Gamma Knife surgery for basal ganglia and thalamic arteriovenous malformations

Clinical article

CHING-HSIAO CHENG, M.D.,1,2 R. WEBSTER CROWLEY, M.D.,1 CHUN-PO YEN, M.D.,1 DAVID SCHLESINGER, PH.D.,1 MARK E. SHAFFREY, M.D.,1 and JASON P. SHEEHAN, M.D., PH.D.1

1Department of Neurological Surgery, University of Virginia Health System, Charlottesville, Virginia; and 2Department of Neurosurgery, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, Taiwan

Object. Gamma Knife surgery (GKS) has emerged as the treatment of choice for small- to medium-sized cerebral arteriovenous malformations (AVMs) in deep locations. The present study aims to investigate the outcomes of GKS for AVMs in the basal ganglia and thalamus.

Methods. Between 1989 and 2007, 85 patients with AVMs in the basal ganglia and 97 in the thalamus underwent GKS and were followed up for more than 2 years. The nidus volumes ranged from 0.1 to 29.4 cm³ (mean 3.4 cm³). The mean margin dose at the initial GKS was 21.3 Gy (range 10–28 Gy). Thirty-six patients underwent repeat GKS for residual AVMs at a median 4 years after initial GKS. The mean margin dose at repeat GKS was 21.1 Gy (range 7.5–27 Gy).

Results. Following a single GKS, total obliteration of the nidus was confirmed on angiograms in 91 patients (50%). In 12 patients (6.6%) a subtotal obliteration was achieved. No flow voids were observed on MR imaging in 14 patients (7.7%). Following single or repeat GKS, total obliteration was angiographically confirmed in 106 patients (58.2%) and subtotal obliteration in 8 patients (4.4%). No flow voids on MR imaging were observed in 18 patients (9.9%). The overall obliteration rates following one or multiple GKSs based on MR imaging or angiography was 68%. A small nidus volume, high margin dose, low number of isocenters, and no history of embolization were significantly associated with an increased rate of obliteration. Twenty-one patients experienced 25 episodes of hemorrhage in 850 risk-years following GKS, yielding an annual hemorrhage rate of 2.9%. Four patients died in this series: 2 due to complications of hemorrhage and 2 due to unrelated diseases. Permanent neurological deficits caused by radiation were noted in 9 patients (4.9%).

Conclusions. Gamma Knife surgery offers a reasonable chance of obliterating basal ganglia and thalamic AVMs and does so with a low risk of complications. It is an optimal treatment option in patients for whom the anticipated risk of microsurgery is too high.


Key Words • arteriovenous malformation • basal ganglia • complication • Gamma Knife • stereotactic radiosurgery • thalamus • vascular disorders

Basal ganglia and thalamic AVMs represent a special group of cerebral vascular anomalies. Their natural history tends to be more aggressive, with an annual hemorrhage risk of approximately 10%1,11,28 compared with the often quoted 2%–4% among AVMs in general.4,22 Additionally, due to the critical neuronal pathways and nuclei in these locations, the morbidity and mortality associated with hemorrhage, steal phenomenon, or mass effect of these AVMs are significant.

Surgery has been the mainstay of treatment for basal ganglia and thalamic AVMs in the preradiosurgery era. A total extirpation of the nidus eliminates the risk of hemorrhage immediately, but the enthusiasm associated with surgery is tempered by the high incidence of associated neurological deficits. Embolization is rarely curative due to the difficulty of accessing the small feeding perforating arteries and a high risk of complications. Therefore, embolization is often used as an adjunct for subsequent definitive treatment. Radiosurgery has emerged as the treatment of choice for small- to medium-sized, deep-seated AVMs. Unlike microsurgical or endovascular procedures, the success of radiosurgery is less dependent on the ability to manage the deep location of the nidus and small feeding arteries or deep draining veins. In the present series, we review a large cohort of patients with basal ganglia/thalamic AVMs treated with GKS and report the obliteration and complication rates. The results were compared with a group of patients who had supratentorial, superficially located AVMs.
Methods

