

**Arteriovenous Malformations in the Basal Ganglia**

—Surgical Indications and Approaches—

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Abstract

We present 18 cases of arteriovenous malformation (AVM) in the basal ganglia. The results of seven cases of definitive surgery are reported in detail and are compared with those of 10 nonsurgical and one nondefinitive surgical cases. Among those available for follow-up, three of the seven nonsurgical and one nondefinitive surgical patients died of rebleeding. On the other hand, all seven patients who underwent definitive surgery are doing well, and their signs and symptoms were not aggravated by surgery. AVMs in the basal ganglia are classified into four types according to site: lateral, medial, mixed, and anteroinferior in relation to the internal capsule. The nidus was totally removed in two medial and four lateral cases. Eight of the 11 nonsurgical and nondefinitive surgical cases were of the mixed type and most had large nidi, and those that were totally removed were small or medium-sized. The surgical accessibility of AVMs in the basal ganglia and the current surgical approaches are discussed in terms of their locations and sizes.

Key words: arteriovenous malformation, basal ganglia

Introduction

The surgical accessibility of arteriovenous malformations (AVMs) in the basal ganglia is a subject of controversy. In the past, AVMs in this area were generally considered inoperable because of possible damage to critical structures. However, with advances in both diagnostic radiology and surgical techniques, cases of successful removal of AVMs in the basal ganglia are being reported with increasing frequency. From such reports it is clear that some AVMs in the basal ganglia can be safely removed. A newer potential treatment for AVMs, still highly experimental, is artificial embolization. In the near future, as therapeutic options expand, neurosurgeons will have to make informed decisions concerning the treatment of these AVMs.

In the hope of contributing to the establishment of treatment criteria, we assessed and compared the results of our surgically and conservatively treated cases of AVM in the basal ganglia. In particular, we evaluated surgical feasibility in terms of the location

**Patients and Methods**

We reviewed the records of 18 patients with AVM in the basal ganglia who had been diagnosed and treated at Kyushu University Hospital from 1964 to 1985. Our series included AVMs occupying the basal ganglia with or without involvement of the thalamus and excluded AVMs confined only to the thalamus. Five patients were females and 13 were males. Their average age at the time of onset was 26 years. The initial insult was hemorrhage in 14 cases, epilepsy in one, transient ischemic attack (TIA) in one, and motor weakness in two. Demographic and clinical data for these patients are listed in Tables 1 and 2.

The AVMs were classified into four types according to location (Fig. 1).

Type 1 (lateral): The nidus of the AVM is situated mainly in the putamen and insula. It is supplied by the lateral lenticulostriate arteries, including the insular branches and/or cortical branches of the middle cerebral artery. It usually drains into superficial

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cortical veins, such as the superficial Sylvian vein or the vein of Labbé.

Type II (medial): The nidus is situated mainly in the head of the caudate nucleus, with one portion facing the anterior horn of the lateral ventricle. The feeding arteries comprise mainly the medial group of lenticulostriate arteries, and the main drainage route is the internal cerebral vein through the caudate vein.

Type III (mixed): The nidus of the AVM is relatively large and involves the thalamus and/or internal capsule in addition to the basal ganglia. It is usually supplied by many arteries, including not only the lenticulostriate arteries and the insular and cortical branches of the middle cerebral artery but also the choroidal and perforating arteries. It sometimes drains into two different venous systems, i.e., the superficial cortical veins, including the Sylvian vein, and the deep cerebral veins, including the internal cerebral vein.

Type IV (anteroinferior): The nidus of the AVM, which is supplied by the recurrent artery of Heubner or the lenticulostriate artery, is located mainly in the anterior perforated substance, particularly anteroinferiorly to the anterior limb of the internal capsule and the putamen. The draining vein is the basal vein of Rosenthal through the deep middle cerebral vein.

In this study there were five AVMs of the lateral type, three of the medial type, nine of the mixed type, and one of the anteroinferior type.

We used Drake’s definition⁹ to characterize the sizes of the nidi. A small nidus is less than 2.5 cm in diameter on angiograms; a medium-sized nidus is between 2.5 cm and 5.0 cm; and a large nidus is over 5.0 cm in diameter. Our 18 cases contained six small, five medium-sized, and seven large nidi. All large nidi were of the mixed type.

