## **ORIGINAL ARTICLE**



# Preservation of cranial nerve function in large and giant trigeminal schwannoma resection: a case series

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## Abstract

**Background** Trigeminal schwannomas (TSs) are intracranial tumors that can cause significant brainstem compression. TS resection can be challenging because of the risk of new neurologic and cranial nerve deficits, especially with large ( $\geq 3$  cm) or giant ( $\geq 4$  cm) TSs. As prior surgical series include TSs of all sizes, we herein present our clinical experience treating large and giant TSs via microsurgical resection.

**Methods** This was a retrospective, single-surgeon case series of adult patients with large or giant TSs treated with micro-surgery in 2012–2023.

**Results** Seven patients underwent microsurgical resection for TSs (1 large, 6 giant; 4 males; mean age  $39 \pm 14$  years). Tumors were classified as type M (middle fossa in the interdural space; 1 case, 14%), type ME (middle fossa with extracranial extension; 3 cases, 43%), type MP (middle and posterior fossae; 2 cases, 29%), or type MPE (middle/posterior fossae and extracranial space; 1 case, 14%). Six patients were treated with a frontotemporal approach (combined with transmastoid craniotomy in the same sitting in one patient and a delayed transmaxillary approach in another), and one patient was treated using an orbitofrontotemporal approach. Gross total resection was achieved in 5 cases (2 near-total resections). Five patients had preoperative facial numbness, and 6 had immediate postoperative facial numbness, including two with worsened or new symptoms. Two patients (28%) demonstrated new non-trigeminal cranial nerve deficits over mean follow-up of 22 months. Overall, 80% of patients with preoperative facial numbness and 83% with facial numbness at any point experienced improvement or resolution during their postoperative course. All patients with preoperative or new postoperative non-trigeminal tumor-related cranial nerve deficits (4/4) experienced improvement or resolution on follow-up. One patient experienced tumor recurrence that has been managed conservatively.

**Conclusions** Microsurgical resection of large or giant TSs can be performed with low morbidity and excellent long-term cranial nerve function.

Keywords Case series · Cranial nerve · Microsurgery · Resection · Trigeminal schwannoma

# Introduction

Trigeminal schwannomas (TSs) are rare (0.07-0.3%) intracranial tumors that arise from Schwann cells of the trigeminal nerve sheath [4, 8, 24]. They usually originate within the Gasserian ganglion in Meckel's cave, in the cavernous sinus, or along the three branches of the trigeminal nerve (V1, V2, V3) [22]. Patients with TSs will commonly present with headache, diplopia, proptosis, facial numbness, and facial pain, with gait disturbances possible if the tumor is large enough to cause brainstem compression [4, 15]. TSs can grow to impressive proportions and may be characterized as giant when spanning  $\geq 4$  cm [19]. In cases of larger size, TSs often impart more severe symptoms and can damage critical surrounding neurovascular structures [25]. To prevent further tumor growth and alleviate symptoms, microsurgical resection is the preferred treatment for large and giant symptomatic TS [6, 21].

Although the rates of morbidity and mortality have substantially improved in recent decades, TS resection remains

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an invasive procedure with a high risk for neurologic deficits, regardless of tumor size [6, 29]. Historically, the goal of open surgery has been to resect the tumor to the maximal safe extent and decompress surrounding neurovascular structures, with the expectation that trigeminal symptoms and other existing deficits may persist. However, in selected cases, excellent outcomes and resolution of other cranial nerve (CN) palsies can be acheived [39]. Although TSs of large or giant size are reported sporadically across previous series, there is a dearth of studies specific to the surgical outcomes and CN preservation in patients with exclusively large and/or giant TSs. Therefore, to more definitively assess the long-term safety and efficacy of open microsurgical management of large or giant TSs, we present a single surgeon's experience in treating 7 patients with large or giant TSs via microsurgical resection and discuss strategies for optimal preservation of CN function.

## Methods

This work was performed with local Institutional Review Board approval (with a waiver of informed consent) and has been reported in line with the PROCESS guidelines [5].

#### Study methodology and patient population

A retrospective, single-institution chart review of the surgeon case log was performed to identify patients with large or giant pathology-confirmed TS (tumors were classified as large if the maximum diameter was  $\geq 3$  cm and giant if the maximum diameter was  $\geq 4$  cm). Patients who underwent microsurgical resection by the senior author from April 2012 to July 2023 were included [19]. Patients < 18 years old were excluded.

