IV. 7. Cerebral Glucose Metabolism Around the Nidus of Arteriovenous Malformations


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Introduction

Already many studies have been carried out to clarify the pathophysiology of arteriovenous malformations (AVMs), especially from the view points of cerebral blood flow\(^1\)\(^-\)\(^4\)\(^,\)\(^6\), however, many uncertainty still remain to be solved. Among them, only a few study has recently been appeared on the literature investigating the cerebral metabolism of the AVM patients\(^12\). In order to shed the light on how the nidus of AVMs affect the surrounding brain tissue, cerebral glucose metabolism of patients with AVMs were studied with positron emission tomography (PET).

Materials and Method

Patients

Ten patients with AVMs (five male and five female, ranging in age from 20 to 56 years old, with a mean age of 33.0 years old) were studied with PET using \(^{18}\)F- fluorodeoxyglucose (FDG). Initial symptoms of the patients were consciousness loss attack or convulsion in eight cases, and headache in two cases. All of the patients did not have any history of intracranial bleeding, and they were all neurologically intact at the time of PET study. The size of the nidus of AVMs on angiography were less than 3 cm in one, between 3 and 6 cm in six and more than 6 cm in three cases. In all patients AVMs were located on cerebral hemispheres.

Scanner and Procedure

PT-931 (CTI, Knoxville, Tennessee)\(^1\)\(^0\) was used, the spatial resolution of the images were 8 mm in FWHM and slice thickness was 7 mm. Following the intravenous
administration of 2 to 6 mCi of $^{18}$FDG, sequential scanning was performed for 40 to 60 minutes, and arterial blood sampling was made during this period. The PET images were reconstructed using a calculated attenuation correction, and regional cerebral glucose metabolism were calculated according to the autoradiographic method of Phelps et al. The results were compared with morphological images of CT and/or MRI.

Results

Cerebral glucose metabolism in the nidus of AVMs were clearly low comparing the surrounding brain, however, in eight cases glucose metabolism of the surrounding brain tissue did not show definite decrease (Fig. 1, 2). Only two cases disclosed more than 20% decrease of glucose metabolism in surrounding brain tissue of nidus comparing with the contralateral cerebral hemisphere, those were $6 \times 5 \times 4$ and $6 \times 4 \times 4$ cm in size of the nidus, and both of the AVMs had multiple feeding arteries from middle cerebral artery and posterior cerebral artery (Fig. 3, 4, 5). Nevertheless, these decreased glucose metabolism of brain tissue were localized only around nidus and extensive hemispheric decreased metabolism or the findings of transtentorial diachisis were not recognized.

In two cases, marked reduction of glucose metabolism was found in bilateral cerebral hemisphere, unaccompanied with cerebellar atrophy on CT and/or MRI (Fig. 6, 7)

Discussion

The clinical symptoms of cerebral AVMs can be classified into two categories; relating and not relating to hemorrhagic attacks. According to the large series of Yasargil, which included over 400 AVMs, about 80% of patients were proved to have the history of bleeding and the rest of 20% complained seizures and other symptoms such as headache without the history of intracranial bleeding. It has been postulated that the brain tissue around the nidus of AVMs is more or less ischemic due to the blood steal to the AVMs, which does not have the normal capillary network resulting in blood shunt from feeding artery to draining vein. And the longstanding ischemic insult brings about the histological changes in the surrounding brain tissue, which leads to seizure attacks or other symptoms except for hemorrhagic attacks. These hypothesis was partly supported by the intraoperative fluorescent angiography and local CBF measurement.

However, there has been only a few reports closely observing the cerebral metabolism in patients with AVMs. In our series of AVM patients, eight cases out of ten did not disclose definite reduction of glucose metabolism in the brain tissue surrounding the nidus. On the other hand, only two cases of large AVMs showed decreased metabolism in the brain tissue
surrounding the nidus. On the other hand, only two cases of large AVMs showed decreased metabolism in the surrounding brain. These results were in contrast to the recent report of Tyler et al.\textsuperscript{12} They studied 17 patients with PET and found wide spread metabolic impairment of glucose, both in ipsilateral regions remote from the lesion and in the contralateral hemispheres in patients with large AVMs. These divergent results were probably due to the different criteria for patient selection between the two studies. In their series of 17 patients, five cases showed the evidence of hemorrhagic history on CT scan, six cases had hemiparesis, two were dysphasia, one showed homonymous hemianopsia, one had organic brain syndrome, and only three cases showed no neurological deficit rather than headache. On the other hand, all the patients did not have the history of hemorrhagic attacks and were neurologically intact at the time of PET study in our series. Therefore, their findings of extensive metabolic impairment were likely due to the reflection of organic brain damage resulted from the intracranial bleeding rather than the direct influence by the AVM itself.

AVMs are believed to be congenital disease, arising during the course of vascular development in fetus.\textsuperscript{11} In the present study, the findings of transtentorial diachisis was not observed in any of the cases, which again support the fact that this lesion is congenital anomaly. Because in many of the acquired cerebral disease, such as brain tumor, intracerebral hemorrhage or cerebral infarction, transtentorial diachisis were frequently seen.

It is another interest that marked bilateral cerebellar hypometabolism without atrophy on CT and/or MRI, was observed in two cases. The average value of cerebellar glucose metabolism in our institute with normal volunteer was 75-90\% of cerebral cortex, however, it was about 50\% in the two cases. This is probably due to the effect of anticonvulsant drug. However, in our series, all of the patients received anticonvulsant, and why the other patients did not reveal cerebellar hypometabolism was not clear. Further studies are required to make the relationship clear between the effect of anticonvulsant and cerebellar hypometabolism.

In recent years, there has been great advancement in the surgical treatment for AVMs, mainly due to the sophisticated microsurgical technique and meticulous preoperative angiographical evaluation. Moreover, appearance of intravascular neurosurgical technique seems promising as an alternative treatment strategy. Nevertheless, the treatment for AVMs, either by surgically and/or intravascular technique, still remains one of the most difficult procedures in the neurosurgical field. The present study clearly demonstrated that metabolic impairment of brain tissue surrounding the AVM nidus was small in many of the cases except for larger AVM in size, when the patient dose not have the history of hemorrhagic attacks and are neurologically intact. These findings again stress the importance to establish the better treatment strategy trying to cure this disease with preserving the vital brain tissue.
References


Fig. 1.