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Clinical Variation in Bilateral Internal Carotid Artery Occlusion through the Unilateral Occlusive Pathology without Surgical Intervention —Report of Three Cases—

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We herein report three cases of bilateral internal carotid artery occlusion (ICAO). All of them developed from unilateral pathology and could be followed until death without surgical intervention. We could observe only three cases of bilateral ICAO developed from unilateral ICAO in the course of over thirty years. Bilateral ICAO was diagnosed by cerebral angiography in the first case and by magnetic resonance angiography in the other two cases. Although each of them would be caused by an atherothrombotic mechanism, they showed various clinical features during the development from unilateral to bilateral ICAO.

Key words: bilateral internal carotid artery occlusion, atherothrombosis, collateral circulation

INTRODUCTION

Different types of angiographic appearance have been reported in internal carotid artery occlusions (ICAO)[1]. In recent years, ICAO has been able to be easily diagnosed by the combination of noninvasive examinations of magnetic resonance (MR) imaging, MR angiography and echography of the carotid artery in place of cerebral angiography.

On the other hand, circulation via the carotid arteries is essential to the maintenance of brain activity and its acute insufficiency, especially in bilateral ICAO, is very critical indeed[2]. But chronic or stepwise bilateral ICAO is not always critical pathology. Therefore, we retrospectively investigated the clinical features in three patients with bilateral ICAOs for over thirty years. Each of them had developed from unilateral pathology and could be followed until death without surgical intervention.

CASE REPORT

Case 1

A man, who died at 68 years of age, had experienced right ICAO at 50 (Fig. 1A), then suffered from bilateral ICAO at 57 (Fig. 1B). The low-density areas of brain computed tomography (CT) appeared in the right hemisphere at 50, but spread over the left hemisphere at 57 and 68 (Fig. 2). He had histories of diabetes mellitus, hyperlipidemia, and smoking without hypertension. He had received antiplatelet ther-

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apy since 50 years of age. When he suffered from bilateral ICAO, his height and weight were 162 cm and 51 kg, respectively. Although the physical examinations were normal, neurological examinations revealed the presence of global aphasia and severe gait disturbance because of right Wernicke-Mann posture and left hemiplegia. Babinski signs were bilaterally positive.

The regional cerebral blood flow (rCBF) using single photon emission computed tomography (SPECT) of N-isopropyl-p-[^{123}I] iodoamphetamine (IMP) performed a month after onset of bilateral ICAO at 57 years of age showed focally decreased in the bilateral cortical anterior cerebral artery (ACA)-middle cerebral artery (MCA) and the MCA-posterior cerebral artery (PCA) watershed territories for both early and delayed image (Fig. 3).

He had continued to receive conservative therapy including antiplatelet therapy after onset of bilateral ICAO. He had a long-lived prognosis for 11.5 years as a result, although he had to be supported in all points of activity of daily living (ADL) at the time he had suffered from bilateral ICAO. He died of chronic heart failure.

Case 2

A man, who died at 84 years of age, had experienced right ICAO at 72 (Fig. 4A), then suffered from bilateral ICAO at 83 (Fig. 5A). He had histories of hypertension and smoking. He had taken antihypertensive and antiplatelet agents until three months before the onset of bilateral ICAO. At the onset of bilateral ICAO, his height and weight were 175 cm and 43 kg, respectively. The physical examinations were normal, and neurological examinations revealed the presence of global aphasia and severe gait disturbance due to the quadriparesis. Babinski signs were bilaterally positive. The NIH Stroke Scale (NIHSS) score was 20.

The rCBF using SPECT of IMP performed at 72 years of age showed focally decreased in the right cortical MCA territory for both early and delayed image (Fig. 4B), but the rCBF using SPECT of technetium-99m ethyl cysteinate dimer (^{99m}Tc-ECD) 18 days after onset of bilateral ICAO showed additionally decreasing in the left MCA territory (Fig. 5B).

He had continued to receive conservative therapy including antiplatelet therapy after onset of bilateral ICAO. Although his left internal carotid artery (ICA) had made a spontaneous recanalization without his realizing it (Fig. 6A and 6B), he also had to be supported in all points of ADL. The SPECT of ^{99m}Tc-ECD after three months of onset of bilateral ICAO showed more decreasing in the left MCA territory than 18 days after (Fig. 6C). After one year, he died of his infectious disease.

Case 3

A man, who died at 80 years of age, had experienced left ICAO of unknown onset (no imaging) and right cerebral infarction at 75 (Fig. 7A and 7B). He had histories of myocardial infarction, hypertension and smoking, and had taken antihypertensive and antiplatelet agents. At the time of this attack, his height and weight were 170 cm and 61 kg, respectively. The physical examinations were normal, and neurological examinations revealed the presence of left side unilateral spatial neglect and gait disturbance due to mild left hemiparesis. Left Babinski sign was positive. The NIHSS score was 5. SPECT was not performed. After several months, he suffered from bilateral ICAO without knowing it (Fig. 8A and 8B).

He had continued to receive conservative therapy including antiplatelet therapy and had

a good clinical course over five years after suffering from bilateral ICAO, but died suddenly due to cardiac arrest of unknown origin.

DISCUSSION

Formerly, most bilateral ICAO has been able to be diagnosed by cerebral angiography, CT scan or autopsy[3)~11)], but nowadays it can be easily diagnosed by MR imaging, MR angiography, echography of carotid artery or their combination[2)12)]. There are many cases of unilateral ICAO, but less chance of diagnosing bilateral ICAO in ischemic cerebrovascular disease, the reported frequency of which was between one and nineteen percent[5)8)~10)]. In our three cases, bilateral ICAO was diagnosed by cerebral angiography or MR angiography.

