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A CASE WITH BILATERAL INTERNAL CAROTID ARTERY OCCLUSION

YUTAKA MAKI, M. D., YOSHITAKA NAKADA, M. D., OSAMU WATANAE, M. D.
TADAO NOSE, M. D., and ETSUKO KINOSHITA, M. D.

Department of Neuropsychiatry, Unit of Neurological Surgery,
School of Medicine, Chiba University, Chiba, Japan

(Director: Prof. Dr. Yutaka Matsumoto)

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Introduction

The disease which are caused by the narrowing or obstruction of main arteries supplying brain with unknown etiology has recently been noticed in neurological clinics.

One is the disease with spontaneous internal carotid occlusion which is noticed by Shimizu & Takeuchi[1~2], and the other is the disease with abnormal intracranial vascular networks which has been notice in these several years in Japan[3~7].

Recently we have experienced a case which showed bilateral occlusion of internal carotid arteries by unknown etiology and did belong to neither of the above mentioned two diseases which are similar to the case.

Case Report

The patient was a 52-year-old male, sailor. Nothing is important in his family history. In his previous history also nothing was noticed but slight head concussion at 16-year-old. He did not smoke but took Sake a half or a third quart a day since about 25-year-old.

In July 1963 (at 48-year-old), he lost his consciousness about several seconds when he tried to deficate. Then, until November 1968 he had experienced such transient attack about three times. The attacks sometimes were accompanied with transient right hemiparesis including right facial palsy, and disturbance of speech, but those recovered spontaneously and completely.

In November 1968, he was admitted to the Chiba University Hospital in order to get through investigation for the disease.

Examination. His general status was good. Blood pressure was 180–90 mmHg and showed no difference between right and left.

There were not any particular signs in his chest and abdomen. The pulsation of his carotid, radial, femoral and other arteries were ease to be felt. Only slight mental retardation and slowness of his body movement were noticed. Laboratory data shown in Table 1 shows somewhat delayed the circulation time. The slight depression at ST waves in ECG were found and the patient was suspected as a coronal disease.

In the EEG, brain rhythm were 8–9 c/s but showed irregular slowing.

Table 1. Laboratory data

RBC		424×10^4	Urine	
WBC		5800	Protein	(±)
Hb		85%	Suger	(−)
Ht		39%	Urobilinogen	(±)
Thrombo		194880		
			PSP	
Serum			15′	17.5%
Total protein		7.0 g/d*l*	30′	27.5%
Al		63.2	60′	15%
Gl	α_1	3.6	120′	15%
	α_2	10.8	Wasserman	(—)
	β	11.4	Venous pressure	
	γ	10.8	Circulation time	
Cholesterol		203 mg/d*l*	Ether	13″
Na		138 mEq/*l*	Decolin	22″
K		4.0 mEq/*l*		
GOT		22 Karmen U		
GPT		18 〃		
MG		4.0×		

X-ray examination: As in Fig. 1 and 1′, common carotid angiograms showed the occlusion of internal carotid arteries at the region of 1 cm distal from common carotid bifurcation, but external carotid artery was filled completely. One second after injection (Fig. 2), the anterior cerebral artery was filled to A_3-portion through the collateral circulation from the external carotid artery.

Two seconds after the infection (Fig. 3) the perforating arteries at the base of brain were seen somewhat dilated and the middle cerebral artery was filled as retrograde fashion through the occipital artery of external carotid artery. The both side were almost the same.

In vertebral angiograms (Fig. 4), the diameters and patterns of the vertebro-basilar system was normal, the posterior cerebral artery was filled retrogradely through leptomeningeal arteries and Ramus spleni (Fig. 5).

