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Subpontic osseous hyperplasia: Three case reports and literature review

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ABSTRACT

Subpontic osseous hyperplasia (SOH) is an ectopic growth of bone occurring on the edentulous ridge beneath a fixed partial denture. The aim of this article is to present three patients with SOH with long-term follow-up and to make a current overview of the literature. Presented maxillary SOH is the second case in the dental literature. Intraorally the lesions were bone-hard and painless swellings in the subpontic space. Radiographically, the maxillary case was a nodular bone growth having similar radiodensity and trabeculation to the adjacent bone with a thin radiopaque border. Mandibular cases were bony growths, which were more radiopaque than the adjacent alveolar ridge. Follow-up radiographs revealed regression in two of the cases. Clinicians should take care not to cause the possible etiologies of SOH such as functional stresses and chronic irritation by the prosthetic treatments and be aware of SOH does not usually require treatment or a biopsy.

Key words: Fixed partial denture, pontic, subpontic osseous hyperplasia

INTRODUCTION

Subpontic osseous hyperplasia (SOH) is the slow-growing, benign proliferation of the alveolar bone beneath the pontic region of a fixed partial denture (FPD). This term is used owing to the microscopic, radiographic, and clinical features of the lesion.[1] The terms subpontic osseous proliferation,[2,3] plateauzation,[4] subpontic osseous hyperostosis,[5] subpontic cartilagenous hyperplasia,[6] subpontic tissue enlargement,[7] and subpontic hyperostosis[8] were also used.

The aim of this article is to present three patients with SOH and to make a current overview of the literature.

CASE REPORTS

Case 1

In year 2005, a 55-year-old female patient presented for renewal of her prosthetic restorations that were placed 15 years ago. Her medical history was significant for a breast cancer surgery and chemotherapy-radiotherapy treatment 4 years ago and for osteoporosis treatment for 3 years. There was a four unit FPD replacing the mandibular right second premolar and first molar teeth. Panoramic radiograph revealed a hemispherical bony growth beneath the pontic of the mandibular first molar, which was partially more radiopaque than the adjacent alveolar ridge and was less trabeculated with the loss of bone marrow areas [Figure 1a]. When the FPD was removed and the patient was reexamined, a bone-hard, painless swelling was detected on palpation. Irritation caused by the pontic was evident on the crestal mucosa [Figure 1b]. The patient was unaware of the condition and she had no complaints related with the bony growth. A clinical diagnosis of SOH was made and the patient was scheduled for follow-up. The patient presented again in year 2011; history and clinical examination revealed that in year 2007 a root canal treatment was made for the mandibular first premolar and the first premolar, and

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the second molar teeth were crowned. After extraction of other carious teeth, maxillary and mandibular removable partial prostheses were constructed. At the time of follow-up appointment, she had no complaints; she was using her removable partial prostheses. Intraoral examination of the previous subpontic area revealed smooth mucosal surface, there was no pain or swelling on palpation. Panoramic [Figure 1c] and periapical radiographs showed that the area which was less trabeculated than the surrounding bone was larger than in the initial radiographs; however, the dome shaped alveolar crest was flattened.

Case 2

A 61-year-old systemically healthy male patient presented to our clinic in year 2006 with a chief complaint of hypersensitive abutment teeth. The prosthetic restoration was placed 3 years ago. After 6 months cementation of the restoration, the patient had noticed a slow-growing swelling beneath the pontic region but as there were no symptoms he did not seek for treatment. Intraoral examination revealed a bone-hard, painless swelling in the subpontic space of a three unit FPD that replace the mandibular left first molar tooth [Figure 2a]. Panoramic radiography revealed a nodular bony growth under the pontic region, which was more radiopaque than the surrounding bone and secondary caries at the tooth 37 [Figure 2b]. When the FPD was removed, irritation caused by the pontic on the crestal mucosa observed [Figure 2c]. A diagnosis of SOH was made on the basis of clinical and radiographic examinations. As the lesion was asymptomatic, the patient refused surgical treatment. The patient was referred to the conservativ e treatment department for secondary caries at tooth 37. In 2007, there were no complaints and there were no dimensional changes in the lesion [Figure 3a]. In 2012, the patient came to renew the FPD. Panoramic radiograph revealed that the bony growth was larger although the density remained almost the same [Figure 3b].

