

Gamma Knife stereotactic radiosurgery for intracranial hemangiopericytomas

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Abstract The purpose of this study is to determine the efficacy of Gamma Knife stereotactic radiosurgery (GK SRS) for intracranial hemangiopericytomas, and to investigate the optimal dose for successful tumor control without adverse effects. We evaluated 17 hemangiopericytomas of nine patients treated with GK SRS between 1999 and 2008. The mean tumor volume was 2.2 cm^3 (range $0.2\text{--}9.9 \text{ cm}^3$), and the mean and median marginal doses were 18.1 and 20 Gy (range 11–22 Gy), respectively, at the 50% isodose line. Mean clinical and radiological follow-up periods were 49 and 34 months, respectively. Successful tumor control was achieved in 14 of 17 lesions (82.4%) at time of last follow-up after GK SRS. Actuarial local tumor control rates at 1, 2, and 5 years after GK SRS were 100%, 84.6%, and 67.7%, respectively. No adverse effects, such as radiation necrosis or marked peritumoral edema, were observed in any patient. Marginal dose (≥ 17 Gy) was the only statistically significant factor for local tumor control on univariate analysis. Extended analysis using lesion data available from previous studies revealed that higher marginal dose (≥ 17 Gy) was also significant ($P = 0.028$). GK SRS provides an effective and safe adjuvant management option for patients with recurrent or residual hemangiopericytomas.

Our results suggest that doses higher than previously used (around 15 Gy) are desirable to achieve better local tumor control of hemangiopericytomas. Close radiological follow-up is also necessary for early detection of small recurrent lesions.

Keywords Hemangiopericytoma · Tumor control · Radiosurgery · Gamma Knife · Radiation dose

Abbreviations

HPC	Hemangiopericytoma
GK SRS	Gamma Knife stereotactic radiosurgery
RT	Radiation therapy
WHO	World Health Organization
MRI	Magnetic resonance imaging

Introduction

Intracranial hemangiopericytomas (HPCs) are rare malignant tumors whose natural history, optimal treatment, and clinical outcome are not yet well understood. Over the past couple of decades, they have been termed “angioblastic meningiomas” or “soft tissue sarcomas.” They are well known to have biologically and clinically aggressive behavior, even after radical resection followed by postoperative adjuvant radiation therapy (RT): local recurrences and extracranial metastases as well as intracranial metastases are common [1–4].

Surgical resection is the treatment of choice for intracranial HPCs. However, it has proven very difficult to cure the disease with surgical resection alone [4–7]. Because intracranial HPCs are known to be radiosensitive, some

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previous studies have suggested a role for adjuvant RT in the treatment of recurrent HPCs [5, 7–9].

However, there are few treatment options for HPCs that recur even after surgical resection followed by adjuvant RT, particularly when the recurrent tumors are located in eloquent or deep-seated areas. Although adjuvant chemotherapy, repeated conformal RT, and radiosurgery have been tried, the efficacy of these adjuvant treatments for recurrent intracranial HPCs remains unclear due to the scarcity and insufficiency of clinical data [10, 11].

Gamma Knife stereotactic radiosurgery (GK SRS) was introduced as a treatment option for the management of intracranial HPCs recurring after surgical resection followed by RT, because GK SRS provides safe delivery of a relatively high dose of radiation to a well-defined target (like HPCs), and it is not difficult to reapply GK SRS for possible recurrences. However, only a few studies using GK SRS for recurrent HPCs have been reported to date [1, 3, 4, 6, 11–16]. Furthermore, the optimal dose for successful local control of HPCs without adverse effects remains unclear.

The aim of this retrospective study was to investigate the efficacy of GK SRS in the treatment of recurrent intracranial HPCs, and to verify the optimal dose for achieving successful local tumor control without adverse effects.

Methods and materials

Patient characteristics

Between August 1999 and February 2008, nine consecutive patients with HPCs underwent GK SRS for the treatment of 17 recurrent or residual tumors. Diagnoses of all patients were confirmed histopathologically after surgical resection before GK SRS. Six patients were female, and the mean age of the patients was 45 years (range 29–61 years).