Biopsy: From the above described findings, this patient was diagnosed as a bilateral occlusion of internal carotid arteries. In order to investigate the etiology, the biopsy of bilateral internal carotids were tried. The arteries were narrowed remarkably at the 1 cm distal portion from the bifurication. The wall of arteries were quite hard and this elasticity was lost completely and the pulsation could not be felt. The blood back flows were not occurred by needle punctures. The biopsies were carried out bilateraly about 1 cm along the vessels. The patient has been administrated the anticoagulant and vasodelatator drugs and maintenanced good condition. Histologically as shown in Fig. 6, the cavity of the arteries were completely occluded. The distruction of muscle fibres and the accompanied increment of connective tissue at the media were main findings. The disarrangement of the elastic fiber were also seen (Fig. 7, 8). The fragmentation of the external elastic layer and the reactive proliferation of adventitia

were also seen. There were not any differences between right and left.

Discussion

The occlusion of unilateral internal carotid artery is not rare disease but the cases of bilateral occlusion as this patient are not so many reported: That is, Batley (1958) 2[8], Groch et al. (1960) 4[9], Field et al. (1961) 16[10] and total numbers are less than 70. In Japan, Kito (1958)[11], Totani (1965)[12], and Kudo (1969)[5] have reported some, but less than 20 cases totaly. This disease could be said relatively rare.

Elvidge et al. (1951)[13] classified this disease from it's processes and symptomes as follows:

1) apoplectic type
2) slowly progressive type
3) transient and recurrent type

And Webster et al.[14] have added as asymptomatic type moreover. This case can be classified as transient and recurrent type, this means a Denny-Brown syndrome. The occurrence of each type are apoplectic type 2, slowly progressive type 1, and transient and recurrent type 2 and this asymptomatic type is quite rare and in Japan only Totani (1965)[12] have reported one.

The sex incidence is said to be larger in male and the age incidence is higher in the aged people in Europe and America, whereas there are two peaks at the young adults and the aged in Japan. It could be assumed that this difference is caused by the frequent occurrence of spontaneous occlusion of internal carotid arteries in Japan.

Table 2. Comparison among three spontaneous occlusive disease groups

		A disease with abnormal intracranial vascular network	Spontaneous occlusion of the internal carotid	Present cases
Age		Children Adult	Young adult Adult	52-year-old 〃
Sex		M<F	M>F	male
Site of arterial leigion	Internal carotid (cervical)	Narrowing	Occlusion	Occlusion
	Internal carotid (intracranial)	Narrowing ~Occlusion	?	Narrowing
	Middle cerebral			
	Anterior cerebral			
Abnormal vascular network		++	?	+
Mainly histopathological change		Intima	Intima	Media

The etiological reasons for the occlusion of the internal carotid artery are assumed to be arteriosclerosis, syphilis, aneurysm, thrombosis and embolus due to the heart disease, periarteric involvement due to tumor, trauma and inflammation, periarteritis

nodosa, polycythemia and cirvical rib etc. In the case of ours, there are not any above mentioned etiology and still unknown.

In order to compare this case with other occlusive disease with unknown etiology, Fig. 2 is prepared. This case is quite similar to the disease with spontaneous internal carotid occlusion from it's age, sex and the region of occlusion. However, this case is also significantly different from the disease with spontaneous internal carotid occlusion concerning the following two points. The first is angiographical findings. There have not been reported in the disease with internal carotid occlusion that the narrowing of the arteries and found in the anterior and middle cerebral arteries and also the extensive collateral circulation by diffuse abnormal arterial networks are observed as in this case. But these findings are quite similar to a disease with abnormal intracranial vascular networks which will be discussed later and these two diseases are difficult to differenciate from their angiograms.

The second, histological finding are different. In this case, the main lesion is at the media, its proliferation, destruction of muscle fiber and distinct scar formation. On the hand, in the disease with spontaneous internal carotid occlusion has its main lesion at the intima, that is; irregular thickening or fibrile thickening of intima accompanied with mucous edematous changes. In the disease with abnormal intracranial vascular networks, histologically the main lesions are found in the intima and the fibroblasts or collagen fibers at the intima increased dominantely, according to the autopsy findings of Ando et al. (1967)[3], Suzuki et al. (1967)[7] and Maki et al. (1967)[6].

