Representation of the Visual Field in the Striate Cortex: Comparison of MR Findings with Visual Field Deficits in Organic Mercury Poisoning (Minamata Disease)

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PURPOSE: To compare MR imaging findings of the striate cortex with visual field deficits in patients with Minamata disease and to reestimate the classical Holmes retinotopic map by using the data obtained from comparing visual field abnormalities with degree of visual cortex atrophy. **METHODS:** MR imaging was performed in eight patients with Minamata disease who had been given a full neuroophthalmic examination, including Goldmann dynamic perimetry. The atrophic portions of the calcarine area were measured in the sagittal plane next to the midsagittal image and represented as a percentage of atrophy of the total length of the calcarine fissure. MR findings were compared with results of a visual field test. **RESULTS:** The visual field test revealed moderate to severe concentric constriction of the visual fields, with central vision ranging from 7° to 42° (mean, 19°). The ventral portion of the calcarine sulcus was significantly dilated on MR images in all patients. A logarithmic correlation was found between the visual field defect and the extent of dilatation of the calcarine fissure. The central 10° and 30° of vision seemed to fill about 20% and 50% of the total surface area of the calcarine cortex, respectively. **CONCLUSION:** Visual field deficits in patients with Minamata disease correlated well with MR findings of the striate cortex. Our data were consistent with the classical Holmes retinotopic map.

Index terms: Brain, magnetic resonance; Magnetic resonance, functional; Nervous system, diseases; Vision

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Minamata disease is a neurologic illness caused by ingestion of contaminated seafood (1, 2). Patients with severe cases have died after a subacute or chronic clinical course, although many patients have survived (3). Magnetic resonance (MR) images show lesions in the striate cortex, cerebellum, and postcentral gyri, which correlate with three of the characteristic clinical manifestations of the disease: constriction of visual field, ataxia, and sensory disturbance (3). The range of visual field in patients with Minamata disease might correlate with the degree and extent of atrophy in the striate cortex on MR images.

The familiar Holmes retinotopic map provides a detailed source of primary data concerning the representation of visual fields in the human striate cortex (4). In this map, the macula occupies the occipital pole, while the periphery of the visual field is located anteriorly. Holmes realized that the central retina, more densely cellular and specialized for best visual acuity, has a relatively expanded representation in the striate cortex.

The purpose of our study was to compare MR imaging findings of the striate cortex with visual field deficits in patients with Minamata disease and to reestimate the classical Holmes retinotopic map by using data obtained from a com-

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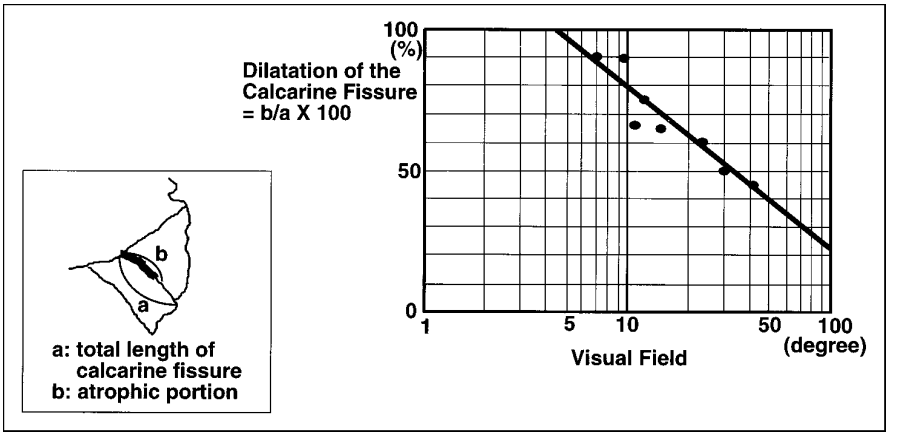

Fig 1. Relationship between the visual field and dilatation of the calcarine fissure.

parison of visual field abnormalities with degree of visual cortex atrophy.

Materials and Methods

MR imaging was performed in eight patients, five men and three women (46 to 59 years old; mean, 51 ± 5 years), with Minamata disease. All patients were initially affected between 1955 and 1958. Their clinical findings, including visual field test results, have been followed up periodically for about 30 years. All patients have had full neuroophthalmic examinations, including Goldmann dynamic perimetry.