A single lesion was observed in four patients, and multiple lesions in five patients. On average, there were two intracranial lesions per patient. There were five HPCs of World Health Organization (WHO) grade II in four patients and 12 anaplastic HPCs of WHO grade III in five patients. GK SRS was performed for 15 recurrent lesions in seven patients who previously underwent surgical resection followed by RT (mean dose 56.9 Gy, range 50.4–61.2 Gy). The remaining two lesions in two patients were treated with GK SRS as an adjuvant therapy for new recurrent or residual lesions after resection without RT. Eight of 17 tumors were locally recurrent tumors at the resection margin of the primary operation; the other nine tumors were intracranial metastatic lesions distant from the original tumor location. Patient characteristics are summarized in Table 1.

GK SRS

GK SRS was performed using a Leksell Gamma Knife (Elekta Instrument, Stockholm, Sweden) model B or C. The planning system was a Leksell GammaPlan™ system (Elekta Instrument), based on 1.0- to 1.5-mm-thick magnetic resonance imaging (MRI) slices with gadolinium contrast enhancement. GK SRS planning was performed on axial images supplemented with reconstructed coronal and sagittal MRI images.

The prescription dose of 15–22 Gy (mean 19.0 Gy, median 20 Gy) at the 50% isodose line was applied to the tumor margin in all patients except two. In these two patients, close proximity of critical neural structures such as the optic nerve reduced the marginal dose at the 50% isodose line to 11 and 12 Gy, respectively. In total, the overall mean marginal dose was 18.1 Gy. A summary of the treatment parameters is given in Table 1.

Follow-up

Follow-up brain MRIs were performed in all nine patients 1 month after GK SRS, and then regularly at 3- to 6-month

Table 1 Summary of patient characteristics and radiosurgical parameters of nine patients with 17 lesions

Age (years)	44.9 ± 14.3 (29–61)*
Male/female	3/6
WHO grade II/III	5/12
Mean/median clinical follow-up duration (months)	48.8/45 (18–82)
Mean/median radiological follow-up duration (months)	34.2/32 (7–82)
Number of previous craniotomies	
1	5
2	4
Mean RT dose (Gy) ^a	56.9 (50.4–61.2)
Gamma Knife radiosurgery	
After surgery ^b	2
After surgery and RT	7
Mean/median tumor volume (cm ³)	2.2/1.0 (0.2–9.9)
Mean/median marginal dose (Gy)	18.1/20.0 (11.0–22.0)
Mean isodose line (%) ^c	50
Mean number of shots per lesion	6.5 (1–13)

RT radiation therapy

* Values are mean ± standard deviation (range)

^a One patient (patient no. 5) underwent RT at another hospital and the RT dose is unknown

^b Two patients underwent GK SRS for residual or recurrent lesions after resection, without RT

^c All isodose lines are 50%

intervals. At each follow-up, tumor volumes were determined from brain MRI scans using Osiris software (version 4.8; Service of Medical Informatics, Geneva University Hospital, Geneva, Switzerland). Tumor volume was computed by specifying an object in a series of regions of interest (ROIs) in a set of multiplanar images, and then summing ROI areas in each plane. The error associated with repeat measurements of tumor volume in each MRI scan was within $\pm 10\%$.

Each tumor was categorized into one of four treatment outcomes after GK SRS: complete response (CR, tumor disappeared); partial response (PR, $\geq 50\%$ decrease in tumor volume compared with measurements taken on day of GK SRS); stable disease (SD, $< 50\%$ reduction or $< 25\%$ increase in tumor volume); or progressive disease (PD, $\geq 25\%$ increase in volume). Local tumor control percentage was defined as the number of lesions in the CR, PR, and SD groups over the total number of lesions.

Both clinical and radiological data were collected up to last follow-up. The overall survival (OS) rate of patients was defined as the time interval between the date of GK SRS and the date of death or the date of last follow-up. The duration of local tumor control was defined as the time interval between the date of GK SRS and the date of local tumor recurrence.

