T2 Shortening in the Visual Cortex: Effect of Aging and Cerebrovascular Disease

Yukunori Korogi, Toshinori Hirai, Yasuyuki Komohara, Tomoko Okuda, Ichiro Ikushima, Mika Kitajima, Yoshinori Shigematu, Takeshi Sugahara, and Mutsumasa Takahashi

PURPOSE: To evaluate the effect of aging and cerebrovascular disease on T2 shortening in the visual cortex at MR imaging. **METHODS:** MR images of 72 neurologically normal subjects (45 men and 27 women, 35 to 92 years old) and 32 patients (13 men and 19 women, 54 to 92 years old) with cerebrovascular disease were evaluated retrospectively. On T2-weighted spin-echo images, the signal intensity of the visual, motor, and sensory cortices was divided into three grades and compared with the signal intensity of the frontal subcortical white matter. **RESULTS:** Decreased signal intensity (grade III) was rarely seen in the visual and sensory cortices of the neurologically normal subjects who were less than 60 years old. The signal intensity of the motor cortex decreased rapidly after the age of 50 years. At 61 to 70 years of age, 53% of these subjects had grade III intensity, and at age 71 years or older, 94% had reached grade III. The frequency of progression from grade I to grade III was lower in the visual cortex than in the motor cortex; 22% of these subjects had grade III appearance at age 61 to 70 years, and at age 71 years or older, 56% had reached grade III. In patients with cerebrovascular disease who were older than 60 years of age, the frequency of grade III signal intensity in the visual cortex was almost equal to that in the neurologically normal subjects. **CONCLUSIONS:** T2 shortening in the visual cortex is frequently seen in neurologically normal older persons. These findings are compatible with a previously reported histochemical study of normal iron deposition in the visual cortex. Cerebrovascular disease has no effect on T2 shortening in the visual cortex.

Index terms: Age and aging; Brain, magnetic resonance

AJNR Am J Neuroradiol 18:711–714, April 1997

Decreased signal intensity found only on T2-weighted magnetic resonance (MR) images can be ascribed to the magnetic susceptibility effect (1). The T2 shortening effects from local magnetic field heterogeneity can be produced by paramagnetic substances. Iron is the most frequently encountered paramagnetic substance in the normal brain (2, 3). With high-field-strength MR imaging, region-specific T2 shortening in the human brain has been reported in association with iron deposition in normal and abnormal conditions (4–9). In healthy adults, decreased signal intensity on T2-weighted images is seen in the globus pallidi, red nuclei, substantia nigra, and dentate nuclei (4–6). This decreased signal intensity is age dependent and has also been reported in abnormal conditions (7–9).

T2 shortening in the motor cortex has been reported in patients with amyotrophic lateral sclerosis (10–12), as well as in healthy older persons (13). We retrospectively studied brain MR images in 72 neurologically normal patients to determine the normal timing and sequence of T2 shortening in the visual cortex. We also evaluated brain MR images in patients with cerebrovascular disease, and compared them with those of the neurologically normal subjects.

Materials and Methods

MR studies of 72 patients (45 men, 27 women) were selected and studied retrospectively from a consecutive

Received July 30, 1996; accepted after revision November 15.

Presented at the annual meeting of the American Society of Neuroradiology, Seattle, Wash, June 1996.

From the Department of Radiology, Kumamoto University School of Medicine, 1–1–1 Honjo, Kumamoto 860, Japan. Address reprint requests to Yukunori Korogi, MD.

