Occlusion of All Four Extracranial Vessels With Minimal Clinical Symptomatology. Case Report

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Abstract: Occlusion of All Four Extracranial Vessels With Minimal Clinical Symptomatology. Case Report

A case of a 55-year-old man with occlusion of both vertebral, left common carotid and right internal carotid arteries is described. Also present was 95 per cent stenosis on the origin of the right external carotid artery with severe atherosclerotic disease on both subclavian and innominate arteries. Clinical symptoms consisted of blindness of the left eye; otherwise the patient was free of neurological signs. It is suggested that not only the presence of extracranial occlusive disease but also the status of the intracranial circulation, the time factor in which the extracranial vessels became occluded, and the adaptability of the individual to establish all possible means of collateral flow play their roles in correlation of extracranial atherosclerotic disease and clinical symptomatology.

Additional Key Words: extracranial cerebral vascular disease, transient ischemic attacks, extracranial collateral circulation, cerebral angiography.

In contemporary neurological practice, aggressive use of angiography in evaluation of patients with cerebral ischemic symptoms often reveals extensive occlusive disease, sometimes with unusual patterns of collateral flow. Many such patients have already suffered severe clinical disability. We wish to present a case which is unique in that very minimal clinical symptoms were present, despite occlusion of both carotid and vertebral arteries, due to extensive extracranial collateral and virtual absence of intracranial disease. Serial angiographical study permitted observation of the chronological evolution of the disorder.

Case Report

This 55-year-old Caucasian man was first admitted to the University Hospital in November, 1961; he was then 45 with a three-year history of increasingly severe claudication coincident with recurring episodes of vertigo and dysarthria. Neurological examination at that time was negative except for the presence of bruits over both subclavian, left carotid, right renal and both femoral arteries. His blood pressure was 200/100 in the left arm and 190/100 in the right arm. Angiographical study revealed stenosis of both subclavian arteries, occlusion of the left vertebral artery and 90% stenosis of the origin of the left internal carotid artery. A left carotid endarterectomy was performed after which he was free of neurological symptoms. Seven weeks later percutaneous left carotid arteriogram revealed satisfactory patency of the surgical region. Translumbar aortogram was then performed and showed 80% stenosis of the right common iliac artery. The renal arteries were free of disease.

In November 1964 he had four episodes of severe vertigo. Cerebral angiography (fig. 1), performed by bilateral axillary approach and direct puncture of the left common carotid artery, then revealed occlusion of the left vertebral artery, 60% stenosis at the origin of the left internal carotid artery, and significant stenosis at the origin of the right vertebral and left subclavian arteries. The bifurcation of the right common
carotid artery was free of arteriosclerotic disease. This procedure was complicated by loss of the radial artery pulsation on the right which was treated with stellate ganglion block.

In November, 1970, he suffered sudden onset of left eye blindness. There was no recovery from this. In June, 1971, he was readmitted to this Center following an episode of mild right hemiparesis and dysarthria. Left optic atrophy was present and there were bruits over the right

carotid artery, both subclavian arteries, the abdominal aorta, and both renal arteries, and an

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**FIGURE 1**

Bilateral axillary arteriography, November, 1964. Right common carotid artery (1), left common carotid artery (2), right vertebral artery (3), left subclavian artery (4), remnant of the left vertebral artery (5).

**FIGURE 2**

Arch study by right axillary approach, June, 1971. Right common carotid artery (1), right vertebral artery (3), left subclavian artery (4), basilar artery (6), right subclavian artery (7), innominate artery (8). Arrows point on the muscle branches of both subclavian arteries and on the cervical muscular collateral network, the only supply of the brain circulation.
almost undetectable pulse in both groins. The left common carotid artery pulse was absent. Arteriography was performed through right axillary artery. Aortography (fig. 2) was followed by selective injections into the left subclavian, right common carotid and right subclavian arteries (figs. 3-5). The study revealed occlusion of the left common carotid artery (fig. 1), both vertebral arteries (figs. 2-4), and right internal carotid artery (fig. 1). The origin of right external carotid artery was stenosed 95% (fig. 5), the proximal portion of the left subclavian artery showed severe arteriosclerotic irregularities and stenosis (fig. 1), and stenosis was seen in the innominate and right subclavian arteries. The entire brain was supplied through muscular branches of both subclavian arteries (figs. 2-6) which through muscular neck arterial collateral network filled the distal

FIGURE 3

Right subclavian injection through right axillary approach, June, 1971. Right vertebral artery (3), basilar artery (6), right subclavian artery (7). Arrows point on enlarged and tortuous muscle branches of the right subclavian artery.