The MR unit used was a 1.5-T superconductive type. T1-weighted images were obtained in axial, coronal, and sagittal projections using the spin-echo technique (500–600/13/2 [repetition time/echo time/excitations]), and T2-weighted images were obtained in axial and coronal sections also with the spin-echo technique (2000–3000/80/1). The section thickness was 5 mm, the field of view was 25 cm, and the matrix size was 192–256 × 256. The atrophic portions of the striate cortex were measured in the sagittal plane next to the midsagittal image and represented as percentage of atrophy of the total length of the calcarine fissure (Fig 1). Coronal and axial images were also used for confirmation of the extent of the lesion. The MR imaging findings were then compared with the patients' visual fields. Pearson's and Spearman's correlation coefficients were calculated between the visual field defects and the extent of dilatation of the calcarine fissure.

Results

The visual field test revealed moderate to severe concentric constriction of the visual fields, with central vision ranging from 7° to 42° (mean, 19°) from the point of fixation. In the most severe cases, the visual fields were identical with bilateral homonymous hemianopsia, with sparing of central vision (Fig 2).

The ventral portion of the calcarine fissures was significantly dilated on all MR images (Fig 2). T2-weighted images showed hyperintense lesions sparing the most posterior portion of the striate cortex. With more severe constriction of the visual fields, there was more posterior dilatation of the calcarine fissure. There was a logarithmic correlation between the visual field defects and the extent of dilatation of the calcarine fissure (Fig 1). The central 10° and 30° of vision seemed to fill about 20% and 50% of the total surface area of the striate cortex, respectively. Correlation coefficients were −.930 for Pearson's ($P = .0002$) and −.976 for Spearman's ($P = .0098$).

Discussion

The striate cortex is specifically affected in Minamata disease (1). Pathologic changes are more severe in the anterior part of both calcarine areas. The normal cortical striation of Gennari is usually destroyed. Disintegration and loss of neurons are the basic findings on microscopic examination, and, in moderate to severe cases, the neurons in the striate cortex almost always disappear entirely (status spongiosus) (1). On MR images, the calcarine fissures are significantly dilated, reflecting atrophy of the visual cortex. Sagittal MR studies clearly depict the location of calcarine involvement. Long T1 and T2 relaxation times within the calcarine cortex may be due to related status spongiosus (3). MR imaging well shows the vulnerability of the striate cortex to methyl mercury.

Representation of the visual field in the occipital striate cortex was delineated by Inouye (5) and subsequently by Holmes and Lister (6). Thereafter, Holmes devised his original scheme, which has gained widespread acceptance (4).

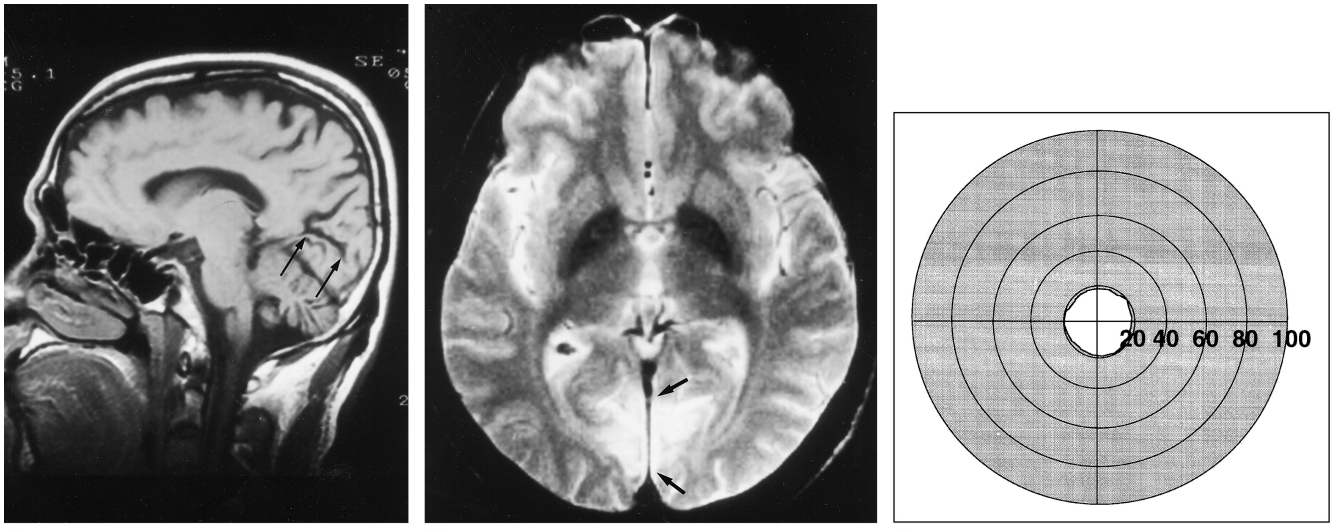

Fig 2. A 45-year-old man with Minamata disease (central vision, 20°).

