

An Overview on Endo-Perio Interrelationship - A Multidisciplinary Approach

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Abstract: The human periodontium and dental pulp cavity are closely connected by their proximity and by the presence of apical and lateral radicular foramina, which permit the passage of pathogens between these two distinct anatomical areas. The interrelationship between endodontic and periodontic lesion has been a subject of speculation, confusion and controversy for many years. Pulpal and periodontal problems are responsible for more than 50% of tooth mortality today. An endo-perio lesion can have a varied pathogenesis which ranges from quite simple to relatively complex one. When a periapical lesion communicates with a deep periodontal pocket, the etiology can be either endodontic or periodontic. The purpose of this review paper is to explain the nature of that relationship. Successful therapy will only result from the establishment of an accurate diagnosis and forming such a diagnosis requires a methodical multi-staged approach.

Keywords: Endo-perio lesions, Periodontal, Pulpal, Diagnosis

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I. Introduction

The pulp-periodontal interrelationship is one in which “there are so many paths of communication that one is tempted to put aside the notion of two distinct anatomical structures and consider them as a single continuous system.”^[1] Pulpal infection may cause a tissue destructive process that proceeds from the apical region of a tooth toward the gingival margin. The relationship between periodontal and pulpal disease was first described by Simring and Goldberg in 1964 as Pulpodontic-periodontic syndrome^[2]. This syndrome can be initiated by either pulpal or periodontal disease and may manifest pulpal and periodontal symptoms [3]. The periodontium and pulp have embryonic, anatomic and functional inter-relationship. In the embryological development of the tooth, the dental follicle, which is the precursor of the periodontium, is in close relationship with the dental papilla from where comes the pulp. They are separated only by Hertwig’s epithelial root sheath prior to root formation. However there is a common area at the future apical foramen. As the development progress there is a direct vascular communication between the pulp and periodontium through the apical foramen and accessory (lateral) canals [4]. Once the tooth has erupted however, removal of cementum by root planing and scaling introduces a further perio/pulpal pathway of communication through exposed dentinal tubules. The other means of communication are Periodontal ligament (Sharpey’s fibres), Alveolar bone, Palatogingival groove, Fractures and perforations and Common vasculolymphatic drainage pathways (Simon 1984) [5].

II. Pathways of communication between Pulp and Periodontium

There are various pathways for the exchange of infectious elements and irritants from the pulp to periodontium or vice versa, leading to the development of endodontic periodontic lesions [6,7]

Pathways of developmental origin (anatomical pathways):

- Apical foramen, accessory canals/lateral canals
- Congenital absence of cementum exposing dentinal tubules
- Developmental grooves

Pathways of pathological origin:

- Empty spaces on root created by Sharpey's fibers
- Root fracture following trauma
- Idiopathic root resorption - internal and external
- Loss of cementum due to external irritants

Pathways of iatrogenic origin:

- Exposure of dentinal tubules following root planning
- Accidental lateral root perforation during endodontic procedures

- Root fractures during endodontic procedures.
- Vertical root fracture

III. Etiology

The main etiological factors for endo-perio lesions are living (bacteria, fungi and viruses) and nonliving pathogens. Along with these, many contributing factors such as trauma, root resorptions, perforations, and dental malformations also play an important role in the development and progression of such lesions [8,9]. Kobayashi et al., (1990) reported that the predominant obligate anaerobes common to both regions are *Streptococcus*, *Peptostreptococcus*, *Eubacterium*, *Bacteroides* and *Fusobacterium* [10]. Other than these microbial findings, similarities in the composition of cellular infiltrates also suggest the existence of communication between the pulp and the periodontal tissues (Bergenholtz, 1978) [11]. These findings infer that cross-contamination between the pulp and periodontal tissues is possible. Although disease transmission from the pulp to the periodontal tissue is possible, the influence of periodontal disease on pulpal status remains controversial (Bergenholtz & Lindhe, 1978).

