Trigeminal Nerve Deficit in Large and Compressive Acoustic Neuromas and Its Correlation with MRI

Findings

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What is This?
Trigeminal Nerve Deficit in Large and Compressive Acoustic Neuromas and Its Correlation with MRI Findings

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Abstract

Objectives. Evaluate the prevalence of preoperative trigeminal nerve deficit in large/compressive acoustic neuromas and try to find a correlation between pre/postoperative magnetic resonance imaging (MRI) findings and pre/postoperative trigeminal nerve deficit.

Study Design. Case series with chart review.

Setting. University medical center.

Subjects and Methods. Retrospective study (1994-2009) including patients with stage 4 or 5 acoustic neuromas (Zininger-Magnan classification). All patients underwent surgical resection. Pre- and postoperative trigeminal symptoms were sought. Imaging criteria were sought on pre- and 3-month postoperative MRI scans. Pearson $\chi^2$ statistical test was used.

Results. Fifty-three patients (27 females, mean 51 years) were operated on. Preoperatively, 3 patients (5.7%) had trigeminal neuralgia, 1 (1.9%) trigeminal anesthesia, and 28 (52.8%) trigeminal hypoesthesia. Sixteen patients (30.2%) had no corneal reflex (ophthalmic branch); keratitis occurred in 1 patient (1.9%). Postoperatively, 2 patients (3.8%) had trigeminal neuralgia, 1 (1.9%) trigeminal anesthesia, and 24 (45.3%) trigeminal hypoesthesia. Twenty-six patients (49%) had no corneal reflex; keratitis occurred in 11 patients (20.7%). Preoperative trigeminal hypoesthesia was statistically correlated with impaction of the tumor on cerebellar peduncles on preoperative MRI. Postoperative trigeminal hypoesthesia was statistically correlated with nonvisibility of the trigeminal nerve on postoperative MRI.

Conclusion. In large/compressive acoustic neuromas, trigeminal nerve deficit has to be sought to avoid corneal complications in particular. Trigeminal hypoesthesia occurs preoperatively in about half of the cases. It remains relatively stable after tumor removal, but there appears to be an increased rate of absent corneal reflex and keratitis postoperatively. We were able to correlate pre/postoperative trigeminal hypoesthesia with pre/postoperative MRI findings.

Keywords

trigeminal nerve, acoustic neuroma, vestibular schwannoma, giant tumor, cerebellopontine angle

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Introduction

The preoperative symptoms and signs as well as the postoperative outcome after surgical removal of large and compressive acoustic neuromas are nowadays well known with less than 1% mortality rate, 31% risk of facial palsy, 37% hearing preservation rate, and 2.5% risk of lower cranial nerve deficit in specialized referral centers.1-3 However, preoperative trigeminal nerve manifestations and postoperative trigeminal nerve sequelae are usually underestimated or sometimes not even mentioned in the studies. Trigeminal nerve involvement should be well defined because of its functional consequences on facial sensation and mastication. More particularly, involvement of its ophthalmic branch (V1) may have dramatic consequences on vision should neuroparalytic keratitis occur.

The purpose of this study was to evaluate the incidence of preoperative trigeminal nerve deficit in large and compressive acoustic neuromas and try to find a correlation between pre- and postoperative magnetic resonance imaging (MRI) findings and pre- and postoperative trigeminal nerve deficit.

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**Methods**

This work is a retrospective study carried out in an academic hospital over 16 years, from 1994 to 2009, which included all patients with an acoustic neuroma stage 4 (21-40 mm, displacing fourth ventricle) or stage 5 (>40 mm, extending beyond midline) using the classification of Zini and Magnan. To measure the size of the tumor, we calculated the largest diameter of the tumor (Figure 1).

All patients included underwent single-stage surgical resection. Pre- and postoperative trigeminal symptoms/signs were sought in all cases. Pre- and postoperative (3 months after surgery) MRIs were performed using the same protocol, pre- and postcontrast T1-weighted spin echo imaging with a slice thickness of 3 mm, T2-weighted spin echo imaging with a slice thickness of 3 mm, heavy-weighted T2 imaging with a slice thickness of 0.5 mm (ie, Drive sequence on Philips MRI scan). All MRI exams were performed with a 1.5 Tesla scan. After receiving the instructions from our referring neuroradiologist on how to visualize the trigeminal nerve in Meckel’s cave on MRI scans, 3 of the coauthors (AK, EL, MM) interpreted the scans while being blinded to trigeminal symptoms/signs. Consequently, there was no bias during MRI scan interpretation.

