

Case Series: Bilateral Homonymous Visual Field Defects Due to Bilateral Ischemic Strokes

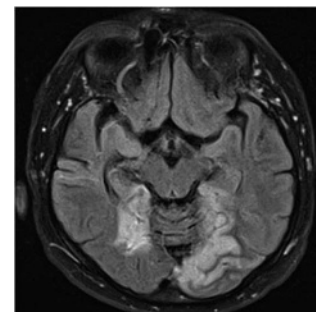
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SIGNIFICANCE: Bilateral strokes are rare and should be considered when patients present with bilateral visual field loss characterized by patterns consistent with right and left-sided homonymous visual field defects. Perimetry, dilated funduscopy, and immediate neuroimaging are mandatory for diagnosis, because patients may present with vague symptoms. These cases reflect the retinotopic features of the striate cortex.

PURPOSE: The purposes of this study were to describe the unusual presentation of bilateral homonymous visual field defects in three patients with bilateral ischemic strokes and to discuss the clinical and neuroanatomical correlations.

CASE REPORTS: Neuro-ophthalmological examination including perimetry and brain magnetic resonance imaging (MRI) was performed in three patients with bilateral homonymous scotomas. Two of three patients presented with superior altitudinal hemianopia, resulting from right and left homonymous superior quadrantanopia due to bilateral occipital strokes below the calcarine fissure. A 57-year-old man (patient 1) with a history of atrial fibrillation presented with driving difficulties. Perimetry revealed bilateral superior altitudinal hemianopia. Brain MRI demonstrated a subacute right occipital stroke and a chronic left occipital stroke, both inferior to the calcarine fissure. An 83-year-old woman (patient 2) presented with reading disorders. Perimetry showed a left incomplete homonymous hemianopia and a right horizontal wedge-shaped homonymous scotoma. Brain MRI showed a chronic ischemic stroke in the left occipital lobe and acute ischemia in the right thalamus. A 40-year-old man (patient 3) was referred with headache, disorientation, and bilateral blurry vision. Perimetry showed bilateral superior altitudinal hemianopia, and MRI demonstrated acute bilateral occipital ischemia. Patients 1 and 2 suffered sequential bilateral strokes and were not aware of the initial scotoma, whereas patient 3 presented with bilateral concurrent strokes.

CONCLUSIONS: Bilateral homonymous visual field defects due to bilateral strokes are rare. Patient history, a careful neuro-ophthalmological examination, and correlation of visual field defect patterns with neuroimaging should prompt the clinician to the presence of this unique entity.



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Field defects respecting the vertical midline indicate chiasmal or retrochiasmal brain damage, with the occipital lobe being the most common lesion location and stroke being the most common etiology.¹ Unilateral damage to the retrochiasmal visual pathway results in homonymous visual field defects, which affect corresponding areas of the contralateral visual field in both eyes and are found in approximately 8% of stroke patients and 25% of traumatic brain injury patients.² Bilateral damage to the retrochiasmal visual pathway causes bilateral homonymous scotomas, which affect both visual hemifields in both eyes.¹ Depending on the degree and the affected site of the optic pathway, various clinical types of bilateral homonymous visual field defects have been described, which are rare and have been infrequently reported.^{3–12} A previous study has reported that only 6% in a large group of patients with homonymous hemianopias had bilateral visual field loss, and up-to-date most published studies represent single case reports.¹ There have been eight case reports of bilateral superior altitudinal hemianopia caused by occipital lobe lesions³; however, the extent of the lesions inside the visual cortex was not always evaluated in those previously reported patients.³ Similarly, in an older report of 15 patients with bilateral homonymous hemianopia, neuroimaging was performed in only five patients.⁴ There are also some additional sporadic case reports of altitudinal hemianopia,

resulting from a combination of bilateral superior or inferior homonymous quadrantanopia.^{5–7}

We here report detailed clinical and radiological findings of three additional cases of bilateral homonymous visual field defects, two patients presenting with superior altitudinal hemianopia, resulting from a combination of right and left superior homonymous quadrantanopia, and a third one with bilateral incomplete homonymous hemianopia. Based on magnetic resonance imaging findings, we evaluated the extent of the lesions affecting the visual pathway and discussed the correlation between the location of the lesions and visual field defects in our patients. The clinical and neuroanatomical correlations in those rare cases aim to further enhance our understanding of the retinotopic features of the striate cortex.