A, T1-weighted sagittal MR image shows significant dilatation (*arrows*) of the ventral portion of the calcarine fissure.

B, T2-weighted axial MR image shows the hyperintense lesions with sparing of the most posterior portion of the striate cortex (*arrows*).

C, Visual fields of the patient as determined by Goldmann perimetry. The central 20° of the visual field was intact.

In the Holmes retinotopic map, about 25% of the surface area of the striate cortex is allocated to the central 15° of vision. The foveal representation is located at the occipital pole, where the striate cortex usually extends about 1 cm onto the lateral convexity of the occipital lobe. The extreme periphery of the visual field is represented anteriorly, at the junction of the calcarine and parietooccipital fissures.

After the introduction of computed tomography (CT) of the brain, the accuracy of the Holmes map was confirmed by clinical-radiologic correlation (7–9). However, these early studies are limited by the poor resolution of early computed tomographic scans. Horton and Hoyt (10) tested the accuracy of Holmes's retinotopic map of the striate cortex by correlating MR images with homonymous field defects in three patients with clearly defined occipital lobe lesions. Their findings indicated that Holmes underestimated the cortical magnification of central vision. In the revised map by Horton and Hoyt, the area subserving central vision was expanded and the area devoted to peripheral vision was reduced. They proposed that the central cortical representation of vision occupies a much larger area. A study by McFadzean et al (11) also supported this revised representation by Horton and Hoyt. In their study, the central 10° of visual field is represented by at least 50% to 60% of the posterior striate cortex.

The striate cortex has been carefully mapped using electrophysiological studies of Old World primate genera (12, 13). These studies have shown similarities to data obtained from other nonhuman primate species in which central vision occupies a large proportion of the striate cortex. For example, in macaque monkeys, the central 15° of vision occupies about 70% of the total surface area of the striate cortex (12, 13). The findings in the human striate cortex by Horton and Hoyt and by McFadzean et al are compatible with microelectrode recordings in the macaque striate cortex (12, 13).

In our study, the visual field deficits in patients with Minamata disease were incompatible with the map devised by Horton and Hoyt. According to Figure 1, the central 10° and 15° of vision fill about 20% and 30% of the surface area of the striate cortex, respectively; whereas in the study by Horton and Hoyt, the central 10° of the visual field is represented by 50% to 60% of the posterior striate cortex. Our cases demonstrate that central vision occupies a smaller proportion of the striate cortex than showed by Horton and Hoyt in their retinotopic map. Our results were closer to the data from the original Holmes map, in which the central 15° of vision occupies

about 25% of the surface area of the striate cortex. The MR imaging findings and the visual field deficits have been correlated in a very limited number of cases in previous studies. Horton and Hoyt studied only three cases and McFadzean et al used axial scans in most cases. In our cases, all MR imaging examinations included axial, coronal, and sagittal views. Our data from eight patients may be more exact than those of previous studies. It is also important to be cautious in relating the anatomic extent of a lesion at MR imaging to the functional perimetric findings, as the former simply outlines disturbances of water content and its distribution within tissues without necessarily implying neuronal death, while the latter is a subjective determination of variation in function at one point in time (11). Because all our patients with Minamata disease have had very long clinical courses, some functional reorganization may have occurred, leading to improved vision. On the other hand, in the patients with relatively acute disease examined by Horton and Hoyt, this reorganization may not have occurred. This difference might explain the discrepancy between our results and theirs.

In severe cases of Minamata disease, the visual fields are identical with bilateral homonymous hemianopsia, with sparing of central vision. Complete bilateral homonymous hemianopsia with sparing of central vision is a specific visual defect caused in most cases by bilateral occipital infarction associated with arteriosclerosis (14). MR images in these patients are essentially the same as in patients with Minamata disease. A mechanism whereby macular sparing arises has been proposed, based on the hypothesis that the posterior pole of the occipital lobe in the striate cortex, which corresponds to the macula, receives a dual blood supply from the posterior and middle cerebral arteries (14).

In conclusion, visual field deficits in our patients with Minamata disease correlated well with MR imaging findings of the striate cortex. The central portion of the visual fields occupied the posterior area as well as a greater proportion of the striate cortex. This study confirms the classical Holmes retinotopic map of the representation of the visual field in the occipital striate cortex, in which the central 10° occupies 20% of the striate cortex posteriorly, and the more peripheral visual field is reduced correspondingly.

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