Patient Population

Between 1989 and 2007, 92 patients with basal ganglia AVMs and 103 with thalamic AVMs underwent radiosurgery at the Lars Leksell Center for Gamma Surgery in Charlottesville. Seven patients with basal ganglia and 6 with thalamic AVMs for whom follow-up was shorter than 2 years post-GKS were excluded, leaving 85 patients with basal ganglion and 97 patients with thalamic AVMs for analysis in this study. During the same period, 615 patients with supratentorial, superficially located AVMs were treated with GKS at our center and had a follow-up exceeding 2 years. These patients served as a comparison group.

In the basal ganglia/thalamic AVM group, there were 88 males and 94 females whose mean age was 28.4 years (range 5–80 years). Forty-seven patients were younger than 18 years of age at the time of GKS. The presenting symptoms leading to the diagnosis of AVM were hemorrhage in 139 patients (76.4%), seizure in 11 (6%), headache in 13 (7.1%), visual field defects in 2 (1.1%), long tract signs in 12 (6.6%), change in consciousness in 1 (0.5%), and hydrocephalus in 1 (0.5%). Three patients (1.6%) were asymptomatic, and their AVMs were an incidental finding. Incomplete resection was carried out in 19 patients before GKS. Thirty-eight patients underwent embolization prior to GKS. In 2 patients fractionated radiotherapy was performed, and in another 2, previous proton-beam radiotherapy was performed. Comparisons between patients with basal ganglia and thalamic AVMs and those with superficial AVMs are detailed in Table 1. Among the intergroup differences were a younger mean age at GKS in the basal ganglia/thalamic group (mean 28.4 vs 35.1 years in the group with superficial AVMs) and a greater number of the basal ganglia/thalamic AVM patients presenting with a hemorrhage (76.4% vs 40.8%).

Arteriovenous Malformation Characteristics

In patients with basal ganglia/thalamic AVMs, the maximum diameters of the nidi ranged from 4 to 69 mm (mean 22 mm) and the volumes ranged from 0.1 to 29.4 cm³ (mean 3.4 cm³). Eighteen nidi had only superficial venous drainage, 145 had only deep venous drainage, and 19 had both. At the time of initial GKS, the Spetzler-Martin grade was II in 11 patients, III in 130 patients, IV in 38 patients, and V in 3 patients. The mean modified radiosurgery-based AVM scale score was 1.20 (range 0.57–3.90). Ten patients had coexistent peri- or intranidal aneurysms. Compared with patients harboring superficial AVMs, more patients with basal ganglia/thalamic AVMs belonged to a higher Spetzler-Martin grade because of the lesions’ eloquent location and often deep venous drainage.

Thirty-six patients with basal ganglia/thalamic AVMs underwent a second GKS performed at a median of 4 years (range 2.0–10.1 years) after the initial GKS for residual AVMs. The maximum diameters of the nidi at the time of repeat GKS ranged from 4 to 47 mm (mean 17.4 mm), and the volumes ranged from 0.1 to 12.7 cm³ (mean 1.7 cm³). Three patients required a third GKS procedure. At the time of the third GKS, the maximum diameter was 10, 15, and 9 mm, and the volume was 0.3, 0.4, and 0.2 cm³, respectively. In the 615 patients with superficial AVMs, 92 underwent a second GKS for a residual nidus, and 3 underwent a third GKS.

Gamma Knife Procedures

The details of the GKS procedure have been reported previously.29,33,38 The Leksell Gamma Unit Model U was used until July 2001 when the Model C (Elekta Instruments, Inc.) replaced it. For the first 2 years, stereotactic biplanar angiography was the only imaging modality available for nidus definition and dose planning. Since 1991, stereotactic MR imaging was routinely used as a supplement to enhance the spatial accuracy of angiography for treatment planning. The Kula software was used for dose planning from 1989 to June 1994, after which Gamma Plan software was used.