Statistical analysis

OS time and actuarial progression-free survival time were calculated by the Kaplan–Meier method. A generalized Wilcoxon rank-sum test was used to compare the difference of local tumor control rates according to the optimal marginal dose, and to determine the correlation of OS with WHO grade. Statistical analysis was performed using SPSS version 12.0 (SPSS Inc., Chicago, IL, USA). Results were regarded as significant for $P < 0.05$.

Results

Detailed patient treatment information is given in Table 2. Mean clinical follow-up duration was 49 months (range 18–82 months), and mean and median radiological follow-up duration were 34.2 and 32 months, respectively (range 7–82 months). After GK SRS, seven of nine patients were alive at last follow-up, but two patients died during the follow-up period (patient nos. 1 and 2). One patient (patient no. 1) developed tumor recurrence and died 45 months after GK SRS. The other patient (patient no. 2) developed multiple intracranial tumors after the initial GK SRS and later underwent two further GK SRS treatments for three new lesions. This patient died 70 months after initial GK SRS, due to dissemination of tumors throughout

the whole neuraxis. Extracranial metastasis was observed in one patient (patient no. 5) during the follow-up period.

MRI volumetric evaluation after GK SRS demonstrated that CR was observed in eight tumors (47.1%), PR in five tumors (29.4%), SD in one tumor (5.9%), and PD in three tumors (17.6%). Local tumor control rate was 82.4%. Three lesions (lesion nos. 1, 4, and 11) were confirmed to be progressive on follow-up brain MRI scans taken 21, 22, and 38 months after GK SRS, respectively.

Of these three recurrent lesions, two lesions (lesion nos. 4 and 11) were managed with repeated GK SRS. Lesion no. 4 was initially treated with a marginal dose of 15 Gy, but progressed 21 months after the initial GK SRS. A second GK SRS was performed with a marginal dose of 20 Gy, after which the tumor was well controlled for 12 months. The other recurrent lesion (lesion no. 11) was treated with an initial marginal dose of 16.5 Gy. Twenty-two months after initial GK SRS, a second treatment was performed with a marginal dose of 16 Gy. The tumor remained unchanged for 22 months, but was finally confirmed as progressive 30 months after the second GK SRS.

OS rates were 100% and 83.3% at 2 and 5 years after GK SRS, respectively. Actuarial tumor control rates, as determined by the Kaplan–Meier method, were 100% at 1 year, 84.6% at 2 years, and 67.7% at 5 years (Fig. 1). There were no significant differences in tumor control rate between WHO grade II HPCs and WHO grade III HPCs ($P = 0.917$). All three failures of local tumor control occurred in the group treated with low marginal dose (< 17 Gy). High marginal dose (≥ 17 Gy) was significantly associated with longer progression-free survival ($P = 0.003$). Mean tumor volume did not significantly differ between the two groups, being 1.98 cm^3 in the low-marginal-dose group, and 2.84 cm^3 in the high-marginal-dose group ($P = 0.630$). The 2-year progression-free survival was 100% for HPCs treated with high marginal dose, compared with 50% for HPCs treated with low marginal dose.

In no case were adverse effects from radiation, such as radiation necrosis or marked peritumoral edema, ever observed in any patient who received GK SRS, as of last follow-up.

Discussion

Surgical resection is generally accepted as the standard treatment of choice in the management of HPCs, although they are hypervascular tumors prone to cause intraoperative hemorrhage, and previous studies have reported surgical mortality rates as high as 9–24% [4, 13, 14, 17]. Complete resection at first operation was strongly correlated with prolongation of time to recurrence and extension of survival duration [2, 6]. However, it is difficult to cure HPCs with surgical resection alone, because of their high

Table 2 Summary of detailed characteristics of patients and lesions, and tumor response

