Postoperative imaging of vestibular schwannomas

Daniel T. G inat, M.D., M.S., 1 and Robert L. Martuza, M.D., F.A.C.S. 2
Departments of 1Radiology and 2Neurosurgery, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts

Symptomatic vestibular schwannomas can be treated with resection (translabyrinthine, retrosigmoid [suboccipital], or middle cranial fossa approaches) or stereotactic radiosurgery. When appropriate, auditory brainstem stimulators can also be implanted in patients with current or impending hearing loss due to bilateral vestibular schwannomas. Imaging plays a prominent role in determining management following these procedures. In this article, the expected postoperative imaging appearances are depicted. The radiological features of complications are also reviewed, including recurrent tumor, fat graft necrosis, CSF leakage, infection, hydrocephalus, cerebral infarction, venous sinus thrombosis, hemorrhage, and temporal lobe and cerebellar contusions.

(http://thejns.org/doi/abs/10.3171/2012.6.FOCUS12150)

Key Words • vestibular schwannoma • magnetic resonance imaging • computed tomography • postoperative imaging

V estibular schwannomas are typically benign, slow-growing tumors that most commonly arise from the region of Scarpa’s ganglion and comprise approximately 85% of cerebellopontine angle masses. 23 Patients with vestibular schwannomas can present with tinnitus, dizziness, unsteadiness, and vertigo, as well as symptoms due to compression effects, including hearing loss, facial and trigeminal nerve dysfunction, and hydrocephalus. 15 Management options include observation with serial imaging, stereotactic radiosurgery, fractionated radiotherapy, microsurgical resection, or a combination of these. Auditory brainstem implants are a useful adjunct in select patients. Radiological imaging plays an important role in guiding management. In this article, we review the imaging features of surgical approaches, auditory brainstem implantation, and stereotactic radiosurgery along with relevant complications in cases performed by several surgeons from various institutions. Indeed, many of the cases presented in this article were referred to our institution for specialized management of complications.

Abbreviation used in this paper: MRV = MR venography.

Surgical Approaches

There are 3 main types of surgical routes that can be implemented for resection of vestibular cranial nerve tumors, including translabyrinthine, retrosigmoid (suboccipital), and middle cranial fossa approaches. 1,21 These routes are detailed as follows: 1) Translabyrinthine resection involves performing mastoidectomy and labyrinthectomy, including resection of the semicircular canals and at least portions of the vestibule. The ipsilateral cerebellum is retracted, and the internal auditory canal is skeletonized to facilitate tumor resection. Once the tumor is removed, the resection bed is packed with a characteristic triangular-shaped fat graft (Fig. 1). 2) The retrosigmoid (suboccipital) approach consists of performing craniotomy or craniectomy with cranioplasty via a retrosigmoid incision inferior to the transverse sinus and medial to the sigmoid sinus. Portions of the mastoid air cells are often traversed, but are sealed intraoperatively using bone wax. In addition, drilling through the posterior wall of the internal auditory canal is performed to access the internal auditory canal. This is often repaired with an adipose graft. Cerebrospinal fluid is drained to promote cer-
ebellar relaxation, and the ipsilateral cerebellum may be retracted. This can result in a characteristically flattened lateral edge of the cerebellar hemisphere in association with prominence of the overlying extraaxial CSF (Fig. 2), which should not be mistaken for an arachnoid cyst or hemorrhage. 3) The middle cranial fossa approach involves performing a temporal craniotomy, retracting the temporal lobe superiorly, and decompressing the internal auditory canal by skeletonizing the roof (Fig. 3). A layer of fat or fascial graft is usually applied to the dural defect overlying the internal auditory canal to seal the internal auditory canal.

The translabyrinthine and the retrosigmoid approaches can be used for all tumor sizes, while the middle cranial fossa approach is useful only for removal of intracanalicular tumors. Hearing preservation can be achieved only via the retrosigmoid and middle cranial fossa approaches. Resection can be classified as gross total if there is no visible tumor remaining, near total if the remaining tumor dimensions are less than $5 \times 5 \times 2$ mm, and subtotal if the remaining tumor is larger than that achieved after near-total resection. Alternatively, near-total resection can be designated as incomplete removal with less than 5% residual tumor volume and subtotal resection as greater than 5% of the original tumor volume. Subtotal resection is associated with a 9 times greater likelihood of tumor recurrence compared with gross-total resection.

