

# An Aneurysm Associated with Moyamoya Disease

—A Followup Study by Computed Tomography—<sup>\*)</sup>

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(Received November 18, 1982)

*Key words: Cerebral aneurysm, CT scan, Moyamoya disease, Ventricular hemorrhage*

## ABSTRACT

We recently experienced a case which had an aneurysm with Moyamoya disease. He developed by a sudden severe headach, vomiting and conscious disturbance. CT was performed, and it revealed hematoma having a diameter of 1.0 cm adjacent to the anterior horn of the lateral ventricle in the head of the left caudate nucleus, and both lateral ventricles, 3rd ventricle, and 4th ventricle were found to be filled with clot. On the 17th day after onset, cerebral angiography was performed and it was found that both internal carotid arteries were strongly stenotic and Moyamoya vessels araised from bifurcation of each internal carotid artery. A spherical aneurysm 3×3 mm in size was seen in the periphery of the Moyamoya vessels, and its location was coincided with intracerebral hematoma on CT. This aneurysm were observed in enhanced CT, and became to smaller and smaller. We performed repeat cerebral angiography on 72nd day after onset, and found the aneurysm to be disappeared.

## INTRODUCTION

Reports of aneurysm associated with Moyamoya disease are relatively rare, but the reported number of cases has exceeded 30. We recently experienced a case which developed by subarachnoid hemorrhage. By CT, intracerebral hematoma and intraventricular hemorrhage were observed and by cerebral angiography, an aneurysm was detected in the periphery of the Moyamoya vessels, but it later disappeared. A report of this case which was followed by CT and cerebral angiography will be presented.

## CASE

The patient is a 31 year old male whose occupation is automobile mechanic. Family history showed his mother to have diabetes mellitus. He gave a past history of bilateral tonsillitis at the age of 25, for which he received tonsillectomy. Otherwise, there is nothing remarkable.

As for his present illness, while repairing a

bus in the afternoon of 30 March 1981 he suddenly developed a severe headache and then he drove his car to see a physician. On his way home, he vomited, developed clouding of consciousness, and thereafter was taken to a hospital. At the hospital he was in an excited state with motor restlessness. About 22 hours after onset, he was referred to this Department.

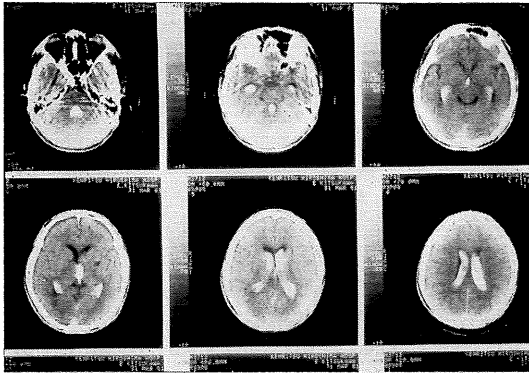
At the time of admission, his eyes were open and he could converse, but he was slightly excited with severe disorientation. He complained of severe headache but was not aware of his disease. Neurological examination showed no abnormality of the cranial nerve with normal muscle strength of both extremities. Deep tendon reflex was normal without any pathologic reflex. Körnig's sign and nuchal rigidity were both positive. Blood chemistry showed leucocytosis (17,000/mm<sup>3</sup>) and hyperglycemia (300 mg/dl). The patient is still under drug therapy under the diagnosis of diabetes mellitus.

