MR imaging of spinal cord and vertebral body infarction.


http://www.ajnr.org/content/13/1/145
MR Imaging of Spinal Cord and Vertebral Body Infarction

William T. C. Yuh,1 E. Eugene Marsh, III2 Angela K. Wang,3 James W. Russell,2 Francis Chiang,4 Timothy M. Koci,4 and Tony J. Ryals1

Purpose: To study the usefulness of MR in the evaluation of spinal cord infarctions and associated findings. Materials and Methods: MR examinations of 12 patients (10 men and two women) were reviewed retrospectively. Onset of symptoms of spinal cord ischemia was abrupt in all patients; MR was performed 8 hr to 4 months after onset. Contrast-enhanced MR was performed in four of the patients. Results: Abnormal MR findings of the spinal cord included abnormal cord signal (11 of 12), best demonstrated on T2-weighted images, and morphologic changes (cord enlargement during the acute phase in nine patients and cord atrophy during the chronic phase in two), best demonstrated on T1-weighted images. Vascular abnormalities (aortic) were detected by MR in four of the 12 patients. Three of these four patients also had abnormal bone marrow signal, predominantly in the anterior half (one) or in multiple areas near the endplate and/or deep medullary portion of the vertebral body involving several vertebrae (two). T1-weighted images were not sensitive in detecting signal changes in either the bone marrow (two of three) or spinal cord (nine of 12). Enhanced MR imaging was performed in four patients (two in the acute phase and two in the chronic phase) and showed diffuse enhancement of the spinal cord proximal to a relatively unenhancing distal conus in one of the two patients imaged during the acute phase. No abnormal enhancement was noted in the other three patients. Conclusion: MR is a useful means of detecting spinal cord infarction and associated vascular and bony changes. The patterns of bone marrow abnormalities reflect the underlying pathophysiology of the blood supply to the spinal cord and bone. The associated vascular and bone marrow abnormalities serve as additional information for the diagnosis of spinal cord infarction.

Index terms: Spinal cord, infarction; Spinal cord, magnetic resonance


Although there are no good epidemiologic data on the prevalence of spinal cord infarctions, they are generally considered to be rare (1–9). Some investigators think they occur more frequently than is commonly reported (5, 6). Ischemic spinal cord infarctions are difficult clinical diagnoses. In the past, radiologic diagnosis has been considered a diagnosis of exclusion. Myelography and computed tomography (CT) can help identify cord swelling, but these techniques are limited in their ability to visualize internal cord structure. In 1971, Di Chiro (7) reported the value of selective spinal cord angiography in improving recognition of occlusive vascular disease affecting the spinal cord. MR has proved to be a valuable noninvasive way to evaluate spinal cord disease. This article discusses the magnetic resonance (MR) findings associated with spinal cord and vertebral body infarctions and reviews the literature.

Materials and Methods

MR examinations of 12 patients with a clinical diagnosis of spinal cord infarction were reviewed retrospectively. There were 10 men and two women 17–85 years old (mean, 56 years). All patients had abrupt onset of symptoms of spinal cord ischemia.

All MR examinations were performed from 8 hr to 4 months after the onset of symptoms. Eleven patients were
Results

Clinical information and MR findings are summarized in Table 1. All 12 patients had a diagnosis of spinal cord infarction, either by clinical judgment (11) or angiography (one) (Figs. 1-4). A possible cause of infarction was identified in nine of the 12 patients: vascular surgery (three), aortic dissection (one), subarachnoid bleeding (one), hypotension (two), strangling (one), and acute disk herniation (one). Three patients had acute spinal cord infarctions without any known precipitating factors. Only one patient (from among the three without any known cause) (case 8) had an angiogram that showed an occluded right lumbar artery (L2). The diagnoses in the other 11 patients were based on clinical presentation and physical examination. All patients had clinical follow-ups of 2 months to 1 year. In four of the 12 patients, only minor improvement was seen in the neurologic deficits.

