

Vertebrobasilar Insufficiency in a Patient with Persistent Trigeminal Artery and Stenosis of the Ipsilateral Carotid Bifurcation

—Case Report—

Yutaka HIRASHIMA, Shunro ENDO, Keiji KOSHU and Akira TAKAKU

Department of Neurosurgery, Toyama Medical and Pharmaceutical University, Toyama

Abstract

A 69-year-old female with frequent episodes of vertigo was found to have persistent trigeminal artery and ulcerative stenosis of the ipsilateral carotid bifurcation. The clinical symptoms, pathogenesis, and treatment of this anomaly are discussed.

Key words: persistent trigeminal artery, vertebrobasilar insufficiency, microemboli, carotid endarterectomy

Introduction

A number of authors^{2,5,8,10,13,14} have described vertebrobasilar insufficiency associated with persistent trigeminal artery. We encountered a patient with persistent trigeminal artery and ulcerative stenosis of the ipsilateral carotid bifurcation presenting obstinate symptom due to vertebrobasilar insufficiency. We reported this case and discussed about the pathogenesis and treatment.

Case Report

A 69-year-old female with a 10-year history of hypertension and transient attacks of vertigo was admitted to our hospital on April 24, 1985 because of impaired consciousness and dysarthria. On admission she was lethargic and had left hemiparesis. Ophthalmologic examination showed conjugate deviation to the right. A carotid murmur was detected in the right neck. A general physical examination revealed no other abnormalities; blood pressure was 140/70 mmHg. Computed tomograms (CT) showed low-density areas in the right basal ganglia and centrum semiovale (Fig. 1).

Cerebral angiography revealed that the left com-

mon carotid artery arose from the right innominate artery, which formed a so-called bovine arch (Fig. 2A). Stenosis and irregularity of the wall were noted at the bilateral carotid bifurcation. The degree of internal carotid artery stenosis was 95% on the right side (Fig. 2B) and 40% on the left side (Fig. 3). Stenosis was also noticed in the M₁ and A₁ portions of the right middle and anterior cerebral arteries. Some circulatory impairment of the vertebrobasilar system was also demonstrated. The right vertebral artery was hypoplastic. The origin of the left vertebral artery was not visible and collateral flow

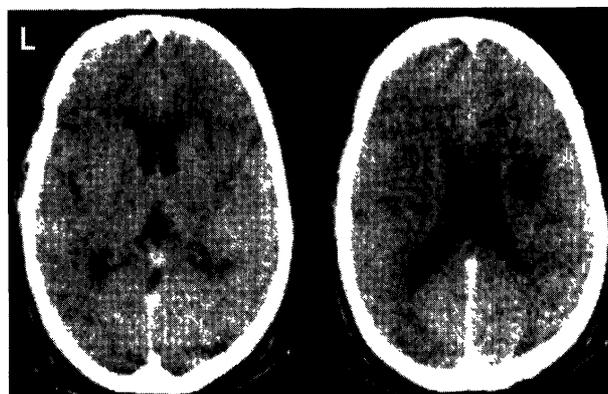


Fig. 1 Precontrast CT scan on admission showing low-density lesions in the right basal ganglia and centrum semiovale.

