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Positional Occlusion of the Vertebral Artery: A Rare Cause of Embolic Stroke

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Summary. A young, previously healthy patient developed embolic occlusions of the posterior cerebral, superior cerebellar, and posterior inferior cerebellar arteries. This patient also demonstrated positional occlusion of the left vertebral artery which may have been the etiology for his embolic stroke.

Key words: Stroke – Positional occlusion of the vertebral artery – Emboli

Turning of the head has been noted to produce occlusion of the contralateral vertebral artery at the C_1-C_2 level [1]. If both vertebral arteries have adequate blood flow the individual is normally asymptomatic. In patients with compromise of the opposite vertebral artery from cervical spondylosis or atherosclerotic disease, turning of the head may produce vertebrobasilar insufficiency. This report concerns a young patient who demonstrated complete occlusion of the vertebral artery upon turning the head and suffered embolic strokes involving the posterior cerebral artery (PCA), superior cerebellar artery (SCA), and posterior inferior cerebellar artery (PICA). We believe that stasis in the vertebral artery, secondary to positional occlusion, produced small thrombi that embolized in major blood vessels, of the posterior circulation.

Case Report

A healthy 19-year-old male experienced intermittent bifrontal headaches and colored flashing lights in his right visual field 2 weeks before admission. On the day before admission he noted the sensation of "the room spinning around" associated with nausea and vomiting. On admission, the neurological examination disclosed right homonymous inferior quadranopsia, lateral and upbeat nystagmus, and listing to the right on tandem gait and Romberg tests. A computed tomographic (CT) scan demonstrated no abnormality. The patient's headache and dizziness slowly resolved and he was discharged after 2 days.

Approximately 24 h after his first admission the patient returned to the hospital complaining of a strange sensation over the left side of his body. On examination he had a complete right homonymous hemianopsia, left dysmetria, and dysdiadokinesia. Nystagmus was noted on lateral gaze bilaterally.

Two weeks later, the patient noted the acute onset of inability to walk, numbness on the right side of the body and the left side of the face, diplopia, nausea, vomiting, and slurred speech. Neurological examination showed vertical nystagmus and left ataxia. A CT scan revealed infarction of the left cerebellar hemisphere. An emergency arteriogram (Fig. 1) demonstrated emboli in the proximal left PCA, SCA, and PICA. Slow flow was also noted in the left vertebral artery at the C_1-C_2 level with the head turned obliquely to the right (Fig. 2).

Over the next 24 h the patient rapidly deteriorated and became obtunded with decerebrate posturing. CT scan (Fig. 3) displayed acute obstructive hydrocephalus with obliteration of the fourth ventricle. An emergency suboccipital crainotomy was performed with partial resection of the left cerebellar hemisphere and decompression of the foramen magnum.

The pathological findings in the operative speciman were of multiple infarcts both acute and long standing. There was no evidence of vasculitis. An extensive workup for the etiology of the emboli was completely negative and included a nuclear medicine cardiac shunt study, echocardiogram, multiple blood cultures, and collagen vascular studies.

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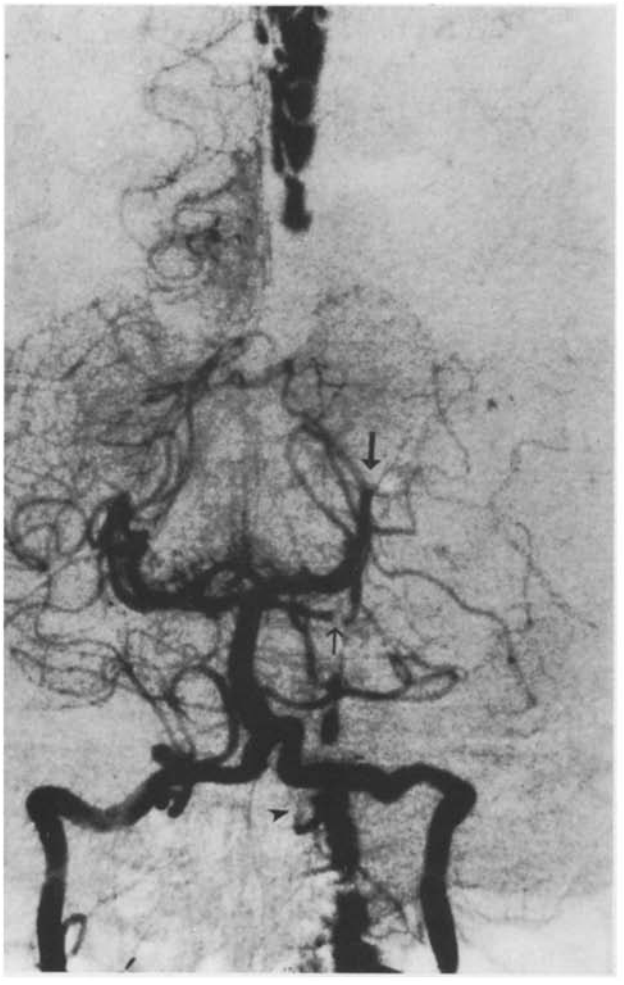