Criteria were analyzed on MRI scans in an attempt to find a correlation with trigeminal symptoms/signs. First, the authors tried to correlate preoperative MRI scans with preoperative trigeminal symptoms/signs. Analyzed criteria were: (1) stage of the tumor (4 or 5), (2) infiltration of the internal acoustic canal (IAC), (3) nonvisibility of ipsilateral trigeminal nerve, (4) tumor cysts or necrosis, (5) infiltration of tentorium cerebelli, (6) peritumoral edema, and (7) impaction of tumor on ipsilateral cerebellar peduncles, in the plane of the middle cerebellar peduncles, which is at the same axial level as the IAC (Figures 1 and 2). Second, the authors tried to correlate postoperative MRI scans with postoperative trigeminal symptoms/signs. Analyzed criteria were: (1) presence of residual tumor, (2) edema of cerebellar peduncles, (3) nonvisibility of ipsilateral trigeminal nerve, and (4) impacted ipsilateral cerebellar peduncles (Figure 3).

Data analysis was performed using the Pearson $\chi^2$ statistical test and was validated by the Department of Statistics of our institution. Study ethics approval was obtained by our Regional Ethics Committee: CECIC Rhône-Alpes-Auvergne, Clermont-Ferrand, IRB 5891.

**Results**

Fifty-three patients were operated on; there were 27 females and 26 males (sex ratio F/M 1.04). The mean age was 51 years (range, 19-78). There was an additional patient operated on for a giant acoustic neuroma, but this patient was excluded from the study because he died on postoperative day 3 secondary to cerebral thrombophlebitis; therefore, the follow-up period was not enough. The neurotologic surgical approach was mainly a translabyrinthine approach: translabyrinthine (44), retrosigmoid (5), transcochlear/transotic (2), and retrolabyrinthine (2).

Preoperatively, the most common symptom was hearing loss, with 44 of 53 patients (83%) having moderate, severe, profound, or total hearing loss. The 3 branches of the trigeminal nerve, namely, ophthalmic branch (V1), maxillary branch (V2), and mandibular branch (V3) were almost equally affected: V1 = 28 cases, V2 = 28 cases, and V3 = 26 cases. All cases of V1 hypoesthesia had some degree of decreased corneal reflex, but only cases with completely absent reflex were mentioned. Absence of corneal reflex (V1 branch) occurred in 16 patients (30.2%), among whom 4 patients had facial nerve paralysis House-Brackmann (HB) grade ≥IV; absence of corneal reflex could thus have been
Figure 2. Preoperative magnetic resonance imaging (MRI) criteria (in addition to tumor stage) that were sought to find any eventual correlation with preoperative trigeminal nerve involvement. (A) and (B): Axial T1 spin echo with injection of gadolinium. Infiltration of internal acoustic canal (IAC). (C): Axial T2 spin echo. Cystic tumor (intense signal). (D): Drive sequence with coronal reconstruction. Infiltration of tentorium cerebelli. (E): Axial T2 spin echo. Comparison of length of both middle cerebellar peduncles. (F) Axial drive sequence. Visibility of trigeminal nerve (V) and peritumoral edema.

Figure 3. Postoperative magnetic resonance imaging (MRI) criteria that were sought to find any eventual correlation with postoperative trigeminal nerve involvement. (A): Axial drive sequence. Visibility of trigeminal nerve (V). (B) Axial T2 spin echo. Peduncular edema. (C) Axial T1 spin echo with injection of gadolinium. Presence of residual tumor and comparison of length of both middle cerebellar peduncles.
due to facial nerve, trigeminal nerve, or both. One patient with absent corneal reflex had normal V1 sensation and of course HB grade ≥IV facial nerve paralysis. Keratitis occurred in 1 patient (1.9%) preoperatively.