## **Collected data**

Demographics and comorbidities were captured from the manual review of all available clinical documentation for all patients who fit the inclusion and exclusion criteria. Presenting symptoms including trigeminal nerve dysfunction and other CN deficits were collected, as were tumor characteristics including size and location. Tumor location was described based on the Kawase classification [40]. Specifically, tumors involving one compartment were classified as type M (middle fossa in the interdural space), type P (posterior fossa in the subdural space), or type E (extracranial in the epidural space). Tumors involving multiple compartments were classified into one of three types: type MP (dumbbell-shaped in the middle and posterior fossa), type ME (dumbbell-shaped in the middle fossa and extracranial space), or type MPE (involving three compartments: the

middle and posterior fossae and extracranial space). Operative details regarding approach, staging, resection status, and intraoperative complications were recorded. Postoperative deficits and their recovery at multiple time points (immediate, 1-month, and last available follow-up) were captured.

## Results

## **Demographics and clinical data**

Seven patients (4 male, 3 female) met study inclusion criteria. The patient ages ranged from 22 to 57 years (mean  $\pm$  standard deviation 38.6  $\pm$  14.3), and body mass index ranged from 20.9 to 42.8 (mean 27.2  $\pm$  7.8) (Table 1). Tumor size ranged from 3 to 7 cm (mean 5.2  $\pm$  1.5; 1 large, 6 giant). Preoperatively, 5/7 patients (71%) had trigeminalspecific symptoms, and 3/7 (43%) had likely tumor-related non-trigeminal cranial neuropathies. The trigeminal-specific symptoms included V1, V2, and V3 numbness with

Table 1 Baseline patient characteristics

Variable	Value
Female sex	3 (43)
Age (years)	$38.6 \pm 14.3$
BMI	$27.2 \pm 7.8$
Pre-existing comorbidities	3 (43)
Tumor size (cm)	$5.2 \pm 1.5$
Average duration of symptoms before surgery (months)	$14.0 \pm 9.7$
Trigeminal-specific deficits	5 (71)
V1 numbness ± pain	3 (43)
V2 numbness $\pm$ pain	4 (57)
V3 numbness $\pm$ pain	1 (14)
Additional tumor-related non-trigeminal cranial neuropathies	3 (43)
CN II	1 (14)
CN III	1 (14)
CN IV	1 (14)
CN VI	1 (14)
CN VIII	1 (14)
CN IX/X	1 (14)
Tumor location (Kawase classification)	
Type M	1 (14)
Type ME	3 (43)
Type MP	2 (29)
Type MPE	1 (14)

Value reported as mean  $\pm$  SD or no. (%)

*BMI* body mass index; *CN* cranial nerve; *M* middle fossa in the interdural space; *ME* dumbbell-shaped in the middle fossa and extracranial space; *MP* dumbbell-shaped in the middle and posterior fossae; *MPE* involving three compartments: the middle and posterior fossae and extracranial space; *SD* standard deviation or without pain (present in 43%, 57%, and 14% of patients, respectively). Non-trigeminal neuropathies included CN III palsy (14%), CN IV palsy (14%), CN VI palsy (14%), decreased palatal raise (14%), hearing loss (14%), and vision loss (14%). The average time from symptom manifestation to surgical intervention was  $14.0 \pm 9.7$  months. No patient had received prior TS treatment. Tumor locations based on the Kawase classification included: 1 M (14%), 3 ME (43%), 2 MP (29%), and 1 MPE (14%). Additional patient-specific details are described in Table 2.

Four patients (57%) were treated with a standalone frontotemporal approach, two patients (29%) were treated with a frontotemporal approach combined with a second approach (transmastoid or transmaxillary), and one patient (14%) was treated using an orbitofrontotemporal approach (Table 3, Supplemental Videos 1 and 2). Combined or extended approaches were used to remove extracranial tumor extensions into the middle ear/jugular foramen (transmastoid), pterygopalatine fossa (transmaxillary), and orbit (orbitofrontotemporal). Only 1 case (patient 1) involved a staged approach (frontotemporal craniotomy for resection of middle fossa inter/intradural and subtemporal tumor, followed by a transmaxillary approach for resection of tumor within the pterygopalatine fossa), with the remaining resections being performed in a single surgery. Five patients (71%) had a gross total resection and 2 (29%) had a near-total resection (adherent thin capsule left on the brainstem and facial nerve, respectively). No intraoperative complications occurred.

