Spontaneous Vertebral Artery Dissection Initially Mimicking Myocardial Infarction

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Background and Purpose: Vertebral and carotid artery dissections may present with very different signs and symptoms, making early recognition difficult. However, diagnosis should be established as soon as possible to prevent unnecessary diagnostic investigations and to institute adequate treatment.

Case Description: A 46-year-old man presented with severe intermittent pain of his left upper arm and general discomfort. During extensive cardiological evaluation for suspected myocardial infarction, a severe brain stem syndrome occurred. Ultrasound Doppler studies detected vertebral artery dissection, which was confirmed by angiography.

Conclusions: The unusual initial presentation of vertebral artery dissection delayed an early diagnosis and adequate treatment. Because noninvasive methods are available today, their applications are recommended in similarly uncharacteristic circumstances. (Stroke 1992;23:1021-1023)

KEY WORDS • cerebral arteries • ultrasonics • vertebral arteries

Dissections of the carotid and vertebral arteries are probably still being misdiagnosed if the clinical features associated with a focal neurological deficit (such as ipsilateral headache or neck pain, Horner's syndrome, tinnitus, and ipsilateral lower cranial nerve involvement) are absent. Ultrasound Doppler examination may be useful under such circumstances; reduced flow velocities and a bidirectional Doppler signal throughout the neck (to-and-fro component) may raise the suspicion of dissection and prompt diagnostic confirmation by angiography. It is important to note that delayed angiography can miss the diagnosis and that magnetic resonance imaging with special neck sections and magnetic resonance angiography (MRA) seem to be diagnostic but have not yet been established as routine methods. Moreover, clinical signs and symptoms vary between carotid and vertebral artery dissections, and the latter may more often be oligosymptomatic. Headache or neck pain is reported similarly, occurring in 60–92% and 67–100% of cases of carotid and vertebral artery dissections, respectively, but Horner's syndrome has been found in 20–58% of carotid artery dissections and is only occasionally reported in vertebral artery dissection. Probably if carotid artery dissection is associated. Oculosympathetic paresis is often subtle or incomplete (presenting as only slight ptosis) and may be overlooked on routine examination. The real incidence of lower cranial nerve lesions involving the extracranial glossohypoglossal nerve is not known and is mentioned only rarely in the literature. Interpretation of angiograms may be more difficult in establishing vertebral than carotid artery dissection, especially if physicians are not alert to the diagnosis or if the dissection changes rapidly. Therefore, diagnosis can be missed even if highly suspicious features are apparent. In a recent case report typical signs, symptoms, and course suggesting carotid artery dissection as the most likely diagnosis were described, but no definite diagnosis was made. To focus this important issue further, we describe an unusual warning symptom that delayed diagnosis because of the suspicion of myocardial infarction.

Case Report

A 46-year-old man noted severe intermittent pain of his left upper arm 7 days before admission. The pain subsided within 24 hours, and no other symptoms were noted. No trauma had preceded the pain.

On the day of admission identical pain recurred in association with vertigo and general weakness. On examination the patient appeared ill, he sweated, and his blood pressure was 160/100 mm Hg. A neurological consultant found only slight spontaneous nystagmus to the right and a gaze-dependent horizontal nystagmus with a rotary component more to the right than to the left. There were no other neurological abnormalities, especially no Horner's syndrome, no dysfunction of cranial nerves IX–XII, and no sensory, motor, or coordination deficits. An electrocardiogram showed ST elevation in an anterolateral distribution, suggesting myocardial ischemia, but remained unchanged on the following days. The serum creatine kinase, aspartate aminotransferase, and lactate dehydrogenase levels did not rise, even on several follow-up examinations.

Five hours later bulbar dysarthria developed, with a shift of the soft palate to the right. There was slight hemiparesis on the right side, with pronounced cerebellar limb ataxia. High-dose intravenous heparin was be-
gun, and nimodipine (60 mg b.i.d.) was administered orally. The patient's pain subsided in the following hours.

A cranial computed tomogram showed no abnormality. Continuous-wave Doppler signal spectrum analysis and duplex-system examination revealed a high-resistance flow pattern and typical bidirectional systolic flow components (to-and-fro component) highly suggestive of vertebral artery dissection (Figure 1). Digital subtraction angiography (Figure 2) confirmed occlusion of the left vertebral artery at the origin, revascularized at level C3a with alternating bidirectional flow, which was also present in the distal vertebral artery. The latter was filled by retrograde flow from the right vertebral artery. The above-mentioned flow abnormalities could also be detected by MRA. T2-weighted magnetic resonance images were consistent with left paramedian pontine infarction. Echocardiography and electrocardiograms were negative.

Over 4 weeks the patient's hemiparesis and ataxia gradually improved. The high-resistance flow pattern gradually resolved, as shown by sequential follow-up Doppler studies, indicating recanalization of the left vertebral artery.

Discussion

In summary, this 46-year-old patient presented with pain and symptoms of myocardial ischemia and initially minor neurological abnormalities, that is, nystagmus. He was referred to a cardiology unit, where extensive evaluation was negative. A short time later severe brain stem infarction occurred.

The clinical picture of spontaneous vertebral artery dissection may be characterized by severe posterior headache or neck pain, usually followed by neurological deficits in hours or days. These deficits may consist of the lateral medullary syndrome or cerebellar infarction, but minimal features are probably not rare. This is particularly true for dissections simultaneously associated with carotid dissections, which may present with ipsilateral neck pain, Horner's syndrome, and contralateral long-tract or sensory signs, all indicating carotid rather than vertebrobasilar territory disease. Since the prevalence of both vessels being involved is unknown and treatment for dissections of the individual arteries may be quite different, diagnostic studies must assess both arterial territories. These studies may consist of magnetic resonance imaging or MRA with appropriate anatomical sections, ultrasound analysis with both duplex and transcranial Doppler examination, and confirmation by four-vessel conventional angiography.

When a patient presents with typical symptomatology, the diagnosis may be suspected on clinical grounds, but there is no good evidence to decide how frequently dissection occurs without all of these clinical suspicions. A recent case of vertebral artery dissection presenting with chest pain was investigated by angiography after a
lumbar puncture showed the presence of red blood cells and raised the question of possible subarachnoid hemorrhage. That case and our own presented with an unusual pain localization, which led to presumption of a cardiac origin of the pain. While the studies were not diagnostic, the question as to whether the pain was of central origin or in fact cardiac cannot be answered with complete reliability. Simultaneous multiple noncephalic dissections have been described, and one can hypothesize whether concomitant coronary artery dissection that had not been reported earlier was a feature in the case under discussion. It is possible that, with a higher index of suspicion, earlier Doppler examination or noninvasive but at present less available MRA might have made the diagnosis and allowed the institution of heparin before the onset of severe brain stem syndrome. In our opinion, this is the treatment of choice—after subarachnoid hemorrhage has been excluded—to prevent thromboembolic complications.

References
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