Published in final edited form as:
Otol Neurotol. 2012 September ; 33(7): 1270–1275. doi:10.1097/MAO.0b013e31825f2365.

Facial and Lower Cranial Neuropathies after Preoperative Embolization of Jugular Foramen Lesions with Ethylene Vinyl Alcohol

Brian C Gartrell, MD [(Primary author)],
University of Wisconsin - Madison, Department of Surgery, Division of Otolaryngology – Head and Neck Surgery, 600 Highland Ave, K4/719 CSC c/o Delight Hensler, Madison, WI 53792-7375, 402-910-4488, Fax: 608-252-0925

Marlan R Hansen, MD,
University of Iowa Department of Otolaryngology – Head and Neck Surgery, 200 Hawkins Dr, 21163 PFP, Iowa City, IA 52242-1078, 319-353-7151

Bruce J Gantz, MD,
University of Iowa Department of Otolaryngology – Head and Neck Surgery, 200 Hawkins Dr, 21158-A PFP, Iowa City, IA 52242-1078, 319-356-2173

Michael B Gluth, MD,
University of Arkansas for Medical Sciences, Department of Otolaryngology – Head and Neck Surgery, 4301 West Markham St, Little Rock Arkansas, 72205, 501-686-5140

Sarah E Mowry, MD,
University of Iowa Hospitals and Clinics, Department of Otolaryngology – Head and Neck Surgery

Beverly L Aagaard-Kienitz, MD,
University of Wisconsin - Madison, Department of Radiology, Section of Neuroradiology, 600 Highland Avenue, E3/366 Clinical Science Center, Madison, WI 53792-3252, 608-263-8329

Mustafa K Baskaya, MD, and
University of Wisconsin - Madison, Department of Neurological Surgery, 600 Highland Avenue K4/822 CSC, Madison, WI 53792, 608-262-7303

Samuel P Gubbels, MD
[1]University of Wisconsin - Madison, Department of Surgery, Division of Otolaryngology – Head and Neck Surgery, Section of Otology and Neurotology &[2]Waisman Center, 600 Highland Ave, BX7375 Clinical Science Center-K4, Madison, WI 53792-3284, 608-265-0494

Brian C Gartrell: BGartrell@uwhealth.org; Marlan R Hansen: marlan-hansen@uiowa.edu; Bruce J Gantz: bruce-gantz@uiowa.edu; Michael B Gluth: MBgluth@uams.edu; Sarah E Mowry: sarah-mowry@uiowa.edu; Beverly L Aagaard-Kienitz: baagaard-kienitz@uwhealth.org; Mustafa K Baskaya: m.baskaya@neurosurg.wisc.edu; Samuel P Gubbels: gubbels@surgery.wisc.edu

Abstract

Objective—To report three unique cases of cranial neuropathy after super-selective arterial embolization of jugular foramen vascular tumors with ethylene vinyl alcohol.

Correspondence: Please send all correspondence, reprint requests, and proofs to the contact information of the corresponding author supplied above.

Permission was obtained for this work by the Institutional Review Boards at each participating institution.

Financial Disclosure: The authors endorse no financial interests related to this manuscript.

Conflicts of Interest: None

Study Design—Clinical capsule report

Setting—Three tertiary academic referral hospitals

Patients—Three patients who underwent superselective arterial embolization (SSE) of head and neck paragangliomas with ethylene vinyl alcohol are described. One individual was treated with primary SSE, while the remaining tumors were treated with preoperative SSE followed by surgical extirpation within 72 hours. All patients were found to have new cranial nerve deficits following SSE.

Results—One patient with isolated complete cranial nerve VII palsy demonstrated no return of function. One individual experienced cranial nerve VII, X, and XII palsies and demonstrated partial recovery of function of the involved facial nerve after 19 months. One subject experienced ipsilateral cranial nerve X and XI palsies after SSE and recovered full function of the spinal accessory nerve within one week, but failed to demonstrate mobility of the ipsilateral true vocal fold.

