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Intolerable Pulse-synchronous Tinnitus Caused by Occlusion of the Contralateral Common Carotid Artery

A Successful Treatment by Aorto-carotid Bypass Surgery

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Summary

A case with severe pulsatile tinnitus of the left side caused by occlusion of the right common carotid artery was reported. Tinnitus in this case was supposed to be due to the rich blood flow of the external carotid systems developed as collateral routes resulting from the occlusion of the right common carotid artery. It subsided with the establishment of a bypass using a vein graft between the ascending aorta and the residual patent portion of the right common carotid artery.

Keywords: Tinnitus; occlusion of the carotid artery; bypass surgery; vein graft.

Introduction

Vascular bruit is generally ausculated ipsilaterally on the scalp or the neck in cases of intracranial arteriovenous malformation, carotid-cavernous fistula, and carotid stenosis, but, tinnitus sometimes occurs contralateral to these vascular lesions, usually as an intracerebral steal phenomenon. In this paper a case with pulsatile tinnitus of the left side caused by occlusion of the right common carotid artery is presented. Tinnitus in this case was supposed not to be due to an intracerebral steal phenomenon, but to an increased blood flow through the external carotid arteries developed as collateral routes, and subsided successfully by a bypass using a vein graft between the ascending aorta and the residual patent portion of the right common carotid artery.

Case Report

A 47 year old male presented with left sided intolerable pulsatile tinnitus of about 9 months duration. Six weeks before admission the patient had a transient sensory disturbance on the left side of the body and a scintillating scotoma of the right eye. On the next day he lost consciousness for short period during a conversation.

On admission he complained of difficulty in speaking and the left sided pulsatile tinnitus, but no neurological deficit was elicited objectively. Blood pressure at the right arm was 170/80 mmHg and 210/100 mmHg on the left. Pulsation of the left common carotid artery was palpable, but that of the right carotid artery was not.

In order to examine the cause of hypertension, angiograms of the whole body were obtained, verifying an aneurysmal dilatation of the aorta near the renal artery, an occluded right superficial femoral artery, an irregular wall of the left superficial femoral artery, and slight stenosis of the origin of both vertebral arteries (Fig. 1). The right common carotid artery was occluded and the right subclavian artery showed arteriosclerotic changes (Fig. 1). The left carotid angiogram revealed a well developed external carotid artery with anastomoses to the right external system (Figs. 2, 3), through which the right internal carotid artery was filled (Fig. 3). Digital subtraction angiography also showed occlusion of the right common carotid artery as mentioned above and the clear filling of the internal carotid artery from the external carotid arteries (Fig. 4). Single photon emmision CT (SPECT) using N-isoprophyl-p-[^{123}I] iodoamphetamine (IMP) showed low perfusion areas in the right parietal region (Fig. 5, left), though X-ray CT showed no paticular changes. From these findings it was clear that the patient suffered from multiple stenotic arterial disease, and his left-sided pulsatile tinnitus was deduced to have occurred due to an increased blood flow in the external carotid arteries developed as collateral circulatory routes resulting from the occlusion of the right common carotid artery. At first an antiplatelet agent was administered, which remained ineffective and tinnitus continued. Then, a bypass operation using a vein graft between the ascending aorta and the distal portion of the right common carotid artery was thought to be the procedure of choice.

Operation. A long linear skin incision from the right side of the neck to the xyphoid process was made, and the ascending aorta and the common carotid artery were exposed. The proxymal portion of the latter was like a string and blood flow was recognized only at the site of the bifurcation, which was about 7 mm long. A small opening was made in the ascending aorta with a punch after partial

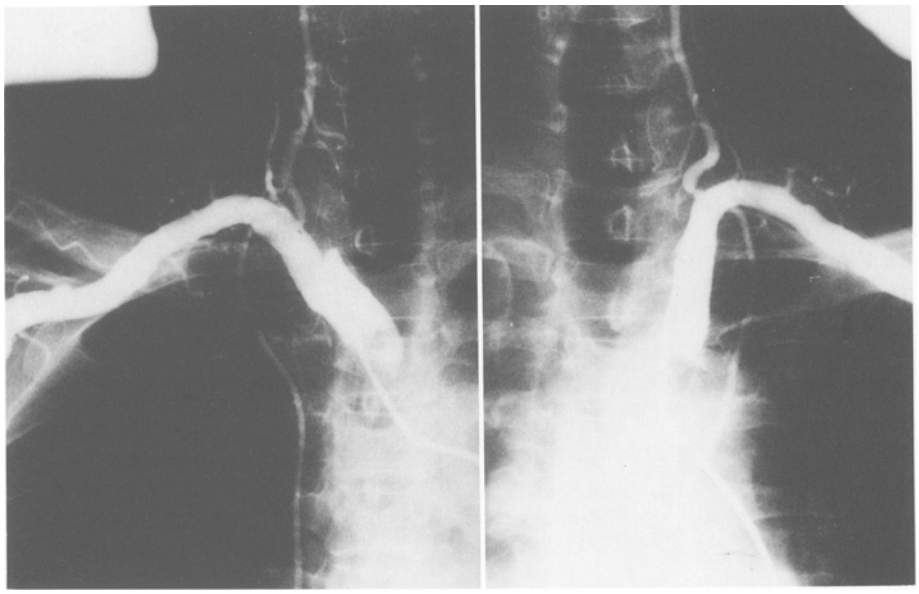

Fig. 1. Stenotic origins of both vertebral arteries and sclerotic changes of the right subclavian artery