This case looks like a quite peculiar one from its biopsy findings eventhough it is difficult to assume the general changes of the patient's body since it is not an autopsy case.

The further investigations are planned to go on.

Summary

The disease which are caused by the narrowing or obstruction of main arteries supplying brain with unknown etiology has recently been noticed in neurological clinics.

One is the disease with sponteneous internal carotid occlusion which is noticed by Shimizu & Takeuchi, and the other is the disease with abnormal intracranial vascular networks which has been noticed in these several years in Japan. Both of the disease are noticed and investigated especially by Japanese neurosurgeons.

A case of 51-year-old male patient which showed clinically Denny-Brown Syndrom since recent 6 years was reported. The angiograms showed bilateral complete occlusion at the proximal cervical portion of internal carotid arteries and also showed extensive collateral circulation similary seen in a disease with abnormal intracranial vascular networks. Histological studies by biopsy is carried out and it appeared that main lesion was not at intima but at media accompanying increment of connective tissue and fragmentation of elastic fibres. Moreover, the partial fragmentation of internal elastica and the thickning of intima was also observed.

From it's clinical symptoms and it's site of occlusion of arteries, this case resembles

with a spontaneous internal carotid occlusion, but from angiographical studies, this rather resembles with a disease with abnormal intracranial vascular networks and histologically, the findings are completely different from them in this case. The cases which are on similar line have not been reported yet.

Keywords: spontaneous arterial occlusive disease, bilateral internal carotid occlusion, angiogram of carotid occlusion, collateral circulation of carotid occlusion

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Fig. 1. Lateral view of right common carotid angiogram.
Arrow shows non-filling of internal carotid artery even at the level C_{2-3}.

Fig. 1′. Lateral view of left side.
Arrow shows the stump of internal carotid artery at the level of C_{3-4}.

Fig. 2. Left lateral view at the 1 sec. after injection.
The anterior cerebral artery reveals by collateral circulation via external carotid artery.

Fig. 2′. Left AP view at the 1 sec. after injection.
Upper arrows shows distal portion of the anterior cerebral artery through anterior temporal superficial artery and lower arrow shows collateral circulation between proximal anterior cerebral artery and external carotid artery.

Fig. 3. Left lateral view at 2 sec. after injection.
Vascular network at the anterior base of brain are seen. Ophthalmic artery (0) is now clearly seen, posterior arrows show leptomeningeal collateral circulation with occipital artery of external carotid through skull and dural meninges.

