self CDE 2 HOURS CREDIT instruction

Subpontic osseous hyperplasia: a case series and literature review

Connie A. Lee, DDS, MS ▪ Michael B. Lee, DDS, MS ▪ Chad R. Matthews, DMD, MS ▪ Dimitris N. Tatakis, DDS, PhD

A subpontic osseous hyperplasia (SOH) is a slow-growing, non-neoplastic bone growth that uniquely affects mandibular posterior edentulous ridges underneath pontics of fixed partial dentures. An SOH can result in significant periodontal and restorative complications, however, it is usually corrected by surgical excision. This report presents a series of SOH cases, illustrates SOH management approaches, and reviews the literature on SOH clinical presentations.

Received: August 25, 2012
Accepted: January 28, 2013

Only a limited number of conditions and deformities manifest themselves on edentulous ridges. In fact, the pertinent subcategory introduced in a classification scheme includes only 6 clinical entities: vertical and/or horizontal ridge deficiency, lack of gingiva/keratinized tissue, gingival/soft tissue enlargement, aberrant frenum/muscle position, decreased vestibular depth, and abnormal color.[1] Of these, only the first (ridge deficiency) can be considered specific to edentulous ridges. Another condition/deformity specific to edentulous ridges is the subpontic osseous hyperplasia (SOH). SOH, first described in 1971 by Calman et al, is a non-neoplastic growth of bone underneath pontics of fixed partial dentures (FPDs); therefore, SOH uniquely affects edentulous ridges bound by FPD abutment teeth.[2]

SOH is relatively uncommon, usually asymptomatic, and can occur from several months to many years after FPD insertion.[3] The lesion typically presents unilaterally, even in the presence of bilateral FPDs, and has a predilection for the mandible, particularly the first molar region.[4-6] Routinely, both the radiographic and histologic features of SOH are consistent with normal, compact, lamellar bone, similar to other exostoses.[2,5-7] The size and shape of an SOH lesion depends on its growth stage, the dimension of the edentulous space, and the position and shape of the inferior border of the associated pontic.[6] Various etiologic factors have been suggested, but the exact etiology of SOH remains unknown.[3,5,6]

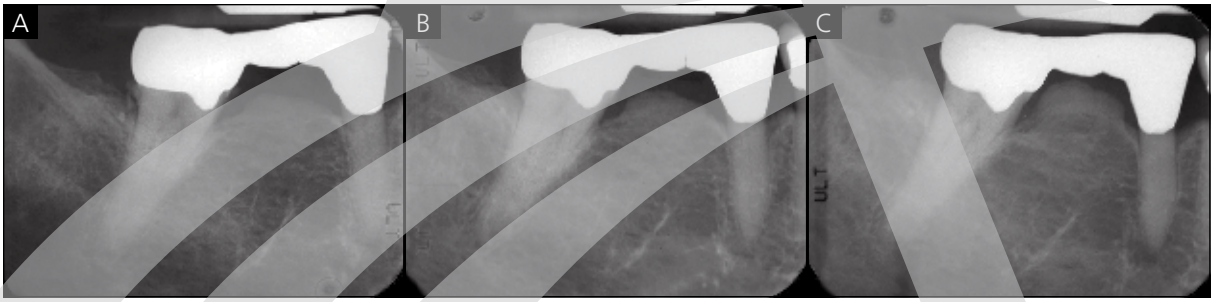

Fig. 1. *(Case No. 1)* Periapical radiographic images of the fixed partial denture (FPD). A. Eleven years prior to treatment. B. Six years prior to treatment. C. Fourteen months prior to treatment. Note the progressive increase in lesion dimensions, the obliteration of the space under the pontic, and the increasing proportion of the radiopaque, cortical bone-like, coronal aspect of the lesion.

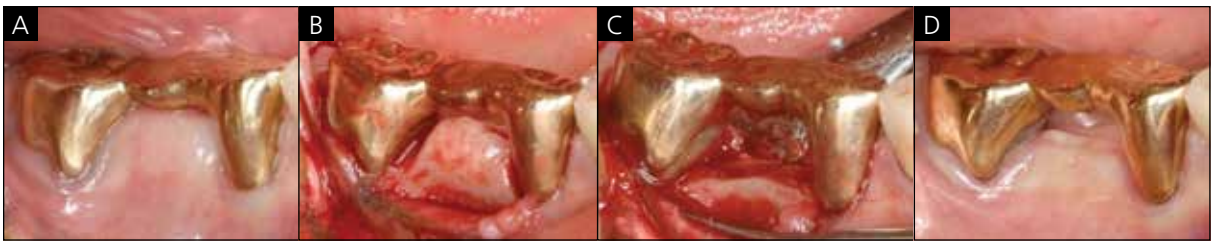

Fig. 2. *(Case No. 1)* Buccal views of the FPD. A. Preoperative. B. Following flap elevation with bony lesion exposed (note the correspondence with Fig. 1C.). C. Following resection. D. At 6 weeks postoperative.