Abnormal findings were seen on MR images of the spinal cord in 11 of 12 patients. These included high signal intensity of the cord on the T2-weighted image in 11 of 12 patients and enlargement of the cord on the T1-weighted image in nine (Figs. 1, 2, and 4). In two patients without cord enlargement, MR studies performed 4 months after the onset of symptoms showed an atrophic cord with abnormal T2 signal. In only three of the 12 patients was abnormal cord signal seen on the T1-weighted image. Abnormalities in neural structures could not be evaluated by MR imaging in one patient because of the relatively low position of the lumbar coil (case 3) (Fig. 3). However, the absence of flow-void phenomena in the distal aorta due to complete occlusion and abnormal bone marrow signal on the T2-weighted image, located predominantly in the anterior portion of the vertebral body, were demonstrated in this patient. These findings support the clinical
diagnosis of spinal cord infarction caused by acute aortic occlusion.

Vascular abnormalities (aortic) were detected in four of the 12 patients. These included complete aortic occlusion (cases 1 and 3), DeBakey III aortic dissection (case 2), and saccular aneurysm with intraluminal clots (case 4). In three of these four patients, vascular abnormalities were first detected by MR. Abnormal bone marrow signal was seen in these three patients also and was demonstrated best on the T2-weighted image (Figs. 1–3). One of the three patients had a lesion involving predominantly the anterior half of one vertebral body (Fig. 3), whereas the other two had multiple bone marrow lesions located near the endplate and/or deep medullary portions of several vertebrae (Figs. 1 and 2). Abnormal bone marrow was demonstrated on the T1-weighted image (Fig. 3) in only one of the three patients in whom abnormal bone marrow was apparent on the T2-weighted image. One patient had a 1-year follow-up MR study that demonstrated persistence of bone marrow findings with a reduction in lesion size and a typical triangular configuration.
located both near the endplate and in the deep medullary portion of the vertebral body (Fig. 1D). These triangular areas may be the most vulnerable regions of the vertebral body during ischemia and may represent the true infarcted area.

Contrast-enhanced MR images were obtained in four of the 12 patients, two patients in the early phase of ischemia and two in the chronic phase. Only one of the two patients in the early phase showed diffuse enhancement of the spinal cord proximal to a sharply delineated, unenhancing distal conus. No significant abnormal enhancement was noted in the other three patients (one early phase, two chronic phase).

Discussion

Thirty-one pairs of segmental arteries and their regional equivalents supply the entire spinal column and surrounding structures including muscle, bone, and neural derivatives (1–19). In most cases, all these tissues at each level, with the exception of the spinal cord territory, receive their major blood supply from the two (one on each
side) segmental arteries or their equivalents at the same and/or adjacent level (7, 8, 10, 17-19) (Figs. 5-7). Branches of the segmental arteries or their equivalents course dorsally and further divide into extraspinal branch arteries to supply muscle, bone, and nerve roots and intraspinal branch arteries (radicular arteries) to supply bone and neural structures including the spinal cord, dural meninges, and epidural structures (8, 10, 17-19) (Fig. 6). The radicular arteries are the first branches of the dorsal division of the segmental arteries or their regional equivalents. They enter the intervertebral foramina as either a single vessel or a number of vessels accompanying the emerging veins and spinal nerves. After entering the spinal canal, these paired radicular arteries on each side of the vertebrae may ultimately divide into a triad of vessels: the posterior central and prelaminar arteries to supply primarily the bone and, inconsistently, a radiculomedullary artery (medullary feeder) to supply primarily the spinal artery of the spinal cord (7, 8, 10, 17-19) (Figs. 5 and 6).

Unlike the paired posterior central and prelaminar arteries, the radiculomedullary artery may occur on only one side and does not always occur

Fig. 4. Case 8: 68-year-old previously healthy man had acute onset of paresthesia progressing to paralysis of both lower limbs within 2 hr.

A, Parasagittal T1-weighted image (350/20) obtained 48 hr after onset of symptoms shows enlargement and decreased signal (straight arrows) of distal spinal cord. Note that renal artery (curved arrow) and inferior border of conus are slightly lower than disk level.

B, Axial T2-weighted image (2000/80) obtained through conus shows abnormal cord signal (arrows) involving predominantly gray matter.

C, Enhanced parasagittal T1-weighted image corresponding to A shows diffuse abnormal enhancement of distal spinal cord (large solid arrows) above disk level and unenhancing distal conus (open arrow) below disk level, which may represent true infarcted area. Note relatively linear cutoff (small arrow) between enhancing proximal and unenhancing distal conus just above disk level.