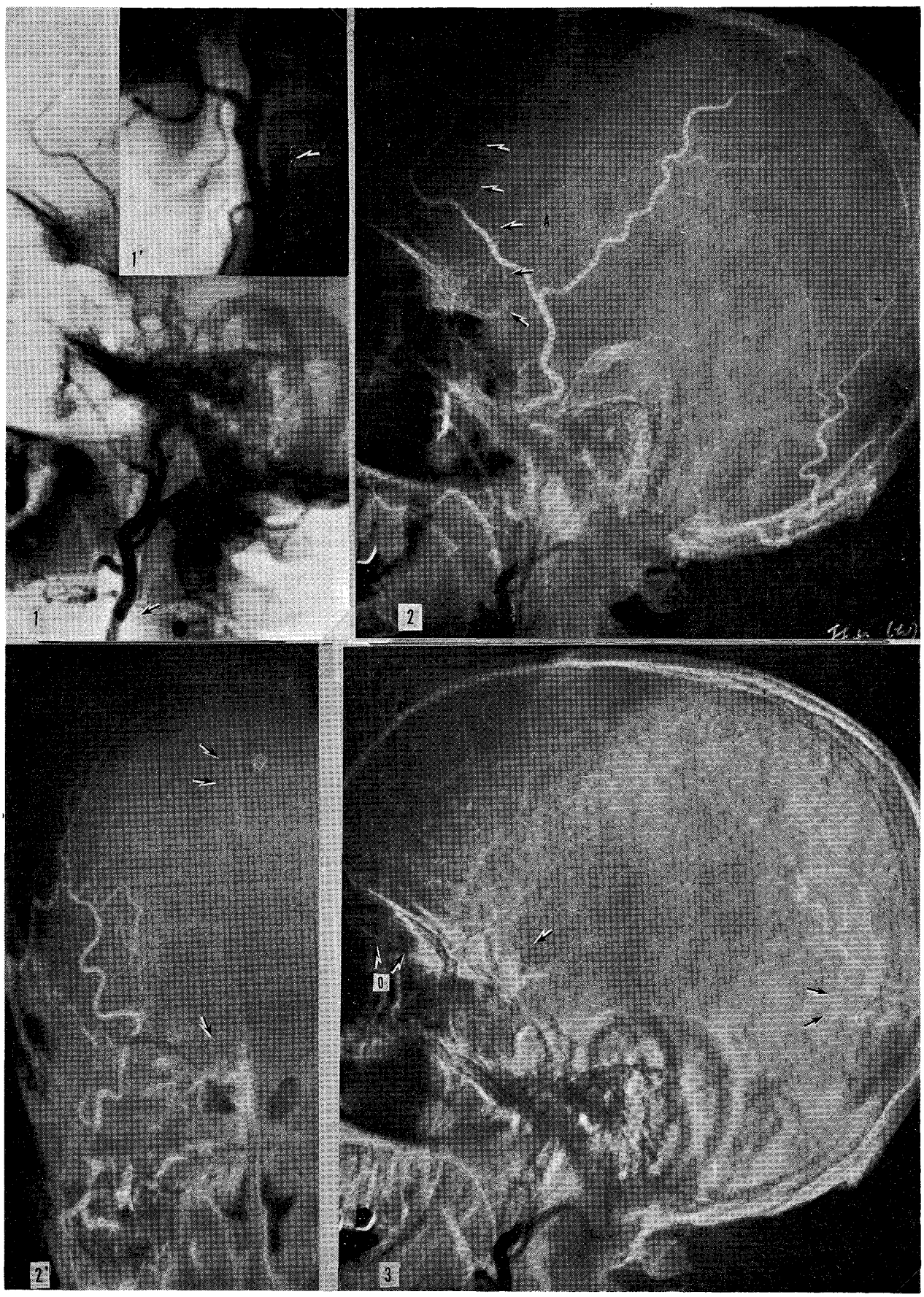
Fig. 4. Lateral view of vertebral angiogram at 1.5 sec. after injection.
Vertebral and basilar arteries show no abnormality in diameter also their pattern. The retrograde filling of anterior and middle cerebral arteries reveal by leptomeningeal arterial anastomosis via ramus splenii.

Fig. 5. Same view at 2 sec. after injection.
The retrograde filling of anterior and middle cerebral arteries are now clearly seen.

Fig. 6. Cross section of internal carotid (Van Gieson stain).
The lumen of the vessel is almost obstructed.

Fig. 7. Van Gieson elastica.