Conclusion—We present the first report documenting facial and lower cranial neuropathies after super-selective embolization of head and neck paragangliomas with EVA. Although it is difficult to draw conclusions from this small number of cases, it is plausible that use of ethylene vinyl alcohol during SSE may result in a higher risk of permanent cranial neuropathy than the use of other well-established and more temporary agents. Knowledge of the arterial supply to the cranial nerves can help the clinician to choose the embolization agent that will provide maximal occlusion while minimizing the risk of complications.

INTRODUCTION

Surgical extirpation with or without radiation therapy represents the treatment of choice for the majority of head and neck paragangliomas. The introduction of preoperative super-selective embolization (SSE) has provided a means to reduce complication risks during surgical removal of vascular tumors by decreasing operative time and blood loss (1). SSE has also become a primary treatment option for head and neck paragangliomas in instances where the patient is unable to have surgery or radiotherapy (2). Several polymers have been utilized to achieve this task including cyanoacrylate glues and polyvinyl alcohol (PVA). Each agent has advantages and disadvantages for embolization. Cyanoacrylate provides excellent permanent occlusion of small vessels due to rapid solidification, however this can lead to poor distal penetration in the target vessel and to potential difficulties in removing the embolization microcatheter (3). In addition, its permanent nature increases the danger of a permanent cranial neuropathy if the polymer migrates to a vessel supplying a cranial nerve or if certain anatomic vascular patterns exist (4). PVA is supplied as solid particles and was introduced as an alternative to cyanoacrylate, as PVA provides more distal penetration into feeding vessels. PVA, however, has demonstrated a significant recanalization rate which often results in only temporary occlusion of target vessels (5). This property of PVA has been found to be useful in embolizing vessels supplying cranial nerves, as any resulting deficit is more likely to be temporary than with liquid agents (4). These concerns led to the investigation of ethylene vinyl alcohol (EVA) as a possible alternative. When dissolved in the solvent dimethyl sulfoxide (DMSO) and mixed with micronized tantalum powder to allow for visualization, a useful new embolization agent was formed (Onyx, ev3, Irvine, CA). Onyx provided a more permanent embolization of target vessels similar to that of cyanoacrylate, but its longer solidification time decreased the risk of microcatheter adherence. In addition, because Onyx forms a polymer of sponge-like consistency when fully solidified, it allows more distal penetration of target vessels similar to that of PVA (3). Onyx was therefore viewed as an agent combining the benefits of cyanoacrylate with those of PVA. In 2005, Onyx became FDA-approved for preoperative SSE of central nervous system AVMs (6). Onyx has since been utilized for other applications including

preoperative SSE of head and neck vascular tumors, the majority of which are glomus jugulare or carotid body tumors. Although Onyx has several advantages, the incidence of facial and lower cranial neuropathies following SSE with Onyx is unknown. In general, cranial neuropathies after SSE are rare. Currently only four cases of facial nerve palsy have been reported in the literature after SSE of glomus jugulare tumors, all with PVA (4,7,8). In all of these cases, partial or full recovery of facial nerve function was observed over time. There are currently no reported cases in the literature of facial or lower cranial nerve palsies after SSE of paragangliomas of the head and neck with Onyx.