Histological analysis confirmed all tumors were World Health Organization (WHO) Grade 1 TSs (Table 4) [34]. No patient experienced any complications unrelated to CN dysfunction. Follow-up of  $\geq 1$  month was available for all patients (mean 22 months). Of the 5 patients (71%) with preoperative facial numbness, none experienced immediate postoperative improvement. Two patients (28%) had worsened or new facial numbness (patients 1 and 4) postoperatively. In total, 6/7 (86%) of patients had postoperative facial numbness, although 5/6 (83%) experienced improvement at 1-month follow-up. Among patients with longer follow-up, 3/4 (75%) experienced complete resolution. Notably, all patients with preoperative or new postoperative V1 hypoesthesia with > 1-month follow-up had resolution by 12 months. No patient required surgical management for V1-related neurotrophic keratitis. All three patients (100%) with pre-existing tumor-related non-trigeminal cranial neuropathies (affecting CNs II, III, IV, VI, VIII, and IX/X) demonstrated symptom improvement at 1-month follow-up, and 2/3 (66%) had complete resolution on long-term follow-up. Two patients (28%) experienced worsened or new immediate postoperative non-trigeminal CN deficits, including CN III and VI palsies (patient 5) and a CN VI palsy (patient 6). Both of these patients had improvement or complete resolution of these CN deficits on follow-up (45 months and 1 month, respectively). One patient (patient 2) experienced tumor recurrence that has been conservatively managed during their 66-month postoperative follow-up. No patients have received adjuvant radiation at any time.

## Illustrative case (patient 4)

A 23-year-old woman with no previous medical concerns presented with a 4-month history of worsening balance with daily falls, dysphagia, hearing loss, headaches, blurry vision, and diplopia. Physical examination demonstrated mild anisocoria with left pupillary dilation, as well as left-sided ophthalmoparesis in the CN III and IV distribution. Palatal raise and hearing was decreased on the left side, and the patient exhibited dysmetria on left-sided finger-to-nose testing. Magnetic resonance imaging (MRI) of the brain demonstrated a giant (7.0×5.5×2.2-cm) left-sided Kawase classification MP tumor extending from the prepontine cistern, expanding the petrous apex/Meckel's cave, and growing into the middle cranial fossa interdural space (Fig. 1). The tumor exerted significant mass effect on surrounding structures, including compression of the brainstem. Considering the progressive and debilitating constellation of symptoms as well as the mass effect on critical neurological structures, resection was offered to and accepted by the patient.

A left frontotemporal craniotomy was performed, followed by an extradural transcavernous approach (Fig. 1). Additional bone was drilled to unroof the superior orbital fissure, foramen rotundum, and foramen ovale. The superior orbital fissure, V1, V2, and V3 were identified, and tumor debulking/resection was performed through the anteromedial and anterolateral triangles and tumor-expanded corridors. In this case, the tumor created a corridor to enable visualization and resection of the posterior fossa component compressing the brainstem. A near-total resection was achieved, including resection of a portion of tumor-infiltrated abducens nerve, which was subsequently primarily repaired. A thin adherent aspect of the capsule was left on the brainstem to avoid injury, with the endoscope used to ensure no hidden residual tumor remained. The tumor was confirmed to be WHO Grade 1 TS on pathological report. The patient had an uneventful hospital course and was discharged to inpatient rehabilitation on postoperative day 4 at her neurologic baseline (including CN III and VI palsies) with new mildly decreased V1-2 sensation. At 1-month follow-up, she had improvement of her left CN III palsy, improved palate movement and hearing, and improved facial numbness. At last follow-up (37 months), she had resolved facial numbress, palatal weakness, and hearing and improvement in her CN III/VI palsies. Longitudinal imaging has shown no evidence of tumor recurrence (Supplemental Video 1).