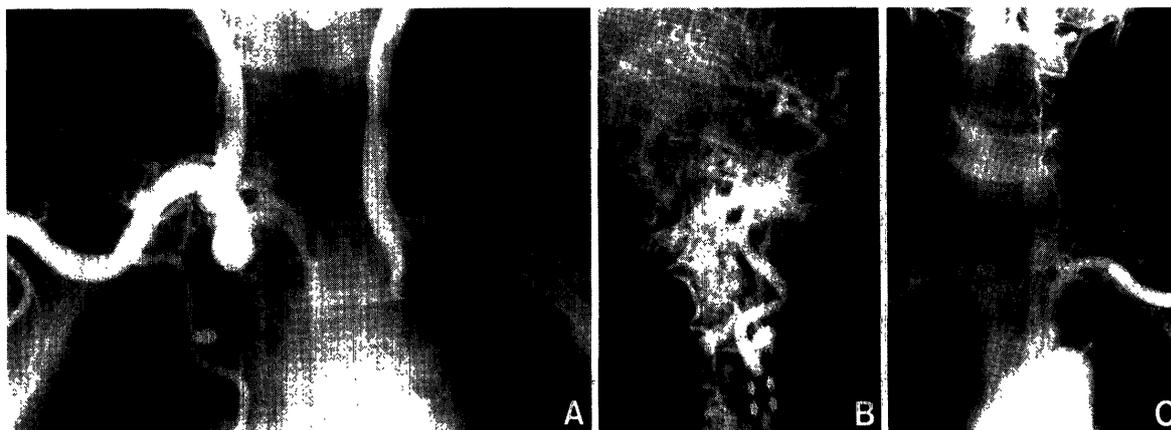


Fig. 2 A: Anteroposterior view of the right retrograde brachial angiogram demonstrating that the left common carotid artery arises from the right innominate artery (bovine arch). B: Lateral view of the right retrograde brachial angiogram demonstrating 95% stenosis of the cervical internal carotid artery (arrows) and the hypoplastic vertebral artery. C: Anteroposterior view of the left retrograde brachial angiogram showing the hypoplastic vertebral artery.

through the ascending cervical artery was evident (Fig. 2C). A primitive left trigeminal artery was present and the basilar artery system appeared to be supplied by this primitive trigeminal artery and the left posterior communicating artery. Stenosis and irregularity of the wall were observed in these arteries as well (Fig. 3).

Conservative treatment resulted in gradual restoration of consciousness and disappearance of the conjugate deviation within 2 weeks of admission. However, motor function on the left side did not improve. Because of the risk of internal carotid artery occlusion and for the purpose of increasing cerebral

blood flow, endarterectomy of the right internal carotid artery and right superior cervical gangliectomy were performed on May 2 and 23, respectively.

The patient's neurological symptoms, especially the motor weakness in her left lower extremity, improved after these surgical procedures, and she was able to walk with a cane about 1 month post-operatively. Unfortunately, however, the attacks of severe vertigo persisted and disrupted her rehabilitation program. Therefore, left carotid endarterectomy was recommended but she refused this surgery and sought treatment at another hospital. During the past 5 years her symptoms of vertebrobasilar insufficiency have progressed, and she remains bedridden.

Discussion

According to the literature, the incidence of persistent trigeminal artery ranges from 0.1 to 0.6%.^{4,6,12} In 1884 Quain described a persistent trigeminal artery in an autopsied patient,¹⁾ and Sutton¹¹⁾ detected the anomaly angiographically in 1950. Saltzman⁹⁾ divided the cerebral blood circulation in cases of persistent trigeminal artery into three types: In type I the bilateral posterior cerebral arteries are supplied by the basilar artery; in type II one of the posterior cerebral arteries is fed by the ipsilateral internal carotid artery; and in type III both posterior cerebral arteries are supplied by the internal carotid artery. Agnoli¹⁾ added a fourth type, in which one posterior cerebral artery is supplied by the contralateral internal carotid artery, and reported that the frequencies are 61% for type I, 26% for type II, 2%

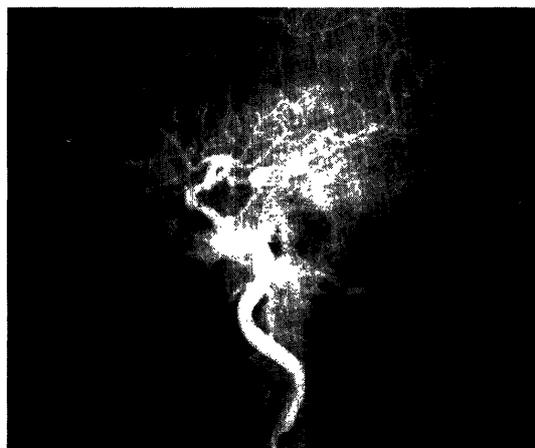


Fig. 3 Lateral view of the left carotid angiogram demonstrating 40% stenosis of the cervical internal carotid artery and severe atherosclerotic changes in the persistent trigeminal artery and posterior communicating artery.