Patient no.	Gender/age (years)	Lesion no.	Diagnosis	Tumor location	Recurrence type after previous treatment	Pre-GK SRS treatment	Volume (cm ³)	Marginal dose (Gy)/no. of shots	Radiological follow-up duration (months)	Tumor response
1	M/35	1	HPC	Medial temporal	Op bed	OP(PR)-RT(5500 cGy)-OP(GTR)	1.5	12/11	38	PD
2	F/30	2	M-HPC	Falx	Distant	OP(NA)-OP(NA)-RT(5940 cGy)	0.5	18/7	44	CR
3		3	M-HPC	Medullar	Distant		0.6	15/9	33	CR
4		4	M-HPC	Medial temporal	Distant		0.4	15/5	33	PD
5		5	M-HPC	Medullar	Distant		0.2	20/4	18	CR
3	M/43	6	HPC	Falx	Distant	OP(GTR)-RT(5400)-OP(GTR)	0.6	20/6	82	PR
7		7	HPC	Tent	Op bed		1.0	20/3	13	CR
4	F/55	8	M-HPC	Torcular	Distant	OP(GTR)-RT(6120 cGy)	6.4	19/13	59	PR
9		9	M-HPC	Occipital	Distant		1.2	20/3	22	PR
10		10	M-HPC	Occipital	Distant		0.6	20/2	22	PR
5	F/61	11	M-HPC	Occipital	Op bed	Op(NA)-RT(NA)-OP(GTR)	9.9	16.5/12	56	PD
12		12	M-HPC	Pituitary stalk	Distant		1.8	11/5	7	PR
6	F/61	13	HPC	Falx	Op bed	OP(GTR)-RT(6120 cGy)	1.4	20/10	51	CR
7	M/60	14	M-HPC	Ventricle	Op bed	RT(5040)-OP(GTR)	0.4	22/3	27	CR
15		15	M-HPC	Ventricle	Op bed		0.3	22/1	27	CR
8	F/30	16	M-HPC	Medial temporal	Op bed	OP(GTR)	1.4	20/5	32	CR
9	F/29	17	M-HPC	Cerebellum	Op bed	OP(STR)	9.8	17/11	17	SD

HPC WHO grade II hemangiopericytoma, M-HPC WHO grade III anaplastic hemangiopericytoma, GK SRS Gamma Knife radiosurgery, OP operation, GTR gross total removal, STR subtotal removal, PR partial removal, RT radiation therapy, CR complete response, PR partial response, SD stable disease, PD progressive disease, NA not available

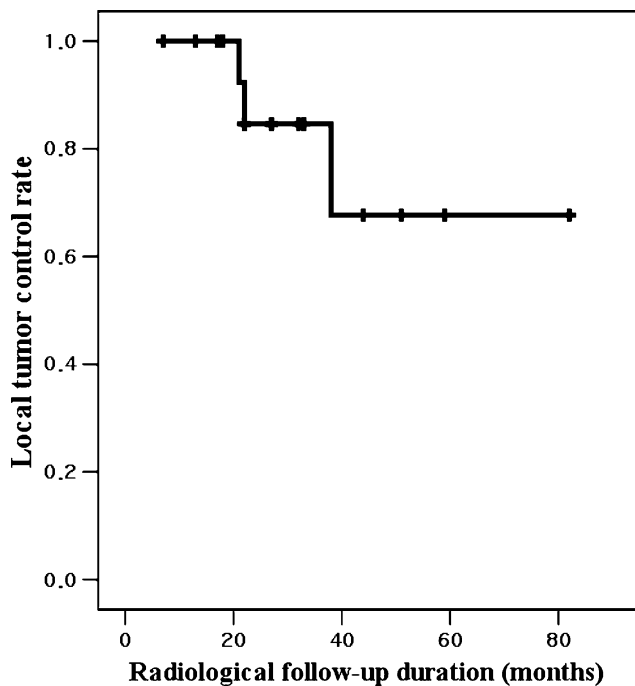


Fig. 1 Actuarial tumor control rates were 100% at 1 year, 84.6% at 2 years, and 67.7% at 5 years. Control rates were analyzed by the Kaplan–Meier method

rate of recurrence, and because intracranial and extracranial metastases after resection are frequent. In order to achieve complete resection of HPCs, some authors have recommended supplementing surgical intervention with preoperative embolization of the feeding vessels, or even preoperative radiotherapy [2, 17]. Because the hypervascular characteristics of HPCs imply “radiosensitivity,” postoperative adjuvant RT appears to significantly decrease the local recurrence rate and to lengthen recurrence-free duration [12, 16]. Guthrie et al. reported that the mean progression-free duration of irradiated HPCs after surgical resection was 74 months, while that of nonirradiated HPCs was 29 months [5, 8, 18]. Full radiation doses of greater than 50 Gy were recommended for best tumor control outcome and to prevent early recurrence after surgical resection [4, 8, 14].