PATIENTS

Case 1

A 51 year old otherwise healthy female presented to the Otolaryngology clinic with a one-year history of right-sided pulsatile tinnitus and progressive ipsilateral hearing loss. Physical exam revealed a pale reddish mass originating from the hypotympanum and involving the lower half of the middle ear space. Cranial nerve exam revealed no deficits. An audiogram indicated a mild-moderate right-sided mixed hearing loss. High-resolution computed tomography (HRCT) of the temporal bone revealed a 1.8 X 2.5 cm erosive mass centered at the right jugular foramen without significant intracranial involvement but with extension to the neck and in close proximity to the vertical segment of the internal carotid artery (ICA) (Figure 1A and 1B). A Fisch type C2 glomus jugulare was suspected and preoperative superselective embolization (SSE) was arranged. The patient initially underwent an attempted contralateral balloon test occlusion (BTO) and suffered an iatrogenic ICA dissection requiring stenting. No neurologic sequelae resulted from this injury. She was placed on anticoagulation and returned 6 months later for repeat angiography and SSE. The external carotid artery (ECA) was entered and the occipital artery was canalized revealing a tortuous pedicle supplying the tumor which was primed with DMSO and embolized with ethylene vinyl alcohol (EVA). During this time, there was reflux of EVA along the microcatheter, however embolization of the pedicle was achieved. Two smaller distal pedicles of the right occipital artery and two pedicles originating from the neuromeningeal division of the ascending pharyngeal artery were embolized with 100–300 micron Beadblock particles, achieving a 90% reduction in tumor blush. Upon awakening the patient demonstrated a right-sided House-Brackmann (HB) grade II facial nerve paresis which progressed to HB grade VI over the next 24 hours. No other cranial neuropathies were identified. Surgery for tumor extirpation was performed 72 hours later utilizing a Fisch type A infratemporal fossa approach including short rerouting of the facial nerve. The entire tumor was removed during this procedure. Following surgery, the patient was found to have a complete paralysis of the right facial nerve without other neurologic deficits. She had no signs of recovery of facial nerve function on last examination 11 weeks postoperatively.

Case 2

A 72 year-old otherwise healthy female presented to the Otolaryngology clinic with a 3-month history of a sudden-onset complete left-sided facial paralysis associated with intermittent ipsilateral pulsatile tinnitus. Her audiogram and the remainder of her physical exam, including cranial nerve exam, were normal. Computed tomography angiography revealed a 1.5 X 2.0 X 2.5 cm vascular neck mass originating from the left carotid sheath with involvement of the poststyloid space and invasion of the adjacent skull base including enlargement of the stylomastoid foramen. There was only minimal penetration into the jugular foramen. Treatment options, including radiotherapy, surgery, or therapeutic SSE with EVA were discussed with the patient, who consented to the latter. This decision was based in large part due to the patient's age, poor general health, and suboptimal candidacy for general anesthesia. During SSE, a small branch of the occipital artery was selectively

embolized with EVA. Next, the ascending pharyngeal artery was canalized and was subsequently determined to be the primary blood supply to the tumor. Branches of the ascending pharyngeal artery, vertebral artery, and internal maxillary artery were selectively embolized with EVA without difficulty with a near-complete reduction in tumor blush. Hours after the procedure, the patient demonstrated hoarseness and dysphagia with clear liquids. Her exam revealed a left true vocal fold paralysis and a cranial nerve XI palsy. Serial cranial nerve exams documented complete resolution of the spinal accessory nerve palsy in one week, however the vagus nerve palsy persisted necessitating ipsilateral medialization injection laryngoplasty. Her last clinic visit at 14 months demonstrated continued complete paralysis of the vagus nerve and facial nerve (HB grade VI).

Case 3

A 38 year-old female presented to the Otolaryngology clinic with a history of left-sided pulsatile tinnitus and progressive hearing loss. During her physical exam, a cranial nerve X paralysis was observed. HRCT of the temporal bone and magnetic resonance imaging (MRI) were performed which indicated an ipsilateral erosive jugular foramen lesion (Figure 2A–2D). The diagnosis of a Fisch type D2 glomus jugulare was established, and the patient was scheduled for SSE prior to surgical extirpation. During SSE, the left postauricular artery was first canalized and a pedicle feeding the tumor was located. The vessel was primed with DMSO, followed by embolization with EVA. In a similar fashion, branches of the left occipital artery, the parietal branch of the left middle meningeal artery, and a feeding branch of the ascending pharyngeal artery were canalized and embolized with EVA. A significant reduction in tumor blush was achieved after embolization. Upon reversal of anesthesia, the patient was noted to display a left-sided HB grade V facial weakness in addition to a new ipsilateral hypoglossal nerve paralysis. Two days later, she underwent resection of the tumor via Fisch Type A infratemporal fossa approach. Surgical exposure revealed invasion of the pars nervosa of the lower cranial nerves, which were resected. Nineteen months after her surgery, the patient has demonstrated recovery of her facial nerve to HB grade II status. She displays no recovery of ipsilateral hypoglossal nerve function and, as expected, no function of the ipsilateral vagus nerve which was nonfunctional preoperatively and was sacrificed during tumor removal.

