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Case Report

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Traumatic Neuroma of the Mental Nerve: A Case Report

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ABSTRACT

Traumatic neuroma is a rare complication of dental extraction and other surgical procedures. Owing to the low prevalence of the entity coupled with fewer number of reported cases, many of the dental professionals are unaware about its exact nature and management. A confirmatory diagnosis of traumatic neuroma requires distinguishing it from other clinical and histopathological entities with similar features. The present report details a case of traumatic neuroma associated with the mental nerve and emphasizes on its pathogenesis and histopathological aspects.

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Introduction

Oral Traumatic neuroma (TN) is one of the most infrequently occurring complications of dental extraction and other surgical procedures [1]. It is defined as a non-neoplastic proliferation of neuronal tissue, secondary to trauma (surgical or accidental), resulting from a failed attempt of the severed nerve fibers to reunite [2]. TN was initially described as 'amputation neuroma', owing to its occurrence following nerve transection [3].

The prevalence of TN is about only 0.3% making it a rare entity and as a result, dental professionals seldom encounter this complication in their practice. As a result, not many dental surgeons are aware about TN, its clinical course, pathogenesis or management [4]. Our case report presents a case of TN with emphasis on its histopathological aspect correlating with the pathogenesis.

Case Report

A 56-year-old male complained of pain in the right mandibular posterior region since two months. He described the pain as dull, aching in nature which got aggravated and became pulsating on chewing food. The patient had undergone dental extraction with mandibular right first molar six months ago in a private dental clinic. He provided a medical history of Type II diabetes mellitus since two years which was currently under control with medications. The patient did not consume tobacco in any form nor did he have any parafunctional habits such as bruxism. There was no family history of temporomandibular disorders or orofacial pain.

On extraoral examination, facial asymmetry or swelling was absent. Intraorally, an oval, nodular lesion having a diameter of about 7 mm was noted in the buccal vestibular region distal to the mandibular right second premolar. The color of mucosa overlying the lesion was normal. The lesion was found to be firm and fixed to underlying tissues on palpation. The temperature of the area with lesion was found to be normal.

A painful response radiating to the chin was elicited on application of pressure to the lesion, without any refractory period, which led to inference of a positive Tinel's sign [5]. The patient described initial pain to be of intensity score 4 on a Visual Analog Scale (range: 0 to 10) which got aggravated to 7 on chewing or provocation. The pain subsided completely on perilesional infiltration of local anesthetic. No significant findings were noted in the intraoral periapical radiograph of mandibular right premolar-molar region.

The differential diagnosis after clinical evaluation comprised of traumatic fibroma, late-stage pyogenic granuloma, Trigeminal neuropathy (TNP), traumatic neuroma, solitary neurofibroma, peripheral encapsulated neuroma or musculogenic tumors. Upon surgical exposure, a well-defined grayish nodular lesion was noted attached to the mental nerve. The lesion was excised, fixed in formalin and submitted for histopathological processing.

On microscopic examination, multiple discrete bundles consisting of neurofibrils and Schwann cells were noted (Figures 2 and 3). These bundles were concentrically surrounded by dense bundles of collagen fibers that interlaced with adjacent set of bundles. Histopathologically, the differential diagnosis included traumatic neuroma, neurofibroma and Schwannoma. Oral rhabdomyoma could still not be ruled out owing to certain areas of hyalinized bundles of fibers resembling muscle tissue.

Immunohistochemistry (IHC) was performed for the confirmation of diagnosis. Immunoexpression for vimentin was found to be strongly positive throughout the tissue indicating its mesenchymal nature (Figure 4). Intense immunostaining of S100 was found only in the discrete nerve bundles indicating that the fibers of intervening fascicles did not comprise of neurofibrils as would have been the case in neurofibroma or Schwannoma (Figures 2 and 3). Completely negative immunoexpression for Desmin ruled out a tumor of muscular origin (Figure 4).

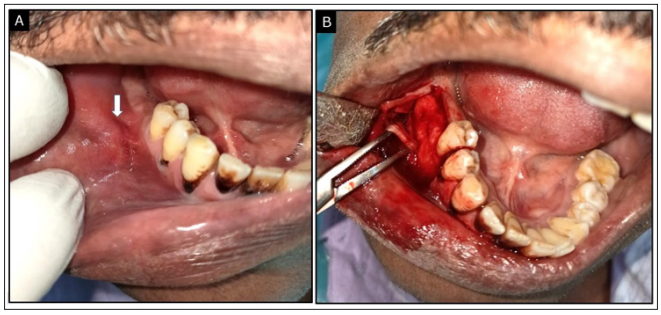

Figure 1: A) Clinically evident nodular lesion in the buccal vestibule region, B) Surgical exposure of lesion associated with the mental nerve