Response of the pulpal tissues to long standing periodontal disease

- Deposition of large quantities of reparative secondary dentine along the pulpal walls,
- Induction of pulpal fibrosis,
- Dystrophic calcifications,
- Reduced vascularity and fewer nerve fibres, (Bender & Seltzer 1972) [12]

Pulpal infection can both initiate and maintain periodontal disease

- Although the effects of pulpal disease on the periodontitis are well documented, a clear-cut relationship between periodontitis and pulpal involvement is less evident. The main obvious route of communication is the apical foramina. Advanced pulpitis will lead to pulp necrosis, which often is accompanied by inflammatory bone resorption at the root apex, as found in cases of apical periodontitis or an apical abscess. This is also known as *retrograde periodontitis* because it represents the periodontal tissue breakdown from an apical to a cervical direction and is the opposite of *orthograde periodontitis* that results from a sulcular infection.[13]

IV. Classification of Endodontic-Periodontic Lesions and how to identify them -

The first classification of endodontic-periodontic lesions based on pathology of origin was proposed by Simon et al. [14] as follows:

1. Primary endodontic lesions
2. Primary periodontic lesions
3. Primary endodontic lesions with secondary periodontal involvement
4. Primary periodontic lesions with secondary endodontic involvement
5. True combined lesions.

Though Simon et al. have classified these lesions into five types but actually last three can be considered as combined lesions. In contrast to combined perio-endo lesions, concomitant pulpal and periodontal lesions reflect the presence of two separate and distinct disease states with different causative factors and with no clinical evidence that one disease state has influenced the other [15].

Recently, Von Arx T and Cochran [16] proposed a clinical treatment classification of perio-endo-furcation lesions based on the role of membrane application in endodontic surgery.

Class I: Periapical bone defect without marginal lesion

- Ia Lingual/palatal cortex not eroded
- Ib Lingual/palatal cortex eroded (with a buccal surgical approach, this will result in a transosseous or through-and-through bone defect)

Class II: Periapical lesion (with or without lingual erosion) and concomitant marginal lesion

- IIa No communication between the separate lesions
- IIb The two lesions are fused = communicating apico-marginal or endodontic-periodontic lesion

Class III: Lateral or furcational lesion (with or without marginal lesion)

- IIIa No communication to alveolar crest/marginal periodontium
- IIIb Communication to alveolar crest/marginal periodontium

Singh [17] classified endo-perio lesions based on the pathogenesis and added the term iatrogenic lesions, usually endodontic lesions produced as a result of treatment modalities. All these classifications are mainly based on the

theoretic pathways explaining how these radiographic lesions are formed. Therefore, by comprehensive understanding of the pathogenesis and investigations, the clinician can make a sound diagnosis, formulate an appropriate treatment plan and assess the prognosis of these lesions [15]

