J Neurooncol (2014) 119:169–176
DOI 10.1007/s11060-014-1470-x

CLINICAL STUDY

Stereotactic radiosurgery of petroclival meningiomas: a multicenter study

Robert Starke · Hideyuki Kano · Dale Ding · Peter Nakaji · Gene H. Barnett · David Mathieu · Veronica Chiang · James B. Yu · Judith Hess · Heyoung L. McBride · Norissa Honea · John Y. K. Lee · Gazanfar Rahmathulla · Wendi A. Evanoff · Michelle Alonso-Basanta · L. Dade Lunsford · Jason P. Sheehan

Received: 10 November 2013 / Accepted: 20 April 2014 / Published online: 13 May 2014

Abstract Petroclival meningiomas are difficult to treat due to their intimate location with critical structures, and complete microsurgical resection is often associated with significant morbidity. In this study, we evaluate the outcomes of petroclival meningiomas treated with Gamma Knife radiosurgery (GKRS) as an adjunct to microsurgery or a primary treatment modality. A multicenter study of 254 patients with a benign petroclival meningioma was conducted through the North American Gamma Knife Consortium. One hundred and forty patients were treated with upfront radiosurgery, and 114 following surgery. Multivariate analysis was used to determine predictors of favorable defined as no tumor progression following radiosurgery and the absence of any new or worsening neurological function. At mean follow up of 71 months (range 6–252), tumor volumes increased in 9 % of tumors, remained stable in 52 %, and decreased in 39 %. Kaplan–Meier actuarial progression free survival rates at 3, 5, 8, 10, and 12 years were 97, 93, 87, 84, and 80 % respectively. At last clinical follow-up, 93.6 % of patients demonstrated no change or improvement in their neurological condition whereas 6.4 % of patients experienced progression of symptoms. Favorable outcome was achieved in 87 % of patients and multivariate predictors of favorable outcome included smaller tumor volume (OR = 0.92; 95 % CI 0.87–0.97, $p = 0.003$), female gender (OR 0.37; 95 % CI 0.15–0.89, $p = 0.027$), no prior radiotherapy (OR 0.03; 95 % CI 0.01–0.36, $p = 0.006$), and decreasing maximal dose (OR 0.92; 95 % CI 0.96–0.98, $p = 0.010$). GKRS of petroclival meningiomas achieves neurological preservation in most patients and with a high rate of tumor control.

Keywords Meningioma · Radiosurgery · Gamma Knife

Abbreviations

GKRS	Gamma Knife radiosurgery
SRS	Stereotactic radiosurgery
MR	Magnetic resonance
CT	Computed tomography
IMRT	Tintensity-modulated radiotherapy
FSRT	Fractionated stereotactic radiotherapy

Introduction

Patients with a petroclival meningioma commonly present with neurological deterioration due to the tumor's close proximity to neurovascular structures. Because of the

R. Starke · D. Ding · J. P. Sheehan (✉)
Department of Neurological Surgery, University of Virginia, Charlottesville, VA 22908, USA
e-mail: jsheehan@virginia.edu

H. Kano · L. D. Lunsford
University of Pittsburgh, Pittsburgh, PA, USA

P. Nakaji · H. L. McBride · N. Honea
Barrow Neurological Institute, Phoenix, AZ, USA

G. H. Barnett · G. Rahmathulla · W. A. Evanoff
Cleveland Clinic, Cleveland, OH, USA

D. Mathieu
University of Sherbrooke, Sherbrooke, QC, Canada

V. Chiang · J. B. Yu · J. Hess
Yale University, New Haven, CT, USA

J. Y. K. Lee · M. Alonso-Basanta
University of Pennsylvania, Philadelphia, PA, USA

availability of neuro-imaging, the diagnosis of these lesions as incidental findings has also increased. Although surgical resection has historically been the primary treatment modality for symptomatic tumors or tumors with progression on serial imaging studies, microsurgery has been associated with significant morbidity and mortality, incomplete resection, and delayed progression [1–26].

Stereotactic radiosurgery (SRS) has emerged as a valuable therapeutic option for selected patients following microsurgery and as a primary treatment modality. Outcomes of petroclival meningiomas treated with surgery and/or radiotherapy are limited to small patient series which limit rigorous statistical analysis [8, 21, 27]. Petroclival meningiomas present unique challenges from a radiosurgical planning standpoint due to their proximity to the brainstem, cranial nerves, and vascular structures and their potential for causing the future development of hydrocephalus. In this multi-institutional study, we assess the outcomes of patients with petroclival meningiomas treated with Gamma Knife radiosurgery (GKRS) to determine long-term predictors of neurological function, tumor progression, and overall outcome.