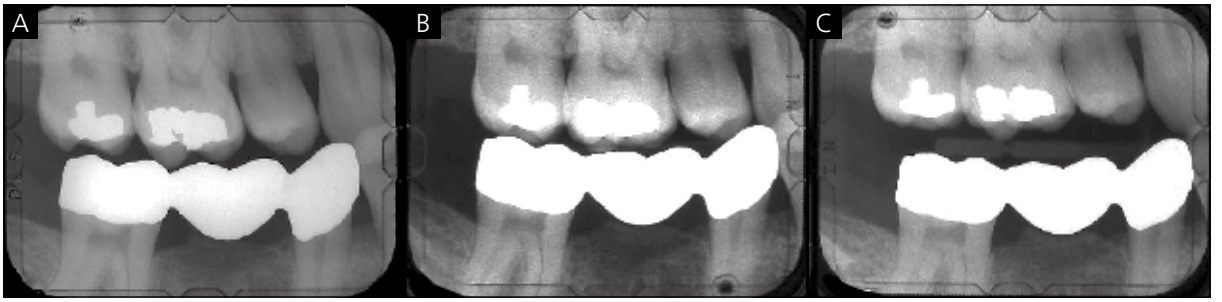

Fig. 3. *(Case No. 2)* Periapical radiographic images of the FPD. A. Seven years prior to initial visit. B. Four years prior to initial visit. C. At initial visit. Note the progressive increase in lesion dimensions and the increasing proportion of the radiopaque, cortical bone-like, coronal aspect of the lesion.

Although SOH lesions *per se* may go unnoticed by patients, increasing difficulty in oral hygiene practices under the FPD is often reported.[3,5,6] Few reported cases include patients that reported discomfort/sensitivity or pain in the area.[6,8-11] The growing size of an SOH may impinge on the FPD pontic(s), leading to loosening or dislodgement of the FPD.[3,5,6] Progressively increasing limitation of access to the FPD abutment teeth could result in caries development.[5] The impaired oral hygiene around SOH-associated teeth may also lead to localized gingival inflammation and periodontal attachment loss.[5,7,8] The periodontal and restorative complications of SOH constitute the main indications for treatment, which routinely consist of surgical excision.[5,6] The purpose of this article is to present a series of SOH cases, illustrate SOH treatment approaches, and review the literature on the clinical presentation of SOH.

Case No. 1

A 79-year-old woman presented to one of the authors with a medical history including hearing loss, frequent urination, chronic obstructive pulmonary disease, arthritis, stroke, and congestive heart failure. She was taking several medications, including atenolol, quinidine, captopril, coumadin, oxycodone, furosemide, and lovastatin. She reported a 45- to 50-year smoking history, quitting at age 69. She also reported receiving dental prophylaxis every 6 months. She denied any history of parafunctional habits or any oral pain.

Oral examination revealed the space under the bar-like pontic of the patient's full gold FPD (replacing tooth No. 30) was obliterated (Fig. 1 and 2). Radiographs obtained from the patient's general dentist indicated that the lesion—already present 11 years prior to presentation to the author—had grown substantially over the years. The lesion was clinically diagnosed as SOH. The radiographs also showed evidence of remaining extraction socket lining, suggesting a slow remodeling of the extraction socket cortical walls. Deep (6-8 mm) probing depths were present on teeth No. 29 and 31. There was also evidence of attachment loss in other localized areas. No tori were present. The patient received nonsurgical mechanical therapy, and maintenance care was recommended.

The patient presented again 14 months after the initial therapy and the probing depths around teeth No. 29 and 31 had increased by 1-2 mm. Surgical removal of the SOH was recommended. At the time of surgery, buccal and lingual full thickness flaps were elevated, the SOH was removed using rotary and hand instruments, flaps were closed with 4-0 silk sutures, and periodontal dressing was applied. Analgesic medication (acetaminophen) and antimicrobial rinse (chlorhexidine) were prescribed. Postoperative follow-ups at 1, 3, and 6 weeks revealed healing within normal limits. The patient did not return for any appointments beyond the 6-week postoperative visit, despite repeated invitations to do so.