AJNR 18:711–714, Apr 1997 0195-6108/97/1804–0711

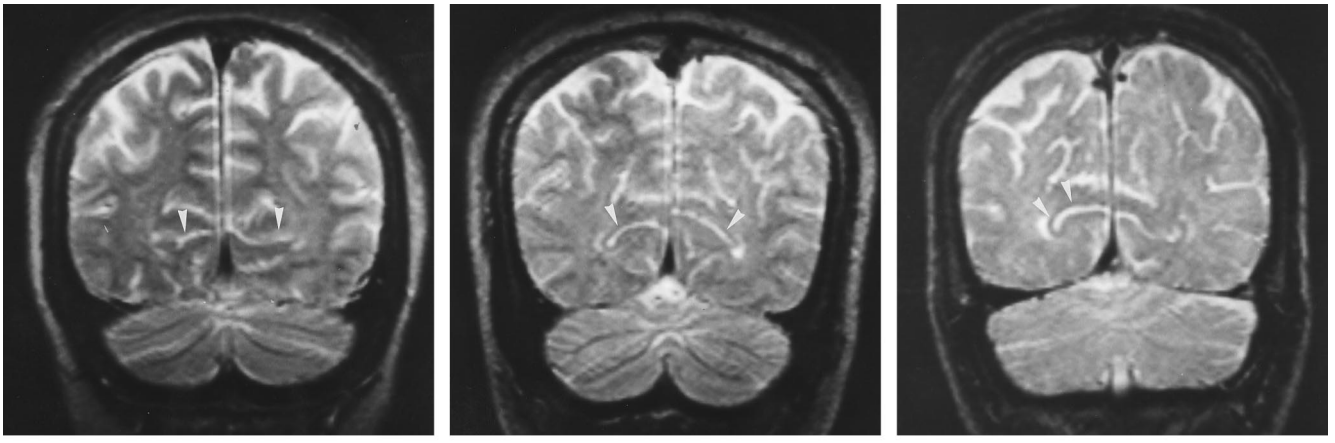

Fig 1. Signal intensity grades of the visual cortex on T2-weighted images (2200/80/1) in neurologically normal subjects.

A, Grade I in a 63-year-old woman. The visual cortex (*arrowheads*) is hyperintense relative to the subcortical white matter and isointense relative to gray matter.

B, Grade II in a 58-year-old man. The visual cortex (*arrowheads*) is isointense compared with the subcortical white matter and hypointense compared with gray matter.

C, Grade III in an 84-year-old man. The visual cortex (*arrowheads*) is definitely hypointense as compared with the subcortical white matter.

series of routine MR brain examinations in which both axial and coronal T2-weighted images were obtained. Criteria for inclusion were no clinical evidence of neurologic disease and a normal brain MR study. We excluded patients with a history of neurologic disease, malignancy, stroke, and brain surgery. Patients' ages ranged from 35 to 92 years: nine patients (five men and four women) were 35 to 50 years old; 15 (eight men and seven women) were 51 to 60 years old; 32 (23 men and nine women) were 61 to 70 years old; and 16 (nine men and seven women) were 71 to 92 years old.

MR images of 32 patients (13 men, 19 women) who had small multiple infarctions of the deep white matter and/or basal ganglia were also studied retrospectively. These lesions were probably due to microangiopathy; those involving major infarction of the territories of the anterior, middle, and/or posterior cerebral arteries were not included. The ages of these patients ranged from 54 to 92 years: three (all women) were 54 to 60 years old; 12 (five men and seven women) were 61 to 70 years old; and 17 (eight men and nine women) were 71 to 92 years old. We excluded patients with metabolic, degenerative, and specific neurologic disorders.

A high-field-strength (1.5-T) MR system was used to obtain both T1- and T2-weighted spin-echo images. Imaging parameters were 600/20/1 (repetition time/echo time/excitations) for T1-weighted images and 2200/80/1 for T2-weighted images. Using a head coil, we acquired multisection images in the axial and coronal planes. In 28 neurologically normal subjects and in 15 patients with cerebrovascular disease, sagittal T2-weighted images were also obtained. All images were obtained with a 22-cm field of view and a 192 × 256 matrix. Section thickness was 5 to 6 mm, with a 1.0- to 1.2-mm intersection gap. The fast spin-echo technique was not used in this study. The motor and sensory cortices were evaluated in the axial plane. Since the calcarine gyrus runs nearly parallel to the axial plane, it was evaluated in the coronal and sagittal planes.

The signal intensity of the calcarine gyrus (striate cortex = visual cortex), precentral gyrus (motor cortex), and postcentral gyrus (sensory cortex) was evaluated by two neuroradiologists without information about clinical data. We used the signal intensity of the precentral and postcentral gyri as background data. The signal intensity of these regions was divided into three grades (13): grade I, signal intensity is equal to that of cortical gray matter (Fig 1A); grade II, signal intensity is less than that of cortical gray matter and equal to or greater than that of subcortical white matter (Fig 1B); and grade III, signal intensity is less than that of subcortical white matter (Fig 1C). The frontal subcortical white matter was used for reference.

