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New-onset facial spasm is associated with treatment failure after radiosurgery in vestibular schwannoma

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Abstract

Background. New-onset facial spasm (NOFS) has been reported to be a post-therapeutic side effect, when treating vestibular schwannoma (VS) with radiosurgery (SRS) and has been linked to post-treatment pseudoprogression. The aim of this study was to identify risk factors for developing NOFS after SRS treatment and to investigate NOFS as a clinical parameter for radiographic tumor response in VS.

Methods. This study included all consecutive patients of $N = 1{,}998$ between 2004 and 2020, which were treated with SRS identified by a prospective registry. Patient and tumor characteristics (ie, sex, age, tumor extension and size, and intracanalicular extension) were analyzed retrospectively. Statistical testing was performed with R Studio.

Results. The incidence of NOFS was 5% overall. In total, 62% were permanent NOFS, whereas 39% recovered spontaneously between 4 and 34 months, postinterventionally. The incidence of NOFS was unrelated to tumor volume—however, previous SRS increased the incidence of NOFS to 20%. In primary SRS therapy, facial spasm was associated with a higher recurrence rate compared to non-NOFS patients in the Kaplan–Meier analysis ($P < 0.001$). Tumor control decreased with increasing tumor size. The rate of pseudoprogression was higher in the group of transient NOFS at 39% compared to permanent NOFS at 18% ($P = 0.032$).

Conclusions. The risk of NOFS was significantly higher in recurrent compared to primary treatment (20% vs 5%) and the majority of NOFS was permanent. The incidence of permanent NOFS was significantly associated with treatment failure. Temporary NOFS was associated with pseudoprogression. Future analysis comparing the risk profile of either treatment option should include facial spasm as a significant VS-related postinterventional symptom. Patients with postinterventional NOFS should be followed-up long-term for higher risk of treatment failure.

Key Points

- The incidence of new-onset facial spasm (NOFS) after Gamma Knife radiosurgery is 5%.
- The incidence of NOFS is significantly higher in recurrent vestibular schwannoma (VS) with 20%.
- New-onset facial spasm is associated with long-term treatment failure after stereotactic radiosurgery in VS.

As vestibular schwannoma (VS) are intracranial neoplasm with a usually benign growth behavior, functional outcome and quality of life are important aspects besides tumor control, when discussing the choice of treatment (radiosurgery (SRS) versus microsurgery) in VS.[1–3] New-onset facial spasm (NOFS) has been reported to be a relevant post-therapeutic side effect

Importance of the Study

To our best knowledge, this is the first study to focus on postinterventional new-onset facial spasm (NOFS) after stereotactic radiosurgery (SRS), that is Gamma Knife radiosurgery. The incidence of NOFS is significantly higher in the treatment of recurrent VS, which have received previous SRS. Moreover, in primary SRS, NOFS was associated with a higher incidence of treatment failure after SRS and a shorter time to recurrence. Our data show that NOFS is a surrogate marker for treatment failure and tumor expansion after SRS therapy in the treatment of primary VS. Patients who develop NOFS should be followed up in the long term.

with an incidence of 5%, when treating VS with SRS.[4] It has been reported that the burden of facial hemispasm—whether they are VS related or not—can be very high in a patient's daily life.[5] However, facial function preservation in the care of VS is pre-dominantly measured and investigated in motoric function (eg, House–Brackman (HB)) and NOFS is more commonly neglected in analyses.

The incidence of radiographic tumor recurrence/progression in VS has been reported to be higher after SRS compared to gross total microsurgical tumor resection.[4] While the tumor response of VS surgery can be easily quantified in postoperative magnetic resonance imaging (MRI), we are lacking clinical or radiographic parameters, which predict positive or negative tumor response after SRS. Nagano et al interpreted the incidence of hemifacial spasm as a clinical sign of nerve deterioration, when radiographic tumor response to Gamma Knife radiosurgery (GKS) was transient volume expansion (also known as pseudoprogression).[6] Therefore, the aim of this study was (1) to identify risk factors for developing NOFS after GKS treatment, (2) to characterize the course and recovery of NOFS, and (3) to investigate NOFS as a clinical parameter for radiographic tumor response in VS after GKS.

