CASE REPORT

Homonymous Sectoranopia: Asymptomatic Presentation of a Lateral Geniculate Nucleus Lesion

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ABSTRACT

This is a rare presentation of brain tumour in the region of the lateral geniculate nucleus (LGN) presenting as a homonymous horizontal sectoranopia (HHS). The case highlights that subtle field defects can be asymptomatic and only detected by formal perimetry. Although homonymous sectoranopia is a rare form of visual field defect, it should be recognised as a potential manifestation of potentially significant intracranial pathology.

Keywords: Homonymous horizontal sectoranopia, glioma, lateral choroidal artery, lateral geniculate nucleus

Homonymous sectoranopia with its sloping boundaries of field defect is very rare, accounting for 0.3% of all homonymous field defects.¹ Typically homonymous sectoranopia occurs as a result of an insult to the lateral geniculate nucleus (LGN), reflecting its retinotopic organisation and distinct regional blood supply.² The fovea is represented by the posterior part of the LGN, with blood supply from the posterolateral choroidal artery (PLChA). The upper and lower quadrants of the visual hemifields are represented by the anterolateral and anteromedial segments of the contralateral LGN, respectively. Both segments are supplied by the anterior choroidal artery (ACA). Therefore, a lesion in the territory of the PLChA results in a contralateral horizontal wedge-shaped defect called homonymous horizontal sectoranopia (HHS), whereas a lesion in the territory of the ACA results in contralateral defects in superior and inferior hemifields, sparing a wedge-shaped horizontal field. Such a defect is termed homonymous quadruple sectoranopia^{2,3} (Figure 1).

A 22-year-old woman was found to have a right HHS on automated perimetry as part of a routine optician check in 2007. She was unaware of any visual defect prior to the examination. She had no past medical history apart from cervical dysplasia with satisfactory yearly follow-up.

Ophthalmological examination revealed normal visual acuity, colour vision, slit-lamp examination of the anterior and posterior segments of her eyes and intra-ocular pressures. Goldmann perimetry revealed a right HHS (Figure 2). The rest of her neurological examination was entirely normal. Funduscopy revealed loss of the nerve fibres in the papillomacular and nasal areas in the right fundus and atrophy of the temporal hemiretina in the left fundus (Figure 3). Optical coherence tomography (OCT) showed that the peripapillary retinal nerve fibre layer thickness (RNFL) in the nasal-temporal area of the right eye (or crossing fibre defect eye) was thinner than those in the left eye. The RNFL in the superior-inferior area of the left eye (or non-crossing fibre defect eye) was

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FIGURE 1 Schematic representation of (a) left homonymous horizontal sectoranopia due to pathology of the posterior pole of the contralateral lateral geniculate nucleus (territory of the posterolateral choroidal artery), and (b) left homonymous quadruple sectoranopia due pathology of the anterolateral and anteromedial parts of the contralateral lateral geniculate nucleus (territory of the anterior choroidal artery).

thinner than those in the right. The findings correspond with the retinotopic organisation and reflect a retrograde degeneration from the LGN (Figure 4).

Magnetic resonance imaging of the brain revealed a 3-cm heterogeneous hyperintense ill-defined mass on T2-weighted images centred below the left putamen and above the choroid fissure, approximately in the region of the LGN (Figure 5). The LGN itself was not visualised and there was mass effect on the local structures, in view of the substantial size of the tumour and its location. In particular, there was mass effect and/or involvement of the posterior part of the optic tract, with thinning and T2 hyperintensity of the optic tract, suggesting retrograde axonal degeneration. In addition, there were further separate foci of signal change similar to the tumour, involving the medial thalamus and superior and inferior colliculi.

Repeat imaging over a 7-year period revealed no significant overall change in tumour size. There was some internal heterogeneity within the tumour with foci of high T1 signal showing susceptibility on T2 images that has progressed over time, indicating calcification or possibly haemorrhage. A low-grade tumour was felt to be the most likely diagnosis.

Her visual acuity, colour vision, and neurological examination remained unchanged, but there was slow objective progression of visual field defect noted over 7-year follow-up, which became almost a complete right homonymous hemianopia. The patient declined biopsy and possible radiotherapy and opted for close monitoring. She was fitted with Eli Peli-style hemianopia prisms for hemianopic field expansion.

This is a rare presentation of brain tumour in the region of the LGN presenting as HHS. However, lack

FIGURE 2 Goldmann perimetry revealing right homonymous horizontal sectoranopia.

FIGURE 3 Right (a) and left (b) fundus photographs showing atrophy of the retinal fibre layer corresponding to the right homonymous horizontal sectoranopia. In the right fundus, there is bow tie atrophy with striae of the nerve fibre layer close to the upper and lower margins of the disc. In the left fundus, there is pallor of the upper and lower temporal parts of the disc.

FIGURE 4 OCT showing retinal nerve fibre layer thickness. There is loss of nasal and temporal fibres in the left eye, and superior and inferior fibres in the right eye corresponding to lesion in the right optic tract or beyond.

FIGURE 5 MRI brain images. Top row: Axial and coronal T2-weighted MRI images taken through the level of the LGN shows the normal LGN on the right side (short arrows) and the tumour on the left side (*) with its epicentre approximately in the region of the LGN but also exerting mass effect on the surrounding structures. Superoinferiorly, it lies between the putamen above and the choroidal fissure below. There is some compression of the temporal stem and mesial temporal lobe. Bottom row – axial and coronal T2-weighted images obtained more inferiorly and anteriorly, demonstrate a normal optic tract on the right side (long dashed arrows) and abnormally thin and hyperintense optic tract on the left side (long solid arrows), presumably due to retrograde axonal degeneration and/or partial involvement.

of histopathological confirmation is a limitation. Most of the reported cases of sectorial sectoranopia occurred as a result of a vascular insult. Although sectoranopia^{4–8} is typically associated with lesions of the LGN, a few cases have been reported as a result of lesions affecting the temporo-occipital or parieto-occipital lobes⁹

as well as optic tract lesions¹⁰ and lesions involving the primary visual cortex with the calcarine fissure.¹¹ However, in this case, the pattern of retinal nerve fibre layer atrophy suggested a left-sided damage to second-order neurons behind the chiasm at the level of either the optic tract or LGN. The sectorial pattern

of the field defect pointed to a lesion in the LGN rather than the optic tract.

The case highlights that subtle field defects can be asymptomatic and only detected by formal perimetry. Although homonymous sectoranopia is a rare form of visual field defect, it should be recognised as a potential manifestation of significant intracranial pathology.

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