Case No. 2

A 47-year-old man presented to the Graduate Periodontology clinic, The Ohio State University, for consultation regarding gingival recession on his maxillary central incisors. Following a clinical examination, and upon further discussion, the patient related increasing difficulty in performing oral hygiene procedures under an FPD that had replaced tooth No. 30. The space under the pontic had been obliterated. Previous radiographs were obtained from the patient's general dentist. Seventeen years prior to the patient's presentation to the authors, tooth No. 30 had been extracted due to caries and pulpal involvement. A 3-unit gold FPD was inserted, and 10 years later, during a visit to his general dentist, a subpontic lesion was noted. Radiographically, the extraction socket lining was still evident and the subpontic osseous mass appeared somewhat less radiopaque than the adjacent alveolar bone. Subsequent radiographs revealed that the lesion had increased in density and mesiodistal length under the pontic (Fig. 3). The lesion was clinically diagnosed as SOH. The patient, who did not have any tori, declined both a proposed removal of the lesion and a proposed treatment for his maxillary central incisors, and did not return to the clinic.

Case No. 3

A 64-year-old woman was referred to the Graduate Periodontology clinic due to localized increased pocketing (5-6 mm). She was systemically healthy and took multivitamin tablets daily. Upon examination, it was noted that the space under her mandibular left full gold bar-like pontic (replacing tooth No. 19) was obliterated; the patient was not able to perform proper hygiene measures in the area. Tooth No. 19 was extracted approximately 30 years prior to presentation. An FPD was inserted approximately 15 years after the

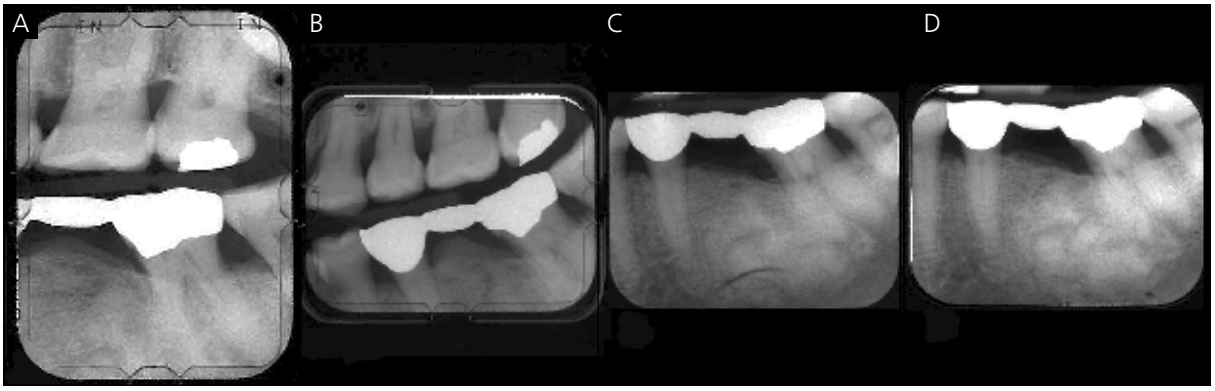

Fig. 4. *(Case No. 3)* Periapical radiographic images of the FPD. A. Three years prior to initial visit. B. At initial visit (note the increase in lesion dimensions and the increased proportion of the radiopaque, cortical bone-like, coronal aspect of the lesion, which corresponds with Fig. 5B). C. Nine months postoperative. D. Eighteen months postoperative, which corresponds with Fig. 5F (note the mixed radiolucent/opaque area of alveolar bone, consistent with focal cemento-osseous dysplasia).

