

Image Diagnosis: An Uncommon Cause of Painful Trigeminal Neuropathy

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CASE PRESENTATION

A 54-year-old man was admitted to the hospital with excruciating paroxysmal pain in the 3 divisions of the right trigeminal territory, triggered by both speaking and eating, with no apparent trigger point. He had presented the year prior (in 2017) with recurrent infections of the upper airway and ears, and hypoacusis.

An initial neurologic examination revealed both sensory deficits and allodynia in the right trigeminal region (V₁, V₂, and V₃), as well as bilateral diminished auditory acuity, with no evidence of other cranial nerve involvement or other focal signs. Furthermore, the ear, nose, and throat examination showed a painless tumefaction in the left side of the soft palate with no fluctuation.

Results of computed tomography showed a bilateral nasopharynx infiltration of lymphoid tissue. Results of subsequent magnetic resonance imaging (MRI) disclosed extensive infiltration of the pharynx with extension to the base of the skull with involvement of the surrounding muscles, right internal carotid artery, and jugular vein. Additional mass extension was observed in the base of the skull involving the right cavernous sinus, the right Meckel cave, and the ipsilateral foramina ovale and rotundum. Moreover, the MRI results showed contrast enhancement of the right trigeminal nerve, including the gasser ganglion (Figure 1).

The histologic examination confirmed the presence of a follicular-type B-cell non-Hodgkin lymphoma. Nevertheless, the staging process excluded a leukaemic component, systemic metastazition, or meningeal invasion (cerebrospinal fluid analysis revealed 5 leukocytes, with normal protein count and negative flow cytometry immunophenotyping).

The patient underwent chemotherapy and exhibited a favorable response. Although the frequency of the algic paroxysms gradually decreased until complete remission, the patient maintained hypoesthesia in the right trigeminal territory. Additionally, given the refractory characteristic of the pain, symptomatic treatment required the use of carbamazepine, corticosteroids, phenytoin, gabapentin, amitriptyline, metamizole (dipyrone), and tramadol.

DISCUSSION

Painful trigeminal neuropathy is a pathology characterized by algic paroxysms associated with neurologic deficits in the trigeminal divisions, and represents secondary disorders that affect cranial nerve V (accounting for 15% of the trigeminal neuralgias).¹

With an incidence ranging between 14% and 63.2% in tumor series, neoplastic perineural spread is one of the many etiologies of this neuropathy.² This form of metastasis occurs through the movement of neoplastic cells into the neural space, affecting predominantly the trigeminal and facial nerves. Given the anatomic proximity to these nerve terminations, it is typically associated with cutaneous and salivary glands tumors.²⁻⁴ The perineural spread can be detected by MRI as an obliteration of the fat planes around the nerve branches and an enhancement and/or enlargement of the nerve.^{2,3}

Despite being a rare situation, this case demonstrates that a painful trigeminal neuropathy can be caused by retropharyngeal lymphomas. Hence, in the case of a trigeminal neuropathy of undetermined

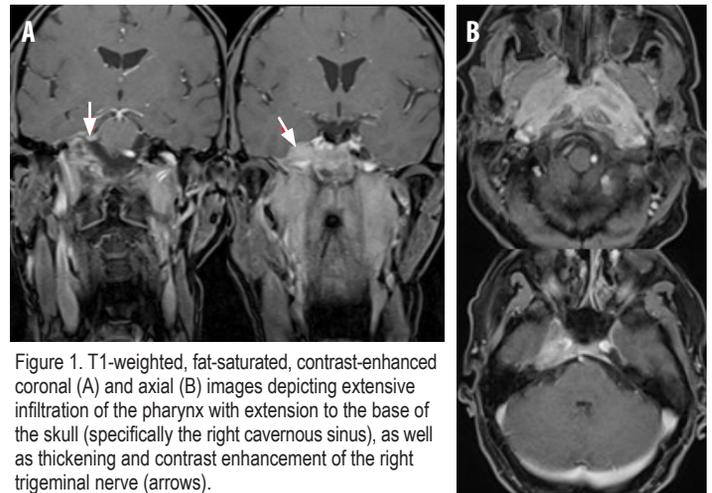


Figure 1. T1-weighted, fat-saturated, contrast-enhanced coronal (A) and axial (B) images depicting extensive infiltration of the pharynx with extension to the base of the skull (specifically the right cavernous sinus), as well as thickening and contrast enhancement of the right trigeminal nerve (arrows).

etiology and recurrent upper respiratory and ear infections, an extensive search for an occult neoplasm of the pharynx or sinonasal sinuses should be performed.⁵ The main takeaway of this image diagnosis is that in a case of a trigeminal neuralgia with associated focal deficits, an MRI is promptly required to determine the underlying etiology. ❖

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

How to Cite this Article

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