OLGU SUNUMU CASE REPORT

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Co-Incidence of Excessive Deep-Bite and Pulpal Necrosis in Mandibular Incisors: Case Series

Aşırı Derin Kapanış ve Alt Kesici Dişlerde Pulpa Nekrozu Birlikteliği: Olgu Serisi

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ABSTRACT In this article, a unique disease pattern in 9 patients involving the simultaneous presence of excessive deep-bite malocclusion (≥50% vertical overlap) and the necrosis of multiple mandibular incisor teeth with large periradicular radiolucency is described. The mandibular central incisors were always implicated and were sometimes joined by the lateral incisors. In the examination, no other significant factor that could contribute to the pathology was found. During centric occlusion, the incisal edges of the incisors either occluded into the palatal gingival crevices of the maxillary teeth (Akerly Class II) or abraded the palatal surfaces of maxillary incisors (Akerly Class IV). Additionally, during protrusive and lateral jaw movements, the necrotic mandibular incisors often contacted their antagonists. Mild to severe attrition was found in the affected mandibular incisors. Changes in the stomatognathic equilibrium, due to the deep-bite malocclusion and the elicited chronic trauma, are assumed to be the underlying factors in this pathology.

Keywords: Endodontics; nonvital tooth; orthodontics; Ingle's syndrome

ÖZET Bu makalede, 9 hastada aşırı derin kapanış (≥%50 dikey örtüşme) ve çok sayıda alt keser dişte pulpa nekrozu ve geniş periradiküler radyolüsensilerin birlikte görüldüğü özel bir hastalık paterni tarif edilmektedir. Alt santral kesici dişlerin her zaman, lateral kesicilerin ise bazen bu durumdan etkilendiği görüldü. Muayenede, patolojiye neden olabilecek başka önemli bir faktör bulunamadı. Sentrik oklüzyonda, alt keserlerin kesici kenarlarının üst keserlerin palatal gingival sulkusuna kapandığı (Akerly Sınıf II) veya üst keserlerin palatal yüzeylerini aşındırdığı izlendi (Akerly Sınıf IV). Ek olarak, ileri ve yan çene hareketleri sırasında, nekrotik alt kesici dişlerin sıklıkla antagonistleriyle temas ettiği görüldü. Etkilenen alt kesicilerde hafif ile şiddetli arasında değişen aşınmalar bulundu. Derin kapanış maloklüzyonu ve ortaya çıkan kronik travmaya bağlı stomatognatik dengedeki değişikliklerin bu patolojinin altında yatan etken olduğu düşünülmektedir.

Anahtar Kelimeler: Endodonti; cansız diş; ortodonti; Ingle sendromu

The reasons for pulp necrosis are often dental caries, dental treatment (*e.g.,* deep restorations, extensive crown preparation), and acute dental trauma (*e.g.,* displacement injuries such as luxation). Similar to acute dental trauma, chronic dental trauma has also been shown to be a reason for inflammatory pulpal changes in experimental studies and a clinical observational study.[1-4] The detrimental effects of chronic dental trauma have also been shown in case reports. In a historical article by Ingle protrusive bruxism, a type of chronic dental trauma, was suggested to be the possible reason for mandibular incisor pathosis with periradicular rarefaction in girls aged between 13 and 16 years, which is known as the Ingle's syndrome.[5,6]

Deep-bite is defined as the excessive vertical overlapping of the incisors in centric occlusion, and it is customary to diagnose deep-bite when the in-

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cisors' overlap exceeds one-third of the crown height of the lower incisors. Vertical overlaps greater than 40% should be considered 'excessive.'[7] A classification of the clinical presentations of the deep-bite was made previously by Akerly, and prosthetic and restorative treatment recommendations were discussed.[8,9] It was reported that deep-bite with an overlap greater than 40% had the potential to cause deleterious effects on the surrounding periodontal structures and temporomandibular joint.[10] However, the impact of traumatic deep-bite on pulpal and periradicular health has remained relatively unknown.

In this case series, we present 9 cases, in which mandibular incisor necrosis was found with no apparent reason except for the presence of deep-bite malocclusion. This case series suggests that chronic trauma generating on the mandibular incisors due to deep-bite malocclusion may be a reason for the pulpal death.

CASE REPORTS

All patients were referred to the clinics of the Department of Endodontics at Gazi University, Ankara. At first glance, the patients invariably had necrotic mandibular incisor teeth with large periradicular lesions and deep-bite malocclusion. Painful symptoms varied among the patients from none to severe. Some of the patients' main concern was the swelling or the presence of an intraoral or extraoral fistula. A written informed consent for further investigation, and permission to use their photographs and clinical data were taken.

