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CASE STUDY

Multiple external root resorptions in a patient with Stage IV, Grade C periodontitis and autoimmune diseases: A case report

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Abstract

Background: Pathological root resorption affects permanent teeth and is usually triggered by infectious-inflammatory stimuli and/or dental trauma. Periodontitis and traumatic occlusion have been reported as possible causative factors of root resorptions, whilst the impact of systemic diseases is less well understood. This case highlights the need for consideration of multiple risk factors, especially when presenting in combination.

Methods and Results: A 62-year-old South Asian female presented with unstable Stage IV Grade C periodontitis, poor oral hygiene and multiple autoimmune conditions including oral lichen planus. Clinical and radiographic examination revealed multiple advanced apical and external root resorptions of the patient's molars associated with periapical bone loss, despite of a minimally restored dental status.

Conclusion: A likely etiology of this patient's multiple root resorptions is the presence of unstable periodontitis with periodontal-endodontic lesions, exacerbated by a dysbalanced immune response to infectious agents. Appropriate monitoring and managing of such patients can prevent or limit the pathological process of inflammatory root resorption.

KEYWORDS
periodontitis, root resorption, molar, autoimmune diseases, traumatic dental occlusion, risk factors

Key points

Why is this case new information?

- This is the first report documenting advanced multiple external inflammatory root resorptions in a periodontitis patient with oral and systemic co-morbidities.

What are the keys to successful management of this case?

- Early diagnosis, prevention and intervention to limit periodontal inflammation, endodontic infection and occlusal trauma.

What are the primary limitations to success in this case?

- Late diagnosis of multiple root resorptions, palliative periodontal treatment due to poor oral hygiene compliance, and poorly controlled systemic inflammation favoring the persistence of a dysregulated immune response to the oral microbiota.

INTRODUCTION

Root resorption is defined as the loss of dentine and cementum through the continued action of osteoclastic cells upon prolonged exposure to mechanical or infectious stimuli, or as a result of root surface damage.[1] If left untreated, this condition often progresses, leading to tooth destruction and loss.

TABLE 1 Patient's medical history and medication intake. IU: International units

Disorder	Medication	Daily dose
Hypertension	Felodipine	2.5 mg
Type II diabetes mellitus	Metformin	500 mg
Sjögren syndrome	Hydroxychloroquine	200 mg
Oral lichen planus	Difflam oral rinse 0.15%	As needed
	Flixonase nasule drops	400 mcg
	Corsodyl mouthwash 0.2%	Once
Lichen Planus	Clobetasol 17-propionate cream 0.05%	Once
Rheumatoid arthritis	Betamethasone valerate cream 0.1%	Once
Asthma	Salbutamol tablets	12 mg
Vitamin D deficiency	Fultium-D3	800 IU
Vitamin B12 deficiency	Hydroxocobalamin	1 mg
Keratoconjunctivitis sicca	Hypromellose eye drops 0.3%	As needed

External root resorption (ERR) is the physiological process leading to active remodeling of cementum and dentine in the primary dentition, but also exists in the permanent dentition in an equilibrium of resorption and cementum deposition, allowing for repair.[1] Pathologic ERR occurs in distinct types with different etiologies and locations. The main types of ERR are inflammatory, replacement/ankylosis, surface, cervical resorption and transient apical breakdown.[1]

Although cases of multiple root resorption have been reported, none of these are similar to the unusual case presented herein. Our literature search was conducted on PubMed and included the following search terms and Boolean operators: 'multiple AND external AND (root AND resorption) AND molar NOT orthod* NOT cervical', 'periodontitis AND (root AND resorption) NOT orthod* NOT cervical'.

