FLUCTUATING RESPONSE OF A CYSTIC VESTIBULAR SCHWANNOMA TO RADIOSURGERY: CASE REPORT

Amy R. de Ipolyi, M.D., Ph.D.
Department of Neurological Surgery, Brain Tumor Research Center, University of California at San Francisco, San Francisco, California

Isaac Yang, M.D.
Department of Neurological Surgery, Brain Tumor Research Center, University of California at San Francisco, San Francisco, California

Anne Buckley, M.D.
Department of Pathology, University of California at San Francisco, San Francisco, California

Nicholas M. Barbaro, M.D.
Department of Neurological Surgery, Brain Tumor Research Center, University of California at San Francisco, San Francisco, California

Steven W. Cheung, M.D.
Department of Otolaryngology-Head and Neck Surgery, University of California at San Francisco, San Francisco, California

Andrew T. Parsa, M.D., Ph.D.
Department of Neurological Surgery, Brain Tumor Research Center, University of California at San Francisco, San Francisco, California

Reprint requests:
Andrew T. Parsa, M.D., Ph.D., Department of Neurological Surgery, University of California, San Francisco, 505 Parnassus Avenue, Room M779, San Francisco, CA 94143-0112.
Email: parsaa@neurosurg.ucsf.edu

Received, August 13, 2007.
Accepted, February 8, 2008.

OBJECTIVE: A vestibular schwannoma (VS) is a benign tumor of the VIIIth cranial nerve that can often be treated by microsurgery or radiosurgery and demonstrates high tumor control rates. Radiosurgery is typically performed as gamma knife surgery (GKS), although other modalities are being applied with increasing frequency. A differentiating feature in responsiveness to microsurgery or GKS is whether the VS is cystic or solid. A cystic VS is less responsive to GKS than a solid VS, representing a challenging clinical problem. GKS treatment of a cystic VS usually results in sustained expansion, sustained regression, or transient expansion followed by sustained regression. In this article, we report an atypical fluctuating course of a cystic VS after GKS, ultimately requiring surgical intervention.

CLINICAL PRESENTATION: A 66-year-old woman presented with asymmetric hearing loss and tinnitus. Magnetic resonance imaging revealed a 2.0-cm unilateral cystic VS within the cerebellopontine angle.

INTERVENTION: After GKS with a 12-Gy dose to the 50% isodose line, the tumor expanded transiently to 3.2 cm and then regressed to 1.0 cm over the next 2 years. She presented several months later with new-onset dizziness, ataxia, and facial numbness. Magnetic resonance imaging revealed a 3.2-cm multicystic VS that compressed the brainstem. After microsurgical tumor excision, the patient's symptoms abated.

CONCLUSION: Our case report is a novel demonstration that a cystic VS that has regressed after GKS is still at risk for expansion. The mechanisms responsible for radiation-induced cystic tumor expansion have not been thoroughly elucidated. The risk of unpredictable tumor enlargement should be discussed with patients when considering GKS for cystic tumors.

KEY WORDS: Cystic vestibular schwannoma, Gamma knife radiosurgery, Microsurgery

Neurosurgery 62:E1164–E1165, 2008 DOI: 10.1227/01.NEU.0000297143.70840.82 www.neurosurgery-online.com

A vestibular schwannoma (VS) is a benign tumor of the VIIIth cranial nerve sheath. These tumors represent 10% of all newly diagnosed intracranial tumors and occur with a frequency of 2000 to 2500 new cases per year in the United States (8). The primary management options for patients with a VS include observation, microsurgical removal, and stereotactic radiosurgery (27). Radiosurgical techniques include single-session gamma knife surgery (GKS) and linear-accelerator (LINAC) technologies, as well as fractionated radiosurgery such as the CyberKnife (Accuray, Sunnyvale, CA) (9). Of these, GKS is the best studied and most widely used (27). Observation is typically reserved for elderly patients who do not experience mass effect because half of all VS increase in size over 3 years (9, 13). In recent decades, surgery has improved as a treatment modality of VS with the introduction of microsurgical techniques and instrumentation, advanced intraoperative monitoring of the facial nerve, and brainstem evoked potential recording (3), whereas GKS has also improved with the introduction of more sophisticated and accurate radiation dosing (11). Compared with microsurgery, potential advantages of GKS include faster recovery, reduced cost, and minimized morbidity and mortality (15, 16). Microsurgical removal of a VS is technically challenging, with surgeons requiring 200 or more procedures to acquire expertise (15). For these reasons, GKS has emerged as a preferred treatment at many centers.

