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Case Report

Radiology Section

Psoriasis-associated Petrous Hyperostosis Causing Trigeminal Neuralgia: A Case Report

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ABSTRACT

Trigeminal Neuralgia (TN) is most commonly caused by Neurovascular Conflict (NVC); bony aetiologies are rare. We report a 31-year-old woman with a 10-year history of psoriasis who presented with a 3-month history of intermittent, severe, paroxysmal right lower jaw pain triggered by chewing, consistent with right V3 TN. Magnetic Resonance Imaging (MRI) of the brain with Constructive Interference in Steady State (CISS) sequence, done after three months of onset of first symptom, demonstrated attenuation of the right cisternal trigeminal nerve, effacement of the right cerebellopontine cistern, and thickening of the right petrous bone. A hypertrophied suprameatal tubercle caused narrowing of the right internal auditory meatus. Mild generalised calvarial hyperostosis was also noted. Computed Tomography (CT) with bony windows confirmed hypertrophic changes at the suprameatal tubercle. A coincidental neurovascular loop was identified adjacent to the right trigeminal nerve. The history of psoriasis, missed at the initial consultation, was elicited on directed enquiry. The constellation of petrous hyperostosis, calvarial thickening, and clinical presentation led to the diagnosis of TN secondary to bony compression in the setting of psoriasis-related bone disease. The patient was treated with oxcarbazepine 300 mg once daily, achieving 60-70% symptomatic relief, and was counselled regarding surgical intervention, including bony decompression and Microvascular Decompression (MVD). This case highlights that psoriasis, a known cause of skeletal hyperostosis, can rarely produce surgically correctable TN through bony compression of the trigeminal nerve at the porus trigeminus. A thorough systemic history, recognition of imaging asymmetry, and precise characterisation of the compressive element are critical to directing appropriate surgical management and avoiding unnecessary nerve-ablative procedures.

Keywords: Computerised tomography, Hyperostosis, Magnetic resonance imaging, Oxcarbazepine, Psoriasis, Trigeminal neuralgia

CASE REPORT

A 31-year-old woman presented with a 3-month history of intermittent, severe, electric shock-like pain in the right lower jaw. The pain was paroxysmal, lasting seconds to minutes, and was consistently triggered by chewing. There was no spontaneous baseline pain between episodes (Type 1 TN). There was no history of burning pain, congestion of the nose, or tearing/redness of the eye. Although chewing was painful, the pain was intermittent, and there was no history of clicking or jaw locking. On neurological examination, her higher mental functions were normal. Detailed trigeminal nerve examination was normal, with no motor weakness or masseter wasting noted. The rest of the neurological examination was normal. There was no short neck, restriction, or painful neck movement. Local examination revealed no tenderness or swelling at the temporomandibular joint. Dermatological review revealed established plaque psoriasis affecting the scalp and extensor surfaces; this history had not been volunteered at the initial consultation and was identified only through directed systemic inquiry at the post-MRI consultation. Differential diagnoses considered were dental caries, temporomandibular joint dysfunction, cluster headache, craniovertebral junction anomaly, cerebellopontine angle tumour/epidermoid, vascular malformation like dolicoectatic basilar/vertebral artery, and the rare possibility of multiple sclerosis.