DISCUSSION

Understanding cranial nerve blood supply is essential when considering deficits after SSE. With respect to the facial nerve, the tympanic and mastoid segments receive an overlapping blood supply from the stylomastoid artery and the petrosal branch of the middle meningeal artery (MMA) (9). In 60% of patients, the stylomastoid artery arises from the occipital artery and in 40% of patients it arises from the postauricular artery (10). This is relevant as the occipital artery is commonly catheterized during SSE. Furthermore, 10% of people lack a blood supply to the geniculate ganglion from the MMA, meaning that the mastoid and tympanic segments receive their blood supply only from the stylomastoid artery (8). This has clinical implications for SSE because in patients with dual blood supply to the facial nerve occlusion of the stylomastoid artery would not be predicted to result in paresis, however without blood supply from the MMA, embolization of the stylomastoid artery would likely result in a facial nerve deficit. The lower cranial nerves receive their extratemporal vascular supply via branches of the ascending pharyngeal artery, which is the predominant blood supply to head and neck paragangliomas in addition to the stylomastoid artery (8). The glossopharyngeal, vagus, and spinal accessory nerves all receive their blood supply at the jugular foramen from the jugular branch of the neuromeningeal trunk of the ascending pharyngeal artery (9). The hypoglossal branch of this same neuromeningeal trunk supplies the hypoglossal nerve (9). Table I provides a summary of these observations.

Because cranial neuropathies after SSE of paragangliomas are rare, it is difficult to quote the risk of such a complication. Vascular anatomic variants, the embolization agent utilized, and intraoperative occurrences such as vasospasm all likely contribute to the development of a post-procedure cranial nerve deficit. When considering vascular anatomy, Figure 3 demonstrates that 6% of patients would be expected to derive their extratemporal facial nerve blood supply from only the stylomastoid artery originating from the occipital artery. These patients would be predicted to be at a high risk of facial palsy. This incidence correlates well with the observations of Marangos (6.7%) and Valavanis (5.7%) in their respective series of patients with glomus jugulare who underwent embolization (8,4). In our series, Case 1 was predicted to display this anatomic variant. It is more difficult to predict the risk of cranial neuropathy after selective embolization of the neuromeningeal trunk of the ascending pharyngeal artery given the relative paucity of occurrences despite its more constant anatomy. It should also be noted that in situations such as Case 3 where branches of the occipital, postauricular, and middle meningeal arteries are all embolized, a higher probabiliby of facial nerve paresis exists after embolization even if no vascular anomalies are present as both overlapping sources of blood supply to the facial nerve are involved.

The embolization agent also influences the risk of developing a cranial neuropathy. If a vessel supplying a cranial nerve is exposed to an embolic material, a permanent cranial nerve palsy will likely result if nonabsorbable polymers are used (4). PVA, however, has been widely implemented in SSE given its association with revascularization (5). Valavanis proposed the concept of “dangerous” and “safe” vessels when choosing embolization agents. Safe arteries were described as those not involved in supplying a cranial nerve, while those feeding a functional nerve were termed dangerous (4). Because most paragangliomas of the head and neck are multicompartmental, embolic agents with different properties can be utilized for each compartment to provide maximal cessation of tumor blood supply while minimizing complication risks. Because permanent agents are the most effective at occluding vessels, they can be implemented in safe arteries. Less permanent agents like Gelfoam or PVA should be selected for dangerous vessels because the risk of permanent deficits is low (7). If SSE is being performed as the primary mode of treatment (as in Case 2 in our series), permanent agents are recommended to reduce the likelihood of revascularization (7).