CT of the head performed at time of admission revealed hematoma having a diameter of

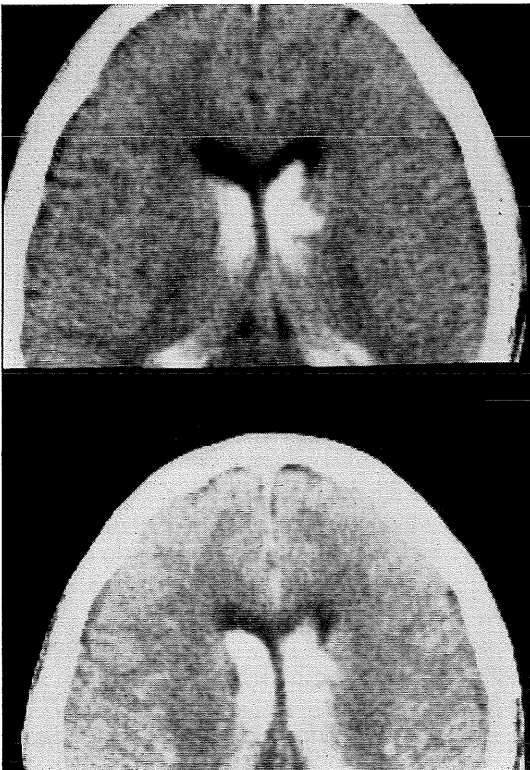
<sup>\*)</sup> 吉原高志, 北岡 保, 富原健司, 木矢克造, 野村雅之: モヤモヤ病に合併した脳動脈瘤——CT による追跡——

1.0 cm adjacent to the anterior horn of the lateral ventricle in the head of left caudate nucleus and both lateral ventricles, 3rd ventricle, and 4th ventricle were filled with clot. Hydrocephalus was not observed. The source of hemorrhage was the head of left caudate nucleus.

The hematoma ruptured the ependyma and penetrated into the anterior horn of the left ventricle where it spread. A small low density



**Fig. 1.** CT scans on admission (on the next day of onset).



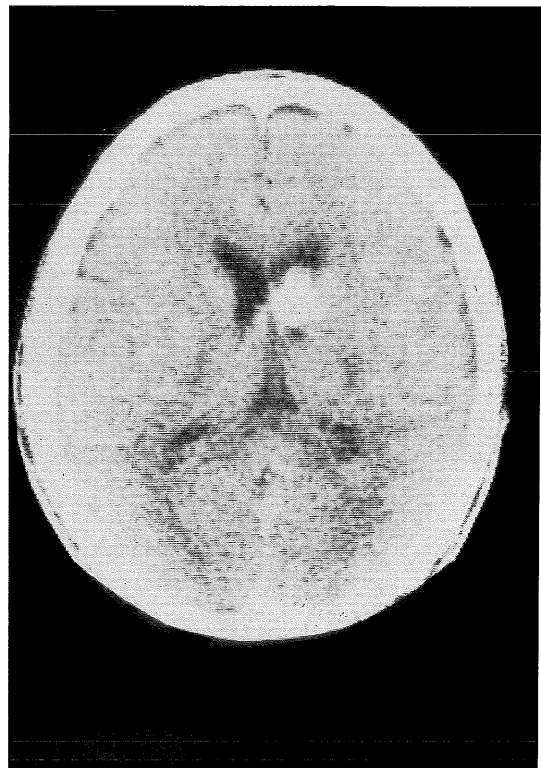
**Fig. 2.** CT scans on admission. Plain CT (upper) and enhanced CT (lower).

area was observed in the left putamen distal from the hemorrhagic site. By enhanced CT, the enhancement of the proximal region of the middle cerebral arteries of both sides was weak and no abnormally enhanced site was detected (Fig. 1). The site of the hematoma which was the source of hemorrhage did not show any change (Fig. 2).

The clinical findings and CT findings strongly suggested Moyamoya disease. As his state of consciousness was relatively good and was improving and hydrocephalus was not observed, his course was followed by conservative therapy. Lethargic tendency was observed until about the 10th day after onset and his response while he was awake gradually became firm and his appetite improved.

By plain CT taken on the 8th day after onset, the hematoma within the ventricle had disappeared and intracerebral hematoma in the left caudal head and the subependymal hematoma adjacent thereto remained as a high density area (Fig. 3).