Treatment Parameters

The treatment parameters, based on 221 GKS procedures in 182 patients with basal ganglia/thalamic AVMs and 710 procedures in 615 patients with superficial AVMs, are shown in Table 1. The treatment parameters for basal ganglia/thalamic AVMs, at the initial GKS, were as follows: mean margin dose 21.3 Gy (range 10–28 Gy); mean maximum dose 38.9 Gy (range 15–50 Gy); mean isodose line 56% (range 40%–95%); and mean number of isocenters 2.8 (range 1–22). The margin and maximum doses used in superficial and basal ganglia/thalamic AVMs were similar.

The treatment parameters of the second GKS in basal ganglia/thalamic AVMs were as follows: mean margin dose 21.1 Gy (range 7.5–27 Gy); mean maximum dose 39.1 Gy (range 15–50 Gy); mean isodose line 56% (range 48%–95%); and number of isocenters 3.9 (range 1–22). The 3 patients each with a small residual nidus undergoing a third GKS were treated with a margin dose of 25, 23, and 23 Gy.

Clinical and Imaging Follow-Up After GKS

Follow-up data were obtained from patients and referring physicians. Clinical and imaging responses, as well as GKS-related complications, were evaluated. The patients treated earlier in the study period were subjected to a vigorous protocol of yearly angiography. Later, with the introduction of MR imaging, angiography was not performed until the nidus were no longer visible on MR images. The MR images were acquired at 6-month intervals for the first 2 years and then yearly afterward. When no flow void signals were detected on MR imaging, suggesting that the AVMs had occluded, angiography was performed to confirm obliteration of the nidus. Patients were then advised to undergo follow-up MR imaging every 5 years to rule out delayed adverse effects.

Statistical Analysis

All statistical analyses were performed using the statistical software package SPSS version 13.0. Comparisons of nominal measurements were made using the chi-square test. Continuous data were compared using
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Imaging Outcome Following GKS

Of the 182 patients with basal ganglion/thalamic AVMs, 65 (35.7%) had a residual nidus demonstrated on imaging. The table below summarizes the variable and parameters in patients with basal ganglion/thalamic and supratentorial/superficial AVMs.

