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Incongruous homonymous hemianopic scotoma

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Abstract

We report a patient presenting with incongruous homonymous hemianopic scotoma due to infarction in the territory of the lateral posterior choroidal artery. Imaging studies showed that the patient had a fresh infarct in the lateral geniculate body causing this unusual visual field defect. © 1999 Elsevier Science B.V. All rights reserved.

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1. Introduction

Neurological symptoms of lesions in the lateral posterior choroidal artery (L-PChA) territory have been characterized by visual field defect, sensory dysfunction, motor weakness, neuropsychological disturbances and hyperkinetic syndrome [1,2]. In particular, quadrantanopia of the lower or upper visual field is frequently observed. However, homonymous horizontal sectoranopia is the most unique abnormality [3]. We presented a patient whose visual field defect was scotomatous, i.e. the peripheral visual field was spared. This field defect strongly suggests a tiny restricted lesion of the L-PChA territory.

2. Case history

A 71-year-old right-handed, hypertensive man could not see the left part of traffic signal while he was driving his car. The next day, he was admitted to our hospital because of the visual field abnormality. Upon admission, his blood

pressure was 160/80 mmHg and his pulse was regular at a rate of 60 beats/min. Neurological examination using the confrontation test revealed homonymous hemianopia on the left side. Other neurological findings were normal. Electrocardiography was normal and cranial CT showed no lesions. Laboratory examination was normal for liver and renal function, and for serum cholesterol, blood glucose, blood cell count, plasma levels of protein S, protein C, antithrombin III, plasminogen, D-dimer, lupus anticoagulant and the anticardiolipin antibody.

Cerebral angiography performed on the 4th day, revealed stenosis of the second segment of the right posterior cerebral artery (PCA) (Fig. 1). The left PCA, basilar artery, middle cerebral arteries, anterior cerebral arteries and vertebral arteries all appeared normal.

On day 5, a visual-field examination using a Goldmann perimeter disclosed left incongruous homonymous horizontal sectoranopia that was restricted to the central regions of the visual field (incongruous homonymous hemianopic scotoma) (Fig. 2). His best corrected visual acuity was 20/20 bilaterally. External, motility, pupillary, slit-lamp, and funduscopy were normal. Optic atrophy was absent.

On day 7, diffusion weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) magnetic resonance imaging (MRI) revealed recent ischemic lesions in the

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Fig. 1. Angiography demonstrating stenosis of second segment of PCA (arrow) 4 days after stroke onset.

right lateral geniculate body (LGB), posterior thalamus and peduncle of the fornix (Fig. 3), which are supplied by the L-PChA.
His visual defect gradually improved, but fields had not fully normalized when examined 2 months after the stroke.

3. Discussion
The incongruous homonymous hemianopic scotoma seen in this patient was most likely caused by infarction of the LGB detected in MRI.