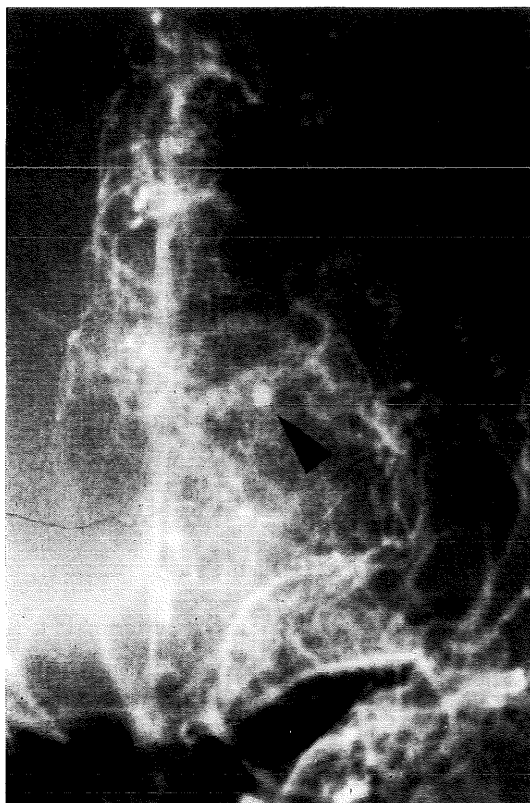
On the 17th day after onset, bilateral internal



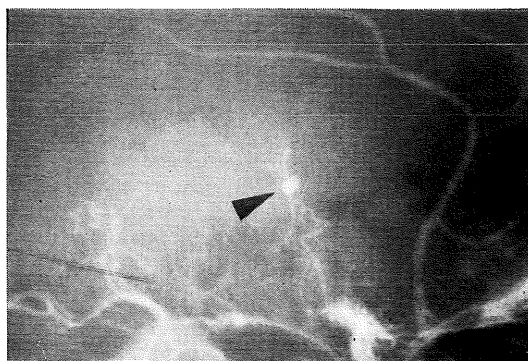
**Fig. 3.** CT scans on the 8th day after onset (plain CT).

carotid angiography and vertebral angiography were performed. Wall irregularity and stenosis of the right and left internal carotid arteries were observed from C<sub>2</sub> to the peripheral region. Both the right and left middle cerebral arteries showed severe stenosis. From M<sub>2</sub> to the peripheral region, orthograde visualization was possible in one part and in the other part retrograde visualization was possible by leptomeningeal anastomosis via the anterior and posterior cerebral arteries. Both the left and right anterior cerebral arteries were larger than normal and on both sides of the bifurcation of the internal carotid artery were seen Moyamoya vessels running upward in the posterior direction. No abnormal findings could be observed in the subtentorial blood vessels.

By left internal carotid angiography, a spherical aneurysm 3×3 mm in size was seen in the periphery of the Moyamoya vessels. In measuring its location, it was 1.0 cm superior and anterior to the venous angle and 1.3 cm lateral to the midline and coincided with the location



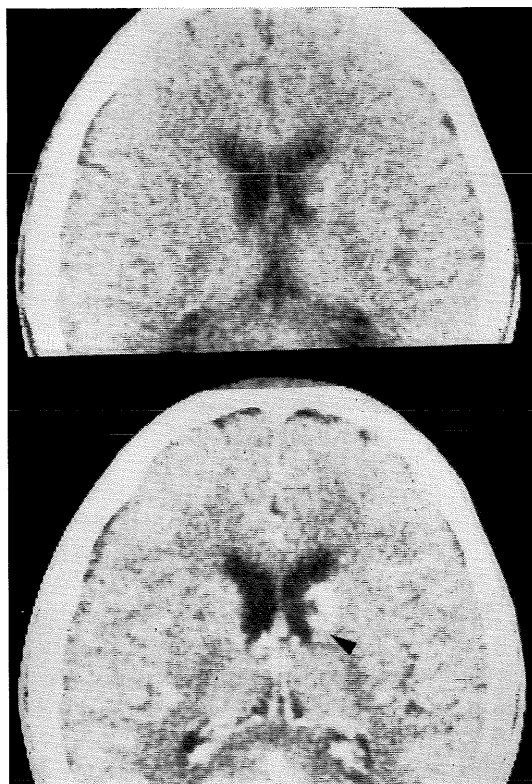
**Fig. 4.** Left carotid angiogram on the 17th day after onset (A-P view). Arrow shows an aneurysm.