As root resorption may be self-limiting in early stages,[2] it is important to recognize and manage risk factors and possible causes of ERR such as occlusal trauma, periodontal, pulpal and periapical infection, as well as systemic inflammation.[3,4]

MATERIALS AND METHODS

Clinical presentation

A 62-year old South Asian female patient presented in 2021 on the undergraduate periodontology teaching clinic at Birmingham Dental School and Hospital, UK, with severe periodontal pocketing. Furthermore, this patient had orally and extraorally manifested autoimmune diseases, namely oral lichen planus (OLP), Sjögren's syndrome and rheumatoid arthritis, along with type II diabetes mellitus, hypertension and asthma, all of which were medication-controlled (Table 1). She had poor oral hygiene owing

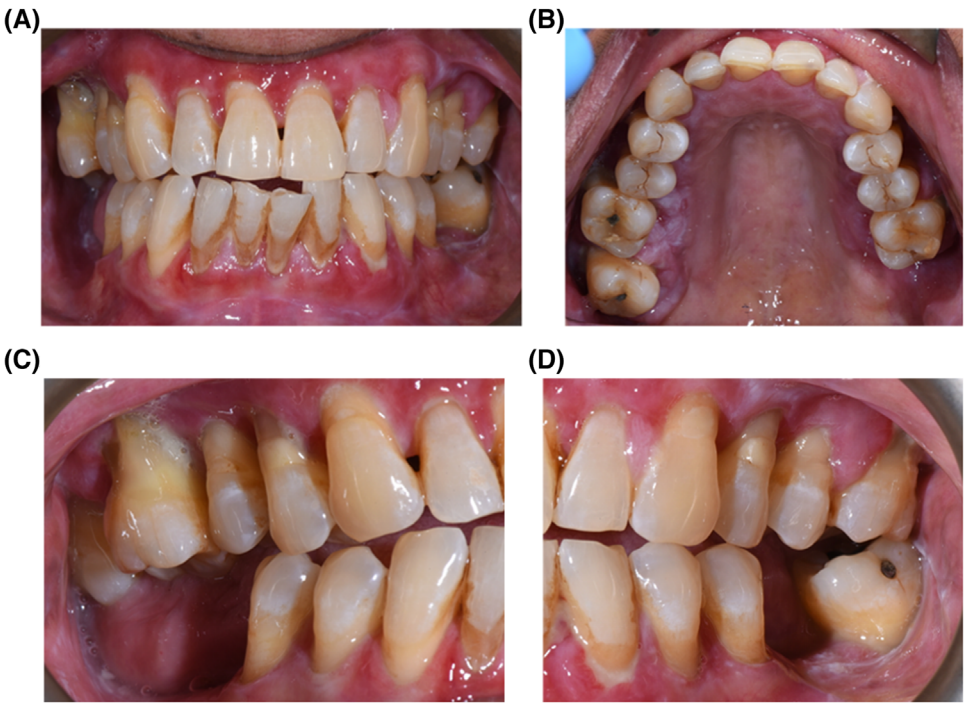

FIGURE 1 Intraoral presentation after extraction of 31. (A) Anterior view with generalized gingival recessions, inflammation, multiple suppurating sites, plaque and calculus. Wickham's striae typical of oral lichen planus are also visible on the oral mucosae. (B) Occlusal view with inflammation and marked swelling of the palatal mucosa adjacent to the molars. (C) Right side view and (D) left side view