However, it is very difficult to treat recurrent HPCs after surgical resection followed by RT. Treatment options in such cases may include repeat surgery, salvage chemotherapy, repeated fractionated stereotactic radiotherapy, or radiosurgery. If there is low risk of significant surgical morbidity or mortality, repeat surgical resection for recurrent HPCs has been recommended as the first treatment option [4]. Salvage chemotherapy with various chemotherapeutic agents, including α -interferon, provided limited efficacy with acceptable toxicity for refractory recurrent HPCs [10, 11]. Although results to date are disappointing, the hypervascular nature of HPCs suggest that antiangiogenic

strategies might be another attractive new therapeutic option for the treatment of recurrent HPCs [11].

GK SRS for HPCs

Since Coffey et al. reported their preliminary study of GK SRS for recurrent HPCs in 1993, only a few studies with limited patient numbers have been reported [1, 3, 4, 6, 11–16]. Most studies reported immediate and dramatic shrinkage of tumors after radiosurgery for HPCs, and local tumor control rates were 46–93% [1, 3, 4, 6, 11–16]. Similarly, the local tumor control rate in the present study was 82.4%, as measured over a mean and median radiological follow-up period of 34 and 32 months, respectively. These results are summarized in Table 3.

During the follow-up period in our study, six lesions in three patients developed new distant metastases in sites different from that initially treated with GK SRS. This observation is consistent with previous reports and shows that GK SRS alone cannot prevent further distant metastases, even when local control is successful [1, 2, 4, 14]. However, RT as well as GK SRS after surgical resection did not seem to prevent further local or neuraxis recurrence [14].

Only two lesions were initially treated with GK SRS without previous RT in our study. These lesions were successfully controlled throughout the follow-up duration, but long-term follow-up data are required to assess the long-term outcome of GK SRS for these lesions. Recently, along with progress in microsurgical techniques, advanced imaging technology has provided an opportunity to detect smaller residual and recurrent HPCs. These trends might make radiosurgical techniques such as GK SRS more attractive for the treatment of residual and recurrent HPCs. Payne et al. emphasized that, in order to minimize GK SRS-related complications for recurrent HPCs, GK SRS should be performed at the first sign of recurrence, when the tumors are small [4]. The mean tumor volume (2.2 cm^3) in the present study is relatively small, and the mean marginal dose (18.1 Gy) is relatively high compared with in previous reports. We used aggressive imaging surveillance techniques to perform GK SRS for these tumors as early as possible, and at as high a marginal dose as possible. Most authors recommended close imaging follow-up after GK SRS for HPCs, because of their high recurrence rates and their propensity to metastasize [4, 14]. Aggressive imaging follow-up at no more than 6-month intervals after GK SRS should be performed to detect early recurrence and new metastases.

Optimal dose for the treatment of recurrent HPCs

The mean marginal doses used in previous studies ranged from 13.5 to 17 Gy [1, 3, 4, 6, 11–16]. Only one study,

Table 3 Summary of previous reports for hemangiopericytomas treated with Gamma Knife radiosurgery

	No. of patients	No. of tumors	Tumor volume mean, range (cm ³)	Marginal dose, mean, range (Gy)	Tumor control rate (%)	Mean/median radiological follow-up duration, range (months)
Coffey et al.	5	11	8.5 (0.4–24.3)	15.5 (12–18)	82	12.7/15 (4–17)
Galanis et al.	10	20	NA	NA (12–18)	90	36/NA (NA)
Payne et al.	10	12	7.6 (0.3–33.6)	14 (2.8–25)	75	25/NA (3–56)
Sheehan et al.	14	15	8.8 (0.3–26.6)	15 (11–20)	80	31.3/21 (5–76)
Ecker et al.	15	45	7.8 (0.4–58.3)	16 (12–21)	93	45.6/NA (NA)
Chang et al. ^a	8	8	NA	20.8 (16–24)	87.5	44/NA (8–77)
Sun et al.	22	58	5.4 (0.1–37.2)	13.5 (10–20)	89.7	26/NA (5–62)
Kano et al.	20	29	4.5 (0.07–34.3)	15.0 (10–20)	72.4	37.9/23.3 (NA)
Olson et al.	21	28	4.6 (0.3–18.7)	17.0 (2.8–22)	46.4	69/68 (2–138)
Current study	9	17	2.2 (0.2–9.9)	18.1 (11–22)	82.4	33.8/32 (7–82)

