

## Anterior Spinal Artery Syndrome

The anterior spinal artery syndrome was first described by Spiller [1] in a case of thrombosis of this artery. The diagnosis usually is made on the basis of clinical findings because the cause of the syndrome remains obscure in most cases [2]. We report a case of anterior spinal artery syndrome in which immediate and 3-year follow-up MR showed a signal abnormality from C1 to T3.

### Case Report

A 17-year-old boy, previously in good health, suddenly had paresthesias in his right hand and arm while he was sitting on his bed doing his homework. Within 5 min, the right arm became paralyzed. After 30 min, he also had paresthesias in his left hand and arm; these became completely paralyzed in 5 min. He took a hot bath but then was unable to get out of the bathtub because of a severe paraparesis. Physical examination showed a flaccid paralysis of the left arm and leg, a proximal weakness of the right arm, a paralysis of the right hand, and a weakness of the right leg. No tendon jerks in the left arm or leg could be elicited. Slight biceps, brachioradial, and knee jerks were present on the right. Vibration sense, position sense, and perception of light touch were completely intact. Perceptions of temperature and pain were absent below T3–T4. Urinary retention occurred the next day, with priapismus. After 14 days, a Horner syndrome developed on the left side. Complete analyses (including

all cultures) of blood, urine, and CSF showed no abnormalities. CSF was obtained twice, by means of a lateral cervical puncture (for the myelogram) and by a lumbar puncture. A plain chest radiograph showed no abnormalities of the aortic arch. A cervical myelogram was normal. MR imaging clearly showed normal vertebrae, a somewhat swollen spinal cord, and a lesion in the anterior part of the cord extending from C4 to C6 (Figs. 1A and 1B).

The patient showed a slow but gradual improvement and was transferred to a spinal cord injury center for further rehabilitation. After 18 months, he was able to walk independently. No incontinence of urine or stools with a normal urge was present. Physical examination showed a weakness only in the triceps, extensor digitorum, and flexor digitorum muscles in the right arm and of the infraspinatus, triceps, extensor digitorum, flexor digitorum, deltoid, supraspinatus, rhomboid, and biceps muscles of the left arm. The other muscles of the left arm were paralyzed. The right leg had not improved much proximally, but distally, muscle power was MRC grade III. The left leg showed a slight distal weakness. The tendon jerks in the left arm and leg were increased, with an ankle and patellar clonus and a Babinski sign on the left. Perceptions of temperature and pain were diminished below level T2–T3. Repeat MR imaging 3 years later showed that the spinal cord had become thinner, especially on the left side (Figs. 1C–1E). The lesion now extended upward to the C1–C2 level.

