MRI OF THE ANTERIOR SPINAL ARTERY SYNDROME

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ABSTRACT

MRI of the anterior spinal artery syndrome was demonstrated at the lower thoracic level and the conus medullaris, in a 63-year-old female diabetic patient. The size and the signal of the cord was not change on T1WI and there was central bright signal at the cord on T2WI. Aortic dissection was noted at the abdominal aorta in the axial images.

CASE REPORT

A 63-year-old female diabetic patient, had sudden onset of back pain and paraplegia, 12 hours prior to the MRI study. The motor power of the lower extremities was of grade 0, the reflex of the lower extremities was absent. There was a decreased pinprick sensation from L1 downward. The touch, proprioceptive and vibration sense was intact. The sphincter tone was loose. The bulbocavernosus test was absent. Clinical diagnosis was anterior two-third spinal syndrome.

MRI study was performed and showed normal size of the lower thoracic cord and the conus medullaris (Fig.1). The signal of the cord on T1WI was normal and the signal was increased in the cord on T2WI and was a central type of hypersignal (Fig. 2,3). The abdominal aortic dissection was shown clearly at the axial view of both T1WI and T2WI (Fig. 3,4). The contrast medium was not given due to short onset-examination interval.

INTRODUCTION

The diagnosis of anterior spinal artery syndrome is made from the characteristic clinical presentation (1,2). Onset is often abrupt with the maximum deficit occurring either immediately or after progression over a few hours. Neurologic deficits include initial flaccid paralysis and depression or absence of muscle stretch reflexes. As time progresses the reflexes become hyperactive and involved muscle groups atrophy. A sensory level is present, below which pain and temperature sensations are not perceived. Sensation of proprioception and vibration, mediated through the dorsal columns in the posterior aspect of the spinal cord is preserved. Bowel and bladder paralysis are common features of the clinical picture. Occlusion of the spinal artery is rare.

We present a case of anterior spinal artery syndrome, studied by MRI.

DISCUSSION

The spinal cord has a unique pattern of blood supply with one anterior and two posterior arteries. In the lower cervical cord and upper thoracic segments of the thoracic cord, the anterior spinal artery is supplied by two or four anterior radicular arteries arising from the vertebral, deep cervical, superior intercostal, and ascending cervical arteries (1,3-5). Radicular arteries are less prominent in the midthoracic cord. The thoracolumbar region is supplied by the great anterior radicular artery of

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Adamkiewicz. The cauda equina is supplied by lower lumbar, iliolumbar and lateral sacral arteries.

The anterior spinal artery supplies approximately 70% of the blood to the cord, including all of the gray matter except for the posterior horns, and the corticospinal tracts. It does this in a centrifugal fashion via numerous central arteries that traverse the anterior median fissure and alternately penetrate the cord to the right or left. Within the inferior of the cord there are no anastomoses, and the central penetrating vessels are essentially end arteries. The paired posterior spinal arteries supply approximately 30% of the cord, including the posterior horns, posterior columns, and a peripheral rim of white matter. They do this in a centripetal fashion via numerous peripheral perforating arteries that are richly connected by anastomotic channels (6-8). The periphery of the gray matter is at the border zone of these centrifugal and centripetal circulations.

The intrinsic arterial supply to the cord is directly proportional to the cross-sectional area of the gray matter, which is most abundant in the thoracolumbar segment. The central gray matter has a much higher metabolic rate than white matter and consequently receives five times more blood flow (6-8).

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 (9). Most spinal cord infarctions occur at the

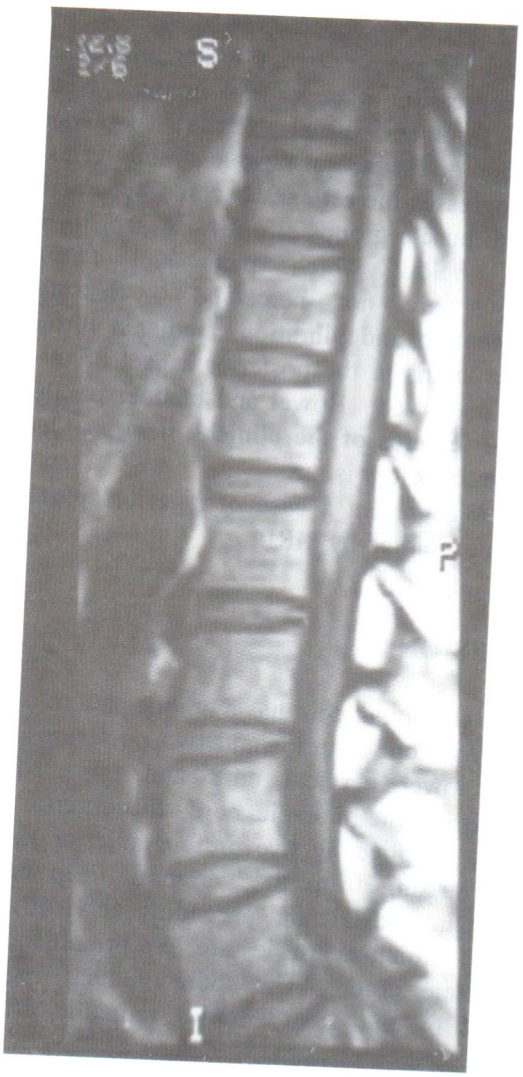

Fig.1 Sagittal view, T1WI MRI study at the thoraco-lumbar junction showed a normal sized cord with unchanged signal pattern