Methods

Study Design and Patient Cohort

This is a retrospective cohort study. Study reporting followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. Patients were identified by a prospectively kept registry. Data were then retrospectively collected. All VS patients in the SRS cohort received Gamma Knife radiosurgery (Leksell Gamma Knife Perfexion—Elekta AB, Stockholm, Sweden) with a prescription dose of 12–13 Gy (Gray) to the 65% isodose line. The local ethics committee approved this analysis and was according to the ethical standards laid down in the Declaration of Helsinki for research involving human subjects.

Data Collection

Tumor size was classified by Koos classification. Recurrence-free survival (RFS) was assessed radiographically by contrast-enhanced MR imaging.[7,8] Tumor volumetry was carried out at each follow-up (FU) timepoint using slice-by-slice manual contouring. The criteria for tumor progression was progredient growth in gadolinium contrast-enhanced MRI (radiographic tumor control). The phenomenon of pseudoprogression after GKS of VS included patients with post-therapeutic tumor volume increase within 2 years, with following decrease smaller than initial tumor volume is tumor volume stability.[9] The tumor extension into the internal auditory canal was classified according to Ohata et al on the contrast-enhanced MRI.[10]

Statistical Analysis

Statistical analysis was performed in R Studio (Version 1.2) using descriptive statistics. To compare the nonnumeric parameters of both groups, the chi-square test was applied. For numeric parameters, Welch's two-sample *t*-test was used. RFS was estimated using the Kaplan–Meier method and compared between cases and controls using a log-rank test. The length of FU for RFS was calculated from the date of radiosurgical intervention to the date of either recurrence or the last clinical visit. Significance was defined as the probability of a two-sided type 1 error being < 5% ($P < 0.05$). Data are presented as mean ± standard deviation (SD) if not indicated otherwise.

Results

Study Cohort

From 2004 and 2020, N = 1,998 VS patients were treated with GKS in this center. A total of 95% (N = 1,897/1,998) were treated for the first time (primary GKS), whereas 5% (N = 100/1,998) received recurrent GKS for tumor recurrence (Figure 1). Overall, 16% (N = 316/1,998) had received previous surgical treatment.

Mean patient age was 59(± 13.73) years in the overall cohort with a significantly younger subgroup of NOFS patients with 56(± 12.15) years compared to 59(± 13.79) years in patient with no NOFS (*CONTROL*) ($P = 0.034$). NOFS was significantly associated with previous GKS and surgical treatment ($P < 0.001$ and $P = 0.036$, respectively) (Table 1). Tumor volume was insignificant in either subgroup ($P = 0.511$). The incidence of NOFS was significantly higher in Koos III VS tumors ($P = 0.011$) and significantly lower in Koos I VS tumors ($P = 0.031$) (Table 1 and Figure 2A).

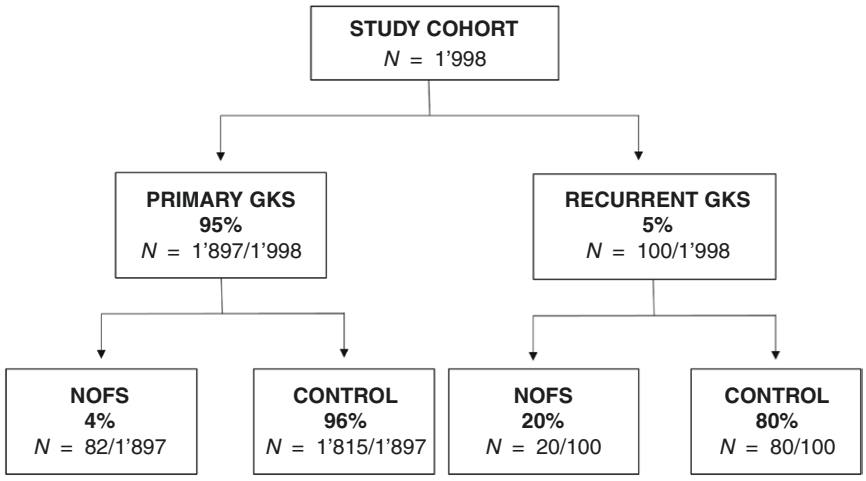

Figure 1. Patient cohort flowchart.

