Clinically Unidentified Dissection of Vertebral Artery as a Cause of Cerebellar Infarction

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Background and Purpose—Dissection of vertebral arteries has been reported in association with minor neck movements without signs of trauma on the surface of the neck. In addition, injury of a vertebral artery can cause brain infarctions. However, few cases have been reported in which fatal brain infarction was due to nonocclusive, clinically undetected, traumatic thrombus formation in a vertebral artery.

Case Description—A 62-year-old man was hit by a car, and a right cerebellar infarction was found the day after the accident. The cause of the infarction could not be detected by angiography. Although the patient recovered favorably after surgical removal of the right lateral hemisphere of the cerebellum, he died suddenly 2 weeks after the accident. An autopsy and a microscopic study revealed pulmonary thromboembolism and organizing traumatic lesions of the right vertebral artery without occlusion or noteworthy stenosis of the artery.

Conclusions—We concluded that the patient sustained traumatic lesions of the right vertebral artery during the traffic accident 2 weeks before death and that his cerebellar infarction was due to a thrombus resulting from these traumatic lesions. (Stroke. 2001;32:1422-1424.)

Key Words: cerebellar infarction ■ dissection ■ trauma ■ vertebral artery

Extracranial dissection of the vertebral arteries is increasingly being recognized as a cause of stroke. Dissection of the vertebral arteries frequently occurs in association with abnormal neck movements. However, injuries to the extracranial vertebral arteries are commonly ignored or overlooked, because the relative inaccessibility of the arteries makes them difficult to examine at autopsy.

Potsch and Bohl reported on some cases in which the autopsy findings revealed fatal dissection of the vertebral arteries. In such cases, the cause of sudden death was brain stem ischemia, which was caused by occlusion of the vertebral or basilar arteries from thromboembolism or dissection. Given that brain infarction can be caused by thrombi from traumatic lesions in the vertebral arteries, even small traumatic lesions in the vertebral arteries could cause brain infarction in the absence of occlusion of the vertebral or basilar arteries; subsequent death would occur from complications. The present study may be the first report in which postmortem microscopic examination has revealed clinically undetected, nonocclusive, traumatic lesions of a vertebral artery as a cause of cerebellar infarction.

Case Report
A 62-year-old man on a bicycle was hit by a car on his left side. Because he had sustained a fracture of the left clavicle, he was admitted to the hospital. On admission, he was fully conscious, with no arrhythmia on ECG and no neurological symptoms. The white blood cell count was 18 730/μL, the red blood cell count was 499 × 10^6/μL, and the platelet count was 27.0 × 10^4/μL. Vertigo, nausea, and vomiting appeared 10 hours after the accident, indicating a cerebellar disorder. The patient lost consciousness approximately 21 hours after the accident. CT and MRI indicated infarctions of the right cerebellum in the area of the posterior inferior cerebellar artery (Figure 1) and of the right occipital lobe in the area of the posterior cerebral artery. Angiography of the right vertebral artery was unsuccessful because the contrast medium could not flow into it, perhaps because of severe brain herniation. An endotracheal intubation was performed, and for cerebral decompression, the right lateral hemisphere of the cerebellum was surgically removed without delay. After the surgery, extubation was performed after the patient was weaned from oxygen. The patient recovered favorably 6 days after the accident. However, 14 days after the accident, he suddenly collapsed without symptoms of chest pain or dyspnea and died before treatment could be initiated.

Postmortem and Pathological Findings
The patient was 165 cm tall and weighed 62 kg. A laceration that had been sutured and was in the process of healing was
found on the left occipital region of the head; this lesion was a result of the accident. The fracture of the left clavicle appeared to be organizing. The right lateral hemisphere of the cerebellum had been removed. An old hemorrhagic infarct was found in the area of the right posterior cerebral artery, but no brain herniation was found. No atherosclerosis was seen in the arteries of the cerebral basal region. The heart weighed 490 g. Neither myocardial infarction nor thrombosis of the coronary arteries was apparent. The valves of the heart were normal, and no intra-atrial thrombus had adhered to the wall of the left or right atrium. No dissecting aneurysm was seen in the aorta or subclavian arteries. There were 2 noteworthy findings: (1) pulmonary thromboembolism and phlebitis of the legs and (2) traumatic lesions in the right vertebral artery.