Table 1 shows a summary of these three bilateral ICAO cases. All of them were thought to be caused by an atherothrombotic mechanism, because they had risk factors of cerebral infarction including diabetes mellitus, hyperlipidemia, hypertension and smoking. In case 1 after onset of bilateral ICAO, various collateral circulation to the right cerebral hemisphere had arisen from the basilar artery (BA) and right external carotid artery (ECA), while those to the left hemisphere had come from BA. On the other hand, collateral circulation from left ECA showed poor images (Fig. 1B). In case 2 and case 3, their collateral circulations after the onset of bilateral ICAO could not be discussed because of no study of cerebral angiography.

In 1981, Sakaki et al.[8)] reported that the patients with bilateral ICAO could be divided into three types. Patients with apoplectic type have

Table 1. Summary of three cases

	Case 1	Case 2	Case 3
Onset age of unilateral ICAO	50 y.o. (Right side)	72 y.o. (Right side)	Unknown (Left side)
Onset age of bilateral ICAO	57 y.o.	83 y.o.	75 y.o.
Onset mechanism of bilateral ICAO	Atherothrombotic (Slowly progressive occlusion in left side)	Atherothrombotic (Sudden-onset occlusion in left side)	Atherothrombotic (Asymptomatic occlusion in right side)
Risk factors	Diabetes mellitus, Hyperlipidemia, Smoking	Hypertension, Smoking	Hypertension, Smoking
Collateral circulation	BA, Right ECA→ Right cerebral hemisphere BA→Left Pcom→Left MCA ↓ Left cerebral hemisphere (Poor collateral circulation from left ECA)	(Could not be discussed because of no study of cerebral angiography)	(Could not be discussed because of no study of cerebral angiography)
Type of bilateral ICAO (Sakaki et al.)[8)]	Apoplectic type	Apoplectic type	? (Could not be well divided)
Therapy for bilateral ICAO	Conservative therapy including antiplatelet therapy	Conservative therapy including antiplatelet therapy	Conservative therapy including antiplatelet therapy
ADL and prognosis	Total assistance (mRS: Grade 5) Long-lived prognosis for 11.5 years	Total assistance (mRS: Grade 5) Survived only one year after onset of bilateral ICAO	Independence (mRS: Grade 1) Long-lived prognosis for 5 years
Cause of death	Chronic heart failure at 68 y.o.	Infectious disease at 84 y.o.	Cardiac arrest of unknown origin at 80 y.o.

ICAO: internal carotid artery occlusion; ADL: activities of daily living; BA: basilar artery; ECA: external carotid artery; Pcom: posterior communicating artery; MCA: middle cerebral artery; mRS: modified Rankin scale

severe neurological deficits acutely after occlusion, their angiographies show impaired or absent collateral circulation, and their CT scans demonstrate diffuse low-density areas bilaterally; with the progressing type, those neurological signs and symptoms grew progressively and more severely; their angiographies showed moderate or adequate retrograde filling with collateral circulations via posterior cerebral arteries and CT scans demonstrated low-density lesions in the bilateral frontal lobes. In the TIA・RIND repeated type, the angiographies of those who had transient ischemic attacks or reversible ischemic neurological deficits repeatedly evidenced good normograde filling of bilateral internal carotid artery with collateral circulation through posterior communicating arteries, and CT scans demonstrated no abnormal low-density areas except findings of brain atrophy[8]. According to Sakaki's division, case 1 and case 2 would be rather Apoplectic type, but case 3 could not be well classified. We think it would be better to define case 3 as another new kind of symptom, for example, asymptomatic type.

Antiplatelet therapy is recommended as an acute therapeutic strategy for bilateral ICAO caused by an atherothrombotic mechanism. Furthermore, one must also manage diabetes mellitus, hyperlipidemia, hypertension and smoking. In the chronic stage, the extracranial-intracranial (EC-IC) bypass surgery is also recommended. However, EC-IC bypass surgeries were not indicated in case 1 and 2, because the modified Rankin Scale (mRS) was Grade 5 (severe disability: bedridden, incontinent, and requiring constant nursing care and attention)[13], respectively. In case 3, surgery was not also appropriate, because the bilateral ICAO was caused asymptomatically at the ripe old age of 75 in spite of Grade 1 of mRS (no significant disability despite symptoms: able to carry out all usual duties and activities)[13] and the patient had no opportunity to perform the rCBF using SPECT.

The cause of death in case 1 and 3 might be of cardiac origin. Lazarides et al. reported that coronary ischemia, rather than stroke, was the main cause of death in patients with bilateral ICAO[10]. Bilateral ICAO shows various clinical features resulting in a different clinical course. When managing bilateral ICAO, one must note the various pathologies. As for bilateral ICAO, further studies will be necessary.

Author's disclosure of potential Conflict of Interest (COI).
Masayuki Izumi: Astra Zeneca and Bayer AG

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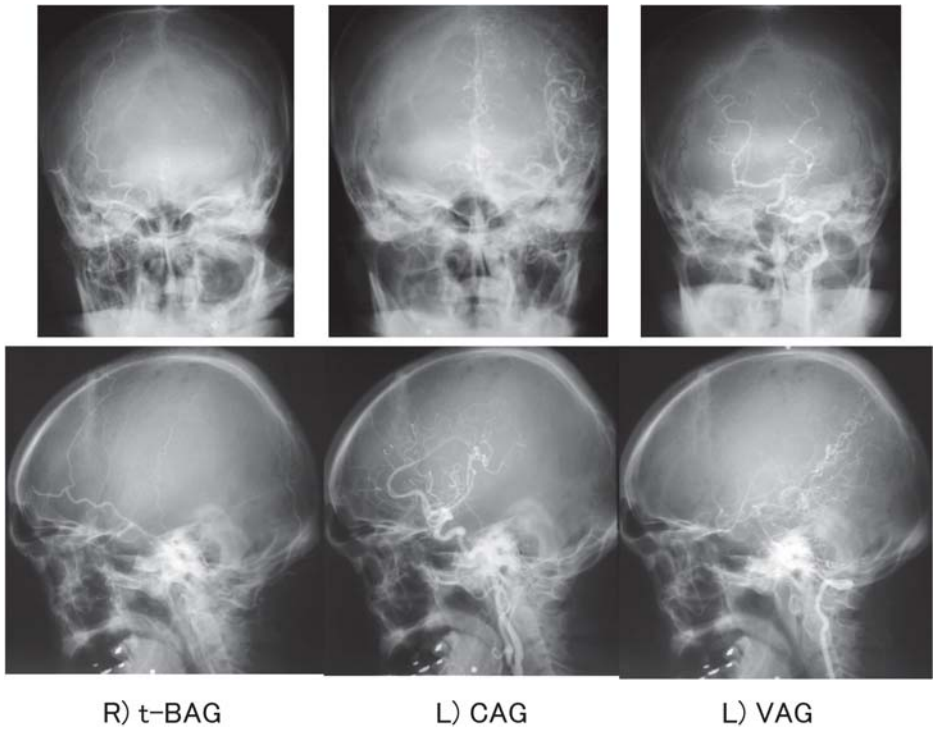