It remains to be determined whether GKS is as effective as microsurgery for the treatment of VS. Tumors that fail radiosurgery may become more difficult to manage with salvage surgery because of tumor fibrosis, loss of the peritumoral arachnoidal plane, adjacent scarring, and an indistinct facial nerve-tumor capsule interface (14, 22). Radiosurgery is associated with a small risk for developing radiation-induced tumors (20). Another potential difficulty in managing VS with GKS is that, although most tumors shrink with time, a relatively large proportion of treated VS expand transiently before shrinking (10). In longitudinal studies, VS reach a peak expansion at 1 year after GKS before shrinking within 2 years (10). Transient enlargement of irradiated VS may reach 180% of the untreated tumor size within 1 year (28). These volumetric changes represent a clinical challenge requiring diligent surveillance and possible surgical intervention (10). In this report, we present a case of a cystic VS treated with GKS. The tumor enlarged transiently, then shrank considerably, and subsequently underwent expansion 2 years later, necessitating microsurgical removal. This case illustrates that shrinkage of a cystic VS treated with GKS does not preclude future expansion.

CASE REPORT

A 66-year-old woman presented with right hearing loss and tinnitus. Her medical history was noncontributory but included subclinical hyperthyroidism, breast cancer 30 years earlier treated with a right total mastectomy, and episodes of palpitations. A preoperative magnetic resonance imaging (MRI) scan with gadolinium showed a right cystic VS, manifested as a cerebellopontine angle extra-axial mass with extension into the right internal auditory canal causing slight canalicular expansion (*Fig. 1A*). The component within the cerebellopontine angle was primarily cystic with peripheral rim enhancement, and measured 1.3 × 2.0 × 1.7 cm; the tumor volume was approximately 4.3 cm^3. GKS was performed using a Leksell stereotactic frame (Elekta, Stockholm, Sweden), with a 12-Gy dose delivered to the 50% isodose line (*Fig. 2*), which has been shown to be the optimal dose range (12–13 Gy) for treating VS while reducing brainstem toxicity (8). The patient tol-

