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Trigeminal neuralgia secondary to epidermoid cyst and neurovascular conflict: An illustrative case with literature review

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Case Report

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ABSTRACT

Background: Trigeminal neuralgia (TN) is a highly disabling facial pain syndrome, historically known as the suicide disease, in which most cases can be cured with appropriate surgical treatment.

Case Description: We present the case of a 43-year-old male farmer with acute, self-limiting episodes of shocklike pain on the left side of the face that started in June of 2021. He was diagnosed with TN and was treated with carbamazepine. Magnetic resonance imaging was performed, which revealed an epidermoid cyst (EC) at the preportine cistern with an extension to the left cerebellopontine angle. The neurosurgery department at our institution was consulted, which performed surgical tumor resection and Vth cranial nerve decompression. During the resection, a neurovascular conflict (NVC) was identified at the root entry zone. After the resection around the nerve and its whole tract was completed, a microvascular decompression (MVD) was performed.

Conclusion: TN secondary to EC in association with a NVC is a rare phenomenon, due to the growth pattern of the EC. TN may remit if an appropriate treatment is carried out. In cases of NVC, an MVD is required apart from an appropriate resection to achieve pain relief.

Keywords: Epidermoid cyst, Microvascular decompression, Neurovascular conflict, Trigeminal neuralgia

INTRODUCTION

Trigeminal neuralgia (TN) is a facial pain syndrome characterized by episodes of intense, lancinating facial pain followed by a period of relief. Between episodes, patients live in fear and anticipation of the next episode, coming to be known as the "suicide disease."[14,18,25]

TN is classified based on the etiology as classical, secondary, and idiopathic.^[4,20] Tumors are responsible for around 6-15% of the patients with secondary TN.^[7,15,22]

Epidermoid cysts (ECs), also called intracranial cholesteatomas, are rare lesions accounting for ≤1-2% of all intracranial tumors and 5-7% of cerebellopontine angle (CPA) lesions, just after schwannomas and meningiomas. Their primary location is in the CPA, followed by the parasellar region.^[1,13,16,18,27,32] EC clinical symptoms are very variable, presenting as hearing loss, pyramidal syndrome, seizures, mono- or polycranial neuropathies, headache, and TN, with the latter being

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the most commonly presented, although this varies across different series.^[13,18,22,27]

ILLUSTRATIVE CASE

We present the case of a 43-year-old male farmer who presents acute, self-limiting episodes of shock-like pain located at the left maxillary branch territory of the trigeminal nerve. The episodes were triggered by chewing, touching the affected area, tooth brushing, and sometimes talking, with an intensity of 10/10, causing the patient to interrupt his daily activities. Initially, he was diagnosed with TN and treated with carbamazepine, 200 mg every eight h, and no magnetic resonance imaging (MRI) was taken. As the disease progressed, his pain became constant and disabling, and even though the patient had good therapeutic attachment, he increased the dose by his own accord, reaching toxic doses (5 tablets [200 mg] 5 times a day) because the pain was insufferable.

Study description

The patient eventually sought a second opinion, this time getting an MRI, where a tumoral lesion with EC characteristics in the CPA was identified, given an II in Revuelta's Classification [Figure 1].^[18]

Due to the diagnosis and the excruciating pain, the patient came to the ER of our institution, where the pain was controlled, and the neurosurgery department was consulted.

The neurosurgery department decided to operate on him as a proprietary case based on the patient's characteristics, doing so two weeks after the initial visit. No other focal deficits or pathologies were present in the patient before the surgery.

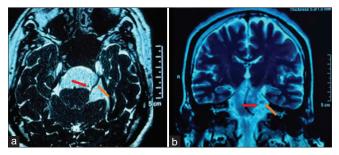


Figure 1: Pre-surgical MRI. Compression of the pons by the EC and the immersion of the Vth CN in the tumor can be appreciated, as well as it being displaced superolaterally. (a) 3D FIESTA: The Vth CN adopted an S shape, and near the REZ, a hyperintense round figure that corresponds to the loop formed by the SCA (red arrow) can be observed. (b) T2: We appreciate a hypointense round which correspond to the SCA (red arrow) as the OV. Laterally, an oval figure that corresponds to the REZ of the Vth CN is seen. CN: Cranial nerve (orange arrow), EC: Epidermoid cyst, MRI: Magnetic resonance imaging, OV: Offending vessel, REZ: Root entry zone, SCA: Superior cerebellar artery (red arrow), 3D FIESTA: Three-dimensional fast imaging employing steady-state acquisition.

