Arteriovenous malformations in the posterior fossa

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We have operated on 12 of 14 cases of arteriovenous malformation (AVM) in the posterior fossa since 1968, with one death. The lesions were in the cerebellum in 10 cases (three anteromedial, one central, three lateral, and three posteromedial), and in the cerebellopontine angle in two; in two cases the lesions were directly related to the brain stem. The AVM's in the anterior part of the cerebellum were operated on through a transtentorial occipital approach.

KEY WORDS • arteriovenous malformation • subarachnoid hemorrhage • posterior fossa • cerebellum • cerebellopontine angle • transtentorial approach

A total of 84 cases of arteriovenous malformations (AVM's) were treated at Kansai Medical University Hospital and Tenri Hospital in the 10 years from April, 1966, to March, 1976. There were 14 cases of AVM's in the posterior fossa (16.7%).

Clinical Features

A summary of the clinical features of each case is given in Table 1. The patients' ages ranged from 24 to 53 years (average 36). There were 10 men and four women.

Location and Size of Lesions

The location of the lesions is shown in Fig. 1. In three cases (Cases 1, 6, and 12) the AVM's were in the anteromedial part of the cerebellum, in one (Case 2, whose AVM was huge) in the center of the cerebellum, in three (Cases 8, 9, and 10) in the posteromedial part of the cerebellum, in three (Cases 4, 5, and 11) in the lateral part, and in two (Cases 3 and 4) in the cerebellopontine angle; in two (Cases 7 and 13) lesions were suspected to be directly related to the brain stem. There was no markedly predominant location for AVM's in the posterior fossa. The size of the lesions was variable: the largest (Case 2) was $5 \times 5 \times 2.5$ cm and the others varied from finger tip to walnut size.

Symptoms and Signs

The initial evaluations were not always done by a neurosurgical specialist; some patients were treated by general physicians for a considerable period of time before transfer to our hospital and, therefore, neurological descriptions were not always complete.

Twelve of 14 cases presented with subarachnoid hemorrhage, and three of them had multiple SAH's. Cases 1 and 2, which were our initial experiences, had six and four attacks of SAH, respectively, and this prob-
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Fig. 1. Location of the arteriovenous malformations of the posterior fossa in 14 cases.

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**TABLE 1**

*Summary of clinical features in 14 cases of posterior fossa AVM*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>Symptoms</th>
<th>Signs</th>
<th>Feeding, Draining Vessels</th>
<th>Location of AVM</th>
<th>Patient’s Position; Approach</th>
<th>Surgical Procedure</th>
<th>Operative Results†</th>
<th>Follow-Up Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36, M</td>
<td>6 SAH's, choked disc</td>
<td>rt SCA, vein of Galen, transv. sinus</td>
<td>upper vermis</td>
<td>prone, occipital, transtentorial</td>
<td>clipping of feeders; total removal</td>
<td>unchanged; concentric contraction of visual field</td>
<td>8 yrs; full work as masseur</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>33, M</td>
<td>4 SAH's, vertigo, diplopia</td>
<td>SCA's, AICA's, rt PICA, vein of Galen</td>
<td>vermis, 4th ventricle</td>
<td>sitting; occipital, transtentorial</td>
<td>partial removal</td>
<td>died 12 wks postop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>32, F</td>
<td>SAH, choked disc, hydroceph. on carotid angiography</td>
<td>rt AICA, rt PICA, straight sinus</td>
<td>rt CP angle, rt cerebral hemisph.</td>
<td>sitting; rt sub-occipital</td>
<td>total removal in two steps; VP shunt</td>
<td>fair; rt VI, VII, VIII nerve weakness; rt ataxia</td>
<td>5 yrs; slight rt VII, VIII nerve weakness; full housework</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>53, M</td>
<td>SAH, dysarthria</td>
<td>lt SCA, lt petrosal vein, lt inferior vein of vermis</td>
<td>lt cerebell. hemisph.</td>
<td>sitting; lt occipital, transtentorial</td>
<td>total removal</td>
<td>excellent</td>
<td>4 yrs; full work in previous occupation</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>48, F</td>
<td>2 SAH's, choked disc, dysarthria, rt III, V, VII, VIII ataxia</td>
<td>rt SCA, rt petrosal vein, rt lat. mesencephalic vein</td>
<td>rt cerebell. hemisph.</td>
<td>sitting; rt occipital, transtentorial</td>
<td>total removal</td>
<td>fair; rt III, VII ataxia; hyperesthesia</td>
<td>4 yrs; full housework</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>52, M</td>
<td>rt hemiparesis, SAH, hydroceph. on carotid angiography</td>
<td>lt SCA, vein of Galen; lt MCA occlusion</td>
<td>lt side of upper vermis</td>
<td>sitting; rt occipital, transtentorial</td>
<td>total removal; VP shunt</td>
<td>no additional deficit postop; rt hemiparesis due to MCA occlusion persisted</td>
<td>4 yrs; rt hemiparesis; unable to work</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>29, M</td>
<td>SAH, unconscious 17 days, diplopia, ataxia, rt VI nystagmus</td>
<td>SCA's, petrosal &amp; transv. sinus</td>
<td>upper vermis &amp; midbrain</td>
<td>—</td>
<td>none</td>
<td>poor</td>
<td>3 yrs; able to walk with support</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>36, M</td>
<td>choked disc, nystagmus, vertigo, ataxia, rt anisocoria, no SAH</td>
<td>rt PICA, straight &amp; occipital sinuses</td>
<td>lower end of vermis</td>
<td>sitting; suboccipital</td>
<td>VA shunt; total removal</td>
<td>good; ataxic gait</td>
<td>7 yrs; full work in previous occupation</td>
<td></td>
</tr>
</tbody>
</table>