Diagnostic procedures used to identify the endo-perio lesion

EXAMINATION	PRIMARY ENDODONTIC LESION	PRIMARY PERIODONTAL LESION	PRIMARY ENDODONTIC SECONDARY PERIODONTAL	PRIMARY PERIODONTAL SECONDARY ENDODONTIC	TRUE COMBINED LESION
VISUAL	Soft tissue - presence of sinus opening. Presence of deep carious lesion/defective restoration/previous root canal treatment/cracks, discoloration, attrition, erosion, abrasions.	Inflamed gingiva/gingival recession around multiple teeth. Accumulation of plaque and subgingival calculus around multiple teeth. Intact teeth. Presence of swelling indicating periodontal abscess	Plaque forms at the gingival margin of the sinus tract and leads to inflammation of marginal gingiva Exudate Root perforation/fracture / misplaced post.	Presence of plaque, subgingival calculus and swelling around multiple teeth. Pus, exudate. Localized or generalized gingival recession and exposure of roots.	Plaque, subgingival calculus and periodontitis will be present in varying degrees. Pus, exudate. Swelling around single or multiple teeth.
PAIN	Sharp	Usually dull ache. Sharp pain only in acute condition.	Usually sharp shooting. Dull ache in chronic condition.	Usually a dull ache. Sharp pain in acute periodontal abscess.	Usually dull ache. Sharp pain only in acute condition.
PALPATION	It doesn't identify whether the inflammatory process is of endodontic or periodontal origin.	Pain on palpation	Pain on palpation	Pain on palpation	Pain on palpation
PERCUSSION	Tender on percussion	Sensitivity of the proprioceptive fibers in an inflamed periodontal ligament will help identify the location of pain	Tender on percussion	Tender on percussion	Tender on percussion
MOBILITY	Fractured teeth often shows mobility	Localized to generalized mobility	Localized mobility	Generalized mobility.	Generalized mobility with high grade mobility to the involved tooth.
PULP VITALITY	A lingering response - irreversible pulpitis. No response - necrotic pulp.	Pulp is vital and responsive to testing.	Negative pulp vitality test	Pulp vitality test may be positive in multi rooted tooth.	Usually negative because of non vital pulp, but Pulp vitality test may be positive in multi rooted tooth.
PERIODONTAL PROBING	A deep narrow solitary pocket in absence of periodontal disease may indicate the presence of a lesion of endodontic origin or vertical root fracture	Multiple wide and deep pockets	Presence of wide solitary pocket, but if periodontal lesion is due to vertical root fracture then solitary deep narrow pocket	Multiple wide and deep pockets	Typical conical periodontal type of probing with the exception that at the base of the periodontal lesion, the probe will abruptly drop further down the lateral root surface and may even extend to the apex of the root.

SINUS TRACING	Points to the apex of the root or furcations of molars	Sinus tract is at the lateral aspect of the root.	Sinus tract mainly at the apex or furcation area.	Sinus tract is at the lateral aspect of the root.	Difficult to trace the origin of the lesion. It may be necessary to raise the flap to determine the etiology.
RADIOGRAPH Y	Presence of deep carious lesion/defective restoration/previous poor root canal treatment/endodontic mishaps/root fracture/root resorption with peri-apical radiolucency.	Vertical bone loss. Bone loss wider coronally.	Presence of deep carious lesion/defective restoration/previous poor root canal treatment/endodontic mishaps/root fracture/root resorption with a wide base radiolucency around the root	Angular bone loss in multiple tooth with a wide base coronally and narrow at the apex of the tooth.	May be similar to vertical root fracture.
CRACKED TOOTH TESTING (using transillumination, wedging, staining)	Painful response on chewing, particularly during release of bite	No symptoms	Painful response on chewing, particularly during release of bite	No symptoms	Painful response on chewing, particularly during release of bite

Nomenclature distinguishes between lesions caused by periodontal pathogens, as seen in chronic periodontitis, and lesions of the apical periodontal tissues associated with endodontic pathology. When the location is distinct and the lesion is discrete, the two are easy to differentiate. When they simultaneously affect the marginal and apical areas of the periodontium, making it essential to ascertain their true cause through differential diagnosis[18].

V. Treatment Planning and Prognosis of Endo-Perio Lesions :

In general, when primary disease of one tissue, i.e. pulp or periodontium, is present and secondary disease is just starting, treat the primary disease[19,9,12,20,21]. When secondary disease is established and chronic, both primary and secondary diseases must be treated. By and large, endodontic therapy precedes periodontal therapy. Periodontal therapy may or may not be required, depending on disease status. The complete healing of destroyed periodontal support can be expected following the treatment of pulpal pathology.

Primary endodontic lesions - For primary endodontic lesions conventional endodontic therapy alone will resolve the lesion. A review of 4-6 months post-operatively should show healing of the periodontal pocket and bony repair [22]. Good prognosis is to be expected if treatment is carried out properly with a focus on infection control. The sinus tract extending into the gingival sulcus or furcation area disappears at an early stage once the affected pulp has been removed and the root canals well cleaned, shaped, and obturated. In case of tooth with large periapical lesion, orthograde endodontic therapy has been advised instead of surgical endodontic therapy[22,23]. Placement of intra-canal medicaments such as calcium hydroxide has found to be very effective in the healing of large periapical lesion. Surgical endodontic therapy has been shown to be unnecessary even in the presence of large periradicular radiolucencies and periodontal abscesses. Invasive periodontal procedures should be avoided as this may cause further injury to the attachment-possibly delaying healing [24].

