

MR demonstration of partial lesions of the lateral geniculate body and its functional intra-nuclear topography

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Abstract

Two rare cases with a partial lesion of the lateral geniculate body (LGB) presumably due to ischemia are demonstrated on high resolution MR imaging. A 62-year-old woman (case 1) presented with left homonymous superior quadrantanopia on Goldmann perimetry. Heavily T2 weighted MR images showed a localized lesion at the lateral portion of the LGB. The visual field defect was macular and horizontal meridian sparing and persisted for 9 years. A 49-year-old woman (case 2) presented with a sudden onset of left homonymous horizontal sectoranopia on Humphrey automated perimetry and heavily T2 weighted images demonstrated a lesion localized at the more medial part of the right LGB. Axons originating from inferior, central and superior retina are essentially located laterally, centrally and medially, respectively, in the LGB, based on the electrophysiological studies of animal experiments and this observation has been applied to humans. This study radiologically shows that a discrete lateral lesion of the LGB produced homonymous upper quadrantanopia in case 1, whereas a more medially located lesion produced homonymous sectoranopia in case 2, and reveals that the axons originating from inferior retina are located more laterally than those from central retina in the human LGB.

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1. Introduction

The human lateral geniculate body (LGB) is the principal thalamic visual center linking the retina and the striate cortex [1]. Determination of axonal projections from the retinal ganglion cells to the LGB has been based on the electrophysiological studies using monkeys and this observation is applied to humans [1,2]. In normal humans, the LGB has been successfully visualized with the advent of superconductive MR apparatus [3,4]. In clinical settings, although MR demonstrations on nuclear lesions have been attempted to correlate with the patterns of visual field defects [5], direct evidence of intra-nuclear topology and visual field defect has not been reported in humans. We report two patients with a discrete lesion affecting a part of LGB. Intra-nuclear

topography of the LGB is discussed in in vivo human brains.

2. MR imaging

T1 and T2 weighted MR images were performed with a 1.5 T MR imaging unit (Sigma; GE Medical Systems, Milwaukee, WI). Scan parameters were as follows: repetition time (ms)/echo time (ms), 500/9 and 2000/80 ms, respectively; field of view, 16 cm × 16 cm; matrix, 256 × 224; slice thickness, 3 mm; gap, 0.5 mm; number of signals acquired, two.

MR apparatus was a Gyroscan ACSNT (Philips) 1.5 T for heavily T2 weighted fast spin echo method. Black and white reversed imaging was used. Sequence parameters were TR 5800 ms and TR 250 ms, 200 mm field of view, 284 × 512 pixel matrix, and slice thickness of 2.5 mm with 0.5 mm interslice gap and four excitations. The axial and coronal sections on MR imaging are essentially parallel

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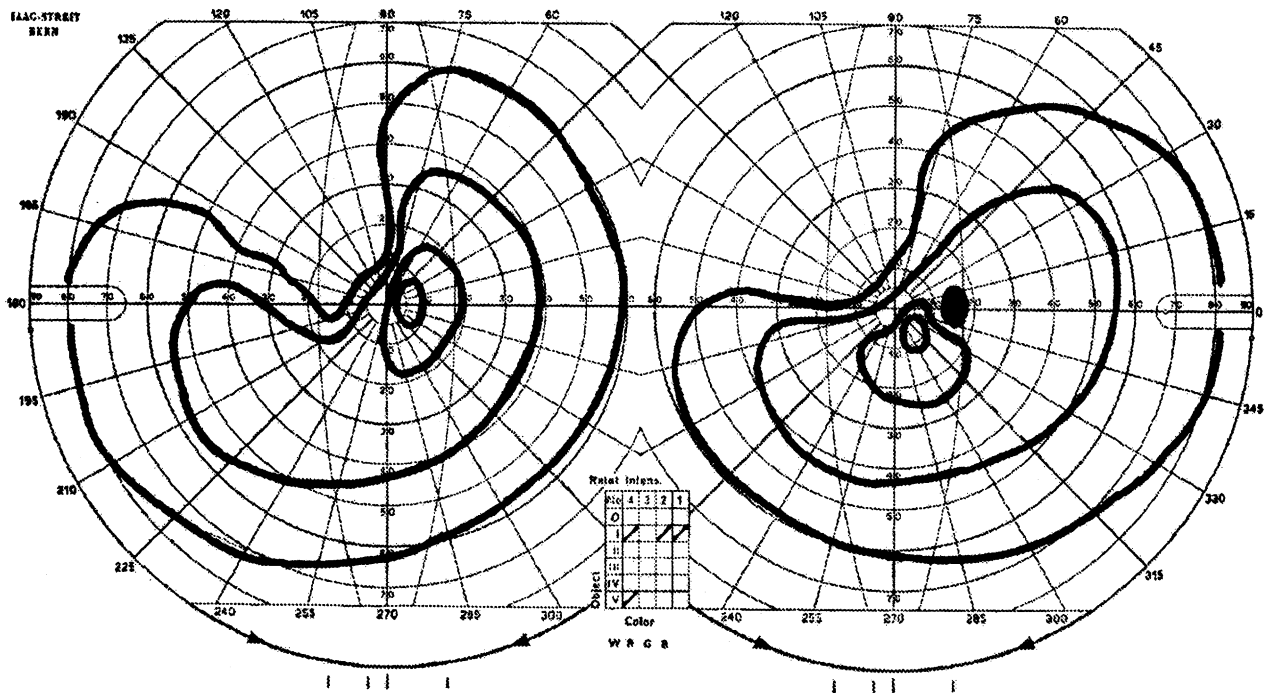


