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Spinal Cord Infarction and Fibrocartilagenous Emboli

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The preceding report by Yuh et al (1) provides an interesting discussion of spinal cord infarction. However, there was no mention in that report of fibrocartilagenous embolic disease as a possible etiology of cord infarction. Our group has recently had the opportunity to study a case as part of a clinical-pathologic conference (2). It is an unusual entity and its preoperative diagnosis is rare, although there are many reports of this abnormality in the non-radiologic literature. For this reason, a re-presentation of that case report and an expanded radiologic discussion is germane to the report by Yuh et al.

Case Report

A 61-year-old obese woman with a 10-year history of hypertension was well until 4 months prior to admission when she developed mild lumbar pain relieved by sitting. The pain recurred while driving and paresthesias developed in her buttocks and rectal region. This was rapidly followed by right leg weakness and an inability to walk.

She was taken to a hospital where physical exam disclosed paraparesis, poor proprioception, areflexia in the lower extremities bilaterally, saddle anesthesia, and impaired pinprick sensation below T12. Complete paraplegia with loss of bladder and bowel function developed shortly thereafter. A lumbar puncture revealed clear, colorless fluid with 68 red blood cells and no white cells per cubic millimeter. A myelogram followed by computed tomography (CT) on the day of admission were normal except for mild enlargement of the conus medullaris. With acute spinal cord compression excluded, a tentative diagnosis of spinal cord infarction was considered.

Magnetic resonance (MR) imaging of the lumbar spine performed on December 26, 1989 revealed mild enlargement of the conus with increased signal on T2-weighted images. There was a central zone of decreased signal, suggesting the presence of blood products. No postcontrast enhancement was observed (Fig. 1). A follow-up study (without contrast) performed on the 6th hospital day showed interval development of a zone of increased signal on T1-weighted images, suggesting the presence of methemoglobin in the distal spinal cord. The area of increased signal on the T2-weighted images was more striking, and increased signal was seen within the T11 vertebral body (Fig. 2).

The patient was transferred to our hospital 9 days later. Examination disclosed full paraplegia with flaccid, areflexic lower extremities and absent pinprick sensation below T12 except for minimal sensation along the inner thighs bilaterally. Lower extremity proprioception was absent. A postcontrast MR study performed on admission revealed enhancement of the conus. Hyperintense signal was present in the T11 vertebral body on the T2-weighted sequence and was more prominent than on the preceding MR (Fig. 3).

T11–L1 laminectomies were performed the following day for exploration of the distal spinal cord. This revealed a cavity, at a depth of 2 mm, that contained blue, slightly reddish, soft, necrotic tissue that was sent for pathologic analysis. The wall of the cavity appeared to be lined with gliotic tissue.

Histologic examination of the necrotic tissue revealed glial fibrils, small blood vessels, a small amount of hemosiderin, scattered ischemic anterior horn cells, lymphocytes and macrophages, findings typical of acute ischemic infarction of

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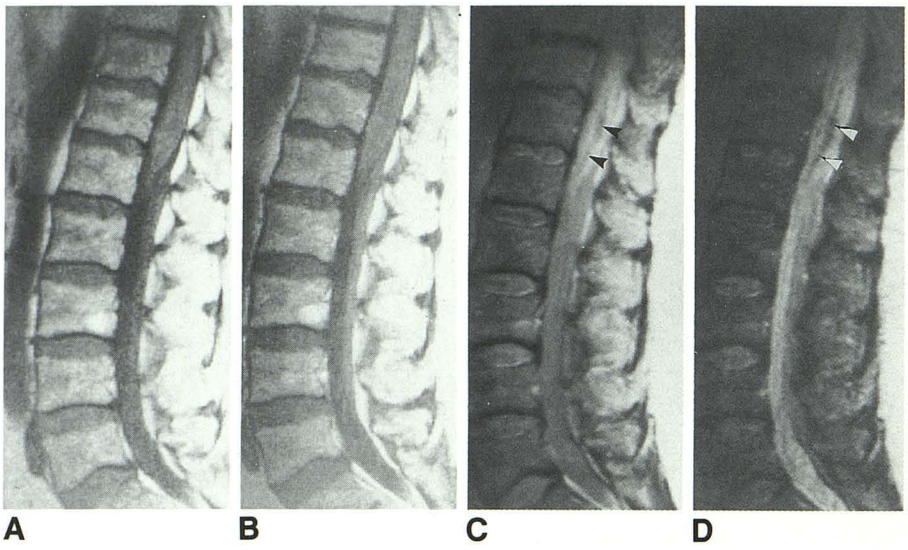

Fig. 1. Day 5 MR study (1.5 T). 500/23 (TR/TE) spin echo sequence showing slight enlargement of the distal cord and conus. The spinal cord signal appears normal with this sequence (*A*). No evidence of enhancement following 20 mL of intravenous gadolinium-DTPA (*B*). First (*C*) and second (*D*) echoes of 2500/45/90 spin echo sequence showing overall increased signal in the distal cord and conus with a central curvilinear zone of decreased signal (*arrowheads*) suggesting the presence of blood products. (Reproduced with permission from the *New England Journal of Medicine* 1991; 324:322–332.)