Primary periodontic lesions - Primary periodontal disease should only be treated by periodontal therapy. In this case, the prognosis depends on the severity of the periodontal disease, efficacy of periodontal therapy and patient response; however, prognosis of primary periodontal lesions is not as favorable as primary endodontic lesions. Primary periodontal lesions should be treated by hygiene phase therapy in the first instance. Subsequently, poor restorations and developmental grooves that are involved in the lesion must be removed. Periodontal surgery should be performed after the completion of hygiene phase therapy if deemed necessary [25]. Since, the presence of an intact cementum layer is important for the protection of the pulp and vigorous surgical periodontal procedures may remove cementum and expose dentinal tubules, which in turn transport irritants, thereby cause pulpal inflammation and necrosis of the dental pulp. Therefore, clinicians should take precautions during periodontal therapy and avoid the use of irritating chemicals, minimize the use of ultrasonics and rotary scaling instruments when <2 mm of dentin thickness remaining. Judicious use of periodontal surgical intervention is advantageous to treat this lesions [26].

Primary endodontic with secondary periodontal lesions - The treatment and prognosis of the tooth with these lesions are different from those of teeth involved with only primary endodontic disease. The prognosis for primary endodontic lesions is good but worsens in the advanced stages of secondary periodontal

involvement. The prognosis then depends on the effectiveness of periodontal treatment and with advancement becomes comparable to that of a true-combined lesion [26]. Tooth with these lesions should first be treated with endodontic and simple hygiene phase therapy. In this case, multi-visit endodontic therapy should be practiced and the placement of intra-canal medicament was found to be very useful in reducing inflammation and favoring repair [27]. Treatment results should be evaluated in 2-3 months and only then further periodontal treatment should be considered. This sequence of treatment allows sufficient time for initial tissue healing and better assessment of the periodontal condition.[28,29] It also reduces the potential risk of introducing bacteria and their byproducts during the initial phase of periodontal healing.

But in cases where healing with only endodontic therapy does not occur then both endodontic and periodontic treatments should be carried out since with endodontic treatment alone, only part of the lesion may heal up to the level of the secondary periodontal lesion. If the endodontic treatment is adequate, the prognosis of primary endodontic disease with secondary periodontal involvement depends primarily on the severity of periodontal involvement, periodontal treatment and patient response.

Primary endodontic lesions with secondary periodontal involvement may also occur as a result of iatrogenic damage such as root perforation or fracture during root canal treatment or placement of pins or posts. Root perforations are treated according to their etiology. The outcome of the treatment of root perforations depends on the size, location, time of diagnosis and treatment, degree of periodontal damage as well as the sealing ability and biocompatibility of the sealer.

Root fractures may also present as primary endodontic lesions with secondary periodontal involvement. These typically occur on root-treated teeth, often with post and crowns. Treatment depends on the tooth type, extent, duration and location of fracture, for example, single rooted tooth with lesions caused by vertical root fracture has a hopeless prognosis and should be extracted [30] while molars can be treated by root resection or hemisection. [31]

Primary periodontal secondary endodontic lesion and true combined lesions - Primary periodontal disease with secondary endodontic involvement and true combined endodontic-periodontal diseases require both endodontic and periodontal regenerative procedures. The success rate of the endodontic-periodontic combined lesion without a concomitant regenerative procedure has been reported to a range from 27% to 37%. [32] Combined lesions can be classified into three types-

1. Tooth with two separate lesions, one endodontic usually periapical and one periodontal with no communication,
2. Teeth with a single lesion that involves both endodontic and periodontal pathosis
3. Teeth with endodontic and periodontal lesions that were once separate but now communicate.

