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Anterior Spinal Artery Syndrome Associated with Severe Stenosis of the Vertebral Artery

Kensuke Suzuki, Kotoo Meguro, Mitsuyoshi Wada, Kei Nakai, and Tadao Nose

Summary: We describe a 55-year-old man with quadriparesis and impaired pain and temperature sensation in whom T2-weighted MR images revealed a high-intensity lesion in the cord at C3–4. Angiography showed occlusion of the right vertebral artery and severe stenosis of the left vertebral artery. We concluded that the stenosis of the vertebral artery led to the anterior spinal artery syndrome and to a disturbance of consciousness.

Anterior spinal artery syndrome, first reported by Spiller et al (1), consists of pain at the level of the lesion, quadriparesis or paraparesis, disturbance of pain and temperature sensation, and urinary incontinence. Foo (2) reported 60 cases in which angiomas, infections, aortic lesions, and obstruction of the anterior spinal artery were responsible for anterior spinal artery syndrome, but angiographic evidence of occlusion of the anterior spinal artery was present in only a few of these patients. We report a case of anterior spinal artery syndrome in which the patient had severe stenosis of a vertebral artery, occlusion of the contralateral vertebral artery, and MR findings of spinal infarct.

Case Report

A 55-year-old man with a 10-year history of diabetes and hypertension experienced pain and paresthesia in his shoulders. The pain gradually increased, and bilateral weakness of the upper and lower limbs developed over a 1-month period. On admission, the patient had quadriplegia, decreased deep tendon reflexes, sensory disturbance of pain and temperature, urinary incontinence, and shoulder pain. An MR study revealed an intramedullary high-intensity lesion on T2-weighted images at the C3–4 region (Fig 1A–D). The lesion did not enhanced after administration of contrast material (Fig 1B).

CSF examination showed two lymphocytes, a normal IgG level, and a protein level of 221 mg/L (normal range, 20 to 30 mg/L). We suspected multiple sclerosis (MS) on the basis of the patient's clinical course, despite the normal IgG level. Treatment with methylprednisolone for 3 days (1000 g/day) improved the symptoms somewhat.

A month after admission, the shoulder pain suddenly recurred, the quadriparesis worsened, and the patient became drowsy. Emergency angiography, performed because the clinical course suggested a vascular accident, revealed occlusion of the right vertebral artery and severe stenosis of the left vertebral artery (Fig 1E). Following the angiographic examination, the patient lapsed into a coma, which was effectively treated with induced hypertension. The angiographic findings strongly suggested concurrent ischemia of the brain stem and spinal cord consequent to the impaired vertebral artery flow. Treatment consisted of percutaneous transluminal angioplasty (PTA), which resulted in satisfactory dilatation of the origin of the left vertebral artery (Fig 1F). The patient became alert despite discontinuation of the induced hypertension, and follow-up angiography 1 month later showed no restenosis of the left vertebral artery.

An MR study performed 15 months after PTA showed the T2 high-intensity lesion at C3–4 and a decrease in the size of the spinal cord due to prior infarction (Fig 1G and H). A repeat brain MR examination showed new abnormalities. The patient's symptoms persisted, but no new neurologic deficits appeared during a 2-year follow-up period.

Discussion

Anterior spinal artery syndrome has characteristic symptoms that consist of sudden onset of flaccid quadriparesis with pain, dissociated sensory loss below the level of the lesion, and bladder dysfunction. The causes of the syndrome reportedly include arteriosclerosis, infection, vasculitis, embolic events, sickle cell anemia, cervical cord herniation, surgery, and trauma (2, 3). Angiographic demonstration of occlusion of the anterior spinal artery is relatively rare (4).

Although the clinical diagnosis of anterior spinal artery syndrome is sometimes difficult and the diagnosis can be reached only by exclusion (5), high-resolution MR imaging can show axial sections of the spine in detail. In our patient, the lesion appeared as a large, single, round area (the so-called "owl's eye" appearance) and was located in the center of the cervical spinal cord, a finding compatible with ischemia of the territory of the anterior spinal artery.

MR images of spinal MS, which is difficult to distinguish from spinal infarct, may show enlargement of the cervical cord with a high-intensity intramedullary lesion on T2-weighted sequences (4, 6, 7). Tartaglino et al (8) reported that the majority of plaques in

