior altitudinal hemianopic scotoma, splitting the macula, with the I12 isopter (Fig. 1). Static automated perimetry (Vision Monitor; Métrievision manufacturer) using a 95 points program and a supra threshold strategy confirmed an absolute...
superior homonymous deficit with a macular sparing (Fig. 2). The field amputation appeared highly congruous with both methods. A high-dose steroid course had not altered the perimetric deficit but yielded a gain of 1/10 of VA in the two eyes. In contrast to the visual symptoms, the neurological examination was unremarkable apart from a mild cerebellar syndrome and some verbal memory decline. Axial MRI (0.5 Tesla) with T2-weighted sequences showed extensive periventricular white matter hypersignals. In particular, there were bilateral lesions lateral to the occipital horns and spreading in the occipital lobes (Fig. 3).

Fig. 1. Goldmann perimetry showing the upper altitudinal hemianopic defect.

Fig. 2. Automated static perimetry in the central 30° showing an absolute superior altitudinal deficit with macular sparing (the numerical values indicate the retinal sensitivity in dB for each point tested; 0 corresponds to no perception).
DISCUSSION

Altitudinal hemianopia is a rare condition which usually points to a bilateral occipital lobe pathology. Our case developed a dense and congruous superior altitudinal hemianopia which was documented with both static and kinetic perimetry. A diagnosis of definite MS according to Poser et al.’s criteria was supported by the clinical and laboratory data. MRI showed diffuse white matter periventricular lesions typical of MS, and no evidence for another condition. MRI lesions spread almost symmetrically in the temporoparietal regions, from the lateral aspect of the occipital horns to the depths of the medial part of the occipital lobes. They were likely to damage the ventral bundle of the optic radiations, resulting in a bilateral dysconnection of the inferior lip of the calcarine fissure from its retino-geniculate inputs. Congruence of the field defect and macular sparing were probably related to the posterior extension of demyelination in the occipital lobes. Reduction of VA, attenuation of contrast sensitivity, dyschromatopsia, abnormal VEP’s and optic disk palor were probably not caused by the lesions in the optic radiations. Although consistent with an optic tract involvement, they were more likely related to a bilateral subacute optic neuritis.

It remains a valuable clinical rule to question a diagnosis of MS when homonymous hemianopia is present. However, the widespread distribution of plaques in the central visual pathways makes it likely that almost every type of visual field deficit could be encountered. In addition to bitemporal heteronymous hemianopia and homonymous hemianopia, our case illustrates that MS can present with altitudinal hemianopia.

REFERENCES