Vertebral Artery Occlusion Following Hyperextension and Rotation of the Head

BY S. OKAWARA, M.D., AND D. NIBBELINK, M.D.

Abstract: Vertebral Artery Occlusion Following Hyperextension and Rotation of the Head

This is a case report of a lateral medullary syndrome of Wallenberg following occlusion of one vertebral artery and stenosis of the opposite artery precipitated by combined motion of hyperextension, rotation, and lateral flexion of the head within physiological limits. Sufficient duration of such head position appeared to initiate thrombus formation following stenosis or occlusion of the vertebral artery at the level of the atlanto-occipital joint. Propagation of this thrombus obstructed the posterior inferior cerebellar artery causing infarction of the lateral medullary region. The pathogenesis of this mechanism is discussed.

Additional Key Words: Wallenberg syndrome, vertebral artery thrombosis, posterior inferior cerebellar artery, atlanto-occipital joint

Medullary or upper cervical spinal cord infarction syndrome following traumatic occlusion of the vertebral artery has been described with cervical fracture,1-3 cervical manipulation,4-7 and calisthenics.8 Decreased blood flow through the vertebral artery without cervical fracture or dislocation is presumed to occur with hyperextension9 and/or excessive rotation9 beyond physiological limits by external forces.1-4-9 However, the mechanism of arterial occlusion, thrombosis, and infarction has not been clarified from post-mortem or clinical findings.8

This article presents a description of a lateral medullary syndrome following sustained physiological hyperextension, rotation, and lateral flexion movements of the head for a period of several hours. The angiographical survey demonstrated complete obstruction of the right vertebral artery associated with severe stenosis of the vertebral artery on the left. The clinical course and angiographical abnormalities provide an interesting sequence of events which suggest a probable mechanism of progression for this type of vascular occlusion.

Case Report
A 43-year-old left-handed male draft designer experienced occasional nonvertiginous dizziness for two years. On March 21, 1974, he painted the ceilings of two rooms in his home, beginning in the early morning and finishing in the late afternoon. While painting, he used a roller or brush in his left hand. Therefore, his head was maintained in hyperextension, rotated to the left, and tilted to the right. Occasionally he used his right hand while his head remained hyperextended, rotated to the right, and tilted to the left. During that afternoon, he noted occasional dizziness as he used his right hand. The remainder of the day was not unusual and he was in no particular distress. The next morning, however, on March 22, 1974, he experienced an abrupt onset of severe headache, unsteadiness, numbness of the right side of his face, difficulty swallowing, and loss of taste on the right side of the tongue. As he attempted to climb into bed, he fell to the floor with marked loss of balance, but noted no change in level of consciousness. On March 23rd he was admitted to a local hospital where his ataxia and headache increased gradually. On admission to University of Iowa Hospitals on March 28th, examination revealed an alert and well-oriented man with a blood pressure of 140/80 mm Hg. The cranial nerve examination revealed absence of corneal reflex, and marked impairment of sensation to touch and pin prick along the entire distribution of the trigeminal nerve on the right side. There was marked impaired sense of taste to salt and sweet substances on the right side of the tongue. The salpingo-pharyngeal muscles moved less on the right, and the tongue deviated to the right upon voluntary protrusion. His voice was nasal, although speech articulation was clear. There was no weakness or sensory impairment of the extremities, and the deep tendon reflexes were normal. No Babinski sign was present. There was severe disturbance on finger-to-nose testing on the right and his gait was markedly ataxic with a tendency to fall to the right.

Hematological evaluation revealed a hematocrit of 47 and white blood count of 9,100 with a normal differential. Sedimentation rate was 26 mm per hour and the urinalysis was normal. Serum-extracted cholesterol was 222 mg/dl and triglycerides were 230 mg/dl. The serum lipoprotein electrophoresis showed mild elevation of the pre-albumin pattern. Tc99 brain scan, x-rays of the skull, and computer-assisted tomography were normal. Audiometric evaluation was also normal. On April 3rd transfemoral vertebral and carotid angiography revealed a threadlike narrowing of the right vertebral artery for 1 cm, followed by complete obstruction at the atlanto-axial level (fig. 1). The left vertebral artery showed marked stenosis at the level of the foramen magnum, but the intracranial portion of the left vertebral and basilar arteries appeared normal (figs. 2A and B). A short stem of the right vertebral artery was demonstrated. The large left posterior inferior cerebellar artery, bilateral anterior inferior cerebellar arteries, bilateral superior cerebellar arteries, and bilateral posterior

