CASE REPORT

Subpontic osseous hyperplasia: report of two cases

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Abstract

Subpontic osseous hyperplasia is a non-neoplastic growth of bone under the pontics of fixed partial dentures, whose pathogenesis is unknown. It is considered a common, not well-known and under-diagnosed lesion. Its clinical and radiographic features are diagnostic, and its recognition will help avoid diagnostic or therapeutic procedures unnecessary to the patients. We present two cases of subpontic osseous hyperplasia and discuss its differential diagnosis and current concepts on pathogenesis and management.

Clinical relevance

A slowly growing enlargement beneath the pontic of a fixed partial denture may represent subpontic osseous hyperplasia, a lesion of unknown pathogenesis that may imitate other developmental or neoplastic diseases. Recognition of its clinical and radiographic features that are diagnostic will protect the patient from unnecessary diagnostic or therapeutic procedures.

Introduction

The non-neoplastic growth of bone under the pontics of fixed partial dentures (FPD) was described as ‘subpontic osseous hyperplasia’ (SOH) by Calman et al. in 1971, and by subsequent authors as plateauitization, subpontic osseous proliferation, subpontic bony deposition, bilateral subpontic osseous hyperplasia, hyperostosis, subpontic hyperostosis and subpontic tissue enlargement. Lee et al. in 2014 reviewed 54 cases of SOH from the English-language literature and added three more cases. No other case has been published since this report, although it is certain that this condition is not rare but under-diagnosed.

SOH usually develops in Caucasians patients of both sexes, with an average age of 56.6 years. It is mostly related with a 3-unit FPD with abutments that are full-coverage crowns,¾ crowns or inlays, and a pontic that is bar-shaped or hygienic. Save for two cases that developed in the area of the maxillary second premolar and first molar, respectively, all other published cases of SOH relate to the mandible. Clinically, there is a slow-growing enlargement beneath the pontic that is covered by normal mucosa and has a flat or lobular surface. The size and shape of the lesion depends on the growth stage, the size of the edentulous area and the shape of the space between the pontic and the ridge. It is usually unilateral, even when there is a FPD on the opposite side; however, bilateral cases have been reported.

The radiographic findings vary. In cases where removal of the lesion was performed, microscopic examination showed normal lamellar bone.
that in one case was in continuity with calcified hyaline cartilage.

We describe the clinical, radiographic and microscopic features of two new cases of SOH.

**Case 1**

A 59-year-old male had an FPD made of gold–palladium fused to porcelain in the left posterior mandible (abutments teeth #35 and #37, pontic tooth #36). The FPD was placed 18 years ago in order to replace a preexisting FPD whose pontic was broken. His medical history included urethral polyps (2003), nephrolithiasis (2008) and hypercholesterolaemia with a daily administration of atorvastatin calcium. The patient did not smoke.

During a routine clinical examination, it was noted that the subpontic space was obliterated, although the alveolar ridge appeared normal (Fig. 1). The occlusion of the FPD and marginal fit of the abutments were satisfactory. A cervical loss of dental substance consistent with abfraction was noted on tooth #25 (Fig. 2). No pathological findings were observed in the rest of the dentition the periodontal tissues and the oral mucosa. Probing depths were \( \leq 3 \) mm. Oral hygiene both in the area of the FPD, as well as in the rest of the oral cavity was good, although cleaning with SuperFloss\textsuperscript{®} was complicated by the close contact of the pontic with the mucosa. A periapical radiograph showed two subpontic growths: a larger proximal one, presenting as a radiopaque mass beginning at the top of the ridge and ‘embracing’ the pontic, and a smaller distal one with trabeculation that was in smooth continuity with the underlying bone and did not reach the pontic (Fig. 3). The study of periapical radiographies taken 18, 14 and 4 years ago showed that the growths were present and enlarged gradually (Fig. 3).

Based on the patient’s medical history and the clinical and imaging findings, a diagnosis of SOH was established. As the patient did not report any discomfort and the growth did not impede FPD function or oral hygiene practices, regular follow-up was decided.