**Fig. 5.** Left carotid angiogram on the 17th day after onset (lateral view). Arrow shows an aneurysm.

of the intracerebral hematoma in the left caudal head. This aneurysm could be observed during the early period from the arterial phase to the venous phase (Fig. 4 and Fig. 5).

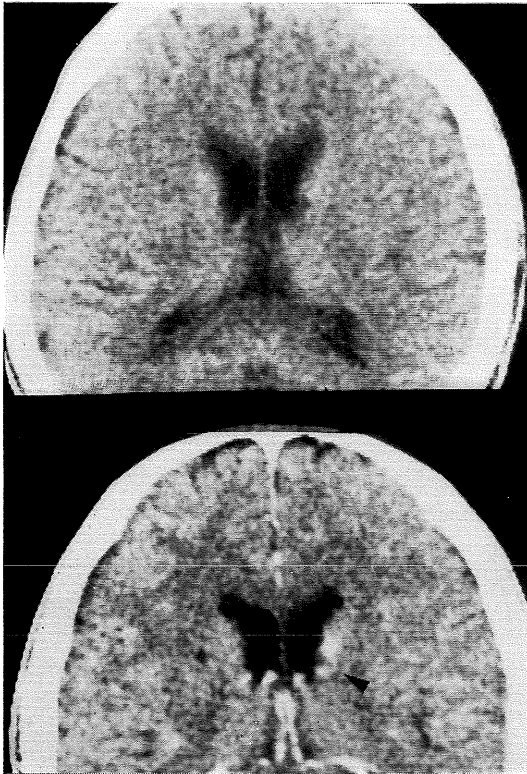
By CT conducted on the 22nd day after onset, the site of the intracerebral hematoma appeared to be a small low density area which was enhanced in a ring shape by the contrast



**Fig. 6.** CT scans on the 22nd day after onset. Plain CT (upper) and enhanced CT (lower). Arrow shows the image of aneurysm.

medium. On the medial side was a small round well demarcated and enhanced area of high density. This was ascertained to be the image of the aneurysm observed on the angiogram (Fig. 6).

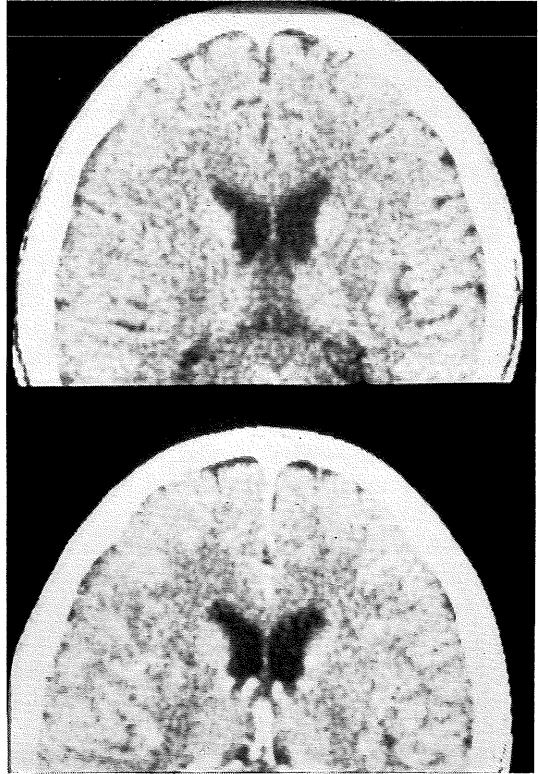
By CT conducted on the 50th day after onset, the site of the hematoma was poorly enhanced and the site of the aneurysm was not enhanced, being an area of low density (Fig. 7).



