Journal of Clinical Neuroscience 24 (2016) 37–42

Contents lists available at ScienceDirect

Journal of Clinical Neuroscience

journal homepage: www.elsevier.com/locate/jocn

Review

Stereotactic radiosurgery for deep intracranial arteriovenous malformations, part 2: Basal ganglia and thalamus arteriovenous malformations

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ARTICLE INFO

Article history:
Received 26 October 2015
Accepted 21 November 2015

Keywords:
Basal ganglia
Gamma Knife radiosurgery
Intracranial arteriovenous malformations
Intracranial hemorrhages
Outcome prediction
Thalamus

ABSTRACT

The aim of this review is to critically analyze the outcomes following stereotactic radiosurgery (SRS) for arteriovenous malformations (AVM) of the basal ganglia and thalamus. The management of these deep-seated lesions continues to challenge neurosurgeons. Basal ganglia and thalamic AVM show a higher risk of hemorrhage, and an associated devastating morbidity and mortality, as compared to AVM in more superficial locations. Any of the currently available treatment modalities may fail or result in iatrogenic neurologic deterioration. Recent evidence from A Randomized Trial of Unruptured Brain AVM (ARUBA) further deters aggressive approaches that carry a significant risk of treatment-related adverse events. Microsurgical resection, endovascular embolization and SRS all play a role in the treatment of AVM. SRS is an effective therapeutic option for AVM of the thalamus and basal ganglia that are deemed high risk for resection. SRS offers acceptable obliteration rates, with generally lower risks of hemorrhage occurring during the latency period compared to the AVM natural history. Considering that incompletely obliterated lesions still harbor the potential for rupture, additional treatments such as repeat SRS and microsurgical resection should be considered when complete obliteration is not achieved by an initial SRS procedure. Patients with AVM of the basal ganglia and thalamus require continued clinical and radiologic observation and follow-up after SRS, even after angiographic obliteration has been confirmed.
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1. Introduction

The annual risk of hemorrhage from an arteriovenous malformation (AVM) has been traditionally cited to be in the range of 2–4% [1–6]. However, AVM hemorrhage risk can vary significantly based on its angio-architectural characteristics and a prior history of rupture. In one report, the annual hemorrhage rates ranged from 0.9% for low-risk patients (defined as those without prior AVM hemorrhage, superficial AVM location, and a component of superficial venous drainage) up to 34.4% for high-risk patients (patients with prior AVM hemorrhage, deep AVM location, and exclusively deep venous drainage) [5,7]. Additionally, deep-seated AVM (thalamic, basal ganglia and brainstem locations) also appear to carry a higher risk of hemorrhage-associated mortality, with rates up to 62.5% [8].

Basal ganglia and thalamic AVM represent a unique subgroup of intracranial vascular malformations, constituting 4–11% of all AVM [4,9–10]. Similar to brainstem AVM [11], the natural history of basal ganglia and thalamic AVM tends to be more aggressive, with an annual hemorrhage risk approaching 10% [5–6,8,12], which is substantially higher than that quoted for AVM in general [1,4]. Furthermore, the age at diagnosis in these patients seems to be younger than that of patients with AVM in other locations [8]. Basal ganglia and thalamic AVM nidi characteristically have deep venous drainage. In addition, AVM with a history of prior hemorrhage are more likely to rupture [5]. Up to 72–91% of basal ganglia and thalamic AVM present with hemorrhage, as compared to approximately 50% for all AVM locations [6,8,9,12]. Fleetwood et al. [6] reported a 9.8% annual bleeding rate in a cohort of 96 patients during a period of 500 patient-years. Sasaki et al. [8] described an 11.4% overall annual hemorrhage rate and a 42.9% mortality rate during a mean follow-up period of 6.6 years. Additionally, due to the critical neuronal pathways in the vicinity of the basal ganglia and thalamus, the morbidity and mortality associated with hemorrhage, steal phenomenon, or mass effect from AVM in these locations are significant.