From the Division of Neurosurgery, Department of Neurology, University of Iowa College of Medicine, Iowa City, Iowa 52242.
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The vertebral artery has a characteristic anatomical course through six foramina transversaria of the cervical vertebrae, passing through the groove on the surface of the arch of the atlas, and then penetrating two strong membranes (atlanto-occipital membrane and cervical dura mater) before entering the cervical subarachnoid space. This artery is particularly susceptible to injury or occlusion following cervical fracture dislocation,\(^2\) cervical traction,\(^3\) hyperextension,\(^4\) and hyperrotation.\(^5-10\) Three main sites of compression associated with fracture dislocation have been described at the level of the fifth and sixth cervical vertebrae, atlanto-axial joint, and atlanto-occipital joint.\(^1\) However, stenosis and/or occlusion of the artery with hyperextension and hyperrotation movements without fracture dislocation are located primarily at the atlanto-occipital joint, atlanto-axial joint,\(^2\) and the foramen magnum.\(^11\) At the atlanto-axial joint the vertebral artery can be narrowed normally or even occluded during normal head rotation to the opposite side as shown by postmortem angiographical studies.\(^12\)

**Discussion**

The vertebral artery was well demonstrated. The carotid angiogram was normal. He was treated conservatively with dexamethasone and he showed slow but gradual improvement. Examination on May 1st revealed marked improvement of speech and swallowing difficulties, but the ataxic gait and loss of facial sensation persisted.

**FIGURE 1**

Right lateral vertebral angiogram performed on thirteenth day following the onset of neurological symptoms. Threadlike narrowing and occlusion are shown at atlanto-axial level. No intracranial circulation is demonstrated.

**FIGURE 2**

Left vertebral angiogram performed on thirteenth day following the onset of neurological symptoms. The anteroposterior film shows the small distal end of the right vertebral artery. Both films show narrowing of the left vertebral artery at the foramen magnum level.

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The vertebral artery is directly covered by m. obliquis capitis inferior and m. intertransversarius between the two foramina transversaria of the atlas and axis. The artery can be compressed by either of these two muscles between the atlas and axis during rotatory movements as it ascends obliquely just medial to the attachments of these muscles at the atlas.\(^2\) The artery then passes through a slit of the strong atlanto-occipital membrane as it travels medially and ventrally in the groove of the atlas, and can be compressed by the membrane between the atlas and occiput during hyperextension. The ischemic syndrome of the brain stem or spinal cord can result from one or more of the following conditions: (1) occlusion of both vertebral arteries, (2) occlusion of one vertebral artery with insufficient contralateral flow in the opposite artery by way of one of its major branches or basilar artery, and (3) thrombosis, embolism or atheroma of the posterior communicating arteries or nearby collateral circulation. This ischemic syndrome has been observed immediately following abrupt hyperextension or excessive rotatory movement of the head without fracture or dislocation.\(^4\) In postmortem findings, the presence of thrombus in the distal vertebral-basilar system\(^6\) suggests that the vertebral arteries can be sufficiently compromised to initiate a propagating thrombus. The distal propagation of a thrombus in an occluded vertebral artery has been well documented in patients with vertebral artery ligation for other reasons.\(^18\)

In the patient described in this report, the movement of the head was neither forced nor powerful, but sufficiently long-lasting so that he had to maintain his head in a hyperextended position, rotated to the left, and tilted to the right while painting the ceiling with his left hand. This position apparently obstructed his right vertebral artery at the atlanto-axial joint as demonstrated by Tatlow,\(^10\) while the patient remained without symptoms while the left vertebral artery could supply the right posterior inferior cerebellar artery. However, he occasionally used his right hand for a short period and kept his head hyperextended, rotated to the right, and tilted to the left. This maneuver apparently obstructed the blood flow in the left vertebral artery. The unsteadiness he experienced in the afternoon as he used his right hand could be interpreted as the result of disturbed blood flow through the left vertebral artery, since in all probability the right vertebral artery was thrombosed. The thrombus had not yet propagated to the origin of the right posterior inferior cerebellar artery at this point. At the end of the day he appeared to have an occluded right vertebral artery at the atlantal level and a stenotic vertebral artery on the left. During the following 12 hours, the thrombus in the right vertebral artery propagated distally because of occlusion proximally. The obstructed blood flow to the right posterior inferior cerebellar artery produced the lateral medullary syndrome the next morning. Propagation of the thrombus could have been enhanced by such factors as low oxygen saturation, structural changes, and a lack of effective collateral circulation as suggested previously by Lindenberg.\(^11\) Although the significance of his dizziness in the past is not clear, such symptoms should be regarded as a warning against sustained self-induced or externally applied hyperextension or rotation of the head in a relatively young patient.

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