

HOMONYMOUS CENTRAL QUADRANTANOPIA CAUSED BY AN EXTRASTRIATE (V2/V3) INFARCTION: A CASE REPORT

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A 66-year-old Taiwanese aboriginal male had complained of right-side blurred vision for 2 months, especially when reading. He had a 10-year history of hypertension and cardiovascular disease. His best-corrected visual acuity was 20/25 in each eye. Ophthalmoscopy revealed asymmetrical cupping, but a normal disc. Humphrey perimetry showed an upper homonymous paracentral quadrantanopic defect. Brain magnetic resonance imaging showed an infarction in the left lower calcarine area over the extrastriate (V2/V3) cortical area and a narrowing of the left middle and posterior cerebral arteries due to severe arteriosclerosis.

Key Words: calcarine fissure, extrastriate cortex, magnetic resonance image, quadrantanopia

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The striate cortex extends along the calcarine fissure on the medial surface of the occipital lobe. The striate cortex also extends anteriorly from the junction between the calcarine fissure and the parieto-occipital sulcus to the occipital pole [1]. The first case of quadrantanopia was reported by Hun in 1887 [2]. One century ago, Inouye reported the first retinotopic map of the striate cortex based on his observations of soldiers with brain injuries during the 1904–1905 Russo-Japanese War. Inouye proposed that the vertical meridian was represented along the margin of the calcarine lips and the horizontal meridian was at the base of the calcarine fissure [3]. Holmes reported a similar map a decade later, and presented clearly the topographic characteristics of the contralateral hemifield

of vision in the striate cortex [4]. According to Holmes' proposed retinotopic organization, a V1-based visual deficit restricted to one quadrant of the visual field (that is, a homonymous quadrantanopia) with a clean border along the horizontal meridian would be the result of a lesion that included either the upper or lower lip of the calcarine sulcus extending precisely to, but not beyond, the horizontal meridian [4]. The upper lip of the calcarine fissure represents the lower visual fields and the lower lip represents the upper visual fields [1]. Logically, Holmes' statement was right and is well accepted, but practically, it is not easy to imagine how such a lesion can sharply respect the winding nature of the calcarine fissure and result in homonymous quadrantanopia.

Based on magnetic resonance imaging (MRI) evidence, Horton and Hoyt proposed another theory of cortically-based homonymous quadrantanopia, whereby the lesion occurs in the extrastriate cortex, rather than in the striate cortex. They reported that an early extrastriate visual area within a hemisphere, such as V2, V3 or V4v, represents a single quadrant of the visual field [5,6]. In animal studies on monkeys, the extrastriate visual striate cortex (V2) completely surrounds



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the primary striate cortex (V1), except for the anterior part, where the temporal crescent is located. The representation of the vertical meridian is located between V1 and V2. Extrastriate visual area 3 (V3) is an area around V1 and V2 that forms a horizontal meridian with V2 and the vertical meridian delimiting the outer border of V3. Thus, V2 becomes a mirror image of V1, and shares the representation of the vertical meridian; V3 is a mirror image of V2 and shares the representation of the horizontal meridian; furthermore, the outer perimeter of V3 is defined by the vertical meridian [7].

Horton and Hoyt proposed that, in humans, the horizontal and vertical meridians are represented in not only the V1 area, but also in V2 and V3 [5,6]. This proposal has been further supported by another report that showed a positive correlation between anatomic location based on functional MRI and the model proposed by Horton and Hoyt [8].

In this paper, we report a case with a unique homonymous central quadrantanopic defect due to an occipital lobe infarction for which, based on MRI studies, the damage was mainly confined to a V2/V3 extrastriate lesion.

CASE PRESENTATION

The patient was a 66-year-old Taiwanese aboriginal man with hypertension and hyperlipidemia who had received an operation for a coronary artery bypass

graft due to severe triple vessel disease. The patient had suffered from blurred vision in the right eye for about 2 months. The blurring condition had persisted, and was affecting his reading ability. On ocular examination, visual acuity was 20/25 in both eyes with best correction. Slit lamp biomicroscopy revealed a mild cataract and a silent anterior segment in each eye. The intraocular pressures were 17 mmHg OD and 16 mmHg OS. The pupils were equal without any relative afferent papillary defect. Fundoscopy revealed slightly enlarged cupping, but a normal neuro-rim. On examination, the visual field was found to be grossly intact by the confrontation test, but analysis using Humphrey perimetry with a central 30–2 threshold program revealed a right upper homonymous central quadrantanopia with macula splitting; this respected the horizontal and vertical meridians and was limited to the central 10 degrees in both eyes. The basic data in the visual field report were: in both eyes, fixation losses 0/17; false POS errors 0%; false NEG errors 0%; mean deviation -5.38 dB (OD) and -3.74 dB (OS) and PSD 4.11 dB (OD) and 4.62 dB (OS) (Figure 1).