Surgical technique

The surgery was performed by Rogelio Revuelta-Gutiérrez (RRG). An extended microasterional approach was taken, involving a 30 mm diameter craniectomy at the internal angle of the transverse and sigmoid sinus. Before opening the dura, the cerebellum was tense, mainly due to the tumor, even after medical intervention by the anesthesiology team. To alleviate some of the tension, the dural opening was started in the inferolateral aspect of the craniectomy, depleting the cisterna magna. Subsequently, the dural opening was extended upwards to reach and deplete the cerebellomedullary cistern. However, multiple adhesions between the cerebellum, cerebellar fossa, and petrous eminence made it difficult to access the cistern.

Once reached, the petrous vein was found to be displaced superficially [Figure 2a] and was used as an anatomical landmark to locate the trigeminal nerve. Nevertheless, it was also relaying over the tumor, which interfered with the visualization of the posterior fossa structures. Initially, the petrous vein was coagulated [Figure 2b], and the tumor capsule was opened to start the tumor debulking. The intratumoral dissection followed the petrosal vein projection to decompress the trigeminal nerve. The tumor removal was achieved using blunt dissection with a Penfield dissector and hydrodissection.

The first structure identified was the trigeminal nerve [Figure 2c], which was displaced and deeply immersed in the tumor. As the debulking progressed around and along the distal part of the trigeminal nerve, the superior cerebellar artery (SCA) was found to be shifted by the tumor, causing vascular compression on the nerve's medial aspect [Figure 2d]. Complete tumor debulking was performed around the trigeminal nerve from the root entry zone (REZ) to the Meckel cavum. A Teflon was placed between the SCA and the nerve [Figure 2e and f]. After ensuring appropriate hemostasis, the surgical site was successfully closed in layers, concluding the procedure without any incidents or accidents.

In the immediate postoperative period, the patient was painfree, with a corneal reflex and no facial hypoesthesia. There were no postoperative cranial nerve (CN) deficits observed. Five days after surgery, the patient was cleared of any complications.

DISCUSSION

Clinically, primary and secondary TN are indistinguishable, but various studies have reported a statistically significant difference in the time of symptom onset, with secondary TN presenting in younger patients.^[3,10] In cases of tumorinduced TN, certain types are more likely to cause specific neurological abnormalities than others. Meningiomas, ECs, and schwannomas tend to be the most common causes of tumor-induced TN, and the prevalence of this phenomenon

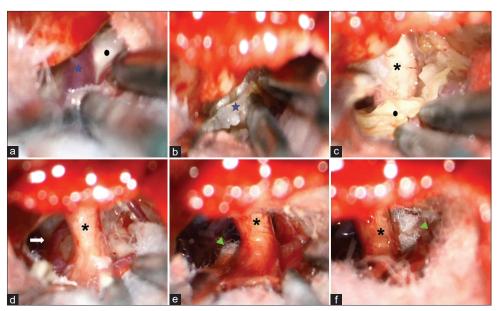


Figure 2: Intraoperative findings. (a) PV overlying the EC. (b) Coagulated PV. (c) Vth CN immersed in the EC. (d) After tumor debulking and capsular resection from the Vth CN, the SCA was exposed and didn't relocate far from the REZ. (e) The MVD was started by placing a Teflon piece through the ventral part of the REZ. (f) MVD was completed by placing a Teflon piece through the dorsal aspect of the REZ. CN: Cranial nerve (asterisk), EC: Epidermoid cyst (circle), MVD: Microvascular decompression (arrowhead), PV: Petrous vein (star), REZ: Root entry zone, SCA: Superior cerebellar artery (arrow).

varies across case series.^[3,6] Microvascular decompression (MVD) effectiveness has been extensively demonstrated in classic TN.^[11,14,19,20] However, in cases of secondary TN, especially those caused by tumors, surgical resection remains the most effective treatment.^[16]

Observations

The presence of NVC and EC is a rare phenomenon, as seen in our patient, has only been seen once in the experience of the main author RRG. The growth pattern of these tumors can explain this. EC tends to exhibit a slow and disseminated growth pattern, conforming to the shape of the subarachnoid space due to its soft and pliable nature before causing substantial compression with mass effect until all available space is occupied. This leads to a delayed diagnosis, with the presence of big and highly disseminated tumors with complex anatomy. This is a much more common phenomenon in other tumors that tend to displace the nerve, such as meningiomas and schwannomas, rather than compress or encase it.^[3,10,16,18,26,30,31]

These anatomical variations have led to various classifications, depending on the author, based on the anatomical relationships between the tumor and the neurovascular structures.