The unique properties of Onyx should be considered when performing SSE. Onyx is known to solidify over a longer period of time than other agents. Although this property is beneficial in preventing microcatheter gluing, in the presence of tortuous vessels (as occurred in Case 1) or vasospasm, this could potentially lead to migration of the material to the vasa vasorum of cranial nerves prior to complete solidification. The use of DMSO as a solvent contributes to these observations. DMSO is known to cause perivascular inflammation, vasospasm, and even vascular necrosis at high concentrations (3,11). Applying low concentrations of DMSO and utilizing a slow injection technique have been proposed to minimize the risk of these complications, which could lead to inadvertent reflux of liquified Onyx to the vasculature of cranial nerves (12). In addition, the recanalization rate of Onyx is not currently known, making it difficult to define its use as a nonpermanent or more lasting agent. While most cases of cranial neuropathy reported in the literature after embolization with PVA are temporary with full or partial recovery, this may not be the case with Onyx. Our case series provides three new cases of cranial neuropathy with Onyx, none of which achieved full recovery of at least one involved nerve. This observation correlates with that of Lv et al. who reported two cases of facial palsy after embolization of intradural arteriovenous fistulas with Onyx, neither of which displayed full recovery with time (13). Further studies are therefore needed to best characterize Onyx with respect to permanence in order to further define its use in the treatment of head and neck vascular tumors.

In order to minimize the risk of a cranial nerve injury with SSE, the authors propose using Onyx with caution when embolizing dangerous vessels known to supply cranial nerves as the likelihood of a nonreversible neuropathy may possibly be higher with Onyx than with other nonpermanent agents. With respect to the facial nerve, the clinician can consider canalizing the MMA and assessing the length of the petrous branch as proposed by Valavanis (4). If a dual blood supply to the facial nerve is confirmed, then the clinician could safely choose to proceed with embolization of the occipital artery with Onyx. If the petrous branch is short or if both the middle meningeal and occipital arteries are to be embolized, Onyx should be used with caution and PVA or Gelfoam should be considered. Similarily, caution must be taken if utilizing Onyx near branches of the neuromeningeal trunk of the ascending pharyngeal artery to avoid a lower cranial neuropathy. Care must also be taken to infuse DMSO slowly prior to Onyx injection to avoid vasospasm and reflux of embolic material. Embolization of venous contributions supplying the tumor such as the inferior petrosal sinus (IPS) has also been proposed as a method to reduce the likelihood of a cranial neuropathy after surgical excision. Warren et al. reported a 38% incidence of new cranial neuropathies after surgical resection when preoperative embolization of the IPS was performed in addition to arterial embolization, compared to an 80% incidence of postoperative cranial neuropathies when only arterial SSE was performed. However, the authors of this study did not mention the embolization agent used in their patients nor any post-embolization cranial neuropathies in their study, presumably as there were none. Therefore it is currently not known if combining venous embolization of the IPS with arterial SSE will influence the incidence of post-embolization cranial nerve deficits (14).

The findings presented in our case descriptions have many implications related to clinical decision making for patients with head and neck paragangliomas in that SSE with EVA should not be considered to be a completely benign, complication-free endeavor. Indeed, the decision to pursue SSE as the chief mode of treatment in Case 2 was made in part based on patient age and projected poor tolerance of lower cranial nerve deficits that often occur with paraganglioma excision, yet such deficits occurred despite this perceived conservative approach. In addition, surgical intervention after preoperative SSE adds additional risk of cranial nerve injury that must be considered. Appropriate counseling must be performed with informed consent prior the intervention decided upon.

CONCLUSION

Ethylene vinyl alcohol has several unique properties that have resulted in its use as an effective agent when performing super-selective arterial embolization of head and neck vascular tumors. The vascular patterns of head and neck tumors that are best suited for use of this agent are still being defined, and the incidence of permanent cranial nerve deficits with EVA has not been compared to other embolic agents. To the authors' knowledge, our case series describes the first known cases of cranial neuropathy associated with SSE of head and neck paragangliomas with Onyx. None of these patients demonstrated full recovery of at least one involved nerve. Caution should be utilized when selecting the best agent to employ when embolizing dangerous vessels known to supply cranial nerves. Patients undergoing SSE should also be counseled on a theoretical 6% risk of harboring a vascular anatomic pattern that may place the facial nerve at increased risk during embolization.

