Gadolinium-Enhanced MR Imaging of Infarction of the Anterior Spinal Cord

We describe a case in which hyperflexion of the neck resulted in temporary insufficient blood supply and subsequent infarction of the anterior part of the spinal cord. Only a few cases of gadolinium-enhanced imaging of cord infarctions have been reported, and these have been associated with arteriovenous fistulas [1].

Case Report

A 42-year-old previously healthy man experienced paresthesias in both hands 5 min after he had flexed his neck extremely far when trying to creep under a barbed wire. Three hours later he was unable to turn a key with his right hand. Twelve hours later he could not sit up in his bed. At admission, neurologic examination showed a complete flaccid paralysis in the C7–T1 myotomes on both sides. Moderate paresis of the upper motor neuron type was present in the left leg. Pain and temperature sensations were lost in the C4–T8 dermatomes; proprioceptive sensation was spared. Plain radiographs of the cervical spine, CT scans of the cervical spinal cord, and duplex sonograms of the vertebrraland carotid arteries were normal. An MR study of the spine was performed within 24 hr of the injury. T2-weighted, 2000/22, 90/2 (TR/TE/excitations), and T1-weighted spin-echo images, 500/20/3, obtained before and after IV administration of Gd-DOTA were normal. During the following days, motor loss in the arms and legs gradually disappeared. The dissociated sensory loss persisted in the C4–T8 dermatomes. Sagittal T1-weighted images obtained 1 week after the injury were normal, but a high-intensity lesion was seen anteriorly in the cervical cord at the C5 level on the sagittal T2-weighted images (Fig. 1A). The presence of pulsation artifacts made it difficult to evaluate the caudal extension of the lesion on these images. However, postcontrast images showed enhancement anteriorly in the cervical cord at levels C4 to C7 (Fig. 1B). The diagnosis of anterior spinal artery syndrome was confirmed, and the MR-based diagnosis of ischemia or infarction was made. A cervicothoracic spinal angiogram excluded an arteriovenous fistula. MR images obtained 3 months later showed cystic lesions anteriorly in the cervical cord from C5 down to C7 (Fig. 1C). Mild paresis of both hands and loss of the sensations of pain and temperature in the C6–T8 dermatomes persisted.

Discussion

Infarction of the cervical spinal cord is rare, especially in young people. Usually the territory of the anterior spinal artery, supplying the ventral two thirds of the spinal cord, is involved [2, 3]. Some known causes of spinal cord infarction in young adults are cardiac surgery (clamping of the aorta), arteriography, spinal surgery, carotid or vertebral artery dissection, polyarteritis nodosa, and endarteritis [3]. Often, infarction is associated with an AVM [1, 2]. In our case, these causes were excluded, and interruption of the blood supply due to the extreme flexion of the neck was hypothesized. Clinical findings associated with infarction of the anterior spinal cord, the anterior spinal artery syndrome, include motor paralysis and dissociated sensory loss below the level of the lesion. Within 2 hr, our patient had motor paralysis of the lower motor neuron type in both arms and of the upper motor neuron type in the left leg and loss of the sensations of pain and temperature below the level of the lesion.

MR imaging of spinal cord infarction has been reported; many of the cases were associated with arteriovenous fistulas [1, 4]. These fistulas are not always visualized on the MR images and should be excluded in young patients with infarction of the spinal cord when no obvious cause is known. The infarcted spinal cord can have an increased diameter, but when the diameter remains normal, as in our case, the radiologist must rely on the signal intensities within the spinal cord. The pathophysiologic changes in neural tissues, leading to the different signal intensities on MR images during the different phases of infarction, have been described [5]. Infarcts first can be seen on T2-weighted images as high-signal lesions during the acute phase. However, in the first hours after the injury, even T2 images can remain normal. Once the blood-cord barrier is affected, enhancement of the ischemic or infarcted region can be seen after IV administration of gadolinium [6]. In this subacute phase, high-intensity lesions are seen on T2-weighted images. In our case, the extent of the infarction was seen better on the contrast-enhanced T1-weighted images during this phase; on T2-weighted images, the caudal extent was more difficult to evaluate because of pulsation artifacts. Similar findings were reported with use of gadolinium in the study [5] in which brain infarctions were visible only on enhanced T1-weighted images. Only three cases of contrast-enhanced infarctions of the spinal cord, all due to arteriovenous fistulas, have been reported [1]. In the chronic phase, these infarctions have a typical low signal on T1-weighted images and high signal on T2-weighted images.

J. W. Casselman
E. Jolie
I. Dehaene
L. Meeus
A. Z. St.-Jan
B-8000 Brugge, Belgium

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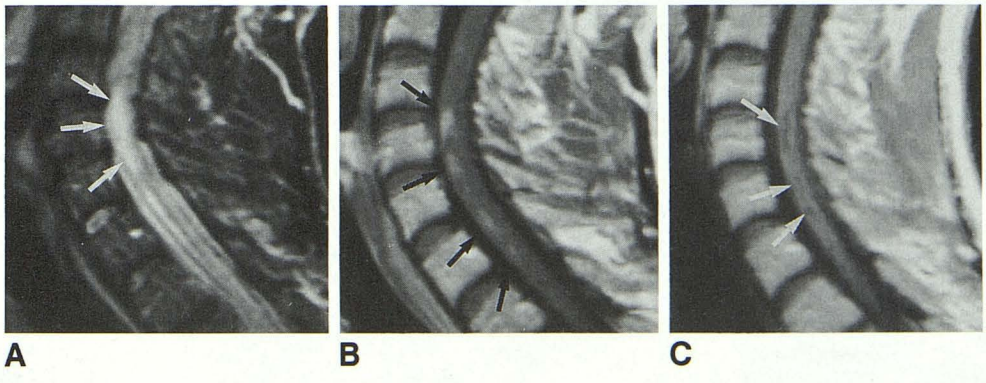

Fig. 1.—Infarction of anterior spinal cord.

***A*, T2-weighted sagittal image (2000/90) of cervical spinal cord 1 week after injury shows a high-intensity lesion (*arrows*) in cord at C5 and C6.**

***B*, Enhanced sagittal T1-weighted image (500/20) during subacute phase (1 week after injury) shows enhancing lesions in anterior part of cervical cord from C4–C5 to C7–T1 (*arrows*).**

***C*, Sagittal T1-weighted image (500/20) 3 months after *B* shows a low-intensity lesion in anterior part of spinal cord at levels C5 and C6 (*arrows*). A similar lesion was seen at C7 level on an adjacent sagittal image.**