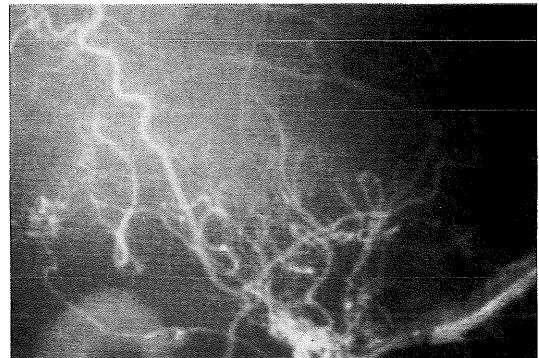
**Fig. 7.** CT scans on the 50th day after onset. Plain CT (upper) and enhanced CT (lower). Arrow shows the image of aneurysm.

The CT findings observed on the 64th day after onset were identical to those observed on the 50th day (Fig. 8).

These findings suggested that the aneurysm had disappeared and therefore on the 72nd day after onset cerebral angiography was repeated, which revealed disappearance of the aneurysm. There was no change in the degree of stenosis of the internal carotid artery, middle cerebral artery, and anterior cerebral artery, but the diameter of the Moyamoya vessels was smaller (Fig. 9 and Fig. 10).

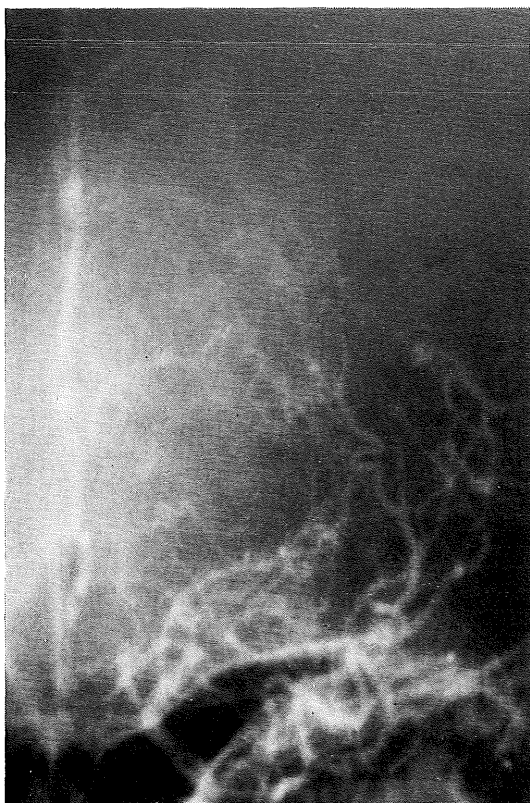


**Fig. 8.** CT scans on the 64th day after onset. Plain CT (upper) and enhanced CT (lower).



**Fig. 10.** Left carotid angiogram on the 72th day after onset (lateral view). Any aneurysm is not seen.

The consciousness of the patient became clear on the 3rd week after onset and on the 83rd day and 129th day after onset right and left STA-MCA anastomosis and encephalomyosynangiosis were performed, respectively. At present about one year after onset the patient is neurologically normal and has return to work without any repeat hemorrhage.



**Fig. 9.** Left carotid angiogram on the 72th day after onset (A-P view). Any aneurysm is not seen.

## DISCUSSION

Moyamoya disease presents on cerebral angiography a finding of stenosis or occlusion of the terminal region of the bilateral internal carotid arteries associated with rete mirabile at the brain base. As rete mirabile at the brain base appear "moyamoya", this condition is called Moyamoya disease. The entity of this disease is yet unknown and is still being debated, but the present concept of this disease is as described below.

1) The cause is still unknown. There is the congenital theory and the acquired theory, but more than one cause might be involved<sup>12, 19, 26</sup>.

2) Clinically, it can be classified into the juvenile type and the adult type. The age of predilection of the former type is 5-6 years and it develops with brain ischemia. The age of predilection of the latter type is 20-50 years and it develops with intracranial hemorrhage<sup>12, 19, 20, 26</sup>.

