



Oral Mandibular Peripheral Osteoma: A Case Report

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ABSTRACT

Peripheral osteomas of the mandible are rare, benign osteogenic tumors characterized by slow-growing cortical or cancellous bone proliferation. This report presents a case of a peripheral osteoma in the mandibular angle and reviews potential etiological factors. A 72-year-old woman presented with a painless, asymptomatic mandibular bone asymmetry and was referred to the oral and maxillofacial department of Isfahan University of Medical Sciences with no visible asymmetric changes in facial soft tissue. The lesion was a 4 cm pedunculated radiopaque mass located on the buccal surface of the left posterior mandible, as confirmed by computed tomography. Surgical excision revealed histopathological features characteristic of lamellar bone admixed with some woven bone, consistent with a diagnosis of peripheral osteoma. Postoperative recovery was smooth, and there was no evidence of recurrence at 1-year follow-up. Peripheral osteomas frequently occur in the mandible and can lead to functional disruptions or cosmetic concerns. Trauma and chronic muscle tension can contribute to their formation, but many cases arise without a clear cause. Typically asymptomatic, surgical removal is the most effective treatment with low recurrence rates. A thorough radiographic assessment is essential; more research is needed to understand their molecular mechanisms.

Keywords: Peripheral osteoma; Mandible; Benign bone tumor; Case report; Risk factors.

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Introduction

Osteoma is a type of osteogenic tumor resulting from bone proliferation. Although it can occur in any bone, it is uncommon to involve the facial bones. This benign tumor is distinguished by its slow and generally confined growth [1,2]. Osteomas are mainly made up of well-formed cancellous or compact bone. Osteomas can develop on the bone surface as periosteal (peripheral or exophytic) osteomas or endosteal (central) osteomas within the medullary cavity [3]. Jaw osteomas are most commonly found in adults, with a particular tendency for the mandibular body and condyle. Lesions in the mandibular body are often located on the lingual surface next to the premolars or molars [2,4,5]. Locations found less frequently include the angle (especially at the inferior border), ramus, and coronoid process. Typically asymptomatic, osteomas are primarily influenced by their size and position. These lesions are usually small, solitary, slow-growing, and painless, often only becoming noticeable during routine check-ups. As they grow, they may enlarge and exacerbate signs and symptoms. Possible complications include facial asymmetry, contralateral mandibular deviation, limited mouth opening, and discomfort [6]. There is debate over whether osteomas are true neoplasms, and not all lesions classified as osteomas may represent a single entity. Some are likely the result of injury, an inflammatory response, or a hamartomatous process. Since some osteomas develop in regions where muscles attach to bone, researchers have proposed that muscle traction might play a role in their formation [7-10]. Although the precise origin of osteomas remains uncertain, numerous potential etiological factors, including traumatic, developmental, congenital, inflammatory, and endocrine causes, have been suggested. Multifocal lesions can occur in connection with Gardner syndrome. Additionally, unilateral jaw osteomas have been observed in relation to encephalocraniocutaneous lipomatosis (Haberland syndrome) [11].

Case Report

A 72-year-old female patient with a history of chronic hypertension has been referred to the Department of Oral and Maxillofacial Medicine. During a consultation with a maxillofacial surgeon regarding a dental implant treatment plan, a radiographic examination revealed a radioopaque mass with well-defined borders measuring approximately 2 × 4 cm in the posterior mandible (Figure 1). The patient's prior radiographs were assessed, revealing that the lesion has been