Fig. 1. Left vertebral arteriogram (AP) demonstrating emboli in the proximal left PCA *(large arrow)*; SCA *(small arrow)*, and PICA *(arrowhead)*. Note reflux into contralateral right vertebral artery

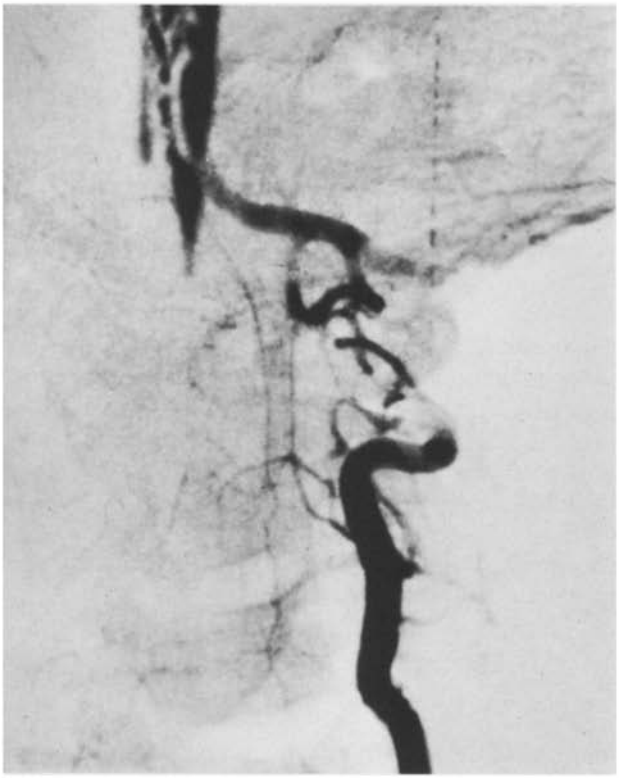

Fig. 2. Left vertebral injection (RPO) showing area of contrast washout by muscular collaterals at C_1-C_2. Decreased flow in left vertebral artery was visualized during angiographic sequence

The patient gradually improved following operation and was discharged on anticoagulants with residual ataxia and listing to the right.

Six months after operation he was readmitted for repeat vertebral angiography. The left PCA and SCA had recanalized. The left PICA was suboptimally visualized. This study, however, was remarkable for total occlusion of the left vertebral artery at the C_1-C_2 level when the head was turned approximately 60° to the right (Fig. 4a). The patient was completely asymptomatic in this position, even though the contrast column remained stagnant in the vertebral artery (Fig. 4b). When the head was placed in the neutral position contrast flowed rapidly into the basilar artery and transiently down the codominant right vertebral artery (Fig. 4c). In retrospect this situation occurred during the first arteriogram when slow flow as noted in the vertebral artery while the head was held in the oblique position.

At present, the patient is neurologically stable and is no longer on anticoagulant medication.

Discussion

It is well recognized that the vertebral artery, on the contralateral side to which the head is turned, may be occluded in three separate locations:

1. The intervertebral foramen above C_5-C_6,
2. the atlantoaxial joint, and
3. the occipitoatlantal joint [2–6]. If both vertebral arteries have adequate flow, the patient remains asymptomatic when one vertebral artery is positionally occluded. If the ipsilateral vertebral artery is hypoplastic and the lumen of the contralateral vertebral artery is decreased by cervical spondylosis or atheromata, symptoms of vertebrobasilar insufficiency or infarction may be produced [7–11]. Ischemia and infarction of the brainstem have been reported follow-

ing chiropractic manipulation of the neck with subsequent occlusion and damage to the vertebrobasilar system [12–15]. Trauma to the neck has been said to precipitate thrombosis of the vertebral artery [16]. Patients with rheumatoid atlantoaxial subluxation may compress the vertebral artery and develop positional vertebrobasilar insufficiency and infarction [17]. This case is remarkable in that despite thorough evaluation for cardiac disease, endocarditis, vasculitis, and drug abuse, no other apparent etiology for the embolic strokes could be detected.

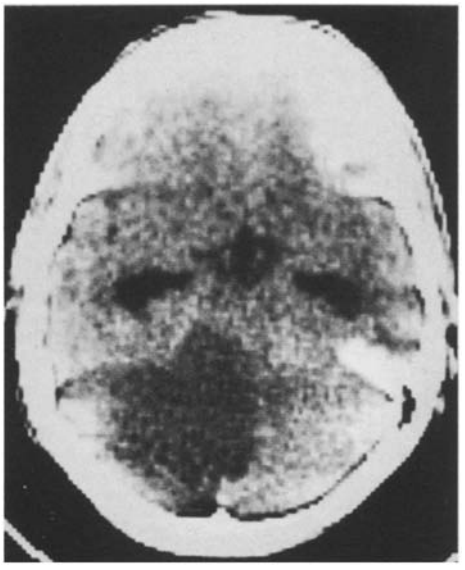

Fig. 3. Plain CT scan with large area of low absorption in left cerebellar hemisphere. There is obliteration of fourth ventricle and dilatation of temporal horns