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Table 1 Cases of definitive surgery for AVM in the basal ganglia

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Initial symptom</th>
<th>Size of nidus</th>
<th>Side</th>
<th>Type of AVM</th>
<th>Result of surgery</th>
<th>Follow-up Assessment</th>
<th>Length</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23</td>
<td>F</td>
<td>hemorrhage</td>
<td>small</td>
<td>rt.</td>
<td>lateral</td>
<td>improved</td>
<td>working (hemiparesis)</td>
<td>19 yr</td>
</tr>
<tr>
<td>2</td>
<td>19</td>
<td>M</td>
<td>hemorrhage</td>
<td>small</td>
<td>rt.</td>
<td>lateral</td>
<td>unchanged</td>
<td>working</td>
<td>13 yr</td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>M</td>
<td>hemorrhage</td>
<td>small</td>
<td>rt.</td>
<td>lateral</td>
<td>improved</td>
<td>working (hemianopsia)</td>
<td>3 yr</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>F</td>
<td>hemorrhage</td>
<td>medium</td>
<td>lt.</td>
<td>lateral</td>
<td>unchanged</td>
<td>working</td>
<td>3 yr</td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>M</td>
<td>hemorrhage</td>
<td>small</td>
<td>rt.</td>
<td>medial</td>
<td>unchanged</td>
<td>working</td>
<td>14 yr</td>
</tr>
<tr>
<td>6</td>
<td>61</td>
<td>M</td>
<td>hemorrhage</td>
<td>medium</td>
<td>lt.</td>
<td>medial</td>
<td>unchanged</td>
<td>working (dementia)</td>
<td>1 yr</td>
</tr>
<tr>
<td>7</td>
<td>27</td>
<td>F</td>
<td>hemorrhage</td>
<td>small</td>
<td>rt.</td>
<td>mixed</td>
<td>unchanged</td>
<td>working (hemiparesis)</td>
<td>3 mo</td>
</tr>
</tbody>
</table>

In Cases 1–6 the AVM was totally removed. In Case 7, the feeding artery was obliterated.

Table 2 Cases of nonsurgical and nondefinitive surgery for AVM in the basal ganglia

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Initial symptom</th>
<th>Side</th>
<th>Type of AVM</th>
<th>Follow-up Assessment</th>
<th>Length</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>6</td>
<td>M</td>
<td>motor weakness</td>
<td>lt.</td>
<td>mixed</td>
<td>unavailable</td>
<td>unavailable</td>
</tr>
<tr>
<td>9</td>
<td>10</td>
<td>M</td>
<td>hemorrhage</td>
<td>rt.</td>
<td>mixed</td>
<td>died</td>
<td>3 yr</td>
</tr>
<tr>
<td>10</td>
<td>9</td>
<td>M</td>
<td>epilepsy</td>
<td>bil.</td>
<td>mixed</td>
<td>died</td>
<td>2 yr</td>
</tr>
<tr>
<td>11</td>
<td>29</td>
<td>M</td>
<td>TIA</td>
<td>lt.</td>
<td>mixed</td>
<td>died</td>
<td>13 yr</td>
</tr>
<tr>
<td>12</td>
<td>22</td>
<td>M</td>
<td>hemorrhage</td>
<td>rt.</td>
<td>mixed</td>
<td>died</td>
<td>16 yr</td>
</tr>
<tr>
<td>13</td>
<td>11</td>
<td>F</td>
<td>hemorrhage</td>
<td>lt.</td>
<td>mixed</td>
<td>working (34 yr)</td>
<td>3 yr</td>
</tr>
<tr>
<td>14</td>
<td>25</td>
<td>M</td>
<td>hemorrhage</td>
<td>lt.</td>
<td>mixed</td>
<td>working (8 yr)</td>
<td>6 yr</td>
</tr>
<tr>
<td>15</td>
<td>29</td>
<td>M</td>
<td>hemorrhage</td>
<td>rt.</td>
<td>lateral</td>
<td>unavailable</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>27</td>
<td>M</td>
<td>motor weakness</td>
<td>rt.</td>
<td>anteroinferior</td>
<td>working (6 yr)</td>
<td>6 yr</td>
</tr>
<tr>
<td>17</td>
<td>21</td>
<td>F</td>
<td>hemorrhage</td>
<td>lt.</td>
<td>medial</td>
<td>unavailable</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>20</td>
<td>M</td>
<td>hemorrhage</td>
<td>rt.</td>
<td>medial</td>
<td>unavailable</td>
<td></td>
</tr>
</tbody>
</table>

Only VP shunt was performed in Case 12 (nondefinitive surgery).
Fig. 1 Classification of AVMs in the basal ganglia according to their locations, feeders, and drainers.