New-Onset Facial Spasm

Mean time of the first initial manifestation of NOFS was 5 months (range 3 weeks–6 months). Of all patients with NOFS, 35% was reversible and therefore transient—while the majority (65%) remained symptomatic at the last follow-up. Age was statistically insignificant, when comparing NOFS with transient (53(± 11.96) years) and persistent (56(± 11.19) years) character ($P = 0.196$). However, patients with transient NOFS had a significantly higher initial tumor volume with 1.75(± 2.31) cm^3 compared to patients with persistent NOFS with 0.91(± 0.78) cm^3 ($P = 0.008$). The incidence of NOFS was 3% ($N = 12/402$) in Koos I, 4% ($N = 39/877$) in Koos II, 7% ($N = 43/610$) in Koos III, and 7% ($N = 8/108$) in Koos IV. Moreover, the incidence of NOFS was proportional to tumor extension into the internal auditory canal (classified in OHATA), with a higher risk of NOFS, when tumor extension was more extensive (Figure 2C). There was no significant difference in the incidence of NOFS depending on the prescription dose of 13 or 12 Gray ($P = 0.429$).

NOFS was significantly associated with previous treatment ($P < 0.001$): however, only in previous SRS with an incidence of 20% of NOFS ($P = 0.001$) and not in previous surgery with 7% incidence ($P = 0.640$).

SRS-Related Tumor Response

The conformity index (Paddick) was comparable in all subgroups (CONTROL and NOFS). The same applied to other radiosurgical parameters shown in Table 2. A Kaplan–Meier analysis on RFS in the overall cohort is shown in Figure 3 according to different tumor size. The best tumor control was ensured in Koos I tumors with an incidence of treatment failure/tumor progression of only 4% (mean-time-to-progression 37(± 36.26) months), treatment failure was 10% (mean-time-to-progression 44(± 7.05) months) in Koos II, 16% (mean-time-to-progression 43(± 37.28) months) in Koos III, and 17% (mean-time-to-progression 34(± 44.80) months) in Koos IV.

The incidence of tumor progression in patients with no NOFS (CONTROL) was at 10% ($N = 185/1,898$), these numbers were significantly higher in the NOFS cohort with 39% ($N = 40/102$). In a Kaplan–Meier analysis, this phenomenon was only present in primary SRS treatment and not in recurrent SRS. Kaplan–Meier analysis is shown in Figure 3. The rate of tumor progression was independent of whether NOFS was transient or permanent with 39% ($N = 26/66$) of tumor progression in permanent and 39% ($N = 14/36$) in transient NOFS ($P = 1$). The Kaplan–Meier analysis stratified by permanent and transient NOFS is shown in Figure 3.

The rate of pseudoprogression was 17% ($N = 333/1,998$) overall, with a significantly increased rate in the NOFS cohort of 25% ($N = 26/102$) compared to 16% ($N = 307/1,896$) in patients with CONTROL ($P = 0.002$). The rate of pseudoprogression was higher in the group of transient NOFS at 39% ($N = 14/36$) compared to permanent NOFS at 18% ($N = 12/66$) ($P = 0.032$).

Discussion

Long-term treatment failure of VS after SRS was 11% overall, but increased significantly with increasing tumor size. NOFS is a SRS therapy-related side effect with an overall incidence of 5%. The risk of NOFS was significantly higher in recurrent SRS with 20% compared to primary SRS with 4%. With 65%, the majority of NOFS was permanent, whereas 35% was temporary. The incidence of NOFS was significantly associated with the incidence of tumor progression overall, which was also reflected in the Kaplan–Meier analysis. Temporary NOFS was associated with post-therapeutic temporary tumor volume increase (pseudoprogression).

Most commonly, the facial function is measured in motoric function and therefore HB. Facial spasm has been more neglected in the past, when reporting post-treatment functional, facial outcome.[2,4,11] In a large comparative study

Table 1. Tumor and Patient Demographics of Patient With and Without NOFS. Values are Presented as the Number of Patients (%) Unless Indicated Otherwise. Significant *P* Values (< 0.05) are Highlighted in Bold

	All (N = 1,998)	Control (N = 1,896)	NOFS (N = 102)	P Value
Age	58.56 (± 13.73)	58.70 (± 13.79)	55.74 (± 12.15)	**0.034***
Female	1,130 (57)	1,064 (56)	66 (65)	0.101
Neurofibromatosis type II	32 (2)	31 (2)	1 (1)	1
Tumor size (Koos classification)				
Koos I	402 (20)	390 (21)	12 (12)	**0.031***
Koos II	877 (43)	838 (44)	39 (38)	0.261
Koos III	610 (31)	567 (30)	43 (42)	**0.011***
Koos IV	109 (6)	101 (5)	8 (8)	0.262
Tumor progression	225 (11)	185 (10)	40 (39)	**<0.001***