Case 3

A 55-year-old systemically healthy male patient attended our clinic in year 2006 for renewal of his maxillary fixed partial dentures, which were made ~15-20 years ago. Intraoral examination revealed

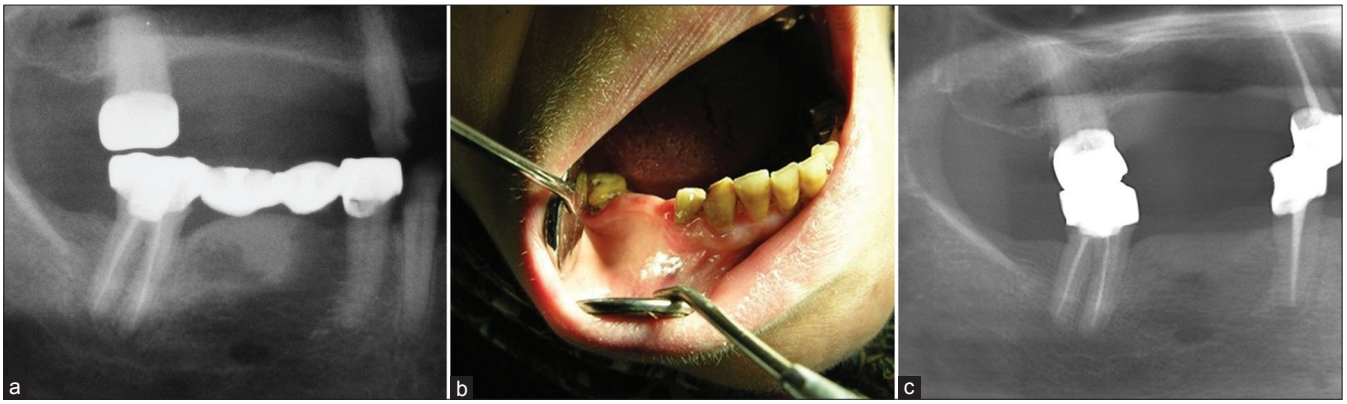

Figure 1: (a) A cropped panoramic radiograph showing hemispherical bony growth beneath the pontic of the mandibular first molar (b) Intraoral photograph showing mucosal irritation caused by the pontic of the mandibular right first molar on the crestal mucosa (c) Follow-up panoramic radiograph demonstrating the subpontic osseous hyperplasia area that is less trabeculated than the surrounding bone

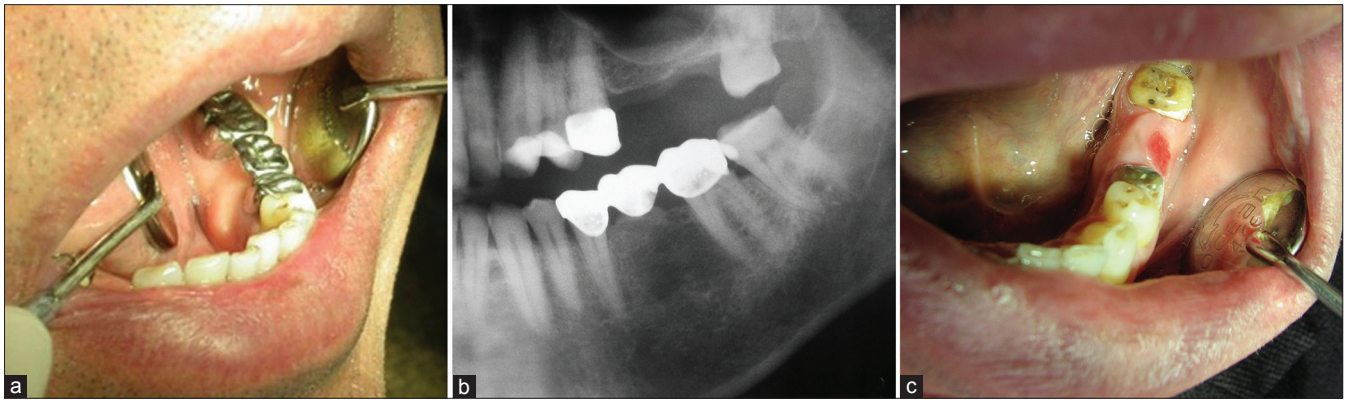

Figure 2: (a) Intraoral photograph showing lingual swelling in the subpontic space (b) Cropped panoramic radiograph showing the radiopaque nodular bony growth in the pontic region (c) Intraoral photograph showing irritation on crestal mucosa

