Subpontic Tissue Enlargement of the Mandible Following Cross-Arch Fixed Partial Denture Reconstruction: An 18-Year Follow-up

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This study aimed to investigate the occurrence of subpontic tissue enlargement (STE) beneath a mandibular fixed partial denture. A 55-year-old Japanese woman received periodontal therapy and cross-arch fixed partial dentures were placed in the maxilla and mandible. After 18 years, STE developed in the left posterior region of the mandible. It was presumed that biomechanical loading in the mandible, along with other factors, might have caused the STE in this particular patient. *Int J Prosthodont 2010;23:243–245.*

Subpontic tissue enlargement (STE), the cause of which is still unclear, is known by many names: osteoma,[1] subpontic hyperostosis,[2–5] plateauization,[6] subpontic osseous proliferation,[7] subpontic bony deposition,[8] reactive subpontine exostoses,[9] and subpontic osseous hyperplasia.[10–14] The characteristic feature of STE is slow and spontaneous bone growth, which is in most cases found in the posterior region of the mandible under the pontic of a fixed partial denture (FPD).

The present report illustrates a case of STE underneath a mandibular cross-arch FPD that had been supported by teeth with reduced periodontal tissue for 18 years.

Case History

In 1991, a 55-year-old Japanese woman sought periodontal therapy at Kato Dental Clinic, Implant Center, Osaka, Japan. She was cooperative and in excellent general health. She had no mandibular tori or palatal torus. Many hopeless teeth had to be extracted due to advanced periodontal disease. However, a few of the maxillary and mandibular teeth were maintained by cross-arch stabilization, as presented in Table 1. After periodontal and endodontic treatment, metal-ceramic cross-arch FPDs were placed in both arches according to routine clinical procedures (Fig 1). As part of the maintenance therapy, professional tooth cleaning and dental care were performed once every 3 to 6 months for 18 years. In 2003, the mandibular right first molar was extracted due to root fracture. Consequently, the mandibular FPD was modified to a cantilever extension on the right side with two pontics from the mandibular right canine (Fig 1b, Table 1).

In 2004, periapical radiographs of the tooth abutments were taken. Changes in the alveolar bone underneath the FPD pontic and around the abutment teeth were noted (Fig 1). At that point, the patient had no complaints regarding the subpontic mucosa. In 2009, she began to feel the pontic impinging on the subpontic mucosa and pain at the mandibular left first premolar and first molar sites. The periapical radiograph taken at that time revealed STE, which impinged on the FPD pontic (Fig 2). The radiograph revealed that spontaneous bone proliferation had developed under the pontic over the course of 18 years.

Soft tissue growth around the abutment teeth was also observed proportional to the bone proliferation over 18 years (Fig 3).

Treatment

Surgical excision and removal of the FPDs or a combination of these modalities have been reported as treatment options for STE.[4,7] When surgical excision is planned, the possibility of STE recurrence should be considered.[7,12] In this case, it was practically impossible to remove the cross-arch FPD.

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Table 1 Course of Cross-Arch FPD Reconstruction

Year	Remarks	Tooth abutments*	
		Maxilla	Mandible
1991	Bonding of cross-arch FPDs	17, 13, 12, 21, 23, 25	35, 33, 31, 43, 46
2003	Extraction of tooth 46*	17, 13, 12, 21, 23, 25	35, 33, 31, 43
2004	No symptoms of STE clinically and radiographically	17, 13, 12, 21, 23, 25	35 ,33 ,31, 43
2009	Development of mandibular STE	17, 13, 12, 21, 23, 25	35, 33, 31, 43

STE = subpontic tissue enlargement.
*FDI tooth-numbering system.

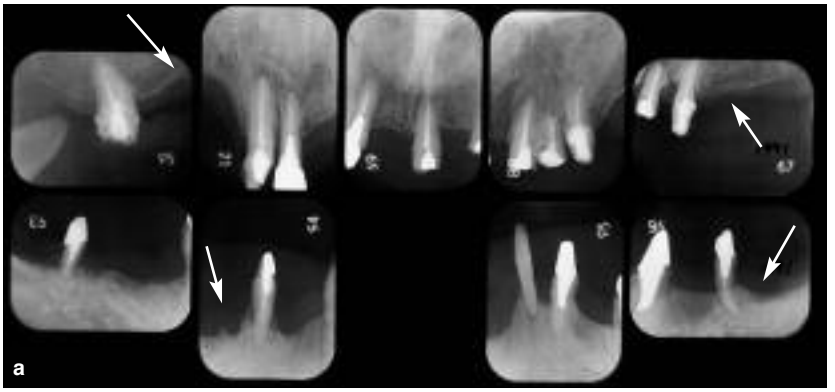


Fig 1 Periapical radiographs taken in **(a)** 1991 and **(b)** 2004. After the FPDs were placed, continuous socket remodeling was observed *(arrows)*. There was no pontic impingement on the subpontic mucosa at the mandiublar left first premolar and first molar at this time.