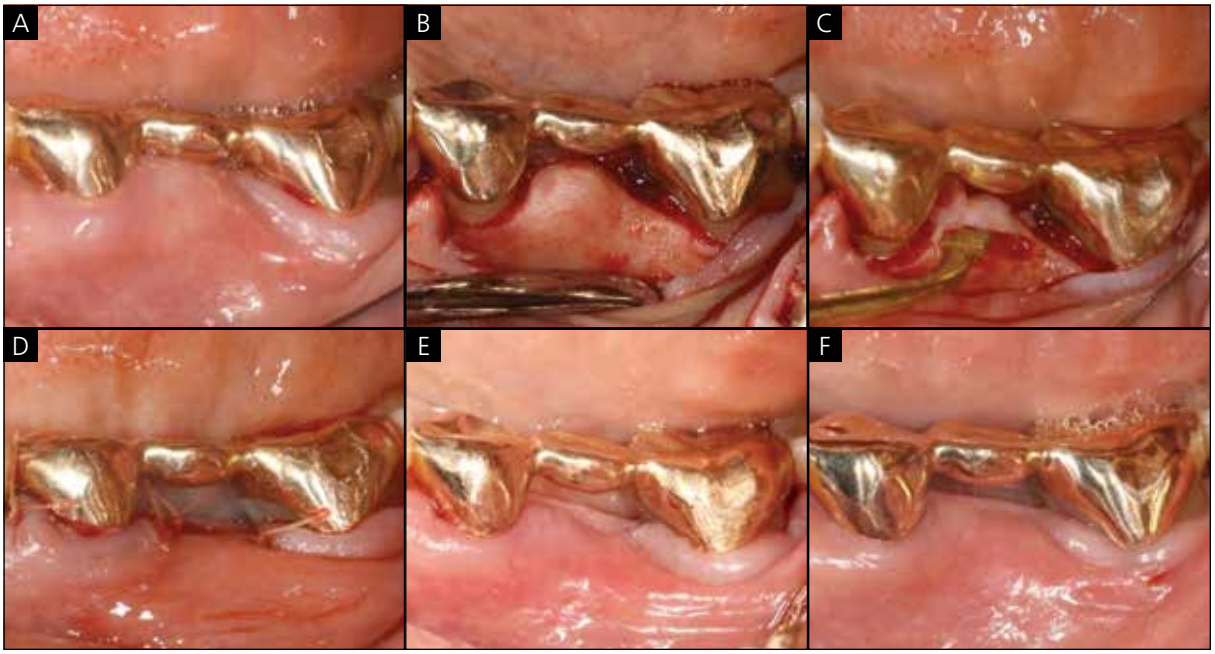

Fig. 5. *(Case No. 3)* Buccal views of the FPD. A. Preoperative. B. Following flap elevation with bony lesion exposed. C. Resection using piezosurgical tip. D. Flaps sutured. E. Nine days postoperative. F. Eighteen months postoperative.

extraction and replaced after 7 years for unknown reasons. Neither discomfort nor pain was reported by the patient. Gingiva was erythematous with some areas of bleeding on dental probing. Probing depths were ≤4 mm, except for tooth No. 17 (mesial, 6 mm) and tooth No. 18 (mesial, 5 mm). There was also a Grade II furcation involvement on tooth No. 18 (lingual). No mobility was noted. Wear facets on all teeth suggested bruxism. The patient's overall hygiene was good and she had a midpalatal torus.

The radiographically evident bony mass was hemispherical in shape and filled the entire subpontic space. Previous radiographs were retrieved, which indicated that the size of the bony mass—clinically diagnosed as SOH—had grown in size during the 3 years prior to presentation. The radiographic appearance of the central portion of the alveolar bone suggested the presence of a benign fibro-osseous lesion, most consistent with focal cemento-osseous dysplasia, which was apparently unchanged during the same period (Fig. 4).

The proposed treatment plan included surgical removal of the SOH, osseous surgery on tooth No. 17, and bone grafting (lingual furcation of tooth No. 18), while

Table. Summary of case reports of subpontic osseous hyperplasia (SOH).