METHODS

Three patients presented with acute visual problems, all of which ultimately represented the same underlying cause. All patients underwent neuro-ophthalmological examination, including best-corrected visual acuity, intraocular pressure, slit-lamp biomicroscopy, and both direct and indirect funduscopy. Visual fields were assessed with standard automated static perimetry,

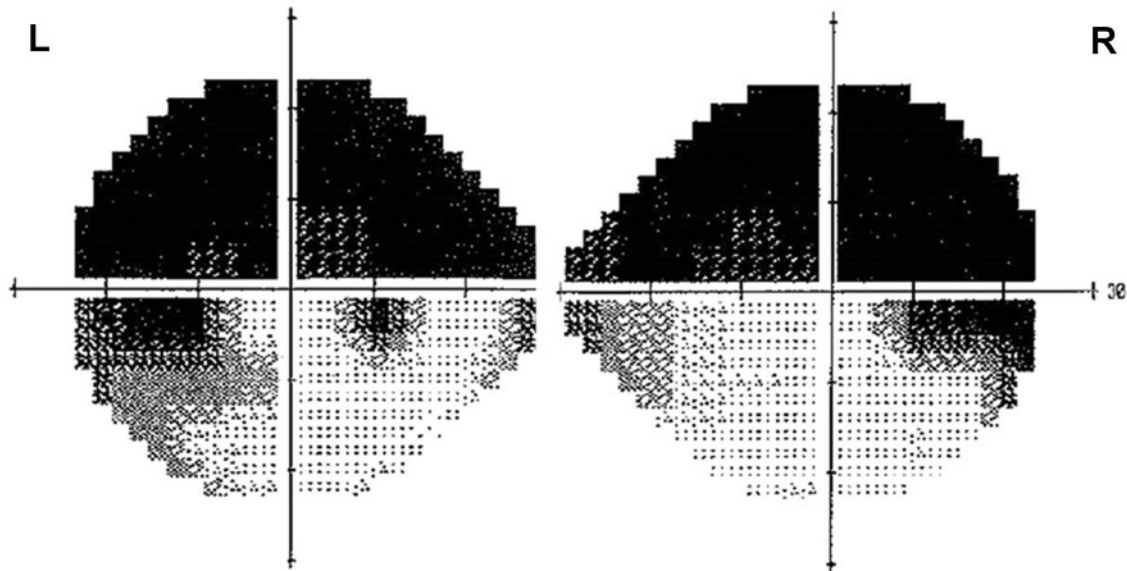


FIGURE 1. Humphrey 24-2 static automated perimetry of patient 1. There is bilateral superior homonymous quadrantanopia, which resembles superior altitudinal visual field defects.

using the central 24-2 and central 30-2 threshold test of the Humphrey field analyzer (Humphrey Field Analyzer, 24-2 and 30-2 Test Protocol; Carl Zeiss Meditec, Dublin, CA). In addition, magnetic resonance imaging with gadolinium was performed. All patients underwent neuropsychological testing for visual hemineglect, visual object agnosia, simultanagnosia, and prosopagnosia. No evidence of visual agnosias was found. The study was performed according to the Declaration of Helsinki. No identifiable health information was included in this case series.

RESULTS

Patient 1

A 57-year-old man presented with driving difficulties for the past 2 days. His medical history was remarkable for atrial fibrillation; he had suffered an ischemic stroke 6 months prior and had been on antihypertensives and aspirin since then. He was not aware of any previous ophthalmological abnormalities and had not seen an eye care practitioner for the past 2 years. On examination, the patient's best-corrected visual acuity was 20/20 in both eyes, with normal ocular motility and normal color vision. Confrontation visual field testing revealed bilateral superior visual field defects, which was confirmed with Humphrey 24-2 automated perimetry as bilateral superior altitudinal hemianopia that respected the horizontal midline (Fig. 1). Slit-lamp biomicroscopy and dilated funduscopy did not reveal any pathological findings. In particular, the optic discs appeared pink with sharp margins and a cup-to-disc ratio of approximately 0.3. No optic disc pallor, edema, hemorrhage, neuroretinal rim damage, or changes of the peripapillary region, which could imply glaucomatous or ischemic optic nerve damage, were noted.