Fig. 8. Van Gieson elastica.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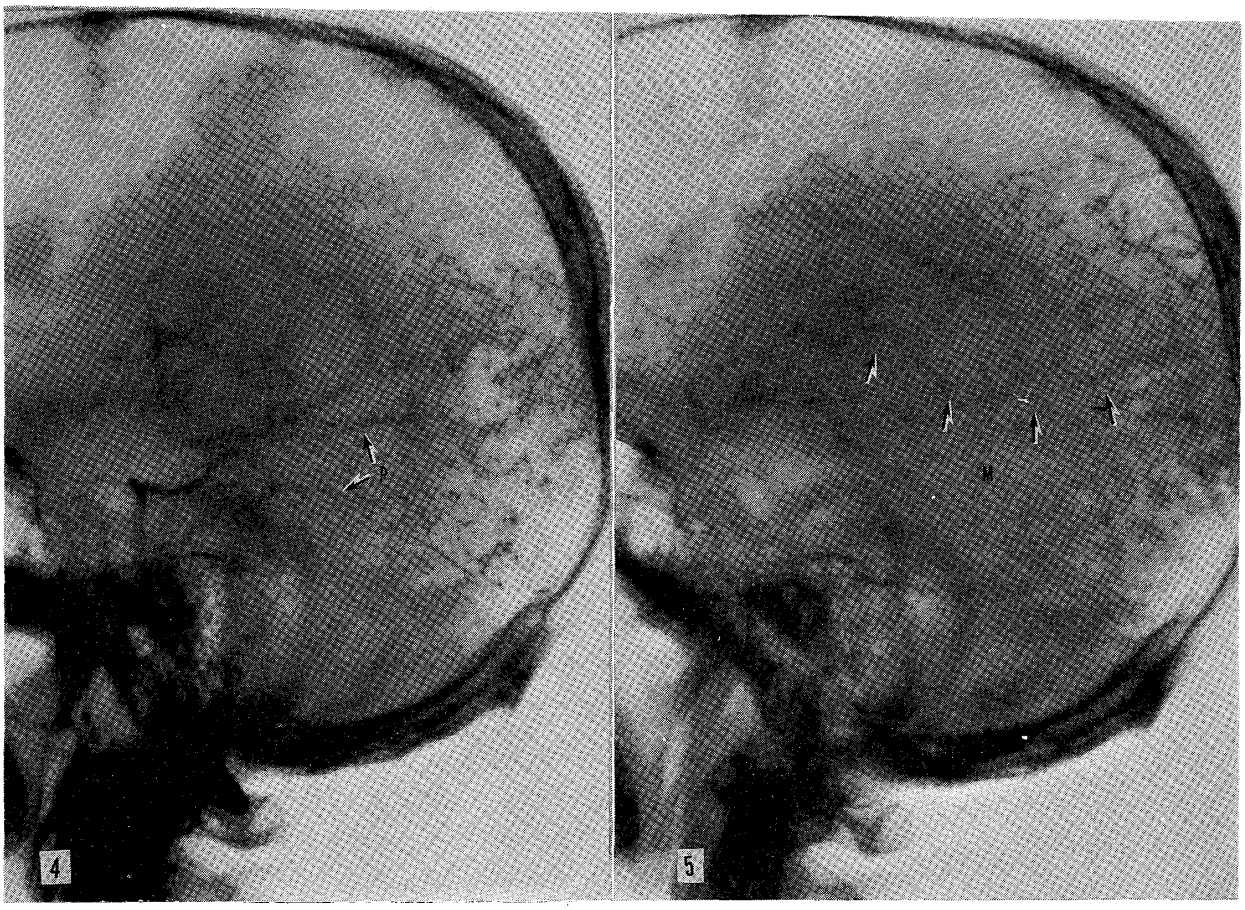
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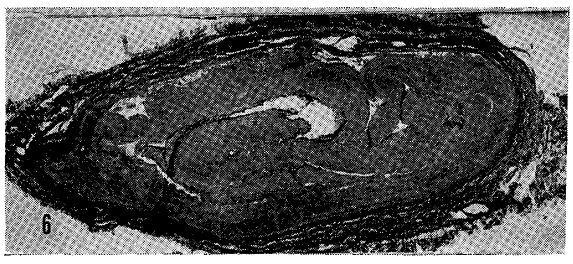
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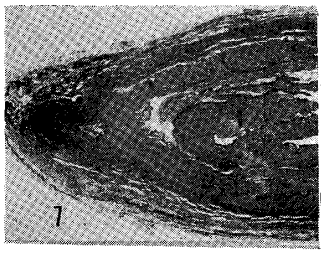
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