On postoperative day 1, a patient needed a second surgery to remove a hematoma in the cerebellopontine angle (CPA). A small tumor remnant (less than 10% of initial tumor volume) was left in 14 patients (26.4%): residual tumor was kept in place due to tumor adhesions to the brainstem, facial and/or trigeminal nerve, cerebellum, or internal carotid artery. Postoperatively, absence of the corneal reflex and hypoesthesia of the face were the main trigeminal signs. Hypoesthesia was also used for correlation with 3-month postoperative MRI findings. The V1 branch was affected as often as V2 and V3 branches (21 cases for each). Absence of corneal reflex (V1 branch) occurred in 26 patients (49%), among whom 23 patients had facial nerve paralysis HB grade ≥IV; absence of corneal reflex could thus be due to facial nerve, trigeminal nerve, or both. Six patients had normal V1 sensation and of course HB grade ≥IV facial nerve paralysis. Keratitis occurred in 11 patients (20.7%) postoperatively, among whom 6 had facial nerve paralysis HB grade <IV. In these cases, keratitis was exclusively neuroparalytic, namely, due to only V1 branch involvement, because patients with HB grade <IV have a fairly good orbicularis oculi motricity. Table 2 summarizes preoperative and early postoperative (at 10 days) clinical manifestations of trigeminal nerve involvement.

The median follow-up period in our institution was of 4 years (6 months-8 years). At 1 year follow-up, 27 patients (51%) had HB grade I-II facial nerve function, 17 patients (32%) had grade III-IV, and 9 patients (17%) had grade V-VI. Also at 1 year, initial preoperative trigeminal hypoesthesia (28 patients) improved or ceased in 10 of 28 patients (35.7%) and worsened in 4 of 28 (14.3%). Postoperatively, there was de novo appearance of trigeminal hypoesthesia in 2 of 53 patients (3.8%) otherwise not having trigeminal symptoms/signs preoperatively. Trigeminal neuralgia ceased in both postoperative cases and trigeminal anesthesia persisted on the long term in the single patient who had early postoperative anesthesia. There was no case of mastication problems on the short or long term.

After analyzing preoperative MRI, there was only 1 criterion that was statistically correlated with preoperative trigeminal nerve hypoesthesia: impacted cerebellar peduncles (cp): $P < .05$ Pearson $\chi^2$ test. This was evaluated by calculating the ratio of ipsilateral cp/contralateral cp. The optimal cut point ratio was 0.36, with both a good specificity (74%) and sensitivity (71%). Tumor infiltration of the IAC was statistically significant ($P < .05$ Pearson $\chi^2$ test) for hearing loss with a proportionate relationship between the degree of hearing loss and extension of the tumor into the IAC. However, there was no correlation between IAC infiltration and trigeminal nerve involvement ($P > .05$ Pearson $\chi^2$ test); paradoxically, there was more trigeminal hypoesthesia in cases of IAC infiltration than in cases with no IAC infiltration (Table 3). Tumor stage, nonvisibility of ipsilateral trigeminal nerve, cystic or necrotic tumor, infiltration of tentorium cerebelli, and peritumoral edema were all not

### Table 1. Summary of Preoperative and Early Postoperative (Day 10) Clinical Manifestations.

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>Preoperative n (%)</th>
<th>Early Postoperative n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal hearing or mild HL</td>
<td>10 (18.9)</td>
<td>0</td>
</tr>
<tr>
<td>Moderate HL</td>
<td>10 (18.9)</td>
<td>2 (3.7)</td>
</tr>
<tr>
<td>Severe HL</td>
<td>11 (20.7)</td>
<td>2 (3.7)</td>
</tr>
<tr>
<td>Profound HL/anacusis</td>
<td>23 (43.4)</td>
<td>50 (94.3)</td>
</tr>
<tr>
<td>Vertigo/dizziness</td>
<td>32 (60.4)</td>
<td>10 (18.9)</td>
</tr>
<tr>
<td>Significant tinnitus</td>
<td>15 (28.3)</td>
<td>10 (18.9)</td>
</tr>
<tr>
<td>Walking disturbances/ataxia</td>
<td>17 (32.1)</td>
<td>8 (15.1)</td>
</tr>
<tr>
<td>Grade I-II facial nerve paralysis</td>
<td>48 (90.6)</td>
<td>9 (17)</td>
</tr>
<tr>
<td>Grade III-IV facial nerve paralysis</td>
<td>3 (5.7)</td>
<td>17 (32.1)</td>
</tr>
<tr>
<td>Grade V-VI facial nerve paralysis</td>
<td>2 (3.7)</td>
<td>27 (50.9)</td>
</tr>
<tr>
<td>Lower cranial nerve deficit</td>
<td>2 (3.7)</td>
<td>2 (3.7)</td>
</tr>
<tr>
<td>Signs of ICHT</td>
<td>14 (26.4)</td>
<td>0</td>
</tr>
</tbody>
</table>