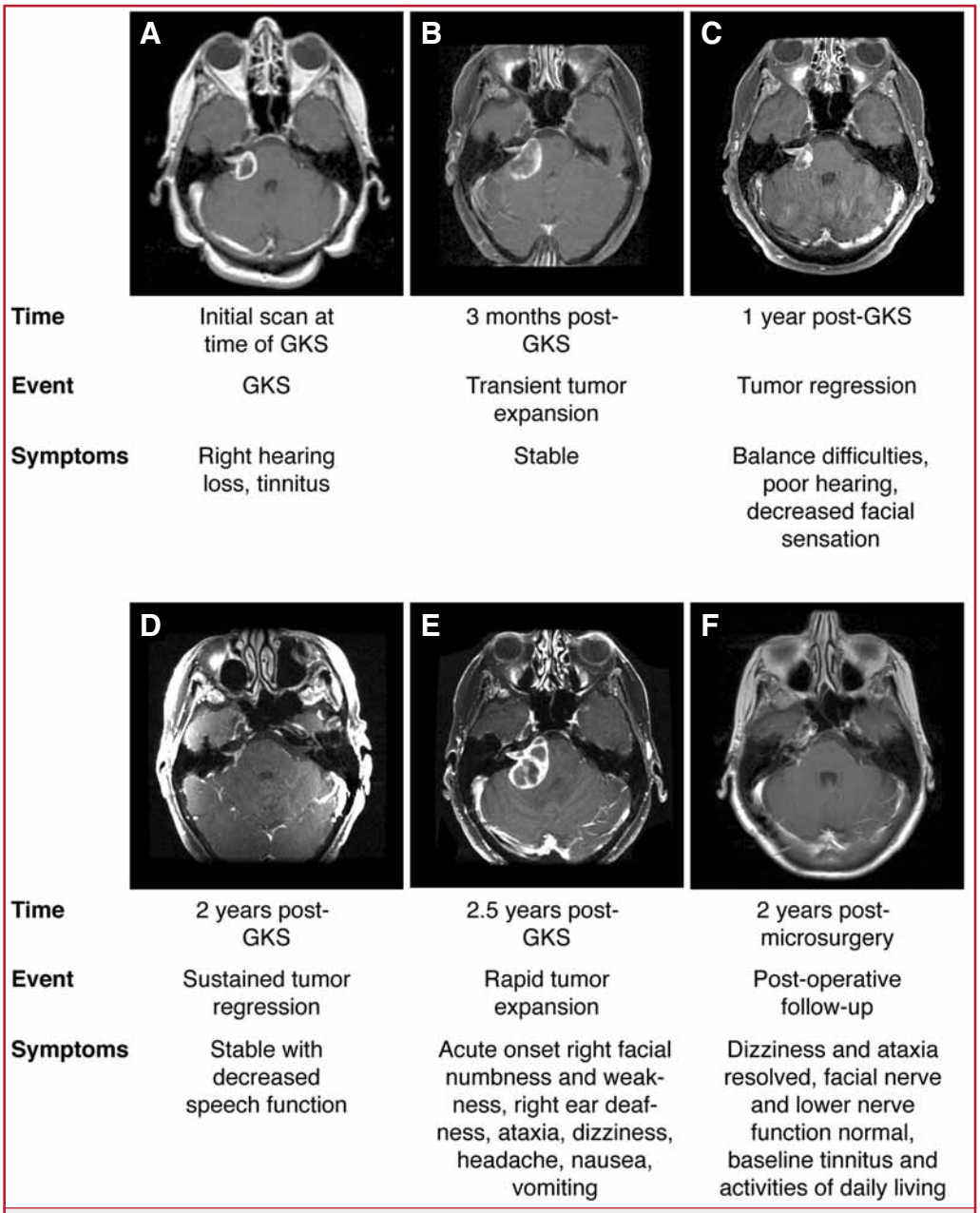

FIGURE 1. *Neuroimaging of the tumor before and after gamma knife surgery (GKS). Gadolinium-enhanced T1-weighted axial images of the patient's cystic vestibular schwannoma (VS) are presented with a timeline and associated therapeutic events and clinical presentation, and demonstrate fluctuating volume changes of cystic VS after GKS. Axial T1-weighted magnetic resonance imaging (MRI) with contrast obtained (**A**) at the initial time of GKS, (**B**) 3 months after GKS, (**C**) 1 year after GKS, (**D**) 2 years after GKS, (**E**) 2.5 years after GKS, and (**F**) 2 years after microsurgical resection of recurrence.*

