Infarction of the Medulla and Cervical Cord After Fitness Exercises

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The clinical association of a left lateral medullary syndrome with left corticospinal and posterior column deficits is reported in the case of a young woman with a left vertebral artery dissection. The signs are explicable on the basis of occlusion of branches of the vertebral artery to both the upper cervical cord and the medulla. The dissection may have been due to sustained rotation of the neck during fitness classes. In view of the undocumented benefits and the potential unwanted effects of this exercise, it is doubtful whether it should be recommended. (Stroke 1989;20:292–294)

A healthy 32-year-old singer had complained of discomfort in her neck with difficulty in turning her head to the right for 2 weeks prior to admission. She had not experienced headache. On the day of admission, she awoke with paresthesias and numbness in her left face, arm, and leg. She believed that her voice was changed, and swallowing was difficult. She vomited and had a brief episode of vertigo. Her left arm and leg quickly became weak so that she could not walk even with assistance, and her left arm was grossly incoordinate.

Examination showed abnormalities only in the nervous system. There was a left Horner’s syndrome with normal facial skin temperature and color. Minor direction-changing horizontal jerk nystagmus with greatest amplitude to the left, diminished left corneal response, reduced light touch and pinprick sensations on the left side of her face, weakness of the left palate and of the left trapezius muscle, and loss of taste on the left side of her tongue were noted. In the motor system, a pyramidal syndrome involved her left arm and leg. There was obvious left cerebellar dysmetria, and on sitting up she fell to the left. Sensations of touch and pinprick were diminished over the right side of her body, and vibration and proprioception were lost in her left arm, trunk, and leg.

The patient was not taking oral contraceptives and had suffered no recent trauma. She was attending fitness classes in which certain exercises involved maximally turning the neck to either side, then holding the head in the rotated position for 1–2 minutes. These exercises had not formerly caused any symptoms.

Normal investigations included routine hematol-ogy and biochemistry, glucose tolerance test, luetic serology, electrocardiogram, isotope brain scan, x-ray films of the chest and skull, and serum lipoprotein electrophoresis. Cervical spine films showed evidence of minor degenerative change with well-preserved disk spaces. A left brachial angiogram showed that the left vertebral artery was threadlike, having a long segment of narrowing in the midcervical area (Figures 1 and 2), indicative of dissection. At the level of C3 there was a marked increase in the caliber of the vertebral artery, indicating collateral input from the ascending cervical branches of the thyrocervical trunk. The left vertebral artery supplied the territory of the posterior inferior cerebellar artery, which was also of very small caliber.

The patient was considered to have ischemic damage to the left lower medulla and to the upper segments of the cervical cord on the left side. She was treated with anticoagulants and later with antiaggregant agents. She made a slow and incomplete recovery, being left with diminished coordination on the left side and an altered voice, noted by her during her professional work. Markedly abnormal dysesthetic sensations have persisted throughout the left side of her body for 6 years following the episode.
FIGURE 1. Left vertebral angiogram. Artery is thinned throughout its length below C3 level. Double lumen cannot be discerned.

Discussion

Because of the almost exclusively left-sided signs (Horner’s syndrome, trigeminal and bulbar nerve involvement and loss of posterior column sensation, and pyramidal involvement in the left limbs), the patient had initially been thought to have a hysterical conversion reaction and was referred to the psychiatry service, where no abnormality of mental state was detected and a neurology consultation was sought. While the symptoms of vertigo, nausea, and vomiting together with the alternating thermoanalgesia, Horner’s syndrome, and left-sided cerebellar signs are classical for the lateral medullary syndrome, the presence of impaired proprioception and vibration sensation on the left, with added left-sided pyramidal signs, indicated that the lesion had extended below the inferior border of the medulla and into the cervical cord, where it would have involved the (now crossed) lateral corticospinal tract and either the posterior columns or the gracile and cuneate nuclei. Damage to the upper cervical cord was also suggested by the weakness of the homolateral trapezius muscle.

Vertebral artery dissections commonly cause headache and a lateral medullary syndrome, which is often delayed following any original neck trauma, but lateral medullary syndromes should not give rise to weakness nor to posterior column dysfunction. When weakness does occur, serious brainstem infarction is the probable reason, the accompanying alterations in consciousness, respiratory disturbance, and diplopia suggesting upward extension of ischemia in the territory of the basilar artery. Combined medial and lateral medullary infarction has been described with vertebral artery dissections, but in such cases the hemiparesis should have been contralateral to the infarct. The combination of signs reported here has been described in only one other case, but the pathogenesis was not discussed by these authors.

While most cases of induced vertebral artery damage reported in the literature have been caused by cervical manipulation, including chiropractic, yoga has been incriminated in three reports.
Neck trauma, bow hunting, athletic injuries, neck hyperextension, and atlantoaxial dislocations have also been incriminated (reviewed in Reference 3), as has head-turning while driving an automobile and while leading a parade. Spontaneous dissections have been regarded as rare, but vessel wall disease such as cystic medial necrosis, arteritis, and fibromuscular hyperplasia may be predisposing causes. 

The pathophysiology of vertebral artery occlusion has been well studied. The vertebral arteries penetrate the atlanto-occipital membrane and the cervical dura, having emerged from the transverse foramina of the first cervical vertebra; it is at this level that at least 50% of rotation occurs during head turning. Angiography during head rotation has shown such vertebral artery compression at C1, associated with the development of symptoms of brainstem ischemia, and even in normal subjects vertebral arterial flow may be reduced or even halted when the head is maximally turned to the opposite side. In the patient reported here, dissection of the left vertebral artery was the cause. Not only did its major branch, the posterior inferior cerebellar artery (PICA) receive less blood as a result of the dissection, but a reduction in blood flow in those branches of the vertebral artery supplying the crossing pyramidal fibers and the gracile and cuneate nuclei in the upper cervical cord must also have been responsible for the development of symptoms on the same side.

In humans, the PICA supplies branches to the lateral and posterior medulla and gives off the posterior spinal artery as well as its major branch to the posterior inferior cerebellum and inferior vermis. However, branches arising directly from the vertebral artery are probably more important in supplying blood to the lateral medulla, and even in normal subjects vertebral arterial flow may be reduced or even halted when the head is maximally turned to the opposite side. In the patient reported here, dissection of the left vertebral artery was the cause. Not only did its major branch, the posterior inferior cerebellar artery (PICA) receive less blood as a result of the dissection, but a reduction in blood flow in those branches of the vertebral artery supplying the crossing pyramidal fibers and the gracile and cuneate nuclei in the upper cervical cord must also have been responsible for the development of symptoms on the same side.

All the clinical features can be explained by vertebral artery dissection, leading to reduced blood flow in its branches to the lateral medulla above and in its bulbous branches to the cervicomedullary junction below. The signs of neocerebellar involvement indicated ischemia of the homolateral inferior cerebellar peduncles or of the lateral lobes, the former being supplied partly by the lateral branches of the vertebral artery and the latter by the PICA. That the paramedian arteries were not involved is suggested by retention of the right pyramidal tract function, these arteries supplying both right and left pyramids and lemnisci. Such sparing was probably due to the bilateral feeding of blood from the vertebral into the anterior spinal arteries.

Thus, it would seem that prolonged head turning to the right damaged and perhaps predisposed to dissection of the left vertebral artery, causing ischemic damage to the left lateral medulla and to the left side of the upper cervical cord. Attention has not previously been drawn to this predictable constellation of clinical signs, which is explicable on the basis of the vascular supply of the lateral medulla and upper cervical cord from the vertebral arteries.

References

Key Words • dissection • vertebral artery • cerebral infarction
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