present for approximately five years and has recently enlarged. The patient reports no discomfort or tingling sensations. Additionally, there is no documented history of trauma or infection, and the patient's medical history does not contain any relevant information. A firm mass, approximately 3 cm in diameter, was palpable during the clinical examination. Based on the radiographic findings and clinical assessment, differential diagnoses included osteoma, osteoblastoma, and osteosarcoma. Given the progression of the lesion, the potential for malignancy, and the patient's discomfort, an excisional biopsy was deemed necessary as part of the treatment plan. Surgery was conducted via extra-oral access under general anesthesia. A 4 cm incision was made in the submental area, and the lesion was excised in three separate pieces (Figure 2). The samples were then sent to pathology for histologic analysis. The patient was closely monitored during the postoperative period, and the recovery was smooth. The patient has been monitored for one year, during which no recurrence has been observed. Pathological examination revealed that the neoformed mass comprises lamellar bone interspersed with some woven bone. The bone exhibits a cortical-type architecture, with osteoblasts and osteocytes appearing inconspicuous. The definitive diagnosis was osteoma. The patient has been monitored for one year, during which no recurrence has been observed.



Figure 1. Lateral CBCT view of the internal aspect of the mandible, revealing the osteoma lesion.



Figure 2. Resected lesion surgical view.

Discussion

Osteomas are benign, slow-growing osteogenic neoplasms characterized by proliferation of compact and/or cancellous bone tissue. Although these tumors can arise throughout the craniofacial skeleton, those that affect the mandible have distinct etiological factors and associated risks [11,12]. This report examines the current scientific understanding of the specific elements contributing to the development of mandibular osteomas, with a particular emphasis on trauma, muscle activity, inflammatory processes, and anatomical predisposition. Despite their benign nature, mandibular osteomas can cause significant functional and aesthetic concerns, depending on their size and location, underscoring their etiopathogenesis for effective diagnosis and management. Radiographically, osteomas typically present as well-defined sclerotic masses, though early lesions may appear radiolucent or exhibit mixed radiolucent-radiopaque characteristics [13,14]. Periosteal osteomas can show a uniformly sclerotic pattern or a sclerotic periphery with central trabeculation [11].

Histopathologically, compact (or “ivory”) osteomas consist of normal-appearing, dense bone with minimal marrow. In contrast, cancellous osteomas are characterized by bony trabeculae and fibrofatty marrow, with prominent osteoblastic activity in some cases. Certain sino-orbital osteomas exhibit “osteoblastoma-like features,” described by enlarged osteoblasts, woven bone, and a loosely organized fibrovascular stroma [11,15,16]. Small, asymptomatic osteomas typically do not require intervention; however, periodic monitoring for any changes is essential. Conservative excision is recommended for more extensive or symptomatic mandibular body osteomas. Frequently presented symptoms of condylar osteomas are generally removed through local resection or condylectomy [17,18]. Symptomatic paranasal sinus osteomas may be excised endoscopically or through an open surgical approach. Recurrence after excision is rare, and no documented cases of malignant transformation have been reported [18,19].

Theories on Etiology and Pathogenesis of Oral Osteomas

The precise cause of oral osteomas is not well understood, with various theories in scientific literature. These lesions can be classified as developmental anomalies, true neoplasms, or reactive lesions from specific stimuli [20]. The developmental theory suggests osteomas are malformations from embryonic development or early postnatal stages. Proponents of the neoplastic

theory note that these lesions exhibit genuine neoplastic traits, showing autonomous growth independent of normal physiological processes. In contrast, the reactive theory posits that osteomas develop in response to local stimuli or environmental factors affecting bone metabolism [11]. Several factors have been associated with the development of oral osteomas, including trauma, muscle strain, and infections [20]. Trauma can trigger inflammation that stimulates osteoblastic activity, leading to abnormal bone formation. Prolonged muscle tension at tendon attachment sites may also promote bone growth as a response to mechanical stress. Chronic infections can trigger inflammatory responses that contribute to excessive osteoblastic proliferation. Additionally, congenital disorders and hormonal changes have been proposed as potential causes, although conclusive evidence for a specific mechanism remains elusive [21]. The literature reports multiple hypothetical mechanisms that may explain the development of mandibular osteomas. The first is the developmental theory, which suggests that osteomas arise from congenital abnormalities that occur during embryonic or early postnatal stages. This is controversial, given that most reported cases occur in adults rather than during the growth stages typically associated with developmental lesions. This timing discrepancy contradicts what is anticipated for genuinely congenital or developmental lesions, which usually become evident during times of active growth [22].