<table>
<thead>
<tr>
<th>Variable</th>
<th>BG/TH AVMs</th>
<th>Superficial AVMs</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of patients</td>
<td>182</td>
<td>615</td>
<td></td>
</tr>
<tr>
<td>patient demographics</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>male/female</td>
<td>88:94 (48.4%:51.6%)</td>
<td>309:306 (50.2%:49.8%)</td>
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</tr>
<tr>
<td>mean age at 1st GKS (yrs)</td>
<td>28.4 (5–80)</td>
<td>35.1 (4–82)</td>
<td>&lt;0.001</td>
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<tr>
<td>prior microsurgery</td>
<td>19 (10.4)</td>
<td>77 (12.5)</td>
<td>0.518</td>
</tr>
<tr>
<td>prior embolization</td>
<td>38 (20.9)</td>
<td>164 (26.7)</td>
<td>0.121</td>
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<tr>
<td>presenting symptoms</td>
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<td></td>
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<tr>
<td>hemorrhage</td>
<td>139 (76.4)</td>
<td>251 (40.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>seizure</td>
<td>11 (6)</td>
<td>203 (33.0)</td>
<td></td>
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<tr>
<td>headache</td>
<td>13 (7.1)</td>
<td>92 (15.0)</td>
<td></td>
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<tr>
<td>neurological deficits</td>
<td>15 (8.2)</td>
<td>33 (5.4)</td>
<td></td>
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<tr>
<td>others</td>
<td>1 (0.5)</td>
<td>17 (2.8)</td>
<td></td>
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<tr>
<td>asymptomatic</td>
<td>3 (1.6)</td>
<td>19 (3.1)</td>
<td></td>
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<tr>
<td>AVM characteristics</td>
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<tr>
<td>mean maximal diameter (mm)</td>
<td>22 (4–69)</td>
<td>24 (2–55)</td>
<td>0.020</td>
</tr>
<tr>
<td>mean nidus vol (cm³)</td>
<td>3.4 (0.1–29.4)</td>
<td>3.9 (0.1–33)</td>
<td>0.054</td>
</tr>
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<td>location</td>
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<tr>
<td>thalamus</td>
<td>97 (53.3)</td>
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<tr>
<td>basal ganglia</td>
<td>85 (46.7)</td>
<td></td>
<td></td>
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<tr>
<td>frontal</td>
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<tr>
<td>occipital</td>
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<tr>
<td>Spetzler-Martin grade</td>
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<td>&lt;0.001</td>
</tr>
<tr>
<td>I</td>
<td>0 (0)</td>
<td>115 (18.7)</td>
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<tr>
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<td>11 (6.0)</td>
<td>266 (43.3)</td>
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<tr>
<td>III</td>
<td>130 (71.4)</td>
<td>200 (32.5)</td>
<td></td>
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<tr>
<td>IV</td>
<td>38 (21)</td>
<td>34 (5.5)</td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>3 (1.6)</td>
<td>0 (0)</td>
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<tr>
<td>treatment parameters*</td>
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<tr>
<td>mean margin dose (Gy)</td>
<td>21.3 (10–28)</td>
<td>21.3 (10–32)</td>
<td>0.957</td>
</tr>
<tr>
<td>mean maximum dose (Gy)</td>
<td>38.9 (15–50)</td>
<td>39.7 (14–60)</td>
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<tr>
<td>mean isodose line (%)</td>
<td>56 (40–95)</td>
<td>55 (30–91)</td>
<td>0.053</td>
</tr>
<tr>
<td>mean isocenters</td>
<td>2.8 (1–22)</td>
<td>3.0 (1–22)</td>
<td>0.361</td>
</tr>
</tbody>
</table>

* Based on 221 and 710 Gamma Knife procedures in basal ganglion/thalamic AVMs and superficial AVMs, respectively.

A t-test. Time to nidus obliteration was calculated using the Kaplan-Meier method. The log-rank test was used to compare the obliteration rates between basal ganglia/thalamic and superficial AVMs. Univariate and multivariate analyses, using Cox proportional hazards models, were computed to evaluate favorable predictors for obliteration of basal ganglia/thalamic AVMs. Relevant factors examined included sex, age, history of embolization, history of hemorrhage, AVM volume, margin dose, maximum dose, number of isocenters, superficial or deep venous drainage, and presence of radiation-induced changes after GKS. Logistic regression was used to analyze factors potentially affecting the occurrence of radiation-induced changes and associated neurological deficits following GKS. Factors examined included sex, age, history of embolization, history of hemorrhage, AVM volume, margin dose, maximum dose, number of isocenters, and superficial or deep venous drainage. A p value of < 0.05 was considered statistically significant.

Results

Imaging Outcome Following GKS

Of the 182 patients with basal ganglia/thalamic AVMs, 65 (35.7%) had a residual nidus demonstrated on
MR imaging or angiography following a single GKS. In 14 patients (7.7%), the last MR imaging revealed an absence of flow voids, but the patients refused to undergo angiography to confirm the obliteration of nidus even when they were instructed to do so. In 91 patients (50%), total obliteration was angiographically confirmed (Fig. 1). Twelve patients (6.6%) had a subtotal obliteration (no visible nidus with a persistent early filling draining vein).

Of 36 patients undergoing repeat GKS, 13 had angiographically confirmed total obliteration. In 5 patients, there were no flow voids on MR imaging. In 16 patients, the nidus remained patent. Two patients had a subtotal obliteration. Of the 3 AVMS treated with a third GKS, 1 remained patent; angiography showed that the second was obliterated, and MR imaging showed no flow voids on the third.