D, Lumbar arteriogram shows occluded right L2 segmental artery (arrows).
at each level. Radicular arteries have an obligatory ascending course since they always follow the nerve roots with increasing obliquity from the cranial to caudal levels (8, 10, 17). Therefore, the levels of spinal cord involvement are frequently not the same as those of the bone marrow, as seen in our patients (Fig. 5). In summary, the vertebral body is supplied mainly by the terminal branches (anterior central artery) derived from the paired segmental arteries at the same level and the posterior central artery derived from the paired radicular arteries at two adjacent levels (Figs. 6 and 7). The blood supply of the spinal cord comes from a limited number of medullary feeders that are derived from a segmental artery or its equivalent at a more caudal level (Figs. 5 and 6).

The blood supply to the entire spinal cord is dependent mainly on three longitudinal arterial trunks: a single anterior spinal artery and paired posterior spinal arteries. These spinal arteries extend from the medulla oblongata to the conus medullaris (1, 4, 7–17) (Fig. 5). Although these longitudinal trunks are usually continuous, the anterior spinal artery is narrowest in the midthoracic region and widest in the cervical region (9). The number of anterior radiculomedullary arteries supplying the anterior spinal artery rarely exceeds nine but ranges from two to 17. The artery of Adamkiewicz is the largest anterior medullary feeder and supplies the thoracolumbar region (Fig. 5). It occurs on the left side in 80% of
Fig. 8. Midparasagittal view of two adjacent vertebral bodies. Upper vertebra shows vascular territories of anterior central (AC) and posterior central (PC) arteries. Lower vertebra shows watershed area (WSA) in deep medullary portion and end arterial territory (EAT) near endplate. Cross-hatched areas have a triangular configuration located within both EAT and WSA and may represent areas more vulnerable to ischemia.

subjects and can arise anywhere between the T5 and L4 levels (T9–L2 in 85%, T9–T11 in 75%, L1–L2 in 10%, and T5–T8 in 15%) (9).

The posterior medullary feeders (radiculomedullary arteries) supplying the posterior spinal artery are more numerous, usually ranging from 10 to 23 (20, 21). The posterior spinal arteries are also supplied by radicular arteries derived from segmental arteries or their regional equivalents (including vertebral arteries and posterior inferior cerebellar arteries). In most cases, the artery of Adamkiewicz supplies the entire lumbosacral cord, including the posterior spinal arteries.

Because of the limited but critical sources of blood supply to the spinal cord, any pathologic processes that interfere with this crucial blood supply may result in ischemia and/or infarction in the spinal cord (1, 3–7, 11, 17–19, 22–36). Most spinal cord infarctions occur at the upper thoracic region or thoracolumbar junction. Infarctions in the latter site may result from occlusion of the artery of Adamkiewicz, which is frequently the only blood supply to the thoracolumbar junction (11, 17). In the upper thoracic regions, medullary feeder arteries are sparse, the anterior spinal artery is narrow, sulcus arteries are fewer and smaller, the spinal canal is narrow, and the spinal cord is in a watershed area between major feeding arteries (1, 8, 9). The vertical extent of spinal cord infarction may have from one to 15 segments, depending on the vascular anatomy of the cord and extent of occlusion. Single segmental infarction is frequently caused by ischemia in the watershed area, as occurs in hypotension (17).

Single segmental infarction may also be seen in diseases that affect the small end arteries, such as emboli or focal vasculopathies. All of our patients with bone marrow abnormalities had vertical cord involvement involving several levels, probably caused by a more extensive disease of the aorta.

Although abnormal spinal cord signals were clearly demonstrated in most of our patients, the earliest MR image was obtained 8 hr after onset. Therefore, the earliest time that abnormal findings can be detected by MR remains to be determined. Only four of our patients had enhanced MR studies performed at various periods of cord ischemia. The efficacy of gadopentetate dimeglumine has yet to be determined, especially with regard to early detection and prognostic value. The cause of the sharp demarcation between the enhancing spinal cord and unenhancing distal conus is unknown (Fig. 4).