The second theoretical framework views osteomas as true neoplasms caused by chronic inflammation. This hypothesis posits that ongoing inflammation instigates osteoblastic proliferation, ultimately leading to tumor formation. However, this theory has also lost acceptance among researchers, as it does not account for the distinctly slow and self-limiting growth pattern in most osteomas. If these lesions were genuinely neoplasms driven by inflammation, more aggressive and unrestricted growth patterns would be expected, which conflict with clinical observations. Moreover, many documented cases do not show signs of any preceding or concurrent inflammatory conditions that could have initiated such a phenomenon [22]. The reactive theory, currently the most widely accepted explanation for peripheral osteomas, merges trauma and muscle activity elements as key contributing factors. This model suggests that an initial traumatic incident leads to subperiosteal bleeding, while concurrent or subsequent traction from muscles elevates the periosteum locally. This combination of elements triggers an osteogenic response that leads to abnormal bone formation. The

masseter muscle often plays a significant role in this process, especially in the formation of osteomas along the angle or body of the mandible. This theory effectively accounts for the tendency of peripheral osteomas to occur along the lower border and buccal region of the mandible, which are particularly vulnerable to traumatic injuries and subject to considerable muscle forces [22].

Mechanisms of Trauma-Induced Osteogenesis as a Primary Risk Factor

Trauma is frequently recognized in the literature as a significant risk factor for the development of mandibular osteomas. Both clinical observations and theoretical biomechanical principles support this correlation. Even minor mandibular impacts can trigger a cascade of biological responses that may lead to osteoma formation. Initially, the traumatic event typically results in subperiosteal hemorrhage, creating a hematoma between the periosteum and the underlying cortical bone. This hematoma gradually organizes and may ossify, potentially serving as the nidus for osteoma development [22].

Interestingly, many patients with mandibular osteomas do not recall a specific traumatic incident, suggesting that significant osteomas may arise from relatively minor traumas that did not necessitate medical attention or remain memorable to the individual. This observation supports the reactive theory, which posits that even minor traumatic events can trigger the pathophysiological processes leading to osteoma formation. Although the patient may forget the initial trauma due to its minor nature, the biological response can continue, potentially sustained by other factors such as muscle activity [22]. Case reports in the literature provide some evidence for the trauma hypothesis. For instance, one documented case involved a 14-year-old boy with a large solitary peripheral osteoma located on the buccal surface of the left posterior mandible, who had experienced facial trauma approximately five years before presentation [23]. However, not all cases demonstrate this association; another report describes a 30-year-old male with a peripheral osteoma of the mandible who had no history of trauma or infection [24]. This inconsistency suggests that while trauma may play a significant role, it is likely not the sole factor in osteoma development.

Biomechanical Influences of Muscle Activity and Anatomical Considerations

According to the reactive theory, muscle activity, par-

ticularly that of the masticatory muscles, plays a significant role in the formation of mandibular osteomas. The masseter muscle is often highlighted due to its considerable force generation and its attachment to the lateral surface of the mandibular ramus and angle. This theory posits that continuous muscle traction locally elevates the periosteum, creating a microenvironment conducive to abnormal bone formation. This mechanical influence may help explain why peripheral osteomas frequently arise at sites corresponding to muscle attachments, particularly along the mandible's lower border or buccal aspect [22].