Materials and methods

Institutional review board approval to participate in this study was obtained individually by medical centers who are members of the North American Gamma Knife Consortium (NAGKC). Collection of the outcomes of 254 patients with petroclival meningiomas treated with GKRS was conducted at each center (Table 1). The following centers contributed to this study: the Cleveland Clinic (N = 2), Yale University (N = 6), Barrow Neurological Institute (N = 30), University of Pittsburgh (N = 161), University of Pennsylvania (N = 11), University of Sherbrooke (N = 16), and University of Virginia (N = 28).

Petroclival meningiomas were defined as tumor whose maximal volume was centered over the region between the petrous apex and the upper two-thirds of the clivus [1, 27]. 44.9 % of the patients included had a histologically diagnosed WHO grade I petroclival meningiomas. Remaining patients had clinical and neuro-imaging features consistent with a benign meningioma. For inclusion, patients were required to have a minimum of 6 months neuro-imaging and clinical follow-up after GKRS. This minimum follow-up period enabled some evaluation of the beneficial and adverse effects from radiosurgery since any effects usually are latent.

Radiosurgical technique

The Gamma Knife Models U, B, C, 4C, or Perfexion® were used depending on the technology available at the time of treatment at participating centers. Median prescription dose delivered to the tumor margin was 13 Gy (range 9–40 Gy; Table 2). Radiosurgical treatment parameters (e.g. margin dose, treatment isodose) were determined individually by each treatment center and included considerations related to tumor volume, proximity to critical structures, pre-existing neurological deficits, and history of previous treatments.

Table 1 Baseline characteristics of patients with petroclival meningiomas treated with GKRS

Characteristic	Total
Female gender	195 (76.8)
Median age	57.1 ± 13.4 years (18–89)
Previous resection	114 (44.9)
Prior radiotherapy	4 (1.6)
Mean time from presentation to GKRS	14.6 ± 33.3 months (0–264)
Initial presentation	
Headache	64 (25.2)
Subjective dizziness	71 (28.0)
CN III/IV/VI	31(14.6)
CN V	23 (9.1)
CN VII	106 (32.6)
CN VIII	70 (27.6)
CN IX/X	14 (5.5)
CN XI	1 (0.4)
CN XII	3 (1.2)
Ataxia	22 (8.7)
Other cerebellar alteration/deficit	5 (2.0)
Body weakness	13 (5.1)
Change body sensation	11 (4.3)
Mean volume	7.8 ± 6.6 cc (0.17–36.1)
Mean follow up	71.1 ± 50.8 months (6–252)

Characteristics are presented as sample size (percent) and mean ± SD (range)

Clinical and neuro-imaging follow-up

Clinical and neuro-imaging evaluations were generally performed at 6 month follow-up intervals for the first 2 years after radiosurgery. Patients without evidence of tumor growth

Table 2 Gamma Knife treatment parameters

Characteristic	Total
Margin dose	13.4 ± 2.4 (9–40) Gy
Maximum dose	27.6 ± 5.5 (18–80) Gy
Isodose line	48.9 % ± 4.6 (30–60)
Isocenters	11.2 ± 6.7 (1–41)
Isodose volume	7.5 ± 6.2 (0.3–33.1)

Characteristics are presented as sample size (percent) and mean ± SD (range)

and or new neurological findings, follow up intervals were subsequently increased to every 1–2 years. The follow-up images were compared with the images obtained at the time of GKRS. Tumor growth (an increase of more than 10 % from the original tumor volume at the time of GKRS) within the planned treatment volume or adjacent to it was defined as tumor progression. Guidelines for imaging review were provided to each center in the NAGKC network and films were independently reviewed at each participating institution.

Statistical analysis

Statistical analyses of categorical variables were carried out using Chi square and Fisher's exact tests. Calculations of normality were assessed statistically and graphically. Statistics of means were carried out using unpaired student *t* test, and Wilcoxon rank sum tests when variables were not normally distributed. Youden indices were used to determine the optimal dichotomized breakpoint of maximal dose to predict favorable outcome and optimal dichotomized breakpoint of tumor volume to predict tumor progression. The following dependent variables were assessed in univariate and multivariate analysis: tumor free progression, worsening or new decline in neurological function, and favorable outcome (no tumor progression and no worsening or new decline in neurological function). The following factors were assessed to determine predictors of the above dependent outcomes: patient age, gender, year of GKRS treatment, time from symptom onset, tumor location, tumor volume, maximal tumor diameter, history of chemotherapy, specific signs and symptoms on presentation and/or at the time of treatment, history of surgery, history of radiotherapy, maximal GKRS dose, marginal GKRS dose, isodose line, number of isocenters, duration of imaging follow up, and duration of clinical follow up. Kaplan–Meier risk of tumor progression was calculated. Factors predictive of tumor progression ($p < 0.15$) were entered into multivariate Cox regression analysis to assess hazard ratios. Clinical covariates predicting new or worsening neurological function with a univariate p value < 0.15 were included in multivariate logistic regression analysis. Additionally, clinical covariates predicting unfavorable outcome with a univariate p value < 0.15 were included in multivariate logistic regression analysis. Clinically significant variables and interaction expansion covariates were further assessed in both Cox and logistic multivariate analyses as necessary.