True-combined lesions should be treated initially as primary endodontic lesions with secondary periodontal involvement. In general, assuming the endodontic therapy is adequate, what is of endodontic origin will heal. Thus the prognosis of combined diseases rests with the efficacy of periodontal therapy [5]. The prognosis of a true-combined perio-endo lesion is often poor or even hopeless, especially when periodontal lesions are chronic with extensive loss of attachment. [33] Prior to surgery, palliative periodontal therapy should be completed and root canal treatment carried out. The prognosis of true combined lesion is often poor or even hopeless, especially when periodontal lesions are chronic and extensive. Though, root amputation, hemisection or bicuspidization may allow the root configurations to be changed sufficiently for a part of the root structure to be saved, however, the operator need to consider various factors before root resection such as tooth function, root filling, anatomy, restorability, bone support around the healthy root and patient's compliance.

The prognosis of an affected tooth can also be improved by increasing bony support, which can be achieved by bone grafting and guided tissue regeneration (GTR). These advanced treatment options are based on responses to conventional periodontal and endodontic treatment over an extended time period. These regenerative procedures with the aid of the microscope, in the treatment of combined lesions have been found to have a success rate of 77.5%. [34] GTR therapy has also been implemented in the endodontic surgeries as a concomitant treatment during the management of the endodontic-periodontal lesions. [35,36,37,38] The decisions and treatment strategy for the application of the regenerative procedures are made at various levels such as pre-surgical, post-root canal treatment, intra-surgical, and post-surgical. Factors influencing treatment outcome should also be considered at each level under patient-specific, defect-specific, and healing categories. [39]

The pre-surgical assessment includes

- establishing and verifying the non-vital status of the pulp,
- the extent and severity of the periodontal destruction, and
- therapeutic prognosis of the planned regenerative procedure.

Once the therapeutic prognosis of the periodontal regenerative procedure is determined to be favorable, then endodontic therapy should be provided. Root canal therapy helps to reduce the mobility of the involved tooth therefore, after a successful root canal therapy; tooth mobility should be further assessed to determine the necessity for splinting. Cortellini et al. [40,41] have recommended splinting of the mobile tooth before GTR procedure.

The intra-surgical assessment should include

- morphology of the periodontal defect,
- defect type,
- material of choice to fill the defect and augment healing,
- control of patient's oral hygiene, and
- wound stabilization. [42,43]

Furthermore, long term follow up is mandatory for these lesions. The ideal therapeutic sequence for the true combined lesion is:

Root canal therapy;

Review after 2 to 3 months;

If lesion is not showing signs of resolving (clinically and radiographically) perform appropriate periodontal therapy;

Review 2 to 3 months after periodontal therapy and re-evaluate radiographically. [44] However, advanced diagnostic tests like cone beam computer tomography to check the conditions of the hard tissues, pulse oximetry for evaluate the true vitality, polymerase chain reaction to identify the specific microbes may add value in proper diagnosis.

VI. Conclusion

A perio-endo lesion can have a varied pathogenesis which ranges from quite simple to relatively complex one. To make a correct diagnosis the clinician should have a thorough understanding and scientific knowledge of these lesions. Successful treatment of lesions of endodontic-periodontic origin is related to the identification of the etiology, control of micro biota present, the immunological characteristics of the individual, and one of the strategies and the possible prognosis is linked to the origin of the process infection (endodontic or periodontal).

CONSENT -

It is not applicable.

ETHICAL APPROVAL -

It is not applicable.

COMPETING INTERESTS -

Authors have declared that no competing interests exist.