Fluid-attenuated inversion recovery magnetic resonance imaging demonstrated bilateral occipital ischemic strokes inferior to the calcarine fissure, corresponding to bilateral superior homonymous quadrantanopia that respected the horizontal midline and led to bilateral altitudinal field loss (Fig. 2). There was a subacute

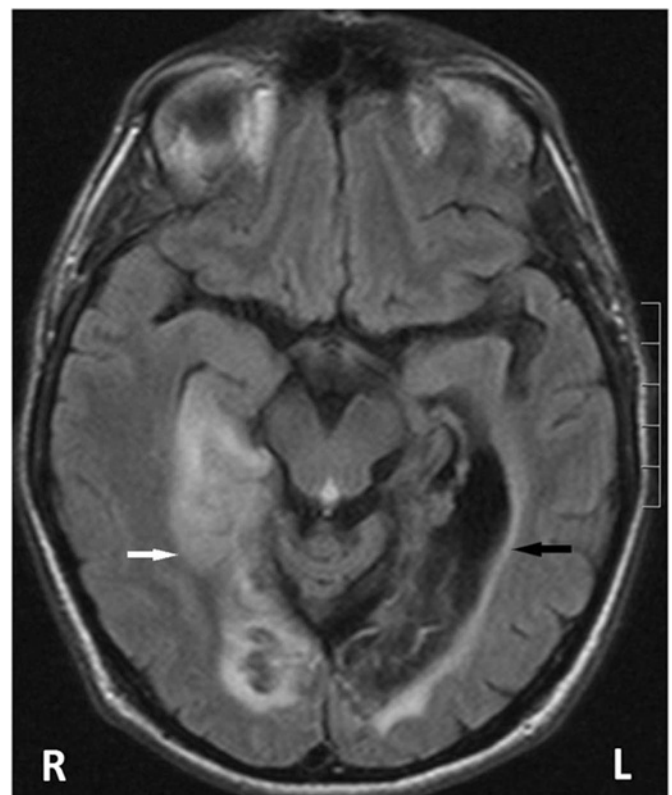


FIGURE 2. Axial fluid-attenuated inversion recovery magnetic resonance imaging of patient 1. There are bilateral lesions of the inferior occipital lobe. There is a subacute right posterior cerebral artery territory infarct involving the right posterior temporal lobe and occipital cortex (white arrow) and an old left posterior cerebral artery territory infarct (black arrow).

right posterior cerebral artery territory infarct involving the right medial/posterior temporal lobe, occipital cortex, and pulvinar of the right thalamus, with a small amount of parenchymal hemorrhagic transformation (Fig. 2, white arrow). The infarct showed mild enhancement after gadolinium injection, which was also suggestive of a subacute stroke. In addition, there was a mature previous left posterior cerebral artery territory infarct with *ex vacuo* expansion of the occipital horn of the left lateral ventricle (Fig. 2, black arrow). The patient was admitted to the hospital for cardiac and neurologic monitoring and was started on dipyridamole and atorvastatin.

Patient 2

An 83-year-old woman presented with a 24-hour history of reading disorders and problems in finding the beginning of lines. She had a history of a cerebral stroke 1 year ago, and her medical history was remarkable for diabetes mellitus, hypertension, and hyperlipidemia. Slit-lamp examination and dilated funduscopy were normal. Humphrey automated static 24-2 perimetry showed a left incomplete incongruous homonymous hemianopia and a right horizontal homonymous sectoranopia with macular sparing (Fig. 3). T2-weighted and fluid-attenuated inversion recovery magnetic resonance imaging showed an acute ischemic infarction of the right thalamus (Fig. 4A, white arrow) and an old infarct of the left occipital lobe (T2 and fluid-attenuated inversion recovery magnetic resonance imaging, Fig. 4A, black arrow, and Fig. 4B). Post-contrast magnetic resonance imaging showed a mild peripheral enhancement in the right thalamus consistent with the acute infarction. The patient was started on oral metformin, chlorthalidone, benazepril, clopidogrel, and atorvastatin under regular cardiological evaluations.