NA not available

^a Their treatment modalities included LINAC (four patients) and Cyberknife (four patients)

reported by Chang et al., applied higher marginal doses (mean marginal dose of 20.5 Gy) than that of our study. However, this study employed different treatment modalities, namely LINAC and Cyberknife [13].

The optimal dose for achieving successful tumor control without adverse radiation effects remains unclear. Shaw et al. reported on the maximum tolerated doses of single-fraction radiosurgery in patients with recurrent, previously irradiated primary brain tumors and brain metastases. Their report gave the maximum tolerable doses as 24, 18, and 15 Gy for tumors ≤ 20 , 21–30, and 31–40 mm in maximum diameter, respectively [19]. Furthermore, relatively high prescription doses of 18 Gy were not significantly related to adverse radiation effects in a report on radiosurgery for malignant meningioma by Ojemann et al. [20]. Considering the malignant characteristics of HPCs, a similar treatment strategy might apply to GK SRS for recurrent HPCs following RT. In the most recent and largest study, which included 22 patients with 51 HPCs, Sun et al. suggested that treatment principles for GK SRS of HPCs should be similar to those used for intracranial metastases, and that an aggressive prescription dose could achieve a reduction in the rate of local recurrence [12]. This finding has been further supported by Sheehan et al., who recommended that a radiation dose of at least 15 Gy should be delivered to the tumor margin for successful local tumor control, and Kano et al., who reported significantly better progression-free survival in HPCs treated with high marginal dose (≥ 14 Gy) [1, 14].

In this study, all three recurrent tumors that were not successfully treated by GK SRS occurred using a marginal dose that was relatively low (12, 15, and 16.5 Gy, respectively). Higher marginal dose (≥ 17 Gy) also significantly correlated with longer progression-free survival. Our results demonstrate that recurrent HPCs should be treated with a relatively high marginal dose to achieve

long-term tumor control, and that close radiological follow-up is necessary to detect early and small recurrent lesions.

In order to overcome the small number of cases in our study, we added lesion data from the previous studies of Olson et al. and Sheehan et al. [3, 14]. These data were acceptable because the treatment parameters used in those studies were sufficiently well described to allow comparison with our data. Four lesions from these studies were excluded from this analysis: two lesions from Olson et al.'s study, treated with marginal doses of 2.8 and 3.3 Gy, respectively; and two lesions from Sheehan's study with tumor volumes of 23 and 26 cm³, respectively. A total of 52 HPCs were included and categorized into two groups according to the marginal dose used. Of the 52 tumors, 31 (59.6%) tumors were treated with high marginal dose (≥ 17 Gy) and 21 tumors (40.4%) were treated with low marginal dose (< 17 Gy). Progression-free survival rates after 2 and 5 years were 96.8% and 91.9%, respectively, in the high-dose group (≥ 17 Gy), in contrast to 70.7% and 61.8%, respectively, in the other group (< 17 Gy) (Fig. 2). This difference was statistically significant ($P = 0.028$). Mean tumor volume was also smaller in the group with high marginal dose (≥ 17 Gy) than in the other group (3.29 and 5.75 cm³, respectively); however, this difference was not significant ($P = 0.088$). Adverse radiation effects related to a relatively high marginal dose were not reported. Although the possibility of different treatment scheme such as target delineation and target covered ratio among different institutes, we could obtain the same critical marginal dose (≥ 17 Gy), which was significantly associated with improved local tumor control on analysis of the expanded data set.