3) There is no established theory whether the juvenile type and the adult type are essentially the same disease or whether they are different disease. This is still being debated<sup>12, 19, 26</sup>.

4) The rete mirabile at the brain base (Moyamoya vessels) are collateral pathways for occluded or narrowed trunk arteries<sup>11, 17</sup>.

5) As for the entity of Moyamoya disease, it is the dominant theory that they are perforators (medial and lateral lenticulostriate arteries) which are dilated and tortuous<sup>11, 29</sup>. There are other theories such as abnormal development of new blood vessels and malformation<sup>12, 19</sup>.

6) As for the prognosis of this disease, both life prognosis and function prognosis are considered to be poor if left to take its natural course.

7) As for therapy, blood vessel anastomosis is surgically performed and angiographically this procedure is effective in improving blood flow. However, at the present stage it is still under review whether surgery would bring about long term improvement in prognosis<sup>3, 25, 28</sup>.

The foregoing are the present concepts of and problem points in Moyamoya disease.

A relatively large number of case reports have been made on Moyamoya disease associated with aneurysm<sup>1-7, 9, 10, 12-16, 18, 21-24, 27, 30-32</sup> (Table 1). In classifying these cases, the associated aneurysm can be divided into two types. The first type is aneurysm which is located in the periphery of the anterior and posterior choroidal arteries or the Moyamoya vessels as in the case presented in this report. The other is saccular aneurysm which develops in the circle of Willis at the brain base. There has been a report of both types being observed in one individual<sup>16</sup>. The latter aneurysm is similar angiographically to aneurysm which develops with subarachnoid hemorrhage, but the frequency of aneurysm of the basilar artery-posterior cerebral artery system is high. The predominant view is that this aneurysm develops from the increase in blood flow volume in the basilar artery-posterior cerebral artery system through the occlusion of the bilateral internal carotid arteries<sup>2, 15, 16</sup>.

Aneurysm at the periphery of the Moyamoya vessels and that of the choroidal arteries are both found in the brain parenchyma and are

**Table 1.** Review of literature for Moyamoya disease with aneurysm

No.	Author, year	case	site of AN.	symptoms, prognosis
1.	Kamisasa, 1972	34y, male	Moyamoya vessel	SAH, AN not disappear, died from rebleeding
2.	Debrun, 1974	18y, male	Internal carotis (cavernous part)	?
3.	Takeyama, 1976	43y, male	A. ch. artery	SAH
4.	Kodama, 1976	16y, female	P. ch. artery	SAH, AN disappeared after 42 days
5.	Kodama, 1976	39y, male	P. ch. artery	SAH, AN disappeared after 11 months
6.	Kodama, 1976	48y, male	P. ch. artery	SAH, AN disappeared after 30 days
7.	Yasargil, 1976	38y, male	A-com. artery	SAH, clipping was performed
8.	Yasargil, 1976	38y, female	ACA (A2)	SAH, clipping was performed
9.	Kodama, 1977	35y, male	Basilar top	SAH, AN not disappeared
10.	Kodama, 1977	42y, male	Basilar top	SAH, wrapping was performed
11.	Lee, 1977	29y, male	Moyamoya vessel	SAH
12.	Tanaka, 1978	18y, male	Moyamoya vessel	SAH, AN disappeared after 9 months
13.	Tanaka, 1978	40y, male	PCA	VH, craniotomy and removal of AN
14.	Tanaka, 1978	57y, female	A. ch. artery	VH, died from first bleeding
15.	Adams, 1979	35y, male	Basilar top	non-rupture AN, clipping was performed
16.	Adams, 1979	34y, male	IC-PC	SAH
17.	Adams, 1979	32y, female	Basilar top	SAH
18.	Adams, 1979	45y, female	Basilar top and PCA	Clipping was performed
19.	Muraki, 1981	22y, female	Moyamoya vessel	VH, died from pulmonary edema
20.	Izawa, 1981	? ?	A. ch. artery	SAH,?
21.	Nakao, 1981	57y, female	?	Intracerebral hematoma in R frontal lobe
22.	Nagamine, 1981	41y, female	Basilar artery (non-rupture)	VH, died
23.	Yamada, 1981	42y, female	A. ch. artery	VH,?
24.	Yamada, 1981	54y, female	A. ch. artery	Intracerebral hematoma (basal ganglia)
25.	Yamada, 1981	37y, male	P. ch. artery	VH,?
26.	Tani, 1981	39y, female	P. ch. artery	VH, AN disappeared after 4 months
27.	Funakoshi, 1981	29y, female	Moyamoya vessel	Hematoma in thalamus, AN disappeared after 80 days
28.	Arimits, 1981	? ?	Moyamoya vessel	? ?
29.	Arimits, 1981	36y, male	Basilar top	? ?
30.	Izawa, 1981	49y, male	Basilar top	SAH, hemianopsia
31.	Matsumoto, 1981	67y, male	Moyamoya vessel	Subcortical hematoma in parietal lobe, craniotomy and removal of AN
32.	Okamoto, 1981	46y, female	Moyamoya vessel	Hematoma in thalamus AN disappeared after STA-MCA bypass
33.	Nagamine, 1981	36y, male	1. Moyamoya vessel 2. IC-PC 3. PCA 4. Basilar artery	VH