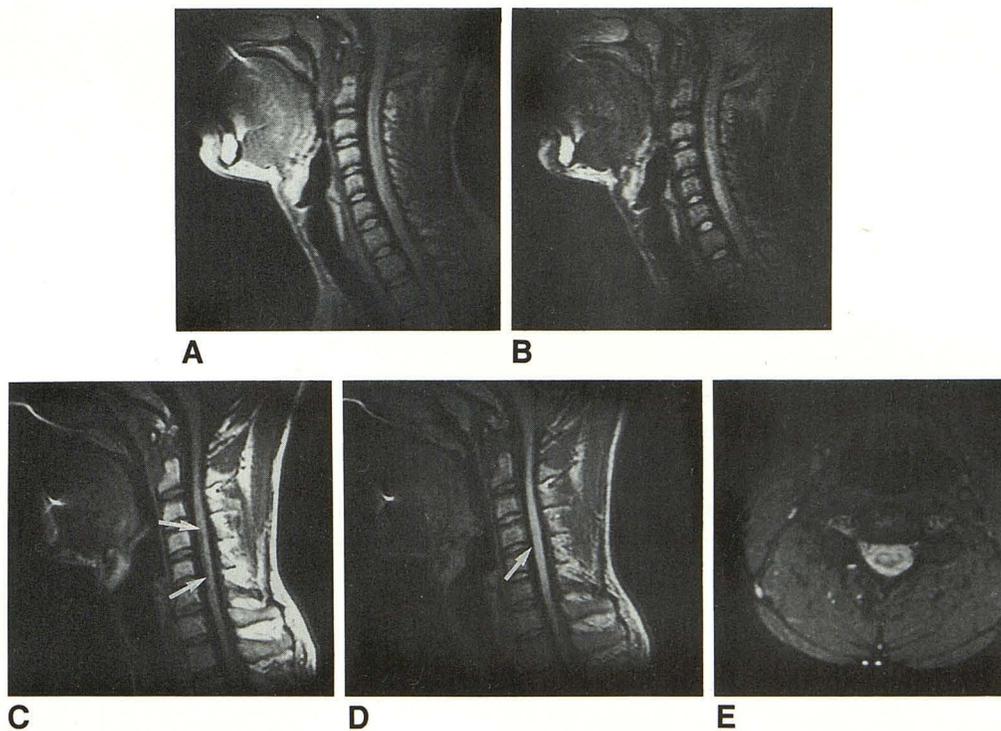


Fig. 1.—Anterior spinal artery syndrome.

A and B, MR images obtained at admission. Sagittal SE image, 350/35/4 (A), of cervical spine shows normal vertebrae and a somewhat swollen spinal cord. Slightly elevated signal intensity in anterior part of cord may be caused by placement of surface coil. T2-weighted sagittal SE image, 2400/96/2 (B), shows a lesion with high signal intensity in anterior part of cord, extending from C4 to C6.

C–E, MR images obtained 3 years after A and B. Midsagittal SE image, 350/35/4 (C), shows spinal cord is thinner than it was earlier. Anterior part (arrows) has a lower signal intensity than in normal cord. Long TR short TE study, 2400/30/2 (D), shows high signal intensity (arrow) in anterior part of cord extending upward to C1–C2 and downward to T3. Midcervical transverse GRE image, 140/35/flip angle 15° (E), shows cord is much thinner than in previous study (A and B), more on left than on right side. Lesion in anterior part of cord has high signal intensity.

## Discussion

The causes of anterior spinal artery syndrome vary widely. Autopsy reports cite causes such as arteriosclerosis, thrombosis due to cervical spondylosis, cholesterol embolus, intervertebral disk embolism, syphilitic vasculitis, and neck trauma [3, 4]. In clinical reports without autopsy findings, other causes also have been mentioned: spinal cord angiomas, aortic occlusion or coarctation, infections, intervertebral disk protrusion, gas embolism, emboli from bacterial endocarditis, use of oral contraceptives, cocaine abuse, systemic lupus erythematosus, and as a complication after surgery for the correction of scoliosis or abdominal aortic surgery [5-7]. In a review of the literature, Foo [2] found 60 cases with adequate clinical information. In 14 patients, the cause remained unknown, as in our patient. The neurologic deficit was less severe and the outlook for recovery seemed more favorable in patients who had postinfectious myelopathy or an unknown cause than in patients who had occlusive disease of the anterior spinal artery. In our patient, the rapid onset of symptoms suggested a vascular cause. However, a spinal angiogram for visualization of both vertebral arteries and the anterior spinal artery seemed to have no therapeutic consequences, especially as MR imaging had excluded any surgically accessible lesions.

The immediate MR images showed a slightly elevated signal intensity in the anterior part of the cord (Fig. 1A), although it is uncertain whether this was caused by placement of the surface coil. On the T2-weighted images (Fig. 1B), a high signal intensity was seen, showing a clear lesion from C4 to C6. An extensive hemorrhage into the spinal cord was highly unlikely, as findings on the myelogram and CSF examination were normal. MR images obtained 3 years later showed thinning of the spinal cord, more pronounced on the left than

on the right, a finding that corresponded with the clinical picture. However, no definitive explanation exists for the upward and downward extension of the lesion. Perhaps this is the first sign of change in spinal cord substance, representing gliosis and microcystic degeneration, which years later could lead to a syringomyelia.

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