Results

The changes in signal intensity in the visual cortex of neurologically normal subjects over time are depicted in Figure 2. Decreased signal intensity (grade III) was rarely seen in the visual and sensory cortices in the these subjects who were less than 60 years old. However, the signal intensity of the motor cortex decreased rapidly after the age of 50 years; at age 61 to 70 years, 53% of these subjects had grade III intensity, and at 71 years and older, 94% had reached grade III. The rate of progression from grade I to grade III was lower in the visual cortex than in the motor cortex; 22% of the subjects had a

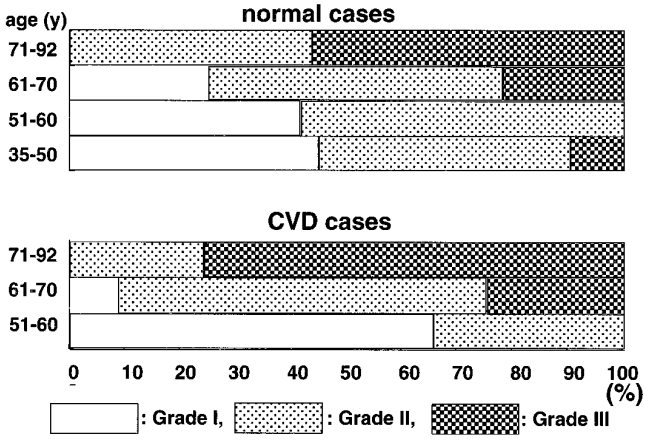

Fig 2. Distribution of grades by age in the visual cortex in neurologically normal subjects (*normal cases*) and in patients with cerebrovascular disease (*CVD cases*). Note that grade III was frequently seen in persons over the age of 70 years. In patients with cerebrovascular disease, the frequency of grade III was almost equal to that in the neurologically normal subjects.

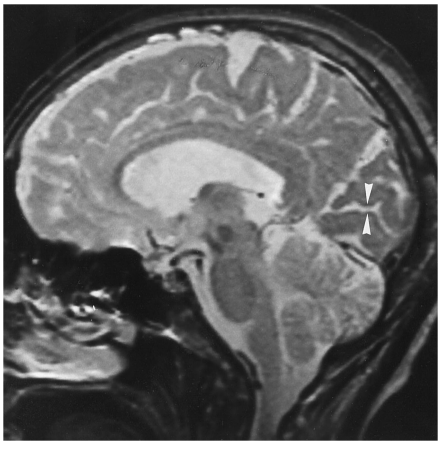

Fig 3. MR image (2200/80/1) in an 86-year-old woman with cerebrovascular disease shows definitely decreased hypointensity (grade III) in the visual cortex (*arrowheads*).

grade III appearance at age 61 to 70 years, and at 71 years and above, 56% had reached grade III in the visual cortex. In the sensory cortex, the frequency of grade III was lowest among the three cortices in each age group.

In patients older than 60 years with cerebrovascular disease, the frequency of grade III signal intensity in the visual cortex was nearly equal to that in the neurologically normal subjects (Fig 3). However, in the motor cortex, 29 (91%) of 32 patients with cerebrovascular disease had grade III intensity.

No abnormal signal intensity was observed on T1-weighted images in either group.

Discussion

In 1922, Spatz (14) described a macroscopic, qualitative histochemical study of brain iron. He divided the regions of the brain into four groups according to their iron content. In 1958, Hallgren and Sourander (15) reported quantitative age-related measurements of nonheme iron in the human brain. They mentioned that the iron content in the cerebral cortices increased with age; in the older people the motor cortex (precentral gyrus) had a mean iron content of approximately 5.0 mg/100 g, closely followed by the occipital cortex, the sensory cortex (postcentral gyrus), and then the rest of the parietal cortex. The temporal and prefrontal cortices showed the lowest iron values; mean iron content was nearly 3.0 mg/100 g. Our results correspond well to the histochemical study by Hallgren and Sourander. A previous study, in which T2 shortening of the cerebral cortices was systematically evaluated, did not include the visual cortex (calcarine gyrus), because this structure runs nearly parallel to the axial plane (13). The visual cortex also showed decreased signal intensity on T2-weighted images when evaluated in the coronal or sagittal plane. In the motor cortex, decreased signal intensity was seen more frequently in this study than in the previously mentioned study (13). Although the reasons are unknown, different MR units and imaging parameters might affect the signal intensity of the cerebral cortex.

There is a preferential progressive increase of iron in the corpus striatum with aging. This increase may be related to a combination of factors, including decreased oxidative phosphorylation, declining oligodendroglial function, decreased dopamine production and turnover, abnormal blood-brain barrier permeability, or accelerated hydroxyl free radical formation with lipid membrane peroxidation (5). Iron may play an important role in neurotransmitter metabolism (4, 5, 16), as it seems to be translocated by axonal transport from regions with high densities of transferrin receptors to regions with high nonheme iron content, such as the striatum (16). A functional decrease of the axonal transport associated with aging may induce iron deposition in the cerebral cortex (13). In our patients with multiple infarctions of deep white matter and/or basal ganglia, the motor cortex reached grade III signal intensity even in those younger than 60 years of age. This indicates

that cerebrovascular disease may promote degeneration of neurons and glial cells in the motor cortex, resulting in iron accumulation (13). In the visual and sensory cortices, however, the frequency of grade III changes in patients with cerebrovascular disease was almost equal to that of the neurologically normal subjects. There may be some differences in the mechanism of iron accumulation between the motor cortex and visual or sensory cortex.