Patient 3

A 40-year-old man was referred with a 24-hour history of headache, disorientation, loss of short-term memory, and bilateral blurry vision. His previous medical history was unremarkable. Slit-lamp biomicroscopy and dilated funduscopy were unremarkable, but Humphrey automated static 30-2 perimetry showed bilateral

superior altitudinal hemianopia, as a result of right and left superior homonymous quadrantanopia (Fig. 5). T2-weighted magnetic resonance imaging demonstrated acute bilateral ischemic infarctions in the posterior cerebral artery territory below the calcarine fissure (Figs. 6A, B), leading to bilateral superior homonymous quadrantanopia. Post-contrast magnetic resonance imaging showed gyral enhancement of the lesions bilaterally, suggestive of acute ischemia. Because of his young age, the patient was referred for cardiological evaluation. Carotid and vertebral artery Doppler ultrasonography scans were normal, as was transesophageal echocardiography, but Holter monitoring revealed premature ventricular and supraventricular contractions, and the patient was started on oral clopidogrel (75 mg) daily. Thrombophilia screening and autoantibody testing including anticardiolipin antibodies were within normal limits.

DISCUSSION

Typically, unilateral lesions of the post-chiasmatic visual pathway result in contralateral homonymous hemianopia or quadrantanopia. Bilateral post-chiasmatic lesions result in homonymous defects of both visual hemifields, as seen in the presented patients.

Bilateral homonymous scotomas are relatively rare and constitute approximately 6% of homonymous visual field defects.¹ Cerebrovascular accidents to the occipital lobe, such as infarction and, less commonly, hemorrhage, are the most common cause, followed by trauma and tumor.^{1,2} All three patients in the present report had cardiovascular risk factors: atrial fibrillation (patient 1), diabetes mellitus, hypertension, hyperlipidemia (patient 2), and cardiac arrhythmia (patient 3). Usually, bilateral infarctions occur sequentially, but in some cases, they can also occur simultaneously.^{1,2} Patients 1 and 2 had suffered consecutive bilateral infarctions, whereas patient 3 presented with simultaneous bilateral infarctions.

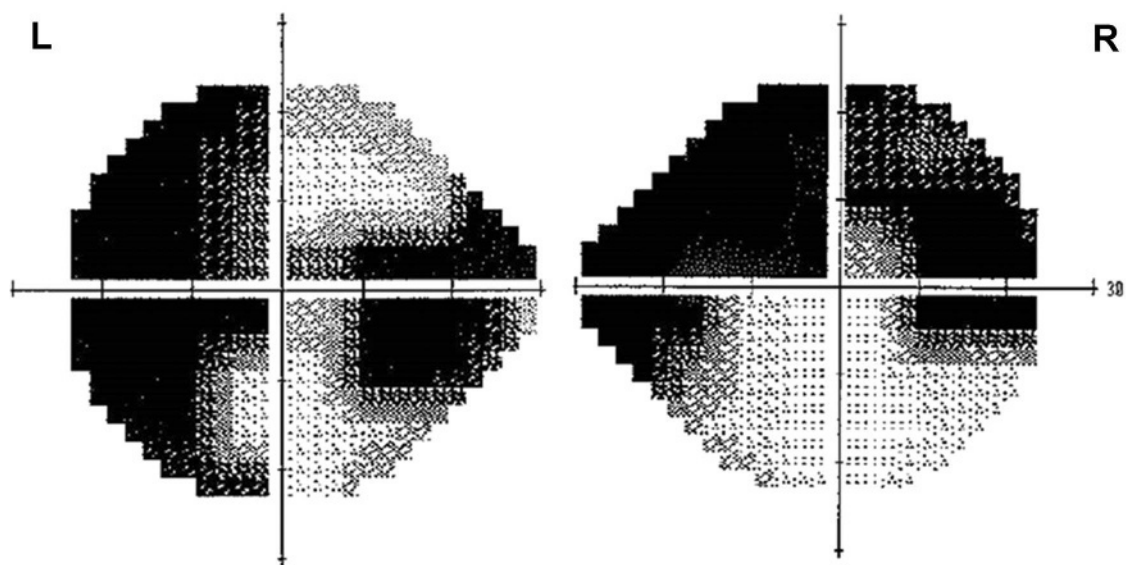


FIGURE 3. Humphrey 24-2 static automated perimetry of patient 2. Bilateral homonymous visual field defects, including a left incomplete homonymous hemianopia and a right horizontal wedge-shaped incongruous homonymous scotoma with macular sparing.