**Abbreviations:** HL, hearing loss; ICHT, intracranial hypertension.

*Significant tinnitus was defined as permanent and incapacitating.

### Table 2. Summary of Preoperative and Early Postoperative (Day 10) Clinical Manifestations of Trigeminal Nerve.

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>Preoperative n (%)</th>
<th>Early Postoperative n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuralgia of the face</td>
<td>3 (5.7)</td>
<td>2 (3.8)</td>
</tr>
<tr>
<td>Anesthesia of the face</td>
<td>1 (1.9)</td>
<td>1 (1.9)</td>
</tr>
<tr>
<td>Hypoesthesia of the face</td>
<td>28 (52.8)</td>
<td>24 (45.3)</td>
</tr>
<tr>
<td>Absence of corneal reflex</td>
<td>16 (30.2)</td>
<td>26 (49)</td>
</tr>
<tr>
<td>Excluding FNP grade ≥IV</td>
<td>12 (22.6)</td>
<td>3 (5.7)</td>
</tr>
<tr>
<td>Keratitis</td>
<td>1 (1.9)</td>
<td>11 (20.7)</td>
</tr>
<tr>
<td>Excluding FNP grade ≥IV</td>
<td>0</td>
<td>6 (11.3)</td>
</tr>
</tbody>
</table>

*FNP grade ≥IV, facial nerve paralysis House-Brackmann grade ≥IV, namely, with absence of eye closure.
Acoustic neuromas or vestibular schwannomas are benign tumors arising in the Schwann cell sheath of the vestibular nerve, at the neurilemmal-neuroglial junction. This junction and thus the origin and extension of the tumor could vary in position. Therefore, clinical manifestations of acoustic neuromas could vary accordingly, in terms of hearing loss, trigeminal nerve deficit, and other cranial nerve involvement. One explanation would be anatomical, due to the vicinity of the trigeminal nerve, followed by the trigeminal nerve, and then the other cranial nerves.

The incidence of giant tumors varies from 2% to 12.5% among all acoustic neuromas in Western countries. In these tumors, the cochlear nerve is the most frequently involved cranial nerve, followed by the trigeminal nerve, and then the lower cranial nerves. Other significant consequences of these large tumors are compression of the brainstem, cerebellum, and middle cerebellar peduncles and occurrence of intracranial hypertension. We found a statistically significant relationship between compression of cerebellar peduncles on preoperative MRI and preoperative trigeminal hypoplasia. The size of ipsilateral and contralateral cerebellar peduncles was measured in the axial plane of the middle cerebellar peduncles, which are in the same plane of the IACs. In the literature, the authors found no straightforward relationship between cerebellar peduncle compression and trigeminal nerve involvement. One explanation would be anatomical, due to the vicinity of the trigeminal nuclei to the cerebellar peduncles, particularly the middle peduncles.

In cases of large and especially giant tumors (stage 5), surgical outcomes are not always optimal, due to compression and adherence to the brainstem, cerebellar peduncles, and cranial nerves. We found a statistical correlation between nonvisibility of the trigeminal nerve on postoperative MRI and postoperative trigeminal hypoplasia. The nonvisibility of the trigeminal nerve on preoperative MRI scan does not necessarily mean that the nerve is damaged, because it can be laminated or just hidden by the tumor. Conversely, postoperative nonvisibility of the nerve might likely reflect nerve damage, because the tumor has been removed, regardless if damage occurred during surgery or was caused by the tumor itself. On the other hand, the nerve could be also scarred down to adjacent structures due to postoperative alterations, making its visibility more difficult than in normal conditions.