In Table 1, we report various series of reports of TN secondary to EC and NVC, confirmed during surgery. There were some other special situations, such as the case

reported by Wakabayashi *et al.*, reported the association between NVC and an EC in a patient with *Tic Convulsif* who underwent a very interesting series, with tumor resection and required MVD.^[28] Barker *et al.* reported 21 patients with TN and tumors, although we did not include this study in the table, as it does not include the tumor type associated with NVC. They reported two patients with EC and five of all patients not presenting NVC. This series is interesting as they described that not every patient required MVD, as the vascular structure was repositioned so as to avoid contact with the REZ after tumor resection, similar to a case presented by Rappaport.^[3,17] Other series where EC&TN are reported were excuded because no NVC were reported.

Not all of the series report which was the offending vessel (OV), but the most common arteries associated with NVC and EC were the anterior inferior cerebellar artery and superior cerebral artery, with some rarer cases reporting veins as the OV, especially the petrosal vein.^[24,29]

This is especially important due to the pathogenesis of TN secondary to EC, and the MVD it's a critical step for pain remission/surgical success. Some authors consider TN secondary to EC to be caused exclusively by an OV, but based on our experience and literature research, it seems to have a multifactorial etiology.^[3,29] Three main pathophysiological mechanisms have been hypothesized for TN genesis, all with subsequent anatomopathological alteration (mainly demyelination) and aberrant conduction: (a) Mass effect

Series	Total study population	Prevalence of EC in patients with TN	Prevalence of NVC in patients with EC & TN	Procedure NVC	Outcomes + (Pain Relief)	Average + follow-up (y)
Kobata, 2002 ⁹	30	93% (n=28/30)	32% (n=9/28)	MVD	Good- excellent	11.5 (3-16)
Meng, 2005 ¹³	124	19% (n=24/124)	13% (n=3/24)	MVD	Good- excellent	3.1 (1-8)
Son, 2010 ⁴	10	100% (n=10/10)	10% (n=1/10)	MVD	Good-excellent	3.75 (0.5-16)
Shulev, 2018 ²²	14	64% (n=9/14)	11% (n=1/9)	MVD	Good- excellent	4.4 (1-10)
Liu, 2017 ¹⁰	35	40% (n=14/35)	21% (n=3/14)	MVD	Good- excellent	4.3 (0.75-10.3
Farhou, 2018 ⁸	32	18% (n=6/32)	50% (n=3/6)	MVD	Good-excellent	3.5 (1-7.6)
Xia, 2014 ²⁹	12	100% (n=12/12)	75% (n=9/12)	MVD	Good- excellent	1.6 (0.25-3)
Zang, 2020 ³¹	36	% (n=27/36)	15% (n=4/27)	MVD	Good-excellent	4.5 (1-7.5)
Zang, 2022 ³²	69	100% (n=69/69)	29% (n=20/69)	MVD or coagulation *	Good- excellent	5.5 (0.5-10.5)

In all the series, EC resections were made in varying degrees, but an additional procedure was required for the NVC. The outcomes described and reported here were based on general outcomes and were specified when available according to the series. *MVD was performed for arterial compression and veins >1 mm, while veins <1 mm were snipped after electrocoagulation. EC: Epidermoid cyst, MVD: Microvascular decompression, TN: Trigeminal neuralgia, y: Years, NVC: Neurovascular conflict

caused by tumor growth, resulting in compression or displacement of the nerve. (b) Chemical irritation is caused by the cellular debris, cholesterol, and keratin inside the tumor. (c) Vascular compression, either by an artery or vein, ipsi- or contralateral to the nerve, or nerve displacement due to tumor growth, leads to an NVC at the REZ.^[3,7,9,13,22] An additional mechanism, although rare due to the nature of EC, is important to mention. It involves compression of the ventral surface of the pons, which can lead to irritation of the trigeminal nuclei.^[22]

Surgical considerations for NVC and EC

Surgery is currently the only curative therapy for EC. It offers the possibility to treat/alleviate the symptoms caused by the tumoral growth and, in many cases, can "cure" the patient or prevent regrowth.^[1,16,18]

EC tends to remain asymptomatic for many years and presents with great extension at the time of diagnosis, representing a complex surgical challenge when total resection is desired, even for experimented hands. Open surgery offers many alternatives for the treatment of TN. In EC, the strategies taken along with tumor resection, which is the primary procedure, depend on the anatomical findings. In cases where salvage surgery is the only option, a radiosurgical procedure directed to the TN or other percutaneous procedures for pain relief can be an alternative.^[1,15,18,26,29,32]