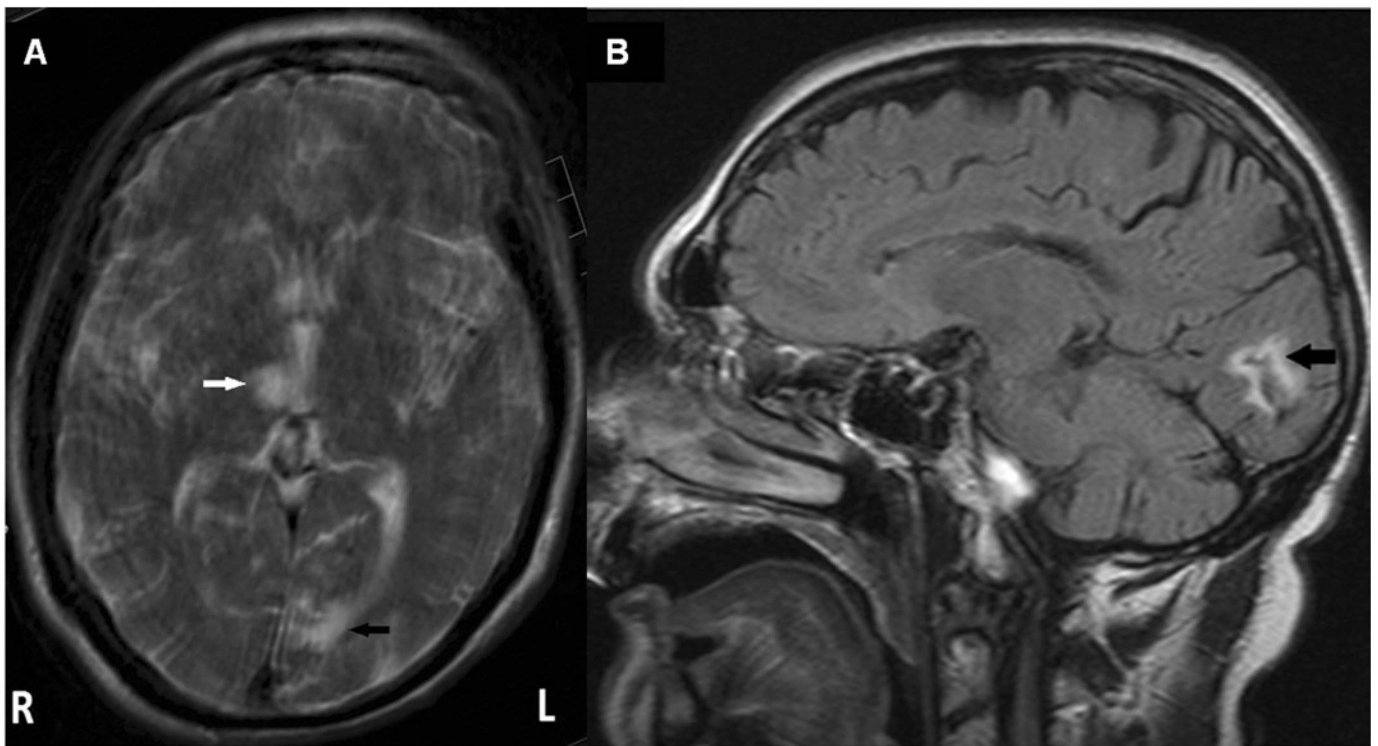


FIGURE 4. Brain magnetic resonance imaging of patient 2. (A) Axial T2 magnetic resonance imaging demonstrates an old left posterior cerebral artery infarct (black arrow) and an acute right paramedian thalamic infarction (white arrow). Blurring of the image is owing to motion artifacts. (B) Sagittal flair magnetic resonance imaging shows the old left posterior cerebral artery infarct (black arrow).