Author	Case No.	Gender	Age (yrs)	Race	Affected area[a]	FPD units	Material	Design	U/Bi lesion	Time[b] (yrs)	Periodontal complications[c]	Tori or exostoses?
Calman et al 1971[2]	1	F	45	-	-	-	-	-	U	-	-	-
Stafne & Gibilisco 1975[12]	2	-	-	-	-	-	-	-	U	-	-	-
Strassler 1981[13]	3	M	58	C	19/30	3	-	Bar	Bi	14	-	-
Burkes et al 1985[3]	4	M	64	-	19/30	3	Gold	Bar	Bi	20	Y	Y
	5	M	-	-	19	3	Gold	Hygienic	U	<1	-	Y
	6	F	58	-	30	3	-	-	U	-	-	N
	7	F	46	-	30	3	Gold	-	U	several	-	Y
	8	F	42	-	20	-	-	Bar	U	9	-	N
	9	M	65	-	30	-	-	-	U	7	Y	N
	10	F	59	-	19/30	5/3	-	Saddle / Hygienic	Bi	-	-	Y
	11	F	49	-	20	3	-	-	U	several	-	Y
	12	M	68	-	19	3	-	Hygienic	U	-	-	Y
Render 1985[15]	13	-	-	-	19	3	Gold	Hygienic	U	-	-	-
Savage & Young 1987[4]	14	M	47	C	30	3	Gold	Hygienic	U	20	N	-
	15	F	60	C	19	-	-	-	U	-	-	-
Takeda et al 1988[7]	16	F	42	A	19/30	-	Silver	Bar	Bi	20	Y	N
Morton & Natkin 1990[6]	17	F	42	C	19/30	-	-	-	Bi	-	-	-
	18	M	79	C	L 2PM-2M	-	-	-	U	-	-	-
	19	M	54	C	R 2PM-1M	3	-	-	U	-	-	-
	20	F	56	C	30	-	-	Bar	U	-	-	-
	21	M	53	C	19	-	-	-	U	-	-	Y
	22	M	42	C	30	-	-	-	U	-	-	Y
	23	M	81	C	19	3	-	Hygienic	U	-	-	-
	24	F	29	A	30	-	-	Bar	U	-	-	-
	25	M	68	C	R 2PM-2M, L 1M-2M	4[c]	-	-	Bi	5	-	Y
	26	M	45	C	19	3	-	Bar	U	5	-	Y
	27	M	55	C	19	-	-	-	U	-	-	-
	28	M	46	C	30	-	-	-	U	-	-	-
	29	F	68	C	19	3	-	-	U	-	-	-
	30	F	51	C	R/L 2PM-1M	3	-	Bar	Bi	-	-	-
	31	F	53	C	L 1M-2M	3	-	Bar	U	-	-	-
	32	M	35	C	19	-	-	-	U	3	-	Y
Appleby 1991[16]	33	F	52	C	30	3	-	-	U	7	-	Y
Abramovitch 1993[18]	34	-	-	-	19	-	-	Hygienic	U	-	Y	-
Ruffin et al 1993[22]	35	F	67	C	19	3	Gold	Bar	U	35	Y	Y
Cataldo & Santis 1993[39]	36	F	55	-	2PM-1PM	4[c]	-	Hygienic	U	7	-	-
Mesaros & Evans 1994[23]	37	F	46	-	19	3	Gold	Hygienic	U	>20	Y	Y

Table continued. Summary of case reports of subpontic osseous hyperplasia (SOH).

Author	Case No.	Gender	Age (yrs)	Race	Affected area[a]	FPD units	Material	Design	U/Bi lesion	Time[b] (yrs)	Periodontal complications[c]	Tori or exostoses?
Cailleteau 1996[17]	38	M	64	A	30	3	Gold	-	U	40	Y	Y
	39	F	79	-	19/30	3/4[c]	-	-	Bi	-	-	-
Daniels 1997[5]	40	M	30	C	19	3	Gold	Hygienic	U	11	N	N
	41	F	38	C	19	3	Gold	Hygienic	U	13	Y	Y
	42	M	44	C	2PM-1M	3	Gold	Bar	U	24	-	N
	43	M	47	B	30	-	PFM	Mod ridge-lap	U	9	Y	Y
	44	M	73	C	30	3	PFM	-	U	10	-	N
Beaumont 1997[14]	45	-	61	-	19/30	-	-	-	Bi	>22	-	-
Bouquot & LaMarche 1999[40]	46	-	-	-	30	-	PFM	-	-	-	-	-
Lorenzana & Hallmon 2000[8]	47	F	56	-	19	3	Gold	Hygienic	U	25	Y	N
Frazier et al 2000[9]	48	M	65	C	3	3	-	-	U	10	Y	-
Ide et al 2003[20]	49	F	65	A	19	3	-	Hygienic	U	15	Y	-
Kessler & Phillips 2006[10]	50	M	-	-	19/30	3	-	-	Bi	3	Y	-
Islam et al 2010[21]	51	F	65	-	19	-	-	Hygienic	U	-	-	-
	52	M	78	-	30	-	-	-	U	-	Y	-
	53	F	80	-	19	3	Gold	Hygienic	U	-	Y	-
Kato et al 2010[11]	54	F	73	A	19	Cant	PFM	-	U	18	-	N
Present study	55	F	79	C	30	3	Gold	Bar	U	>10	Y	N
	56	M	47	C	30	3	Gold	Hygienic	U	17	Y	N
	57	F	64	A	19	3	Gold	Bar	U	8	Y	Y
Summary	57	52% F[d] 48% M[d]	Mean 56.6[e]	18% A[f] 3% B[f] 79% C[f]					18% Bi[g] 82% U[g]			62% Y[h] 38% N[h]

Abbreviations: -, indicates missing or unknown data; A, Asian; B, Black; C, Caucasian; F, female; M, male; FPD, fixed partial denture; Cant, cantilever; M, molar; PM premolar; PFM, porcelain-fused-to-metal; U, unilateral; Bi, bilateral;Y, yes; N, no.