Results

Clinical outcomes

During a mean follow-up of 71.1 months (range 6–252 months), 6.4 % of patients had new or worsening neurological function, 27.1 % had improvement in neurological function, and 66.5 % had no change in neurological function. Specific improvement and decline in signs, symptoms, and cranial nerve functions are displayed in Table 3 and 4.

Table 3 Overall clinical and tumor outcomes

Characteristic	Outcome
Mean pre-GKRS tumor volume	7.8 ± 6.6 (0.17–36.1)
Mean post-GKRS tumor volume	6.2 ± 5.9 (0.4–33)
Tumor size	
Decrease tumor volume	98 (39.2)
No change in volume	129 (51.6)
Increase tumor volume	2 (9.2)
Clinical outcome	
No change in outcome	157 (66.5)
Improvement in outcome	64 (27.1)
New or worsening neurological function	15 (6.4)
Favorable overall outcome	202 (86.7)
Unfavorable overall outcome	31 (13.3)
Post-GKRS hydrocephalus	7 (2.8)
Post-GKRS ventriculoperitoneal shunt	7 (2.8)
Post-GKRS radiotherapy	1 (0.4)
Post-GKRS surgery	8 (3.2)

Table 4 Specific alterations in clinical signs and symptoms following Gamma Knife

Characteristic	Presentation	New or worsening function
Initial presentation		
Subjective dizziness	71 (28.0)	7 (2.8)
CN III/IV/VI	31(14.6)	6 (2.4)
CN V	23 (9.1)	10 (3.9)
CN VII	106 (32.6)	3 (1.2)
CN VIII	70 (27.6)	1 (0.4)
CN IX/X	14 (5.5)	3 (1.2)
CN XI	1 (0.4)	0
CN XII	3 (1.2)	2 (0.8)
Ataxia	22 (8.7)	5 (2.0)
Other cerebellar alteration/deficit	5 (2.0)	2 (0.8)
Body weakness	13 (5.1)	3 (1.2)
Change body sensation	11 (4.3)	1 (0.4)

Patient, tumor, and GKRS planning variables predictive of new or worsening neurological function following GKRS in univariate included male gender, presentation, and increasing prescription dose, and increasing volume. In multivariate analysis, those with pre-existing dizziness and imbalance (OR 3.47; 95 % CI 1.02–11.72, $p = 0.015$) and those with pre-existing alterations in either visual function or movement (OR 5.28; 95 % CI 1.44–19.41, $p = 0.012$)

Table 5 Factors predictive of new/worsening symptoms in multivariate analysis

Pre-GKRS variables	Odds ratio (95 % CI, *p value*)
Dizziness or imbalance	3.47 (1.02–11.72, 0.015)
Alteration visual function or movement	5.28 (1.44–19.41, 0.012)

were most likely to worsen following GKRS (Table 5). There was a trend towards new or worsening neurological function with increasing volume in multivariate analysis (OR 1.07; 95 % CI 1.00–1.14, $p = 0.065$) when controlling for the above baseline alterations in function. Additionally, patients with tumor progression were more likely to experience new or worsening neurological function (OR 4.59; 95 % CI 1.31–16.06, $p = 0.017$).

Radiologic outcome

Mean tumor volume decreased from 7.8 cc (range 0.2–36.1) prior to GKRS to 6.2 cc (range 0.4–33) following GKRS (Table 3). At last follow-up, tumor volumes increased in 9 % of tumors, remained stable in 52 %, and decreased in 39 %. In Kaplan–Meier analysis, actuarial progression free survival at 3, 5, 8, 10, and 12 years was 97, 93, 87, 84, and 80 %, respectively (Fig. 1a). Factors predictive of tumor progression in univariate analysis included male gender, increasing time from symptom onset, history of radiotherapy, increasing volume, decreasing maximal dose, and decreasing isodose percent. Absence of prior surgical resection was not predictive of tumor progression (HR 1.34; 95 % CI 0.58–3.12, $p = 0.495$; Fig. 1b). Regarding tumor control, the optimal breakpoint for tumor volume occurred at 8 cc. Patients with a tumor volume greater than or equal to 8 cc (i.e. 40 % of the study population) were significantly more likely to have tumor progression (HR 2.77; 95 % CI 1.16–6.61, $p = 0.022$, Fig. 1c).