Patients 1 and 3 had bilateral lesions of the occipital lobe below the calcarine fissure, which led to bilateral superior altitudinal field loss from a combination of right and left homonymous superior quadrantanopia. According to the retinotopic organization described

by Horton and Hoyt,¹³ the primary visual cortex contains a topographic representation of the contralateral hemifield of vision. The upper and lower visual quadrants are represented in the lower and upper calcarine banks, respectively, separated by the horizontal

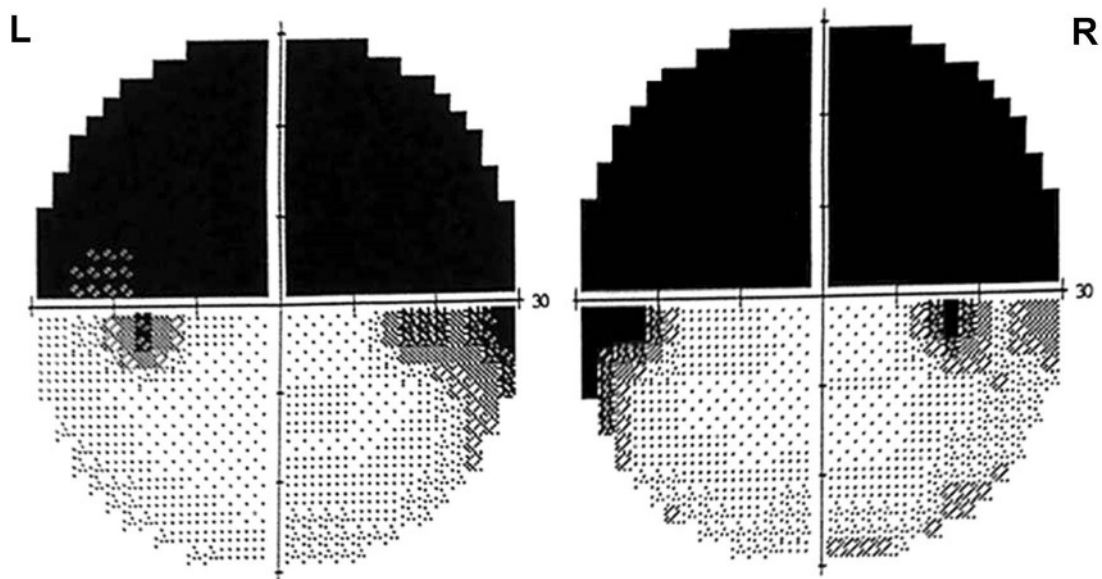


FIGURE 5. Humphrey automated static 30-2 perimetry of patient 3. There is bilateral superior altitudinal hemianopia resulting from bilateral superior homonymous quadrantanopia.