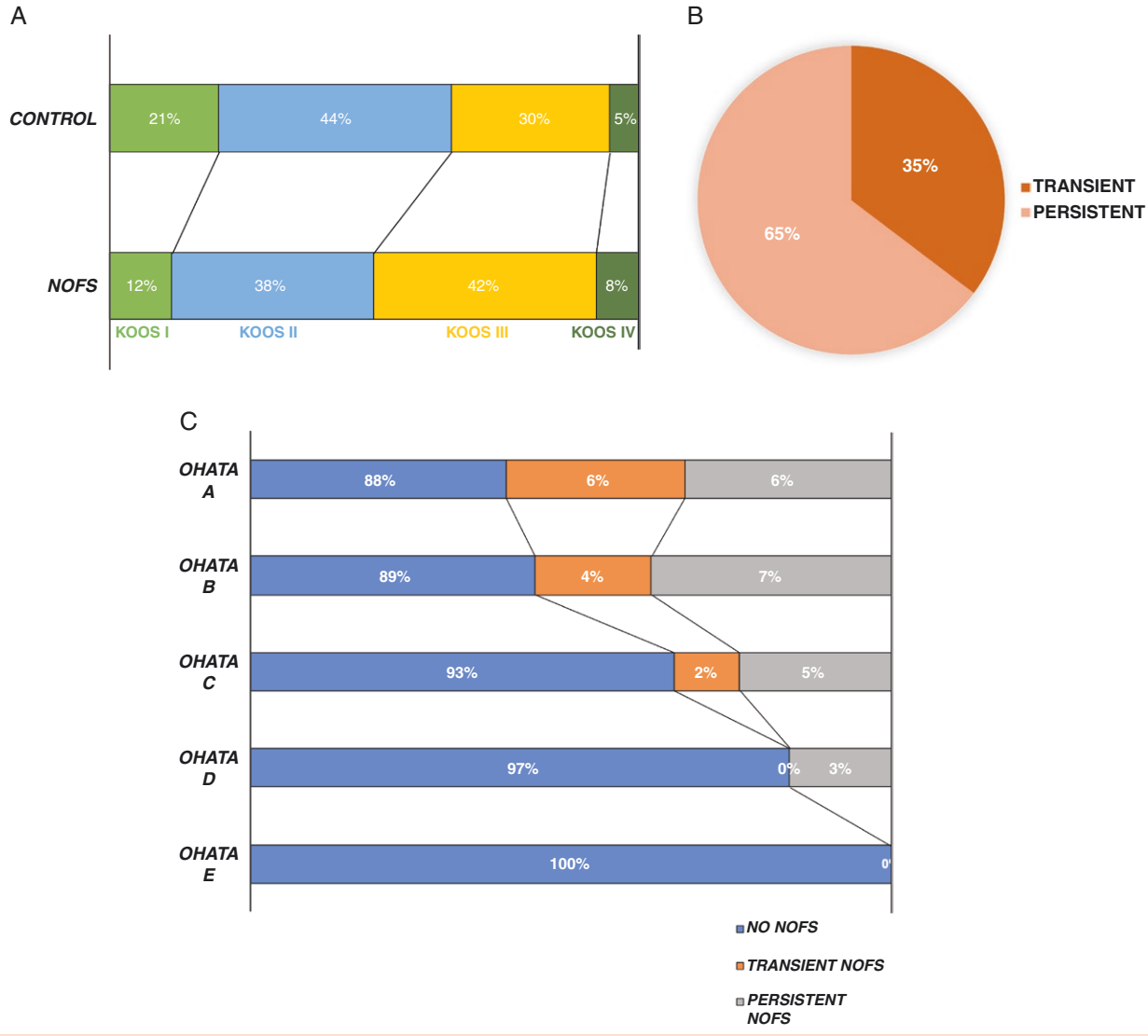

Figure 2. (A) shows the difference in size distribution in NOFS compared to CONTROL. (B) shows a pie chart with the proportions of transient and persistent NOFS after SRS treatment of VS. (C) shows the compared incidence of transient and persistent NOFS in different OHATA classes.