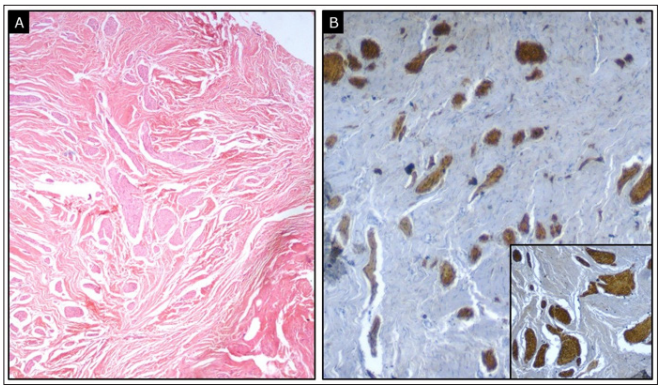

Figure 2: A) Discrete bundles of nerve surrounded by dense fibrous connective tissue stroma (H and E, 10x); B) Immunopositivity of nerve bundles for S100 (10x)

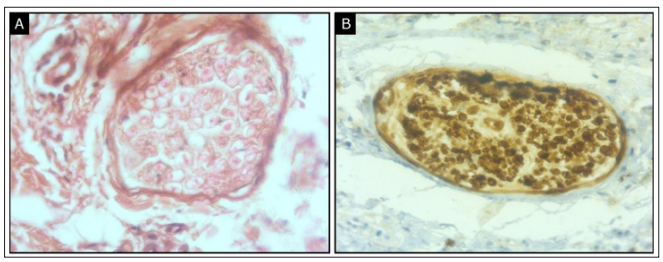

Figure 3: A) Nerve bundle consisting of neurofibrils and Schwann cells surrounded by perineural thickening of stromal fibers. (H and E, 40x); B) Immunopositivity of nerve cells but not perineural stromal fibers for S100 (10x)

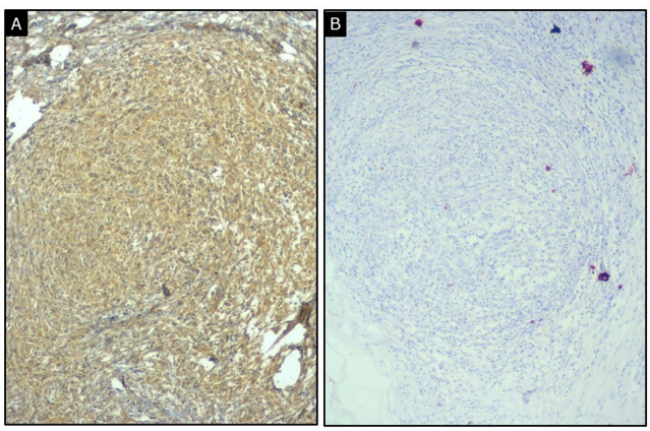

Figure 4: A) Diffuse positive immunostaining in the cytoplasm of cells throughout the tissue for Vimentin B) Completely negative immunostaining for Desmin

After careful consideration of the clinical symptoms, positive Tinel's test, histopathological features and pattern of staining by IHC, a final diagnosis of TN was imparted for the case.

Discussion

Upon transection, the distal axons of a nerve undergo Wallerian degeneration, whereas the proximal axons and Schwann cells proliferate in an attempt to re-establish the connection with the distal segment. For reinnervation to occur, the proximal axons are required to grow through the tubes of proliferating Schwann cells and reach the axons of distal segment [6]. Wound healing is compromised in conditions such as diabetes mellitus, which was present in the present case. As a result, healing occurs by secondary intention leading to delay in filling of the wound and subsequent scar formation. It is hypothesized that the regenerating nerve fibers may extend into such wounds during the prolonged proliferative phase of healing by secondary intention.

The scar tissue produces a barrier for the proliferating proximal axons seeking the distal segment of the nerve. The regenerating axons become irregularly dispersed throughout the proliferating wound and scar tissue forming spirals and end discs [6]. As the myofibroblasts of the scar tissue contract, the peri-neural and epineural tissues proliferate in order to protect the nerve from injury due to compression. This produces a concentric condensation of fibrous tissue around the discrete nerve fascicles, which can be noted in the histopathology of TN.

Overall, the nerve fibers get immersed in scar tissue with concentric condensations of fibrous stroma around the discrete neural fascicles. The thwarted attempt of regenerating neuraxes of a severed nerve by scar tissue ultimately results in a disorganized tangle of neural and connective tissue fibers that extend into the surrounding soft tissues [6]. This manifests clinically as a grayish-white, smooth nodule that is fixed to surrounding structures, as noted in the present case [7].

More than half of the patients with TN report with pain of varied intensity, as was also noted in the present case [7]. The compression of nerve fibers by the contracting scar tissue has been postulated as a reason for the pain-related symptoms in TN. The slow-growing course of fibrous tissue allows for compensatory proliferation of epineurium and the symptoms begin only after a threshold is reached [6]. Inflammatory cells have also been implicated in contributing to the pain in TN, however, previous studies have failed to identify a definite correlation between the extent of inflammation and the patient's clinical symptoms [8]. Inflammatory cells were absent in the present case and yet the patient experienced continuous pain.