*AVM = arteriovenous malformation; SCA = superior cerebellar artery; AICA = anterior inferior cerebellar artery; PICA = posterior inferior cerebellar artery; MCA = middle cerebral artery; CP = cerebellopontine; VA = ventriculoatrial; VP = ventriculoperitoneal.

†Operative results include postoperative vertebral angiography findings.
TABLE 1 (Continued)*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age</th>
<th>Symptoms, Signs</th>
<th>Feeding, Draining Vessels</th>
<th>Location of AVM</th>
<th>Patient’s Position; Approach</th>
<th>Surgical Procedure</th>
<th>Operative Results†</th>
<th>Follow-Up Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>M</td>
<td>43</td>
<td>choked disc, diplopia, rt V, VIII, XII, hydroceph., on carotid angiography, no SAH</td>
<td>rt AICA, rt PICA, rt inf. vein of vermis, straight sinus</td>
<td>rt tonsil</td>
<td>sitting; suboccipital</td>
<td>VA shunt; total removal</td>
<td>excellent</td>
<td>6 yrs; full work in previous occupation</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>24</td>
<td>SAH’s</td>
<td>rt PICA, straight sinus</td>
<td>rt cerebell, hemisph., paramedian dorsal surface</td>
<td>sitting; suboccipital</td>
<td>subtotal, then total removal</td>
<td>excellent</td>
<td>5 yrs; full housework</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>28</td>
<td>SAH, lt ataxia, diplopia</td>
<td>lt SCA, lt PICA, lt transv. sinus, straight sinus</td>
<td>lt lat. surface of cerebell.</td>
<td>sitting; suboccipital</td>
<td>subtotal removal</td>
<td>excellent; small remnant of AVM</td>
<td>3 yrs; full work as nurse</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>26</td>
<td>SAH, vertigo, dysarthria, diplopia, rt ataxia</td>
<td>rt SCA, vein of Galen, transv. sinus</td>
<td>upper vermis</td>
<td>prone; rt occipital, transtentorial</td>
<td>total removal</td>
<td>lt homonymous hemianopsia; good</td>
<td>3 yrs; full work as shopman</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>28</td>
<td>SAH, lt hemiplegia, lt hypotension, dysarthria</td>
<td>rt AICA, rt SCA, petrosal vein, straight sinus</td>
<td>pons rt side (?)</td>
<td>—</td>
<td>none</td>
<td>unchanged; poor</td>
<td>1 yr; no occupation</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>37</td>
<td>SAH</td>
<td>rt AICA, rt SCA, vein of Galen</td>
<td>rt CP angle, rt surface of pons</td>
<td>sitting; suboccipital</td>
<td>total removal; rt VIII severed</td>
<td>excellent; except for deafness on rt</td>
<td>1 yr; full work as shopman</td>
</tr>
</tbody>
</table>

*AVM = arteriovenous malformation; SCA = superior cerebellar artery; AICA = anterior inferior cerebellar artery; PICA = posterior inferior cerebellar artery; MCA = middle cerebral artery; CP = cerebellopontine; VA = ventriculoatrial; VP = ventriculoperitoneal.
†Operative results include postoperative vertebral angiography findings.

ably was due to our delay in recommending surgery, because of our limited experience with cerebellar AVM’s at the time. Only two cases (Cases 8 and 9) presented with evidence of progressive increase in intracranial pressure, simulating cerebellar tumors.

