Commentary

Postmortem (Specimen) MR

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Sharing a common interest in normal and pathologic anatomy, diagnostic radiology and anatomic pathology are disciplines that become more closely related with each technological advance in imaging. As is illustrated in the study by Grafe et al. [1] in this issue of the AJNR, the paths of radiology and pathology sometimes even intersect. This study can be viewed either as a radiologic investigation with pathologic correlation or as principally a pathologic study with radiologic correlation. In such an investigation, specimens are imaged postmortem so that studies of a series of specimens of a specific disease entity no longer must wait for those rare instances in which patients who are autopsied happened to have been studied radiologically shortly before death. MR can record the multiplicity and three-dimensional extent of the lesions, whereas the histologic examination focuses on what it does best: a high-resolution but basically two-dimensional study of what the lesion is. Abundant evidence is now available that MR signal characteristics of normal and abnormal brains change surprisingly little after formalin fixation of this organ and that postmortem MR of the brain or other organs is a legitimate endeavor. The article by Grafe et al. reemphasizes this point and should encourage those interdisciplinary studies that capitalize on the advantages of both disciplines.

This correlative endeavor raises a number of important technical points. One is the assurance that the lesions studied histologically are those that were seen radiologically. In the study by Grafe et al., the brains were cut in the plane of the MR scan to assure this congruence. In certain circumstances, my colleagues and I have found it also is useful to examine the brain by MR after it has been sectioned. This can be done by restacking the sectioned brain and recording a series of images or by imaging each slice individually. By either means, but particularly the latter, the slice that contains a given radiologic lesion can be identified easily.

Another issue is related to the presence of radiologic false-positives. In our experience, such occurrences usually relate to foci of increased signal intensity, as was noted by Grafe et al. Overlooked lesions such as small infarcts or old slitlike hemorrhages are common causes of this seeming lack of correlation between radiologic and histologic findings. These occult foci, however, can be detected by having the pathologist do additional sectioning and a careful examination of the tissue slices. Of course, this proceeds more confidently if it is certain that the radiologic abnormality is contained within the tissue slice being examined for histologic studies. Other causes of false-positives include small areas of demyelination or neoplasia that are in some instances difficult, if not impossible, to visualize with the naked eye. Such lesions may even be missed in standard H and E–stained sections because the edema that provokes high signal intensity is not well seen in such preparations. A section stained for myelin may be required, particularly a large histologic section in which areas of edema or pallor can be contrasted with more normal dark staining of distant white matter or that of the edema-resistant compact white matter subcortical arcuate fibers (U fibers).

In the series of Grafe et al., the false-positives were small punctate foci of signal hyperintensity and presumably represented vessels or perivascular spaces. Such vessels with surrounding spaces appear bright on T2-weighted images in both clinical and postmortem scans. They are most evident at the base of the brain but often are noted in the superior cerebral hemisphere, where they define the course of the

This article is a commentary on the preceding article by Grafe et al.

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penetrating vessels. These are bilaterally symmetrical and generally are not considered abnormal by the pathologist, however impressive they are on MR images.

In the study by Grafe et al., clusters of phagocytes, “glial nodules,” were of special concern because they were not seen on MR but were associated with significant changes in mentation. In the setting of AIDS, these foci of inflammation are usually a consequence of disseminated cytomegalovirus infection. This form of cytomegalovirus encephalitis contrasts with the large necrotizing and often calcifying foci seen in the brain of premature infants or the necrotizing foci seen in some adult AIDS patients. In the case of cytomegalovirus infection, the glial nodules often include a virus-containing cell, usually a neuron, surrounded by a cluster of phagocytes. The nodules are minute, and it is not surprising that they are not spatially resolvable by MR. In addition, because these structures occur in the gray matter, surrounding vasogenic edema is slight, and there may not be a sufficient halo of water to enlarge the overall size of the lesion into range of detection by MR.

REFERENCE