After the tentative diagnosis of a left occipital lobe lesion, the patient was subjected to brain MRI, which revealed an infarction of the occipital lobe with the lesion being 21 mm in length, 19 mm in width and 12 mm thick. The lesion was located in the V2/V3 cortical area below the lower lip of the calcarine sulcus (V1) and adjacent to the posterior occipital pole.

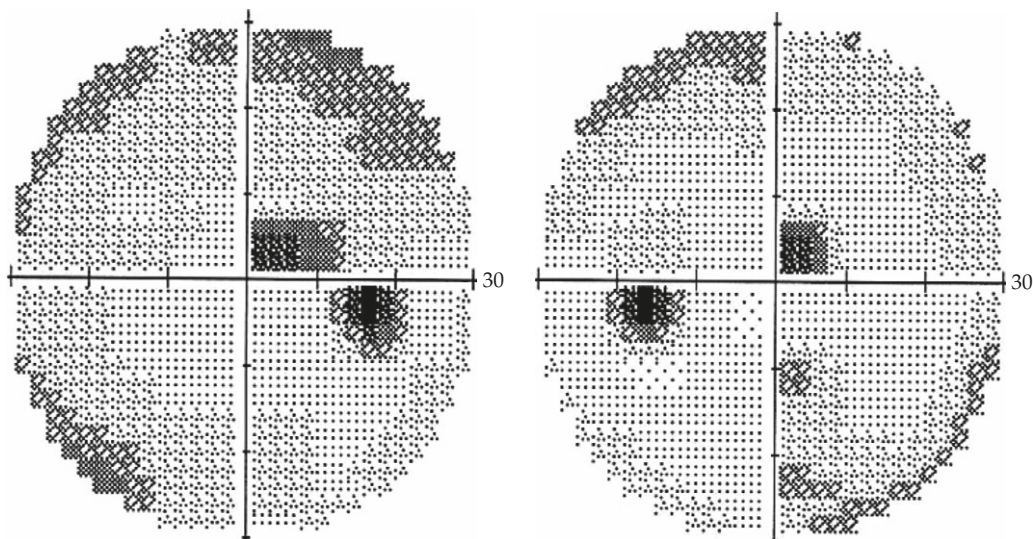


Figure 1. Humphrey perimetry (central 30–2 threshold program) revealed a right-upper homonymous scotomatous defect of 10° with macula splitting that respected the horizontal and vertical meridians.

Brain magnetic resonance angiography (MRA) also showed a narrowing of the left middle and posterior cerebral arteries due to severe arteriosclerosis (Figure 2). Although the patient did not have other obvious neurologic signs, he was referred to the neurological department for further medical care of the stroke.

DISCUSSION

The striate cortex can be further classified into anterior, intermediate and posterior parts. The anterior part lies adjacent to the parieto-occipital fissure and reflects the temporal crescent of the contralateral visual field. The posterior lesions are located in the

posterior 50–60% of the striate cortex, which includes the occipital pole and operculum and represent the central macula area of vision [9].

Over time, the Holmes' map has been modified to reflect reality, with the foveal region occupying more striate cortex of the occipital lobe than the peripheral area. In macaque monkeys, the central 15° of vision fills about 70% of the total surface area of the striate cortex. In human studies, 25% of the surface area of striate cortex presents the central 15° [3–5]. Another study has reported that the central 10° of the visual field is represented in humans by at least 50–60% of the posterior striate cortex [10]. Our patient's visual field defect also showed a characteristic magnification of the macula map in the striate cortex. The infarcted