Fig. 1. The left-sided homonymous upper quadrantanopia was noted in Goldmann perimetry 8 years after the onset.

to and perpendicular to the line connecting the centers of anterior and posterior commissures, respectively (Fig. 1).

3. Case report

3.1. Case 1

A 62-year-old woman, with a past history of hypertension, complained of a sudden onset of blurred vision on

the left visual field in January 1992. She was free of other complaints. Left homonymous upper quadrantanopsia was found on ophthalmological examination. She was then referred to us for further evaluation. On admission she was free of neurological deficits except for the visual field defect. Goldmann perimetry showed left-sided homonymous upper quadrantanopsia. Ocular fungi were normal. CT scan revealed a low density lesion at the right postero-lateral thalamus. MRI revealed a localized high intensity lesion in T2

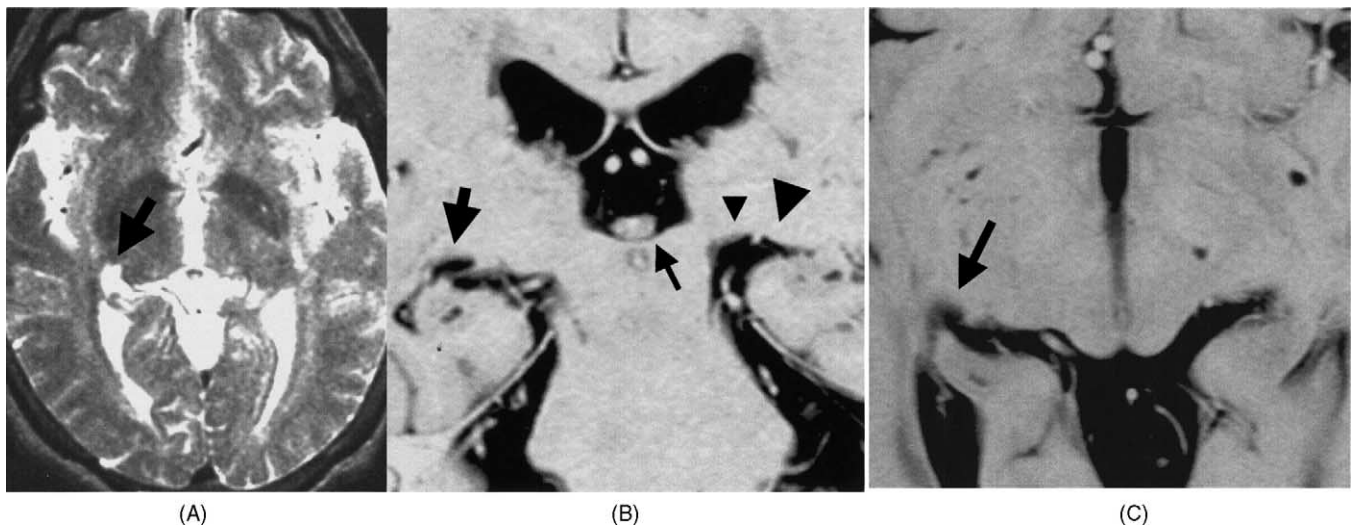


Fig. 2. (A) Two weeks after the onset. T2 weighted axial image showed a localized lesion at the posterior and lateral thalamus (arrow). (B) Eight years after the onset. Coronal image at the pineal body (small arrow), one slice