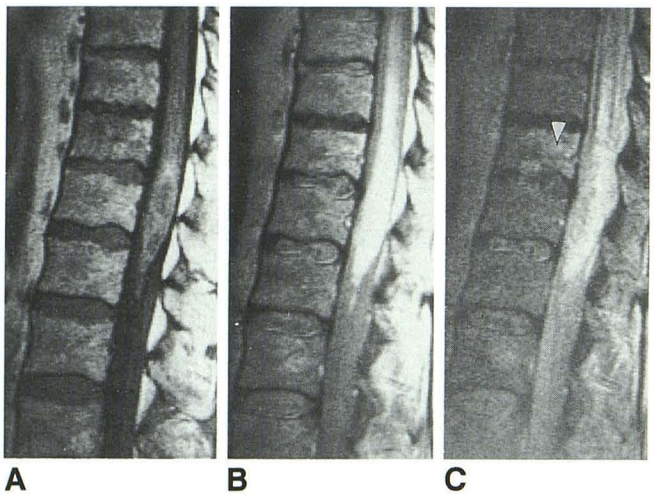

Fig. 2. Day 12 MR study (1.5 T). 500/17 (TR/TE) spin echo sequence (*A*) showing a heterogeneous, somewhat "rim-like" zone of slightly increased signal in the distal cord favoring the presence of methemoglobin not present on the first MR study (Fig. 1). The intensity of increased signal on the first (*B*) and second (*C*) echoes of a 2000/45/90 spin echo sequence, is greater than on the first study. The curvilinear region of decreased signal is much less obvious. Note increased signal in T11 (*arrowhead*). (Reproduced with permission from the *New England Journal of Medicine* 1991;324:322–332.)

the spinal cord. The presence of ischemic anterior horn cells indicated that the infarct at least involved the anterior horn of the spinal cord in the territory of the anterior spinal artery (Fig. 4). Several small blood vessels within the tissue, 20–50 microns in diameter, some arterioles, others thin-walled venules, were distended and occluded by embolic material that was acellular, partly fibrillar, and mildly vacuolated (Fig. 5). This material, on hematoxylin and eosin staining, ranged in color from light gray-blue to a deeper lavender, and, on staining with Alcian blue, the material was a deep azure blue. *The staining characteristics were identical to those of degenerated fibrocartilage derived from the nucleus pulposus of an intervertebral disc.*

In a survey of 5500 postmortem examinations performed on patients with neurologic disease from the Charles-Foix Neuropathological Laboratory at the Salpetriere Hospital, Paris, from 1950 to 1977, only 10 cases of ischemic infarction of the cord were identified (3). Fibrocartilagenous embolic disease was not documented in any of these cases nor was it thought to be the etiology of infarction in any of the cases presented by Yuh et al (1) Fibrocartilagenous embolism is a rare source of spinal cord infarction. Other etiologies include hypotension, atherosclerosis, aortic surgery, dissecting aneurysm, angiography, vertebral occlusion or dissection, syphilis, arteritis, sickle cell anemia, polycythemia, leukemia, spinal trauma, caisson disease, spinal tumor, diabetes, tuberculosis, arachnoiditis, meningitis, pregnancy, disc herniation, vascular malformation, and thrombophlebitis (4–6). Twenty-three pathologically proven cases of spinal cord infarction secondary to fibrocartilagenous emboli have been reported including eight men and 13 women ranging in age from 15 to 75 years (7). Arterial emboli were found in 13 cases, venous emboli in four cases and combined arterial and venous emboli in six cases. Adequate antecedent information was available in only 14 of 23 cases, with 10 of the 14 reporting minor head or neck trauma. Extensive spinal fracture