Results

I. Definitive surgical cases

Eight of the 18 patients underwent surgery (Tables 1 and 2). Six of the AVMs were totally removed, and in one case (Case 7) the feeding artery was occluded. Those seven cases were included in the definitive surgical group (Table 1). The remaining surgical case (Case 12), in which only ventriculoperitoneal (VP) shunt and external drainage were performed, was assigned to the nondefinitive surgical group (Table 2) and will be discussed later.

Total removal was attempted only when the AVM was lateral (four cases) or medial (two cases). The removed nidi were small in four and medium-sized in two cases. With the medial type, the nidus, which faced the lateral ventricles, was removed through the lateral ventricle, in one case via the transfrontal transcortical approach and in the other through the anterior interhemispheric transcallosal approach. In cases of the lateral type, total removal of the AVM and evacuation of the hematoma were accomplished via the trans-Sylvian approach.

Three of the six patients who underwent total re-  
moval of the AVM had presented with slight hemi-  
paresis, hemianopsia, or dementia preoperatively;  
none experienced worsening of these signs after sur-  
gery. All six are doing well, and four have returned  
to their jobs. One woman (Case 1) married and now  
has three children. In the remaining patient (Case  
6), who had six episodes of hemorrhage, urinary in-  
continence and memory deficit improved after VP  
shunt.

Two cases in which the nidus was totally removed  
are described here.

Case 6 (medial AVM): This 61-year-old male had  
suffered six intraventricular hemorrhages and was re-  
ferred to our institution because of gait disturbance,  
urinary incontinence, and memory disturbance. Pre-  
contrast computed tomographic (CT) scans demon-  
strated dilated ventricles (Fig. 2A). The nidus of an  
AVM with a large draining vein in the head of the  
left caudate nucleus was enhanced after intravenous  
injection of contrast medium (Fig. 2B). Angiograms  
disclosed that the AVM was supplied by the medial  
and lateral groups of the lenticulostriate arteries and  
drained into the internal cerebral vein through the  
caudate vein (Fig. 3). The nidus was totally removed  
through the anterior interhemispheric transcallosal  
approach, and a VP shunt was emplaced. The  
urinary incontinence and memory disturbance im-  
proved. The AVM was not present on postoperative  
angiograms (Fig. 4).

Case 4 (lateral AVM): This 44-year-old woman

Fig. 2 Case 6. A: A precontrast CT scan shows  
symmetrically dilated lateral and third ventri-  
cles. B: In the postcontrast study a small iso-  
density mass in the head of the caudate nu-  
cleus and the left caudate vein are enhanced.  
The choroid plexuses in the trigones of the  
lateral ventricles are also enhanced.
suddenly developed right hemiparesis, aphasia, and drowsiness. Precontrast CT scans showed a midline shift and a large hematoma in the lateral part of the left basal ganglia (Fig. 5). The hematoma was evacuated through the trans-Sylvian approach. As a large, red vein was found during this emergency procedure, angiography was performed postoperatively. The left carotid angiogram revealed an AVM in the posterior portion of the left putamen and insula (Fig. 6). It was fed by the insular branches of the middle cerebral artery and drained into the vein of Labbé through a vein in the Sylvian fissure. An aneurysm was discovered at the junction of the left internal carotid and posterior communicating arteries. The AVM was totally removed through the hematoma cavity and the aneurysmal neck was clipped via a left frontotemporoparietal craniotomy. Postoperative angiograms showed no AVM (Fig. 7). She has no deficits except for mild difficulty in auditory comprehension of complicated numbers, and is working in an office.

In one case a feeder was obliterated because angiography indicated that it was the sole supplier.

Case 7 (mixed AVM): This 27-year-old female dentist suddenly developed left hemiplegia, hemihypesthesia, and drowsiness. Precontrast CT scans revealed a large hematoma in the right basal ganglia, with intraventricular hemorrhage. Right carotid angiography revealed a small AVM in the right basal ganglia, which appeared to be supplied by only one branch of the lateral group of lenticulostriate arteries (Fig. 8A). It drained into the internal cerebral vein through the right thalamostriate vein. Two months after onset of her symptoms she was transferred to our institution. Neurological examination revealed left hemiparesis and hemihypesthesia. She could walk only with the support of a full leg brace. Precontrast CT scans obtained 2 1/2 months after onset demonstrated a small, low-density area at the site of the previous hematoma (Fig. 9A), in which the small nidus of an AVM was enhanced (Fig. 9B). It involved the body of the caudate nucleus and the posterior limb of the internal capsule. Because angiography indicated that the lesion was supplied by a single artery, we performed a frontotemporal craniotomy and obliterated this feeding artery by coagulation at the anterior perforated substance. Her neurological status remained un-

Fig. 3 Case 6. Preoperative left internal carotid angiograms, anteroposterior (left) and lateral (right) views. The AVM in the head of the caudate nucleus is supplied by the medial and lateral groups of the lenticulostriate arteries and drains into the caudate vein.