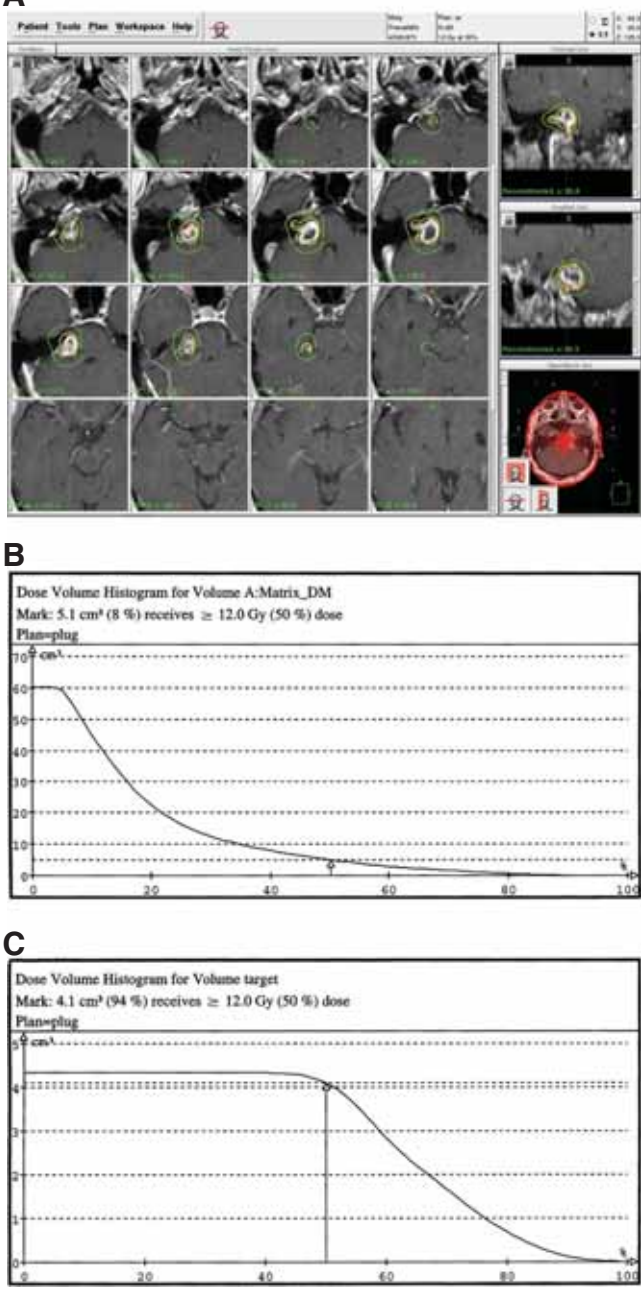

FIGURE 2. *Gamma knife treatment plan with dosing curves.* **A**, *target dosing using axial T1-weighted MRI scan with contrast.* **B**, *dose volume histogram for volume.* **C**, *dose volume histogram for volume target.*

erated the procedure well and, after brief monitoring, was discharged the same day. A follow-up MRI scan 3 months after radiosurgery revealed an increase in the size of the cerebellopontine mass to 2.5 × 3.2 × 1.9 cm, causing increased mass effect on the pons and right middle cerebellar peduncle (*Fig. 1B*).

Nearly 1 year after GKS, an MRI scan revealed regression in tumor size, with the largest diameter measuring 2.0 cm, notably decreased from 3.2 cm on the previous scan (*Fig. 1C*). The cystic portions remained evident on the scan, although there was significantly less mass effect on the brainstem. Clinically, the patient experienced intermittent balance difficulties, poor hearing on the right side, and decreased sensation of the face. Facial and extremity motor functions were normal. After 1 additional year, the patient was clinically stable. An MRI scan revealed a further decrease in tumor size, where the largest diameter was reduced to 1.0 cm, compared with 2.0 cm on the previous scan (*Fig. 1D*).

Six months later, the patient presented with sudden deterioration. She experienced acute onset of right facial numbness, decreased right hearing, dizziness, and ataxia. On examination, she had impaired right corneal reflex, right anacusis, right mild facial weakness, right decreased facial sensation in the trigeminal nerve regions V2 and V3, and profoundly poor tandem gait. MRI revealed an expansion of the cerebellopontine angle mass (*Fig. 1E*). The largest diameter measured 3.2 cm, compared with 1.0 cm from the previous scan. The mass was predominantly multicystic with rim enhancement and expanded the right internal auditory canal. There appeared to be increased mass effect on the brainstem.

Microsurgery was performed using a retrosigmoid approach. A large cystic tumor with entrapped cerebrospinal fluid was encountered, and the tumor was removed. A small amount of tumor that was adherent to the brainstem was not excised to minimize postoperative morbidity. Intraoperative frozen and permanent tumor samples were submitted for pathological review. The formalin-fixed, paraffin-embedded tumor tissue was cut into serial sections 5 μm thick. One section was stained with hematoxylin and eosin (*Fig. 3A*), and an adjacent section was analyzed for S-100 expression (*Fig. 3B*) using a rabbit polyclonal antibody (no. Z-0311; DakoCytomation, Carpinteria, CA) and counterstained with hematoxylin. Pathological examination confirmed the diagnosis of a VS, as S-100 histochemical staining is characteristic (1, 4). Postoperative MRI revealed that the tumor had been debulked with the intracanalicular component unchanged.

One month after microsurgery, the patient's dizziness resolved completely and her gait ataxia improved. One year later, there was no recurrence of the tumor or change in the intracanalicular component, and the previously identified cystic fluid collection in the resection cavity was no longer visible on MRI scans (*Fig. 1F*). There were residual focal sensory deficits in the right V1–V2 distribution, but facial nerve function was normal (House-Brackmann Scale Grade I). The lower cranial nerves were also intact. Hearing remained poor and her underlying tinnitus returned to baseline intensity and quality. The patient was able to resume her normal activities of daily living.

DISCUSSION

In this article, we report a case of a cystic VS that fluctuated in volume after GKS, first expanding and shrinking, then rapidly expanding 2 years later, necessitating microsurgical removal. The post-GKS course of VS is typically either: 1) regression, 2) transient enlargement then regression, 3) sustained enlargement, or 4) no change (10, 16, 18, 21). Tumor regression usually occurs within 3 years, with marked regression by 5 years, although some VS continue to expand, particularly if they are cystic (5). There are no previous reports of enlargement followed by regression and subsequent enlargement. Although the significant regression of the cystic VS presented here might have been interpreted as a typical reassuring course, several months later, it expanded rapidly and unpredictably. This case illustrates that shrinkage of a cystic VS after