In multivariate analysis, covariates predictive of tumor progression included increasing time from diagnosis (HR 1.01; 95 % CI 1.00–1.02, $p = 0.001$), history of prior radiation (HR 8.64; 95 % CI 1.90–39.27, $p = 0.005$), increasing tumor volume (HR 1.07; 95 % CI 1.03–1.13, $p = 0.002$), and decreasing maximal dose (HR 1.08; 95 % CI 1.02–1.13, $p = 0.004$; Table 6). When controlling for other independent predictors, tumors with a volume greater than 8 cc were 3.7 times more likely to have tumor progression (95 % CI 1.5–9.2, $p = 0.006$). Resection prior to GKRS was not predictive of long-term tumor control in multivariate analysis when controlling for other variables.

Further treatments after radiosurgery

During follow-up, one patient underwent fractionated radiation therapy because of out of field tumor progression.

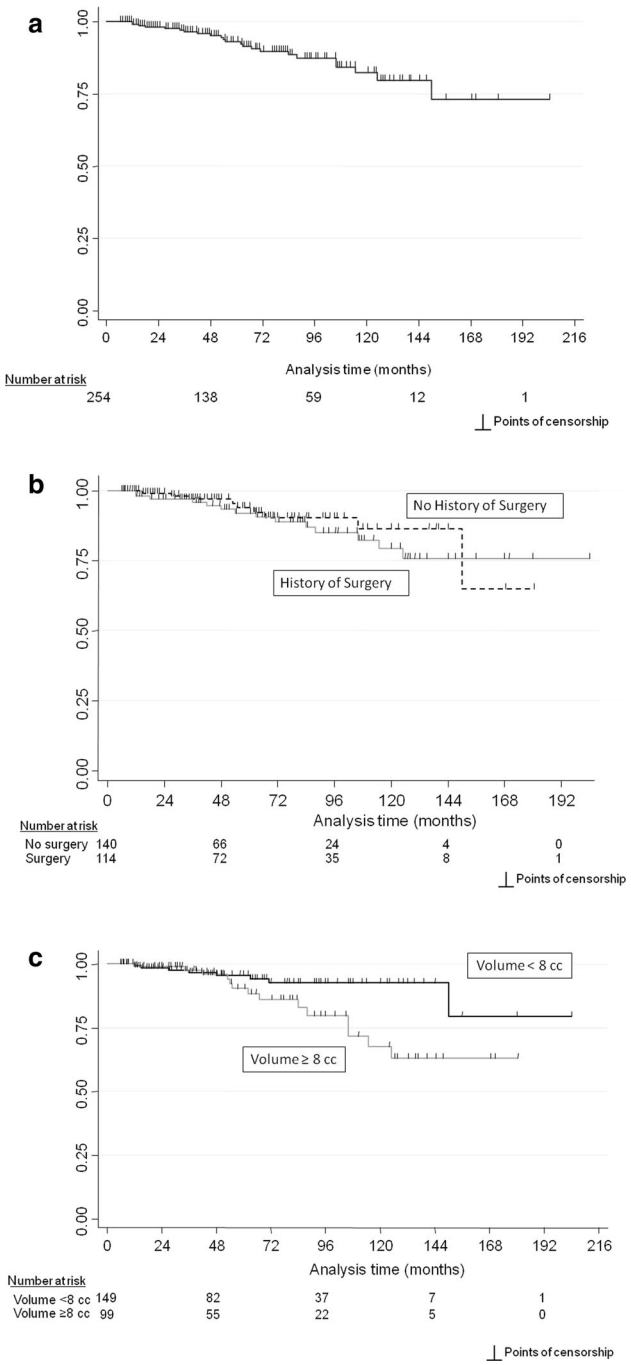

Fig. 1 **a** Tumor free progression after Gamma Knife radiosurgery. **b** Tumor free progression in patients with and without a history of microsurgical resection. **c** Progression free tumor survival in patients with tumors <8 cc and those ≥8 cc ($p = 0.022$). The integers along the X-axis denote the number of patients reaching each of the major time milestones