FIGURE 4

Right subclavian injection, later phase. Right posterior cerebral artery (1), right middle cerebral artery (2), left middle cerebral artery (3), anterior cerebral arteries (4), left posterior cerebral artery (5), basilar artery (6).
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FIGURE 5
Right subclavian injection, later phase, lateral view. Right common carotid artery (1), both middle cerebral arteries (2), right vertebral artery (3), anterior cerebral arteries (4), both posterior cerebral arteries (5), basilar artery (6), right external carotid artery (7), partially supplied through the occipital artery. Anterior arrow points on the stenosis of the origin of the right external carotid artery. Posterior arrow points on the collaterals which supply the distal segment of the right vertebral artery.

Discussion
Weibel and Fields describe a complex case of extracranial atherosclerotic occlusive disease. Their patient was a 62-year-old man with a segment of the right vertebral artery. Through the posterior communicating arteries the contrast went from the basilar artery into both middle and anterior cerebral arteries (figs. 4-6). No communication between the right external carotid artery and intracranial circulation was present.

He was re-examined clinically in September, 1971. He was working four to six hours a day (bank executive). He was blind in his left eye; the slurring of the speech and right hemiparesis had disappeared. His primary limiting difficulty has been claudication in both lower extremities. Blood pressure is controlled at 140/90 bilaterally and the anticoagulant treatment with Coumadin continues.

FIGURE 6
Schematic diagram showing occlusion of the left common carotid artery, right internal and both vertebral arteries. Additionally stenosis of the origin of the right external carotid artery, marked segmental narrowing at the origin of the right common and right and left subclavian arteries. The only supply of the brain is through the arterial cervical network from the ascending cervical artery and muscle branches of the thyrocervical trunk bilaterally into the right vertebral artery. Direction of flow is indicated by arrows.
one-year history of lightheadedness which was related to looking upward. Two months prior to admission, he had suddenly developed diplopia, following extension of his head and neck, and was unconscious for a brief period. Upon regaining consciousness, he had left facial weakness, slurred speech and a tendency to stagger to the left. He had right carotid and bilateral subclavian bruits. He was studied by means of direct percutaneous angiography. On the right common carotid artery injection, the internal carotid was seen to be occluded with good filling of the external carotid. The siphon of the internal carotid artery was filled by way of the internal maxillary artery. The most distal portion of the right vertebral was opacified by way of an anastomosis from the external carotid artery. Faint filling of the basilar artery was achieved. The left common carotid artery was also studied by direct puncture. The internal carotid was found occluded. However, there was good collateral circulation through the orbit between the branches of well-opacified external carotid and ophthalmic arteries into the carotid siphon. The distal portion of the left vertebral artery was again supplied through collaterals via the occipital artery. From this study the authors concluded that there was occlusion of both internal carotid arteries and assumed occlusion of the proximal portion of both vertebral arteries. The aorta and its principal branches were not visualized angiographically, however. We could add that collateral filling is not proof of occlusion of the proximal portion of the vessel. Usually collateral flow is well established before the vessel is occluded.

Mishkin\textsuperscript{3} described two cases with occlusion of both internal carotid and left vertebral arteries. The first patient had already had a completed stroke. In this case both external carotid arteries were patent and participated in the supply of intracranial circulation. The second patient demonstrated only moderate right hemiparesis. Again both external carotid arteries were patent.

Meyer, Sheehan and Bauer\textsuperscript{4} reported a case of a 50-year-old woman with bilateral occlusion of the internal carotid arteries and tortuous and irregular lumina of the cervical portion of both vertebral arteries. Their patient was aphasic, blind, stuporous, hemiparetic, hemianopic and hemianalgesic.

Our case is exceptional in both angiographical findings and clinical history. A man with occlusion of all four extracranial arteries is free of cerebral symptoms with the exception of left eye blindness. Such extensive extracranial arteriosclerotic disease has never been reported in a patient with such limited symptomatology. In our judgment the absence of clinical symptoms here is due to the absence of intracranial disease, and the remarkable extracranial collaterals. The correlation of cerebral symptoms and demonstrable extracranial arterial disease is dependent not only on the state of all the vessels through the intracerebral and extracerebral course but also on the time factor in which the extracerebral vessels became occluded and on the adaptability of the individual to establish all possible means of collateral blood flow.

References

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