DEMOGRAPHICS, HABITS, AND EXPOSURES

All patients were Eurasian in origin. Of the 9 cases presented here, patients were mostly female in their third or fourth decade and had no systemic disease (except Case no. 3, who had hyperthyroidism and anemia) (Table 1). Mostly, they had no parafunctional habits. According to the defined criteria, 2 of the patients reported both wake- and sleep-bruxism.[11] There were no histories of acute dental trauma and orthodontic treatment.

ENDODONTIC EXAMINATION

All clinical and radiographic examinations were carried out by an endodontic specialty trainee, and a specialist endodontist with at least 10-years of experience. The necrotic teeth had no caries, restorations, fractures, or cracks. In almost all cases, the mandibular central incisors were affected. The mandibular lateral incisors were also involved in some patients (Table 1, Figure 1). There was attrition in the incisal aspects of many of the affected mandibular incisors, the degree of which varied from mild to severe. Periodontal examination was also done (*i.e.* probing depths were measured and gingival bleeding was observed). Marginal gingival inflammation was present in most of the patients. Some of the necrotic teeth exhibited increased mobility. The patients reported pain on percussion and palpation consistent with the clinical classification of the periradicular pathology (Table 1). Swelling or the presence of or an intraoral or extraoral sinus tract was another common finding. Upon these findings, it was decided that all patients required primary endodontic treatment, except one who required endodontic re-treatment and the completion of an unfinished root canal treatment (Case no. 4, Figure 1). We learned that the patient had pain and swelling due to her mandibular incisor teeth before the previous treatment was initiated 1.5 years ago.

ORTHODONTIC EXAMINATION

The orthodontic examination was done clinically and through orthodontic study casts by a specialist orthodontist with 10-years of experience. All quantitative measurements were performed on the casts. The patients had either an Angle Class I or Class II malocclusion, mostly with mild anterior-crowding (Table 1). The most striking characteristic of the patients was that they all had excessive deep-bite (Figure 1). The vertical overlap of the mandibular incisors by the maxillary incisors in centric occlusion was ≥50% in all patients. The overjet measurements ranged from 2 to 5 mm. Intraoral frontal, profile, and mandibular arch photographs were taken from all patients in order to document the clinical presentation of deep-bite, overjet, and the mandibular incisor teeth, respectively. Akerly classifications were either Class II or IV. Three patients were classified as Akerly Class II (lower incisors' incisal edge occlude into palatal gingival crevices of maxillary teeth), and 6 patients

TABLE 1: Overview of the demographic and clinical information of the patients (all Eurasian).

Case no	sex	Parafunctional habits	Bruxism	Attrition	Periodontal status	Pulpal status	Periradicular status	Angle classification	Overjet	Overbite†	Crowding (intercanine; mandible/maxilla)	Akerly classification (incisor relationship)
1	26, F	No	No	Severe	Gingivitis	31, 41 nonvital	CAA (intraoral sinus tract)	Class II Div 2	3 mm	90%	-1.5/-3 mm	II
2	23, F	No	Yes	Severe	Gingivitis	31, 32, 41, 42 nonvital	AAP	Class I	2.5 mm	69%	-2/0 mm	IV
3	28, F	No	No	Mild	Gingivitis	41, 42 nonvital	AAP	Class I	2 mm	69%	+2/+2 mm	IV
4	27, F	No	Yes	Mild	Gingivitis	41 unfinished-, 31, 42 finished-RCT	AAA	Class II Div 1	4.5 mm	50%	0/0 mm	IV
5	38, F	No	No	Mild	Healthy	31, 41 nonvital	CAA (intraoral sinus tract)	Class II Div 1	5 mm	72%	-0.5/-0.5 mm	IV
6	31, M	No	No	Severe	Gingivitis	31, 41 nonvital	CAA (extraoral sinus tract)	Class II Div 1	3.5 mm	95%	-2/0 mm	II
7	22, F	Nail-biting	No	Mild	Gingivitis	31, 32, 41, 42 nonvital	AAA	Class I	3 mm	74%	-3/-3 mm	II
8	25, F	No	No	No	Gingivitis	31, 41, 42 nonvital	AAP	Class I	3 mm	59%	-4/0 mm	IV
9	22, F	No	No	Severe	Gingivitis	31, 41 nonvital	SAP	Class II Div 2	2 mm	69%	-3/-2 mm	IV

†For each patient, the greatest overbite percentage measured among the incisors is presented; F: Female; M: Male; AAA: Acute apical abscess; SAP: Symptomatic apical periodontitis; CAA: Chronic apical abscess; AAP: Asymptomatic apical periodontitis; RCT: Root canal treatment.

were classified as Akerly Class IV (lower incisor causing progressive abrasion of palatal surfaces of maxillary teeth). Additionally, during protrusive and lateral jaw movements, the necrotic mandibular incisors generally contacted the maxillary anterior teeth.