for type III, and 11% for type IV. He also noted that 82% of his patients exhibited bilateral or unilateral hypoplasia or aplasia of the posterior communicating arteries. The bilateral vertebral arteries are also likely to be hypoplastic or aplastic in the presence of a persistent trigeminal artery.¹⁰⁾

The angiograms of our patient showed aplasia of the posterior communicating artery contralateral to the persistent trigeminal artery and hypoplasia of the bilateral vertebral arteries. The anterior communicating artery also was aplastic, and the entire basilar system was supplied by the persistent trigeminal artery (type II according to Saltzman's classification) and the ipsilateral posterior communicating artery. Furthermore, the left common carotid artery in this case originated from the right innominate artery (Fig. 4). The reported incidence of this anomaly is 6 to 8%.^{3,10)}

The symptomatology of persistent trigeminal artery may be related to its pathogenesis as follows: 1) subarachnoid hemorrhage due to cerebral aneurysm and arteriovenous malformation; 2) complications of other vascular anomalies; 3) cerebral ischemia due to impaired cerebral circulation; and 4) cranial nerve palsies (III-VI). The attacks of vertigo suffered by our patient are considered to belong to the third category. Such symptoms of transient vertebrobasilar ischemia associated with persistent arteries have been reported by a number of other investigators.^{2,5,8,10,13,14)} Gilmartin⁷⁾ described a patient

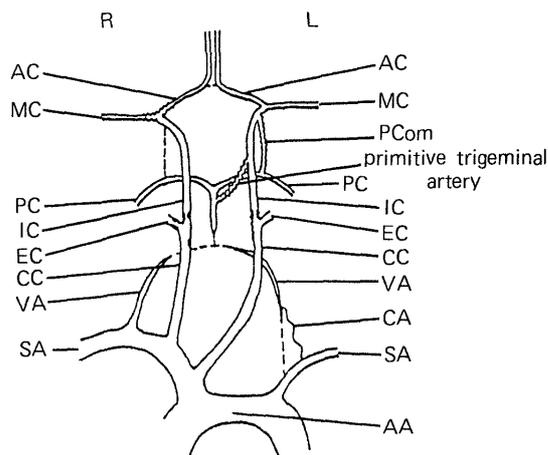


Fig. 4 Schematic representation of the arteriogram. AC: anterior cerebral artery, MC: middle cerebral artery, PC: posterior cerebral artery, IC: internal carotid artery, EC: external carotid artery, CC: common carotid artery, VA: vertebral artery, PCom: posterior communicating artery, CA: cervical ascending artery, SA: subclavian artery, AA: aortic arch.

with persistent hypoglossal artery and stenosis of the cervical internal carotid artery who died of infarction in the vertebrobasilar area. It has been suggested that the ischemia of the vertebrobasilar system in cases of persistent arteries is attributable to impairment of blood flow and/or microembolus formation associated with atherosclerotic change in the proximal arteries.^{10,13)} Ulcerative change in the ipsilateral cervical internal carotid artery is regarded as particularly important.¹³⁾ Waller *et al.*¹³⁾ and Stern *et al.*¹⁰⁾ obtained favorable results with endarterectomy for atherosclerotic lesions in the cervical portion of the internal carotid artery ipsilateral to the persistent trigeminal artery.

In our case a proposed endarterectomy for atherosclerosis of the left cervical internal carotid artery was refused, and the patient's symptoms continued without improvement. Her clinical course strongly suggests that carotid endarterectomy should be performed to relieve vertebrobasilar insufficiency in patients with persistent trigeminal artery and carotid lesions.

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Address reprint requests to: Y. Hirashima, M.D., Department of Neurosurgery, Toyama Medical and Pharmaceutical University, 2630 Sugitani, Toyama 930-01, Japan.