Some neurological signs observed immediately after SAH suggested the source of bleeding was in the posterior fossa. Four cases (Cases 1, 3, 5, 6) had evidence of intracranial hypertension concomitant with dilated ventricles on carotid angiography or papilledema. Persisting vertigo was present in two cases (Cases 2 and 12), dysarthria including slurred speech in four (Cases 4, 5, 12, 13), and diplopia in four (Cases 2, 7, 11, 12). Nystagmus was observed in one patient (Case 7), and ataxic hand movement in four (Cases 5, 7, 11, 12). Multiple cranial nerve signs were present in one case (Case 5, in which the AVM was situated in the right cerebellar hemisphere) and a long-tract sign in one
(Case 13, pontine lesion). Only two cases (Cases 10 and 14) had uncomplicated SAH without any signs suggesting the source of bleeding.

The other two cases without SAH manifested progressive increase in intracranial pressure (Cases 8 and 9); they also had vertigo, nystagmus, diplopia, anisocoria, multiple cranial nerve signs, choked discs, and evidence of the dilated ventricles on the carotid angiogram, but neither had any signs suggestive of AVM.

**Feeding and Draining Vessels**

Main arterial feeders of the lesions were variable. The AVM's located in the anterior part of the cerebellum were fed by unilateral or bilateral superior cerebellar arteries (SCA's) depending on the distance from the midline (Cases 1, 4, 5, 6, 7, 12). When the lesion was large and extended more caudally (for instance, in Case 2), the anterior inferior cerebellar artery (AICA), or the posterior inferior cerebellar artery (PICA), or both participated in its blood supply. Cerebellopontine angle lesions (Cases 3 and 14) and a lesion on the lateral part of the pons (Case 13) were mainly fed from the ipsilateral AICA and supplemented by the PICA or SCA. Lesions on the dorsomedial aspect of the cerebellum (Cases 8, 9, and 10) were supplied by the PICA; if the lesion extended more rostrally, the AICA also participated in feeding. The AVM situated on the lateral margin of the cerebellar hemisphere (Case 11) was supplied by the PICA and a long feeder from the SCA.

Venous drainage was more variable. The AVM's in the median part of the cerebellum including the vermis usually drained into the vein of Galen or the straight sinus. In some lesions of the upper vermis (Cases 1 and 12), however, venous drainage split into two routes, one via the vein of Galen and the other via the transverse sinus. It seems natural that AVM's located more laterally usually drain into the petrous vein and the transverse sinus (Cases 4, 5, 11, 13), but it was unexpected that a rostral lesion of the vermis, probably in contact with the midbrain, had draining routes to the petrosal and transverse sinuses (Case 7). Also, a cerebellopontine-angle lesion drained into the straight sinus in one case (Case 10) and into the vein of Galen in the other (Case 14).

**Surgical Approach**

The surgical approach we used depended upon the location of the lesions. One group included AVM's on the anterior aspect of the cerebellum, and the other group, those in other areas. The first group included rostral lesions of the vermis, those of the anteromedial part of the hemisphere, and a midbrain lesion. The second group consisted of AVM's of the dorsolateral part of the cerebellum, cerebellopontine angle, pons, and medulla. These lesions are easily accessible through the usual suboccipital craniectomy.

We prefer the transtentorial approach through an occipital supratentorial opening for lesions in the anterior aspect. The tentorium cerebelli is incised in an L-shape. If the lesion is on the right side, the long arm of the L is near and parallel to the straight sinus. If the lesion is on the left, the L-shape is reversed. The tentorial flap is turned laterally and the occipital lobe is elevated at a point about 5 cm from the occipital tip. With the incised tentorial edge retracted and elevated with stay sutures, there is easy access to both sides of the anteromedial part of the cerebellum. If extension of the medial lesion to the other side is not large, it can be approached from the side where the mass is larger. If more space is needed, the cerebral tissue may be partly removed in semilunar fashion at the edge under the retractor without affecting the visual fields. Evacuation of ventricular fluid does not always provide adequate surgical exposure if internal hydrocephalus does not exist.

Early in this series we operated with the patient in the sitting position. We opened the suboccipital region and incised the dura in the lower part of the posterior fossa to drain blood accumulated during surgery; external decompression of the posterior fossa was performed by dural patch graft after removal of the lesion. However, we have found that cerebellar edema after removal of an AVM (unlike extirpation of a cerebellar tumor) is not usually excessive; blood from the lesion can be prevented from flowing into the lower space by sealing with cotton sheets. Recently we have preferred to have the patient in the
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prone position and to perform unilateral occipital craniotomy without opening the suboccipital space. This approach was used in six cases (Cases 1, 2, 4, 5, 6, 12) in which the lesion lay in the anterior part of the cerebellum.