Fig. 4 Case 6. Postoperative left internal carotid angiograms, anteroposterior (left) and lateral (right) views. The AVM was totally removed and is not demonstrated.

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Fig. 5 Case 4. The precontrast study shows a hematoma in the lateral portion of the basal ganglia. The midline has shifted from left to right.
changed following this procedure. Postoperative angiograms showed that the feeding artery that had been visualized preoperatively had disappeared and that the nidus had been reduced. However, they also revealed other small feeders from the recurrent artery of Heubner, the insular branch of the middle cerebral artery, and the anterior choroidal artery, none of which had been seen before (Fig. 8B).

II. Nonsurgical and nondefinitive surgical cases

This group consists of 10 nonsurgical cases and one nondefinitive surgical case (Case 12) (Table 2). Radical therapy was not attempted in these patients for the following reasons: i) the symptoms were not of hemorrhage but of epilepsy or TIA; ii) a relatively large nidus involved the internal capsule and/or the thalamus; iii) there were several different feeding arteries, including the choroidal artery and/or the recurrent artery of Heubner; and/or iv) there were two different venous drainage routes, e.g., the deep cerebral venous and superficial cortical venous systems. In eight of the 11 cases the AVMs were of the mixed type. Seven of these had large nidi and the eighth had a small nidus. None of the mixed type of AVM was approached directly.
An example of the mixed type is shown in Fig. 10 (Case 8). The angiograms disclose a large AVM supplied by the medial and lateral groups of the left lenticulostriate arteries and insular branches of the left middle cerebral artery and located in the left basal ganglia and thalamus.

We were able to follow eight of the 11 patients in this group. Four died of rebleeding at 2, 3, 13, and 16 years after the onset of symptoms. In Case 18, the medium-sized AVM in the head of the caudate nucleus was not operated on because at that time the surgical microscope had not yet been introduced.

Discussion

Several authors have discussed the risks of rebleeding and mortality in the patient with AVM. Graf et al. reported 17 deaths among 134 patients (12.7%) with bleeding AVMs. According to Svien and McRae, 11 deaths among 68 patients (16%) who did not undergo definitive surgery were attributed to the AVM. They calculate the mortality in patients with cerebral AVM to be between 10 and 20%. Only one report has mentioned the natural history of AVMs in the basal ganglia.

In our experience, four of the eight nonsurgical and nondefinitive surgical patients (50%) with AVMs in the basal ganglia whom we were able to follow died of hemorrhage. All four AVMs were of the mixed type. Saito reported that nine of 14 nonsurgical patients with AVMs in the basal ganglia had rebleeding, and that six (43%) died of hemorrhage. When definitive surgery is not performed, the mortality from AVMs in the basal ganglia appears to be higher than that for cerebral AVMs as a whole. Pellettiere et al. stated that deeply situated AVMs seem to have a somewhat greater tendency to bleed than the superficial ones. Among their cases, six of the eight patients who died had deeply situated AVMs. On the other hand, all of our patients who underwent definitive surgical treatment of the nidus are doing well. We conclude that, in terms of survival, direct surgery seems to be superior to nonsurgical treatment.

The subjects in this study all had AVMs in the basal ganglia with or without involvement of the thalamus. Although AVMs in the thalamus are sometimes included with those in the basal ganglia, we excluded AVMs confined solely to the thalamus. Basal ganglia AVMs are classified into four types. The medial type have also been referred to as a lateral ventricle or paraventricular AVMs. AVMs of the lateral type are situated very near the site of so-called Sylvian fissure AVMs.

In the past the great majority of AVMs fed by the lenticulostriate arteries were considered inoperable. In six of our patients, however, the nidi were totally removed without postoperative neurological deficits. It appears that the feasibility of surgery depends in large part on the location of the nidus, as does the surgical approach.

A small or medium-sized AVM of the lateral type can be totally and safely removed through the trans-Sylvian approach even if the nidus is located in the dominant hemisphere, as was demonstrated in our Case 4. However, as Sang has warned, for very large AVMs the trans-Sylvian approach does not allow adequate control of intraventricular hemorrhage during surgery. Huge AVMs of the basal ganglia, even those that are situated laterally, are generally considered inoperable.