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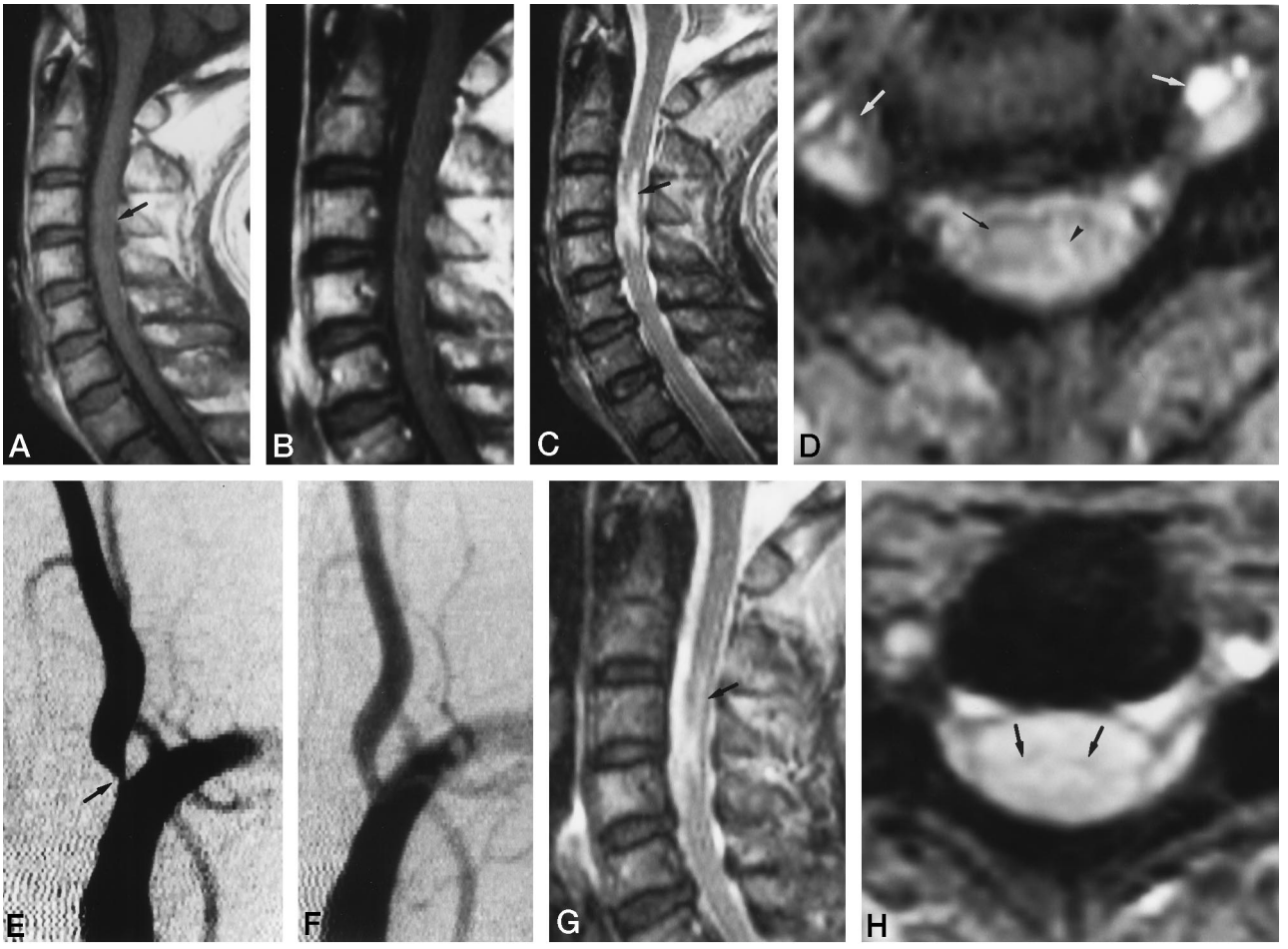

FIG 1. 55-year-old man with anterior spinal artery syndrome.

A, T1-weighted (500/20 [TR/TE]) MR image on admission shows low-intensity lesion at C3–4 (*arrow*).

B, Corresponding MR image after contrast administration shows no enhancement of the lesion.

C, T2-weighted (3500/110) MR image on admission shows high-signal-intensity lesion in the C2–3 region, including the anterior part of the spinal cord (*arrow*).

D, Axial T2-weighted MR image (500/20; field echo, 256; flip angle, 25°) shows a rounded high-signal-intensity area on right side of the gray matter in the C3–4 region (*black arrow*). Linear abnormality on left side may be artifactual (*arrowhead*). Signal voids of vertebral arteries are absent (*white arrows*).

E, Angiogram of left subclavian artery shows severe stenosis at origin of left vertebral artery (*arrow*).

F, Angiogram of left subclavian artery after PTA shows decreased stenosis of left vertebral artery.

G, Sagittal T2-weighted (3500/110) MR image 15 months after PTA shows persistence of high-signal lesion (*arrow*).

H, Axial T2-weighted (500/20) MR image at C-3 15 months after PTA shows decrease in size of spinal cord.

spinal MS are multiple, two vertebral body segments or less in length, peripherally located, and occupy less than 50% of the cross-sectional area of the cord. The lesions of spinal MS usually reside in the white matter, which is located peripherally in the spine.

In our case, the symptoms appeared gradually and the results of CSF analysis were abnormal, prompting the initial diagnosis of MS; however, the recurrence of symptoms, including sudden onset of shoulder pain, quadriparesis with a dissociated sensory deficit, and disturbance in consciousness, suggested a vascular event. In patients with cervical spondylitis, the concentration of protein in CSF may increase without elevating the IgG level (9). Furthermore, MS was unlikely in this case, because the clinical symptoms and T2 high-intensity intramedullary lesion on cervical MR images remained unchanged for 15 months and follow-up brain MR studies revealed no new lesions. The patient's deterioration in consciousness and the motor and sensory deficits strongly suggested that both brain stem and cervical cord ischemia was due to the impaired vertebral artery flow. PTA of the stenotic left vertebral artery resulted in improvement in the patient's level of consciousness, but his other neurologic symptoms remained.

Conclusion

In this case, ischemia of the brain stem and cervical spine caused stenosis of a vertebral artery and occlusion of the contralateral vertebral artery. Although the patient's clinical course and CSF data confused the diagnosis, axial high-resolution MR images showed a lesion with a large, single, round area (the

so-called "owl's eye" appearance) located in the center of the cervical spinal cord. This finding was convincing in helping us distinguish spinal infarct from spinal MS.

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