Figure 1A. Case 1: Cerebral angiography at 50 years of age showed right ICAO.

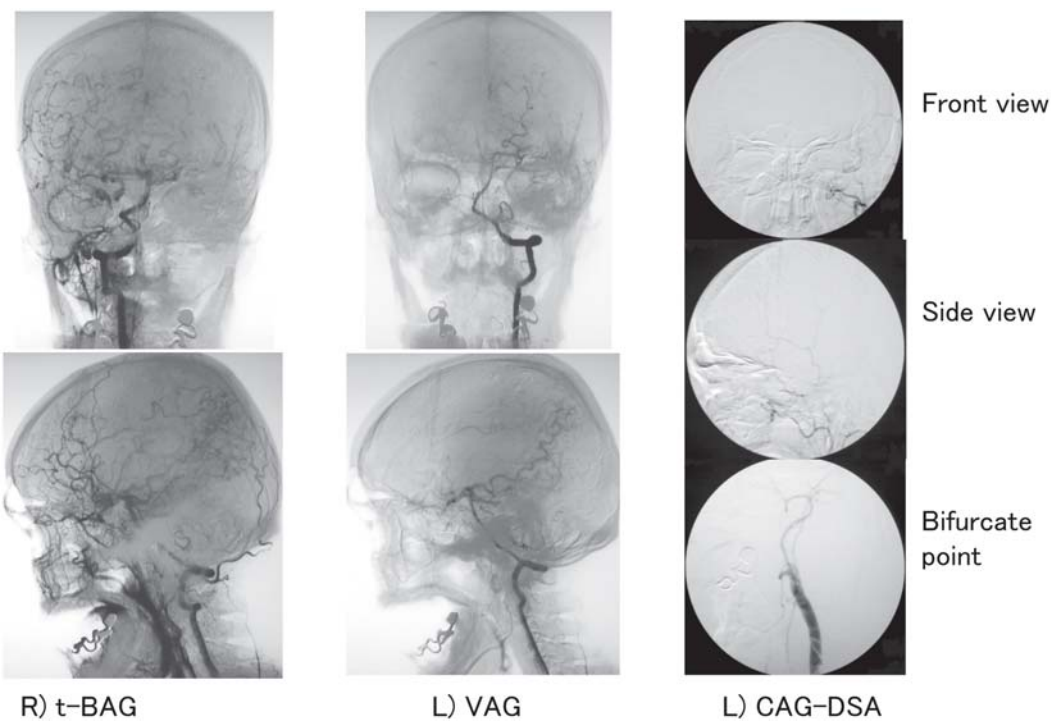

Figure 1B. Case 1: Cerebral angiography at 57 years of age showed bilateral ICAO.

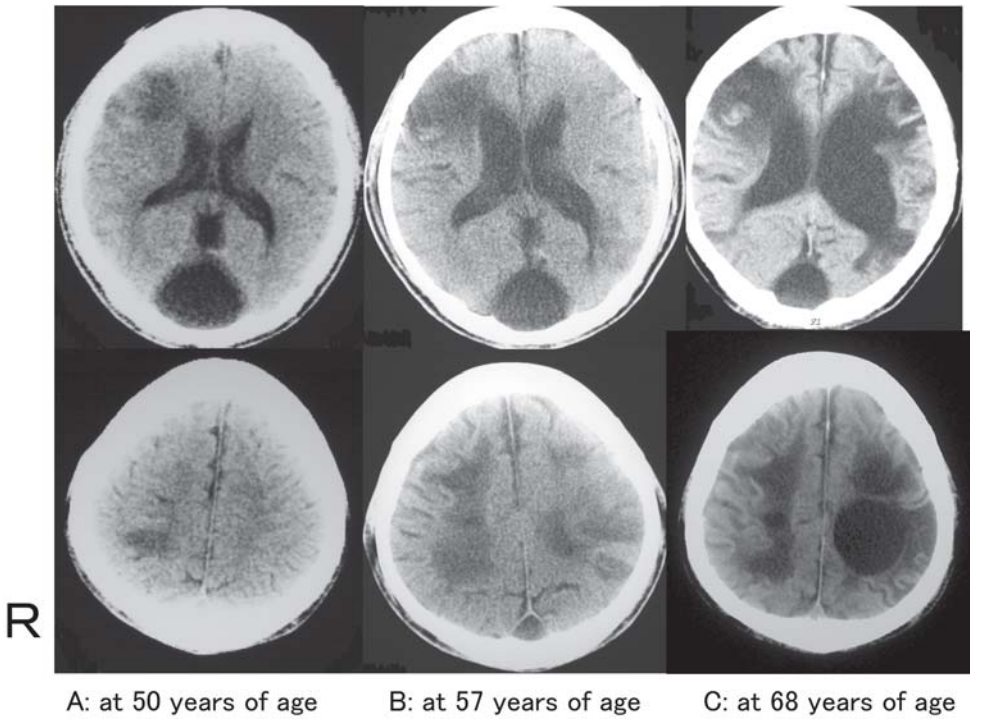

Figure 2. Case 1: Brain CT at 50 (A), 57 (B), and 68 (C) years of age. Low-density areas appeared in right hemisphere at 50, but spread over left hemisphere at 57 and 68.