In the medial type, when a small or medium-sized nidus is located near the lateral ventricle, it can be removed through the transventricular approach, without postoperative deficits. There have been several recent reports of successful removal of AVMs of the head of the caudate nucleus. Thus, AVMs of the medial type are now considered operable. There are two possible routes to the lateral ventricle, the transcortical and the interhemispheric transcallosal. The transcortical approach is appropriate when there is a hematoma in the frontal lobe. Rutka and Tucker reported such a case and emphasized the easy, safe removal of the AVM through the pre-existing hematoma cavity.

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the absence of a hematoma, the anterior interhemispheric transcallosal approach is advisable because it does not necessitate a cortical incision. Incision of the anterior portion of the corpus callosum reportedly causes no neurological deficits. 8,9,15,25 Waga et al.28 encountered complications after performing a frontal cortical incision to reach an AVM in the caudate head of the dominant hemisphere, and they also recommend an anterior transcallosal approach.

Concerning the mixed type of AVM, in which the nidus involves the thalamus and/or internal capsule, only a few cases of radical treatment have been reported.4,11,20,21,24,27 These AVMs usually contain large nidi, making total removal without deficits difficult by a direct approach. Successful surgery for mixed AVMs and those located in the thalamus has been reported by Drake,4) Ralston and Papa-theodorou,20) Ribaric,21) Sang,24) Viale et al.,27) and Wilson et al.29) Viale et al.27) removed a right-side thalamus AVM of the mixed type by two-staged operations involving perional and transventricular approaches. Following the second operation the patient developed a left spastic hemiparesis, but was able to return to work after rehabilitation. In two instances, during exploration of hematomas in the thalamus, Wilson et al.29) discovered and removed cryptic AVMs. Both of these patients’ symptoms temporarily worsened postoperatively but improved later. On the other hand, Drake4) reported four cases of surgically removed thalamic AVMs, one of which was large and situated in the upper dominant thalamus. The nidus was removed through the splenium into the trigone of the lateral ventricle, and the postoperative hemisensory deficit and hemiparesis resolved almost completely. Drake4) suggested, as did Filatov et al.,3) that small AVMs lying in the anterior or posterosuperior thalamus might be excised safely without major deficits. We have not approached AVMs of the mixed type directly. However, our findings concerning AVMs of the mixed type indicate that, in young patients in whom the nidus is small and hemorrhaging is frequent, removal is the best treatment option. In such patients, mortality due to bleeding is very high. We followed seven of nine patients with mixed AVMs; four (57%) died of rebleeding. However, we feel that surgery is not indicated for elderly patients or those in whom hemorrhage has not occurred. Sang24) reported success with aggressive, multistage removal of huge AVMs of the mixed type and the use of barbiturate coma for cerebral protection. Ribaric21) described a case of a mixed AVM in which one feeder was clipped and the AVM disappeared according to postoperative angiograms. In that case the AVM was supplied by a single artery. In our Case 7, obliteration of the feeder at the anterior perforated substance failed to eliminate the AVM; postoperative angiograms revealed the presence of other small feeders.

AVMs of the anteroinferior type are supplied mainly by the recurrent artery of Heubner and therefore are usually not considered for aggressive surgery. Nonetheless, Garrido and Stein7) totally removed an AVM in the region of the right anterior perforated substance and basal ganglia. Postoperatively, the patient had only transient hemiparesis, which resolved completely after several months. These authors concluded that some AVMs in this critical brain region can be removed, provided that major arteries are not directly involved. Andreussi et al.11) and Viale et al.27) have reported similar cases. Viale et al.27) noted that removal of some proximal striate AVMs is relatively safe. However, Andreussi et al.11) stressed that intraoperative angiographic monitoring is necessary in most cases to ensure the identification of the feeding vessels.

According to our own surgical results and those reported in the literature, lateral and medial AVMs in the basal ganglia are candidates for total removal. Some anteroinferior and mixed AVMs are removable — specifically, those that have small nidi associated with hematoma cavities and do not involve major arteries. Since conservative treatments result in a very high mortality rate, some mixed AVMs accompanied by recurrent hemorrhage should perhaps be treated aggressively, by direct, staged operation, feeder ligation, or artificial embolization. New techniques for artificial embolization are being developed and applied even to deep-seated AVMs. In the near future we will again have to evaluate and compare the various treatment approaches to AVMs in the basal ganglia.

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