Bibliography

- [1]. Khan R.N., Kumar A., Chadgal S. Majid Jan S. ENDO-PERIO INTERRELATIONSHIP- AN OVERVIEW. International Journal of Information Research and Review. 2017.;04(3)-3895-3898.
- [2]. Simring M, Goldberg M. The pulpal pocket approach: retrogradeperiodontitis. J Periodontol 1964; 35: 22-48.
- [3]. Meshack RA. Tavane P. Srinivasa TS .Guru R. A Systematic review of Effectiveness of Combined Perio – Endo Interventions . J. Adv Oral Research 2011;2(3):5-9.
- [4]. Pitt Ford TR. Harty's Endodontics in Clinical Practice. 5th Edition 2009; 215
- [5]. Rotstein I. Simon JH. The endo-perio lesion: a critical appraisal of the disease condition. Endodontic Topics 2006, 13, 34–56
- [6]. Mjör IA, Nordahl I. The density and branching of dentinal tubules in human teeth. Arch Oral Biol 1996;41:401-12.
- [7]. Zehnder M, Gold SI, Hasselgren G. Pathologic interactions in pulpal and periodontal tissues. J Clin Periodontol 2002;29:663-71.
- [8]. Sunitha VR, Emmadi P, Namasivayam A, Thyegarajan R, Rajaraman V. The periodontal-endodontic continuum: A review. J Conserv Dent 2008;11:54-62.
- [9]. Rotstein I, Simon JH. Diagnosis, prognosis and decision-making in the treatment of combined periodontal-endodontic lesions. Periodontol 2000 2004;34:165-203.
- [10]. Kobayashi T, Hayashi A, Yoshikawa R, Okuda K, Hara K. The microbial flora from root canals and periodontal pockets of non-vital teeth associated with advanced periodontitis. Int Endod J. 1990 Mar;23(2):100-6.
- [11]. Bergenholtz, Lindhe J. 1978. Effect of experimentally induced marginal periodontitis and periodontal scaling on the dental pulp. J ClinPeriodontol,5:59–73.
- [12]. Bender IB, Seltzer S. The effect of periodontal disease on the pulp. Oral Surg Oral Med Oral Pathol. 1972 Mar;33(3):458-74.
- [13]. Newman MG. Takei HH. Klokkevold PR. Carranza FA. Carranza's Clinical Periodontology. Tenth edition.2006:871-880.
- [14]. Simon JH, Glick DH, Frank AL. The relationship of endodontic-periodontic lesions. J Periodontol 1972;43:202-8.
- [15]. Parolia A, Gait TC..Porto ICCM. Mala K. Endo-perio lesion: A dilemma from 19th until 21st century. Journal of Interdisciplinary Dentistry.2013, 3(1):2-11