a poor-fitting five unit FPD replacing the maxillary right canin and the second molar. Beneath the second premolar pontic, a well-defined, nodular bone growth was detected on the periapical radiograph [Figure 4a]. The radiodensity and trabeculation of the lesion was not different from the adjacent bone: However, a thin radiopaque border between the alveolar crest and the nodular growth could be detected. The contour of the alveolar ridge was normal under the first premolar and first molar pontics. The patient was reexamined after the restorations were removed and a well-defined, nodular, painless bony growth, which was covered with normal appearing mucosa, was detected on the second premolar region [Figure 4b]. The hard tissue mass was clinically diagnosed as SOH. In year 2007, a panoramic radiograph was taken and there was neither a change in the size or in the radiographic pattern of the lesion and the patient's poor-fitting prosthetic restorations were renewed [Figure 5a]. In 2011, a periapical radiograph was taken and regression of the bony growth was noticed [Figure 5b].

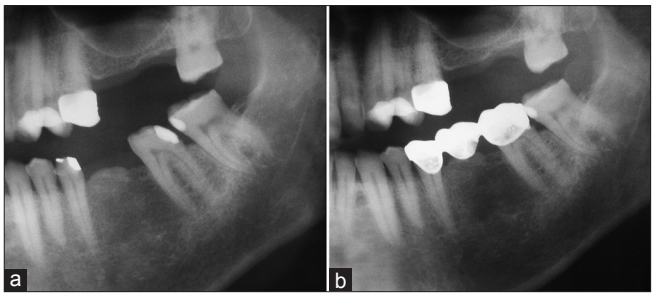

Figure 3: (a) Follow-up panoramic radiograph indicating no dimensional change in the lesion taken in the year 2007 (b) Follow-up panoramic radiograph showing growth of the lesion taken in the year 2012

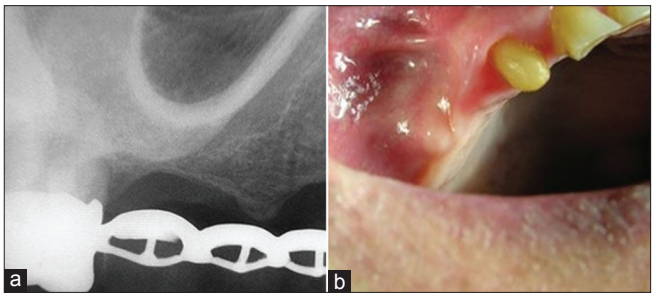

Figure 4: (a) Cropped periapical radiograph showing the nodular bone growth beneath the maxillary right second premolar pontic (b) Intraoral photograph showing the bony growth, which was covered with normal appearing mucosa on the second premolar region

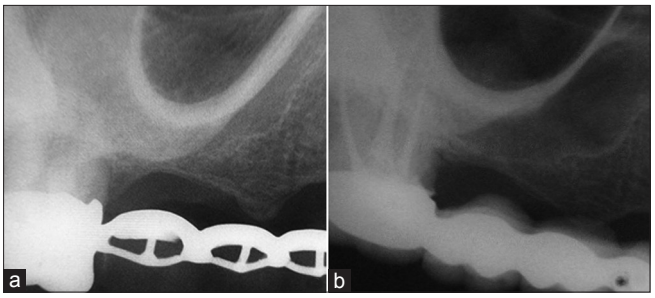

Figure 5: (a) Follow-up periapical radiograph showing no change in lesion taken in the year 2007 (b) Follow-up periapical radiograph taken in the year 2011 showing regression of the bony growth

DISCUSSION

SOH can be diagnosed according to patient's history together with the clinical and radiographic findings, and it was stated that the presence of a FPD encourages a diagnosis of SOH. However, it must always be kept in mind that other pathoses may also be incidentally located beneath a pontic.[9] SOH may simulate osteitis deformans, osteomyelitis and Gardner's syndrome.[1] Suspected lesions should be biopsied and definitive diagnosis can be made by histopathologic examination.