Table 2. Treatment Details in Both Groups (CONTROL and NOFS)

	CONTROL (*N* = 1,896)	NOFS (*N* = 102)	*P* Value
Tumor volume (ccm)	1.35 (± 1.88)	1.57 (± 1.72)	0.228
Conformity index (Paddick)	0.76 (± 0.12)	0.80 (± 0.26)	0.127
Therapeutical dose (Gy)	12.81 (± 0.45)	12.85 (± 0.36)	0.256
Isodose (%)	62.16 (± 5.28)	62.17 (± 5.36)	0.987
Maximal dose (Gy)	20.75 (± 1.72)	20.81 (± 1.77)	0.721
Mean dose (Gy)	16.35 (± 0.92)	17.06 (± 4.86)	0.242

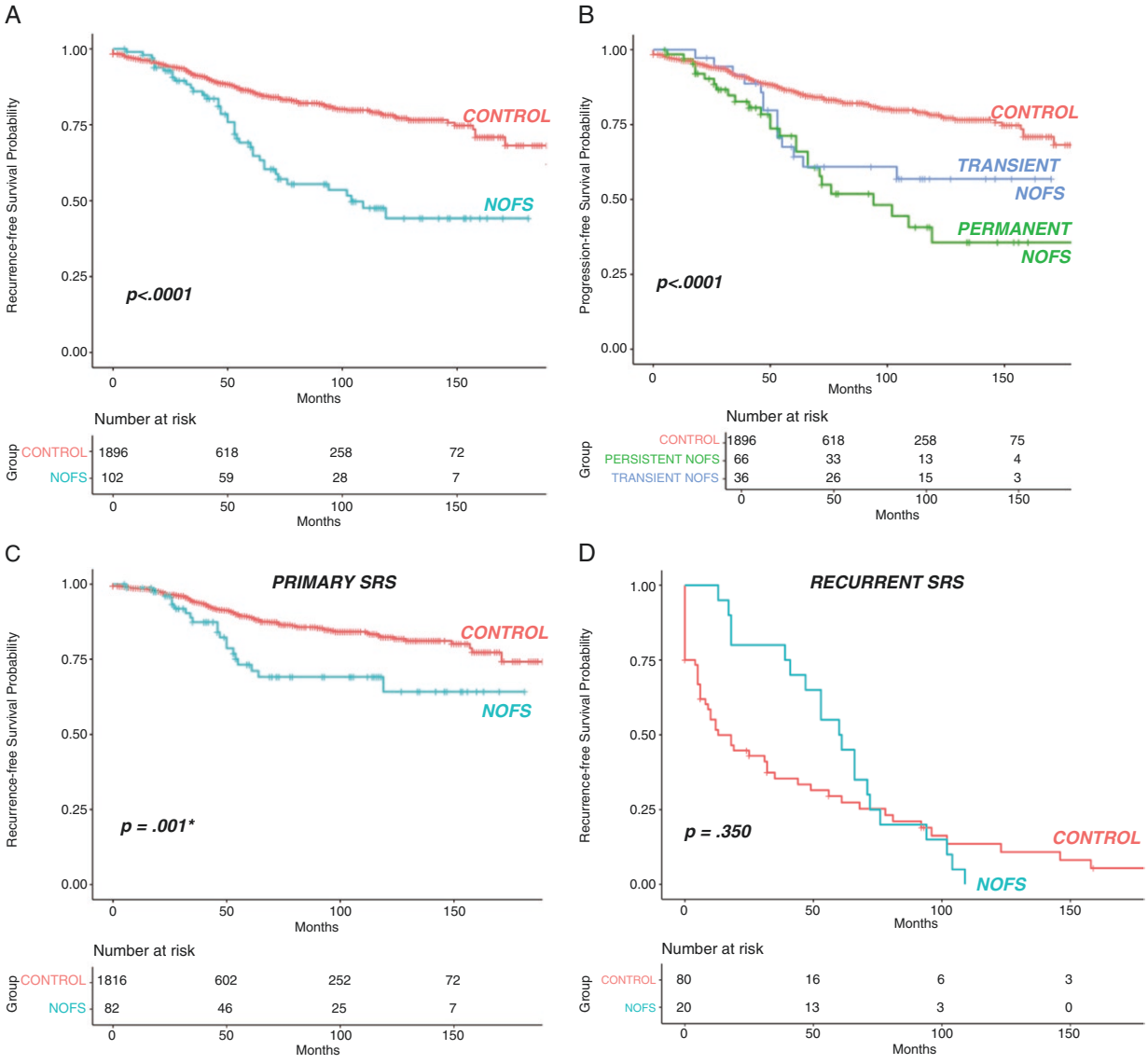

Figure 3. (A) shows a Kaplan–Meier analysis comparing progression-free survival in NOFS patients compared to CONTROL with no NOFS when treated with SRS. (B) shows a Kaplan–Meier analysis stratified by transient and permanent NOFS. (C) shows progression-free survival only in primary SRS. (C) shows Kaplan–Meier analysis in only recurrent SRS.