[a]Time since FPD insertion

[b]Oral hygiene difficulty; increased probing depth and/or gingival inflammation around abutment teeth.

[c]FPD with 2 pontics and 2 abutments

[d]Based only on those studies who reported this information (n = 52)

[e]Based only on those studies who reported this information (n = 51)

[f]Based only on those studies who reported this information (n = 34)

[g]Based only on those studies who reported this information (n = 56)

[h]Based only on those studies who reported this information (n = 29)

retaining the existing FPD. At the time of surgery, full-thickness flaps were elevated, the area was debrided, and the teeth were root planed. A piezoelectric instrument was used to resect the SOH. The remaining alveolar ridge, which was clinically normal, was smoothed and recontoured parallel to the pontic. The removed SOH was ground with a bone mill and used as an autogenous particulate bone graft in the treatment of tooth No. 18. Primary flap closure was obtained with polyglycolic acid sutures. The patient was prescribed chlorhexidine gluconate oral rinse 0.12% BID, acetaminophen 500 mg (5-6 times daily, or as needed for pain), and amoxicillin 250 mg TID/7 days. Postoperative healing was uneventful (Fig. 5).

The patient was placed on regular maintenance recalls every 3 months. She reported much easier hygiene practice in the area. Six months postsurgery, all probing depths were ≤4 mm. Furcation involvement on tooth No. 18 was improved, but not eliminated. At 18 months posttreatment, a radiographic assessment suggested the possibility of SOH recurrence (Fig. 4).

Discussion and literature review

The present report documented a series of radiopaque lesions that occurred under FPD pontics. The lesions were clinically diagnosed as SOH, and all occurred in the mandibular first molar area. The management of this slow-growing, benign lesion is by surgical excision, using hand, rotary, or piezoelectric instruments. The bone excised during the treatment of this exostosis-like tissue growth can be a source of autogenous bone graft, as reported in the third case.

SOH, first described by Calman in 1971, has been reported in the literature under various names, including *osteoma, hyperostosis, plateauization, subpontic osseous proliferation, subpontic bony deposition, reactive subpontine exostosis, subpontic hyperostosis, pontic hyperostosis, hyperostosis alveolaris externa, and subpontic tissue enlargement.*[2-4,6-18]

The Table summarizes the limited number of reported SOH cases in chronological order. Among those cases that reported age and race, the average patient diagnosed with SOH was Caucasian (~80%) and 56.6 years of age. SOH manifests equally in both genders, mostly as a unilateral lesion (82% of the cases in the Table that reported on lesions) and overwhelmingly in the mandibular first molar area, under an FPD that is typically 3 units with a bar/hygienic pontic design, and made of gold. Although on rare occasions, SOH has been reported as early as within the first year of FPD placement, the majority of cases have an FPD history >7 years.[3]

Many reports, the present one included, have noted that the radiographic appearance of SOH is more radiopaque than the underlying ridge, with a tendency for the radiopacity to increase with time. Others have noted a mixed radiopaque/radiolucent appearance, while some have reported a thin radiolucent line separating the ridge from the growth.[3,4,6-8,18-21]

The typical approach to SOH management has been to excise the bony growth, as was performed in this report.[3,5,7-9,16] Recurrence after excision is rare, as is spontaneous regression.[3,5,16] As part of SOH management, some authors have suggested removal of the FPD and replacement of the missing teeth with implants.[3,5,8,9,16]

The etiology of OH has not been definitively established. The most commonly suggested etiologic factors have been functional/occlusal stress, chronic localized inflammation/tissue irritation, or a combination thereof.[2-7,11,12,20,21] The possible role of genetics has also been suggested.[3,6,11,17,21] Soft tissue impingement, a functionally created vacuum under the pontic, trauma from oral hygiene practices under the pontic, generation of electric currents, and muscle insertion or hyperactivity have also been suggested as etiologic factors.[6,8,11,15,17,22,23]