**Case 2**

A 59-year-old female complained of difficulty in performing oral hygiene procedures in the area #33-36, where she had an FPD (abutments teeth #33 and #36, pontics teeth #34 and #35). The FPD was placed ‘many’ years ago. The patient’s medical history was non-contributory.

On clinical examination, a hard swelling covered by normal mucosa was seen in the buccal area of the pontic, occluding the subpontic space (Fig. 4). No other pathological findings were observed in the rest of the dentition, the periodontal tissues and the oral mucosa. The occlusion of the FPD and marginal fit of the abutments were satisfactory. Although oral hygiene was generally good, the gingivae of the abutments adjacent to the edentulous area were enlarged, erythematous and mildly haemorrhagic. A panoramic radiograph showed bone with normal trabeculation that was in contact with the pontic (Fig. 5).

Based on the clinical and imaging findings, SOH was diagnosed. Due to the size of the lesion and the patient’s difficulty in performing oral hygiene procedures, total surgical removal was decided. After removal of the FPD, an erythematous ‘impression’ on the gingivae where the pontic was contacting was revealed (Fig. 6).

Under local infiltration anaesthesia, lingual and buccal full thickness mucoperiosteal flaps were reflected and the bone was removed with a surgical bur. Post-operative healing was uneventful and the anatomy of the area was restored. One year after the removal, no recurrence of the lesion was observed.

The tissue specimen was fixed in 10% buffered formalin, decalcified in Osteodec (Bio-Optica, Miano, s.p.a., Italy), embedded in paraffin, and 5 \( \mu \)m thick sections were stained with haematoxylin and eosin.
The microscopic examination showed trabeculae of cellular, fibrous bone surrounding medullary spaces containing fibro-fatty tissue (Fig. 7). A diagnosis consistent with subpontic osseous hyperplasia was established.

**Discussion**

In the two aforementioned cases, the history and clinical, radiographic and microscopic findings were consistent with SOH\(^1\). In accordance with previously reported cases\(^3\,9\), our cases presented in adults, developed in the area of the first mandibular molar, were covered by normal mucosa and presented radiographically as osseous hyperplasia. In the first case, the patient had a 3-unit FPD and in the second, a 4-unit FPD. In the review of Lee *et al.*\(^9\), 30 out of 35 cases where this information was reported developed in association with a 3-unit FPD, and 3 cases
with a 4-unit FPD. The FPDs were placed several years before presentation, as in previously published cases where this period ranged from <1 year\textsuperscript{3} to approximately 40 years\textsuperscript{12}, with an average of <7 years\textsuperscript{9}. In our first case, the growth was detected incidentally during the patient’s regular re-examination, as it did not cause any discomfort\textsuperscript{6,12}, while in our second case, the patient could not perform oral hygiene procedures, as has been occasionally observed\textsuperscript{3,6,9,2,13,15}, predisposing to the development of caries or periodontal disease in the abutment teeth\textsuperscript{5,15,16}. FPD dislodgement\textsuperscript{3,6,15} or discomfort and pain in the area of the hyperplasia are rare.

Radiographically, the presence of two distinct lesions, as noted in the first case, is unusual and has been reported only in one case\textsuperscript{13}. The difference in the imaging findings between the two cases is not unusual, as the radiographic picture may vary considerably. The lesions are described as radiopaque, compact without trabeculation, cortical, sclerotic, trabeculated or mixed, while the underlying bone may be in continuity with the lesion, separated from it with a thin discernible line, or show a sclerosing reaction\textsuperscript{9,11}.

The differential diagnosis includes exostosis, peripheral osteoma and low malignancy osteosarcoma. Exostoses are more frequently bilateral on the lingual surface of the mandible (torus mandibularis) or the midline of the hard palate (torus palatinus) and more rarely on the labial gingivae of the maxilla or the mandible\textsuperscript{17}. As hamartomas, they do not usually have a tendency to grow. In the cases presented herein, the location and gradual growth are not consistent with exostosis. It is noted, however, that some authors consider subpontic osseous hyperplasia to be a form of exostosis\textsuperscript{13,17}.