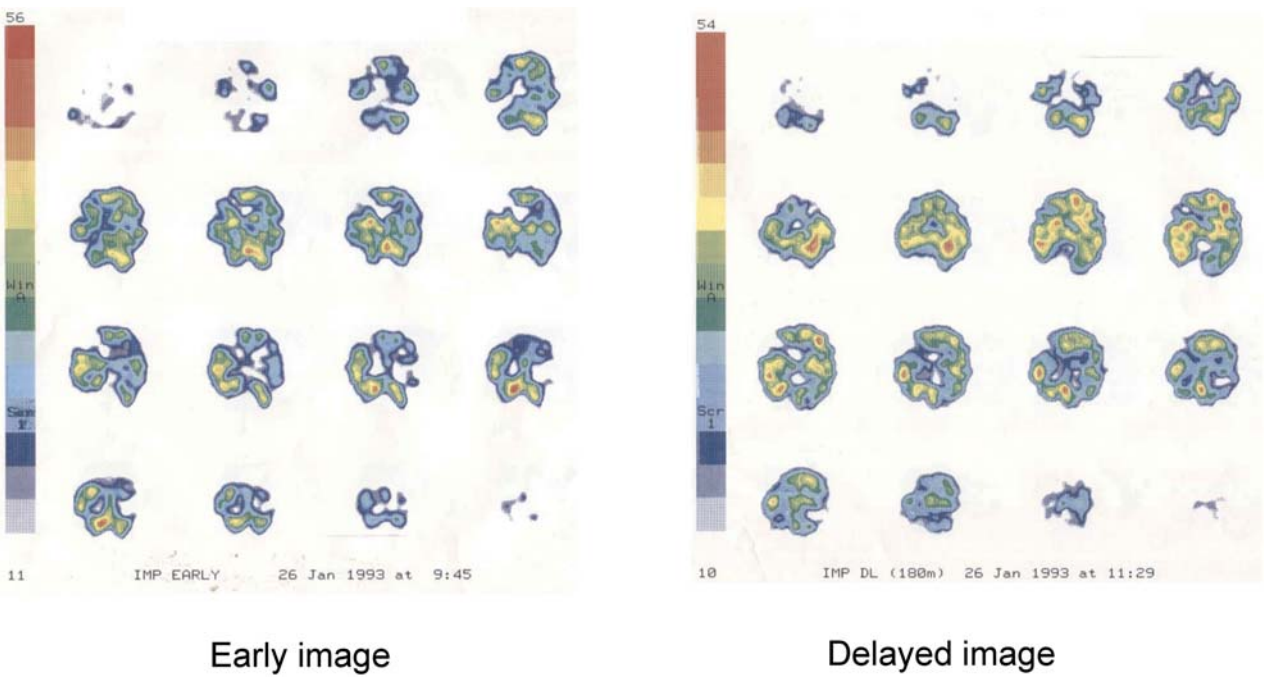

Figure 3. Case 1: The rCBF using SPECT of IMP performed a month after onset of bilateral ICAO at 57 years of age showed focally decreased in the bilateral cortical ACA-MCA and MCA-PCA watershed territories for both early and delayed image.

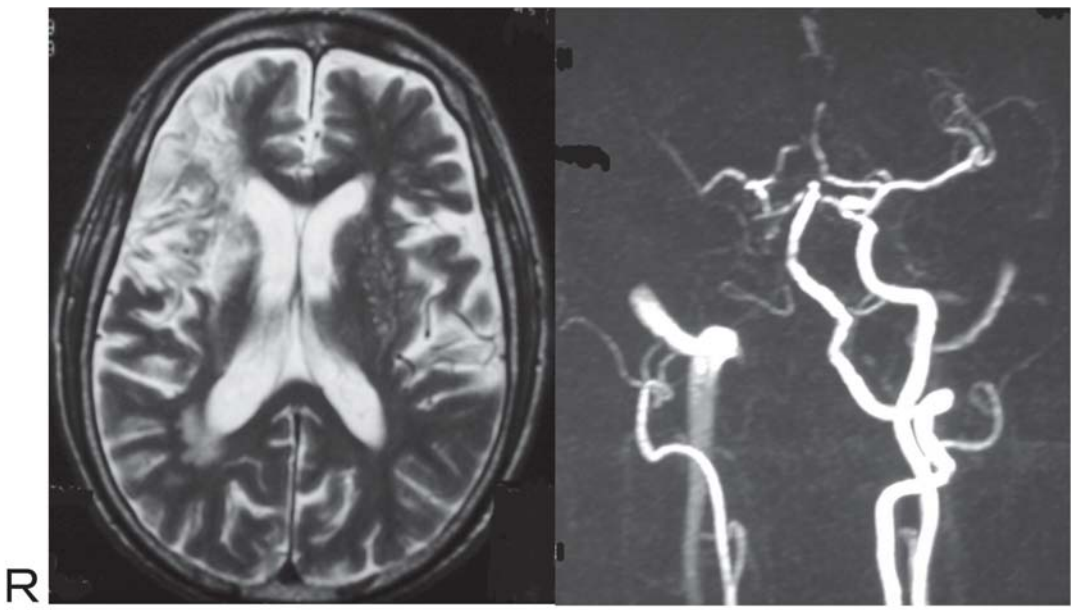

Figure 4A. Case 2: Brain MR imaging and MR angiography at 72 years of age showed right cerebral infarction and right ICAO.