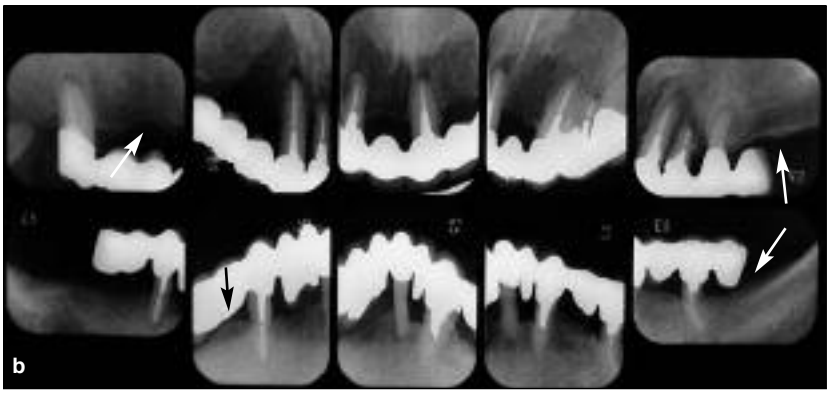
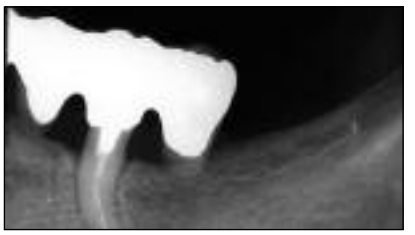


Fig 2 *(below)* Periapical radiograph of the mandibular left second premolar taken in 2009. The spontaneous growth of the subpontic alveolar bone over the course of 18 years resulted in impingement of the subpontic alveolar mucosa.



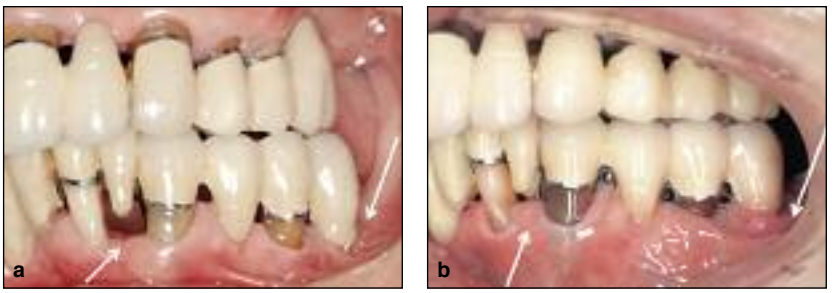


Fig 3 *(left)* Lateral view of the patient in **(a)** 1992 and **(b)** 2009. Healthy periodontal tissues were maintained for 18 years and spontaneous soft tissue growth was also observed *(arrows)*.

A nonsteroidal anti-inflammatory pain medication was prescribed as a symptomatic treatment for the pain and professional cleaning of the subpontic mucosa was performed. The authors regularly reviewed the patient without surgical intervention because the patient's discomfort had subsided after treatment. If the tissue had shown further enlargement, reduction of the bottom of the pontic or surgical excision might have been required.

Discussion

The clinical and radiographic presentation, combined with a complete dental history, is sufficient to diagnose most cases of STE.[14] As with torus mandibulari or osteoma, STE might simulate other radiopaque lesions of the jaw such as osteitis deformans (Paget disease), osteomyelitis, fibrous dysplasia, and Gardner syndrome.

Although the location in the mandible and the presence of an FPD encourages a diagnosis of STE, an excisional biopsy specimen should be submitted for histologic evaluation to make a definitive diagnosis.[2,14]

Based on the literature on STE made available from 1971 to 2009, 43 cases, including the present one, have been reported.[1–13] Very little information is available on the incidence or prevalence of STE. In the authors' clinic, they have encountered a total of three cases of STE in their 20 years of experience. The other two cases were related to osseointegrated implants. The present case report was part of a study on FPDs conducted by general practitioners at a private dental clinic from 1991 to 2009. A total of 446 patients received a tooth-supported FPD with more than two units and 267 patients were treated with an implant-supported FPD having more than two units. Since all of these patients could not be reviewed regularly, there are no data regarding the prevalence of STE in that clinic.

STE is most common in the posterior mandible. However, one case has been reported in the maxillary molar region.[13] The FPDs associated with STE were of three or four units in most cases, except that of the present report. This reflects the relative frequency with which smaller FPDs are fabricated compared to the number of patients treated with full-arch reconstructions.

Although the etiologies of STE, such as biomechanical stress, chronic irritation, genetic predisposition, and any combination of these factors, have been discussed, the main cause is still unclear.[13,14] Wasson et al[14] reported that the etiology of STE could be multifactorial and genetic predisposition is precipitated by unique stress patterns or other stimuli. Spontaneous growth of the alveolar bone in the posterior mandible after implant insertion has also been reported.[15–19] Five (19%) of 27 patients saw alveolar bone growth during an observation period of 6 to 66 months.[15] In another case report, vertical bone growth of 2.5 to 3 mm was observed over 32 months after an implant-supported FPD was placed.[16] In these reports, the probable cause of bone proliferation could be a physiologic adaptation to the increased mechanical load after implant-supported FPD insertion.[15-19] Although there are differences in the supporting mode, such as osseointegration or periodontal tissue, the radiographic observations in the present case revealed socket remodeling beneath the prosthesis over 10 years (Fig 1). The possibility of developing STE with gradual growth might have already existed in 2004. The spontaneous bone growth that eventually reached the underside of the pontic and continued until 2009 may confirm the diagnosis of STE in the present case.

Conclusion

The authors believe that the cause of STE in the present case was not only biomechanical loading to the mandible, but also included other factors such as chronic irritation and genetic predisposition.

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