The prevalent distribution of abnormal bone marrow signal as seen in our limited number of cases has not been described previously. The recognition of abnormal bone marrow signal may be an important finding in establishing a vascular cause for an acute spinal cord syndrome. The blood supply of the bony spine includes anterior central, posterior central, prelaminar, and postlaminar branches (Fig. 6). The radicular arteries (intraspinal branch arteries) usually provide the greater part of the blood supply to the vertebral body (posterior central arteries) and arch (prelaminar arteries).

Anterior central arteries consist of many terminal branch vessels that come directly off the segmental arteries (Fig. 6). They penetrate the cortical bone of the vertebral body and supply the anterior and anterolateral portions. The central and posterior portions of the vertebral body are supplied mainly by four posterior central arteries. These arteries further divide into cranial and caudal branches, which supply two adjacent vertebral bodies (17–19) (Figs. 6 and 7). The cranial branches run upward and medially across the posterior surface of the vertebral body to enter a foramen near the center of the body. The caudal branches run downward and medially a similar distance toward the center of the body of the next distal vertebra. There are, therefore, four diagonal arteries, two from each side, converging centrally to enter the posterior surface to supply
artery occlusion may not always result in bone ischemia because of the excellent collateral circulation of the vertebral body (duplicate anterior central and quadruplicate posterior central arteries) (Fig. 4). Therefore, a wide disparity may exist between the level of spinal cord involvement and that of bone involvement, especially in patients who have a broad range of aortic disease with various degrees of occlusion at different vertebral levels.

The MR findings of avascular necrosis (AVN) in the region of the femoral head, hand, knee, and foot have been reported previously and typically show low signal intensity on T1-weighted images and high signal intensity on T2-weighted images (37–43). The MR appearance of vertebral body ischemia caused by aortic occlusion or dissection has not been described previously. This may be related to the difficult clinical and radiologic diagnoses in patients with acute aortic occlusion. There is only one report of MR findings of vertebral AVN, which was caused by recent trauma in five patients with radiologic evidence of compressed vertebrae containing an intravertebral vacuum cleft indicative of AVN (44).

All vertebral lesions in our patients demonstrated high signal intensity on T2-weighted images that was less obvious on T1-weighted images. In addition, a low-signal peripheral ring frequently seen in AVN was not seen in our limited number of cases during the acute phase. These are not characteristic MR findings of AVN involving the extraspinal bony structures, as reported previously (37–43). During the early phase of AVN, inflammatory cells, necrosis, and edema are the major components in the area of ischemia, whereas new bone formation and fibrosis predominate in the later phases (37, 38, 42, 44). Because these elements have different appearances on MR images, variability in the MR findings among patients and over time is to be expected, depending on which phase of AVN is present. All three patients with bone marrow abnormalities were imaged during a very early phase of ischemia (within first 48 hr), when necrosis, inflammation, and edema were predominant in the ischemic area without significant fibrosis and new bone formation. Therefore, our MR findings of bone ischemia are somewhat different from those of AVN in the extraspinal bone in that T2-weighted images were more sensitive than T1-weighted images and a peripheral low-signal ring was not seen during the acute phase of bone ischemia.
In conclusion, we found MR to be useful for detecting spinal cord infarction. Abnormal MR findings include abnormal signal of the spinal cord, best demonstrated on T2-weighted images, and changes in cord morphology (swelling or atrophy), best demonstrated on T1-weighted images. Other MR signs of ischemia include vascular or bone marrow abnormalities, which may serve as additional information to facilitate a correct diagnosis. The T1-weighted image is not as sensitive as the T2-weighted image in detecting cord signal changes or bone marrow abnormalities. The earliest time that MR can detect spinal cord infarction and the prognostic value of MR imaging with and without gadopentetate dimeglumine require further study.

Acknowledgments

We thank Daniel Loes, MD, and Yutaka Sato, MD, for reviewing the manuscript and Ronald Bergman, PhD, for assistance in the preparation of the anatomic drawings.

References

5. Albert ML, Greer WER, Kantowitz W. Paraplegia secondary to hypotension and cardiac arrest in a patient who has had previous thoracic surgery. Neurology 1969;19:915–918
Please see the Commentary by Mikulis on page 155 in this issue.