A. ch. artery=Anterior choroidal artery, P. ch. artery=Posterior choroidal artery, A-com=Anterior communicating artery, ACA=Anterior cerebral artery, PCA=Posterior cerebral artery, IC-PC=Internal carotis-posterior communicating artery, SAH=Subarachnoid hemorrhage, VH=Ventricular hemorrhage

located close to the lateral ventricular wall<sup>7,23)</sup>. If located at the peripheral region of the Mo-

yamoya vessels, as in our case, hemorrhage develops from the caudal head and putamen



and hematoma penetrates into the anterior horn of the lateral ventricle, and if located at the peripheral region of the anterior and posterior choroidal arteries, it penetrates into the inferior horn and posterior horn. Some authors have classified them into aneurysm of the peripheral region of the Moyamoya vessels and that of the peripheral region of the choroidal artery<sup>21, 30)</sup>, but if we were to accept the view that Moyamoya vessels are dilated collateral pathway of the lenticulostriate arteries<sup>11, 17, 29)</sup>, they are perhaps aneurysms which develop by the same mechanism, and if we were to accept the view that Moyamoya vessels are newly developed abnormal blood vessels, this may be considered to be a meaningful classification. In either case the cerebral angiographic patterns are identical and the clinical symptoms which develop from ventricular hemorrhage do not appear to be different.

There are only a few reports on the frequency of cerebral aneurysm at the peripheral region of the Moyamoya vessels and that of the anterior and posterior choroidal arteries. According to Kudo<sup>31)</sup>, aneurysm having a diameter of 1 mm or more has been detected in one case out of 11 cases of juvenile type Moyamoya disease and in 4 cases out of 13 cases of adult type Moyamoya disease, giving a frequency of 31% for the adult type. If the detected cases of aneurysm having a diameter of less than 1 mm are included, the frequency would be 2 cases out of 11 cases of the juvenile type and 7 cases out of 13 cases of the adult type to increase the frequency rate. These aneurysms having a diameter of less than one mm were detected by enlarged cerebral angiograms and include cases without a history of intracranial hemorrhage. Through detailed observation, the frequency of aneurysm is found to be much higher than expected.

In reviewing the reported cases individually, the disease develops from either ventricular hemorrhage or subarachnoid hemorrhage and through the angiograms taken at time of hemorrhage the aneurysm is detected. Ventricular hemorrhage has been confirmed by CT or angiography, but before the introduction of CT, it is considered that among the cases in which the disease was assumed to have developed from subarachnoid hemorrhage there should be cases of ventricular hemorrhage.