The goal of treating AVM with stereotactic radiosurgery (SRS) is to obliterate the AVM nidus, abolishing any risk for future

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http://dx.doi.org/10.1016/j.jocn.2015.11.006
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hemorrhage. The mechanisms of AVM obliteration following SRS include progressive intimal thickening, thrombosis of irradiated vessels, and eventual concentric narrowing and occlusion of the vascular lumen [13]. Obliteration depends on a multitude of factors, most importantly nidus volume, angio-architectural features (compactness of the AVM and amount of normal intervening brain tissue), and radiosurgical margin dose [1]. The aim of this second part of our review series is to discuss the role of SRS in the management of deep-seated AVM, with a focus on basal ganglia and thalamic AVM. An overview of the different scoring systems and scales commonly used in outcome prediction (obliteration and clinical outcome) is provided in the first part of this series (SRS for the management of brainstem AVM) [11].

2. SRS for basal ganglia and thalamic AVM

In recent years, SRS has emerged as a mainstay therapy for small to medium-sized (less than 3 cm in maximal diameter or 10–15 cm^3 in volume), deep-seated AVM (Fig. 1). Its minimally invasive nature and favorable outcomes, both in terms of obliteration and complication rates, renders it the preferred treatment for these lesions. A systematic review of the literature regarding SRS for AVM of the basal ganglia and thalamus is presented in Table 1. Across the series reported [8,10,14–17], a total of 521 treated patients were reviewed. The median obliteration rate after the first SRS session was 61.9% (range 40–85.7%). The median nidus volume was 3.4 ml (range 2.7–4.7 ml) and the median prescribed margin dose was 20 Gy (range 16.2–21.3 Gy). Sasaki et al. [8] evaluated a cohort of 60 patients with basal ganglia and thalamic AVM. The authors reported an 86% complete obliteration actuarial rate at 2.5 years. The reported final clinical outcomes were also favorable, with 73% of patients returning to work, and 20% living independently.

In contrast Pollock et al. [14] and Andrade-Souza et al. [15] reported lower obliteration rates. In a cohort of 56 deep AVM, comprised of basal ganglia (n = 10), thalamic (n = 30), and brainstem (n = 16), Pollock et al. [14] reported complete obliteration in 43% of patients after primary SRS at a median follow-up of 45 months. The actuarial obliteration rate after one or more SRS procedures was 47% at 3 years and 66% at 4 years. Less favorable results (in terms of obliteration rates and radiation-induced changes [RIC] induced deficits) were found to be associated with a lower prescription dose (median margin dose 18 Gy) [14]. Andrade-Souza et al. [15] analyzed a cohort of 42 AVM in the basal ganglia, internal capsule, and thalamus treated with linear accelerator-based SRS using a mean margin dose of 16.2 Gy. Complete obliteration rate was reported as 62% (determined by angiography or MRI). Koga et al. [16] analyzed a cohort of 48 thalamic AVM and reported obliteration rates of 65% and 74% after one or two SRS sessions, respectively, with a 5 year obliteration rate of 82%. Higher obliteration rates were noted in patients with a prior AVM hemorrhage. Less favorable outcome was noted in patients harboring larger volume nidi and those treated with lower margin dose.

We evaluated a series of 182 patients with basal ganglia (n = 85) and thalamic (n = 97) AVM treated with SRS and followed for a minimum of 2 years [10]. The mean AVM volume was 3.4 cm^3 (range 0.1–29.4 cm^3), and the mean margin dose for the first SRS procedure was 21.3 Gy (range 10–28 Gy). Repeat SRS for residual AVM was performed in 20% of patients at a median of 4 years after the first SRS session. The mean margin dose for the repeat SRS procedure was 21.1 Gy (range 7.5–27 Gy). Complete obliteration following a single SRS treatment was demonstrated on angiography in 50%. Subtotal obliteration occurred in 7% of patients, and no flow voids were observed on MRI in an additional 8%. Following a single or repeat SRS procedure, the angiographic obliteration rate was 58%, subtotal obliteration was achieved in 4%, and an absence of flow voids on MRI was observed in 10%. The cumulative obliteration rate after one or multiple SRS sessions was 68%. Smaller nidi volume, higher margin dose, lower number of isocenters, and a lack of prior AVM embolization were significantly associated with obliteration. The annual post-SRS hemorrhage rate was 2.9% (21 patients experienced 25 hemorrhages in 850 risk-years). Permanent neurological deficits caused by symptomatic RIC were noted in 5% [10].