The surgical objective/approach varies between schools and surgeons; nonetheless, we should keep in mind what Bucy cleverly described: "It is important, however, that the wall of the cyst... which is the only living, growing part of the neoplasm, must be removed completely in order to prevent recurrence."^[1,5] Nowadays, this has become widely accepted and described, although it is not always the best option, especially as new CN deficits are the most frequently presented complications, as well as the ones we want to avoid the most as they greatly interfere with the quality of life. In our experience, the manipulation of CNs secondary to a radical approach should be avoided and only considered when convenient for the patient, even though capsular and tumoral residues are left behind.^[15,18,21,23]

Many authors are in favor of an aggressive approach, as it may offer the patient a possible reintervention in a much longer period, reduce surgical comorbidity, and could even cause tumoral remission in some cases. We need to be conscious of the patient's circumstances and expectations, the economic, human, and technical resources available, and the surgeon's expertise.^[3,21,23,26] Nowadays, the implementation of intraoperative neuromonitoring opened the door to an aggressive but safe resection, but even in the hands of experts, violation of the arachnoid membrane, coagulation or bleeding can disrupt normal nerve functioning, leading to unwanted outcomes.^[18,19,21,23,27,30]

We consider that a moderate resection, focusing on symptomatic nerves, is the ideal surgery, looking for symptomatic remission (pain freedom), prevention of new deficits, and preservation of functionality, especially when the patient is previously completely functional, as in the case of our patient. This is the ideal approach for cases where the resources and circumstances are unfavorable but patient circumstances are optimal.

This focused resection with subtotal tumor removal is backed up by the linear growth hypothesis and the content rate of debris accumulation, especially because, even though the surgeon may have certainty of total capsular resection, sometimes it may be out of our hands. As the linear growth hypothesis postulates, some microscopic flakes may be left, and assuming that only a single cell remains after surgical resection, the patient would be at risk for recurrence at a period equal to his age at the time of the surgery plus nine months.^[2,26,30] In addition, every patient with subtotal resection should be followed up closely, as a significant volume can accumulate before the symptom threshold is reached.^[3,21]

For a focused surgery approach, a clear, wide pathway is made around the nerve with complete rectification of the neuroaxis and visualization of the nerve pathway before exiting through Meckel's Cavum, along with a complete tumoral capsule resection from the affected nerve. Because, if left behind, TN may persist, as it has been seen in some series.^[9,32] As we previously described, these tumors may present some other mechanisms of pain pathogenesis, so a meticulous nerve exploration with an intentional, systematic search may guarantee etiology identification, allowing its elimination and secondary pain relief.

As other authors have described [Table 1], we agree that the decision to perform an MVD should take place after nerve decompression when the OV keeps compressing the nerve. In our experience, when an artery or a unique vein is the OV, MVD is the gold standard, but when it is a venous complex, we consider selective coagulation a more effective alternative.^[19]

Zhang Z *et al.* described their approach to intraoperative findings in a systematic way, which we consider a good alternative for systematization during surgery. They advocate for nerve combing in cases of severe invasion of the nerve with cholesteatomatous tissue, as a simple tumoral resection won't alleviate pain.^[32] We consider that in special situations, such as when the tumoral capsule is too adhered to the nerve or there is a severe invasion of the nerve, either neuropraxia or nerve combing can be performed, according to the surgeon's skills.^[20]

A helpful aid in the previously mentioned cases is the endoscope, which can eliminate blind spots during tumor removal and can help spot hidden NVC. Endoscopic assistance maximizes illumination and magnification around the tip of the endoscope and with this, allows visualization of corners and microsurgical blind spots. The caveat of this tool is the potential risk of damaging delicate structures in the posterior fossa due to new blind spots found behind the rod tip. In the hands of experienced surgeons, this risk can be minimal. This tool is useful but not necessary and should be used with caution so as not to damage vital structures during its use.^[12]

CONCLUSION

We operate on patients, not images. The extent of tumor and capsular resection does not solely determine a successful surgery. Instead, our primary goal is to provide symptom relief, particularly in cases of EC where the growth is typically slow and benign. Considering this, a focused approach is reasonable, especially when dealing with patients who are only presenting TN. When addressing TN, it is crucial to perform EC resection alongside an intentional search for NVC, even though NVC is rare in EC cases, as alleviation of pain relies on identifying and addressing it. As the surgery progresses, alternative surgical strategies such as neuropraxia or nerve combing might be considered in specific situations, as well as the aid of an endoscope which can be useful. but not necessary, always prioritizing patient safety.

They are his nerves, not yours.

-Dr. Revuelta GR.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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