Pulmonary Thromboembolism and Phlebitis of the Legs
Thromboembolism was found in the left and right pulmonary arteries. In the pulmonary arteries and the right femoral artery, thrombi that did not adhere to the vessel wall were found. In the left and right great saphenous veins, thrombi adhered to the vessel walls.

Traumatic Lesions in the Right Vertebral Artery
We investigated the vertebral arteries by the method described by Bromilow and Burns and Johnson et al. No fractures of the vertebral bone or occlusive thrombi of the vertebral or basal arteries were seen. At the level of the sixth cervical vertebra, the right vertebral artery contained a small spot of intramural hemorrhage and a tear of the intima, without any visible thrombus (Figure 2). This lesion was not occlusive or significantly stenotic. No atherosclerosis was seen in this artery. Microscopically, there were at least 3 organizing injuries of the intima (Figures 3A, 3B, and 3C), which ran perpendicular to the blood flow.

A grossly apparent intimal injury involved a tear of the intima and the inner portion of the media; this tear was covered by fibroblasts, collagen fibers, and endothelial cells (Figure 3A). Around this large injury were 2 small intimal tears (Figures 3B and 3C). A small organized thrombus was seen (Figure 3B). The arterial wall was dissected into 2 portions by an organizing hemorrhage, and the dissection in the tunica media appeared to extend proximally from the visible injury (Figure 3A). Proliferating endothelial cells surrounded the false lumen of the dissection (Figure 3D), and granulation tissues containing collagen fibers were seen around the injuries (Figure 3E). Thus, the multiple injuries in the right vertebral artery were in the process of healing, indicating that they had occurred during the traffic accident 2 weeks before death. The stretching of the artery seems to have torn it perpendicular to the blood flow.
Discussion

The patient had no past history of atrial flutter or fibrillation, and no intra-atrial thrombus was found. In addition, the atherosclerosis in the cerebral basal region was minimal. Together, these data suggest that an intrinsic cause of the cerebellar infarction was unlikely.

Our study revealed that the right vertebral artery lacked any atherosclerotic lesion that was occlusive or stenotic. However, there were traumatic lesions of the artery at the level of the sixth cervical vertebra; again, these lesions were not occlusive or stenotic. Considering that no occlusive thrombus was seen in the lesions, a thrombus that came from the traumatic lesions of this artery was assumed to be the cause of the right cerebellar infarction. Extension or flexion movement of the neck during the collision with the car likely tore the intima of the right vertebral artery, and platelets exposed to the subendothelial collagen were activated. In turn, thrombi were formed in the revealed media, causing a right cerebellar infarction. The long confinement subsequently caused phlebothrombosis of the legs, and this complication led to sudden death from pulmonary thromboembolism.

Although there have been some reports in which autopsy has revealed fatal dissection of the vertebral arteries, in such cases the cause of sudden death was brain stem ischemia, which was caused by occlusion of the vertebral or basilar arteries resulting from thrombosis or dissection. The present case was clearly different in that no occlusive thrombus of the vertebral or basilar arteries was seen. Potsch and Bohl have proposed that extracranial injuries of the vertebral arteries can be divided into acute and late groups. In the present case, the sudden death appeared to have a late course. However, our subject’s cause of death was not brain stem ischemia due to occlusion of the basilar or vertebral arteries, which Potsch and Bohl proposed was characteristic of the late group.

In the present case, postmortem examination was important in determining whether the brain infarction and death were due to an extrinsic cause (the traffic accident) or an intrinsic one (atherosclerosis). This differentiation was particularly important because such traumatic lesions of the vertebral artery may not be detectable by CT, MRI, or angiography. In consideration of the fact that dissection of the vertebral arteries frequently occurs in association with abnormal neck movements (without signs of trauma on the surface of body), the vertebral arteries should be examined at autopsy whenever the cause of brain stem or cerebellar infarction is unclear.

References

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