- [16]. Von Arx T, Cochran DL. Rationale for the application of the GTR principle using a barrier membrane in endodontic surgery: A proposal of classification and literature review. *Int J Periodontics Restorative Dent* 2001;21:127-39.
- [17]. Singh P. Endo-perio dilemma: A brief review. *Dent Res J (Isfahan)* 2011;8:39-47
- [18]. Edoardo F. *Endo-Periodontal Lesions*. London: Quintessence Publishing; 2011. p. 1-2.
- [19]. Richard E Walton and Mahmoud Torabinejad. *Principles and Practice of Endodontics*. 3rd Edition Philadelphia W B Saunders Company; 2002 pp. 467-84.
- [20]. Harrington GW, Steiner DR, Ammons WF. The periodontal-endodontic controversy. *Periodontol* 2000 2002;30:123-30.
- [21]. Meng HX. Periodontic-endodontic lesions. *Ann Periodontol* 1999;4:84-9
- [22]. Stock CJR, Nehammer CF. Endodontics in practice. *British Dental Journal* 1990: 62-66 .
- [23]. Whyman RA. Endodontic-periodontic lesions. Part II: Management. *N Z Dent J* 1988;84:109-11.
- [24]. Solomon C, Chalfin H, Kellert M et al. The endodontic-periodontal lesion: a rational approach to treatment. *Journal of the American Dental Association* 1995;126:473-479 .
- [25]. Anand V, Govila V, Gulati M. Endo-Perio Lesion: Part II (The Treatment) – A Review. *Archives of Dental Sciences*. 2012;3(1):10-16.
- [26]. Mhairi RW. The pathogenesis and treatment of endo-perio lesions. *CPD Dent* 2001;2:77-104.
- [27]. Carrotte P. Endodontics: Part 9. Calcium hydroxide, root resorption, endo-perio lesions. *Br Dent J* 2004;197:735-43.
- [28]. Paul BF, Hutter JW. The endodontic-periodontal continuum revisited: New insights into etiology, diagnosis and treatment. *J Am Dent Assoc* 1997;128:1541-8.
- [29]. Chapple IL, Lumley PJ. The periodontal-endodontic interface. *Dent Update* 1999;26:331-6, 338, 340.
- [30]. Solomon C, Chalfin H, Kellert M, Weseley P. The endodontic-periodontal lesion: A rational approach to treatment. *J Am Dent Assoc* 1995;126:473-9.
- [31]. Moule AJ, Kahler B. Diagnosis and management of teeth with vertical root fractures. *Aust Dent J* 1999;44:75-87.
- [32]. Oh SL, Fouad AF, Park SH. Treatment strategy for guided tissue regeneration in combined endodontic-periodontal lesions: Case report and review. *J Endod* 2009;35:1331-6.
- [33]. Christie WH, Holthuis AF. The endo-perio problem in dental practice: diagnosis and prognosis. *Journal of the American Dental Association* 1990;56:1005-1011.
- [34]. Kim E, Song JS, Jung IY, Lee SJ, Kim S. Prospective clinical study evaluating endodontic microsurgery outcomes for cases with lesions of endodontic origin compared with cases with lesions of combined periodontal-endodontic origin. *J Endod* 2008;34:546-51.
- [35]. Taschieri S, Del Fabbro M, Testori T, Saita M, Weinstein R. Efficacy of guided tissue regeneration in the management of through-and-through lesions following surgical endodontics: A preliminary study. *Int J Periodontics Restorative Dent* 2008;28:265-71.
- [36]. Britain SK, Arx TV, Schenk RK, Buser D, Nummikowski P, Cochran DL. The use of guided tissue regeneration principles in endodontic surgery for induced chronic periodontic-endodontic lesions: A clinical, radiographic, and histologic evaluation. *J Periodontol* 2005;76:450-60.
- [37]. Kerezoudis NP, Siskos GJ, Tsatsas V. Bilateral buccal radicular groove in maxillary incisors: Case report. *Int Endod J* 2003;36:898-906.
- [38]. John V, Warner NA, Blanchard SB. Periodontal-endodontic interdisciplinary treatment: A case report. *Compend Contin Educ Dent* 2004;25:601-2,604.
- [39]. Bashutski JD, Wang HL. Periodontal and endodontic regeneration. *J Endod* 2009;35:321-8.
- [40]. Cortellini P, Tonetti MS, Lang NP, Suvan JE, Zucchelli G, Vangsted T, et al. The simplified papilla preservation flap in the regenerative treatment of deep intrabony defects: Clinical outcomes and postoperative morbidity. *J Periodontol* 2001;72:1702-12.
- [41]. Schulz A, Hilgers RD, Niedermeier W. The effect of splinting of teeth in combination with reconstructive periodontal surgery in humans. *Clin Oral Investig* 2000;4:98-105.
- [42]. Tonetti MS, Prato GP, Cortellini P. Factors affecting the healing response of intrabony defects following guided tissue regeneration and access flap surgery. *J Clin Periodontol* 1996;23:548-56.
- [43]. Trombelli L, Kim CK, Zimmerman GJ, Wikesjö UM. Retrospective analysis of factors related to clinical outcome of guided tissue regeneration procedures in intrabony defects. *J Clin Periodontol* 1997;24:366-71.
- [44]. Iain LC Chapple, Philip J Lumely. The periodontal-endodontic interface. *Dental update* 1999;26:331-341.

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