Seven patients required a ventriculoperitoneal shunt. Only one patient who required a shunt had tumor progression during the follow-up. Seven patients had a single microsurgical resection after radiosurgery because of tumor progression and one patient underwent two resections. All

Table 6 Factors predictive of tumor progression in multivariate analysis

Pre-GKRS Variables	Hazard ratio (95 % CI, *p value*)
Time from symptom onset	1.01 (1.00–1.02, 0.001)
Volume	1.07 (1.03–1.13, 0.002)
Prior radiotherapy	8.64 (1.90–39.27, 0.005)
Maximal dose	1.08 (1.02–1.13, 0.004)

Table 7 Factors predictive of favorable outcome in multivariate analysis

Variables	Odds ratio (95 % CI, *p* value)
Volume	0.92 (0.87–0.97, 0.003)
Male gender	0.37 (0.15–0.89, 0.027)
Prior radiotherapy	0.03 (0.01–0.36, 0.006)
Decreasing maximal dose	0.92 (0.96–0.98, 0.010)

tumors resected after radiosurgery were confirmed WHO grade I meningiomas.

Other serious complications

In the current series, there was no evidence of vascular injury or brainstem ischemia as a result of radiosurgery. Additionally, there were no cases of malignant transformation of an existing meningioma nor radiation related tumor development.

Overall outcome after stereotactic radiosurgery

Favorable outcome (tumor control along with neurological stability or improvement) was achieved in 87 % of patients and unfavorable outcome in 13 %. Of those with an unfavorable outcome, 1.7 % exhibited both tumor progression and neurological decline. Univariate predictors of favorable outcome included female gender, no prior surgery, no prior radiotherapy, decreasing time from symptom onset, presentation, decreasing volume, decreasing maximal dose, and increasing isodose line. Patients with a history of surgery before radiosurgery were 1.8 times more likely to have an unfavorable outcome in univariate analysis (95 % CI 1.07–3.01, $p = 0.026$), but prior resection was not an independent predictor of outcome. Patients with a tumor volume greater than 10 cc were 2.3 times more likely to have an unfavorable outcome (95 % CI 1.03–5.10, $p = 0.041$). The rates of favorable outcome for those patients with a tumor volume greater than versus less than 10 cc were 73.9 and 55.2 %, respectively. Multivariate predictors of favorable outcome included decreasing volume (OR 0.92; 95 % CI 0.87–0.97, $p = 0.003$), female gender (OR 0.37; 95 % CI 0.15–0.89, $p = 0.027$), no prior radiation therapy (OR 0.03; 95 % CI 0.01–0.36, $p = 0.006$), and decreasing maximal dose (OR 0.92; 95 % CI 0.96–0.98, $p = 0.010$) (Table 7).

Regarding a favorable outcome, the optimal dose breakpoint occurred with a maximal dose of 31 Gy. Patients treated with a maximal dose greater than 31 Gy were 77 % less likely to achieve a favorable outcome (OR 0.33; 95 % CI 0.14–0.80, $p = 0.014$). Twenty-two of 200 patients (11.0 %) treated with a maximal dose less than or equal to 31 Gy had unfavorable outcome versus 9 of 33 patients (27.3 %) patients treated with a maximal dose greater than 31 Gy ($p = 0.011$). When controlling for independent predictors of favorable outcome (i.e. tumor volume, female gender, no prior radiation therapy), those treated with a maximal dose above 31 Gy were 84 % less likely to achieve a favorable outcome than those treated with a maximal dose of 31 Gy or less (95 % CI 0.06–0.44, $p < 0.001$).

Discussion

Symptomatic petroclival meningiomas are among the most difficult skull base tumors to successfully manage. Their various growth pattern and the adjacent neurovascular structures contribute to the difficulty in obtaining long-term tumor control while avoiding neurological morbidity and mortality. The management paradigm of deep-seated skull base tumors, such as petroclival meningiomas, has generally shifted away from attempts at aggressive gross total surgical resection to a more tempered approach. The goal of the initial subtotal surgical debulking is to decompress critical structures such as the brainstem, cranial nerves, and basilar artery complex in order to facilitate subsequent radiosurgery for residual tumor [26].

Surgical resection

Complete surgical resection may be curative; however, the morbidity and mortality associated with surgical resection of petroclival meningiomas can be very high. Nanda et al. [28] reported 50 petroclival meningioma patients who underwent surgical resection and indicated that 44 % of patients sustained new postoperative cranial neuropathies including 14 % who suffered permanent cranial nerve palsies. Additional postoperative complications included hydrocephalus requiring cerebrospinal fluid (CSF) diversion (16 %), CSF leak (4 %), and wound dehiscence (2 %). Of the 31 patients with at least 6 months radiologic follow-up (mean 22 months), 19 % had tumor progression or recurrence at a median time to recurrence of 84 months [28].