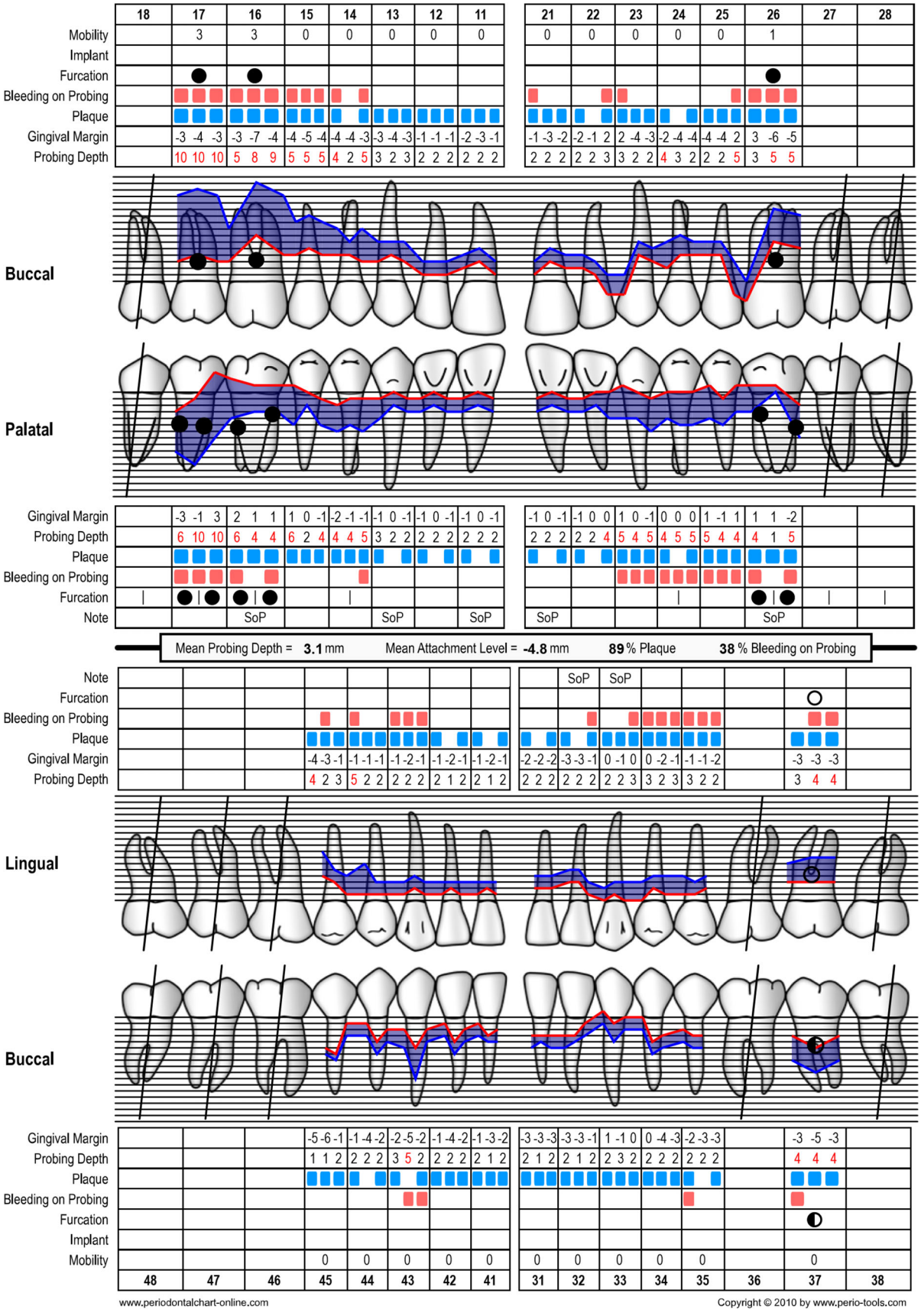

FIGURE 2 6-point pocket chart (recorded after extraction of 31) with BoP, SoP, plaque score, gingival recessions, mobilities, furcation involvement (black circle: Grade III, half-circle: Grade II, white circle: Grade I). Chart created with an online periodontal charting tool (perio-tools.com, School of Dental Medicine of the University of Bern) perio-tools.com, School of Dental Medicine of the University of Bern