As compared with other series of large acoustic neuromas, the present series shows more frequent preoperative involvement of trigeminal nerve, even in the stage 4 group. Also, postoperative trigeminal nerve deficit was not always mentioned. The reason for this could be that postoperatively other morbidities and cranial nerve deficits (facial, cochlear) deter the clinician’s attention from seeking trigeminal nerve symptoms. Mid- and long-term follow-up of patients in institutions other than those where surgery was performed might be another reason for the underestimation of trigeminal nerve symptoms and signs.

There are some sparse publications in the literature reporting on single cases of acoustic neuroma and trigeminal nerve symptoms, without suggesting the underlying mechanisms of nerve involvement. Some explanations provided by other larger studies are that the tumor exerts direct pressure on the trigeminal nerve, which causes demyelination of sensory roots and neuralgia. Another mechanism is that the tumor pushes the nerve against an artery (superior cerebellar or basilar) or vice versa, thus mimicking a neurovascular conflict. It has been suggested that large tumors affect the trigeminal nerve mainly by direct compression, whereas smaller tumors cause neurovascular conflict. Other risk factors mentioned in the literature are tumor growth toward the brainstem in an upward direction and cyst formation, although the latter was not statistically correlated with trigeminal nerve involvement in the present series. Moffat et al divided acoustic neuromas into 3 categories according to their morphology and extension from the IAC to the CPA: dumbbell, cone, and lollipop-shaped tumors, respectively. The more the tumor was out of the IAC and extending into the CPA, the more the trigeminal nerve was involved, and the less the hearing was affected. In the present study, we found a statistical correlation between IAC infiltration and hearing loss, but not trigeminal nerve involvement. It has been suggested that when the tumor extends out of the porus acusticus, it touches the trigeminal nerve when its size reaches 20 mm. Patients present late with trigeminal and cerebellar signs more frequently than hearing loss.

According to the literature, the ophthalmic branch (V1) of the trigeminal nerve seems to be less frequently affected than the maxillary (V2) and mandibular (V3) branches. In the present series, V1 was almost as frequently affected as V2 and V3 pre- and postoperatively. V1 involvement is a serious complication due to absence of corneal reflex,
which exposes the patient to eye dryness and sometimes keratitis, especially if associated with facial nerve paralysis, HB grade IV and above, where there is no eye closure. Furthermore, keratitis becomes painless because of loss of sensation, which causes late presentation and corneal damage. This is a serious complication that is often underestimated in the literature. Moffat et al. reported a 100% rate of decreased corneal reflex in patients with lollipop tumors, but the percentage of keratitis was not mentioned. In the present series, the percentage of keratitis increased dramatically after surgery, due to the increase in the rate of facial nerve paralysis and worsening of facial nerve function postoperatively (1 case HB ≥ IV preoperatively vs 5 cases HB ≥ IV postoperatively), and also to worsening of trigeminal nerve function, secondary to surgical dissection and tumor removal. Therefore, every time there is V1 hypoesthesia and/or facial nerve paralysis HB grade ≥ IV, the authors recommend eye care as well as regular ophthalmologic follow-up.

Conclusion
In large and compressive acoustic neuromas, trigeminal nerve deficit has to be sought to diagnose an eventual facial hypoesthesia and particularly avoid corneal complications. Trigeminal hypoesthesia occurs preoperatively in about half of the cases. It remains relatively stable after tumor removal, but there appears to be an increased rate of absent corneal reflex and keratitis postoperatively. Preoperative trigeminal hypoesthesia was statistically correlated with tumor impaction on cerebellar peduncles on preoperative MRI. Postoperative trigeminal hypoesthesia was statistically correlated with nonvisibility of the trigeminal nerve on postoperative MRI.

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Author Contributions
Alexandre Karkas, data acquisition and analysis, manuscript drafting and definitive writing, responsible for data accuracy, final approval; Eléa Lamblin, data acquisition and analysis, manuscript drafting; Mikael Meyer, data acquisition and analysis, manuscript drafting; Emmanuel Gay, data acquisition and analysis, manuscript drafting; Jessica Ternier, data acquisition and analysis, manuscript corrections, final approval.

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