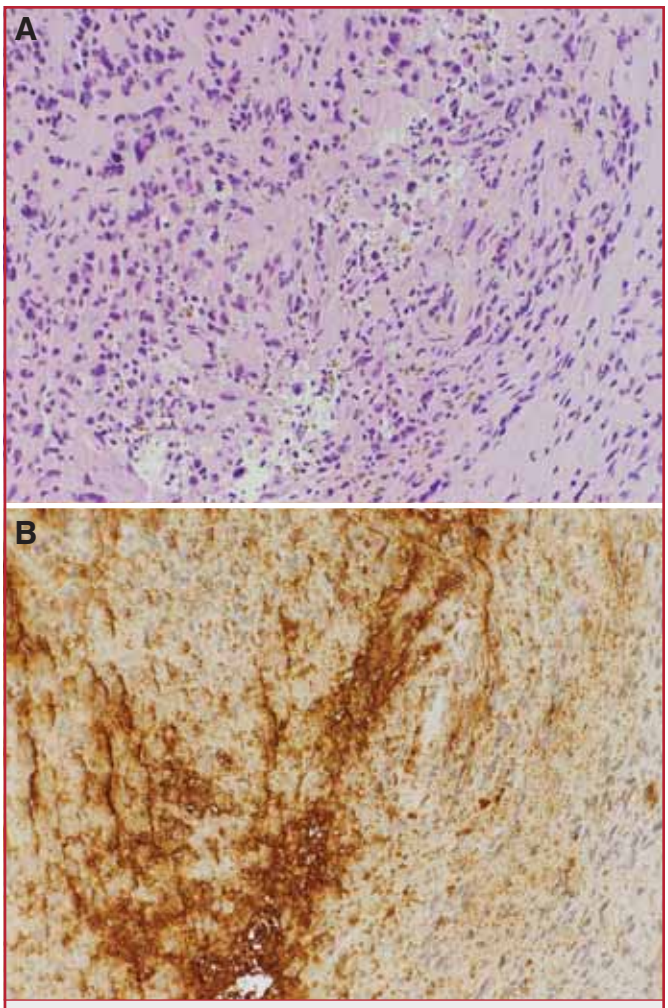

FIGURE 3. *Histopathological examination of the resected tumor.* **A**, *hematoxylin and eosin stain showing schwannoma cell features with some degenerative change and focal hemosiderin deposition indicating remote hemorrhage.* **B**, *S-100 immunohistochemical stain showing diffuse positive staining in tumor cells characteristic of a schwannoma. Original magnification,* ×*200.*

GKS does not guarantee continued tumor regression. A cystic VS must be monitored closely radiographically, even years after GKS has apparently induced tumor shrinkage.

Numerous studies have shown that GKS performed according to current technical standards is comparable to microsurgery for small- and medium-size VS (10), with tumor control rates exceeding 90% (3, 8, 10, 15, 16, 23–26, 28). GKS may result in fewer complications compared with open microsurgery, such as cranial neuropathies and hearing loss (15, 16). Open microsurgery carries the risk of stroke, intracerebral hemorrhage, cardiac arrest, postoperative pain, meningitis, and cerebrospinal fluid leakage (9, 15, 18, 25).

A cystic VS presents a therapeutic conundrum, as it often responds poorly to both microsurgery and GKS. Cystic VS, representing 4 to 15% of all VS cases (1, 2, 4, 6), are characterized by large size and rapid expansion; short clinical history (<2 yr); and atypical initial symptoms, such as facial pain or numbness, dysgeusia, or facial palsy (1, 6, 12). Compared with solid tumors, cystic tumors have a worse microsurgical outcome (4) and tend to behave more unpredictably after irradiation (10). Preexisting cystic VS components may expand markedly after GKS, necessitating surgical removal (23–26). In one study of six patients with cystic VS treated with GKS, all six tumors enlarged after the procedure, causing new-onset cranial neuropathies (12). The authors cautioned against the use of GKS for cystic VS.

The biological mechanisms underlying the delayed regression of VS after GKS are poorly understood but may be attributable to delayed sequelae of damage to deoxyribonucleic acid and vessels. Irradiation damages deoxyribonucleic acid, triggering delayed apoptosis and growth arrest of slowly dividing tumors cells (7). Radiation also causes cellular proliferation of arterioles and small arteries, leading to small artery occlusion (7, 11, 17, 19) and prominent interstitial fibrin accumulation within vascular walls (3), inducing vasculitis (22). These vascular changes may cause tumor cell ischemia and hypoxic cell death. The fact that effects of irradiation on apoptotic pathways and vasculature are not immediate may explain why VS regression is delayed after GKS.