To date, the efficacy of repeated GK SRS for failed cases is disappointing. Sheehan et al. reported that repeated

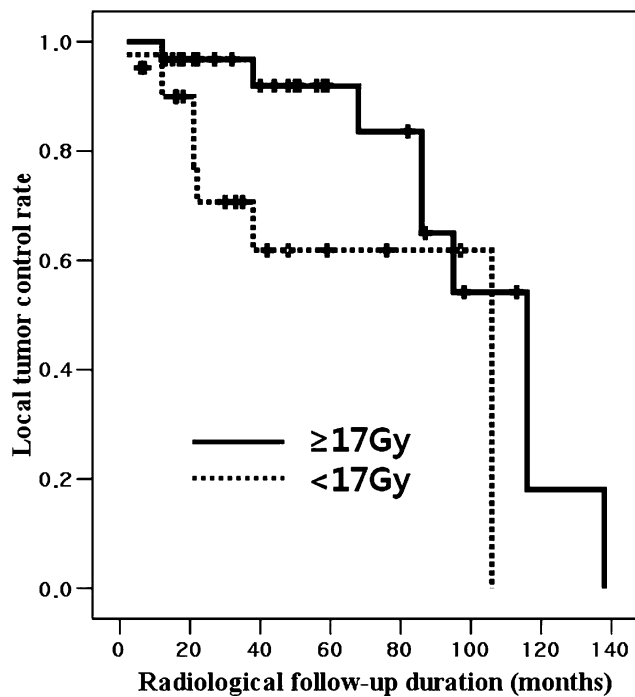


Fig. 2 Kaplan–Meier curves demonstrating the statistical difference between groups treated with different marginal doses (<17, \geq 17 Gy). Data from Olson et al. and Sheehan et al. are also included [3, 14]. Marginal dose of \geq 17 Gy was significantly associated with longer progression-free survival ($P = 0.028$)

GK SRS was unsuccessful in achieving tumor control in two patients where the initial GK SRS was unsuccessful [14]. In our study, one of two lesions treated with repeated GK SRS progressed 30 months after the second GK SRS; however, the lesion was treated with a low marginal dose (16 Gy) due to the relatively large tumor volume. The optimal treatment strategy for tumors that do not respond to initial GK SRS treatment should be considered in further studies.

Complications

As noted above, the possibility of additional adverse radiation effects is one of the most concerning aspects of GK SRS for recurrent HPCs, because most patients have had previous RT with more than 50 Gy. Payne et al. performed GK SRS with marginal dose decreased by 2–4 Gy to recurrent HPCs already treated with RT [4]. In our study, no additional adverse effects from radiation were observed. In particular, patients could receive multiple GK SRS treatments for different recurrences without adverse effects, such as pronounced edema or radiation necrosis, throughout their follow-up periods. This observation is supported by previous reports [4, 14]. However, long-term adverse effects cannot be excluded, and patients should therefore undergo close long-term follow-up.

Limitations of this study

This study is retrospective and limited to a small number of cases. Additionally, mean radiological follow-up duration was 34 months. Therefore, we cannot be certain that the treatment of choice for recurrent intracranial HPCs is GK SRS, or that the results presented herein represent final tumor control rates. Because of the scarcity of these tumors, a further well-designed, multi-institutional study with long-term follow-up is mandatory for determining optimal treatment strategy and dose for recurrent intracranial HPCs.

Conclusion

GK SRS provides an effective and safe adjuvant management option for patients with recurrent or residual HPCs. The results of our careful study demonstrate that doses higher than previously used (around 15 Gy) are desirable for better local tumor control of HPCs without severe adverse radiation effects. Furthermore, the marginal dose is mainly determined by tumor volume, and it is important to identify tumor recurrence at an early stage by close follow-up imaging after treatment.