posterior to the posterior commissure. On the left side, medial and lateral bulges show the medial and lateral geniculate body (small and large arrow heads), respectively. On the right side, the lateral part of the bulge has a low intensity lesion (large arrow), compatible with the infarction. (C) Eight years after the onset. On axial images, lateral part of the lateral geniculate body has a low intensity lesion (arrow).

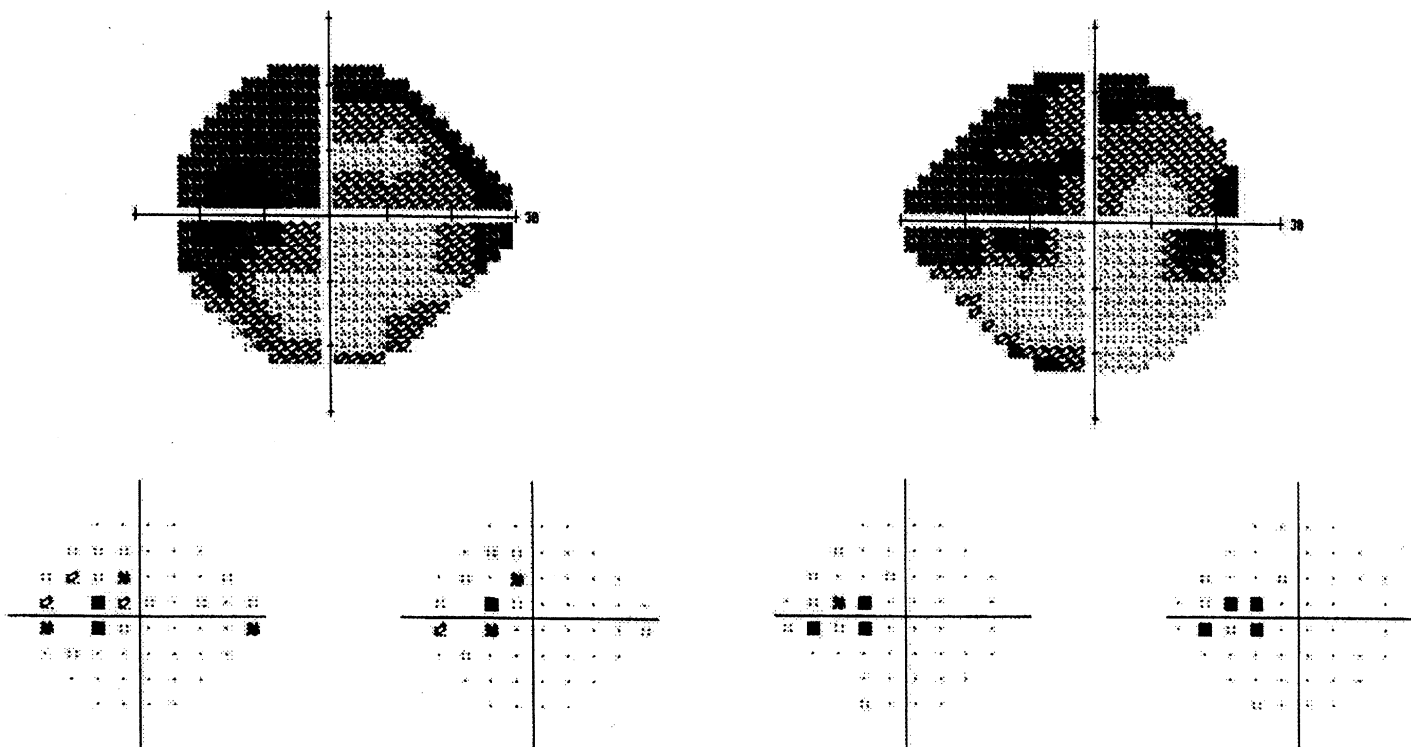


Fig. 3. One month after the onset. Humphrey automated perimetry program 24-2 (54 points at 6° grid within 30° visual field) revealed left homonymous horizontal sectoranopia on total and pattern deviation maps (lower).

weighted image (Fig. 2A). The patient was diagnosed as having an infarction at the LGB. MRA revealed no obstruction or stenosis at the major vessels. In the outpatient clinic, she was treated with ticlopidine 200 mg per day. The same visual field defect has been persistent for the next 8 years. Heavily T2 weighted MR imaging revealed a lesion located at the lateral part of the right LGB at the two coronal slices including the posterior commissure (Fig. 2B and C).