Image: controlling integration         Integration         Image: controlling integration         Controling i	Case	Age/ Sex	Medical	Symptoms			Tumor description				Deficits		
I         HM         Num         LV12 numbers         Num         LV13 muthers         Num         Num         Num         Numbers         Num         Numbers         Num         Num </th <th></th> <th></th> <th>comorbidities</th> <th>Initial</th> <th>Subsequent</th> <th>Duration (months)</th> <th>Location</th> <th>Kawase Classification</th> <th>Size (cm)</th> <th>CN V structures impacted</th> <th>CN V deficits</th> <th>Additional cranial neuropathies</th> <th>Other neurological deficits</th>			comorbidities	Initial	Subsequent	Duration (months)	Location	Kawase Classification	Size (cm)	CN V structures impacted	CN V deficits	Additional cranial neuropathies	Other neurological deficits
2       2Pro       Boyostic potostic potosti	_	47/M	None	L V1,2 numbness and pain	None	12	Left middle fossa interdural and intradural, infratemporal/ pterygopalatine fossae	ME	9	V1-3, ganglion, root	Decreased L corneal reflex, altered V1, 2 sensation	None	None
3       3/M       Note       R V1-3 facial       Note       R V1-3 facial       Unductors       Interluctor         4       2.97       Note       Balanceisae       Svallovits       1       1       V1-3 gangior       R V1-3 facial       Interluctor         6       2.97       Note       Balanceisae       Svallovits       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1       1	0	22/F	Polycystic ovarian syndrome	R proptosis	V1 numbness	24	Right middle fossa interdural, orbit	ME	4.4	V1-3, ganglion	R V1 numbness	None	None
4       23/F       None       Balance issues       Swallowing, HA, since interdual, int	ς.	37/M	None	R V I-3 facial numbness	None	Q	Right middle fossa interdural, posterior fossa subdural, infratemporal/ pterygopalatine fossae, middle car, jugular foramen	MPE	Q	V1-3, ganglion	R V1-3 facial numbness	Unrelated decreased hearing on R (trauma as a child)	None
5       54/F       Hypothyroid.       L visual loss       None       4       Left middle fossa       M       3       V1-3, ganglion       None       L CN II       None         6       57/M       Asthma       R V2 numbness       Unsteady gait       24       Right middle       ME       5       V1-3, ganglion       R V2 numbness       None       None         7       23/M       Asthma       R V2 numbness       Unsteady gait       24       Right middle       ME       5       V1-3, ganglion       R V2 numbness       None         7       29/M       None       L V2 numbness       Diplopia       24       Left middle fossa       MP       4       V1-3, ganglion       LV2 numbness       None       None         7       29/M       None       L V2 numbness       Diplopia       24       Left middle fossa       MP       4       V1-3, ganglion       LV2 numbness       LCN IV       None         8       29/M       None       L V2 numbness       Diplopia       24       Left middle fossa       Not	4	23/F	None	Balance issues	Swallowing difficulty, HA, blurry vision, diplopia, hearing issues	4	Left middle fossa interdural, posterior fossa subdural causing significant brainstem compression	dW	7	V1-3, ganglion, root	None	L CN III, VI, VIII, IX/X	None
6       57/M       Asthma       R V2 numbness       Unsteady gait       24       Right middle       ME       5       V1-3, ganglion,       R V2 numbness       None       None         7       29/M       None       L V2 numbness       Diplopia       24       Left middle fossa       None       V1-3, ganglion,       R V2 numbness       None       None         7       29/M       None       L V2 numbness       Diplopia       24       Left middle fossa       MP       4       V1-3, ganglion,       L V2 numbness       L CNIV       None         1       29/M       None       L V2 numbness       Diplopia       24       Left middle fossa       MP       4       V1-3, ganglion,       L V2 numbness       L CNIV       None         1       29/M       None       L V2 numbness       Diplopia       24       Left middle fossa       Not       L N-3, ganglion,       L V-3, ganglion,       L N-3, ganglion,       L N-3, ganglion,       L N-13, ganglion,       None         1       29/M       None       L V2 numbness       Diplopia       24       Interfunal,       None       L N-13, ganglion,       L N-14, gan	Ś	54/F	Hypothyroid- ism	L visual loss	None	4	Left middle fossa interdural	М	n	V1-3, ganglion	None	L CN II	None
7 29/M None L V2 numbness Diplopia 24 Left middle fossa MP 4 V1-3, ganglion, L V2 numbness L CN IV None interdural, root posterior fossa subdural causing significant brainstem compression	Q	57/M	Asthma	R V2 numbness	Unsteady gait	24	Right middle fossa interdural, posterior fossa subdural causing significant brainstem compression	ME	Ś	V1-3, ganglion, root	R V2 numbness	None	None
	Г	29/M	None	L V2 numbness	Diplopia	24	Left middle fossa interdural, posterior fossa subdural causing significant brainstem compression	ЧМ	4	V1-3, ganglion, root	L V2 numbness	L CN IV	None