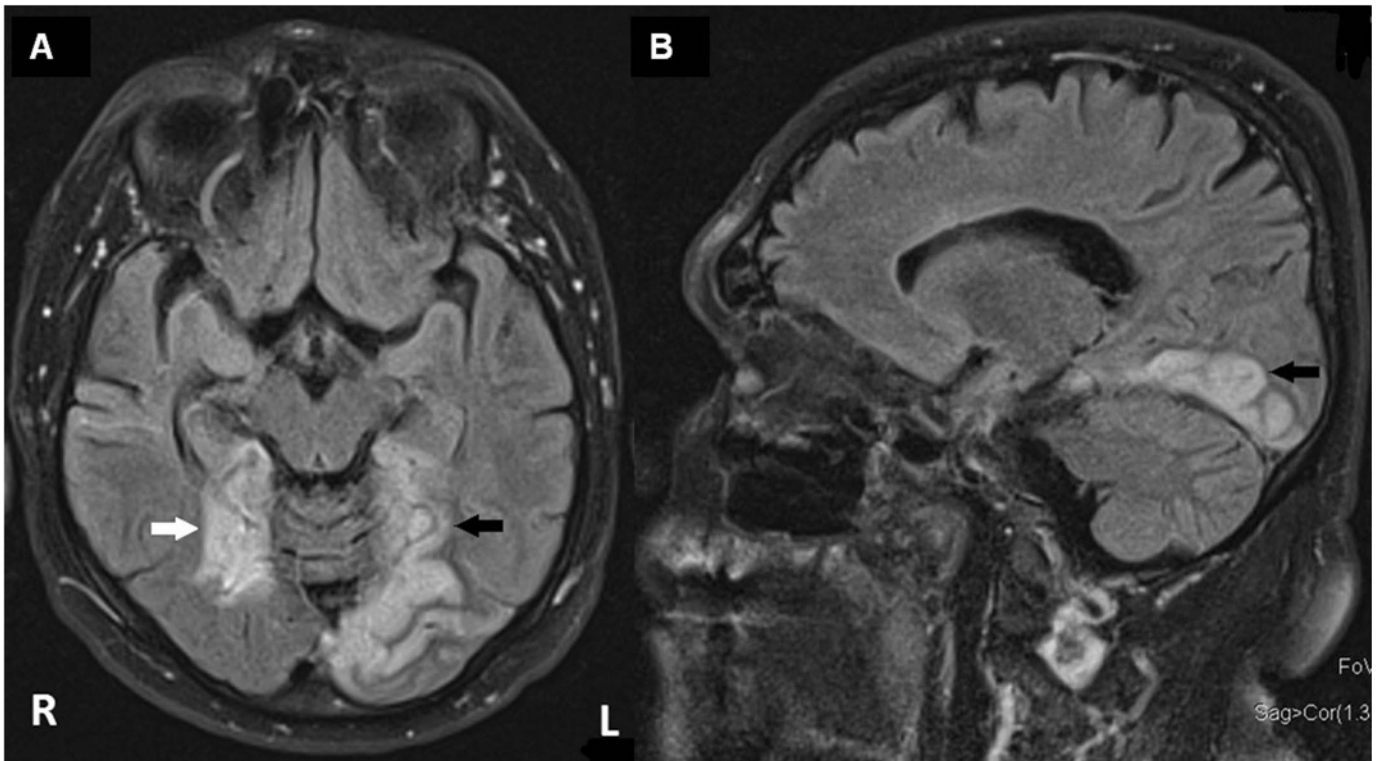


FIGURE 6. Brain magnetic resonance imaging of patient 3. (A) Axial T2 magnetic resonance imaging demonstrates acute bilateral ischemic infarctions in the posterior cerebral artery territory. (B) The infarctions are located below the calcarine fissure, as shown in sagittal T2 magnetic resonance imaging (black arrow).

meridian along the base of the calcarine fissure.¹³ The superior altitudinal hemianopia of patients 1 and 3 is, namely, a combination of a right homonymous superior quadrantanopia and a left homonymous superior quadrantanopia, resulting from occipital ischemic infarctions of the posterior cerebral artery inferior to the calcarine fissure.¹² Alternatively, if both lesions were located above the calcarine fissure, the result would be an inferior altitudinal hemianopia.

It is hypothesized that the lower striatal cortex is more sensitive to ischemia because the upper cortex has a more developed collateral circulation.¹ Rarely, bilateral superior altitudinal hemianopia has been reported in multiple sclerosis.¹ On the other hand, bilateral inferior altitudinal defects usually result from tumors and traumas, such as bullet wounds. Bilateral inferior altitudinal visual field defects have been also described after cardiac surgery and Creutzfeldt-Jakob disease.^{9,11} Finally, crossed homonymous quadrantanopias (“checkerboard” visual fields) present in lesions of the superior occipital lobe (above the calcarine fissure) on one side and the inferior occipital lobe (below the calcarine fissure) on the opposite side.¹ In summary, the acute onset, the good visual acuity, and the normal fundus findings in patients 1 and 3 suggest that the superior bilateral altitudinal hemianopia resulted from a combination of right and left superior homonymous quadrantanopia, which was caused by bilateral occipital infarctions. In addition, the high congruence of the visual field defects is consistent with a lesion of the occipital lobe or the posterior visual radiation and makes a pre-chiasmal or chiasmal cause unlikely.

Common pre-chiasmal causes of bilateral altitudinal hemianopias include bilateral anterior ischemic optic neuropathy and bilateral glaucomatous optic neuropathy, which are associated with abnormal

optic disc findings. The onset of arteritic or nonarteritic anterior ischemic optic neuropathy is usually unilateral, and the condition is accompanied by painless visual loss, optic disc edema, a relative afferent pupillary defect, and an altitudinal visual field defect (typically inferior).¹² In addition, in bilateral anterior ischemic optic neuropathy, the altitudinal inferior defects are usually asymmetrical. Bilateral symmetrical altitudinal hemianopia in bilateral anterior ischemic optic neuropathy or optic neuritis is extremely rare.⁸ Bilateral altitudinal visual field defects have been also reported in bilateral glaucoma, optic nerve head drusen, and tilted discs.^{14,15} Those conditions are not acute and can be diagnosed on the basis of pathological optic disc findings.