In all, following one or multiple GKSs, a total obliteration was angiographically confirmed in 106 patients (58.2%) and subtotal obliteration in 8 (4.4%). Forty-eight patients (26.4%) still had a patent residual nidus. In 18 patients (9.9%), obliteration of the nidi was assumed by the fact that there were no flow voids visible on MR imaging. The overall obliteration rates based on MR imaging or angiography was 68%. The actuarial angiographic obliteration rate was 41.2% and 62.7%, respectively, at 3 and 5 years following GKS (Fig. 2).

Factors Related to Nidus Obliteration

The analysis of factors related to nidus obliteration was based on outcome after the initial GKS. Patients with no MR imaging evidence of residual AVM but without angiographic confirmation of this were excluded from the analysis. Nidi shown to be angiographically obliterated after the initial GKS were compared with those that were still patent (including those in patients with a patent nidus shown either on MR imaging or angiography and those in patients with subtotally obliterated AVMs). Univariate analysis demonstrated that the absence of prior embolization (p = 0.001), small nidus volume (p < 0.001), high margin dose (p < 0.001), and low number of isocenters (p < 0.001) were associated with increased AVM obliteration. Sex (p = 0.544), age (p = 0.640), locations of the nidus (p = 0.871), maximum dose (p = 0.114), and presence of radiation-induced changes (p = 0.899) were not related to the nidus obliteration. In multivariate analysis, the absence of prior embolization (p = 0.038), small nidus

Fig. 1. Angiograms obtained in a 32-year-old man. A and B: Lateral and frontal projections showing a right thalamic AVM that presented with a hemorrhage. In the GKS procedure, a margin dose of 25 Gy was used. C and D: Lateral and frontal projections demonstrating the AVM nidus obliterated completely 3 years after treatment.
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**Clinical Outcome Following GKS**

The clinical follow-up ranged from 24 to 266 months (median 85.8 months). Following GKS, 17 patients had 1 hemorrhage and 4 patients had 2 hemorrhages. In total, 21 patients experienced 25 hemorrhages in 850.3 risk-years (assuming patients with completely obliterated AVMs were no longer at risk for hemorrhage), yielding an annual hemorrhage rate of 2.9%. None of the patients in whom MR imaging demonstrated an absence of flow voids had a hemorrhage. As we previously reported, none of our patients with a subtotally obliterated nidus experienced a hemorrhage.

Three patients with persistent AVMs suffered clinical deterioration due to mass effect or steal phenomenon. Four patients died in this series: 2 due to complications of hemorrhage and 2, whose AVMs had been obliterated, died of diseases unrelated to the AVMs.

No acute complication was noted except for occasional frame-related headache. Excluding 8 patients treated before MR imaging was available, radiation-induced changes visualized as increased T2 signals surrounding the nidus were observed in 62 (35.6%) of 174 patients (Fig. 3). Half of these patients had mild radiation-induced changes presenting as a few millimeters of increased T2 signals surrounding the nidus. Half of the patients had moderate to severe radiation changes causing local mass effects or midline shift. These changes were noticed after a mean of 12.6 months following GKS (range 3–124 months). The changes resolved after a mean of 19.2 months (range 3–64 months). No history of hemorrhage before GKS (p = 0.002) was the only factor that was statistically significant in association with radiation-induced changes.

Twenty-six patients with the imaging finding of radiation-induced changes were asymptomatic, 8 presented with headache only, and 18 (9.9%) developed new or worsened neurological deficits. Two patient developed visual field defects, 15 presented with hemiparesis, and 1 had a change in the level of consciousness. Patients with neurological deficits were treated with corticosteroids. Among those with neurological deficits, 9 (4.9%) made a full recovery, but another 9 (4.9%) still had residual neurological deficits at the last follow-up (Fig. 4). One patient developed a large cyst that required drainage 6 years after GKS. Another 2 patients each had a small, asymptomatic cyst. No patients developed a secondary tumor.