In the cases reported by Funakoshi et al.<sup>4, 22)</sup>, aneurysm was present at the peripheral region of the Moyamoya vessels and during repeat cerebral angiography the aneurysm ruptured and thus the penetration of the contrast medium into the ventricle was observed by chance. This is a case in which it was directly confirmed that the source of hemorrhage was aneurysm. The other methods of confirming the source of hemorrhage are CT, ventriculography, and autopsy<sup>7, 23, 30)</sup>, and through these procedures hemorrhage from the aneurysm has been confirmed. In our case, it was concluded from a comparative review of the CT findings and angiographic findings that the source of hemorrhage was aneurysm at the peripheral region of the Moyamoya vessels.

As for the fate of aneurysm, in many cases spontaneous disappearance has been observed in 1-9 months. There have been two reported cases in which the aneurysm was removed by direct operation<sup>5, 30)</sup>, and one case of disappearance following vascular anastomosis<sup>21)</sup>. In the case reported by Kamisasa et al.<sup>21)</sup> the aneurysm did not disappear for three years and during this period the size of the aneurysm increased. The patient died three years later with repeat rupture. In our case the aneurysm disappeared about 2½ months after onset and at present even after about one year after onset there has been no repeat hemorrhage.

As for the cause of aneurysm, due to the excessive blood flow load on the Moyamoya vessels serving as collateral pathway, a localized dilatation of the vascular wall develops to bring rise to rupture. Pathologically, rupture of the elastica interna and destruction of the tunica media structure have been observed with excessive enlargement of the lumen<sup>8, 11, 17, 20, 29)</sup>. Rupture of the elastica tunica and destruction of the tunica media structure are findings commonly observed in the wall of the Moyamoya vessels<sup>20, 29)</sup>, and it is considered that the site with severe dilatation ruptures which is observed as aneurysm. Angiographically, there are two divided views regarding the wall of the aneurysm; the first is the view that it is a hematoma cavity formed by hemorrhage, that is, it is a pseudoaneurysm, and the second is the view that it is a true aneurysm surrounded by arterial wall<sup>29, 30)</sup>. The former has not been confirmed histologically, but it is supported by

the fact that spontaneous disappearance has been observed and that the aneurysm density remains to the venous phase on the angiogram<sup>9)</sup>. On the contrary, in view of the fact that there are cases in which the size continues to enlarge without disappearing, it may be possible that the two exist depending on the case.

The presence or absence of repeat hemorrhage is of importance in deciding conduct of therapy. It is generally said that the frequency of repeat hemorrhage of the Moyamoya vessels is lower than that of aneurysm of the circle of Willis of the brain base, but if repeat hemorrhage should occur, the situation is critical. As aneurysm at the peripheral region of the Moyamoya vessels and that of the anterior and posterior choroidal arteries have disappeared in many of the cases, it is considered that direct operation is not necessary for several months following hemorrhage. However, in cases where the aneurysm does not disappear for an extended period and in cases where the aneurysm enlarges<sup>7,23)</sup>, there is the possibility of repeat hemorrhage as reported in the literature and thus direct operation would have to be taken into consideration. It is considered that STA-MCA anastomosis and encephalomyo-synangiosis are procedures beneficial in preventing repeat hemorrhage by reducing the blood flow load on the Moyamoya vessels<sup>8,21,25,28)</sup>.

It is necessary to repeat angiography to confirm whether aneurysm has disappeared. In the case of high resolution CT, confirmation of whether the aneurysm has disappeared or not can be made by the use of contrast enhancement<sup>24)</sup>. In our case CT was conducted every 7-14 days and angiography was done at the time when the high density area of the aneurysm was reduced in size. By these procedures, disappearance of the aneurysm was confirmed. CT, being far less invasive to the patient than angiography, is regarded to be an effective procedure in following aneurysm. As described above, it is common for aneurysm to spontaneously disappear, but there is a possibility of repeat hemorrhage of the aneurysm with a large diameter which does not disappear even after a lapse of much time. Aneurysm of this nature requires followup observation. CT is an effective procedure for such followup.

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