Rheumatoid arthritis and positional vertebrobasilar insufficiency

Case report

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The authors report a case of positional occlusions of the vertebral arteries in a 45-year-old patient with juvenile rheumatoid arthritis. The occlusions were documented angiographically by placing the head in various positions during digital subtraction angiography using aortic arch injections.

KEY WORDS • rheumatoid arthritis • vertebrobasilar insufficiency • digital subtraction angiography • anomalous artery

RHEUMATOID arthritis producing atlantoaxial instability in the cervical spine may lead to neurological symptoms by producing compressive myelopathy or, more rarely, by causing vertebrobasilar insufficiency. To our knowledge, no cases of vertebrobasilar insufficiency associated with cervical rheumatoid arthritis have been documented angiographically. We present a case of bilateral vertebral artery narrowing due to downward subluxation of C-1 on C-2, with further compromise during head extension, and with complete occlusion of the ipsilateral vertebral artery during head rotation to the right or left.

Case Report

This 45-year-old man with a history of juvenile rheumatoid arthritis presented to the University of Arizona Medical Center for evaluation of intermittent episodes of dysarthria, dysconjugate gaze, and alterations of consciousness. He had been in his usual state of health until 3 months prior to admission when he noted the onset of episodes of being "unaware of the environment," which lasted from several minutes to an hour. Some of these episodes were associated with dysarthria. Friends had noticed that at times during these spells he had dysconjugate gaze lasting for a few seconds. Five weeks before admission, he had fallen and fractured his left eighth rib without loss of consciousness or head injury. In the 3 weeks prior to admission these episodes had been increasing in frequency and were occurring twice daily in the 3 days before admission. He denied any aura, tonic-clonic movements, or true loss of consciousness in association with these episodes, although he did report post-event disorientation. He also denied headache, vomiting, mental changes, or focal weakness or numbness. He had recently placed his television on the floor because he thought that looking down and to the right prevented the occurrence of his spells.

His medical history was significant for juvenile rheumatoid arthritis since 5 years of age, gastric ulcers and subtotal gastrectomy 10 years prior to admission, and endocarditis 7 years prior to admission. He denied any history of epilepsy, diabetes, or stroke. Medications at the time of admission included atenolol (50 mg orally every morning), adrenocorticotropic hormone (20 units subcutaneously every other day), hydrochlorothiazide (50 mg orally every morning), cimetidine (200 mg orally every 12 hours), pentazocine (50 mg orally every 4 hours), and added potassium daily.

Examination. The patient's general physical examination was unremarkable except for marked rheumatoid deformities of the hands and feet, with limitation of motion of the elbows and shoulders, and moderate edema in both feet. Neurological evaluation was normal except for a mild left hemiparesis, slight left-sided hyperreflexia, and an equivocal Babinski response on the left. No bruits were appreciated.
Laboratory studies were all normal except for a serum potassium level of 2.1 mEq/liter. Two blood cultures yielded no growth. Chest x-ray films revealed mild cardiac enlargement and rheumatoid degenerative changes in both shoulders. An electrocardiogram was normal.

The patient was admitted to the hospital and treated with oral potassium. An awake and sleep electroencephalogram with nasopharyngeal leads was normal. An enhanced computerized tomography scan revealed mild cerebral atrophy.

Cervical spine films showed upward telescoping of the odontoid process into the C-1 ring secondary to chronic erosive and hypertrophic arthritic changes of the C1–2 facet joints, generalized narrowing of the other cervical facet joints with mild hypertrophic changes, generalized ligamentous laxity, an old mild C-6 compression fracture, and disc space narrowing at C5–6 and C6–7. Cervical spine tomograms confirmed the plain film findings and also revealed mild upward displacement of the tip of the odontoid process into the foramen magnum (Fig. 1). Flexion and extension views did not show any significant atlantoaxial instability.

Intra-arterial digital subtraction angiography using aortic arch injections was performed on the 6th hospital day. This study revealed incidental findings of an anomalous common origin of the two common carotid arteries and an aberrant right subclavian artery. There were no atherosclerotic changes in the carotid circulation. With the patient’s head turned to the right there was opacification of a moderate-sized left vertebral artery but no evidence of a right vertebral artery. A second injection with the head turned to the left showed filling of a small right vertebral artery, but no opacification of the left (Fig. 2). When the head was slightly flexed during anteroposterior imaging, the left vertebral artery filled briskly but the right filled very slowly. With the head moderately extended, there was no visualization of the right vertebral artery, and the left vertebral and basilar arteries showed markedly delayed opacification in comparison with the carotid arteries.