To sum up, through elaborate examination, other possibilities for pulpal necrosis were ruled out in all cases, leaving extensive deep-bite as the most possible reason.

DISCUSSION

In the dental literature, deep-bite has been a concern for the structural integrity of the dentition (presence of caries, defective restorations, fractures, etc.).[12] Furthermore, deep-bite can cause periodontal problems (localized gingival recession or gingival enlargement, loss of attachment, tooth mobility, etc.) and restorative-prosthetic difficulties (loss of interocclusal space, tooth wear, esthetic and functional problems, etc.).[8,13] Only recently, a cross-sectional study reported an association between the presence of severe fremitus and pulpal/periapical problems, and further suggested that deep-bite patients needed significantly more root canal treatments compared to controls.[14]

The chronic trauma revealed in this case series as deep-bite malocclusion is considered to be the main reason for the mandibular incisor pathology. Speculatively, because the maxillary incisors are more voluminous than the mandibular incisors, and also because of the different force vectors imposed on the dentoalveolar structures, the mandibular incisors are likely to have more tendency to become affected. Probably, with an asymptomatic inflammation of the pulp, pulpal degenerations emerge, and the blood supply of the tooth gradually becomes impaired. Further, if the necessary vascular support cannot be provided, the eventual pulp necrosis occurs.[3] Subsequent bacterial invasion ultimately results in an infected root canal system, and unless it is treated, in apical periodontitis with its known clinical and radiographic findings.

Another sophisticated speculation on the mechanism of this occurrence may be based on the stomatognathic equilibrium. Deep-bite alters the consistency of chewing cycle kinematics, and pro-