An excellent outcome (complete obliteration of the AVM with no new deficits) was documented in 105 patients (57.7%). The mean modified radiosurgery-based AVM scale score was 1.09 in patients with excellent outcome compared with 1.36 in patients whose outcome was not excellent (including those with AVM obliteration and new deficits and all patients with residual AVM). The difference is statistically significant (p < 0.001).

**Comparison With Superficial AVMs**

The imaging outcomes following a single GKS in patients with superficial AVMs were as follows: 321 patients (52.2%) had angiographically confirmed total obliteration; 58 (9.4%) had MR imaging–confirmed obliteration; 34 (5.5%) had subtotal obliteration; and 202 (32.8%) still had a patent nidus. Following one or multiple GKSs, angiography confirmed a total obliteration in 364 (59.2%) and subtotal obliteration in 25 (4.1%) patients. One hundred fifty-seven patients (25.5%) still had a patent residual nidus. In 69 patients (11.2%), obliteration of the nidus was assumed based on MR imaging. There was no difference in angiographic obliteration rates between patients with basal ganglia/thalamic AVMs and superficial AVMs (p = 0.826) (Fig. 2).

In 260 (43.8%) of 594 patients with superficial AVMs for whom follow-up MR images were available, radiation-induced changes developed. There was a trend that patients with superficial AVMs were more likely to develop radiation-induced changes (p = 0.056). In patients with basal ganglia/thalamic AVMs in whom radiation changes developed, 32 of 62 were moderate to severe (compared with 101 of 260 patients with superficial AVMs). The difference was not statistically significant (p = 0.085). Eighteen patients with basal ganglia/thalamic AVMs had neurological deficits associated with radiation-induced changes, which were significantly higher than that in those with superficial AVMs (32 of 615) (p = 0.035) (Fig. 5).

**Discussion**

**Natural History of AVMs in the Basal Ganglia and Thalamus**

Basal ganglia and thalamic AVMs constitute 4.3%–11% of all AVMs. Studies have shown that deep-seated AVMs, nidi with deep venous drainage, and AVMs with a history of hemorrhage are more likely to rupture. Although half of all AVMs present with hemorrhage, 72%–91% of basal ganglia and thalamic AVMs bleed before they are diagnosed. The annual hemorrhage risk for AVMs in all locations has been reported in the range of 2%–4%. In contrast, basal gan-
glia and thalamic AVMs seem to carry a higher risk of hemorrhage. Fleetwood et al. evaluated 96 patients with basal ganglia and thalamic AVMs between the time of their detection and management and reported a 9.8% annual bleeding rate during 500 patient-years. Sasaki et al. followed up 15 patients with basal ganglia and thalamic AVMs treated conservatively and described an overall hemorrhage rate of 71.4% (annual rate 11.4%) and a mortality rate of 42.9% during a mean follow-up period of 6.6 years. Furthermore, the age at diagnosis in patients with basal ganglia and thalamic AVMs seems to be younger than that of patients with AVMs in other locations. The high cumulative hemorrhage risk and the significant neurological deficits associated with each hemorrhage in patients with basal ganglia and thalamic AVMs often impel neurosurgeons to take on the formidable challenge to treat AVMs in the basal ganglion and thalamus.

Microsurgery for Deep-Seated AVMs

Current treatment options for basal ganglia and thalamic AVMs include microsurgery, embolization, radiosurgery, and a combination of these methods. Microsurgery has been the mainstay of treatment for cerebral AVMs because a successful surgery eliminates the patient’s risk of hemorrhage immediately. In addition to the size of the nidus, location and venous drainage dictate the success of microsurgery. Deep feeding perforating arteries that are usually friable, poorly visualized, and located in eloquent white matter tracts also pose significant difficulty for a successful microsurgical extirpation of AVMs.

