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AIDS-Associated Myelopathy: Clinical Severity, MR Findings, and Underlying Etiologies

It is well recognized that patients with AIDS can present with a myelopathy unrelated to a tumor, opportunistic infection, or vascular disease. Because in such cases it is uncommon to obtain pathologic correlation and because we seldom determine the actual cause of the clinical and MR findings, the term ''AIDS-associated myelopathy'' has been used to reflect this uncertainty. Are such cases of spinal cord dysfunction analogous to brain involvement in HIV; ie, an HIV myelitis? Are the findings nutritional/metabolic in nature, attributable to a treatment regimen, or are they a combination of these and other indirect factors? Although Chong et al, in this issue of the *AJNR* (page 1412), do not attempt to answer these questions, they describe the variable MR findings in the spinal cord in AIDS-associated myelopathy, and correlate these findings with the clinical status of the patient. Despite the small number of patients in each of their MR and clinical categories, one can recognize a trend toward more striking MR-revealed changes (abnormal cord signal and spinal cord atrophy) found in the more severely affected patients. It is surprising and somewhat counterintuitive that MR imaging findings of a patient so impaired that independent ambulation was not possible could show no spinal cord abnormalities. Equally surprising is the finding that a patient with a mild myelopathy could have pronounced MR findings with cord atrophy and intrinsic spinal cord signal abnormalities. Clearly there is a lot to the story of AIDS-associated myelopathy that is not understood and deserves further investigation.

One of the first issues to address is the relationship between spinal cord abnormalities and abnormalities (both clinically and MR-revealed) in the brain; ie, what is the incidence of concurrent dementia (AIDS dementia complex) in such myelopathic patients and what are the MR-revealed features of HIV encephalopathy? A strong association between HIV encephalopathy and AIDS-associated myelopathy would be reflected in some commonality of MR findings in the brain and spinal cord, namely varying degrees of atrophy, abnormal signal on T2-weighted images, and a lack of abnormal contrast enhancement. If this association were found to exist, the efficacy of a common treatment regimen would be informative. As a collolary, one could determine if newer antiviral treatment strategies would have an equal effect on brain and spinal cord lesions as determined by follow-up MR imaging. On the other hand, if no significant association between brain and spinal cord MR findings were present, it would be difficult to imply that an HIV infection of just the spinal cord were present. In such a case, one could suggest a more peripheral site of primary abnormality, with a secondary effect on the spinal cord.

An obvious deficiency of Chong's article, and one that is readily acknowledged by the authors, is a lack of pathologic analysis. Figure 4, which is reproduced from a textbook, shows the histopathologic features ascribed to an AIDS-associated myelopathy. Although predominant lateral and posterior column involvement is seen in that figure, one cannot help but be struck by the fact that in the MR images presented by Chong, a similar anatomic imaging distribution is not seen. MR spatial and contrast resolution for detecting such a pattern of white matter tract abnormalities is widely available and it is known from previously published articles that selective spinal cord tract disease can be seen in a variety of abnormalities. The authors offer no explanation for this apparent lack of tract involvement on their MR images, but a reasonable guess would be that such nonselectivity speaks more for an HIV myelitis than for a vacuolar myelopathy (VM).

VM is frequently the clinical diagnosis suggested when a patient with AIDS presents with a myelopathy. In this situation, the predominate pathologic feature is vacuolation of myelin sheaths. In VM, there is an absence of demyelination and inflammation and, because of this, it is unlikely that VM is a direct viral effect. An HIV myelitis, therefore, is best considered an entity distinct from a VM. The possible cause of an AIDS-associated myelopathy becomes even more complex when one recognizes the existence of another condition termed tract pallor (TP), which is frequently seen in association with a sensory neuropathy. Here neither demyelination nor vacuolation of white matter is present; but rather, there is simply a decrease or loss of myelin staining when specimens are analyzed by histologic methods. The sensitivity of MR imaging of various degrees of pathologically proved TP is not known. Other etiologic factors to consider when encountering a patient with AIDS-associated myelopathy could be an, as of yet, undetermined metabolic abnormality (either direct or indirect) leading to this myelopathic condition and the associated MR findings.