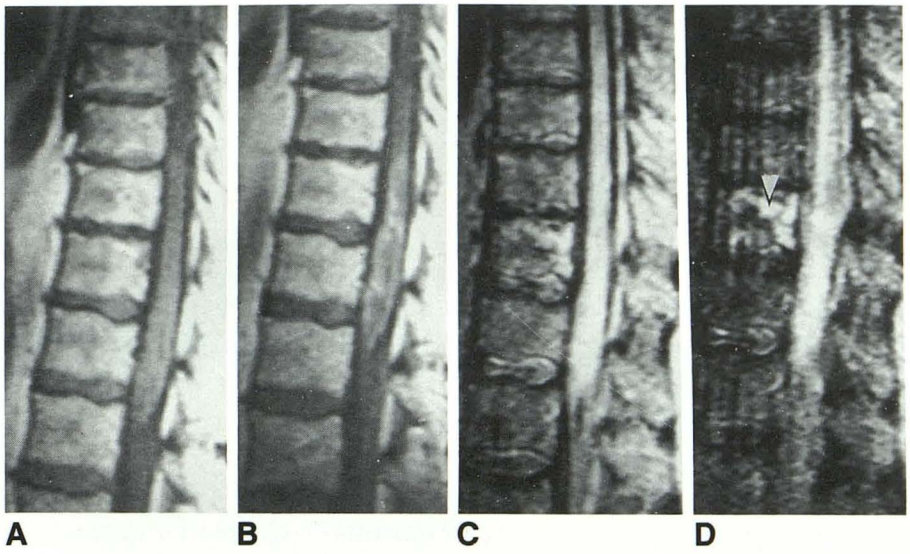

Fig. 3. Day 21 MR study (0.6 T). Pre- (*A*) and post- (*B*) 10 mL of gadolinium-DTPA 400/22 (TR/TE) sequences reveal heterogeneous enhancement from T11 to the tip of the conus. Hyperintense signal is present from T8 to the conus on the first (*C*) and second (*D*) echoes of the 2000/60/120 spin echo sequence. Note continued evolution of increased signal in T11 (*arrowhead*). The signal is greater and more extensive compared to the day 12 examination. (Reproduced with permission from the *New England Journal of Medicine* 1991;324:322–332.)

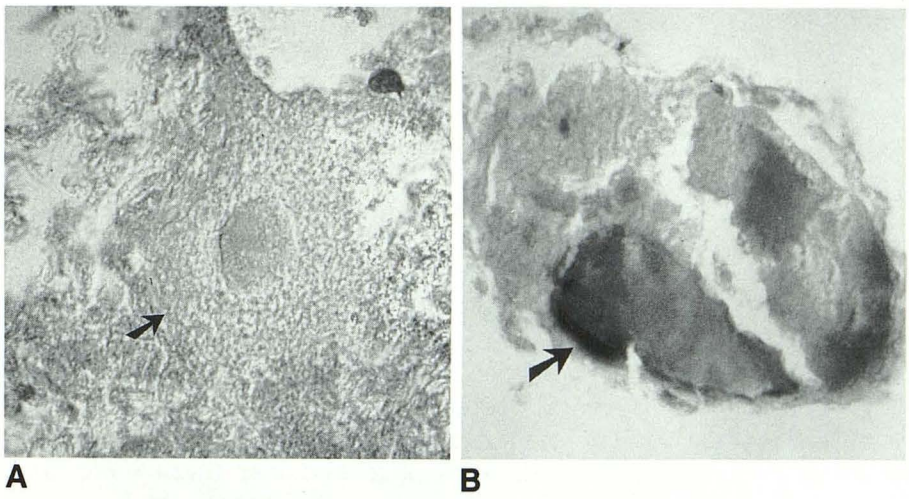

Fig. 4. Necrotic glial fibrils, ischemic anterior horn cell (*arrow*) filled with clumps of lipofuscin pigment, and scattered lymphocytes contained within the biopsy tissue. Original magnification × 800, hematoxylin and eosin stain. (Reproduced with permission from the *New England Journal of Medicine* 1991; 324:322–332.)

Fig. 5. A small artery occluded by deeply staining, fibrocartilagenous embolic material (*arrow*). Original magnification × 800, Alcian blue stain.

or dislocation were not present in these cases, although an occult fracture was observed in the case reported by Srigley et al (8). In our case, there was no evidence of Schmorl nodes, loss of disk space height, or disk herniation. Lifting or carrying was associated with at least two of the cases (9–10). Although our patient performed some minor lifting, it is not clear if this was a predisposing factor. No specific lifting event was correlated with the onset of symptoms. In general, onset of symptoms is acute consisting of severe neck or back pain rapidly followed by a neurologic deficit that becomes maximal within hours. Subsequent recovery is poor (11).

The neuropathologic diagnosis of fibrocartilagenous emboli can be difficult since detection of emboli requires specific attention to all of the regional vascular structures. This is quite time consuming and labor intensive, sometimes requiring assessment of innumerable sections for detection. The sparsity of reported cases may in part be attributed to this problem, and the overall incidence of fibrocartilagenous spinal cord infarction may be much higher than currently reported.