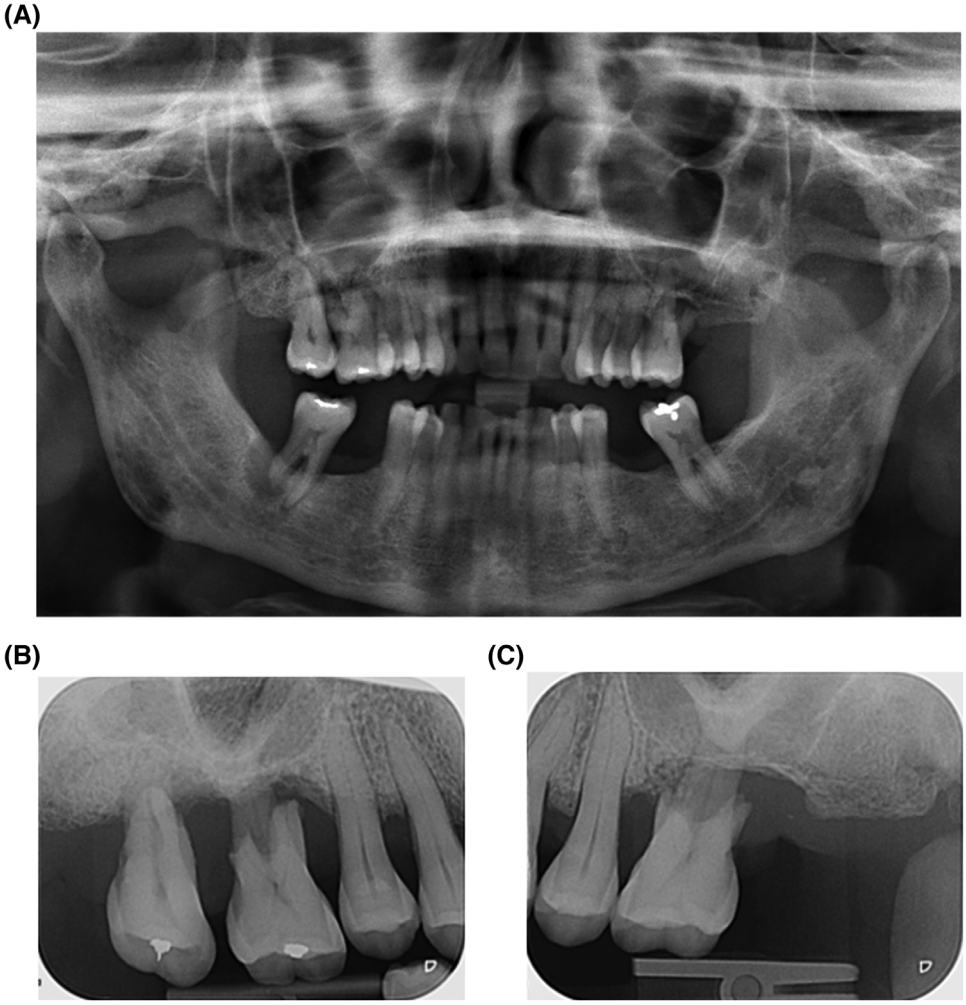

FIGURE 3 Dental panoramic tomogram (DPT) and long cone periapical radiographs of the upper posterior teeth. (A) DPT of the patient's dentition revealing generalized horizontal bone loss and an apical radiolucency associated with 31, which was then extracted. (B) 2, 3, and (C) 14, all displaying more severe resorption of the buccal roots and Grade III furcation involvement. Crescentic bone loss, which is characteristic in secondary occlusal trauma is seen on 3 and 14, along with generalized horizontal bone loss ranging from 40% to 75%

to pain and discomfort as a result of OLP and associated desquamative gingivitis. This patient had undergone multiple courses of periodontal treatment.

Temporomandibular joint dysfunction (TMD) with bone remodeling had been diagnosed in 2015. Furthermore, this patient had suffered from left side chronic generalized sinusitis in 2015, which was attributed to periapical and periodontal pathology of her upper left molars. On computed tomography scans taken in 2015, early signs of root resorption could be observed. The patient had the upper left second molar (15) removed subsequently, but disagreed to having further teeth extracted. She had also been a patient at the Oral Medicine Department at Birmingham Dental Hospital, for treatment of OLP.

