Compromise of blood flow in the vertebral arteries associated with head rotation is generally considered an uncommon cause of vertebrobasilar insufficiency. It can occur at virtually any level of the cervical spine. We present two cases of transient total occlusion of the left vertebral artery at the atlantoaxial joint during head rotation to the right. In one case, the contralateral vertebral artery was severely hypoplastic, and surgical C1–C2 fusion to protect the larger left vertebral artery resulted in relief of the clinical symptoms related to head rotation.

Case Reports

Case 1

A 55-year-old man had a 1-year history of 10–12 episodes of total visual loss lasting 2–3 min. These episodes occurred when he turned his head far toward the right (the first time while backing up his truck). He reported no other neurologic symptoms and had an unremarkable neurologic examination. Cerebral angiography done in the standard manner with the head and neck in neutral position demonstrated a normal left vertebral artery (fig. 1A). However, total occlusion of the dominant left vertebral artery occurred at the foramen transversarium of the axis during marked rotation of the head toward the right (fig. 1B). The right vertebral artery was markedly hypoplastic on a congenital basis. The left posterior communicating artery was not visualized on either vertebral or left common carotid angiography and was presumably very small in caliber. The right posterior cerebral artery was opacified exclusively through a large right posterior communicating artery during right common carotid angiography. The proximal segment of the right posterior cerebral artery was not visualized on the vertebral angiography, indicating severe congenital hypoplasia of this segment of the circle of Willis. Thus, there was no readily available collateral pathway to the posterior circulation from the intracranial carotid circulation. It was believed that the patient

Fig. 1.—Case 1. Cerebral angiograms in frontal projection with left subclavian artery injection. A, In neutral position without head rotation, left vertebral artery appears normal. B, With head rotated toward right, 10 sec after contrast injection, occlusion of left vertebral artery is seen at level of foramen transversarium of axis. C, 3 months after C1–C2 fusion, with head rotated toward right: No compromise of left vertebral artery is seen.
would benefit from C1–C2 fusion to prevent a potential vertebralbasilar region ischemic infarct. Complete patency of the left vertebral artery during head rotation towards the right was demonstrated at follow-up angiography 3 months after surgery (fig. 1C). The patient has been asymptomatic since surgery 6 months ago.

Case 2

A 61-year-old man had had a neck injury in 1938 that resulted in profound weakness of his left arm. Recent complaints included difficulty in walking, dizziness, and dimming of vision, particularly in the left eye. These symptoms seemed to be worse with rotation of the head. His physical examination showed an ataxic gait, bilateral nystagmus on horizontal gaze, marked hearing loss in the right ear, and muscle wasting of the left shoulder girdle and arm. No cranial abnormality was seen on computed tomography. Cerebral angiography demonstrated total occlusion of the left vertebral artery only during rotation of the head to the right (fig. 2), whereas the normal right vertebral artery of moderate size was unaffected by head rotation. The relation between the rotational occlusion and the patient’s other symptoms was considered uncertain, and no surgery was done. No new symptoms have appeared during 1½ years of follow-up.

Discussion

Faris et al. [1] described unilateral vertebral artery occlusion during head turning in 11 of 43 asymptomatic male volunteers; however, the angiograms were not illustrated, and the levels of the occlusion were not specified. With rotational obstruction of a vertebral artery, symptoms of vertebrobasilar insufficiency are unlikely to occur unless the contralateral vertebral artery is either hypoplastic or occluded [2]. Significant compromise by atherosclerotic disease, osteophytic spurring, or absent or poor collateral flow from the carotid circulations also may contribute to the patient’s susceptibility.

There are many accounts of rotational vertebral artery compromise at the middle and lower cervical levels. Common etiologies include compression by cervical osteophytes [3–6], trauma [7, 8], and constriction by the longus colli and scalenus muscles just before the artery enters the foramen transversarium of the sixth cervical vertebra [9–13].

Compromise of the vertebral artery at the atlantoaxial joint after chiropractic manipulation [14–18] and in patients with rheumatoid arthritis and atlantoaxial subluxation [19] has been described. Okawara and Nibbelink [20] and Grossman and Davis [21] reported single cases in which unilateral occlusion of the vertebral artery occurred with rotation of the head to the opposite side. In both cases it was believed that positional occlusion led to formation of thrombi, which became the source of intracranial emboli to the posterior circulation. Barton and Margolis [22] described two patients in whom head rotation resulted in a narrowing of the contralateral vertebral artery at the C1–C2 level.

Cadaver studies have demonstrated that head rotation causes narrowing of the contralateral vertebral artery at the C1–C2 level [23–27]. The ipsilateral atlantoaxial articulation is fixed during rotation of the head, whereas the atlas moves both downward and forward in relation to the axis on the opposite side [26]. It has been hypothesized that stretching of the vertebral artery associated with this movement at the atlantoaxial joint may produce narrowing or occlusion of the artery [22].

In both of our patients, occlusion of the vertebral artery occurred at the level of the foramen transversarium of the axis. In the first case, the combination of recurrent symptoms, complete occlusion of the left vertebral artery with head rotation, severe congenital hypoplasia of the right vertebral artery, and lack of readily available collateral flow from the carotid to posterior circulation prompted the decision to recommend surgical intervention. A C1–C2 fusion was performed to limit atlantoaxial rotary movement and possible stretching and/or injury of the vertebral artery. It was elected to follow the other patient clinically because of his history, complex symptoms, and the moderate size of his right ver-
tebral artery (which was unaffected by head turning to either side).

Our case 1 is unique in that the patient showed clinical improvement after C1–C2 fusion, further substantiating the concept of vertebrobasilar insufficiency secondary to rotational compromise of the vertebral artery. Although it is not practical to suggest that rotational views be obtained during angiography in every patient with symptoms of vertebrobasilar insufficiency, such views should be obtained in cases with appropriate history, since the affected vertebral artery appeared perfectly normal on standard angiography performed in neutral position in both of our patients.

One of us (T. O. G.) gained extensive experience performing vertebral angiography in children under general anesthesia in the pre–computed tomography era. On quite a few occasions, during test injections to check the catheter position fluoroscopically, complete or nearly complete vertebral artery obstruction of the flow of contrast material at the atlantoaxial level was noted during marked contralateral head rotation, without arterial spasm in the region of the catheter tip placed in the mid–lower cervical region; resumption of normal flow occurred when the head returned to the neutral position. Because of this observation and the report by Faris et al. [1] that rotational obstruction may occur in a significant number of asymptomatic patients, careful evaluation of the contralateral vertebral artery and the relation of rotation to clinical symptoms must be undertaken before deciding in favor of surgical intervention.

ACKNOWLEDGMENT

We thank Sandra Ressler for secretarial assistance.

REFERENCES

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