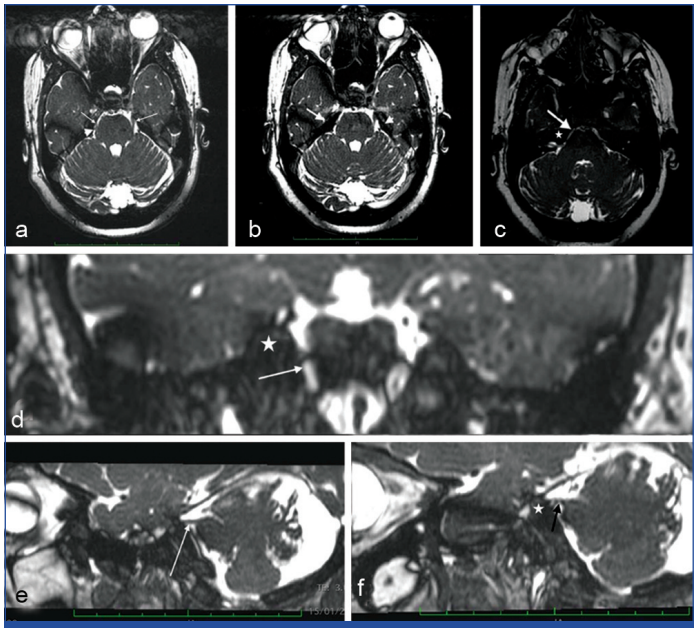
The MRI of the brain with a CISS sequence was performed using a 1.5T GE scanner (GE Healthcare, Milwaukee, WI, USA). Compared with the left-side, the cisternal segment of the right trigeminal nerve was poorly defined and attenuated [Table/Fig-1]. The right cerebellopontine cistern was effaced [Table/Fig-1a,d,f]. A hypertrophied suprameatal tubercle on the anterosuperior aspect of the right internal auditory meatus caused narrowing of the nerve, with the posterior margin of the right petrous bone being contiguous with this structure [Table/Fig-1c,2a]. Thickening of the right petrous bone narrowed the adjacent Cerebrospinal Fluid (CSF) cistern and compressed the cisternal trigeminal nerve. Mild generalised calvarial hyperostosis was identified [Table/Fig-1a]. A vascular loop was noted in proximity to the right trigeminal nerve [Table/Fig-1a,f]. The left trigeminal nerve was normal for comparison [Table/Fig-1a,e]. CT brain with bony windows confirmed hypertrophy of the right suprameatal tubercle [Table/Fig-2a,b].

A formal nerve conduction study was deemed unnecessary, as an MRI evaluation revealed no diagnostic dilemma.

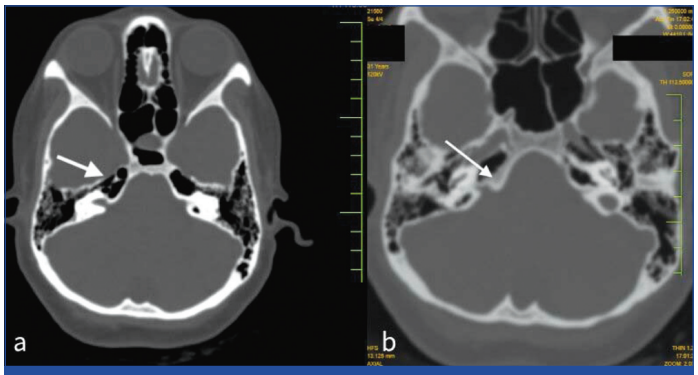
The combination of petrous hyperostosis, suprameatal tubercle hypertrophy, calvarial thickening, and a history of psoriasis led to the diagnosis of TN secondary to bony compression in psoriatic bone disease. The patient was initiated on oxcarbazepine 300 mg once daily and achieved 60-70% symptomatic relief. She has been on medical management for the last three months. There has been no need to escalate the drug dosage to date. She has been counselled for bony decompression (drilling of the petrous bone at the porus trigeminus) with concomitant MVD to address the coincidental neurovascular loop, which, as per the management team, is the only complete and lasting pain relief solution for her. As of the time of this report, she had not provided consent for surgical treatment.

DISCUSSION

The TN is characterised by paroxysmal, unilateral, and severe facial pain. NVC is the most common etiology, usually due to aberrant vascular loops compressing the trigeminal root at the pontine trigeminal junction [1]. Secondary causes account for a minority of cases and include cerebellopontine angle tumours, demyelinating disease, and bony compression. Psoriasis is a chronic systemic inflammatory disease with recognised skeletal manifestations, including arthropathy and hyperostosis; the latter is most



[Table/Fig-1]: a) Axial CISS MRI image showing a well-defined left trigeminal nerve and a thin, ill-defined right cisternal trigeminal nerve (arrow). A vascular loop is seen in proximity to the right nerve (arrowhead). Note the mild generalised calvarial thickening; b) Axial T2-weighted MRI showing thickening of the right petrous bone (arrow) compared with that of the left (arrowhead); c) An axial CISS MRI image showing a hypertrophied right suprameatal tubercle narrowing the internal acoustic meatus (asterisk) and effacement of the right cerebellopontine cistern (arrow); d) A coronal CISS MRI image showing that the right petrous bone at the level of the porus trigeminus is higher (more rostral) than that on the left (asterisk). The right cerebellopontine cistern is effaced compared to the left (arrow); e) A sagittal CISS MRI image showing the normal left trigeminal nerve traversing the cistern from the pons to the porus trigeminus (arrow); f) A sagittal CISS MRI image showing the right trigeminal nerve with poor definition (arrow) and a tall petrous apex (asterisk).