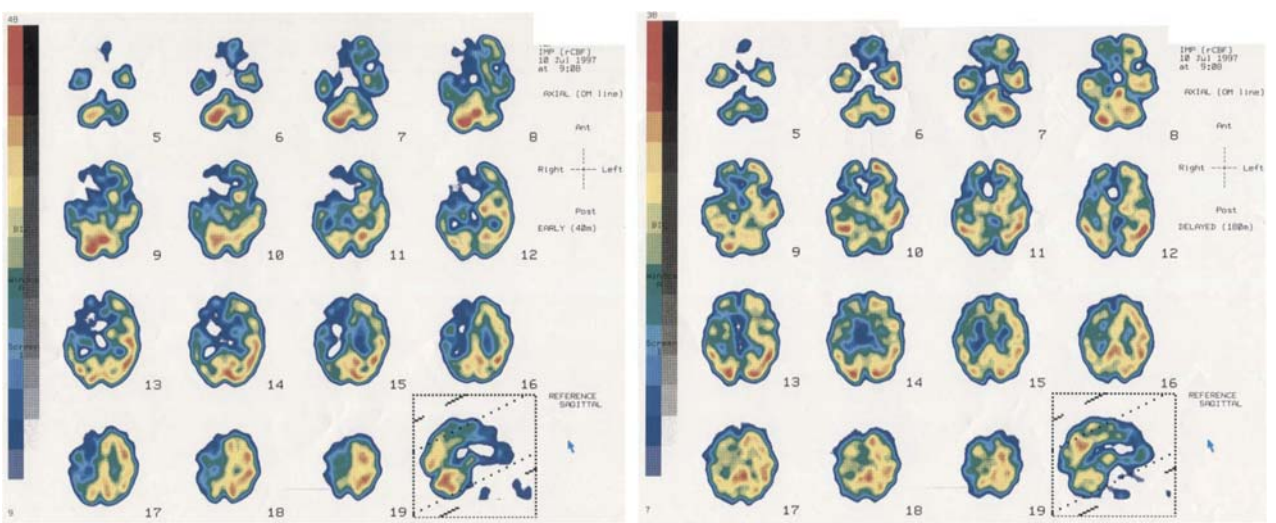

Figure 4B. Case 2: The rCBF using SPECT of IMP performed at 72 years of age showed focally decreased in the right MCA territory for both early and delayed image.

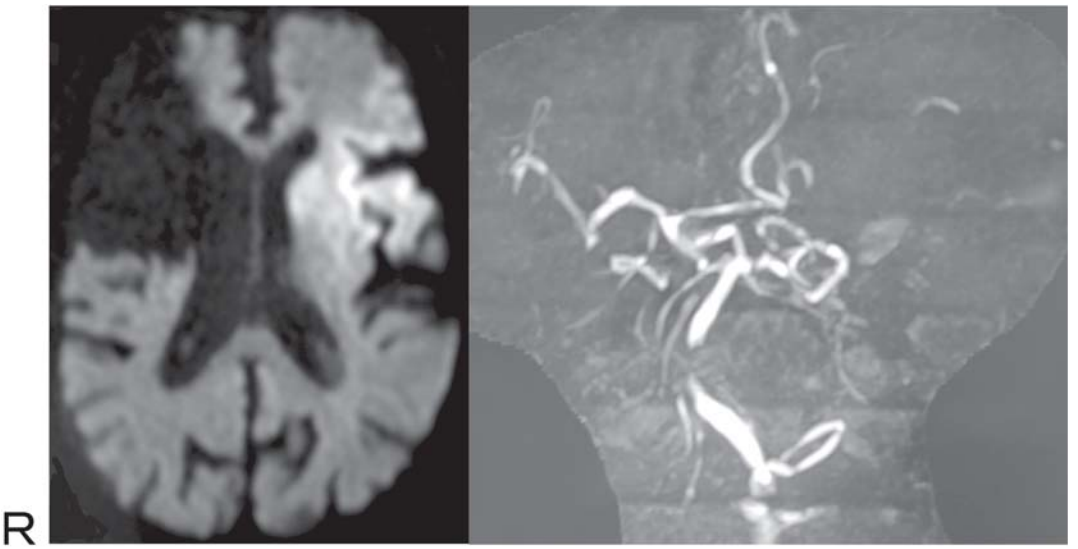

Figure 5A. Case 2: Brain MR imaging and MR angiography at 83 years of age showed acute left cerebral infarction and bilateral ICAO.

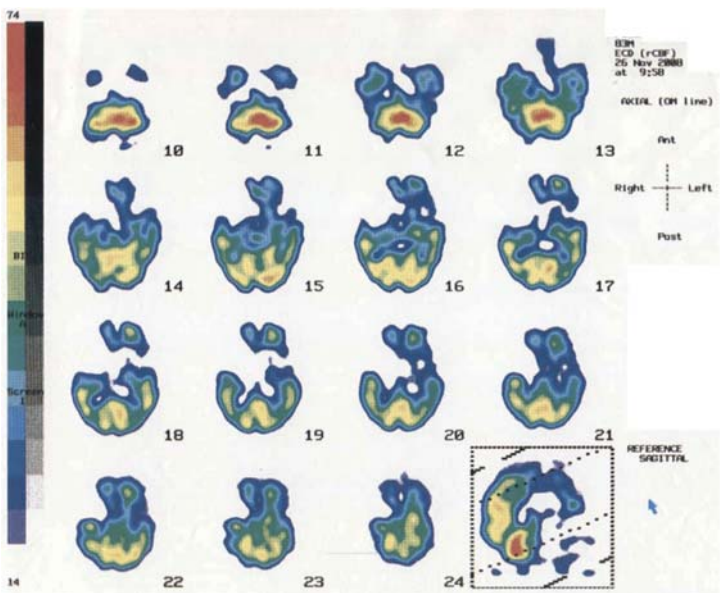

Figure 5B. Case 2: The rCBF using SPECT of ^{99m}Tc-ECD 18 days after onset of bilateral ICAO showed additionally decreasing in the left MCA territory.