Possible mechanisms of cystic tumor expansion after GKS include osmotic mechanisms from vascular damage, which can induce extravasation of serum proteins into the extracellular matrix. Transient radiation-induced elevation in protein levels may also increase transudation from tumor vessels. Together, these factors increase the osmotic force favoring fluid accumulation in cystic spaces. Furthermore, coagulation necrosis of the tumor could enhance osmotic tendencies and lead to edema (6, 25). Another explanation for the reportedly poor response of cystic VS to GKS is that these cystic tumors were actually misdiagnosed solid VS-associated arachnoid cysts. Arachnoid cysts do not respond to radiation, and proteinaceous tumor debris could theoretically cause local cerebrospinal fluid flow disruptions and consequent cyst enlargement. Finally, tumor cell expansion is another possible but unlikely mechanism of cystic VS expansion because a cystic VS has significantly fewer Ki-67-positive cells (by a factor of 36) than a solid VS, which is a direct measure of proliferative activity (1, 2). More work is needed to delineate the mechanisms of cystic tumor component expansion after irradiation.

CONCLUSION

In conclusion, a literature review reveals that the mechanisms underlying VS expansion and regression after irradiation are not fully understood, and that a cystic VS may enlarge more frequently than a solid VS after GKS. This case report is a unique description of a cystic VS that expanded transiently, regressed significantly over 2 years, then unpredictably and rapidly expanded, necessitating microsurgical removal. Sustained regression after transient expansion does not preclude the possibility of subsequent sustained and rapid enlargement. This risk should be addressed with patients when discussing the relative benefits of radiosurgery and micro-

surgery for the treatment of a cystic VS. When GKS is chosen, close clinical monitoring at regular intervals is prudent. Future studies in molecular and biological markers of cystic VS may help to identify the tumors prone to undergo cystic enlargement after radiosurgery.