Kano et al. [17] analyzed a cohort of 133 basal ganglia (n = 56) and thalamic (n = 77) AVM, with a median volume of 2.7 cm^3 (range 0.1–20.7 cm^3), which were treated with Gamma Knife SRS (Elekta AB, Stockholm, Sweden) using a median margin dose of 20 Gy (range 15–25 Gy). Most of the patients in this report (85%) suffered a prior AVM hemorrhage. Complete AVM obliteration was documented in 59% and 47% of patients on MRI and angiography, respectively, with a median follow-up duration of 61 months (range 2–265 months). The actuarial obliteration rates were 57%, 70%, 72%, and 72% at 3, 4, 5, and 10 years, respectively. Factors shown to be associated with obliteration were basal ganglia AVM location, smaller AVM volume, and higher margin dose. Hemorrhage during the latency occurred in 11%, and 5% died. The actuarial post-SRS hemorrhage rates were 4.5%, 6.2%, 9.0%, 11.2%, and 15.4% at 1, 2, 3, 5, and 10 years, respectively, with an annual hemorrhage rate of 4.7%. Permanent neurological deficits secondary to symptomatic RIC developed in 5%. A SRS-induced cyst developed in one patient 56 months after treatment. Factors found to be associated with symptomatic RIC were larger AVM volume, lower margin dose, and higher radiosurgery-based AVM score [17].

2.1. Hemorrhage after SRS

Whether SRS reduces the rate of hemorrhage during the latency period remains controversial [10,18–20]. We previously found a 3% annual hemorrhage rate during the latency period after SRS, which is comparable to the 2–4% annual hemorrhage risk for AVM in all locations [10,21]. However, the natural history of basal ganglia and thalamic AVM may be worse than AVM in more superficial locations, with annual hemorrhage rates approaching 10% [5,6,8,12]. Several studies have indicated that SRS decreases the hemorrhage risk of basal ganglia and thalamic AVM, even prior to obliteration [10,15,18,20,22]. The series presented in Table 1, describing the course and outcome of 521 patients with basal ganglia and thalamic AVM, report hemorrhage during the latency period to occur in a median of 11.5% of patients (range 2.1–14.3) with a median annual risk of bleeding within the first year after SRS of 3.3% (range 0.36–9.5) [8,10,14–17].

Pollock et al. [20] and Andrade-Souza et al. [15] reported relatively high hemorrhage rates in the first year after SRS. This bleeding rate decreased substantially after the second year post-SRS. Kano et al. [17] reported a 0–9.5% risk of latency period hemorrhage for basal ganglia and thalamus AVM during the first year post-SRS. This figure dropped to 0–4.7% during the second year post-SRS. The authors [17] noted that 25% of total observed hemorrhages occurred 3 or more years after SRS. In their study, lower margin dose and larger AVM volume were significantly associated with latency period hemorrhage. We previously showed that the incidence of latency period hemorrhage was reduced as compared to the rate of hemorrhage from diagnosis to SRS. This effect was even more pronounced in patients with a ruptured AVM [23].

2.2. SRS-induced complications

RIC present as increased perinidal T2 hyperintense signal on MRI, and are well described following AVM radiosurgery. Such RIC may be asymptomatic, or associated with neurological morbidity. Deep-seated nidi, including those in the basal ganglia

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