Using modern microsurgical techniques, several authors have reported high obliteration rates for basal ganglia and thalamic AVMs with reasonable rates of morbidity and mortality. Gross et al. reviewed the published microsurgical series of basal ganglia and thalamic AVMs and reported that the overall complete resection rate was 91% (193 of 212 patients) and that the overall surgical mortality rate was 2.4% (5 of 212 patients). Among the 4 largest series (15–39 surgically treated patients), the early postoperative morbidity rate ranged from 13% to 33%. The mortality rate was 0% in 1 series but ranged from 2.6% to 6.3% in the other 3 series. The permanent long-term morbidity rate ranged from 6.3% to 33%, with 1 study reporting 0% permanent morbidity. Of note, patients across these surgical series were carefully selected on the basis of several favorable factors, such as young age, small nidus size, lesion close to the ventricular ependymal surface or insular cortex, and most importantly, a history of hemorrhage that already caused preoperative neurological deficits. A parenchymal hematoma helps the surgeon in the process of dissecting the AVMs from the surrounding tissue, and an acute/subacute blood clot or the resultant encephalomalacia usually serves as a natural corridor to access the nidus while preventing new deficits.

Neurosurgeons are more reluctant to pursue surgery...
in patients with unruptured AVMs due to a high risk of new neurological deficits. In 15 patients surgically treated by Sasaki et al.,28 3 of 7 patients without preoperative paresis developed weakness, 5 of 6 patients developed new speech impairment (1 permanent), and 5 of 8 undergoing a transtemporal or transparietal approach developed permanent visual field deficits. Tew et al.34 did not operate on any patients who were neurologically intact.
Embolization of Basal Ganglia and Thalamic AVMs

The efficacy of embolization of basal ganglia and thalamic AVMs is largely limited by the small, acutely angled feeding perforating arteries and their relatively low-flow rates. In general, endovascular treatment is rarely used alone as a curative procedure but instead as an adjuvant therapy to decrease blood supply to the AVMs before microsurgery or to diminish the size of the nidus before radiosurgery. The authors of small series of basal ganglia and thalamic AVMs treated by embolization alone reported a complete obliteration rate ranging between 0% and 24% and an incidence of permanent neurological deficits ranging from 10.5% to 60%. New liquid embolic material and refined endovascular techniques will likely improve the outcome of endovascular treatment for deep-seated AVMs or effectively reduce the size or flow for subsequent definitive treatment. Adoption of sodium amytal injection and neuromonitoring during endovascular procedures has been reported to avoid complications.

Gamma Knife Surgery for Basal Ganglia and Thalamic AVMs

Radiosurgery has become the preferred therapy for small- to medium-sized and deeply located AVMs because of its minimized invasiveness and reasonable outcomes. Several authors have evaluated the results of radiosurgery for patients with basal ganglia and thalamic AVMs (Table 2). Sasaki et al. performed radiosurgery in 60 patients with basal ganglia and thalamic AVMs and reported an 85.7% actuarial rate of complete obliteration at 2.5 years. The final clinical outcomes were also favorable. Forty-four patients (73.3%) returned to work, 12 (20%) lived independently, and only 4 (6.7%) were dependent. Nicolato et al. reported similar favorable results in 21 patients with an angiographically documented obliteration rate of 81%.

In contrast, Pollock et al. and Andrade-Souza et al. cited lower obliteration rates in their studies. In a series of 56 patients with deep (basal ganglia, thalamus, and brainstem) AVMs treated with GKS by Pollock et al., angiographically or MR imaging–confirmed obliteration of the AVM was noted in 40% of patients with basal ganglia and thalamic AVMs after 1 radiosurgical procedure. The obliteration rate increased to 55% following repeat GKS. The authors attributed their less favorable results to a more conservative dose prescription (median margin dose 18 Gy). Excellent outcome (total obliteration of AVMs without neurological deficits) was achieved in 22 patients (39%) after a single radiosurgical procedure and in 27 (48%) after 1 or more procedures. Andrade-Souza et al. reported their results of LINAC-based radiosurgery in 42 patients with AVMs in the basal ganglia, internal capsule, and thalamus. The mean margin dose prescribed was 16.2 Gy. The overall obliteration rate was 61.9%, confirmed either by angiography or MR imaging. Koga and colleagues reported a 65% obliteration rate following 1 GKS and 74% obliteration rate following repeat GKS. The margin dose used was 21 Gy.