Arteriovenous malformations in the other sites in the posterior fossa were operated on through conventional suboccipital craniotomy. Removal of the lesion on the dorsal or lateral surface of the cerebellum posed no technical problems (Cases 8, 9, 10, 11); however, for those in the cerebellopontine angle there were some difficulties in separating the tough abnormal vessels from fragile cranial nerve roots (Cases 3 and 14). No attempt at surgery was made in two cases in which lesions were suspected to be in or adherent to the brain stem from findings of vertebral angiograms and clinical pictures (Cases 7 and 13). These patients are now being considered for surgery since Drake's recent report suggests that they have a good chance of recovery. The grade of ventricular dilatation was estimated in every case by carotid angiography, which was carried out even if the AVM was bound by the first vertebral angiography. Operation was performed under the operative microscope, and disappearance of the lesion or existence of its remnants was confirmed with postoperative vertebral angiography.

Discussion

There are not many reports concerning operative treatment and follow-up results of patients with posterior fossa AVM's. Chou, et al.,1 Drake,2 and Green and Vaughan suggested several important points in the clinical aspect of AVM's in the posterior fossa. We have seen 14 AVM's in the posterior fossa since 1968, 12 of which were operated on.

Since most of our cases were transferred from other hospitals, the initial signs following SAH were not checked by a specialist and no systematic analysis of the initial signs could be made. Nevertheless, as with hypertensive intracerebellar hemorrhage, prolonged vertigo with vomiting following an attack of SAH suggests the source of hemorrhage to be in the posterior fossa. Diplopia, dysarthria, and marked nystagmus after SAH are also of diagnostic value. Motor ataxia, if tested, may also be an important sign. However, location of the AVM within the posterior fossa cannot be determined from initial neurological signs. Choked discs were observed in five cases and hydrocephalus was found by carotid angiography in three of 14 cases. Four patients underwent a shunting operation for hydrocephalus. Two (Cases 1 and 5) who had choked discs did not need shunts, probably because of the reestablishment of cerebrospinal fluid flow following the total removal of the lesions.

From the point of view of the surgical approach, the AVM's could be divided into two groups. In one group the AVM's lay on the anterior aspect of the cerebellum and could not be seen from behind, and in the other they lay on the dorsal surface of the cerebellum or at the cerebellopontine angle and could be reached by a suboccipital route. The problem is the choice of approach to the lesion in the anterior cerebellar area. Drake2 reached the lateral wall of the brain stem through an opening of the tentorium under the posterior portion of the temporal lobe. We reached the lesion through a more dorsomedial opening elevating the occipital lobe 5 cm lateral to the occipital pole, because the target was the rostral vermis of the anteromedial part of the cerebellar hemisphere.

Another approach to the lesion is between the tentorium and upper surface of the cerebellum from behind. By this approach, a lower edge of the transverse sinus will restrict visibility, but if a medial part of the transverse sinus at the junction to the confluence is cut and the tentorium and occipital lobe elevated, there is more space. In this approach, bridging veins from the occipital lobe to the tentorium are preserved and venous drainage to the ipsilateral transverse sinus remains. This route is reasonable only when the contralateral transverse sinus is sufficiently developed. In Case 1, reopening the occipital craniotomy resulted in irregular, concentric narrowing of the visual fields secondary to separation of adhesions between the occipital cortex and dura. A one-stage operation is advisable, and if a remnant of the lesion is found on the postoperative angiogram, the surgical approach must be altered.

In our experience, cerebellar edema after removal of an AVM is not as marked as that noted after removal of a malignant tumor from the cerebellum. However, edema of the cerebrum, which has been elevated by a
retractor, is more troublesome, and in severe cases, external decompression or occipital lobectomy is inevitable.

Recovery from ataxia and dysarthria after surgical removal of the lesion was almost complete within several months or a year in every case. It is our impression that this recovery is better with AVM's than with tumors. Only in unoperated cases of AVM's in or near the brain stem was recovery from neurological deficits limited; and these patients could not return to normal life. Drake\(^2\) mentioned that there was good cleavage between the mass of the AVM and the pial membrane of the brain stem in the majority of cases, and that recovery from neurological deficits after surgery was also surprisingly good. Operative treatment may be beneficial for these cases, and we have it under consideration. One patient with an AVM in the rostral vermis with concomitant left hemiplegia due to occlusion of the middle cerebral artery could not return to his previous occupation because of hemiplegia. In this case also, removal of the cerebellar AVM did not relieve the neurological deficit.

**Conclusions**

Twelve of 14 cases with AVM's in the posterior fossa have been operated on since 1968 with one death. Surgical risk with these lesions is not necessarily higher than that with cerebral AVM's, because postoperative cerebellar edema is not so severe and recovery of cerebellar dysfunction after surgery is good. Although there are few reports of treatment of posterior fossa AVM's up to now, a more courageous decision for removal is recommended.

**References**


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