CASE REPORT

Age-related White Matter Hyperintensities Attenuated by Compression from a Chronic Subdural Hematoma: Possible Contribution of Brain Interstitial Fluid to the Formation of Leukoaraiosis

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The origin of patchy white matter hyperintensities commonly seen in the elderly on magnetic resonance (MR) images with long repetition time (TR) is still controversial. We describe MR findings in older patients in whom white matter hyperintensities were attenuated by compression of the cerebral hemisphere from a chronic subdural hematoma. These sequential MR findings substantiate the hypothesis that leukoaraiosis may arise when drainage of the bulk flow of brain interstitial fluid is disturbed.

Keywords: brain interstitial fluid, chronic subdural hematoma, leukoaraiosis

Introduction

Patchy white matter hyperintensities on magnetic resonance (MR) images with long repetition time (TR), termed leukoaraiosis, are commonly seen in the elderly.1 However, their origin remains controversial. The reported pathological correlations include noncystic infarction,2 myelin rarefaction of ischemic origin,3,4 myelin pallor and dilatation of perivascular spaces,5 gliosis and demyelination presumably from chronic vascular insufficiency,2 and spongiosis, gliosis and demyelination associated with periventricular venous collagenosis.6 Impairment of interstitial fluid (ISF) transportation may lead to increased interstitial water content in the white matter of patients with leukoaraiosis.3,7

Case Report

Case 1

Fluid-attenuated inversion recovery (FLAIR) MR images of an 84-year-old man admitted to the hospital with weakness of his extremities showed symmetrical irregular periventricular and confluent deep white matter hyperintensities (Fig. 1a, b). After admission, he developed right convexity chronic subdural hematoma. MR images disclosed compression to the right cerebral hemisphere by a convexity hematoma associated with attenuation of the ipsilateral white matter hyperintensities. Quantitative measurement revealed neither laterality nor significant interval changes of the signal intensity of the bilateral patchy white matter lesions (Fig. 1c, d). After evacuation of the hematoma, postoperative MR images revealed resolution of the cerebral compression and reappearance of symmetrical white matter hyperintensities (Fig. 1e, f).

Case 2

An 80-year-old man developed chronic left subdural hematoma. Early postoperative MR images showed moderate residual hematoma compressing the left cerebral hemisphere and white matter hyperintensities less prominent on the ipsilateral left hemisphere than the contralateral right hemisphere (Fig. 2a, b). Follow-up MR images obtained 6 months later disclosed resolution of the subdural hematoma and symmetrical irregular periventricular white matter hyperintensities extending into deep white matter (Fig. 2c, d).

Discussion

In our elderly patients, cerebral compression by a chronic subdural hematoma attenuated white matter hyperintensities. To our best knowledge, serial MR findings of such attenuation have not been
Fig. 1. An 84-year-old man with right convexity chronic subdural hematoma (Case 1). (a) Axial and (b) coronal fluid attenuated inversion recovery (FLAIR) images show bilateral symmetrical irregular periventricular and confluent deep white matter hyperintensities. Right basal ganglia lacunar infarct is also seen. (c) Axial and (d) coronal magnetic resonance (MR) images obtained 5 weeks later disclosed right convexity subdural hematoma compressing the right cerebral hemisphere. Periventricular and deep white matter hyperintensities are less prominent on the ipsilateral side of the subdural hematoma. Postoperative (e) axial and (f) coronal MR images reveal resolution of the cerebral compression and reappearance of symmetrical white matter hyperintensities.
Fig. 2. An 80-year-old man with left convexity chronic subdural hematoma (Case 2). Early postoperative (a) axial and (b) coronal fluid attenuated inversion recovery (FLAIR) images demonstrate moderate residual left convexity subdural hematoma. The left cerebral hemisphere is compressed by a subdural hematoma, and white matter hyperintensities are less prominent on the ipsilateral than contralateral side of the hematoma. (c) Axial and (d) coronal follow-up magnetic resonance (MR) images disclosed resolution of the cerebral compression and symmetrical white matter hyperintensities.

Reported in the English literature.

Heterogeneous histopathologic changes are reported to be associated with incidental white matter hyperintensities. Ischemic brain damage and arteriosclerotic disease are usually detected within irregular periventricular and confluent deep white matter changes.\textsuperscript{3,4} Deep and subcortical white matter hyperintensity reflects myelin pallor and dilatation of perivascular spaces.\textsuperscript{5} Moody and colleagues demonstrated periventricular venous collagenosis in leukoaraiosis. This age-related noninflammatory venous stenosis may promote development of spongiosis, gliosis, and demyelination of the white matter from chronic edema.\textsuperscript{6} Alternatively, leukoaraiosis may arise from disturbance of the cerebrospinal fluid (CSF) circulation. Roman suggested that increased CSF accumulation in the ventricle raises interstitial pressure in the periventricular parenchyma and causes ischemia to the white matter.\textsuperscript{8} Leakage of CSF into the brain parenchyma caused by damage to the ependymal lining may also result in development of leukoaraiosis.

Impaired bulk flow of interstitial brain water may contribute to an increase in interstitial water content of the white matter that may lead to the formation of white matter hyperintensities. Blood plasma is the main source of interstitial brain
water, and interstitial fluid is formed by the brain capillary endothelium. A portion of CSF may recycle and join the circulating ISF, draining back via venous perivascular spaces and axon tracts into the CSF compartments. Age-related amyloid deposition within the interstitial fluid pathways may impede normal ISF circulation and drainage, leading to higher water content in the cerebral white matter.10

Compression of the cerebral hemisphere by a chronic subdural hematoma attenuated white matter hyperintensities in our elderly patients. Similar to our patients, Itasaka’s group also reported less prominent leukoaraiosis at the ipsilateral side with unilateral chronic subdural hematoma.11 These findings substantiate the theory that increased interstitial water may partly contribute to leukoaraiosis. Because of the incompressibility of the constituents of the skull, the sum of the intracranial volumes of blood, brain, CSF, and other components, such as hematoma, must remain constant. Fluid in the central nervous system is distributed in the intracellular and extracellular spaces of the brain parenchyma, CSF, and intravascular compartment.12 The cerebral blood volume comprises 3–4% of the brain. The CSF spaces over the cerebral convexities could account for part of the changes.13 Whereas the gray matter is relatively enriched in nerve cell bodies and has a low compliance, the white matter contains mostly axons and has a high compliance, behaving like a sponge.13 This sponge-like nature of the white matter enables the brain to adjust to compression by shrinkage of the extracellular space. The increased ISF in the white matter compressed by subdural hematoma in our patients may have squeezed into the CSF compartment and thereby attenuated the patchy white matter hyperintensities. Decompression by hematoma evacuation could allow the reappearance of almost symmetrical cerebral white matter changes.

Conclusion

We described the attenuation of white matter hyperintensities in elderly patients by cerebral compression from a chronic subdural hematoma. Serial MR findings substantiate the theory that white matter hyperintensities may develop when drainage of the bulk flow of brain ISF is disturbed.

References