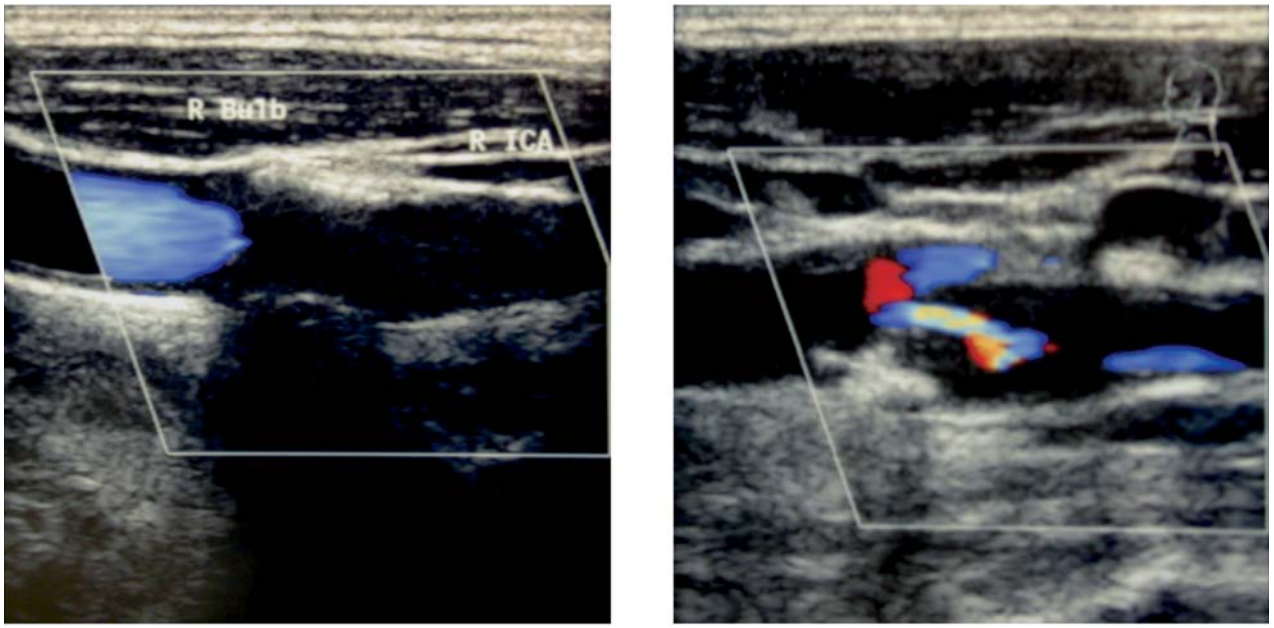

Figure 6A. Case 2: Echography of carotid artery after 20 days of onset of bilateral ICAO showed a spontaneous recanalization of left ICA.

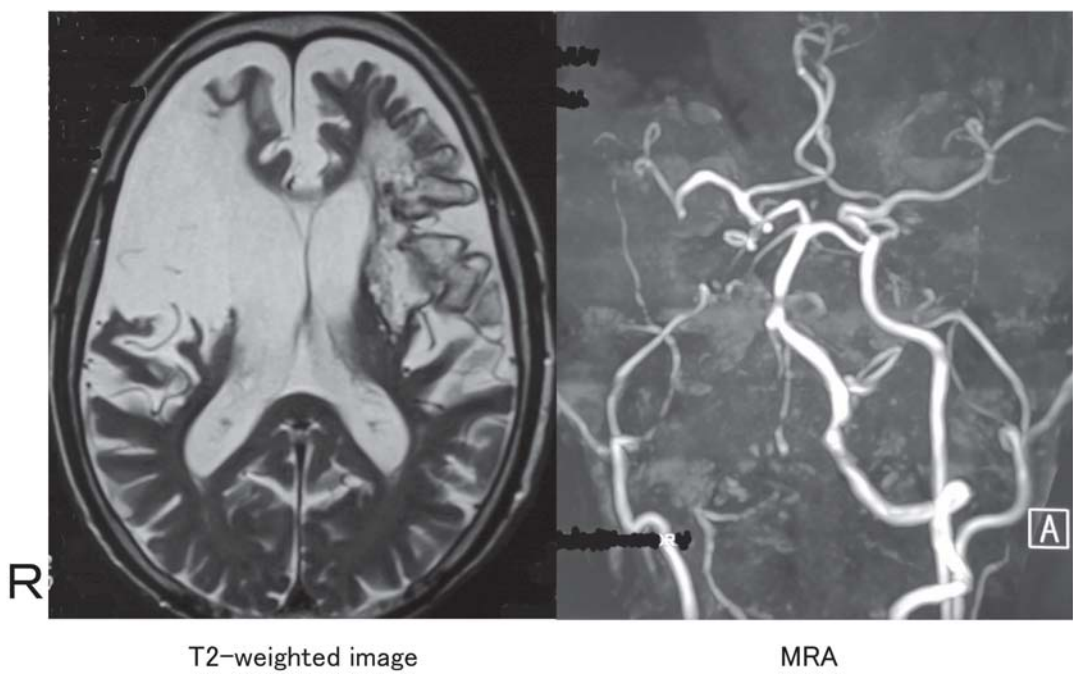

Figure 6B. Case 2: Brain MR imaging and MR angiography after three months of onset of bilateral ICAO showed bilateral old cerebral infarction and a spontaneous recanalization of left ICA.

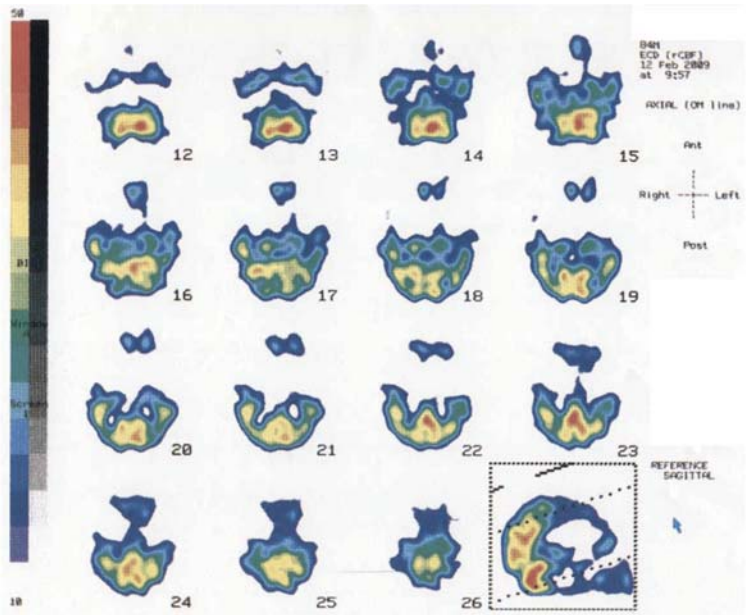

Figure 6C. Case 2: The rCBF using SPECT of ^{99m}Tc-ECD after three months of onset of bilateral ICAO showed more decreasing in the left MCA territory than 18 days after.

