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Diagnostic challenges in numb chin syndrome caused by mandibular intraosseous schwannoma

Numb chin syndrome (NCS, mental neuropathy or V3 sensory neuropathy) poses diagnostic challenges for clinicians, often resulting in significant patient distress due to the abrupt loss of sensation in the lower lip and chin.[1] This case report explored an exceptional instance where an intraosseous schwannoma (IS), mimicking periapical infectious disease, contributed to NCS.

A 58-year-old Taiwanese male presented with persistent right chin numbness for two months. Past medical history yielded nothing particular. Clinical examination revealed a residual root of tooth 45 with a distinctive periapical radiolucent lesion involving the inferior alveolar nerve (Fig. 1A). Cone-beam computed tomography revealed a bone lesion eroding the mandibular canal. Despite tooth 45 extraction, ongoing sensory impairment raised the suspicion of a carcinoma, leading to a surgical intervention. During tumor dissection under general anesthesia, the mass exhibited a rubbery texture, easily detaching from the main trunk of the inferior alveolar nerve, suggesting a central schwannoma. Careful excision of the tumor was performed (Fig. 1B and C). Histopathological examination showed Antoni A and type B tumor tissues and Verocay bodies (Fig. 1D and E). The immunohistochemical stain exhibited that the tumor schwannoma cells were diffusely positive for S-100 protein (Fig. 1F). The patient immediately recovered from NCS, validated by subjective function, light touch, and sharp-blunt discrimination tests one day postsurgery, guaranteeing restored sensory function in the chin and lower lip. This recovery from numbness persisted during a six-month follow-up.

This case emphasized the diagnostic challenges in NCS, highlighting the necessity for a thorough differential diagnosis involving iatrogenic treatment, infection, inflammatory disease, benign tumor, and cancer. The rare occurrence of mandibular IS-induced NCS reminds us that a benign tumor, particularly a schwannoma, may cause NCS.

Carter et al.'s study of 372 cases of V3 sensory neuropathy found that 89.5% cases are caused by iatrogenic treatments and 10.5% cases are due to non-iatrogenic etiologies including infections, systemic inflammatory diseases, and malignancies.[2] This provides guidance for differential diagnoses. Conversely, Dru Perkins et al.'s review of 88 jaw IS cases revealed that 14% cases of mandibular IS may result in paresthesia (a sign of NCS), highlighting clinical heterogeneity across medical professions.[3] This underscores that IS, a benign tumor, can also lead to NCS, particularly when involving the mandibular canal, as observed in our case.

Surgical approaches for mandible IS vary based on tumor location and its relation to the inferior alveolar nerve. Chi et al. and DeLeonibus et al. reported post-surgery sensory loss or paresthesia in 3/14 and 12/28 cases, respectively. The majority of the mandible IS surgeries causes no sensory morbidity.[4,5] Immediate NCS recovery after tumor removal was rare. In our case, conservative excision of the central schwannoma led to immediate NCS recovery, highlighting the importance of considering IS in the clinical practice and recognizing cases that may require complex resections. This report underscored the importance of clinicians being vigilant about the rare entity in NCS. Cautious surgical excision of the IS has proven to be successful in treating IS-induced NCS.

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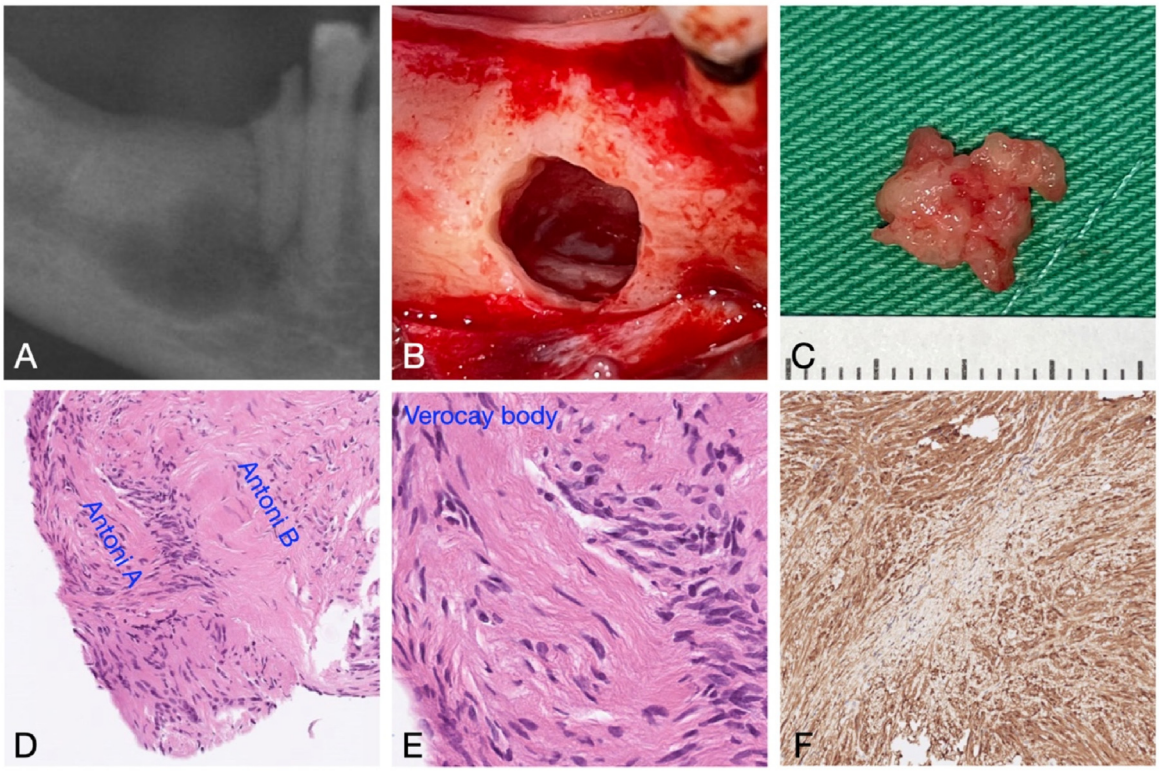

Figure 1 Radiographic, clinical, and gross photographs as well as histopathological microphotographs of our case. (A) Panoramic radiograph displayed a well-defined unilocular periapical radiolucent lesion superimposed with the inferior alveolar canal. (B) Surgical operation involving mucoperiosteal flap, bone window, and excision of the tumor. The mental foramen and mental nerve were situated at the mesial side of the bone window and the inferior alveolar nerve was located at the inferior portion of the bone cavity. (C) The excised schwannoma exhibited as a pale-colored solid tumor. (D) Histopathological microphotographs illustrating a hypercellular area with streaming fascicles and a palisaded arrangement of schwannoma cells (Antoni A tissue), and a hypocellular area with less organized and loose myxomatous stroma (Antoni B tissue). (E) Verocay body shown in the central acellular eosinophilic area. (H&E; original magnification; D, 20×; E, 40×) (F) Immunohistochemical stain revealed that the tumor schwannoma cells were diffusely positive for S-100 protein. (Immunostain; original magnification; F, 20×).

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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