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References

- Kano H, Nirranjan A, Kondziolka D, Flickinger JC, Lunsford LD (2008) Adjuvant stereotactic radiosurgery after resection of intracranial hemangiopericytomas. *Int J Radiat Oncol Biol Phys* 72: 1333–1339
- Kim JH, Jung HW, Kim YS, Kim CJ, Hwang SK, Paek SH, Kim DG, Kwun BD (2003) Meningeal hemangiopericytomas: long-term outcome and biological behavior. *Surg Neurol* 59:47–53 (discussion 53–44)
- Olson C, Yen CP, Schlesinger D, Sheehan J (2009) Radiosurgery for intracranial hemangiopericytomas: outcomes after initial and repeat Gamma Knife surgery. *J Neurosurg*. doi:10.3171/2009.3.JNS0923
- Payne BR, Prasad D, Steiner M, Steiner L (2000) Gamma surgery for hemangiopericytomas. *Acta Neurochir (Wien)* 142:527–536 (discussion 536–527)
- Bastin KT, Mehta MP (1992) Meningeal hemangiopericytoma: defining the role for radiation therapy. *J Neurooncol* 14:277–287
- Galanis E, Buckner JC, Scheithauer BW, Kimmel DW, Schomberg PJ, Piepgras DG (1998) Management of recurrent meningeal hemangiopericytoma. *Cancer* 82:1915–1920
- Soyuer S, Chang EL, Seleck U, McCutcheon IE, Maor MH (2004) Intracranial meningeal hemangiopericytoma: the role of radiotherapy: report of 29 cases and review of the literature. *Cancer* 100:1491–1497

8. Dufour H, Metellus P, Fuentes S, Murracchiole X, Regis J, Figarella-Branger D, Grisoli F (2001) Meningeal hemangiopericytoma: a retrospective study of 21 patients with special review of postoperative external radiotherapy. *Neurosurgery* 48:756–762 (discussion 762–753)
9. Kirn DH, Kramer A (1996) Long-term freedom from disease progression with interferon alfa therapy in two patients with malignant hemangiopericytoma. *J Natl Cancer Inst* 88:764–765
10. Chamberlain MC, Glantz MJ (2008) Sequential salvage chemotherapy for recurrent intracranial hemangiopericytoma. *Neurosurgery* 63:720–726 (author reply 726–727)
11. Ecker RD, Marsh WR, Pollock BE, Kurtkaya-Yapici O, McClelland R, Scheithauer BW, Buckner JC (2003) Hemangiopericytoma in the central nervous system: treatment, pathological features, and long-term follow up in 38 patients. *J Neurosurg* 98:1182–1187
12. Sun S, Liu A, Wang C (2009) Gamma Knife radiosurgery for recurrent and residual meningeal hemangiopericytomas. *Stereotact Funct Neurosurg* 87:114–119
13. Chang SD, Sakamoto GT (2003) The role of radiosurgery for hemangiopericytomas. *Neurosurg Focus* 14:e14
14. Sheehan J, Kondziolka D, Flickinger J, Lunsford LD (2002) Radiosurgery for treatment of recurrent intracranial hemangiopericytomas. *Neurosurgery* 51:905–910 (discussion 910–911)
15. Alen JF, Lobato RD, Gomez PA, Boto GR, Lagares A, Ramos A, Ricoy JR (2001) Intracranial hemangiopericytoma: study of 12 cases. *Acta Neurochir (Wien)* 143:575–586
16. Coffey RJ, Cascino TL, Shaw EG (1993) Radiosurgical treatment of recurrent hemangiopericytomas of the meninges: preliminary results. *J Neurosurg* 78:903–908
17. Jaaskelainen J, Servo A, Haltia M, Wahlstrom T, Valtonen S (1985) Intracranial hemangiopericytoma: radiology, surgery, radiotherapy, and outcome in 21 patients. *Surg Neurol* 23: 227–236
18. Guthrie BL, Ebersold MJ, Scheithauer BW, Shaw EG (1989) Meningeal hemangiopericytoma: histopathological features, treatment, and long-term follow-up of 44 cases. *Neurosurgery* 25: 514–522
19. Shaw E, Scott C, Souhami L, Dinapoli R, Kline R, Loeffler J, Farnan N (2000) Single dose radiosurgical treatment of recurrent previously irradiated primary brain tumors and brain metastases: final report of RTOG protocol 90-05. *Int J Radiat Oncol Biol Phys* 47:291–298
20. Ojemann SG, Sneed PK, Larson DA, Gutin PH, Berger MS, Verhey L, Smith V, Petti P, Wara W, Park E, McDermott MW (2000) Radiosurgery for malignant meningioma: results in 22 patients. *J Neurosurg* 93(Suppl 3):62–67