Several theories have been advanced to explain the entry of disk material into the vasculature of the spinal cord including: 1) lateral disk rupture with penetration of disk material into the adjacent radicular artery and hence into the cord microcirculation (12), 2) axial loading of the spinal column associated with elevated intradiscal pressure and injection of disk material into small arteries that are present specifically within degenerating disks or in the disks of children, followed by retrograde flow into the radicular arteries and hence into the arterial system of the cord (12) and 3) extrusion

of degenerated disk material present within Schmorl nodes into the veins of the vertebral body, followed by retrograde migration of this material into the venous circulation of the cord (13). Spinal arteriovenous communications have been invoked to explain the presence of concurrent arterial and venous emboli seen in 25% of cases (14, 15). Vuia and Alexianu (15) showed that arteriovenous shunts exist between arteries and veins surrounding the spinal ganglia and nerve roots, as well as between arteries and veins in the epidural space. The shunt vessels were reported to "exceed capillary caliber." Mechanistically, the following scenario might ensue: axial loading on the spinal column could push disk material through endplate defects into the venous sinuses of the vertebral body marrow cavity. Disk material could then flow or be forced into epidural veins via the basivertebral plexus, aided by the pressure effects from axial loading. Once in the epidural venous system, access to the arterial system occurs through the arteriovenous shunts in the epidural space, again aided by elevated venous pressures. Disk material then could be carried into the cord circulation by normal arterial flow patterns. Although Schmorl nodes and degenerative changes in the endplates and nucleus pulposus are common, embolization of disk material is a rare phenomenon. The reason for this may be that spinal A-V communications are uncommon, or that the high axial loading pressures necessary to produce retrograde flow in existing A-V shunts are difficult to achieve. Alternatively, many cases of acute transverse myelitis may in fact be unrecognized embolic spinal cord infarction.

Radiologic assessment with myelography and CT in this group of patients has been unrevealing (7, 8, 11, 13). Even mild cord swelling, as observed in our case on the CT myelogram, has not, to the best of our knowledge, been reported with these methods. Four cases of MR imaging in spinal cord infarction have been reported previously. Kestle et al (7) indicated that MR investigation performed in their pathologically proven case approximately 9 days after onset of conus infarction "did not help with the diagnosis." The appearance of the conus, the type of MR examination, and the quality of the MR examination were neither shown nor discussed. MR imaging was performed in one case by Brown et al (16) and in one case by Vandertop et al (17) shortly after onset of cervical cord infarction (exact time unspecified in each case), demonstrating cervical cord enlargement with increased signal on the T2-weighted spin-echo sequences. There was no evidence of increased signal on T1-weighted images or decreased signal on T2-weighted images to suggest the presence of hemorrhage. Three-year follow-up in the case of Vandertop et al showed cervical cord atrophy, with high signal on the long TR short TE sequence presumably representing gliosis. Casselman et al reported a cervical cord infarct that had normal signal on T1 and T2-weighted sequences within 24 hours of the infarct (18). Follow-up imaging at 1 week in this case demonstrated normal signal on the T1-weighted images, but increased signal on the T2-weighted images. There were enhancing lesions in the anterior portion of the cord following gadolinium administration. No mention was made of abnormal signal in the vertebral bodies in any of these reported cases.

Normal or decreased signal on T1-weighted images and increased signal on T2-weighted images, as described in the case of Brown et al, the case of Yuh et al, and as seen in our case, are the dominant signal changes in spinal cord infarction, but are nonspecific since numerous pathologic states including neoplasms and inflammatory disease have identical findings. Increased signal on T1-weighted images (day 12) and decreased signal on T2-weighted images (day 5) were not reported in any of the cases presented by Yuh et al., or any of the previously mentioned authors. However, none of the cases of Yuh et al were imaged in the subacute phase of the infarct (greater than 4 days and less than 4 months). The 1-week follow-up study in the case of Casselman et al did not show evidence of hemorrhage. The presence of the curvilinear zone of decreased signal on T2-weighted images in our case (Fig. 1) suggested hemorrhage (intact deoxygenated red blood cells or ferritin/hemosiderin), or flow void in an abnormal vascular structure such as an arteriovenous malformation. Because of these findings, it was not possible to exclude a treatable lesion, and surgery was undertaken.