Important questions left unanswered by Chong et al, which may be fertile ground for future investigation, include: 1) do MR abnormalities correlate more strongly with the length of time the patient has been myelopathic or do they correlate more strongly with the severity of the myelopathy itself?; 2) can a longitudinal study of these patients show stability or worsening of the MR find-

ings?; 3) can selective white matter tract involvement be seen on MR images early in the patient's myelopathic state?; and 4) can magnetization transfer ratios (MTRs) be applied to normal-appearing cord tissue to show decreased MTRs in affected spinal cords? Although these and other issues are left unresolved by Chong's investigation, the article can serve as a springboard for important studies. Future studies could investigate the effect of newer treatment protocols for the imaging of patients with AIDS-associated myelopathy and the precise distribution of signal abnormalities in the cord in both the early and late stages of this complex disease.

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Three Pathways between the Sacroiliac Joint and Neural Structures Exist

At the beginning of this century, pain from the sacroiliac joints had been considered the main source of low back pain and radiculopathy. Since the discovery and acceptance of the lumbar-disk-complex model of radicular back pain, the theory that the sacroiliac joint contributes to a low back pain syndrome remains controversial and poorly understood as part of a broad category of nondiscogenic low back pain.

Fortin et al present in this issue of the *AJNR* (page 1429) an intriquing hypothesis asserting that pathways of communication exist between the sacroiliac joints and several neural structures. Tracing extravasation patterns on sacroiliac arthrograms and postarthrogram CT, the authors have delineated pathways in which contrast material from the sacroiliac joint communicates posteriorly with the first dorsal foramima, ventrally with the lumbosacral plexus, and dorsally along the sacral ala to the fifth lumbar epiradicular sheath. Drawing from the discogenic model of low back pain, the authors suggest that sacroiliac capsular irritation and cytokine release may cause adjacent neural insult by these communications. Furthermore, the variety of structures these pathways lead to may in turn explain the variety of symptoms and signs possible from sacroiliac disease.

Sacroiliac arthrography is an uncommon procedure in most radiology departments that often falls between specialty lines of neuroradiology, musculoskeletal radiology, and body imaging, because patients with sacroiliac pain come from a variety of orthopedic, neursurgical, neurologic, and rehabilitation specialty referrals. Most of these procedures include injection of anesthetic or corticosteroids, with any reduction of a patient's symptoms indicating the sacroiliac joint as the source of pain. Extravasation is very common in these procedures, and the patterns of extravasation described by the authors frequently are observed in clinical practice. Furthermore, although the validity of pain reproduction and reduction with anesthetic in the setting of extravasation may be challenged, it nonetheless occurs. The notion that these communications by arthrography provide the mechanism for pain arising from sacroiliac disease is unproved but still attractive.

Among the most frustrating conditions in medicine is atypical or nonradicular low back pain. Unlike the patient with persistent low back pain and a radiculopathy matching a structural lesion seen at imaging, in which one may be relatively confident of a relationship between that finding and symptoms, patients with atypical lumbosacral junction pain frustrate clinicians and radiologists. Without radicular symptoms, or with a radiculopathy that does not match a structural lesion, a scenario occurs in which management often is directed by the results of provocative injections of disks, facets, and sacroiliac joints. To make matters worse, asymptomatic imaging abnormalities are common at the lumbosacral junction, including disk herniations causing nerve compression that may misdirect treatment. Sifting through the significant and insignificant imaging findings of the spine and sacroiliac joints in light of a complicated or inconsistent clinical history of low back pain is a very difficult challenge. In this setting, Fortin's observations may provide a starting point to reexamine the nature of atypical radicular pain, particularly with close correlation of injection data and specific pain patterns. Previous work by the author correlating pain maps from sacroiliac injection in volunteers and patient-drawn pain maps in individuals with atypical lumbosacral pain warrant a close read by anyone imaging or treating patients in whom lumbosacral and sacroiliac pain must be differentiated.

Despite this, caution must be used before one should accept the authors' hypothesis. The precise mechanism of pain from any joint may involve not only capsular irritation, but also direct subchondral bone irritation through cartilage loss and marrow edema. In patients with seronegative spondyloarthropathies involving the sacroiliac joints, as well as in individuals with post-traumatic and degenerative sacroiliac pain, the subchondral plate frequently is compromised, and this mechanism cannot be disregarded. Indeed, one could argue that stimulation of subchondral, pressure-sensitive pain receptors in bone could account for much of the local pain observed in sacroiliac disease, with communication to adjacent neural structures accounting only for the radicular component in those individuals with mixed local and radicular pain. This would not account, however, for the authors' prior observation of pain in an identical distribution in