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Pure Homonymous Hemianopia due to Anterior Choroidal Artery Territory Infarction

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Key Words

Cerebrovascular disorders · Anterior choroidal artery · Hemianopia · Magnetic resonance imaging

Abstract

The most consistently observed neurological deficits in the anterior choroidal artery (AChA) territory infarction are pure motor or sensorimotor syndromes. Visual field defects and higher cortical dysfunction are occasionally accompanied, but pure homonymous hemianopia without motor and sensory symptom has never been reported yet. We present 2 patients with pure homonymous hemianopia, whose MRI disclosed cerebral infarction in the well-known territory of the AChA. In most patients with ischemic stroke, pure homonymous hemianopia indicates infarction in the posterior circulation, particularly in the posterior cerebral artery territory. However, the present cases provide evidence that it can also be caused by infarction in the anterior circulation, i.e. the AChA.

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The anterior choroidal artery (AChA) supplies structures that are crucial for motor, sensory and visual functions [1–4]. The most consistently observed neurological

deficits in the AChA infarction are hemiplegia and hemisensory loss [5–10]. Pure homonymous hemianopia without motor and sensory symptoms has not yet been reported. We report clinical and neuroimaging findings of 2 patients showing pure homonymous hemianopia due to the AChA territory infarction.

Case Reports

Patient 1

A 67-year-old man with a 20-year history of diabetes mellitus was admitted to hospital because of the acute-onset left visual field defect while watching television a few hours earlier. On admission, he was alert, and blood pressure was 130/80 mm Hg. Physical and neurological examinations were normal, except for left visual field defect. Visual acuity and color vision were intact. Laboratory tests were normal, except for high blood glucose level, 177 mg/dl. Perimetry disclosed a complete left homonymous hemianopia splitting macular vision with homonymous visual field defects in the uppermost portion of right visual field (fig. 1a). Brain magnetic resonance imaging (MRI) showed increased signal intensity in the region of the right lateral geniculate body (LGB) and the uncus of the temporal lobe (fig. 1b, c). Digital subtraction angiography (DSA) on day 5 revealed luminal irregularities in the cavernous portion of the right internal carotid artery with patent AChA. Transesophageal echocardiography showed ulcerative atheromatous plaques in the aortic arch. Full-dose heparin was given intravenously for 5 days, followed by oral antiplatelet therapy.

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Fig. 1. a Visual field tests using the Humphrey instrument in patient 1 showed complete left homonymous hemianopia. **b**, **c** Conventional T₂-weighted fluid attenuated inversion recovery (FLAIR) axial images in patient 1 showed increased signal intensities in right optic tract and lateral geniculate body.





Patient 2

A 63-year-old man (patient 2) with a 20-year history of diabetes mellitus was admitted to the hospital because of acute onset left visual field defect, which had developed 1 day earlier. On admission, his blood pressure was 140/80 mm Hg. Physical and neurological examinations were normal, except for left visual field defect. Perimetry revealed a complete left homonymous hemianopia splitting ma-

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cular vision (fig. 1d). Brain MRI disclosed a high signal intensity lesion in the area of the right LGB and the uncus of the temporal lobe (fig. 1e, f). No vascular lesions were observed on magnetic resonance angiography.



Fig. 1. d Visual field tests in patient 2 showed complete left homonymous hemianopia. **e**, **f** Conventional T_2 -weighted FLAIR axial images in patient 2 showed increased signal intensities in the right optic tract and lateral geniculate body.





Discussion

The diagnosis of AChA territory infarction has been made according to brain CT or MRI findings [5, 7, 8, 10] because most patients showed normal angiogram with patent AChA [7, 8]. According to known templates of cerebral vascular territories [2–4], the location of infarcts on MRI in our patients, the uncus of the temporal lobe and LGB, was clearly within the AChA territory. Thus, the negative angiogram did not limit the diagnosis of AChA infarction in our patients.

Pure Homonymous Hemianopia

Among 80 patients described in recent papers reviewing AChA territory infarction [5-9], hemiplegia and hemisensory deficits were observed in 76 (95%) and 47 (59%) patients, respectively, while only 16 (20%) patients showed visual field defects. Hupperts et al. [10] also reported that, among their 77 patients with AChA territory infarction, 56 patients (73%) had either pure motor or sensorimotor syndrome, but did not mention the frequency of visual field defects. Some patients also showed dysfunctions of higher cortical modalities such as anosognosia, visuospatial neglect, aphasia, and so on, depending on the side of the lesion [5-10]. With detailed neurological examination, our patients had no defects in motor and sensory functions, but only showed homonymous hemianopia. Sometimes, sensory symptoms of ischemic stroke are transient lasting only a few days or limited to paresthesia, but the short time delay between the onset of symptom and neurological examination in our patients (less than one day) could be enough to exclude transient sensory involvement. Although we did not perform a detailed neuropsychological assessment, higher cortical dysfunction related to AChA territory infarction could be excluded by bedside check of mental function, which was carefully and repeatedly conducted. With an extensive Medline search, we could not find any reported case with AChA territory infarction presenting pure homonymous hemianopia.

The AChA and its branches constantly supply the optic tract, lateral part of the LGB, posterior two thirds of the posterior limb of the internal capsule (PLIC), most of the globus pallidus, the origin of the optic radiation and the middle one third of the cerebral peduncle [1]. However, most AChA territory infarcts, even in patients with total obstruction [9], involved only a part of these structures because of individual anatomical variation and relatively rich collaterals from near arteries. The posterior two thirds of the PLIC has been the most consistently involved area in AChA infarction at autopsy studies [6], which appears to be the reason why hemiplegia is the most frequently observed deficit. The infarctions in our patients were located in the uncus of the temporal lobe and the LGB, but avoided the PLIC. The sparing of the PLIC appeared to free these patients from hemiplegia.

The AChA supplies visual fibers at three separate locations; the optic tract, the origin of the geniculocalcarine tract and the LGB [6]. Visual field defects related to AChA territory infarction are variable and may be an upper quadrantanopia [7, 11] sectoranopia [11], or homonymous hemianopia with or without macular sparing [5-7, 11]. Since the LGB lesions are known to produce a unique syndrome, named quadruple sectoranopia [11], homonymous hemianopia observed in our patients appeared to result from either the posterior optic tract or the early optic radiation involvement rather than pure LGB lesion. Since quadruple sectoranopia sometimes crossed the midline in the upper or lower peripheral portion [11], visual field defects shown in patient 1 suggested that ischemic lesion involved LGB as well as either the posterior optic tract or the early optic radiation, i.e. complete hemianopia plus quadruple sectoranopia. In most patients with ischemic stroke, pure homonymous hemianopia indicates infarction in the posterior circulation, particularly in the posterior cerebral arterial territory. However, our patients provide evidence that it can also be caused by infarction within the anterior circulation, i.e the AChA.

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