## Discussion

Large and giant TSs are rare entities that have been sporadically described in the literature. To the best of our knowledge, this is the first report comprising patients with exclusively large and giant TSs. In these 7 patients whose large/giant TSs were managed via microsurgery, we observed a 0% intraoperative complication rate, a 0%mortality rate, and an 80% cure rate over the available follow-up. Although putting these rates into context is difficult because of the current lack of multiple-patient studies describing exclusively large or giant TSs, similarly low adverse event rates are reported among studies that assessed resection of larger TSs in conjunction with TSs < 3 cm [3, 12, 32]. This reflects the great improvement of modern microsurgical techniques compared with the unacceptably high (up to 25%) morbidity and mortality reported in earlier TS surgical analyses [9, 27, 33]. More importantly, our data suggest that microsurgery for large/ giant TSs can be an effective treatment with minimal longterm negative effects. With an emphasis on factors particular to increased tumor size, we discuss important considerations that must be factored to optimize TS management.

#### Trigeminal schwannoma management strategies

In accordance with the guidance of Niranjan et al. [26], we believe that observation should be primarily considered for small asymptomatic intracranial tumors, including TSs. However, for symptomatic or progressive TSs, treatment modalities include stereotactic radiosurgery (SRS) and resection (using open microsurgical or endoscopic techniques), with gross-total resection being the gold standard because it imparts the best long-term chance of cure [10, 29, 37]. A recent meta-analysis of 553 patients treated with SRS demonstrated a 92.3% rate of tumor control over an estimated > 4-year follow-up with radiosurgery, with TS neuralgia improving in 63.5% of cases; however, tumor progression rates after SRS were 9.4% and clinical worsening was reported in 10.7% of cases [28]. Considering the high cure rate achieved through microsurgery (>90%), SRS has been accepted as an adjuvant therapy for larger symptomatic or recurrent tumors and as a primary intervention for small, asymptomatic, or inaccessible TS [6, 16]. For large or giant TSs, microsurgery is the only treatment modality able to remove local mass effect, making it the preferred first-line intervention.

All cases in the present series were managed microsurgically because each patient exhibited undesirable symptoms related to their TS and all patients had tumors  $\geq$  3 cm in size. SRS was not used in these patients as either primary or adjuvant therapy. Two patients required separate surgical approaches for complete resection (patients 1 and 3, one staged/one in a single operation) owing to significant tumor size and multicompartment extension. Although a single-trajectory operation is used whenever possible, combined approaches may be required for TSs that are excessively large or dumbbell-shaped (especially when spanning multiple intracranial compartments) or for tumors that are highly vascular, adherent, or fibrous [11, 36, 40].

#### Surgical approaches to large and giant TSs

Common surgical approaches for large and giant TS resection include the frontotemporal, subtemporal, fronto-orbital, and retrosigmoid approaches, as well as the anterior transmaxillary (Caldwell-Luc) and the more recently developed expanded endonasal endoscopic approach. Because of its flexibility and simplicity, the frontotemporal (or pterional) approach and its variations are widely employed for supratentorial tumors and can likewise be an effective approach for accessing TSs predominantly within the middle cranial fossa [23, 31]. This approach can be modified as needed to include access to the cavernous sinus, superior orbital fissure, orbit, foramen rotundum, foramen ovale, and infratemporal fossa by inclusion of an extradural frontopolar dissection and selective drilling of the lesser/greater sphenoid wings, orbital roof, and middle fossa floor [1, 2, 13, 23]. Orbital bar removal can also be included as needed. Transcavernous variations of the frontotemporal approach, as predominately used in this series, are especially useful for large and giant TSs because of expanded operative corridors from bony erosion and widening of cavernous sinus triangles (particularly the anteromedial and anterolateral triangles and anterior petrosal corridor as illustrated in patient 4) and Meckel's cave. This technique can be used to resect selected tumors that include a large posterior fossa component by focusing the approach along the long access of the tumor and using the large tumor cavity and widened bony corridors during resection. In such cases, the orientation of the tumor body relative to the trigeminal branches can help the surgeon anticipate the location of the tumor/nerve interface, which is better visualized after debulking. Similarly, tumor from the intracranial cavity can be followed anteriorly along V2 or V3 to remove extracranial components in selected cases to avoid a second approach. Although manipulation of the trigeminal branches is usually well tolerated because of their organized structure, avoidance of aggressive manipulation of the more disorganized trigeminal ganglion is important to maximize CN V outcomes. Although TSs primarily within the region of Meckel's cave or the posterior fossa can also be approached using a subtemporal (temporal craniotomy, with or without anterior petrosectomy) or retrosigmoid approach

