Is Peripheral Spinal Cord Hypointensity a Sign of Venous Hypertensive Myelopathy?

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To describe a heretofore unnoticed finding, specific to a particular diagnosis, has always been a challenge for radiologists. When validated over time by different observers, these ''signs'' become part of the language of our specialty. Radiology is replete with such signs because they may render an otherwise difficult diagnosis easier, may help one appreciate the anatomy and pathologic features of an underlying abnormality, and most importantly, may quickly direct the treating physician to the proper diagnosis. In this issue of the *AJNR* (page 781), Hurst and Grossman posit that peripheral hypointensity of the spinal cord on T2-weighted images suggests the presence of a venous hypertensive myelopathy, and imply that with this ''sign'' one should consider the possibility of an underlying spinal dural arteriovenous fistula (SDAVF). That conclusion and their article deserves comment.

The diagnosis and treatment of SDAVFs have received considerable attention in the literature over the past few years. Establishing the correct diagnosis early in a patient's course, before there are irreversible neurologic deficits, falls primarily on the shoulders of the neuroradiologist because these patients often present with a progressive paraparesis of unknown etiology. Although the MR signs of an SDAVF are now well recognized (a combination of enlarged and tortuous veins that are most prominent along the posterior cord surface, moderate enhancement in an enlarged cord, and increased signal in the center of the cord on T2-weighted images), there is a range of severity of these abnormalities, which may parallel the degree of clinical severity. As a result, less prominent SDAVFs with less flagrant MR findings could escape detection, particularly early in the course of the disease. Having another ''sign'' more specific for this abnormality clearly would be of great value when the veins along the posterior surface of the cord are nearly normal or questionably prominent, and when increased intensity in the cord on T2-weighted images and enhancement on the postcontrast studies might suggest another diagnosis, such as an infiltrating glioma or a myelitis. Recognizing the possibility of an SDAVF would then lead to a spinal MRA, catheter angiography, or both and, by establishing the diagnosis of an SDAVF, there would be early surgical or endovascular intervention that, if successful, would reverse neurologic deficits.

Two issues arise, however, when considering the value and significance of this proposed sign. The first and most important is whether the finding of peripheral cord hypointensity represents a real pathologic alteration. Is it a result of slow venous flow in the venous and capillary system of the cord, an accumulation of paramagnetic substances in the cord, or is this hypointensity a visual phenomenon accentuated by the silhouetting of the periphery of the cord between the bright CSF in the subarachnoid space and the high signal of the abnormal cord? If one assumes that this observation represents a true finding and is sustained by other observers, the second issue to be addressed is what, in fact, this peripheral hypointensity represents.

Concerning the first issue, two approaches could be taken to validate Hurst and Grossman's sign. A series of T2-weighted scans in which abnormal high signal within the center of the cord with various causes other than SDAVFs could be analyzed subjectively. The object would be to determine whether the peripheral cord signal was visually equivalent to an area of normal cord or whether in these cases the periphery of the cord was perceived as hypointense. If the latter situation prevailed, then one would believe that this was simply a visual phenomenon, whereas if the former situation prevailed, one might conclude that peripheral cord hypointensity could be specific to a venous hypertensive myelopathy. A more objective approach would be a quantitative determination of the signals across the width of the spinal canal, both at the level of a proven SDAVF and at the level of the normal cord above the SDAVF. A significantly different signal of the periphery of the cord at these two levels would help confirm this sign.

Concerning the second issue, pathologic correlation was not available in any of the authors' cases; however, if this sign is confirmed by future studies, then venous and capillary engorgement possibly combined with the presence of paramagnetic substances may be a logical explanation for this observation. One would then be hard pressed to think of other diseases that would give such a uniform distribution of lowered signal intensity on T2-weighted images. Over time, investigations by others will be needed before we can add this cord hypointense periphery finding to the legions of ''signs'' in radiology.

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