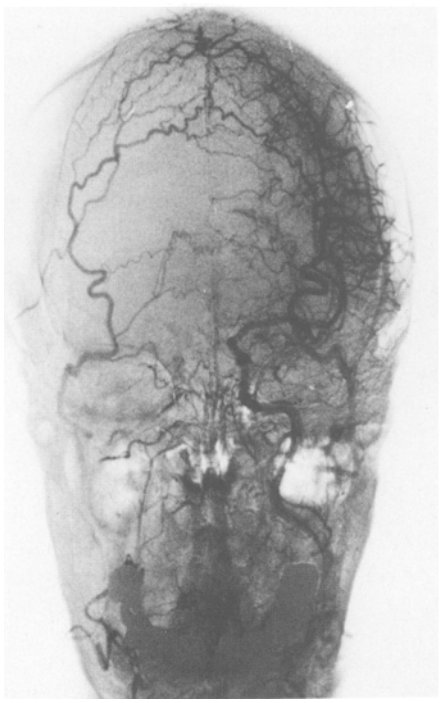

Fig. 2. Well developed external carotid systems

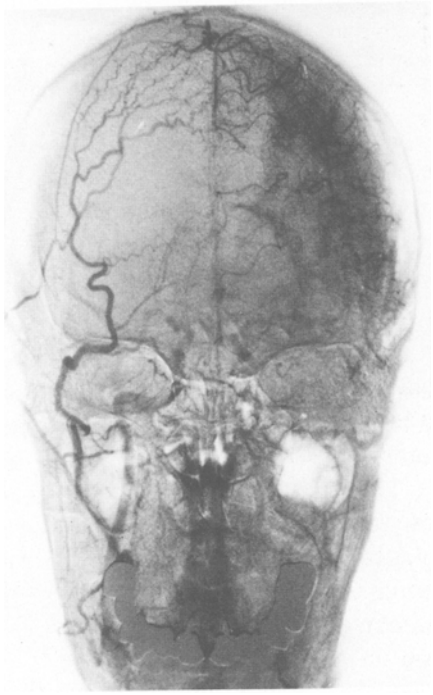

Fig. 3. The internal carotid artery is filled through the external carotid systems

clamping and a femoro-popliteal vein graft of about 20 cm length taken from the right leg was anastomosed in an end-to-side fashion, and then a similar anastomosis was performed between the other end of the graft and the patent portion of the right common carotid artery. Pulsation of the vein graft was satisfactory after the completion of both anastomoses. Revascularization was thus successfully achieved.

Postoperative course. Soon after awaking from the anaesthetic the tinnitus subsided. One week after the operation the patient was transferred to the department of cardiology for treatment of his hypertension. Digital subtraction angiography performed one month after the operation showed the patent vein graft (Fig. 6), and the low perfusion areas recognized in the preoperative IMP SPECT disappeared postoperatively (Fig. 5, right).

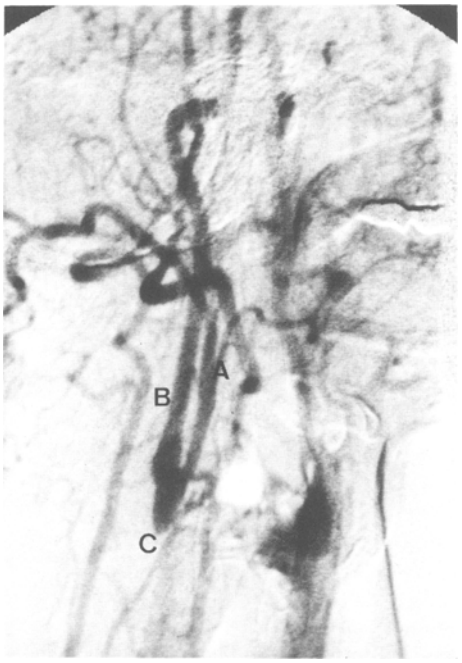

Fig. 4. Digital subtraction angiography. *A* external carotid systems, *B* right internal carotid artery, *C* occluded right common carotid artery

At follow-up ten months later the patient showed no neurological deficit. Tinnitus had disappeared and the vein graft in the right neck pulsated well.