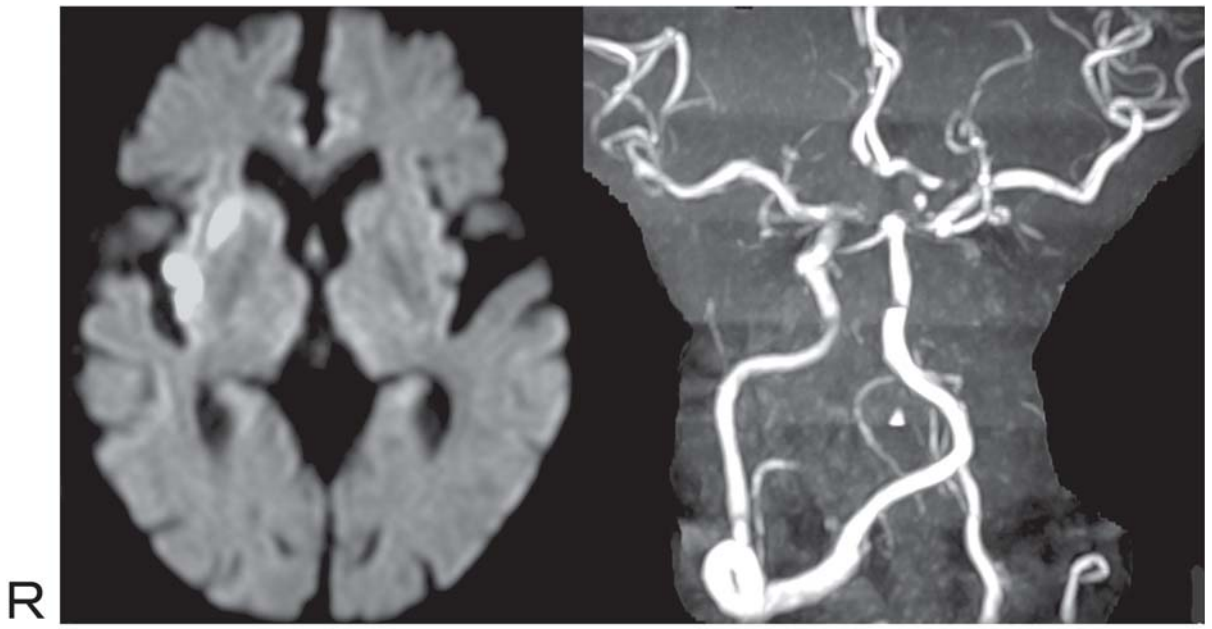

Figure 7A. Case 3: Brain MR imaging and MR angiography at 75 years of age showed acute right cerebral infarction and left ICAO.

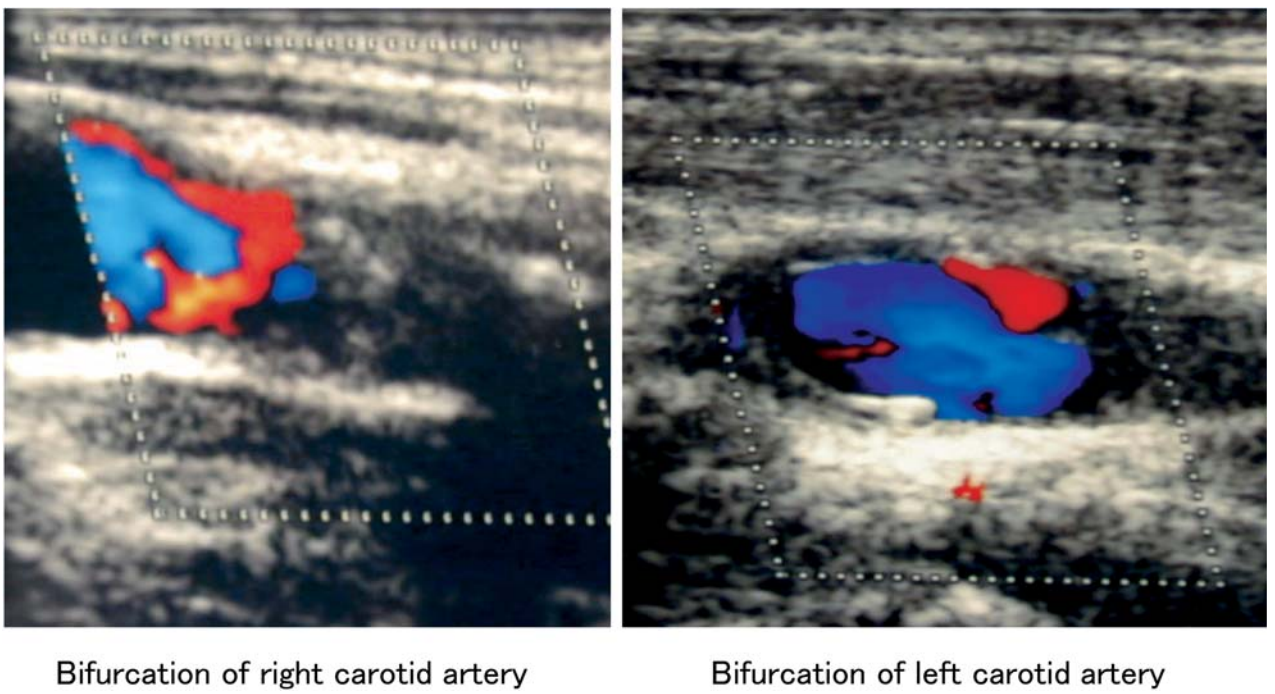

Figure 7B. Case 3: Echography of carotid artery at 75 years of age showed left ICAO.