SOH was first described in 1971 by Calman[10] and this entity can be seen both in men and women with a wide age range. Together with the cases presented, the mean age of the patients is 56.08 (min: 25, max: 81) and the female/male ratio is almost equal (25 females and 24 males).[11,12] SOH mostly occur on the mandible, under the first molar pontics.[1,3,5-8,11-17] There are eight bilateral cases reported in the literature.[2-5,12,18] Our third patient is the second case with a SOH located in the maxillary arch.[15] Currently, it is not exactly known if there is a real preponderance of SOH for mandible, but these two cases suggest that SOH is not unique for mandible.

Genetic pre-determination, functional stresses, and chronic irritation were suggested as etiologic factors.[2,12] Appleby[8] stated that remission of the lesion after the loss of FPD in his case supports the theory of functional stresses or stimuli as an etiologic factor. In two of our cases remission of SOH was noticed and these cases also support the functional stress theory as a cause of SOH. However, remission does not exclude the presence of other possible etiologic factors and the exact etiology of this proliferation is still not known.

In some cases, the patients were unaware of the condition.[2,8-12] In other cases, complaints relevant to the FPD such as loose FPD, broken connector of FPD, complaints in the area of abutment teeth and pain under the pontic were present and lead to diagnosis.[2,11,12,17,18] Inability to floss under the pontic[5,6,11,13-15,17] was the most frequent complaint related with SOH and a few

patients were complaining of bone growths[2,5,7,12,16,17] under the pontic. In the cases presented, only one of the patients was aware of the condition and noticed a slow-growing swelling beneath the pontic.

Clinically, subpontic soft-tissue is in intimate contact with the convex surface of the pontic or subpontic space is completely obliterated.[6,11] The lesion presents as a bony growth beneath the pontic.[2,5,8,12,14,17] Although mostly solitary nodular[2] and hemispherical[13,18] lesions, SOH can also be bilobed.[2,13] On palpation the lesion was described as firm and as bone-like hard[6,17,18] and this feature was also detected in our cases.

On the radiograph, expansion that reduces the subpontic area is generally seen more radiopaque than the neighboring alveolar bone. In some cases, the transition between the bony growth and the adjacent alveolar bone was smooth and in others there was a clearly defined junction; in some patients a thin radiolucent line separated the mass from the underlying bone. In a number of patients a sclerotic reaction was observed in the bone of the edentulous ridge adjacent to the bony growth.[6,12,17] Sclerotic reaction was detected only in our first case. The radiographic features of SOH are shown in Table 1.

The size of the bony growth is dependent on the width of the pontic span, the height of the inferior border of the pontic above the edentulous ridge and on the stage of growth at which the hyperostosis is observed. In some patients, the bony growth demonstrated no change in size after initial observation,[12,17] whereas in other patients the bony growth revealed increases in size at an evaluations[2,3,5,7,11,12,18] In one case, the FPD had fallen out and incidental remission of SOH was observed.[8] In the first case presented, the lesion was flattened after removal of the FPD and construction of a removable partial denture, and in the third case the lesion regressed after the poor-fitting FPD was replaced. In our second case, bony growth became larger in 5 years although the density remained almost the same.

Histopathologic features of SOH are similar to that of normal compact bone or dense cortical bone with trabecular bone and reported to be similar to other exostoses.[1,11,15,16,18] Our cases were not biopsied; however, the lesions were clinically and radiographically typical of SOH and two of them showed remission after construction of new prostheses, thus confirming the initial diagnosis. SOH must be surgically removed and biopsied when it does not allow denture making, produces aesthetic and phonetic problems, causes chronic irritation, interrupts oral hygiene, and causes progressing of periodontal inflammatory disease.[19]

Table 1: Radiographic features of subpontic osseous hyperplasia

Increased radiopacity, dense[1,2,4,9,12,16,18]
Compact without a trabecular pattern, cortical, sclerotic[1,2,16]
Trabeculated[1,2,9]
Mottled radiopaque/radiolucent[6,12,17]
Smooth[1,2,4,9,18]
Nodular, lobulated, bilobed[1,2,9,12]
Dome shaped[12]
Saucer shaped[12]

Treatment options include surgical excision and bone recontouring and construction of a new FPD.[2,6,13,16,18] Follow-up is recommended because of possible recurrence.[16] In one case, two endosseous implant bodies were placed in as the patient had demonstrated a recurrence of SOH and no radiographic evidence of recurrence was noted 6 months after placement.[11]

In conclusion, clinicians should take care not to elucidate the possible causes of SOH such as functional stresses and chronic irritation by the prosthetic treatments and be aware of SOH does not usually require treatment or a biopsy.

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