On examination, this patient had a minimally restored dentition with several missing molars. The reasons for previous lower first molar extractions were not known. Lone-standing lower second molars were in occlusion with the upper molars, leading to secondary occlusal trauma. High generalized plaque levels and gingival recessions as well as calculus were present, along with generalized OLP-related desquamation. An erythematous swelling associated with the buccal mucosa of the upper left first molar (14) was noted, indicative of a lateral periodontal abscess (Figure 1).

Grade III mobile upper right first and second molars (2, 3), and a Grade I mobile 14 were noted, using Miller's mobility classification.[5] A six-point pocket chart revealed generalized severe probing depths with bleeding on probing (BoP) and suppuration on probing (SoP), as well as furcation involvement of all molars (Figure 2). Relevant radiographs (Figure 3) and special tests were undertaken. Radiographically, apical and external root resorptions of the upper molars and the lower right second molar (31) were identified. The following diagnoses were made:

1. Generalized periodontitis, Stage IV, Grade C, currently unstable, risk factors: stress, poor oral hygiene, type II diabetes, rheumatoid arthritis.[6,7]
2. Upper molars and 31 with hopeless prognosis[8] due to advanced attachment loss, mobilities, pulp necroses, periodontal-endodontic lesions, and external

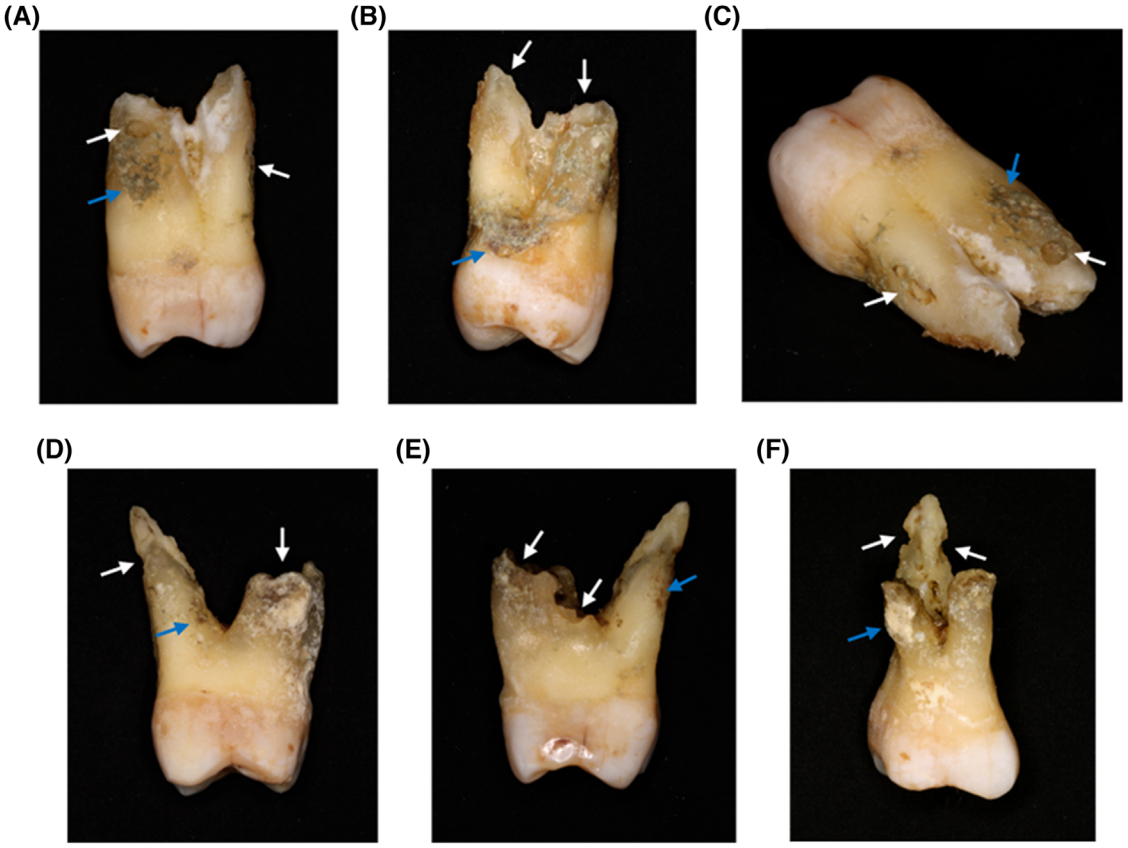

FIGURE 4 Extracted upper right (upper panel) and upper left (lower panel) first molars. Apical and external resorption lesions are seen (white arrows), along with subgingival calculus (blue arrows). (A) Mesial view, (B) distal view, (C) palatal and mesiobuccal roots with external resorption lesions. (D) Mesial view, (E) distal view, (F) buccal view

inflammatory resorptions [4] predominantly in the apical and mid thirds of the roots, accompanied by asymptomatic apical periodontitis[9] in the case of 31.

Case management

The patient consented to undergo extractions of her upper molars and 31 in a staged approach (Figure 4). Subsequently, she continued to undergo periodontal treatment with oral hygiene re-instructions and re-motivation.

RESULTS

This patient's periodontal condition was improved, with pocket depths not exceeding 5 mm and reduced BoP. However, periodontitis was not fully stabilized due to the patient's inability to achieve a lower plaque score, and owed to her high systemic inflammatory burden. Therefore, and due to a remaining shortened dental arch on the right side, this patient did not undergo tooth replacement[10] and enrolled in palliative periodontal care,[11] whilst also continuing treatment for OLP.

DISCUSSION