between SRS and microsurgery in 2023, Tatagiba et al described facial spasm not only as an SRS-related but SRS-specific side effect with an incidence of 0% in the surgically treated VS patients.[4] Other studies have reported a risk for NOFS after VS surgery.[12–14] However, the difference between Tatagiba et al and the other studies, which report

Table 3. Literature Review on NOFS (New-Onset Facial Spasm) Rate

Name	Year	Study Design	Patient Number	Modality	Treatment	NOFS Rate
Bennion[10]	2015	Retrospective	45	SRT	Primary treatment	8%
Boari[11]	2014	Retrospective	379	GKS	Primary treatment	1%
Collen[12]	2011	Retrospective	119	SRS and SRT	Primary and recurrent treatment	3%
Dzierzecki[13]	2020	Retrospective	136	GKS	Primary treatment	2%
Fu[14]	2018	Retrospective	38	GKS	Recurrent treatment	13%
Hayhurst[15]	2012	Retrospective	80	GKS	Primary treatment	1%
Iwai[16]	2008	Retrospective	248	GKS	Primary treatment	4%
Karam[17]	2013	Retrospective	37	CBK	Primary treatment	5%
Kim[18]	2017	Retrospective	235	GKS	Primary and recurrent treatment	11%
Lerner[19]	2020	Retrospective	133	GKS	Primary treatment	13%
Liscak[20]	2009	Retrospective	32	GKS	Primary treatment	9%
„	„	„	24	GKS	Recurrent treatment	13%
Litvack[21]	2003	Retrospective	129	GKS	Primary and recurrent treatment	6%
Nagano[6]	2008	Retrospective	100	GKS	Primary and recurrent treatment	17%
Hayhurst[9]	2012	Retrospective	75	GKS	Primary and recurrent treatment	1%
Noren[22]	1998	Retrospective	530	GKS	Primary treatment	2%
Régis[23]	2002	Prospective	500	GKS	Primary treatment	3%
Thomas[24]	2007	Prospective	34	fSRT	Primary treatment	12%
Our study	*2024*	*Retrospective*	*1,815*	*GKS*	*Primary treatment*	*4%*
„	„	„	*100*	*GKS*	*Recurrent treatment*	*20%*

Abbreviations: CBK = Cyber Knife; GKS = Gamma Knife radiosurgery; fSRT = fractionated stereotactic radiotherapy; *SRS = Stereotactic radiosurgery.

surgery-related NOFS is the surgical approach: Tatagiba et al only used the retrosigmoid approach for surgical resection, whereas surgery-related NOFS was reported in studies with a trans-labyrinthine approach for the surgical resection.[12–14] Therefore, a retrosigmoid approach, may protect postoperative NOFS compared to the middle fossa approach—possibly due to the ability to better surgically protect the facial nerve through a later visualization after tumor decompression through a posterior fossa approach.

NOFS in primary SRS was reported postinterventionally in between 1% and 13% in the current literature.[13,15–24] Concordant to our study, recurrent SRS was reported significantly with higher rates of NOFS.[21,25] However, NOFS was mostly not the primary outcome and the criteria for NOFS are not described in detail in these studies.[13,15–24,26] This is especially important, when discriminating NOFS from facial fasciculation (a milder form, which often occurs in recovering facial palsies). The lack of importance of NOFS in the study design may explain the large range of reported incidences in the literature as shown in Table 3 in a literature review. Postinterventional electrophysiological examination (electromyography) may improve future studies and their significance, when studying NOFS.