Stereotactic radiosurgery in the management of petroclival meningiomas

The present multicenter study comprises the largest petroclival meningioma SRS series reported. GKRS affords excellent tumor control rates of 93 % at 5 years and 84 % at 10 years. Additionally, there was a 21 % decrease in mean tumor volume. In the current study and based upon multivariate analysis, predictors of tumor progression were: (1) increased time from symptom onset, (2) prior radiation therapy, (3) increased tumor volume, and (4) decreased maximal dose. Previously irradiated meningiomas have been shown to be more radioresistant to radiosurgery [29]. It remains unclear whether a subset of meningiomas are in fact more radioresistant or that prior radiation reduces susceptibility to additional ionizing radiation. Alternately, this finding may be an artifact of selecting and delivering a reduced radiosurgical dose to previously irradiated meningiomas.

Larger tumor volumes hinder the ability to deliver the optimal prescription dose to the tumor margin while at the same time delivering a dose within the tolerance of adjacent structures such as the brainstem or adjacent neurovascular structures [30]. A lower marginal dose will correspondingly reduce the odds of successful tumor control [31]. However, the upper limit of the tumor margin radiosurgical dose must be adjusted by the adjacent brainstem and cranial nerves. The present study found that a maximal dose of less than or equal to 31 Gy was associated with a favorable outcome. If a 50 % isodose line is adopted (mean 49 Gy in present study), an optimal margin dose to a WHO grade I petroclival meningioma would be approximately 15 Gy. Prior studies have also found similar dose regimens to provide an effective and safe outcome after single session radiosurgery [31–33]. Although evaluation of previously treated patients may provide some guidelines for GKRS treatment planning, both patient and tumor characteristics for each patient must be evaluated individually to determine the optimal strategy. Increasing dose increases the chances of achieving tumor control, but this must be balanced against the risk of radiation induced injury associated with increasing radiosurgical margin dose.

Cranial nerve risks to GKRS

Over a relatively long mean follow-up period of 71 months, we observed a 6 % rate of neurological deterioration. CN VII was the most common baseline deficit but new or worsening CN V function was the most common during the post-SRS period (Table 4). Also, multivariate analysis identified dysequilibrium symptoms such as dizziness and imbalance ($p = 0.015$) and alterations in visual function or movement ($p = 0.012$) at presentation as independent predictors of delayed clinical deterioration. Dizziness and imbalance are typically the result of dysfunction of the cerebellar pathways traversing the brainstem although damage to the vestibular portion of cranial nerve VIII may also result in similar symptoms. Ocular motility dysfunction may result from compression of the abducent nerve, and motor impairment may be secondary to compression of the cerebral peduncles or the corticospinal tracts as they pass through the pons. It is likely that patients with larger tumors, especially those with neurological dysfunction at the time of presentation, are more likely to develop new or worsened neurological morbidity after SRS. Patients with tumor progression after radiosurgery were at increased risk for neurological decline ($p = 0.017$).

The rate of favorable outcome (tumor control and stabilization or improvement in neurological function) in this study was 87 %. From the multivariate analysis of this study, increased tumor volume and prior radiation therapy were statistically associated with an unfavorable outcome after GKRS (Table 5). Female patients were more likely to have a favorable outcome after GKRS. While we do not know to what extent estrogen and progesterone effects may have led to this finding, the observation bears further investigation. For patients with large, symptomatic petroclival tumors, cytoreductive surgery prior to radiosurgery may be beneficial if additional morbidity can be avoided. Both tumor progression and toxicity associated with SRS after prior radiation therapy lead to increased risk of unfavorable outcomes after SRS.

In a single center GKRS study of 137 patients with intracranial meningiomas, a tumor volume over 10 cc was the only independent predictor of poorer survival [34]. Roche et al. evaluated the GKRS outcomes for 32 patients with petroclival meningiomas [21]. The rate of tumor control over a mean following period of 53 months was 100 %, and the rate of favorable outcome was 94 %. These authors did note that two patients suffered stroke symptoms related to pontine infarcts after radiosurgery. Flannery et al. reported outcomes for a series of 168 petroclival meningioma patients with a median follow-up of 72 months after GKRS [35]. Those patients are also included in the present report. The rates of progression-free survival at 5 and 10 years were 91 and 86 %, respectively, which are similar to the tumor control rates reported in this study. The rate of neurological deterioration was 15 %, and tumor volumes of at least 8 cc and male gender were significant predictors of tumor progression.