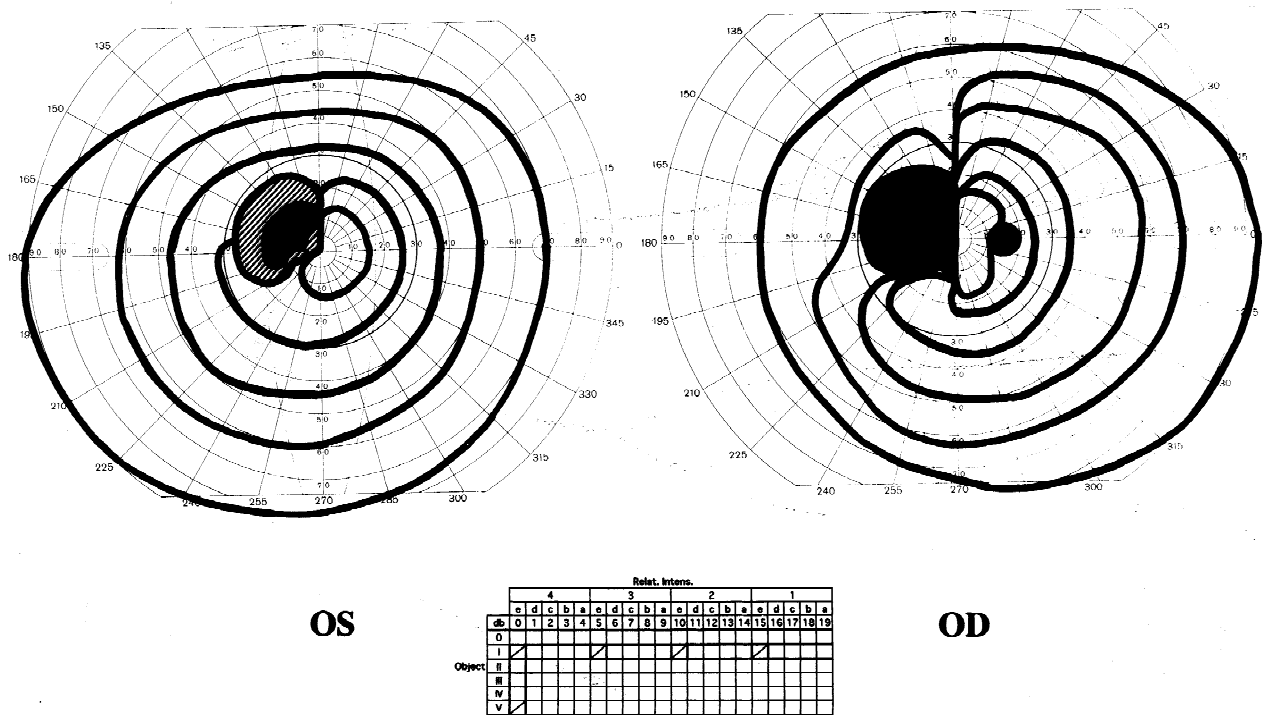


Fig. 2. Visual-field testing using Goldmann perimeter revealed incongruous homonymous hemianopic scotoma on the left side 5 days after stroke onset. Black areas and semi-shaded areas indicate absolute and relative scotoma, respectively.