Although C1–2 fusion was considered as therapy to limit positional compromise of the vertebral arteries, the patient refused any surgical intervention. He was thus placed in a hard cervical collar and began on a course of 75 mg oral aspirin daily with resolution of his symptoms. He was discharged on the 8th hospital day. During the subsequent 10 months, he has not had...
Vertebrobasilar insufficiency in rheumatoid arthritis

similar spells while wearing his collar but he still has episodes of confusion and dysarthria with certain head positions when the collar is off.

Discussion

The most common cause of vertebrobasilar insufficiency is atherosclerosis, accounting for 80% of cases. Other causes include cerebral dysautoregulation and postural hypotension, chiropractic cervical manipulation, sustained physiological positioning, cervical vertebral fracture, yoga positions, athletic injuries, and neck hyperextension while painting. Review of the literature also revealed vertebrobasilar insufficiency as being diagnosed on clinical grounds or at postmortem examination in patients with rheumatoid arthritis.

It is well recognized that with contralateral head rotation there are three possible sites for mechanical compression of the vertebral artery after it enters the foramen transversarium of C-6: at the level of the C5–6 intervertebral foramen, at the atlantoaxial joint, and at the occipitoatlantal joint. If both vertebral arteries have adequate flow with the head in a neutral position, the patient generally remains asymptomatic during the period of mechanical compression of the vertebral artery. However, if the vertebral artery on the side to which the head is rotated is hypoplastic, ischemic symptoms may occur during head turning.

The usual explanation of vertebral artery narrowing or occlusion at C1–2 during head turning is forward subluxation of the contralateral facet of the atlas on the axis, producing stretching of the vertebral artery between the foramen transversarium of C-2 and C-1. This is usually also the site of vertebral artery dissection or occlusion during chiropractic cervical manipulation.

The first published angiographic demonstration of atlantoaxial vertebral artery occlusion during contralateral rotation of the head was that of Ouchi and Ohara. Subsequently, Barton and Margolis and Yang, et al. each added two cases of transient occlusion of a vertebral artery at the atlantoaxial joint during head rotation to the opposite side. Okawara and Nibbelink and Grossmann and Davis each presented a single case of positional occlusion of the vertebral artery at the C1–2 level which they believed led to transient stagnation and thrombus formation and subsequently produced embolic strokes.

The morbidity secondary to cervical spine involvement by rheumatoid arthritis can vary in severity, including a sore neck, "drop attacks," transient vertebrobasilar insufficiency, and compression of the spinal cord or brain stem. Although cervical spine subluxation is a well-recognized complication of rheumatoid arthritis, only recently has there been a trend of thought that death in rheumatoid patients has been wrongly attributed to other causes when in fact it was secondary to damage of the vertebral arteries or spinal cord. Mortality is very rare, but has been documented to occur from insults such as vertebral artery thrombosis and progressive herniation of the odontoid process into the foramen magnum with compression of the medulla oblongata.

The mechanism of vertebrobasilar insufficiency with changes in head position in rheumatoid arthritis patients can be due to a number of causes. It has been postulated that atlantoaxial instability and subluxation could produce increased tortuosity and attenuation of the vertebral artery as it passes around the lateral mass of C-1, especially with head rotation. Eccentric upward herniation of the odontoid process could also pinch off the vertebral artery as it passes through the foramen magnum.

It is interesting that in our case, vertebral artery compression occurred on the side toward which the head was turned and also during extension of the head, and was relieved with flexion. The mechanism of obstruction was not due to gross atlantoaxial instability and subluxation; instead, it was most likely secondary to a combination of "burned out" rheumatoid destructive changes of the C1–2 facet joint and secondary hypertrophic osteophytic spurring and mild capsular laxity. The foramen transversarium of C-2 lies immediately below the lateral margin of the C1–2 facet joint. After the vertebral artery passes through the foramen transversarium of C-2 it turns laterally and passes toward the foramen transversarium of C-1. We postulate that, in our patient, as the vertebral arteries turned laterally they were encroached upon by the overlying osteophytic spurs of the atlantoaxial joints. With head turning, there was further compression of the ipsilateral vertebral artery due to slight posterior and inferior translation of the inferior facet of C-1 on the superior facet of C-2. Inferior and posterior translation also occurred bilaterally with extension of the head, with resultant compression of both vertebral arteries. With flexion of the head, the facet joint of C-1 slid forward enough on C-2 to relieve the compression on the vertebral arteries.

Finally, it should be emphasized that, in this patient as well as in several other cases we have studied, intraarterial digital subtraction angiography with simple aortic arch injections has proven to be an excellent angiographic screening technique for suspected positional occlusion of the vertebral artery. It can be performed safely and relatively inexpensively as an outpatient procedure using small (No. 4 French) catheters, it requires only 20 to 25 ml of a 60% contrast solution for each injection to provide consistently good studies, and it produces very little patient discomfort.

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