[Table/Fig-2]: a) CT brain bony window image confirms bony hypertrophy with pneumatisation at the petrous bone on the right-side; b) CT brain bony window image confirms bony hypertrophy at the right suprameatal tubercle (arrow).

conspicuously seen in the context of Synovitis, Acne, Pustulosis, Hyperostosis, and Osteitis (SAPHO syndrome) [2].

The incidence of TN is approximately 4.3 per 100,000 per annum. It peaks around age 60-70 years, and its prevalence increases with age [1]. TN in patients under 40 years of age should prompt a deliberate search for secondary causes, as NVC tends to present in an older demographic [3]. Bony compression of the trigeminal nerve, although rare, is a well-documented mechanism. Cadaveric studies have identified several susceptible anatomical sites: the porus trigeminus [4], the suprameatal tubercle [5], Meckel's cave, the cavernous internal carotid artery, the foramen rotundum, and the foramen ovale [6].

As per the case report by Hirata S et al., a similar case of petrous bony hypertrophy was identified, which required drilling for satisfactory decompression. The patient had satisfactory pain relief post-surgery [7].

A case report by Abdulla E et al., revealed a clival osteoma as a cause of TN. The patient had complete resolution of symptoms postsurgery [8].

Another case by Lim P et al., demonstrated an intraosseous meningioma along the petrous bone as the culprit [9].

Khormi GJ et al., reported a case of aneurysmal bone cyst of the temporal bone causing ipsilateral TN [10].

Hamrick F et al., eloquently described the management of TN caused by a bone spur [11].

According to a case series by Shenouda EF et al., 15 patients presented with typical TN, had preoperative imaging suggestive of vascular compression of the trigeminal nerve root, and underwent standard retromastoid craniotomy. The juxta-petrous portion of the trigeminal nerve root was obscured by petrous endostosis. The NVC was revealed and dealt with after drilling of the endostosis in 11 patients, and four patients had endoscopic-assisted exploration of the region of the endostosis [12].

The first report of the association between Paget's disease and TN was given by Gardner WJ and Dohn DF. They reported bilateral TN and bilateral hemifacial spasm as a result of secondary basilar impression in a known case of Paget's disease [13].

The present case demonstrates that hyperostosis of the petrous bone and suprameatal tubercle could act as the primary compressive elements in the setting of psoriatic bone disease. Although the patient did not fulfill the criteria for SAPHO syndrome [2], psoriasis-related hyperostosis is a recognised entity, and calvarial thickening provided additional supportive evidence. The coincidental neurovascular loop, had it been the only finding, would likely have led to MVD alone without addressing the critical bony component. As we could not perform a bone biopsy to rule out other causes of this bone hypertrophy, such as fibrous dysplasia or Camurati-Engelmann disease, the inference rests on the temporal coexistence of psoriasis and petrous hyperostosis in this case. We acknowledge this limitation to the report.

The MRI with a CISS sequence is the imaging modality of choice for evaluating TN [14]. Effacement of the cerebellopontine cistern and attenuation of the cisternal nerve compared with the contralateral normal side should prompt a thorough search for the underlying lesion. CT is indispensable for characterising the bony element. Failure to decompress this component during surgery may result in persistent symptoms and avoidable recourse to ablative procedures, such as radiofrequency ablation or alcohol injection, which carry the risk of anesthesia dolorosa and motor fiber damage in young patients [15].

CONCLUSION(S)

Psoriasis-related petrous hyperostosis is a rare but surgically correctable cause of TN. This case adds to the evidence that bony aetiologies of TN must be actively considered, particularly in younger patients.

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