Acknowledgments

The project described was supported by NIH-NICHD P30HD03352 and the Clinical and Translational Science Award (CTSA) program, previously through the National Center for Research Resources (NCRR) grant 1UL1RR025011, and now by the National Center for Advancing Translational Sciences (NCATS), grant

9U54TR000021. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH.

References

1. Murphy TP, Brackmann DE. Effects of Preoperative Embolization on Glomus Jugulare Tumors. Laryngoscope. 1989 Dec.99:1244–1247. [PubMed: 2557501]
2. Tasar M, Yetiser S. Glomus Tumors: Therapeutic Role of Selective Embolization. J Craniofac Surg. 2004 May; 15(3):497–505. [PubMed: 15111818]
3. Ayad M. Onyx: A Unique Neuroembolic Agent. Expert Review of Medical Devices. 2006; 3(6): 705–715. [PubMed: 17280535]
4. Valavanis A. Preoperative Embolization of the Head and Neck: Indications, Patient Selection, Goals, and Precautions. American Journal of Neuroradiology. 1986; 7:943–952. [PubMed: 3096120]
5. Pauw BKH, Makek MS, Fisch U, Valavanis A. Preoperative Embolization of Paragangliomas (Glomus Tumors) of the Head and Neck: Histopathologic and Clinical Features. Skull Base Surgery. 1993 Jan; 3(1):37–44. [PubMed: 17170888]
6. Thiex R, Wu I, Mulliken JB, Greene AK, Rahbar R, Orbach DB. Safety and Clinical Efficacy of Onyx for Embolization of Extracranial Head and Neck Vascular Anomalies. American Journal of Neuroradiology. 2011 Jun-Jul;32:1082–1086. [PubMed: 21454409]
7. Herdman RCD, Gillespie JE, Ramsden RT. Facial Palsy after Glomus Tumor Embolization. The Journal of Laryngology and Otology. 1993 Oct.107:963–966. [PubMed: 8263405]
8. Marangos NM, Schumacher M. Facial Palsy after Glomus Jugulare Tumor Embolization. The Journal of Laryngology and Otology. 1999 Mar.113:268–270. [PubMed: 10435142]
9. Ozanne A, Pereira V, Krings T, Toulgoat F, Lasjaunias P. Arterial Vascularization of the Cranial Nerves. Neurimag Clin N Am. 2008; 18:431–439.
10. Djindjian, R.; Merland, JJ. Super-selective Arteriography of the External Carotid Artery. Berlin: Springer-Verlag; 1978. p. 14-123.
11. Chaloupka JC, Huddle DC, Alderman J, Fink S, Hammond R, Vinters HV. A Reexamination of the Angiotoxicity of Superselective Injection of DMSO in the Swine Rete Embolization Model. American Journal of Neuroradiology. 1999 Mar; 20(3):401–410. [PubMed: 10219404]
12. Pamuk AG, Saatci I, Cekirge HS, Aypar U. A Contribution to the Controversy over Dimethyl Sulfoxide Toxicity: Anesthesia Monitoring Results in Patients Treated with Onyx Embolization for Intracranial Aneurysms. Neuroradiology. 2005 May; 47(5):380–386. [PubMed: 15868171]
13. Lv X, Jiang C, Zhang J, Li Y, Wu Z. Complications Related to Percutaneous Transarterial Embolization of Intracranial Dural Arteriovenous Fistulas in 40 Patients. American Journal of Neuroradiology. 2009 Mar; 30(3):462–468. [PubMed: 19131416]
14. Warren FM III, McCool RR, Hunt JO, Hu N, Ng PP, Buchmann LP, Shelton C. Preoperative Embolization of the Inferior Petrosal Sinus in Surgery for Glomus Jugulare Tumors. Otol Neurotol. 2011 Dec; 32(9):1538–1541. [PubMed: 22072258]