Study limitations

Our multicenter study is the largest radiosurgery series of petroclival meningioma to date. The large number of

patients with significant radiologic and clinical follow-up allows detailed statistical analysis for predictors or tumor progression, neurological dysfunction, and overall favorable outcome. However, this study remains limited by its retrospective nature and therefore by the selection and treatment biases of the physicians and institutions involved. The treatment period of this study spans over 20 years. In that time, GKRS technology and techniques have evolved accordingly to improve the accuracy of radiosurgical targeting. We are unable to reliably account for the effect of changes in GKRS units, refinement of dose planning and dose selection, and targeting strategies on the reported outcomes. While we excluded all patients with a known histological diagnosis of an aggressive meningioma 55 % of the tumors in this study were treated on the basis of neuroimaging features and clinical characteristics alone. Therefore, the slight possibility remains that a small fraction of tumors treated with GKRS were WHO grade II or III meningiomas or were extra-axial skull base tumors other than meningiomas, such as schwannomas.

Conclusion

GKRS for petroclival meningiomas is associated with high rates of long-term tumor control. Those with a smaller tumor volume and no radiation therapy are most likely to have favorable outcomes. In addition, certain presenting symptoms such as cerebellar, visual, facial, and motor dysfunction should be considered when selecting patients as they portend a greater risk for an unfavorable outcome after radiosurgery.

Conflict of interest Dr. Lunsford is a consultant for and stockholder in Elekta AB.

References

1. Al-Mefty O, Fox JL, Smith RR (1988) Petrosal approach for petroclival meningiomas. Neurosurgery 22:510–517
2. Ammirati M, Samii M (1992) Presigmoid sinus approach to petroclival meningiomas. Skull Base Surg 2:124–128
3. Bambakidis NC, Kakarla UK, Kim LJ, Nakaji P, Porter RW, Daspit CP, Spetzler RF (2007) Evolution of surgical approaches in the treatment of petroclival meningiomas: a retrospective review. Neurosurgery 61:202–209; discussion 209–211
4. Carvalho GA, Matthies C, Tatagiba M, Eghbal R, Samii M (2000) Impact of computed tomographic and magnetic resonance imaging findings on surgical outcome in petroclival meningiomas. Neurosurgery 47:1287–1294; discussion 1294–1285
5. Couldwell WT, Fukushima T, Giannotta SL, Weiss MH (1996) Petroclival meningiomas: surgical experience in 109 cases. J Neurosurg 84:20–28
6. Diluna ML, Bulsara KR (2011) Surgery for petroclival meningiomas: a comprehensive review of outcomes in the skull base surgery era. Skull Base 20:337–342
7. Erkmen K, Pravdenkova S, Al-Mefty O (2005) Surgical management of petroclival meningiomas: factors determining the choice of approach. Neurosurg Focus 19:E7
8. Flannery TJ, Kano H, Lunsford LD, Sirin S, Tormenti M, Niranjan A, Flickinger JC, Kondziolka D (2009) Long-term control of petroclival meningiomas through radiosurgery. J Neurosurg 112:957–964
9. Goel A (1999) Extended lateral subtemporal approach for petroclival meningiomas: report of experience with 24 cases. Br J Neurosurg 13:270–275
10. Hitselberger WE, Horn KL, Hankinson H, Brackmann DE, House WF (1993) The middle fossa transpetrous approach for petroclival meningiomas. Skull Base Surg 3:130–135
11. Ichimura S, Kawase T, Onozuka S, Yoshida K, Ohira T (2008) Four subtypes of petroclival meningiomas: differences in symptoms and operative findings using the anterior transpetrosal approach. Acta Neurochir 150:637–645
12. Kano H, Awan NR, Flannery TJ, Iyer A, Flickinger JC, Lunsford LD, Kondziolka D (2010) Stereotactic radiosurgery for patients with trigeminal neuralgia associated with petroclival meningiomas. Stereotact Funct Neurosurg 89:17–24
13. Kawase T, Shiobara R, Toya S (1994) Middle fossa transpetrosal-transtentorial approaches for petroclival meningiomas selective pyramid resection and radicality. Acta Neurochir 129:113–120
14. Li MH, Hong T, Li YY, Zhou DW, Zeng EM, Xu GS (2010) Surgical management of petroclival meningiomas invading into cavernous sinus. Zhonghua Yi Xue Za Zhi 90:295–297
15. Little KM, Friedman AH, Sampson JH, Wanibuchi M, Fukushima T (2005) Surgical management of petroclival meningiomas: defining resection goals based on risk of neurological morbidity and tumor recurrence rates in 137 patients. Neurosurgery 56:546–559 discussion 546–559
16. Mathiesen T, Gerlich A, Kihlstrom L, Svensson M, Bagger-Sjoback D (2007) Effects of using combined transpetrosal surgical approaches to treat petroclival meningiomas. Neurosurgery 60:982–991; discussion 991–982
17. Nanda A, Javalkar V, Banerjee AD (2010) Petroclival meningiomas: study on outcomes, complications and recurrence rates. J Neurosurg 114:1268–1277
18. Natarajan SK, Sekhar LN, Schessel D, Morita A (2007) Petroclival meningiomas: multimodality treatment and outcomes at long-term follow-up. Neurosurgery 60:965–979; discussion 979–981
19. Park CK, Jung HW, Kim JE, Paek SH, Kim DG (2006) The selection of the optimal therapeutic strategy for petroclival meningiomas. Surg Neurol 66:160–165; discussion 165–166
20. Ramina R, Neto MC, Fernandes YB, Silva EB, Mattei TA, Aguiar PH (2008) Surgical removal of small petroclival meningiomas. Acta Neurochir 150:431–438; discussion 438–439
21. Roche PH, Pellet W, Fuentes S, Thomassin JM, Regis J (2003) Gamma knife radiosurgical management of petroclival meningiomas results and indications. Acta Neurochir (Wien) 145:883–888 discussion 888
22. Samii M, Gerganov V, Giordano M, Samii A (2010) Two step approach for surgical removal of petroclival meningiomas with large supratentorial extension. Neurosurg Rev 34:173–179
23. Sekhar LN, Wright DC, Richardson R, Monacci W (1996) Petroclival and foramen magnum meningiomas: surgical approaches and pitfalls. J Neurooncol 29:249–259
24. Spallone A, Makhmudov UB, Mukhamedjanov DJ, Tcherekajev VA (1999) Petroclival meningioma. An attempt to define the role of skull base approaches in their surgical management. Surg Neurol 51:412–419; discussion 419–420
25. Tatagiba M, Samii M, Matthies C, Vorkapic P (1996) Management of petroclival meningiomas: a critical analysis of surgical treatment. Acta Neurochir Suppl 65:92–94