Compressive chiasmal lesions, that is, sellar and suprasellar tumors, arteriosclerotic internal carotid arteries, and aneurysms of the anterior cerebral artery, are rare but can also lead to bilateral, inferior or superior, altitudinal hemianopias.¹² Chiasmal lesions are typically associated with asymmetrical visual acuity loss, optic atrophy, and a relative afferent pupillary defect.⁸ Bilateral occipital lesions can also lead to reduced visual acuity, which is, however, symmetrical. In addition, in those cases (post-geniculate lesions), the fundoscopic findings are normal, and there is usually no relative afferent pupillary defect.⁹

Optical coherence tomography imaging can be useful as a supplementary diagnostic tool for assessing changes in the ganglion cell layer–inner plexiform layer complex and the retinal nerve fiber layer due to intracranial lesions at different locations within the visual pathway.^{16,17} A recent study of patients with intracranial lesions affecting the optic chiasm or the optic tract and/or lateral geniculate nucleus showed that clinical abnormalities in those patients are more likely to demonstrate abnormalities of ganglion cell

layer–inner plexiform layer complex than global peripapillary retinal nerve fiber layer thickness and that papillomacular bundle thickness measurement appears to be a valuable tool to detect abnormalities of the anterior visual pathways.¹⁶ Similar findings have been described in patients with retrogeniculate lesions in the posterior visual pathway, who showed significant thinning of the ganglion cell layer in the projecting sector of the retina mapping to the brain lesion.¹⁷

Patient 2 presented with a different pattern of bilateral homonymous scotomas, comprising a left incomplete homonymous hemianopia and a right horizontal wedge-shaped homonymous scotoma. The left incomplete incongruous homonymous hemianopia was attributed to an acute infarction of the right thalamus (Fig. 4A, white arrow). Visual field defects in cases of thalamic infarcts have been described rarely and mainly comprise incongruous homonymous hemianopias and complete homonymous hemianopias.¹ In addition, the right horizontal sectoranopia in patient 2 corresponded to an old left posterior cerebral artery infarction. Horizontal sectoranopias, which lie along the horizontal midline, are rare and are mainly encountered in lesions of the lateral geniculate nucleus¹; however, in patient 2, the underlying cause is damage to the intermediate portion of the occipital lobe in the region of the calcarine fissure, which has been described less frequently.² Patient 2 also had macular sparing to the right hemifield, which is associated with preservation of the occipital tip.¹³

Interestingly, patients 1 and 2 were not aware of the homonymous visual field defects after the initial stroke, and they presented to the ophthalmology department only after the second stroke, which caused further deterioration of their visual field. Except for the

visual symptoms, unilateral occipital lobe lesions are generally not associated with other neurological problems. Hence, patients are not always aware of the accompanying homonymous visual field defects until they suffer a second event in the contralateral hemisphere weeks to years later, causing a more pronounced visual impairment. On the other hand, patient 3 presented in the acute setting because he developed simultaneous bilateral infarctions leading to bilateral superior altitudinal hemianopia.

CONCLUSIONS

Bilateral cerebral infarcts should be considered in acute, symmetrical, bilateral visual field defects, especially in cases with preserved visual acuity and unremarkable fundus findings. These cases are rare and reflect the retinotopic features of the striate cortex. Patients may present with vague symptoms because homonymous visual field defects can be encountered as an isolated finding and do not always clearly point toward the underlying cerebral lesion. A thorough clinical examination including perimetry and dilated funduscopy will aid the clinician to promptly identify unexplained vision loss due to bilateral homonymous scotomas. Such dense visual field defects with no corresponding optic disc or retinal abnormalities should indicate a need for prompt neuroimaging, as they may indicate bilateral homonymous hemianopias. Efficient and rapid diagnosis of the exact type and cause of those rare bilateral homonymous hemianopias remains crucial in modern stroke therapy.

ARTICLE INFORMATION

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Drs. Tsironi and Asproudis contributed equally to this work.

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