The patient presented herein suffered from three autoimmune conditions and type II diabetes. These conditions have a local and systemic dysregulated inflammatory host response in common, during which several inflammatory cytokines are released.[6] Resorption of mineralized tissues is executed by osteoclasts and regulated through a complex cell-to-cell signaling system, which is triggered by inflammatory mediators and cytokines, but also by lipopolysaccharides of periodontal pathogens, leading to increased osteoclast activity.[12]

Although periodontitis and periodontal treatment have been suggested as risk factors for external cervical resorption,[13] a clear correlation with external inflammatory resorption has not been described in the literature. One of the well-studied risk factors for ERR is apical periodontitis.[14] This patient presented with periapical lesions without having exhibited carious lesions or extensive restorations, making a diagnosis of multiple periodontal-endodontic lesions plausible. These may have led to pulpal and periapical pathology, resulting in ERR. ERR lesions themselves act as plaque-retentive factors when associated with a periodontal pocket and are sites of exudation of serum

proteins serving as substrates for periodontitis-promoting bacteria, thus exacerbating inflammation in these areas.

Teeth affected by periodontitis were found to have an increased incidence of root resorption when in direct contact with an opposing tooth, and occlusal trauma also exacerbates periodontal attachment loss.[15] The more severe resorption of the buccal roots seen in this patient may be explained by more occlusal loading of the buccal cusps as well as by more attachment loss on these roots compared to the palatal roots.

CONCLUSION

In this patient, the combination of periodontitis with periodontal-endodontic lesions, traumatic occlusion and systemic inflammation may have shifted the equilibrium between resorption and cementum deposition to favor ERR, whilst the ERR itself might have further advanced the existing periodontitis, leading to a vicious cycle of tissue inflammation and destruction. However, these possible etiological factors are of a hypothetical nature and need to be corroborated in well-designed observational studies, which are currently scarce. In this case, an earlier diagnosis, intervention such as endodontic treatment, and preventive measures such as fabrication of a Michigan splint may have helped to retain this patient's molars.

AUTHOR CONTRIBUTIONS

All authors have made substantial contributions to the data collection and writing of this case report. BST and HI carried out and JH oversaw patient examinations and treatments. BST, HI, and JH drafted, revised, and approved the manuscript.

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