Table 3	Operative	characteristics
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Case	Surgical indication	Surgical approach	Staged approach	Resection status	Intraoperative complications
1	Facial pain and numbness, giant tumor size	<ol> <li>Left frontotemporal craniotomy</li> <li>Left transmaxillary approach to the pterygopalatine fossa</li> </ol>	Yes (4 months between stages)	Initial: partial; Second-stage: GTR	None
2	Proptosis, facial numbness, giant tumor size	Right orbitofrontotemporal craniotomy	No	GTR	None
3	Facial numbness, giant tumor size	<ol> <li>Right frontotemporal craniotomy</li> <li>Right transmastoid craniotomy</li> </ol>	No	GTR	None
4	Difficulty ambulating, hearing issues, diplopia, giant tumor size	Left frontotemporal craniotomy	No	Near-total	None
5	Vision loss, large tumor size	Left frontotemporal craniotomy	No	GTR	None
6	Facial numbness and gait unsteadiness, giant tumor size	Right frontotemporal craniotomy	No	GTR	None
7	Facial numbness, diplopia, giant tumor size	Left frontotemporal craniotomy	No	Near-total	None

GTR gross-total resection

[38, 40, 41], they were not used in this series of type ME, MP, and MPE TSs (all with a large middle fossa component) and no type P tumors. For TSs that span multiple compartments, combined approaches can nonetheless be used as needed (in a single-stage or staged manner) to avoid performing tumor resection with suboptimal visualization. Endoscopic assistance was also used freely in this series to enhance intraoperative visualization and assess for hidden residual tumor.

Expanded endoscopic endonasal approaches have been increasingly reported for TSs located within Meckel's cave and with extension into the infratemporal fossa [7, 17, 20, 30, 37, 42, 43]. This minimally invasive approach can limit brain retraction and potentially reduce the manipulation of trigeminal structures, with good outcomes reported even for large or giant TSs [37, 42]. Such approaches can nonetheless have steep learning curves and require coordination of a dedicated skull base team for their safe performance. Given the relative rarity of large or giant TSs, the efficacy of endoscopic endonasal resection versus traditional microsurgery remains insufficiently elucidated [30].

#### Neurological outcomes

Facial numbness and/or paresthesias are perhaps the most common preoperative clinical symptoms [3, 29, 43]. In our study, five (71%) patients had preoperative facial numbness and two (28%) had worsened or new facial numbness postoperatively. These results resemble those of Aftahy et al. [3], who described the management of 55 TSs (> 50% being large or giant TSs) and reported a 23% rate of new trigeminal numbness after microsurgery. Regarding symptom improvement, in our study, 80% of patients with preoperative facial numbness and 83% of patients with facial numbness at any point experienced improvement or resolution during their postoperative course. These findings compare favorably with data from other series, in which many cases qualified as large or giant TSs, reporting an 11-44% rate of improvement in facial numbness postoperatively (follow-up periods ranging from 3 months to 11 years) [6, 13, 15, 18, 35]. Additionally, although four (57%) patients in our study had V1 symptoms either pre- or postoperatively, none required surgical treatment for neurotrophic keratitis, a serious potential complication of V1 neuropathy [14]. Affected patients were successfully managed with artificial tears as needed. Demonstrating the potential for similarly good outcomes for other non-trigeminal CN deficits after microsurgery, all patients with preoperative or postoperative non-trigeminal deficits experienced improvement or resolution on follow-up in our study. These results also potentially compare favorably with those of Zhang et al. [43], who reported a 42-patient cohort comprising mostly giant TSs (>50%) that demonstrated a 74% rate of improvement or stability of pre-existing non-trigeminal neuropathies; however, these data were recorded at the time of postoperative discharge and the authors did not comment on the long-term course of these deficits. Our findings are also comparable with those of Al-Mefty et al. [6], who reported 75-100% improvement in non-trigeminal CN deficits in a series of 25 patients (a portion of which were giant TSs) after a follow-up period ranging from 3 to 134 months (mean 33 months).