Magnetic resonance imaging is routinely performed after resection, mainly to assess for residual or recurrent tumor, as well as suspected complications. A typical postoperative MRI examination consists of an internal auditory canal protocol that includes thin-section axial precontrast T1-weighted imaging, axial and coronal thin-section postcontrast fat-suppressed T1-weighted imaging, T2-weighted fast spin echo, T2-weighted FLAIR, diffusion-weighted imaging, and cisternography sequences, such as CISS (constructive interference in steady state) or FIESTA (fast imaging employing steady-state acquisition), at the level of the cerebellopontine angle. The use of fat suppression is helpful for distinguishing enhancing tumor from the intrinsic high signal intensity of the fat grafts, which are commonly implanted in the surgical beds during tumor resection to minimize CSF leakage. Sequences can be added or modified based on the particular indication, such as whole-brain field of view for widespread complications or MRV for venous sinus thrombosis. Computed tomography also plays a role in postoperative imaging for vestibular schwannoma resection, particularly for defining the altered bony anatomy and hemorrhage.

Linear enhancement (Fig. 4) corresponding to scar and/or granulation tissue in the surgical bed is apparent on MRI in the majority of cases during the first 6 postoperative months and can persist for 1 year or longer. On the other hand, the presence of nodular enhancement (Fig. 5) on the initial postoperative MRI studies is associated with a 16-fold higher risk for eventual recurrence compared with a linear pattern of enhancement. It is also important to assess the entire extent of the surgical approach for nodular enhancement that may represent intraoperative tumor seeding (Fig. 6). It is sometimes difficult to distinguish between the different patterns of enhancement on baseline MRI, and follow-up imaging is useful for elucidating the nature of the enhancement.

Other complications related to surgical treatment of vestibular schwannomas that can be readily assessed on
Postoperative imaging of vestibular schwannomas

imaging include fat graft necrosis, CSF leakage, infection, cerebral infarction, venous sinus thrombosis, hemorrhage, cerebellar atrophy, and endolymphatic fluid loss. Risk factors for complications include underlying severe general and/or neurological morbidity, cystic tumor, and major cranial nerve deficits.17

Fat graft necrosis occurs in approximately 1% of cases of vestibular schwannoma surgery in which a fat graft is used to eliminate dead space and reinforce dural closure.24 Sequelae of fat necrosis can occur within days to years after surgery and include sterile liquefied fat fistula, CSF leakage, lipoid meningitis, and graft shrinkage.9,24 Patients with these complications generally experience favorable outcomes with appropriate management.24 On imaging, fat necrosis can appear as fragmentation of the fat graft, allowing infiltration by fluid (Fig. 7). The fluid can extend to the scalp incision. Aseptic lipoid meningitis can present with meningismus and appears as punctate ovoid droplets of fat density or signal on imaging (Fig. 8).9

Cerebrospinal fluid leakage occurs in approximately 1%–8% of cases following schwannoma resection.5,12,14 Cerebrospinal fluid otorrhea can sometimes occur due to mastoid air cell entry despite the application of bone wax.21 In the appropriate clinical setting, opacification of the ipsilateral mastoid air cells and middle ear with craniotomy defect that traverses the mastoid air cells suggests mastoid entry as the source of CSF leak (Fig. 9). Cerebral spinal fluid wound leakage usually occurs over the craniotomy or cranioplasty site and can result in pseudomeningoceles. These lesions display fluid attenuation on CT and CSF signal intensity on MRI (Fig. 10). Since pseudomeningoceles can become large, potentially

Fig. 4. Linear enhancement. Axial postcontrast fat-suppressed T1-weighted MRI study showing linear dural enhancement within the left internal auditory canal (arrow) and in the posterior fossa (arrowhead).

Fig. 5. Nodular enhancement. Axial postcontrast fat-suppressed T1-weighted MRI study showing a rounded focus of enhancement within the left internal auditory canal (arrow).

Fig. 6. Intraoperative tumor seeding. Axial postcontrast fat-suppressed T1-weighted MRI study showing a progressively enlarging enhancing nodule anterior to the fat graft (arrow), which was not present preoperatively. Recurrent tumor is present in the right cerebellopontine angle (arrowhead).