Discussion

Various ischaemic symptoms are caused by extra- and intracranial stenotic and occlusive vascular lesions, which are somtimes accompanied by pulsatile tinnitus[5, 9]. Holgate *et al.*[5] emphasized the turbulent blood flow in or around the temporal bone as the cause of the pulsatile tinnitus reviewing the anatomy of temporal bone blood supply and classified the causative mechanisms of this type of tinnitus as follows; 1) conditions with increased flow volumes, and 2) conditions with laminar narrowing or irregularity.

The tinnitus of our case is thought to have been caused by turbulences in the left external carotid systems due to the increased blood flow developed as collateral circulatory routes resulting from the occlusion of the contralateral common carotid artery. Holgate *et al.*[5], Hirata[4] *et al.*, and Miyagi[7] *et al.* reported cases of pulsatile tinnitus caused by contralateral internal carotid artery occlusion. In their cases blood to the hemisphere of the occluded carotid artery were supplied through intracranial collateral routes from the intact internal carotid system. However, to the best of our knowledge, no case of pulsatile tinnitus with increased blood flow of the external carotid systems developed as collateral routes has so far been reported.

A variety of surgical procedures[3] in cases of occlusion of the common carotid artery have been reported and among these bypass surgery between the subclavian and carotid arteries using a vein graft has usually been performed[1, 3]. But owing to the sclerotic changes in the right subclavian artery the bypass operation between the ascending aorta and the common carotid artery was considered to be preferable.

There are debating reports about vein graft anastomosis. The patency of a vein graft is related to the severity of distal atherosclerotic disease, the number of vessels available for runoff, the diameter of the recipient artery, the length of the graft, graft flow, use of anticoagulation or antiplatelet agents, and the technique of vein harvesting[2, 10, 11]. Most studies report approximately 10% of cases occlude soon after operation and at one year, occlusion rates range from 10 to 35%, with a yearly attrition of 2 to 3% per year thereafter[11]. Though patency of our vein graft has been maintained for 10 months after operation and an antiplatelet agent has been administered, a precise follow-up study will

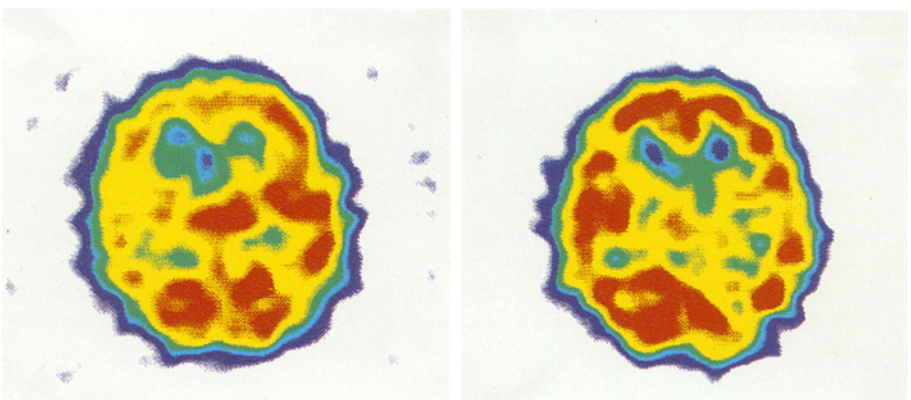

Fig. 5. Pre- and postoperative SPECT findings showing improvement of cerebral blood flow in the right hemisphere. (Left: preoperative SPECT, right: postoperative SPECT)

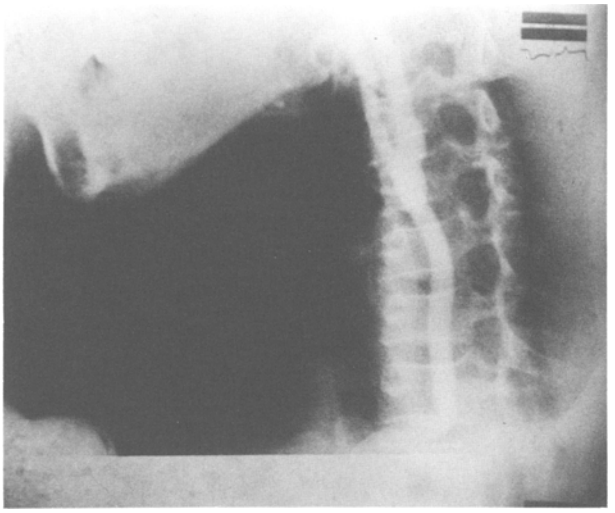

Fig. 6. Patent vein graft

be necessary, because the vein graft used in the operation was relatively long. Artificial vessel grafts might also be used, but the patency of grafts of such a small diameter has been said to be uncertain[8]. For this reason a vein graft was used in this case. The low perfusion shown in the preoperative SPECT was considered to be the cause of the haemodynamic TIA, which disappeared with the increased cerebral blood flow after the operation, and the postoperative SPECT also normalized.

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