Though not thoroughly investigated in the past, tumor volume expansion—transient (pseudoprogression) or permanent (tumor progression) has been linked with positive NOFS status in the past.[6,27] Pollock et al reported NOFS to be temporary and not associated with tumor progression.[28]

Compared to other publications, our study included the largest group of patients (Table 3) in their analysis. Our uniquely large patient number of almost 2,000 cases allows the differentiation between transient and permanent NOFS in the form of subgroup analysis. In our study, transient NOFS was associated with pseudoprogression (therefore, transient tumor volume increase, which was more often presented in larger tumors), and permanent NOFS with continuous tumor progression/treatment failure. In both cases, NOFS was a clinical parameter for tumor volume expansion (transient and progressive). We therefore suggest permanent NOFS to be a clinical predictive parameter for failed SRS and these patients should receive long-term MRI follow-up for screening for treatment failure.

Even though the incidence of NOFS was significantly increased in VS patients, who received recurrent SRS, this particular group of patients did not present with a significantly higher rate of long-term tumor progression. This suggests a different pathophysiology of NOFS in recurrent SRS compared to NOFS in primary SRS: tumor volume increase (by pseudoprogression or continuous tumor progression/treatment failure) may cause local irritation and stretching of the facial nerve against the internal auditory canal in primary SRS, but repeated radiation may cause cranial nerve neuropathy as observed in the past.[27–30] In the latter case, NOFS is not the result of volume expansion and is not associated with tumor progression nor pseudoprogression.

This study shows that NOFS—if observed can be used as a clinical parameter for tumor volume expansion. Therefore, future analyses comparing the risk profile of either treatment option should include NOFS as a significant VS-related postinterventional symptom as a parameter to predict radiosurgical treatment response/failure.

The discussion on the choice of treatment (microsurgery, SRS, and combination therapy) is still ongoing.[31] The rate of successful tumor response after SRS has been reported to be different in different subgroups.[4] Therefore, biomarkers—may they be clinical, radiographic, or even liquid (in blood or cerebrospinal fluids), should be evaluated in the future to better predict treatment success in SRS in VS management. Finding nonresponder VS tumors to SRS may be the key to further refine the framework for personalized treatment decision in VS management.

Limitations of this Study

This study is limited by its nature of retrospective design. However, the patient cohort size is the largest ever published in the literature reporting on the question of NOFS. The large patient number of almost 2,000 patients allows detailed subgroup analysis in transient and permanent NOFS, which is a novelty considering the literature published in the past about the issue of NOFS. Future studies should investigate the bony anatomy of the internal auditory canal to investigate tumor expansion as a possible pathophysiology of the link between NOFS and volume increase.

Conclusion

Long-term treatment failure of VS after SRS was 11% overall but increased significantly with increasing tumor size. NOFS is a SRS-related side effect with an overall incidence of 5%. The risk for NOFS was significantly higher in recurrent treatment with an increased incidence of 20% compared to primary treatment with only 4%. The majority of NOFS was permanent, whereas 35% was temporary. The incidence of permanent NOFS was significantly associated with the incidence of tumor progression and therefore treatment failure, which was reflected in the Kaplan–Meier analysis. Temporary NOFS was associated with pseudoprogression. Future analysis comparing risk profiles of VS treatment options (microsurgery and radiosurgery) should include facial spasm as a significant VS-related postinterventional symptom. And patients with postinterventional NOFS should be followed up in the long-term to discover possible treatment failure.

Keywords

vestibular schwannoma | acoustic neuroma | outcome | stereotactic radiosurgery

LAY SUMMARY

Vestibular schwannomas are benign brain tumors that can cause hearing loss and balance problems. They can be treated with a focused radiation technique called stereotactic radiosurgery (SRS). Some patients experience a side effect called facial spasms after this treatment. The authors of this study wanted to learn how often facial spasms happen, what causes them, and if they are linked to changes in the tumor. To do this, they reviewed data from 1,998 patients treated with SRS. Their results showed that facial spasms occurred in about 5% of patients, and most cases were permanent. The size of the tumor that was treated did not seem to affect the risk of developing facial spasms. However, patients who had received SRS more than once were much more likely to develop facial spasms (in 20% of these patients). Patients with permanent facial spasms were also more likely to have their tumor continue to grow after treatment (=treatment failure).

Conflict of interest statement. The authors reported no conflict of interest.

Author Contributions

Conception and design: Tatagiba, Horstmann, Wang; Acquisition of data: Wang, Horstmann, van Eck; Analysis and interpretation of data: Wang, Tatagiba; Statistical analysis: Wang; Drafting the article: Wang; Critically revising the article: Naros, van Eck, Horstmann; Reviewed submitted version of the manuscript: Tatagiba.

Data Availability

All data and materials are available and can be provided upon reasonable request.

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