3.2. Case 2

A 49-year-old woman, free of risk factors for strokes, suddenly presented with difficulty to see on her left visual field during dinner on July 2001. She did not have headache or nausea. Although the subjective complaint continued, she was found to be free of visual field defect on confrontation at a nearby neurological institute. Four weeks later, in our institute, left homonymous, horizontal sectoranopia was found on Humphrey automated perimetry (Fig. 3), although Goldmann perimetry failed in detecting the visual field defect. She did not have any other neurological deficits. T1 weighted MR imaging revealed a localized lesion at around the posterior part of the right thalamus (Fig. 4A). Heavily T2 weighted MR images demonstrated a lesion localized at the rather medial part of right LGB, starting two slices anterior to the posterior commissure section (Fig. 4B and C). MRA did not reveal any abnormality on major vessels. Although she did not have any risk factors for cerebral stroke, she was suspected to have a lacunar infarction, due to sudden onset of the sign and characteristic visual field defect caused by occluded posterior choroidal artery. The same visual field defect has persisted for 6 months.

4. Discussion

4.1. Anatomy of LGB on MRI

In this MR study, the location of the LGB was mainly determined by use of the following two landmarks: (1) adjacent neural structures such as posterior commissure, pineal body and medial geniculate body and (2) characteristic shape of lateral and medial geniculate bodies in the ambient cistern.

The posterior commissure is present almost at the middle of the LGB in anterior–posterior dimension [4,6,7]. Since the LGB is 9 mm in antero-posterior dimension [6,7], it is visible one or two slices (3 mm in slice thickness) both in front of and at the back of the coronal section of the posterior commissure. Tamraz et al. emphasized a constant detection of the LGB at the section passing the posterior commissure [3]. The pineal body is located one slice posterior to the posterior commissure in coronal section [6,7].

As the optic tract runs along the cerebral peduncle posteriorly, it gradually relocates laterally off the midline and, at around the posterior one-third of the brain stem, it ends in the LGB, a ovoid small cup-shaped mass, making a hump, protruding inferiorly, beside the brain stem, on coronal sections [4,6,7] (Fig. 4B). More posteriorly at the coronal section including the posterior commissure, the medial geniculate body becomes evident as another small bulge on the medial side of the LGB [4,6,7]. Accordingly, there are two small humps on this coronal section at the posterior commissure section [6,7] (Fig. 2B). This is due to the anatomical observation that the medial geniculate body is located medially and a few mm posteriorly to the LGB [6,7]. Based on those radio-anatomical landmarks, the lesions in our two cases were determined to be located in the LGB. In case 1, the lesion was located at the lateral