Among the aforementioned series, those with a larger
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nidus treated with a lower dose appeared to have less favorable outcomes. Both factors also correlated with poor outcome in our series. Additionally, patients who underwent a prior embolization had a lower rate of total obliteration in our series. The negative effect of preradiosurgical embolization has been reported in the literature.1,2

Hemorrhage Following Radiosurgery

Whether radiosurgery reduces the hemorrhage rate during the latency period remains debated.12,15,25 In the present series, the hemorrhage rate following GKS was 2.9%, which, although comparable to the 2%–4% risk associated with the natural course of AVMs in general, seems to be lower than that of the natural history of basal ganglia and thalamic AVMs. Pollock et al.25 and Andrade-Souza et al.3 reported a relative high hemorrhage rate in the 1st year, but the bleeding rate dropped significantly after the 2nd year following radiosurgery. Our recent study showed that the postradiosurgical hemorrhage rate reduced significantly compared with the rate between the detection of the AVMs and GKS. The effect was more prominent in the group of patients with a prior hemorrhage.37

Complications of Radiosurgery for Basal Ganglia and Thalamic AVMs

Radiation-induced changes presenting as increased T2 signals on MR imaging are not uncommon following radiosurgery for AVMs. Although most of the changes are asymptomatic, they are intuitively more likely to cause neurological deficits in patients with basal ganglia and thalamic AVMs. Sasaki et al.28 reported that 12 (20%) of 60 patients had imaging finding of radiation-induced edema, and 8 (13.3%) suffered radiation-induced neurological deficits, of which 3 (5%) were permanent. Nicolato et al.21 reported a 4% rate of permanent radiation-induced morbidity in their series of 26 patients. In 35 patients with basal ganglia and thalamic AVMs reported by Pollock et al.,7 7 (20%) suffered permanent radiation-related neurological deficits. In the series of Andrade-Souza et al.,3 8 patients (19%) had radiation-induced complications, and the deficits were permanent in 5 (11.9%). In our study, signal changes on follow-up MR imaging could be observed in 62 patients (35.6%), but only 9 patients (4.9%) suffered permanent neurological deficits.

A combination of angiography/MR imaging to accurately delineate the nidus and a conformal irradiation are keys to maximizing the radiation effects while minimizing the complications. Tractography has recently been incorporated in the treatment planning for deep-seated AVM to prevent adverse effects.16 The long-term benefits of this technology remain to be seen.

Conclusions

In young patients with small and ruptured basal ganglia and thalamic AVMs, microsurgery would be the treatment of choice, given the fact that patients with basal ganglia and thalamic AVMs tend to suffer a rehemorrhage and their cumulative morbidity and mortality rates are significant. In experienced hands, the AVM nidus might be extirpated while keeping the risk of new neurological deficits low. In patients whose expected risk of microsurgery is high, radiosurgery can be an alternative. Radiosurgery offers a reasonable chance of obliterating basal ganglia and thalamic AVMs and does so with a low risk of complications. In our series, an angiographically confirmed obliteration of the nidus was achieved in 58% of the patients. In the best-case scenario, 75% of patients, including those with angiographically and MR imaging–confirmed total obliteration and those with subtotal obliteration were free from the risk of hemorrhage. The 5% rate of permanent morbidity in our series was relatively low. For patients with a residual nidus, multimodality therapy including embolization, microsurgery, or repeat GKS may be necessary.

Disclosure

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