Osteoma is a benign bone tumour that may be characterized as central or peripheral (periosteal) depending on its location, and compact or spongy, depending on its histological features\textsuperscript{17,18}. Peripheral osteoma is mainly located in the paranasal sinuses and rarely in the jaws, more frequently labially to the maxilla. It manifests as a solitary painless tumour with a distinct border, is covered by normal mucosa and grows slowly. Its radiographic image is similar to exostoses. No case of peripheral osteoma in relation to subpontic osseous hyperplasia was found in the pertinent literature. However, it is noted that in the second case presented in this article, where removal of the growth and histological examination was required, the appearance of fibrous bone with fibro-fatty tissue is more consistent with spongy osteoma, in contrast with previous reports.
that describe a hyperplastic lamellar bone similar to exostoses\textsuperscript{5,6,15}.

Finally, the radiographic findings in both cases presented, rule out endosteal and peripheral periosteal osteosarcoma. The extremely rare parosteal osteosarcoma may have the form of a lobated tumour covered by normal mucosa, grows slowly and presents radiographically as a radiopaque mass with a clear border that adheres to the underlying bone without infiltrating it\textsuperscript{17,19}. However, in the cases presented herein, the slow growth of the lesion excludes the possibility of a malignant neoplasm.

Genetic predisposition and reactive bone formation in response to functional stress are the aetiologic factors most commonly implicated in the pathogenesis of SOH\textsuperscript{9,13}. Genetic predisposition is supported by the coexistence of SOH with exostoses in 62\% of the cases, whereas exostoses occur in \textless10\% to 40\% of the population\textsuperscript{3,6,9,13}. In addition, it is suggested that in some cases of SOH, enlarged maxillary lingual exostoses or mandibular buccal exostoses participate in the formation of the growth\textsuperscript{3}. Reactive bone formation due to stress forces exerted in the area of the pontic by muscle hyperactivity may justify the formation of the growth, as well as its detection exclusively in the mandible\textsuperscript{9}, but it cannot explain the absence of the growth from contralateral FDP in the same patient\textsuperscript{3,13}. In case 1, the presence of abfractions in the opposing arch is indicative of ‘overload’. Subperiosteal bone deposition due to mild chronic inflammation developing in the edentulous ridge because of the presence of the pontic has been discussed by many authors. The presence of the pontic creates favourable conditions for the development of inflammation of the mucosa, even under a hygienic pontic, which may be worsened by trauma from oral hygiene practices\textsuperscript{9,13}. However, in the cases where a biopsy was performed, inflammation was not a common finding\textsuperscript{12}. Other possible aetiologic factors that have been mentioned are the negative pressure created under the pontic and electric currents\textsuperscript{9}. The role of the material of the FDP has not been explored, even though, as shown in the review of Lee \textit{et al.}\textsuperscript{9}, out of the 21 cases, 15 cases employed a gold alloy, 5 cases porcelain fused to metal and 1 silver.

Aesthetic or functional reasons such as difficulty in the retention or maintenance of the FDP, or speech, or when the diagnosis has to be confirmed, surgical removal and restoration of the osseous border of the edentulous ridge is required\textsuperscript{3}. Replacement of the FDP or substitution with osseointegrated implants has been suggested, but not documented, by some authors\textsuperscript{3,5,15,16}. Recurrence of SOH even after FPD removal\textsuperscript{11,15} or total excision\textsuperscript{3,9}, and the use of the removed bone of the growth as an autologous transplant in another position\textsuperscript{9} have been reported. In the first case presented in this article, the absence of discomfort led to the decision to continue follow-up, while in the second case, the lesion was surgically excised and the FDP was replaced.

In conclusion, SOH is an under-diagnosed lesion of unknown pathogenesis that presents diagnostic clinical and radiographic features, whose recognition will help to avoid unnecessary diagnostic or therapeutic procedures. Functional and aesthetic considerations dictate the decision to remove the lesion.

**Conflict of interest**

None declared.

**References**