REFERENCES

1. Charabi S, Klinken L, Tos M, Thomsen J: Histopathology and growth pattern of cystic acoustic neuromas. **Laryngoscope** 104:1348–1352, 1994.
2. Charabi S, Tos M, Thomsen J, Rygaard J, Fundova P, Charabi B: Cystic vestibular schwannoma: Clinical and experimental studies. **Acta Otolaryngol Suppl** 543:11–13, 2000.
3. Chung WY, Liu KD, Shiau CY, Wu HM, Wang LW, Guo WY, Ho DM, Pan DH: Gamma knife surgery for vestibular schwannoma: 10-year experience of 195 cases. **J Neurosurg** 102 [Suppl]:87–96, 2005.
4. Fundová P, Charabi S, Tos M, Thomsen J: Cystic vestibular schwannoma: Surgical outcome. **J Laryngol Otol** 114:935–939, 2000.
5. Hasegawa T, Kida Y, Yoshimoto M, Koike J, Goto K: Evaluation of tumor expansion after stereotactic radiosurgery in patients harboring vestibular schwannomas. **Neurosurgery** 58:1119–1128, 2006.
6. Kameyama S, Tanaka R, Kawaguchi T, Fukuda M, Oyanagi K: Cystic acoustic neurinomas: Studies of 14 cases. **Acta Neurochir (Wien)** 138:695–699, 1996.
7. Linskey ME: Stereotactic radiosurgery versus stereotactic radiotherapy for patients with vestibular schwannoma: A Leksell Gamma Knife Society 2000 debate. **J Neurosurg** 93 [Suppl 3]:90–95, 2000.
8. Lunsford LD, Niranjan A, Flickinger JC, Maitz A, Kondziolka D: Radiosurgery of vestibular schwannomas: Summary of experience in 829 cases. **J Neurosurg** 102 [Suppl]:195–199, 2005.
9. Møller P, Myrseth E, Pedersen PH, Larsen JL, Krakenes J, Moen G: Acoustic neuroma: Treatment modalities. Surgery, gamma-knife or observation? **Acta Otolaryngol Suppl** 543:34–37, 2000.
10. Nakamura H, Jokura H, Takahashi K, Boku N, Akabane A, Yoshimoto T: Serial follow-up MR imaging after gamma knife radiosurgery for vestibular schwannoma. **AJNR Am J Neuroradiol** 21:1540–1546, 2000.
11. Paek SH, Chung HT, Jeong SS, Park CK, Kim CY, Kim JE, Kim DG, Jung HW: Hearing preservation after gamma knife stereotactic radiosurgery of vestibular schwannoma. **Cancer** 104:580–590, 2005.
12. Pendl G, Ganz JC, Kitz K, Eustacchio S: Acoustic neurinomas with macrocysts treated with Gamma Knife radiosurgery. **Stereotact Funct Neurosurg** 66 [Suppl 1]:103–111, 1996.
13. Pollock BE, Lunsford LD, Flickinger JC, Clyde BL, Kondziolka D: Vestibular schwannoma management: Part. Failed microsurgery and the role of delayed stereotactic radiosurgery. **J Neurosurg** 89:944–948, 1998.
14. Pollock BE, Lunsford LD, Kondziolka D, Sekula R, Subach BR, Foote RL, Flickinger JC: Vestibular schwannoma management. Part II. Failed radiosurgery and the role of delayed microsurgery. **J Neurosurg** 89:949–955, 1998.
15. *Deleted in proof.*
16. Prasad D, Steiner M, Steiner L: Gamma surgery for vestibular schwannoma. **J Neurosurg** 92:745–759, 2000.
17. Schneider BF, Eberhard DA, Steiner LE: Histopathology of arteriovenous malformations after gamma knife radiosurgery. **J Neurosurg** 87:352–357, 1997.
18. Selch MT, Pedroso A, Lee SP, Solberg TD, Agazaryan N, Cabatan-Awang C, DeSalles AA: Stereotactic radiotherapy for the treatment of acoustic neuromas. **J Neurosurg** 101 [Suppl 3]:362–372, 2004.
19. Seo Y, Fukuoka S, Nakagawara J, Takanashi M, Takahashi S, Suematsu K, Nakamura J: Effect of Gamma Knife radiosurgery on acoustic neurinomas. Assessment by 99mTc-DTPA-human serum albumin-and 201TlCl-single photon emission computed tomography. **Stereotact Funct Neurosurg** 66 [Suppl 1]:93–102, 1996.
20. Shin M, Ueki K, Kurita H, Kirino T: Malignant transformation of a vestibular schwannoma after gamma knife radiosurgery. **Lancet** 360:309–310, 2002.
21. Shirato H, Sakamoto T, Takeichi N, Aoyama H, Suzuki K, Kagei K, Nishioka T, Fukuda S, Sawamura Y, Miyasaka K: Fractionated stereotactic radiotherapy for vestibular schwannoma (VS): Comparison between cystic-type and solid-type VS. **Int J Radiat Oncol Biol Phys** 48:1395–1401, 2000.
22. Slattery WH 3rd, Brackmann DE: Results of surgery following stereotactic irradiation for acoustic neuromas. **Am J Otol** 16:315–321, 1995.
23. Unger F, Walch C, Haselsberger K, Papaefthymiou G, Trummer M, Eustacchio S, Pendl G: Radiosurgery of vestibular schwannomas: A minimally invasive alternative to microsurgery. **Acta Neurochir (Wien)** 141:1281–1286, 1999.
24. Unger F, Walch C, Papaefthymiou G, Eustacchio S, Feichtinger K, Quehenberger F, Pendl G: Long term results of radiosurgery for vestibular schwannomas. **Zentralbl Neurochir** 63:52–58, 2002.
25. Unger F, Walch C, Papaefthymiou G, Feichtinger K, Trummer M, Pendl G: Radiosurgery of residual and recurrent vestibular schwannomas. **Acta Neurochir (Wien)** 144:671–677, 2002.
26. Unger F, Walch C, Schröttner O, Eustacchio S, Sutter B, Pendl G: Cranial nerve preservation after radiosurgery of vestibular schwannomas. **Acta Neurochir Suppl** 84:77–83, 2002.
27. Wackym PA: Stereotactic radiosurgery, microsurgery, and expectant management of acoustic neuroma: Basis for informed consent. **Otolaryngol Clin North Am** 38:653–670, 2005.
28. Wowra B, Muacevic A, Jess-Hempen A, Hempel JM, Müller-Schunk S, Tonn JC: Outpatient gamma knife surgery for vestibular schwannoma: Definition of the therapeutic profile based on a 10-year experience. **J Neurosurg** 102 [Suppl]:114–118, 2005.

