jnm/CASE REPORT

DIMINISHED CEREBRAL PERFUSION RESULTING FROM KINKING OF THE INTERNAL CAROTID ARTERY

Richard T. Trackler and Adrian G. Mikulicich

Sharp Memorial Hospital, San Diego, California

Kinking of the internal carotid artery resulted in diminished cerebral perfusion of ^{99m}Tc-pertechnetate given as an intravenous bolus to a patient with recurrent hemiparesis. The subsequent static images were normal.

A 76-year-old white woman was admitted to Sharp Memorial Hospital following the acute onset of left hemiparesis and numbness of the left side of her face. Her admitting blood pressure was 206/94. She had had one previous admission to this institution 6 months before for similar symptomatology. She had been seen in the hospital emergency room 1 month before for syncope and transient left upper extremity weakness.

Brain imaging, using a scintillation camera, was accomplished in vertex position. A bolus of 15 mCi of ^{99m}Tc-pertechnetate was injected intravenously following rapid release of a blood pressure cuff applied to the arm. Following an 8-sec delay, serial 3-sec exposure Polaroid scintiphotos were obtained (Fig. 1). There was evidence for diminished vascular perfusion to the right cerebral hemisphere. Similar findings were demonstrated on the radionuclide venous cerebral angiogram performed during the patient's first hospital admission. Delayed static images, accomplished with the gamma camera 2 hr postinjection of tracer, were negative on both admissions.

The clinical impressions were recurrent, transient ischemic episodes, and hypertension. She recovered completely symptomatically in the hospital. It was elected to manage her medically with anticoagulants and antihypertensive medication as an outpatient. She was admitted subsequently for elective arteriography with the expectation of demonstrating significant extracranial atherosclerotic, occlusive vascular disease.

Aortic arch and selective right common carotid arteriograms were performed. Only mild atherosclerotic narrowing of the distal right common carotid artery was apparent. Sequential film series in Towne position following arch injection revealed delayed opacification of the right intracranial vessels compared with the left intracranial vessels. The right middle cerebral artery was opacified about 2 sec later than the left middle cerebral artery. Selective opacification of the right common carotid artery revealed marked coiling and some kinking of the right internal carotid artery adjacent to the C1-C2 vertebrae (Fig. 2). Both anterior cerebral arteries filled from the left.

Received July 25, 1973; revision accepted Jan. 31, 1974.

For reprints contact: Richard Trackler, Dept. of Radiology, Sharp Memorial Hospital, San Diego, Calif. 92123.

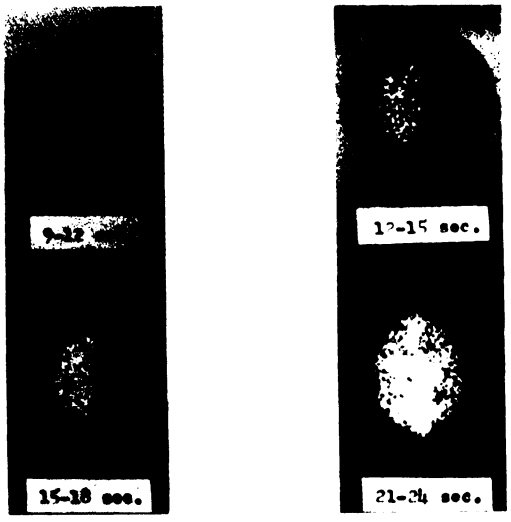

FIG. 1. Serial scintillation camera images with head in vertex position following rapid intravenous injection of ^{99m}Tc demonstrate diminished perfusion of right cerebral hemisphere.

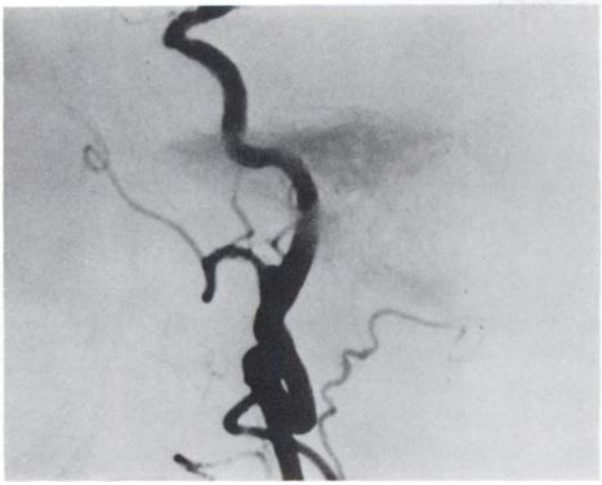

FIG. 2. Right common carotid arteriogram, lateral view, demonstrating marked tortuosity and kinking of internal carotid artery adjacent to C1-C2 vertebrae.

DISCUSSION

The demonstration of reduced cerebral perfusion accompanied by negative static scintillation images generally favors a cerebrovascular accident (*1,2*). Dissimilar perfusion between the two cerebral hemispheres may be associated with obstructive atherosclerotic lesions which are not necessarily symmetrical. Extracranial obstructive carotid arterial disease was suspected in this patient but angiography revealed only minimal narrowing of the distal right common carotid artery. Marked coiling with some kinking of the right internal carotid artery accounted for both reduced perfusion of the right cerebral hemisphere on the intravenous radionuclide angiogram and on the arteriographic studies.

Patients with kinked internal carotid arteries complain of multiple strokes (*3*). Some investigators believe that symptoms occur rarely when severe internal carotid coiling is present unless atherosclerotic occlusive disease is also present in the carotid arteries or vertebrobasilar system (*4*). Evidence for significant extracranial artherosclerotic disease was not demonstrated on brachiocephalic arteriography in this patient.

Histological studies of kinked internal carotid arteries have been reported by Boström and Greitz (*5*). The stenosis associated with intraluminal fold-like protrusions in kinked internal carotid arteries is caused by infolding of the entire wall of the artery made up of all anatomic layers. There is loss of elastic tissue and smooth muscle in the media accompanied by increased ground substance and fibrous tissue. The proposed theory by Boström and Greitz for this stenosis is a mechanical one occurring in arteries elongated by a degenerative process in the media.

Coiled and kinked internal carotid arteries represent another potential etiology for diminished perfusion of the brain as demonstrated both on radionuclide cerebral angiograms and conventional arteriograms.

ACKNOWLEDGMENTS

The authors wish to thank Sherry Nickerson, James Deitemeyer, and Rick Asaro for technical assistance.

REFERENCES

1. ROSENTHALL L: Intravenous and intracarotid radionuclide and cerebral angiography. *Semin Nucl Med* 1: 70–84, 1971

2. BELL E, MCAFEE J: Principles of brain scan interpretation. In *Progress in Nuclear Medicine, Neuro-Nuclear Medicine*, Potchen E, McCreedy V, eds, Baltimore, Md, University Park Press, 1972, pp 249–286

3. DERRICK JR, SMITH T: Carotid kinking as a cause of cerebral insufficiency. *Circulation* 25: 849–853, 1962

4. WEIBEL J, FIELDS W: Tortuosity, coiling and kinking of the internal carotid artery. Relationship of morphological variation to cerebrovascular insufficiency. *Neurology* (Minneap) 15: 462–468, 1965

5. BOSTRÖM K, GREITZ T: Kinking of the internal carotid artery. A roentgenologic and histologic study. *Acta Radiol [diag] [Suppl] (Stockh)* 6: 105–112, 1967