Overall, our data support the safe and effective performance of microsurgery for large and giant TSs, with improvement and long-term preservation of CN function an achievable goal in many cases. The goals of treatment should nonetheless always be tailored to individual patient circumstances, symptom severity, and tumor characteristics, and patients should be fully informed of the potential risks. Surgeons must remember that in patients who are largely

Case	WHO grade	Postoperative	Neurological deficits*			Length	Tumor regrowth/ recurrence	Adjuvant
		complications	Immediate postoperative	At 1 month postoperative	At last F/U	of F/U (months)		radiation
-	1	None	Stable L V1-2 numbness; <b>new</b> V3 numbness^	Improved L V1 and V3 numb- ness; stable L V2 numbness^	Resolved L V1 and V3 numb- ness; stable L V2 numbness	6^	No	No
5	1	None	Stable R V1 numbness	Improved R V1 numbness	None; resolved R V1 numbness	66	GTR with secondary intraorbital recurrence	No
ю	1	None	Stable R V1-3 numbness	Stable R V1-3 numbness	NA	1	No	No
4	-	None	Stable L CN III, VI, VIII, IX/X palsies; <b>new L V1-2 numb-</b> <b>ness</b>	Improved <b>L V1-2 numbness</b> ; improved L CN III, VI, VIII, IX/X palsies	Resolved L V1-2 numbness and CN VIII, IX/X palsies; improved L CN III and VI palsies	37	No	No
S	1	None	Improved L visual acuity (CN II); new L CN III, VI palsies	Stable L CN III, VI palsies; improved L visual acuity (CN II)	None; resolved L CN II, <b>III, VI</b> palsies	45	No	No
9	1	None	Stable R V2 numbness; new CN VI palsy	Improved R V2 numbness, gait, and CN VI palsy	NA	1	No	No
2	1	None	Stable L V2 numbness and left CN IV palsy	Improved L V2 numbness and L CN IV palsy	Improved L V2 numbness; resolved L CN IV palsy	2	No	No
WHC *Defi	Norld Health	Organization; L	left; R right; F/U follow-up; CN cr. ratively are in holdface type	anial nerve; NA not available				

Deficits that appeared only postoperatively are in boldface type ^Relative to initial surgery. No new neurologic deficits seen after the second-stage surgery

 Table 4
 Postoperative course



**Fig. 1** Preoperative imaging and operative course. A 23-year-old woman presented with a 4-month history of balance disturbance, dysphagia, hearing loss, headaches, blurry vision, and diplopia. Pre-operative imaging depicted a giant tumor centered at Meckel's cave causing significant compression of the brainstem and surrounding structures (A: axial T2-wieghted MRI without contrast; B: sagittal T1-weighted MRI with contrast). A left frontotemporal craniotomy was performed (C: incision; D: after bone flap removal and drilling of the sphenoid wing to the meningo-orbital band). An extradural transcavernous dissection was performed, including additional bone removal to expose the superior orbital fissure, V2, and V3 (E). The

tumor was removed through the anteromedial and anterolateral triangles and tumor-expanded corridors (F) An abdominal fat graft was used to assist with a watertight closure (G). The patient had an uneventful hospital course and was discharged on postoperative day 4 at her neurologic baseline. (H) One-week postoperative CT with contrast demonstrates bony resection in addition to residual blood products, with no evidence of tumor residual. (I) Axial T1-weighted MRI with contrast at 3 days postoperatively. At 1-month follow-up, the patient had improvement of her left CN III palsy, hearing, and facial numbness, with imaging showing no tumor residual. See Supplemental Video 1

asymptomatic preoperatively, any postoperative neurological deficits are an unwelcome development and can greatly diminish quality of life [26]. However, in patients experiencing brainstem compression or vision disruption in addition to trigeminal symptoms (such as the illustrative case outlined above), if only facial numbress remains postoperatively, this should be considered an excellent outcome by all parties [26].

#### Limitations

Limitations of this work include its small size, retrospective nature, and single-surgeon design, potentially limiting its broad applicability. Additionally, follow-up was heterogenous in the overall cohort (a product of our large geographic catchment area that includes multiple US states) and was not available for one patient, limiting the assessment of CN improvement for that case.

# Conclusion

Microsurgical resection of large and giant TSs can be performed with low rates of morbidity and mortality and excellent CN outcomes.

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Data availability All data are presented in this report.

Code availability Not applicable.

## Declarations

Ethics approval This work was performed with the approval of the University of Utah institutional review board with a waiver of informed consent.

**Consent to participate** The patient consented to participate.

**Consent for publication** The patient consented to the publication of this case and video.

Conflicts of interest None.

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