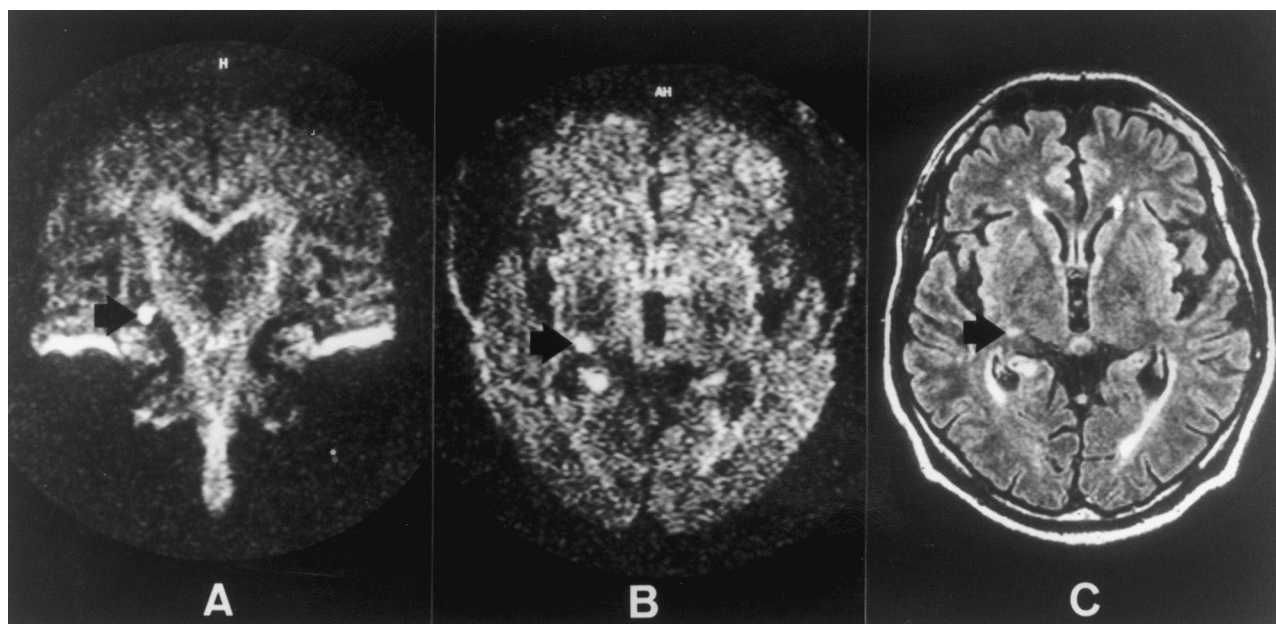


Fig. 3. Diffusion weighted imaging (DWI) (A,B) and fluid-attenuated inversion recovery (FLAIR) (C) magnetic resonance imaging (MRI) demonstrating an infarct in LGB (arrow) 7 days after stroke onset. The occipital lobes appear normal.

Isolated visual field defect without concomitant involvement of the superficial PCA territory is always associated with the L-PChA territory infarct and correlated well with involvement of the LGB. The most specific visual field defect caused by infarction in the territory of the L-PChA is homonymous horizontal sectoranopia [3–5].

The upper and lower retinal quadrants project into the medial and lateral parts of the LGB, respectively [5]. A lesion limited to the lateral part of the LGB results in upper quadrantanopia, while a lesion of the medial part produces lower quadrantanopia. The LGB is supplied by two main arteries, one main artery is the anterior choroidal artery (AChA) and the other is L-PChA [3–5]. Homonymous horizontal sectoranopia may occur when the central part of the LGB, which is supplied by the L-PChA, is involved.

In published case series, the visual field defect always extended to the periphery [1,3–5]. However, our patient presented with incongruous homonymous hemianopic scotoma with infarction in the territory of the L-PChA. The macular fibers project into the caudal region of the LGB, and the peripheral visual field is represented serially along the caudo-rostral axis [6]. Therefore, a lesion restricted to the central and caudal part of the LGB can cause such a scotomatous visual field defect. In patients with LGB infarction, visual field defect is characterized by the homonymous horizontal sectoranopia. However, the upper or lower quadrantanopia and hemianopia are more frequently found in these patients [1]. These findings clearly

indicated that the areas of the LGB irrigated by the L-PChA are variable among individuals. It is conceivable that the L-PChA mainly irrigated the posterior pole of the LGB in the present patient, and the anterior part of the LGB was supplied by the other artery (AChA).

Lesions in the occipital cortex usually produce congruous visual field defects, while abnormalities of more proximal retrochiasmal areas result in incongruous defects [7]. Scotomatous visual field defects are most frequently caused by cortical lesions, and the defects are congruous in patients with cortical lesions. Therefore, incongruous and scotomatous visual field defects are the rare phenomenon. The LGB has six laminae and these layers are numbered one through six in a ventral-dorsal direction, with the ipsilateral retinal fibers terminating in layers two, three, and five, and the crossed fibers terminating in layers one, four, and six [7]. Uncrossed retinal projections terminate in one group of the LGB laminae while crossed retinal projections terminate in another. The laminar structure of the LGB provides the anatomical basis for the occurrence of incongruous visual field defects in a case of partial lesion of the LGB [7]. Patients with occipital tip lesions or fundus pathology occasionally have incongruous visual field defects [8]. Our patient had neither occipital tip lesions nor fundus pathology.

Incongruous homonymous hemianopic scotoma have not been reported yet. There are two theoretical reasons why scotomatous defects are uncommon following LGB infarcts. First, it is assumed that incongruous homonymous hemianopic scotoma is a subtype of homonymous horizon-

tal sectoranopia, a rare defect in its own right. Second, only very small lesions in the LGB could produce the scotomatous defect. But such small lesions could not have been reliably documented prior to the advent of such MRI techniques as DWI and FLAIR [9,10]. DWI is useful for distinguishing acute lesions from chronic ones [9]. FLAIR offers advantages in detection of acute infarcts that contact to the cerebrospinal fluid [10].

The most common cause of infarction in the L-PChA territory is small-artery disease [1]. However, our patient had angiographic evidence of stenosis of the second segment of PCA, due possibly to an atherosclerotic plaque. Therefore, the present infarction appears to have been atherothrombotic.

Based on the present data, we believe that an ischemic lesion due to occlusion of the L-PChA should be considered in patients presenting with incongruous homonymous hemianopic scotoma.

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