Among the potential etiologic/contributing factors mentioned above, functional stress and genetics appear to be the ones supported by suggestive findings.[24-26] The shape and dimensions of the mandible change during opening and closing, possibly resulting in additional functional stresses in the FPD area, thus becoming the trigger for bone growth.[24,25] Ralph & Caputo showed that stress patterns become concentrated on the cortical plates when vertical loads are applied in the mandible.[26] These facts, along with the differences between maxilla and mandible with respect to cortical bone content, cortical plate thickness, and shape changes during mouth movement could explain the almost exclusive presentation of SOH in the mandible. Most SOH cases occur under bar-like or hygienic pontics; it is possible that such pontic designs allow for higher bending stress distribution on the edentulous ridge, due to connectors that are thinner in comparison to other pontics. To date, SOH has not been reported under short-span FPDs supported by implants placed in the posterior mandible. However, bone growth in the molar region of the mandible has been reported under the cantilever portion of fixed full arch prostheses supported by interforaminal implants.[27,28] This coincident bone growth suggests that the molar region of the mandible may be particularly susceptible to reactive bone formation in response to functional stresses, regardless of whether the stress originates from tooth-supported posterior FPDs, as in the case of SOH, or implant-supported fixed full arch prostheses.

Of the cases in the Table that reported on tori/exotosis incidence, 62% reported tori/extosis and 38% did not. In subjects >20 years of age, the reported clinical prevalence of tori from various large-scale studies ranged from <10% to 15%-25%, and as high as 40% occasionally in select populations.[19,29-33] Therefore, there appears to be a strong association between SOH and tori/exostoses, as previously suggested.[3] One of the 3 cases presented in this report was associated with a palatal torus. In this context, it should be noted that presence of tori has been associated with other oral exostoses and a greater height of interdental alveolar bone, regardless of the presence or absence of occlusal stress (parafunctional habits).[34-36] It is possible that SOH shares the same genetic predisposing factors implicated in tori development.[37,38] The strong association between SOH and tori/exostoses is also reflected in the similar complications they present under FPDs, such as hyperostosis and chronic pain.[39,40]

In summary, SOH is a slow-growing, benign, osseous lesion distinctly associated with mandibular posterior edentulous ridges bounded by FPD abutment teeth. SOH can result in significant periodontal and restorative complications, therefore patients presenting with SOH should be appropriately and promptly managed.

Author information

Dr. C. Lee is in private practice in Maple Grove, Minnesota. Dr. M. Lee is in private practice in Redlands, California. Dr. Matthews is in private practice in Rock Hill, South Carolina. Drs. C. Lee and Matthews are former residents, Division of Periodontology, College of Dentistry, The Ohio State University, Columbus, where Dr. Tatakis is a professor and program director.

References

1. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol.* 1999;4(1):1-6.
2. Calman HI, Eisenberg M, Grodjesk JE, Szerlip L. Shades of white. Interpretation of radiopacities. *Dent Radiogr Photogr.* 1971;44(1):3-10.
3. Burkes EJ Jr., Marbry DL, Brooks RE. Subpontic osseous proliferation. *J Prosthet Dent.* 1985;53(6):780-785.
4. Savage NW, Young WG. Reactive subpontine exostoses. *Oral Surg Oral Med Oral Pathol.* 1987;63(4):498-499.
5. Daniels WC. Subpontic osseous hyperplasia: a five-patient report. *J Prosthodont.* 1997;6(2):137-143.
6. Morton TH, Jr., Natkin E. Hyperostosis and fixed partial denture pontics: report of 16 patients and review of literature. *J Prosthet Dent.* 1990;64(5):539-547.
7. Takeda Y, Itagaki M, Ishibashi K. Bilateral subpontic osseous hyperplasia. A case report. *J Periodontol.* 1988;59(5):311-314.