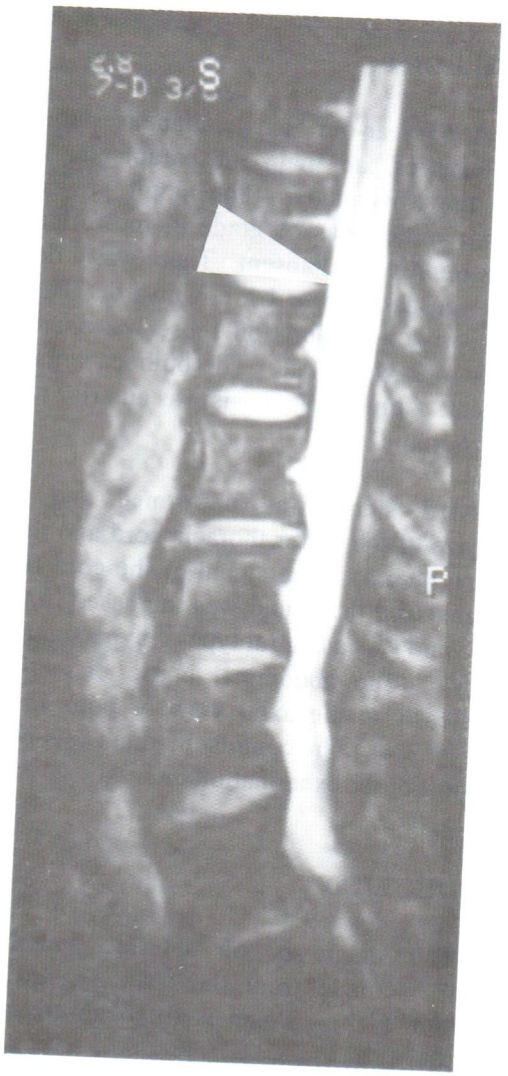

Fig.2 Sagittal view, T2WI MRI study at the thoraco-lumbar junction showed an increased signal diffusely in the central part of the cord

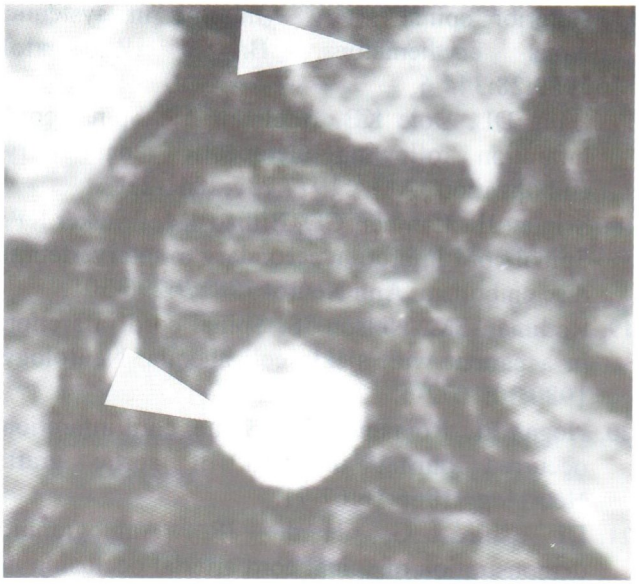

Fig.3 Axial view, T2WI, showed again a hypersignal central type at the cord and an abdominal aortic dissection

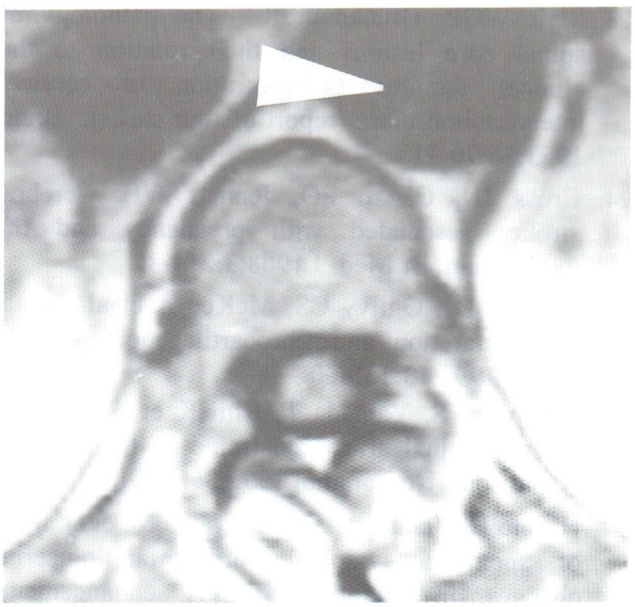

Fig. 4. Axial view MRI showed an abdominal aortic dissection.

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 (10). 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 (11). 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 (12). Single segmental infarction may also be seen in diseases that affect the small end arteries, such as emboli or focal vasculopathies.

Spontaneous anterior spinal cord infarction primarily affects individuals with severe atherosclerotic disease or aortic dissection (13), infection (14), vasculitis (15,16), embolic events (17,18), sickle cell anemia (19), surgery (20), radiation (21), trauma (22), bulging disc (23), associated with pregnancy (24).

Patient age in reported cases ranges from 15 to 75 years (2). Treatment of spinal cord infarction is mainly supportive. Early diagnosis and differentiation of venous versus arterial vaso-occlusive 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 veno-occlusive disease may improve the outcome (9,3). 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.

Magnetic resonance imaging is a sensitive modality in evaluating the spinal cord for infarction. T1-weighted MR scans in acute cord infarction may demonstrate an enlarged cord. Central or anterior intramedullary high signal is typically present on T2WI. Enhancement following contrast may be initially absent but occurs a few days to a few weeks following symptom onset (25-28). Follow-up scans may show focal cord atrophy with myelomalacia and residual high signal intensity on T2WI (27).

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