Focal Lesions of the Splenium of the Corpus Callosum: A Common Finding on FLAIR MR

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Purpose
On a routine axial FLAIR imaging, several elderly patients were noted to have unexplained high signal in the splenium of the corpus callosum without significant surrounding white matter abnormalities. Previous reports have described lesions of the splenium but in the setting of an underlying disease such as multiple sclerosis or tumor spread (1). We initiated a prospective examination of adult patients using sagittal FLAIR imaging to determine the incidence of the finding and its significance.

Materials & Methods
In the course of routine clinical imaging, sagittal and axial FLAIR scans (TR 10,002/TE 166/TI 2200) were included in the imaging protocols of 37 adult patients. No scans of patients with obvious pathology such as tumor involving the corpus or typical imaging features of MS were included in this study. The average age of the study group was 57 years, range 22–93 years. The scans were graded for the degree of involvement of the splenium on the axial exam from zero to three with zero reflecting no high signal in the splenium.

Two additional cases are included both of whom had axial FLAIR scans with high signal in the splenium noted prior to a scheduled autopsy. In one case there was grade 3 involvement. The splenium of the corpus callosum was sectioned sagittally and stained with hematoxylin and eosin (H&E) in both cases.

Results
In 9/36 there were 2 or 3 signal abnormalities evident in the splenium. The sagittal FLAIR scan demonstrated the abnormality along the anterior undersurface of the splenium. Because of the proximity of high signal in the ventricle these lesions are inapparent on T2 imaging. Seven of these cases had received prior radiation therapy to the brain. The other two were aged 79 and 93. Six of 36 had linear high signal in the splenium (grade 1). Half of these cases had a history of CVA or TIA. Their average age was 66 years. The group with no signal abnormality (20/36) included a variety of diagnoses but were younger, average age 48 years.

The two autopsy cases both demonstrated abnormalities of the splenium on FLAIR but one was grade 1 (age 80 years) the other grade 3 (age 63 years). Examination of the under surface of the grade 3 splenium revealed a band of isomorphic gliosis of variable thickness which began at the posterior inferior aspect of the splenium in the midline and extended anteriorly on the undersurface of the corpus callosum a few centimeters. Comparison of these changes with the histologic appearance of the inferior surface of the splenium from the grade 1 case demonstrated the absence of this thick band of gliotic corpus callosum. The abnormalities suggest a slow loss
of axons and their associated myelin sheaths from this zone of the posterior corpus callosum. The pathogenesis of these alterations is not evident from the histologic examination, other than to suggest that it was an indolent process.

**Conclusion**
High signal lesions of the splenium of the corpus callosum are frequently seen on FLAIR imaging in adults without evidence of tumor invasion or demyelination. These lesions are most evident after radiation therapy but can be seen also in elderly patients and younger individuals with a history of cerebrovascular disease. The findings we report at autopsy are not specific but suggest that the signal abnormality does not reflect a prior ischemic insult or leukoariosis.

**References**