The history suggests that the patient had multiple embolic events. The first occurred approximately 2 weeks before his initial hospital admission and resulted in right inferior quadrananopsia. This was not visualized on the first CT scan. Two weeks after his initial admission the patient developed embolic stroke demonstrated angiographically involving the left PICA, SCA, and PCA. This resulted in sufficient posterior fossa mass effect to produce acute obstructive hydrocephalus, decerebrate posturing, and obtundation requiring emergency surgical decompression of the posterior fossa. The fact that all the clinical symptoms occurred in the left vertebral artery distribution strongly points to it as the source of the emboli. It would be most unusual for cardiac emboli to lodge repeatedly in branches of the vertebrobasilar system.

We believe that this patient had a vertebral artery that was completely occludable without producing symptoms. Figure 4b is taken 10 min after contrast injection while the patient maintained his head in the oblique position. Throughout this period of time he remained asymptomatic. The posterior circulation was perfused from a codominant right vertebral ar-

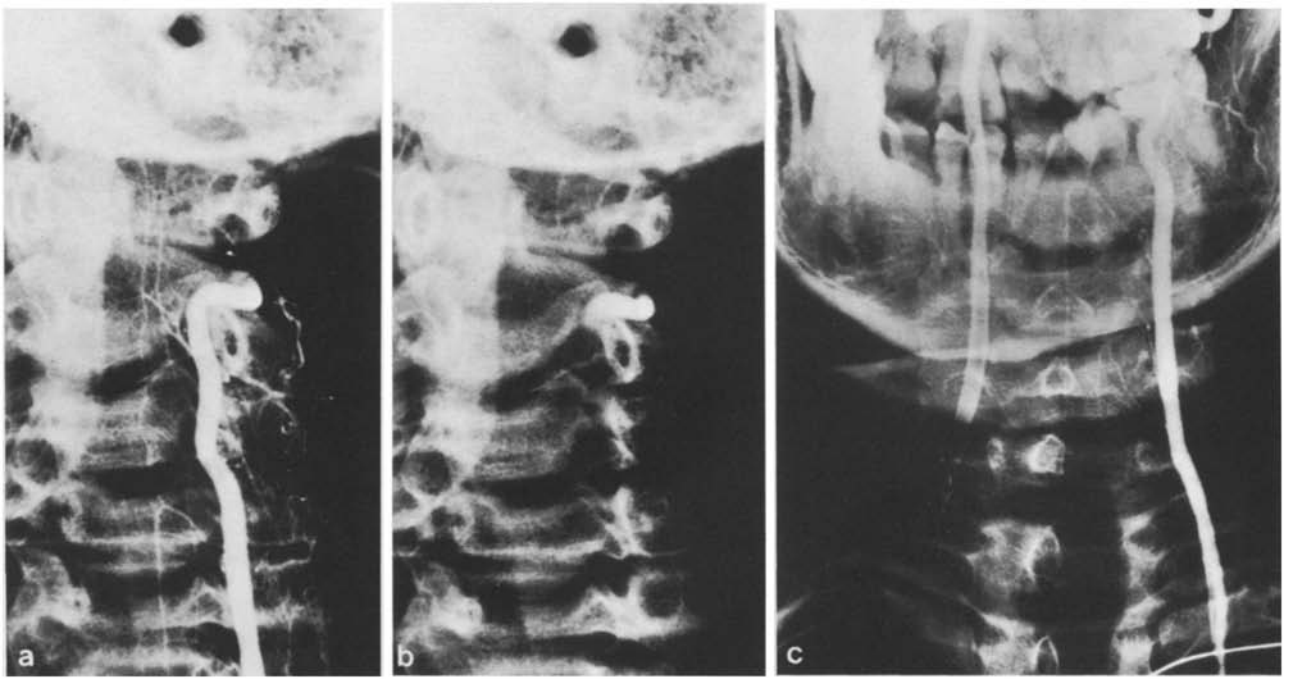

Fig. 4a–c. a Left vertebral injection (RPO at 60°) revealing stagnation of contrast column. **b** Same position 10 min later with contrast still stagnant at C_1-C_2. **c** Repeat injection with head in AP position illustrating normal flow in left vertebral artery and transient reflux down codominant right vertebral artery

tery, which was refluxed from the left vertebral injection when the patient's head was placed in the neutral position (Fig. 4c) This was also the situation at the time of the original arteriogram.

Obstruction of the vertebral artery at a normal atlantoaxial joint results from forward subluxation of the contralateral side of the atlas on the axis [1, 18]. Our patient did not suffer from vertebrobasilar insufficiency with head rotation, rather, this patient's occludable vertebral artery was apparently the source of stasis producing small thrombi and subsequent embolism. We would conclude that although a patient with an occludable vertebral artery may be asymptomatic with respect to cerebral blood flow, this vertebral artery should be considered as a rare source for embolic stroke. We feel that this unique case represents such a situation.

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