8. Lorenzana ER, Hallmon WW. Subpontic osseous hyperplasia: a case report. *Quintessence Int.* 2000;31(1):57-61.
9. Frazier KB, Baker PS, Abdelsayed R, Potter B. A case report of subpontic osseous hyperplasia in the maxillary arch. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2000;89(1):73-76.
10. Kessler HP, Phillips D. Oral and maxillofacial pathology case of the month. Bilateral supbontic osseous hyperplasia. *Tex Dent J.* 2006;123(12):1153, 1156-1159.
11. Kato S, Kato M, Hanamoto H. Subpontic tissue enlargement of the mandible following cross-arch fixed partial denture reconstruction: an 18-year follow-up. *Int J Prosthodont.* 2010;23(3):243-245.
12. Stafne EC, Gibilisco JA. *Oral Roentgenographic Diagnosis.* 4th ed. Philadelphia: W.B. Saunders; 1975.
13. Strassler HE. Bilateral plateauitization. *Oral Surg Oral Med Oral Pathol.* 1981;52(2):222.
14. Beaumont RH. Subpontic osseous proliferation over a period of 22 years: a case report. *Northwest Dent.* 1997;76(6):34-35.
15. Render PJ. Bony deposition under a fixed partial denture. *J Prosthet Dent.* 1985;54(4):524-525.
16. Appleby DC. Investigating incidental remission of subpontic hyperostosis. *J Am Dent Assoc.* 1991;122(12):61-62.
17. Cailleteau JG. Subpontic hyperostosis. *J Endod.* 1996;22(3):147-149.
18. Abramovitch K. Roentgen ray anomalies [pontic hyperostosis]. *J Gt Houst Dent Soc.* 1993;64(7):4.
19. Bouquot JE, Gundlach KK. Oral exophytic lesions in 23,616 white Americans over 35 years of age. *Oral Surg Oral Med Oral Pathol.* 1986;62(3):284-291.
20. Ide F, Horie N, Shimoyama T. Subpontic cartilagenous hyperplasia of the mandible. *Oral Dis.* 2003;9(4):224-225.
21. Islam MN, Cohen DM, Waite MT, Bhattacharyya I. Three cases of subpontic osseous hyperplasia of the mandible: a report. *Quintessence Int.* 2010;41(4):299-302.
22. Ruffin SA, Waldrop TC, Aufdemorte TB. Diagnosis and treatment of subpontic osseous hyperplasia. Report of a case. *Oral Surg Oral Med Oral Pathol.* 1993;76(1):68-72.
23. Mesaros AJ Jr., Evans DB. Subpontic osseous hyperplasia. *Gen Dent.* 1994;42(3):264-266.
24. McDowell JA, Regli CP. A quantitative analysis of the decrease in width of the mandibular arch during forced movements of the mandible. *J Dent Res.* 1961;40(6):1183-1185.
25. Regli CP, Kelly EK. The phenomenon of decreased mandibular arch width in opening movements. *J Prosthet Dent.* 1967;17(1):49-53.
26. Ralph JP, Caputo AA. Analysis of stress patterns in the human mandible. *J Dent Res.* 1975;54(4):814-821.
27. Taylor TD. Osteogenesis of the mandible associated with implant reconstruction: a patient report. *Int J Oral Maxillofac Implants.* 1989;4(3):227-231.
28. Nakai H, Niimi A, Ueda M. Osseous proliferation of the mandible after placement of endosseous implants. *Int J Oral Maxillofac Implants.* 2000;15(3):419-424.
29. Haugen LK. Palatine and mandibular tori. A morphologic study in the current Norwegian population. *Acta Odontol Scand.* 1992;50(2):65-77.
30. Yaacob H, Tirmzi H, Ismail K. The prevalence of oral tori in Malaysians. *J Oral Med.* 1983;38(1):40-42.
31. Gorsky M, Raviv M, Kfir E, Moskona D. Prevalence of *torus palatinus* in a population of young and adult Israelis. *Arch Oral Biol.* 1996;41(6):623-625.
32. Darwazeh AM, Pillai K. Oral lesions in a Jordanian population. *Int Dent J.* 1998;48(2):84-88.
33. Eggen S, Natvig B, Gasemyr J. Variation in *torus palatinus* prevalence in Norway. *Scand J Dent Res.* 1994;102(1):54-59.
34. Jainkittivong A, Langlais RP. Buccal and palatal exostoses: prevalence and concurrence with tori. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2000;90(1):48-53.
35. Sawair FA, Shayyab MH, Al-Rababah MA, Saku T. Prevalence and clinical characteristics of tori and jaw exostoses in a teaching hospital in Jordan. *Saudi Med J.* 2009;30(12):1557-1562.
36. Eggen S. Correlated characteristics of the jaws: association between *torus mandibularis* and marginal alveolar bone height. *Acta Odontol Scand.* 1992;50(1):1-6.
37. Eggen S. *Torus mandibularis*: an estimation of the degree of genetic determination. *Acta Odontol Scand.* 1989;47(6):409-415.
38. Gorsky M, Bukai A, Shohat M. Genetic influence on the prevalence of *torus palatinus. Am J Med Genet.* 1998;75(2):138-140.
39. Cataldo E, Santis HS. A clinico-pathologic presentation. Hyperostosis. *J Mass Dent Soc.* 1993;42(1):5, 50.
40. Bouquot JE, LaMarche MG. Ischemic osteonecrosis under fixed partial denture pontics: radiographic and microscopic features in 38 patients with chronic pain. *J Prosthet Dent.* 1999;81(2):148-158.