A steady barrage of impulses originating in TNs was found associated with unmyelinated afferent nerve fibers [3]. Additionally, application of even slight pressure can lead to generation of bursts of neuronal discharge which accounts for the positive Tinel's sign in TN. Further supporting evidence was provided by previous researchers that demonstrated predominance of small unmyelinated nerve axons in TN [9]. The periodic pain generated from ephaptic cross-talk between the entrapped nerve bundles close resembles the paroxysmal pain noted in TNP. However, the absence of a refractory period did not favor the diagnosis of TNP.

Rationally, only the fibers that are capable of regeneration would have the ability to form such a mass.

Therefore, TN can be formed only by fibers of the sensory nerves or sensory fibers of the mixed nerves, and cannot occur in motor nerves which have very limited regenerative potential [6]. Mental nerve is most frequently involved in TN, which was also noted in the present case. TN commonly tends to occur near the mental foramen area, tongue or lower lip [3,7]. The present case also displays formation of tumor mass close to the mental foramen, coinciding with the site of damage to the nerve during the extraction of mandibular right first molar.

A recent survey reported that a significant percentage of dental professionals were unaware of these facts about formation of TN, and also whether it is benign or malignant. The relative unawareness pertaining to TN amongst general dental practitioners can be attributed to low prevalence of TN and paucity of reported cases. These, in turn, may be because of certain reasons: (i) Most of the extraction procedures are atraumatic or not traumatic enough to produce TN, (ii) Factors complicating wound healing are present only in occasional cases, (iii) A certain percentage of neuromas may possibly remain asymptomatic and therefore, remain undiscovered. The fact that TNs have been accidentally discovered during surgical exposure for treatment of other lesions supports this possibility [10].

Similarly, data from a recent survey has found that more than half of the dental professionals were unaware about the treatment modalities available for TN [4]. Surgical management of TN require nerve-sparing exeresis with careful manipulation to avoid severance of the nerve fibers. Suturing of the severed portion to the parent nerve can help in re-innervation and restoration of normal sensation [3,5]. Use of prosthetic stents, local infiltration of steroids, sympathetic ganglion block and ultrasonic therapy have been implicated in management of TN with fair degree of success [11].

Conclusion

Although uncommon, oral TN is a complication of dental extraction and other surgical procedures that is challenging to manage. Clarity pertaining to the pathogenesis of TN can enable dental surgeons to better correlate with the clinical course of traumatic neuroma. Our case report would aid surgeons as well as pathologists in diagnosis of cases of TN and differentiating them from other clinically or histologially similar entities.

References

1. Guerrieri P, Oliveira A, Arosio F, Murgia MS, Viganò L, et al. (2021) Vascular-nerve complications following oral surgery: hematoma and neuroma: A literature review. Int J Appl Dent Sci 7: 104-107.
2. Deng W, Chen SL, Huang DY (2009) Traumatic neuroma of mental nerve following chin augmentation. Int J Oral Maxillofac Surg 38: 1324-1326.
3. Sist Jr TC, Greene GW (1981) Traumatic neuroma of the oral cavity: report of thirty-one new cases and review of the literature. Oral Surg Oral Med Oral Pathol 51: 394-402.
4. Devi VA, Dhanraj M, Jain AR (2018) Knowledge attitude and practice of dental practitioners in the management of traumatic neuroma-a survey. Drug Invent Today 10: 542-545.
5. Neumeister MW, Winters JN (2020) Neuroma. Clinics in plastic surgery 47: 279-283.
6. Foltán R, Klíma K, Špačková J, Šedý J (2008) Mechanism of traumatic neuroma development. Med hypotheses 71: 572-576.
7. Anbinder AL, Paiva TV, Mariano RC, Quirino MR (2009) Traumatic neuroma of the lower lip. West Indian Med J 58:

604-606.

8. Vora AR, Bodell SM, Loescher AR, Smith KG, Robinson PP, et al. (2007) Inflammatory cell accumulation in traumatic neuromas of the human lingual nerve. Arch Oral Biol 52: 74-82.
9. Mathews GJ, Osterholm JL (1972) Painful traumatic neuromas. Surg Clin North Am 52: 1313-1328.
10. Peszkowski MJ, Larsson Å (1990) Extraosseous and intraosseous oral traumatic neuromas and their association with tooth extraction. J Oral Maxillofac Surg 48: 963-967.
11. Arribas-García I, Alcalá-Galiano Rubio A, Gutiérrez R, Montalvo Moreno JJ (2008) Traumatic neuroma of the inferior alveolar nerve: a case report. Med Oral Patol Oral Cir Bucal 13:186-188.