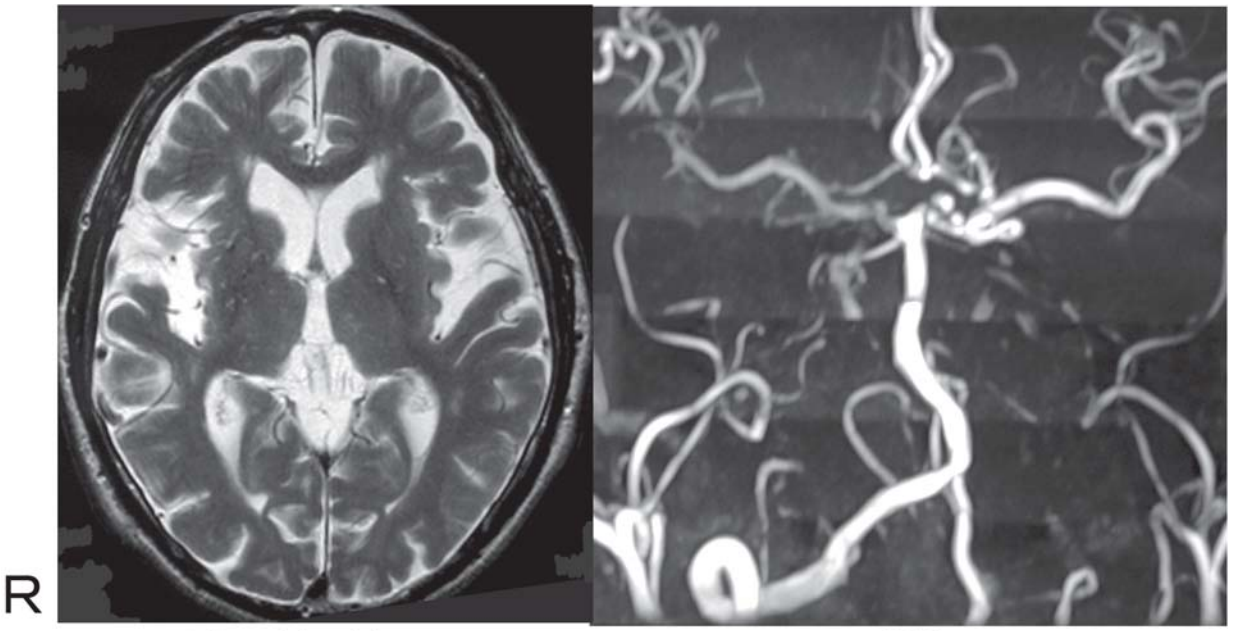

Figure 8A. Case 3: Brain MR imaging and MR angiography after 7 months showed old right cerebral infarction and bilateral ICAO.

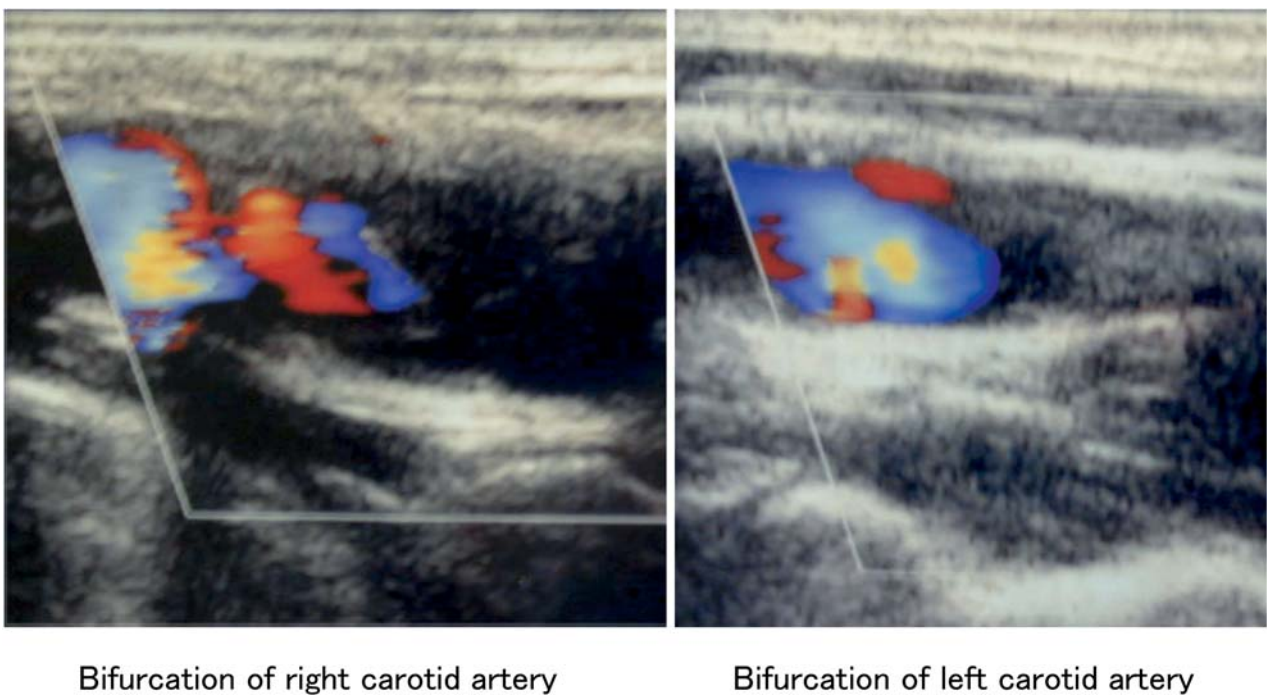

Figure 8B. Case 3: Echography after 7 months showed bilateral ICAO.