Our study has some limitations. First, iron content in the white matter, which we used as a reference standard, also increases with age. Decreased signal intensity (grade III) was not observed in the parietal, temporal, and prefrontal cortices. This may be due to the fact that iron content in the frontal white matter is higher than that in the parietal, temporal, and prefrontal cortices. Although changes in surrounding structures might have some effect on our visual grading, we believe that our grades (especially grade III) correlate with T2 shortening caused by iron accumulation, because our study is compatible with the histochemical quantitative study of brain iron. The second limitation is related to the issue of partial volume effect. We used images in which section thickness was 5 to 6 mm with a 1.0- to 1.2-mm intersection gap. Cortical atrophy, associated with aging, may also affect the signal intensity of the cortices (13).

In conclusion, we have determined the normal timing and sequence of T2 shortening in the visual cortex. In the older neurologically normal subjects, T2 shortening in the visual cortex was seen frequently. This finding is compatible with a previously reported histochemical study of normal iron deposition. Presence of cerebrovascular disease did not affect the decreased signal intensity in the visual cortex.

References

1. Gillis P, Koenig SH. Transverse relaxation of solvent protons induced by magnetized spheres: application to ferritin, erythrocytes, and magnetite. *Magn Reson Med* 1987;5:323– 345
2. Gomori JM, Grossman RI, Goldberg HI, et al. Intracranial hematomas: imaging with high-field MR. *Radiology* 1985;157:87–93
3. Bizzi A, Brooks RA, Brunetti A, et al. Role of iron and ferritin in MR imaging of the brain: a study in primates, at different field strengths. *Radiology* 1990;177:59–65
4. Drayer BP, Burger P, Darwin RH, Riederer SJ, Herfkens R, Johnson GA. Magnetic resonance imaging of brain iron. *AJNR Am J Neuroradiol* 1986;7:373–380
5. Drayer BP. Imaging of the aging brain, I: normal findings. *Radiology* 1988;166:785–796
6. Aoki S, Okada Y, Nishimura K, et al. Normal deposition of brain iron in childhood and adolescence: MR imaging at 1.5 T. *Radiology* 1989;172:381–385
7. Drayer BP, Olanow W, Burger P, Johnson GA, Herfkens R, Riederer S. Parkinson plus syndrome: diagnosis using high field MR imaging of brain iron. *Radiology* 1986;159:493–498
8. Pastakia B, Polinsky R, Di Chiro G, Simmons JT, Brown R, Wener L. Multiple system atrophy (Shy-Drager syndrome): MR imaging. *Radiology* 1986;159:499–502
9. Drayer BP. Imaging of the aging brain, II: pathologic conditions. *Radiology* 1988;166:797–806
10. Oba H, Araki T, Ohtomo K, et al. Amyotrophic lateral sclerosis: T2 shortening in motor cortex at MR imaging. *Radiology* 1993; 189:843–846
11. Ishikawa K, Nagura H, Yokota T, Yamanouchi H. Signal loss in the motor cortex on magnetic resonance images in amyotrophic lateral sclerosis. *Ann Neurol* 1993;33:218–222
12. Cheung G, Gawal MJ, Cooper PW, Farb RI, Ang LC. Amyotrophic lateral sclerosis: correlation of clinical and MR imaging findings. *Radiology* 1995;194:263–270
13. Hirai T, Korogi Y, Sakamoto Y, et al. T2 shortening in motor cortex: effect of aging and cerebrovascular disease. *Radiology* 1996;199:799–803
14. Spatz H. Uber den eisennachweis im gehirn, besonders in zentren des extrapyramidal-motorischen systems. *Z Ges Neurol Psychiatry* 1922;77:261–390
15. Hallgren B, Sourander P. The effect of age on the non-haemin iron in the human brain. *J Neurochem* 1958;3:41–51
16. Morris CM, Candy JM, Bloxham CA, Edwardson JA. Immunocytochemical localisation of transferrin in the human brain. *Acta Anat* 1992;143:14–18