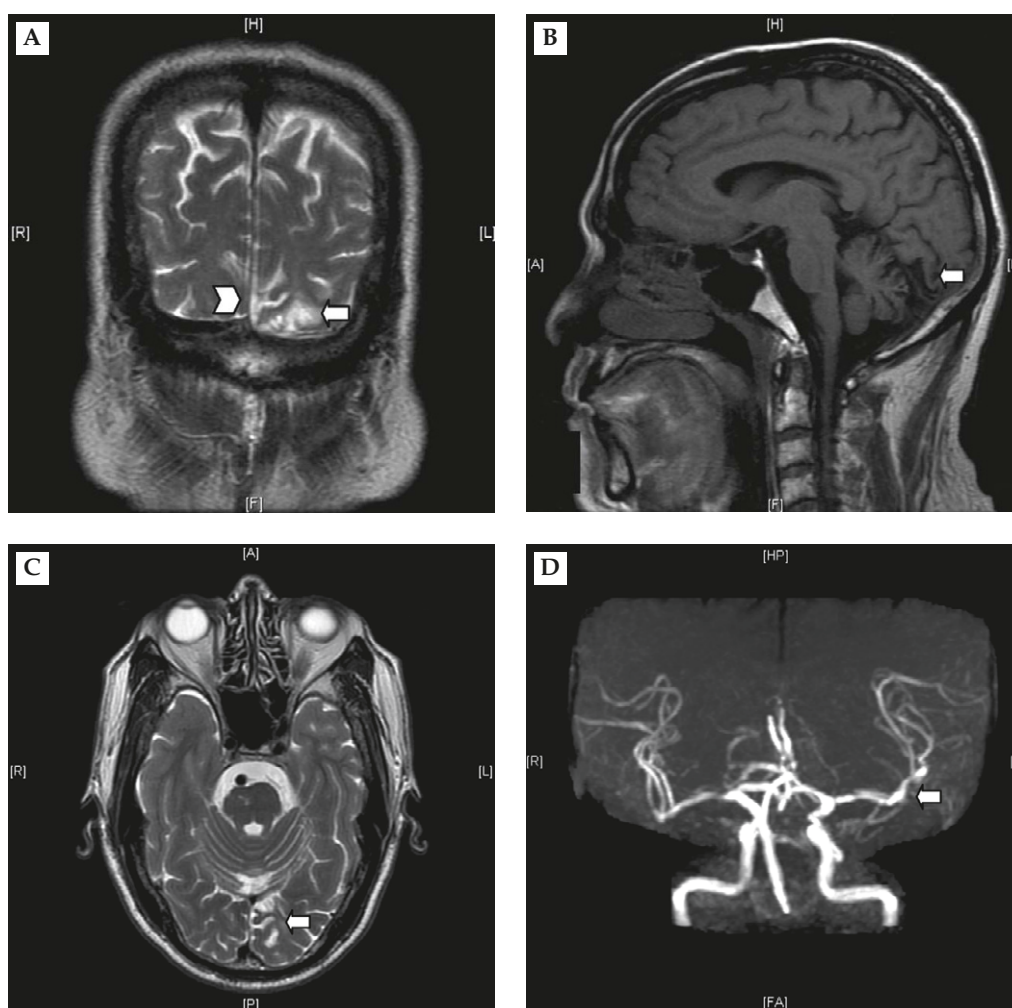


Figure 2. (A) T2-weighted coronal magnetic resonance imaging (MRI) documents an area of infarction (arrow) below the lower lip of the left calcarine sulcus (arrowhead) with V1 relatively spared. (B) T2-FLARE image of the left parasagittal sections shows an infarct (arrow) below the lower bank of the calcarine sulcus, specifically an extrastriate lesion (V2/3). (C) T2-weighted axial MRI shows an increased signal (arrow) in the left calcarine bank along the interhemispheric fissure. (D) Brain magnetic resonance angiography shows a narrowing of the left middle and posterior cerebral arteries due to severe arteriosclerosis.

lesion measured 21 mm in length (about half the length of the calcarine fissure) in the MRI images, but the quadrantanopia defect affected only 10° of the central visual field.

Daniel and Whitteridge invented the term “linear magnification factor” to refer to the millimeters of cortex representing 1° of visual field at any given eccentricity (E). They also reported a ratio of more than 40:1 between the fovea and periphery (60° eccentricity). An advanced formula covering the visual field coordinates of a scotoma will provide the size in millimeters squared of the corresponding cortical lesion, which for humans is $M_{\text{area}} = 300 / (E + 0.75)^2$ [11]. According to the formula, a lesion with an area of 2.59 cm² should be found in the cortex of our patient. Based on the MRI image obtained in this study, the area of the defect was about 2.5 cm².