26. Zentner J, Meyer B, Vieweg U, Herberhold C, Schramm J (1997) Petroclival meningiomas: is radical resection always the best option? J Neurol Neurosurg Psychiatry 62:341–345
27. Subach BR, Lunsford LD, Kondziolka D, Maitz AH, Flickinger JC (1998) Management of petroclival meningiomas by stereotactic radiosurgery. Neurosurgery 42:437–443; discussion 443–435
28. Nanda A, Javalkar V, Banerjee AD (2011) Petroclival meningiomas: study on outcomes, complications and recurrence rates. J Neurosurg 114:1268–1277
29. Pollock BE, Stafford SL, Link MJ, Garces YI, Foote RL (2012) Stereotactic radiosurgery of World Health Organization grade II and III intracranial meningiomas: treatment results on the basis of a 22-year experience. Cancer 118:1048–1054
30. Kondziolka D, Flickinger JC, Lunsford LD (2008) The principles of skull base radiosurgery. Neurosurg Focus 24:E11
31. Starke RM, Williams BJ, Hiles C, Nguyen JH, Elsharkawy MY, Sheehan JP (2012) Gamma knife surgery for skull base meningiomas. J Neurosurg 116:588–597
32. Kollova A, Liscak R, Novotny J Jr, Vladyka V, Simonova G, Janouskova L (2007) Gamma Knife surgery for benign meningioma. J Neurosurg 107:325–336
33. Pollock BE, Stafford SL, Link MJ, Brown PD, Garces YI, Foote RL (2012) Single-fraction radiosurgery of benign intracranial meningiomas. Neurosurgery 71:604–612; discussion 613
34. DiBiase SJ, Kwok Y, Yovino S, Arena C, Naqvi S, Temple R, Regine WF, Amin P, Guo C, Chin LS (2004) Factors predicting local tumor control after gamma knife stereotactic radiosurgery for benign intracranial meningiomas. Int J Radiat Oncol Biol Phys 60:1515–1519
35. Flannery TJ, Kano H, Lunsford LD, Sirin S, Tormenti M, Niranjan A, Flickinger JC, Kondziolka D (2010) Long-term control of petroclival meningiomas through radiosurgery. J Neurosurg 112:957–964