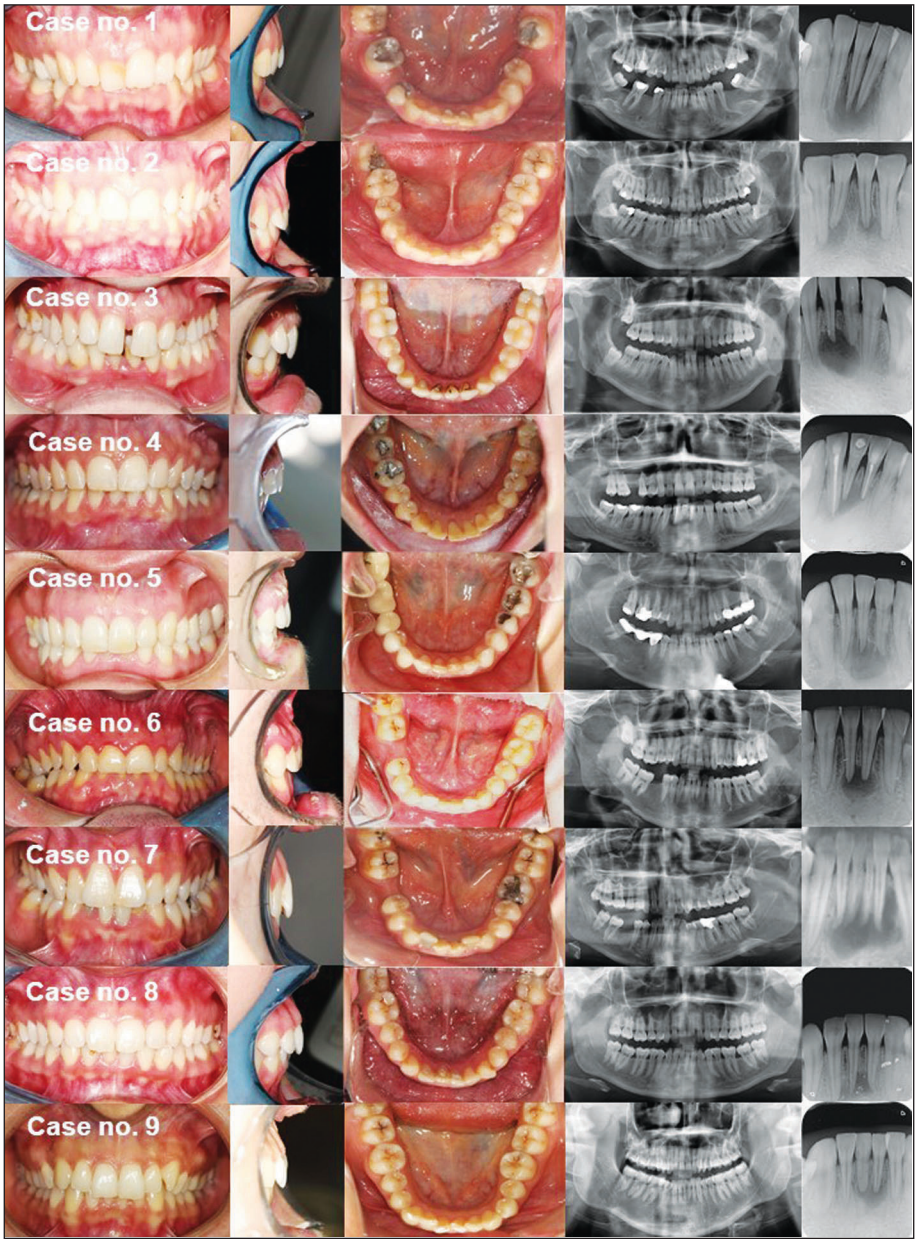

FIGURE 1: Pre-operative photographs and radiographs of cases no. 1 to 9 (age, sex and ethnicity information is given in Table 1); from left to right: frontal and profile photographs showing the extent of the deep-bite malocclusion and overjet, respectively; the mandibular arch; and the pre-operative panoramic and periapical radiographs showing the periradicular radiolucencies. The mandibular arch photographs of cases 1, 3 and 8 were, however, taken between visits. Note that the last image for Case no. 7 is a CBCT-acquired reformatted panoramic image. Enhancements (i.e. adjustments for brightness and colour balance) have been done on the oral photographs.
Camera: Canon EOS 60D body with Canon EF 100 mm f/2.8 Macro USM lens, Tokyo, Japan; panoramic radiography: Sirona Orthophos XG, Bensheim, Germany; periapical radiography: 2+ PSP plate, scanned by Vista Scan, Dürr Dental, Bietigheim-Bissingen, Germany, CBCT: Promax 3D Mid (Planmeca, Helsinki, Finland), FOV size: 20×10 cm, 90 kVp, 12 mA, 0.4 mm^3 voxel size, analyzed with Romexis 2.7.0.R software (Planmeca).

portionately greater condylar rotation is expected during the early phase of mouth-opening in deep-bite cases.[15] Therefore, alterations in the temporomandibular joint movement and the steep incisal guidance may cause abnormal occlusal forces exceeding the regenerative capacity of incisors, inducing pathological changes in the dental pulp tissue, and consequently resulting in their necrosis.

Revealed first by Ingle, protrusive bruxism, as another form of chronic dental trauma, has been suggested to be a reason for mandibular incisor pathosis associated with periradicular lesions.[5,6] Our cases were similar to Ingle's cases in some aspects but differed mainly by the following findings: the patients did not necessarily present with bruxism, and the teeth associated with the periradicular lesion were always necrotic. And, excessive deep-bite was the common finding in our cases. Unfortunately, the deep-bite status of the cases was not reported in Ingle's papers.[5,6]

In conclusion, there may be an association between excessive deep-bite and mandibular incisor pulp necrosis. Systematic research is required on this relationship. If this relationship can be confirmed, an early orthodontic treatment of deep-bite malocclusion might be recommended for the pulpal and periradicular health of mandibular incisors.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Güven Kayaoğlu, Büşra Serçe Fikirli;* ***Design:*** *Güven Kayaoğlu, Büşra Serçe Fikirli, Emine Kaygısız;* ***Control/Supervision:*** *Güven Kayaoğlu;* ***Data Collection and/or Processing:*** *Güven Kayaoğlu, Büşra Serçe Fikirli, Emine Kaygısız;* ***Analysis and/or Interpretation:*** *Güven Kayaoğlu, Büşra Serçe Fikirli, Emine Kaygısız;* ***Literature Review:*** *Güven Kayaoğlu, Emine Kaygısız;* ***Writing the Article:*** *Güven Kayaoğlu, Emine Kaygısız;* ***Critical Review:*** *Güven Kayaoğlu, Büşra Serçe Fikirli, Emine Kaygısız.*

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