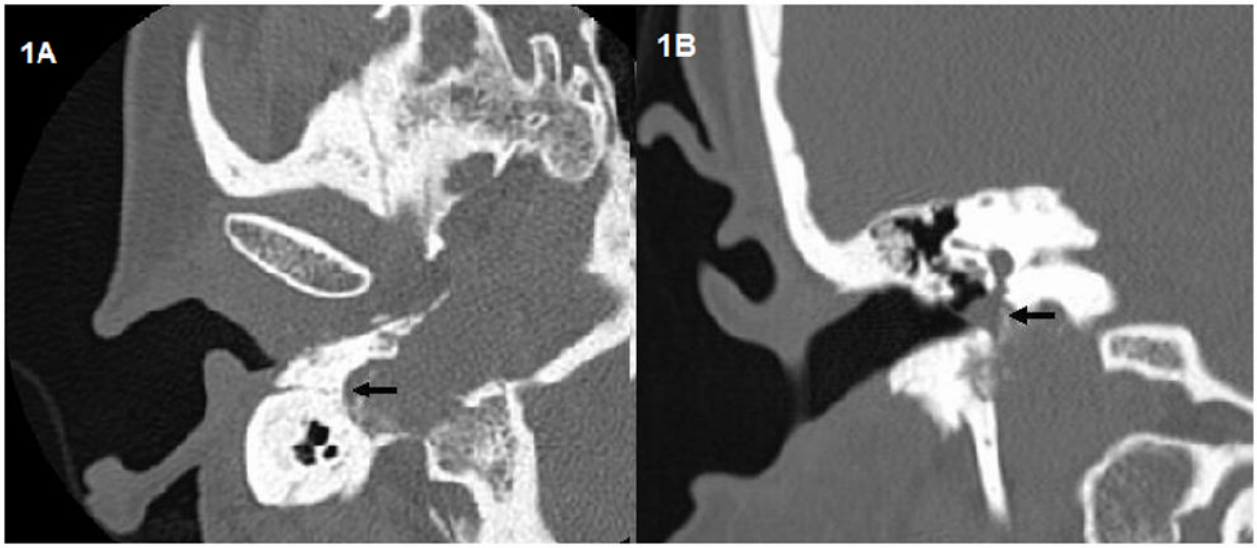

Figure 1.
HRCT image demonstrating a 1.8 X 2.5 cm erosive right jugular foramen mass consistent with a Fisch type C2 glomus jugulare tumor with involvement of the mastoid segment of the facial nerve (1A, arrow). The mass was found to be eroding through the hypotympanum into the middle ear space (1B, arrow).