There was lack of enhancement after gadolinium-DTPA infusion on day 5 in our case, but enhancement was noted on day 21. One of two patients in the series of Yuh et al who received gadolinium in the early phase (<4 days) enhanced in a zone thought to be peripheral to the infarct; the two remaining patients scanned at greater than 4 months did not enhance. Enhancement in the anterior aspect of the cord was observed in the case of Casselman et al 1 week after onset of

symptoms, but was not observed in the first 24 hours. Although cerebral infarcts typically enhance with gadolinium, the enhancement may not appear within the first 5 days. Imakita et al (19) reported enhancement in three of seven cerebral infarcts between 4 hours and 5 days, whereas nine of nine infarcts demonstrated enhancement between days 6 and 10. From the limited spinal cord data, it seems that the timing of enhancement in cord infarction may be similar to cerebral infarction, where lack of enhancement in the acute phase is followed by enhancement in the subacute phase, and may be related to poor contrast delivery to the infarct or to delayed breakdown of the blood-spine barrier. These observations emphasize that the signal abnormalities and enhancement pattern could be quite complex resulting in broad differential diagnostic possibilities, including neoplasia, inflammation, or vascular malformation, with or without hemorrhagic components. If an infarct is indeed present, then the occurrence of hemorrhage may be an indicator of the type of infarct (ie, venous vs arterial). Those infarcts that were thought to be the result of anterior spinal artery occlusion showed no evidence of hemorrhage (although time of imaging may not have been optimal to detect the transitory components of the hemorrhage). Venous infarcts tend to be hemorrhagic. Hemorrhage in our case may have been the sequelae of emboli present in the venous system.

An important finding in the cases of Yuh et al, and in our case, was the presence of increased signal on T2-weighted images in one or multiple vertebral bodies. This abnormal signal was not present on day 5 in our case, but was seen to evolve on days 12 and 21 (Figs. 2 and 3). The etiology of this finding in our case is uncertain since confirmation is lacking. However, several explanations exist including: 1) axial loading of the spinal column resulting in an acute compression fracture with intravertebral disk herniation, or 2) infarction of the vertebral body as a result of vasoocclusion from disk emboli. Vasoocclusion seems the most plausible explanation since each of the cases of Yuh et al with this finding had serious aortic disease, including aortic occlusion in two cases and aortic dissection in one case. *Abnormal signal, or more importantly, evolution of signal abnormality in a vertebral body associated with a nonexpansile or slightly expansile cord lesion should suggest the possibility of spinal cord infarction.* Confusion between infarction and degenerative disease of the vertebral body should not occur, since the pattern of signal change is distinct from degenerative disease due to the geometry of the vascular supply to the vertebral body (1). The presence of vertebral body pathology in association with intramedullary disease of the spinal cord without evidence of direct epidural or transdural extension between the two pathologic regions tends to reduce the likelihood of other diagnoses such as neoplasm, infection, and vascular malformation. In fact, the abnormal signal in the vertebral body may be at a lower level from the infarct because the origin of the supplying radiculomedullary artery is usually more caudal (1).

From the clinical point of view, spinal cord infarction due to occlusion of the anterior spinal artery versus infarction due to fibrocartilagenous emboli may be quite different. Fibrocartilagenous embolic disease would create difficulty in diagnosis from the standpoint of neurologic examination, since the dissociation of sensory modalities seen with the classic anterior spinal artery syndrome may not be present (3). These emboli can extend into the anterior and posterior arterial systems that are linked via the vasa corona, and can enter the venous system, leading to unpredictable regional ischemia in the cord.

Treatment of spinal cord infarction is mainly supportive. Not until progress is made with therapeutic agents that limit the damage resulting from ischemic injury will tissue rescue be feasible. Early diagnosis and differentiation of venous versus arterial vasoocclusive disease may be beneficial since venous, nonhemorrhagic infarction due to thrombosis progresses more slowly and is more protracted than the arterial counterpart, indicating that intervention with thrombolytic agents early in the course of thrombotic venoocclusive disease may improve the outcome (4). It is now possible to detect spinal cord infarction early in the course of the disease, but differentiating arterial from venous sources awaits further study.

Spinal cord infarcts should be considered in the differential diagnosis of all patients with slightly expansile cord lesions associated with T1 and T2 prolongation in the appropriate clinical setting. Serial imaging can document the evolution of hemorrhage and breakdown of the "blood-cord" barrier, and can demonstrate evolving signal changes in nearby vertebral bodies that should help to confirm the diagnosis. Knowledge of the MR imaging findings in spinal cord infarction should perhaps lead to early diagnosis and, there-

fore, more effective management of patients with spinal cord infarction. In the future, noninvasive imaging methods may become available for visualizing the anterior spinal arterial system. Our current philosophy concerning compressive lesions of the spinal cord may need to be expanded to include impairment of flow within the anterior spinal artery as an important source of spinal cord dysfunction.

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