The observed distribution pattern of mandibular osteomas further supports the biomechanical rationale. These lesions preferentially involve the mandibular body, angle, and condyle regions, which endure significant masticatory forces during normal function. The persistent mechanical stimulation in these areas may foster conditions favorable for excessive bone formation, especially when combined with other initiating factors such as trauma. This interplay between traumatic stimuli and ongoing mechanical forces provides a compelling explanation for the development of peripheral mandibular osteomas [22]. Moreover, the mandible's anatomical configuration may enhance its susceptibility to osteoma formation. As the sole mobile bone in the craniofacial skeleton, the mandible experiences complex biomechanical forces during mastication, speech, and other functional activities. These forces are not evenly distributed across the bone, with certain regions experiencing greater mechanical stress than others [25-27]. The concentration of forces at specific anatomical locations may create conditions that encourage abnormal bone proliferation, potentially explaining the predilection of osteomas for particular sites within the mandible.

Inflammatory, Infectious, and Endocrine Influences as Additional Contributing Factors

In addition to trauma and muscle activity, several other factors have been suggested to contribute to the formation of mandibular osteomas. Inflammatory mechanisms have been identified as potential contributors, creating a localized environment enriched with cytokines and growth factors that may promote osteoblastic activity. Similarly, infectious processes have been proposed as triggers for excessive bone activity that could lead to osteoma development [24]. However, the precise mechanisms through which inflammation or infection might facilitate osteoma formation remain incompletely understood, and numerous cases lack ev-

idence of prior inflammatory or infectious conditions. Endocrine factors have also been highlighted as possible contributors, though the literature has not thoroughly characterized specific endocrine abnormalities associated with an increased risk of mandibular osteoma. The endocrine hypothesis suggests that altering hormones that regulate bone metabolism could create conditions favorable for abnormal bone proliferation. Nonetheless, this hypothesis remains theoretical and lacks supporting evidence [11,28,29]. It is essential to recognize that, although numerous factors have been proposed, many cases of mandibular osteoma occur without identifiable risk factors [27,30-32]. This observation suggests a multifactorial etiology, potentially involving genetic predispositions or other unrecognized influences. The inconsistent presence of proposed risk factors across documented cases underscores the complexity of osteoma pathogenesis and highlights the need for further research to clarify the specific mechanisms involved.

Syndromic Associations and Genetic Considerations

While most mandibular osteomas present as solitary lesions, multiple osteomas may be associated with Gardner's syndrome, an autosomal dominant disorder characterized by intestinal polyposis, soft-tissue tumors, and skeletal abnormalities [33]. Patients with this syndrome often develop several osteomas in the skull and mandible, frequently arising before the onset of intestinal polyps. This association emphasizes the potential genetic influences on osteoma development in specific circumstances, although such cases are relatively uncommon [11]. The syndromic association suggests that genetic factors may also contribute to the development of sporadic mandibular osteomas [33,34]. Certain genetic variations may predispose individuals to abnormal bone growth due to environmental factors such as trauma or mechanical stress. However, the existing literature has not thoroughly explored specific genetic markers associated with an increased risk of non-syndromic mandibular osteomas [11,22].

Clinical Implications for Preventive Strategies and Therapeutic Approaches

Identifying the risk factors linked to mandibular osteomas is essential for effective prevention and management. Although complete prevention may be unrealistic, mitigating potential risk factors is practical. This includes using protective measures during high-risk activities, especially in contact sports or jobs with a higher risk of facial injury [35,36]. For patients diagnosed with osteomas, management decisions should consider

ongoing risk factors that may affect lesion behavior or recurrence risk. Mandibular osteomas generally exhibit low growth potential and infrequently recur following complete excision. Therefore, it is advisable to address modifiable risk factors, particularly when a conservative treatment approach is favored [18,36,37].

Conclusion

According to the literature, mandibular osteomas are primarily associated with trauma and muscle activity. The reactive theory suggests that trauma can lead to subperiosteal bleeding, while muscle tension promotes abnormal bone growth, particularly in areas affected by these forces. Additionally, inflammation and hormonal changes might contribute, though their roles are less defined. Understanding these factors and their interactions is essential for developing preventive strategies.

Conflict of Interest

There is no conflict of interest to declare.

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