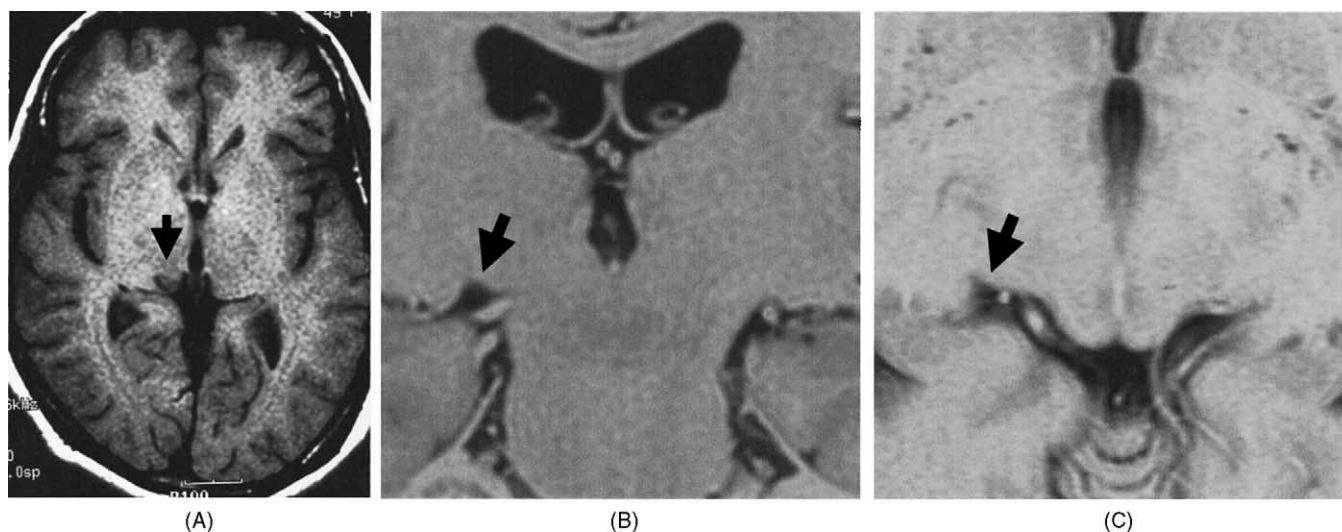


Fig. 4. (A) T1 weighted axial image. A localized low signal intensity is visible at the posterior thalamus (arrow). Heavily T2 weighted coronal (B) and axial (C) slices. (B) The coronal image shows one slice anterior to the section including the posterior commissure. Inverted triangle-shaped low intensity (arrow) is visible at the rather medial part of the LGB. (C) The low intensity lesion is visible at the rather medial part of the lateral geniculate body (arrow).

and rather middle (anterio-posterior dimension) part of the nucleus (Fig. 2). In case 2, as the main lesion was located rather anteriorly, at the one slice anterior to the section of posterior commissure (Fig. 4B), and was located more medially in the nucleus, compared with the case 1.

4.2. The vascular lesion in the LGB and visual field defect

Within each LGB, there is a deviation from the generalization that upper retinal fibers remain above and lower retinal fibers remain below throughout the visual pathways [1]. During the process of evolutionary development, the LGB has rotated 90°. Consequently, axons originating from inferior, central and superior retina are located laterally, centrally and medially, respectively, in the LGB. This intra-nuclear topography is based on primate's electrophysiological studies [1,2].

In case 1, it was evident that the lesion was limited to the lateral to the nucleus, injuring axons from the inferior fovea and resulting in the superior quadrantanopia. In case 2 who presented with the horizontal sectoranopia, the main lesion was present more medially compared with the lesion in case 1. Further radiological delineation of whether it is central or medial in the LGB was difficult in case 2. This is due to failure of direct visualization of the nucleus by use of IR MR imaging, probably due to complex structure of the LGB consisting of both gray and white matters [1,4]. Based on the characteristic visual field defect, it is speculated that the lesion is located at the central part of the LGB in case 2.

This study radiologically reveals that the axons originating from inferior retina are located more laterally than those from central retina in the human LGB.

Causes of the lesion were suspected to be infarction in both cases, although only MRA was carried out and revealed that no major trunk arteries were stenotic or occluded. The LGB has dual blood supplies both from anterior choroidal artery of internal carotid artery and lateral posterior choroidal artery of posterior cerebral artery [1,8–10]. The anterior choroidal artery usually supplies the medial and lateral aspects. Accordingly, ischemia of anterior choroidal artery causes superior homonymous quadrantanopia, inferior homonymous quadrantanopia or both [8–11]. Thus, in

case 1, a responsible vessel was considered to be anterior choroidal artery, in particular, twigs supplying the lateral part of the LGB. Lateral posterior choroidal artery supplies the remainder of the nucleus (essentially the central part of the LGB), which has projecting nerve fibers from the horizontal portion of the retina. Thus, the ischemic lesion of the artery produces wedge-shaped homonymous hemianopia or horizontal sectoranopia [8–11], which is the case in case 2. Partial involvement of the anterior central part of the nucleus may be responsible for the visual field defect, detectable only by automated perimetry.

In conclusion, this study radiologically reveals that the axons originating from inferior retina are located more laterally than those from central retina in the human LGB.

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