Fig. 7. Fat graft necrosis with CSF fistula. This patient presented with drainage of CSF from the wound after schwannoma resection. Axial CT image (left) and axial T2-weighted MRI study (right) showing striated bands of fluid (arrows) traversing the fat graft, extending to the incision site (arrowheads).

Fig. 8. Aseptic lipoid meningitis. Axial T2-weighted image showing punctate ovoid droplets of fat density on the inner table of the skull base (arrows).
extending toward the apex superiorly and into the posterior neck soft tissues inferiorly, it is important to adjust the imaging field of view accordingly to include the entire lesion, perhaps with the aid of external markers.

Wound infection occurs in up to approximately 4% of cases.11 Although the diagnosis can be made clinically, imaging is useful for delineating the extent of disease and establishing prompt diagnosis and treatment, which is crucial for preventing severe morbidity. On MRI, postoperative wound infections can appear as rim-enhancing fluid collections (Fig. 11) with variable presence of restricted diffusion. There may also be intracranial involvement, which can manifest as extra- or intraaxial abscess, labyrinthitis (Fig. 12), and meningitis with communicating hydrocephalus (Fig. 13). On MRI, infectious labyrinthitis can manifest as diffuse enhancement of the cochlea, vestibule, and/or the semicircular canals, as well as loss of endolymphatic fluid on T2-weighted sequences. Labyrinthitis ossificans can eventually develop, which appears as hyperdensity within the inner ear structures. Diminished labyrinthine fluid signal on MRI is associated with postoperative hearing loss.23,25 The imaging differential diagnosis for loss of endolymphatic fluid signal includes labyrinthine fenestration, vascular injury, and blood products.21 It is important to compare these findings with the aid of preoperative imaging since schwannomas can produce these signal abnormalities.7,22

Arterial injury during vestibular schwannoma resection is rare and tends to involve the anterior inferior cerebellar artery and less often the posterior inferior cerebellar artery with retrosigmoid and translabyrinthine approaches and the middle cerebral artery branches with the middle cranial fossa approach. This can sometimes result in cerebral infarction, which produces restricted diffusion and swelling in the acute and subacute stages (Fig. 14). Venous parenchymal injury sometimes occurs in the lateral pons and middle cerebral peduncle during removal of adhesive schwannomas and can manifest as an infarct on imaging.21

Venous sinus thrombosis has been reported in up to 5% of suboccipital craniotomies and translabyrinthine craniectomies for tumor resection.10 The transverse and sigmoid sinuses on the treated side are most often affected, while the contralateral sinuses are typically spared. Magnetic resonance imaging with MRV is a suitable, noninvasive modality for obtaining the diagnosis (Fig. 15). On T1-weighted MRI studies, the thrombus can display a high signal if it is in the subacute stage.6 Consequently, contrast-enhanced MRV can lead to false-negative results. However, on 3D time-of-flight MRV, the loss of flow-related enhancement confirms the diagnosis.6 Computed tomography venography is also sensitive for delineating venous sinus thromboses, which appear as filling defects. Patients can present with headache, visual obscuration, and papilledema due to elevated intracranial pressure, even if the affected venous sinus is nondominant.20 Symptomatic treatment consists of acetazolamide and steroids; however, endovascular thrombolysis or anticoagulation may be warranted in the acute postoperative period.10

Postoperative hemorrhage can result from venous injury and occurs in approximately 0.6% of cases.18 Subdural and brainstem hematomas have been reported in approximately 0.4% and 0.1% of cases, respectively.18 Noncontrast CT scanning is adequate for assessing acute hemorrhage, which appears hyperdense (Fig. 16). If the hemorrhage is sufficiently large, there can be mass effect
Postoperative imaging of vestibular schwannomas

Fig. 10. Pseudomeningocele. Axial T2-weighted (A), axial T1-weighted (B), and sagittal T1-weighted (C) MRI studies showing a large subgaleal fluid collection (asterisk) that communicates with the intracranial CSF across the left retrosigmoid craniotomy.

Fig. 11. Wound infection. Axial postcontrast T1-weighted MRI study showing rim-enhancing fluid collections in the subcutaneous tissues overlying the right retrosigmoid craniotomy and in the epidural space (arrow).