The posterior pole of the occipital lobe is supposed to have a dual blood supply with collateral circulation from the middle and posterior cerebral arteries; this has been proposed to be a macula-sparing phenomenon and can be observed in cases with a small infarct lesion and without mass compression. Macula-sparing types of occipital lobe lesions will occur when a lesion is located at the occipital pole and operculum, because the occipital pole and operculum are supplied by the posterior temporal or parieto-occipital branch of the posterior cerebral artery, or an occipital branch of the middle cerebral artery; the presence of such collateral circulation will preserve the function of the occipital pole. However, in 50% of normal brains, the entire striate cortex, including the occipital pole, is supplied by the calcarine branch of the posterior cerebral artery, and macula splitting occurs after a vascular infarct in such patients [9]. In our patient, MRA demonstrated severe narrowing of the middle cerebral artery and the posterior cerebral artery, and these factors may have added to the ischemic change induced in the occipital pole and delayed re-perfusion from the collateral circulation. The presence of these factors might explain the macula-splitting character of the visual field defect in our patient.

The lower striate cortex is more vulnerable to ischemic damage. The characteristic vascular anatomy and poor development of the collateral circulation in the lower cortical area may explain this area's vulnerability to infarcts. The mechanisms underlying such infarcts include embolism from cardiac disease and plaques in the vertebrobasilar system [5].

Wong and Sharpe compared the accuracy of manual kinetic (tangent screen and the Goldmann perimeter) and automated static perimetry (Humphrey perimeter) among patients with occipital lobe lesions [12]. After comparing the results with those from MRI, all three perimetric techniques were found to be satisfactory screening tests for the detection of occipital lesions. They also proposed that the tangent screen and Goldmann perimetry approaches provided more information about the location and extent of lesions in the postgeniculate visual pathway than the Humphrey perimeter method [12]. However, clinically, the Humphrey perimeter approach is still the most common, general, fast and reliable screening tool used for the visual pathway. Furthermore, it is not easy to detect a homonymous paracentral quadrantanopic defect as occurred in our patient using the confrontation method only. Thus, autoperimetry still has value when detecting a specific pattern of defect.

In a large survey of homonymous hemianopias, homonymous scotomatous defects, such as homonymous central quadrantanopia with macula splitting, made up 12% of the total number of events. The main lesion sites for a homonymous scotomatous defect were the occipital lobe (50%), optic radiation (30%), and the optic tract (10%) [13]. However, a superior homonymous quadrantanopia caused by a temporal lobe lesion with damage to Meyer's loop would induce an optic radiationally-filled defect. The characteristics of such a visual field defect would be incongruous with a sloping border and a failure to respect precisely the horizontal meridian. Homonymous quadrantanopia respecting the horizontal meridian ought to indicate striate or extrastriate cortical disease [10]. The precise MRI images of the coronal and sagittal sections obtained in this case provided better spatial resolution than a computed tomography scan, and this allowed better correlation with the visual field defect [14,15].

An infarct of the extrastriate cortex over the V2/V3 area would result in a homonymous central quadrantanopia. Our MRI studies provide evidence of a V2/V3 lesion with relative sparing of V1. The present homonymous central quadrantanopic defect is relatively small, but it has affected reading ability. Variation in the vessels supplying the visual cortex across the posterior occipital lobe may contribute to the macula-splitting phenomenon in our patient.

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第二及第三皮質病灶引致的同側中心象限盲 — 病例報告

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一位六十六歲原住民男性主訴近兩個月以來右眼視力模糊，尤其是在閱讀時。病患有高血壓及心臟病約十年之久，兩眼最佳矯正視力均為零點八，直接眼底鏡檢查發現兩側視神經盤之視杯大小不對稱，視野檢查發現有雙眼同側中心上象限盲，其來源由腦部核磁共振影像推測為左大腦枕葉距狀溝下方之第二及第三皮質處因動脈粥狀硬化所引致之中及後大腦動脈狹窄所造成之梗塞。

關鍵詞：距狀溝，紋外皮質，核磁共振造影，象限偏盲

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