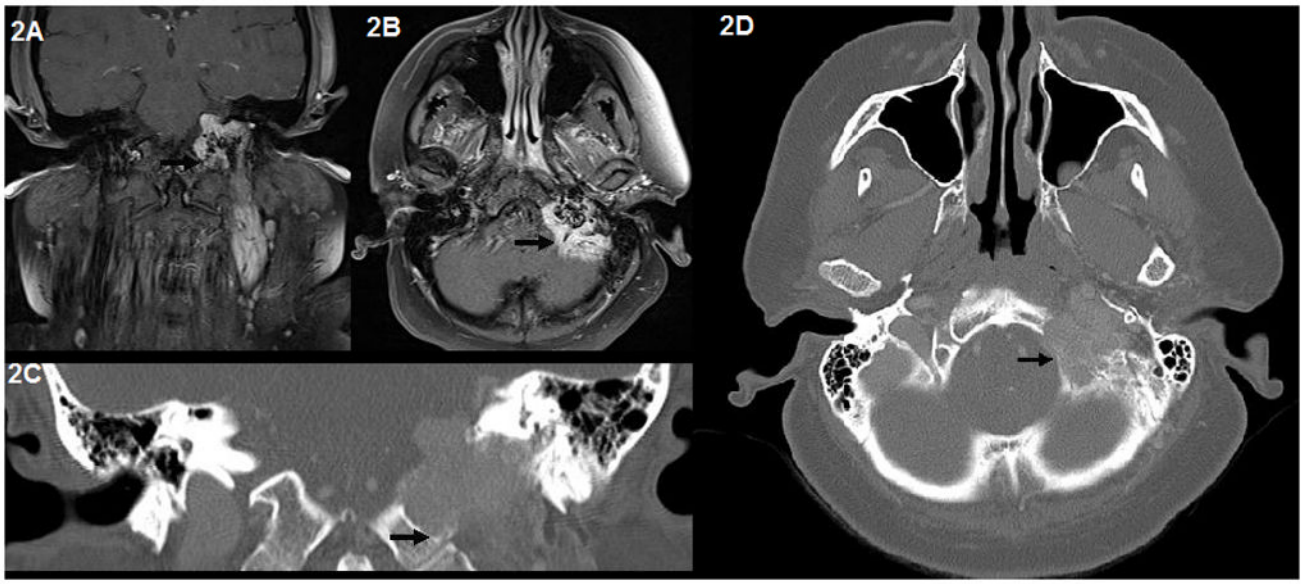

Figure 2.
Coronal (2A) and Axial (2B) T1-weighted MRI sequences and HRCT images (2C and 2D) demonstrating an erosive left-sided jugular foramen lesion (arrows). Physical exam at the time of presentation revealed an ipsilateral true vocal fold paralysis, indicating involvement of cranial nerve X.

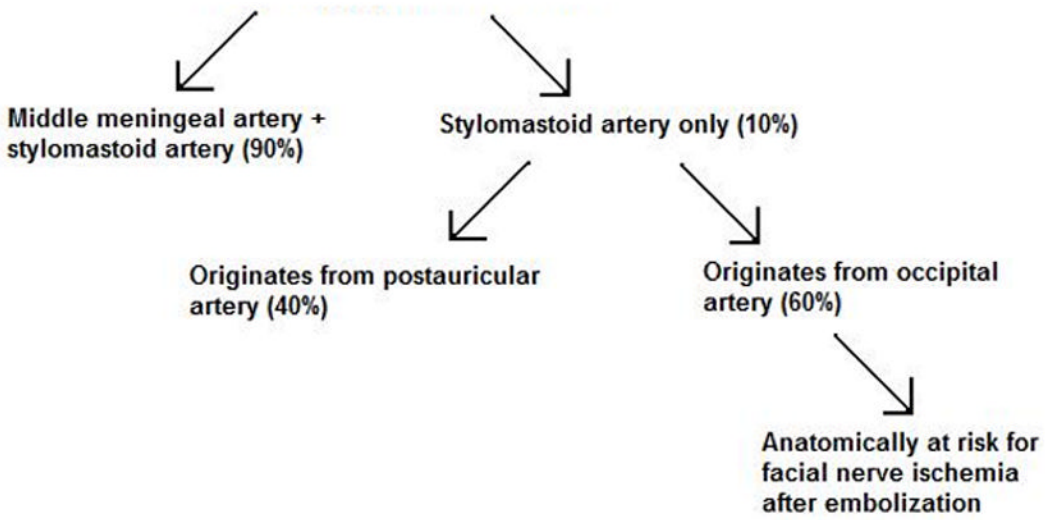

Figure 3.
Anatomic Variants of Facial Nerve Blood Supply. Ten percent of patients would be predicted to carry the stylomastoid artery as the sole primary blood source to the facial nerve. Six percent of all patients would therefore be predicted to have a stylomastoid artery originating from the occipital artery. These patients would be at higher risk for facial nerve ischemia after super-selective embolization of the occipital artery.

Table I

Vascular Supply to the Facial Nerve and Lower Cranial Nerves

Nerve	Blood Supply
Facial Nerve (VII)	Petrosal branch of middle meningeal artery, stylomastoid branch of occipital artery OR postauricular artery
Glossopharyngeal Nerve (IX)	Jugular branch of neuromeningeal trunk of ascending pharyngeal artery
Vagus Nerve (X)	Jugular branch of neuromeningeal trunk of ascending pharyngeal artery
Spinal Accessory Nerve (XI)	Jugular branch of neuromeningeal trunk of ascending pharyngeal artery
Hypoglossal Nerve (XII)	Hypoglossal branch of neuromeningeal trunk of ascending pharyngeal artery