Fig. 12. Labyrinthitis. A and B: Preoperative postcontrast fat-suppressed axial T1-weighted MRI study (A) and CISS sequence (B) showing a right vestibular schwannoma with preservation of the endolymphatic fluid signal and no abnormal labyrinthine enhancement. The patient developed postoperative Klebsiella meningitis. C: Postoperative postcontrast fat-suppressed axial T1-weighted MRI study showing subtotal resection of the tumor and new avid enhancement in the cochlea (arrow) and vestibule (arrowhead). D: Corresponding CISS sequence showing loss of the fluid signal in the right cochlea (arrow). E: Axial CT image obtained 1 year later showing hyperdensity within the cochlea (arrow), consistent with labyrinthitis ossificans.

upon the brainstem and obstructive hydrocephalus. Organizing hematomas can last several years after surgery and occasionally mimic recurrent tumor.8

Auditory Brainstem Implant

Auditory brainstem implants are typically implanted via craniotomy at the time of tumor removal and are intended to facilitate lip reading and, to some extent, enable direct speech comprehension.20 The devices are used when the contralateral ear provides no hearing or if there is concern of contralateral hearing loss, such as in patients with neurofibromatosis Type 2. The main components of a typical auditory brainstem implant include the receiver-stimulator, grounding ball electrode, and stimulator electrode, which is positioned over the affected cochlear nucleus (Fig. 17), usually via the lateral recess of the fourth ventricle. Computed tomography is well suited to assess whether the device components are intact. However, considerable streak artifact produced by
the hardware can limit precise determination of implant positioning. On the other hand, thin-section T2-weighted images are useful for delineating the intracranial course of the stimulator electrode. Complications related to auditory brainstem implant malposition include suboptimal production of auditory stimuli, CSF leak along the course of the wire, and nonauditory stimuli, such as trigeminal neuralgia.19

Stereotactic Radiosurgery

Stereotactic radiosurgery (for example, proton beam and Gamma Knife surgery) for vestibular schwannomas consists of delivering a focused radiation dose to halt tumor growth.13,16 The imaging response to radiosurgery is widely variable. With regard to tumor volume, transient enlargement is observed in 30%–41% of patients, no change or sustained regression in 34%–82%, alternating enlargement and regression in 13%, and continuous enlargement in 12%–16%.13,16 Transient enlargement generally occurs within 2 years after radiosurgery and is often followed by regression.13,16 Changes in tumor enhancement characteristics include transient loss of enhancement in 84% of patients (Fig. 18), continuous increase in enhancement in 5%, and no change in enhancement in 11%.13 An increase in the proportion of tumor cystic components does not appear to correlate with the effectiveness of the treatment.11 New areas of T2 hyperintensity in the adjacent brain parenchyma can appear in about 30% of cases on average 12 months after treatment.
Postoperative imaging of vestibular schwannomas

Fig. 17. Auditory brainstem implant. A: Scout image showing the components of the device, including the receiver stimulator (arrow), stimulator electrode (white arrowhead), and grounding electrode (black arrowhead). B and C: Axial CT images in the bone (B) and soft-tissue (C) windows showing that the electrode (arrows) produces considerable streak artifact, which makes precise localization difficult. D: The position of the electrode (arrow) is better delineated on the axial T2-weighted MRI.

and eventually resolve in most cases. Rarely, secondary neoplasms can arise as a result of the radiation exposure, including sarcomas and meningiomas.

Conclusions

Radiological imaging plays an important role in evaluating patients with vestibular schwannomas after microsurgery, auditory brainstem implantation, and stereotactic radiosurgery. Familiarity with expected and complicated imaging findings is necessary for optimal posttreatment management.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Ginat. Analysis and interpretation of data: both authors. Drafting the article: Ginat. Critically revising the article: both authors. Reviewed submitted version of manuscript: both authors. Approved the final version of the manuscript on behalf of both authors: Ginat.

References


Neurosurg Focus / Volume 33 / September 2012

Accepted June 21, 2012.
Please include this information when citing this paper: DOI: 10.3171/2012.6.FOCUS12150.
Address correspondence to: Daniel T. Ginat, M.D., M.S., Department of Radiology, Massachusetts General Hospital, 55 Fruit Street, Boston, Massachusetts 02114. email: ginatd01@gmail.com.