Acknowledgments

We thank Andrew Bollen, M.D., for interpretation of the immunohistochemistry; David A. Larson, M.D., Ph.D., and Vernon Smith, Ph.D., for radiosurgical expertise; and Lijun Ma, Ph.D., for acquiring the gamma knife radiation treatment plan.

COMMENTS

The authors describe a fluctuation in size of an acoustic neuroma that has undergone radiosurgery. In this case, there was not only initial enlargement of the tumor before postradiation shrinkage, but the tumor also enlarged again several years later, necessitating surgical resection. I also feel that tumors with a cystic component may have a more variable course than their solid tumor counterparts after radiosurgery. This may result in large part from the fact that even a dying tumor may continue to cause an increase in cyst volume, which results in overall tumor expansion. At our institution, we have seen cystic hemangioblastomas behave in a similar fashion, in which the solid portion of the tumor appears to be decreasing in size after radiosurgery, but the cystic portion of the tumor increases in size, necessitating surgical intervention.

Steven D. Chang
Stanford, California

de Ipolyi et al. describe a case of fluctuating size changes in a vestibular schwannoma after radiosurgery, eventually requiring surgery. This is an interesting and different posttreatment course. Vestibular schwannomas most commonly shrink slowly after radiosurgery. Sometimes there is a period of transient enlargement, followed by shrinkage. Here, enlargement was followed by shrinkage, then by dramatic enlargement, requiring surgery. Ultimately, this case is just a radiosurgical failure, albeit with a strange radiographic course. Radiosurgery will prevent subsequent tumor growth in more than 90% of patients. These results appear to be durable (at least with good 10-year follow-up data). In our series, 99% of patients have required no further surgical intervention after radiosurgery (in other words, enlargement is usually minimal and asymptomatic). Complication rates (facial and trigeminal nerve) are both less than 1%. These results compare favorably with those of the best reported surgical series.

William A. Friedman
Gainesville, Florida

This is an important case report that focuses on cystic vestibular schwannomas, which are often clinically less favorable than similar solid tumors. In addition, this report demonstrates again that radiation therapy has delayed risks, so that consistent clinical and imaging follow-up remain important in the management of patients treated with this modality.

Samuel H. Selesnick
Neuro-otologist

Philip H. Gutin
New York, New York

Stereotactic radiosurgery (SRS) can provide long-term control of acoustic neuromas with preservation of cranial nerve function. After SRS, most of the irradiated acoustic tumors decrease in size over time. In the original series by Kondziolka et al. (1), 73.8, 48.4, and 38.1% of tumors, mostly solid type, remained unchanged in size at 1, 3, and 5 years after SRS, respectively. Interestingly, during the first 3 years, the proportion of patients with enlarged tumors increased from 0.7 to 3.1%. Any increase in tumor size after therapy may represent either true tumor regrowth or expansion of the tumor margins as the central portion of the tumor become necrotic. The treatment for cystic acoustic neuromas is tricky, whether by SRS or microsurgery. The cysts in these tumors are almost always part of the tumor, and it is possible to exclude them in treatment planning or to leave part of the cyst wall behind during surgery. For reasons that are not clear, cystic vestibular schwannomas enlarge transiently more commonly than solid tumors after SRS. Shirato et al. (2) reported an actuarial 3-year rate of enlargement greater than 2.0 mm of 45% for cystic and 25% for solid tumors. However, the subsequent tumor reduction rate in cystic schwannomas was better, and the overall actuarial tumor control rate with no need for additional surgery was 92% at 5 years in their series. The case report by de Ipolyi et al. illustrates the fluctuating radiological response of cystic vestibular schwannomas after radiosurgery with an initial response and then an enlargement later needing surgical intervention. Conclusions are difficult to extract from this single case report, because individual tracking of the reported radiological failures are rarely reported. However, it does highlight the point that initial radiological response does not always predict long-term tumor control with cystic tumors and long-term follow-up is necessary.

Ashwatha Narayana
Radiation Oncologist

John G. Golfinos
New York, New York

1. Kondziolka D, Lunsford LD, McLaughlin MR, Flickinger JC: Long-term outcomes after radiosurgery for acoustic neuromas. **N Engl J Med** 339:1426–1433, 1998.
2. Shirato H, Sakamoto T, Takeichi N, Aoyama H, Suzuki K, Kagei K, Nishioka T, Fukuda S, Sawamura Y, Miyasaka K: Fractionated stereotactic radiotherapy for vestibular schwannoma (VS): Comparison between cystic-type and solid-type VS. **Int J Radiat Oncol Biol Phys** 48:1395–1401, 2000.