

CASE REPORT**MANAGEMENT OF ENDO-PERIO LESION- A CASE SERIES****VB Mandlik¹****ABSTRACT**

The relationship between periodontal and endodontic disease has been a subject of speculation for many years. The Pulp and the periodontium are developmentally and functionally closely related to each other. The simultaneous existence of pulpal and periodontal disease can complicate diagnosis and treatment planning. The success of both periodontal and endodontic therapy depends on the elimination of both disease processes, whether they exist separately or as a combined lesion. Treatment of such lesions may be simple root canal therapy with basic periodontal therapy. In certain complex cases there may be a need to undertake regenerative or resective approach. This paper tries to bring out four such cases of endo perio lesion.

INTRODUCTION

The term endodontic-periodontal lesions have been used to describe lesions due to inflammatory products found in varying degrees in both pulp and periodontal tissues^{1,2}. The pulp and the periodontium have common embryonic derivation and are anatomically related to each other via the lateral, apical canals and neuro -vascular apparatus which creates pathway for exchange of noxious agents between the two thereby creating the endo-perio lesion and are responsible for more than 50% tooth mortality³.

The diagnosis between endodontic and periodontic lesion is seldom difficult, since endodontic lesion most often produce severe pain that is localized to tooth. Symptoms of plaque induced periodontitis are usually minor, and the sign of disease is confined to marginal periodontium, but when these lesions occur as combine lesion then diagnosis may be challenging as clinical symptoms may sometime be confusing and their etiology misinterpreted⁴.

The establishment of correct diagnosis may also be complicated by the fact that both periodontal and endodontic lesions can simultaneously affect the same tooth. In such situations, one of the lesion may either be the result of or cause of other or the two lesions may constitute two separate processes which have developed independently. In view of the above establishing of correct diagnosis is important in order to preclude unnecessary and even detrimental treatment^{5,6}.

CASE I

A 43 year old male patient whose medical history was noncontributory reported to dental OPD with chief complaint of chronic food impaction with pain and swelling in relation to his lower back teeth on left side since one month. On examination there was open contact between tooth 36 and 37, probing pocket depth was 9 mm (Fig. 1 A to B), there was mild tenderness on percussion and grade II mobility in tooth 36 grade I mobility in tooth 37. Pus discharge was noticed on buccal aspect of tooth 36. IOPA revealed extensive bone loss along the distal root which was extending to apex, the vitality test with hot gutta percha was done and tooth 36 and tooth 37 were found to be nonvital. It was diagnosed as case of primary periodontal lesion with secondary endodontic involvement. Endodontic treatment was taken up first and the patient was followed up for one month (Fig. 1 C-D).

After one-month definitive periodontal therapy was undertaken. Full thickness mucoperiosteal flap was raised and all granulation tissue were removed with help of curette, the defect was exposed, severe bone loss was noted around the distal root of tooth 36.

Hemisectioning of tooth 36 was done with rotary instrument and diamond bur extending from furcal area towards coronal area. Maximum care was taken to avoid injury to interradicular bone and adequate coronal tooth structure was conserved with root mesial half of the tooth for future restoration. Distal half of tooth 36 was removed with crown and corresponding root. HA bone substitute was placed in the defect with resorbable barrier membrane (Fig. 1 E to F). The flap was repositioned and sutured were placed.

The patient was prescribed Tab Amoxicillin 500 TDS

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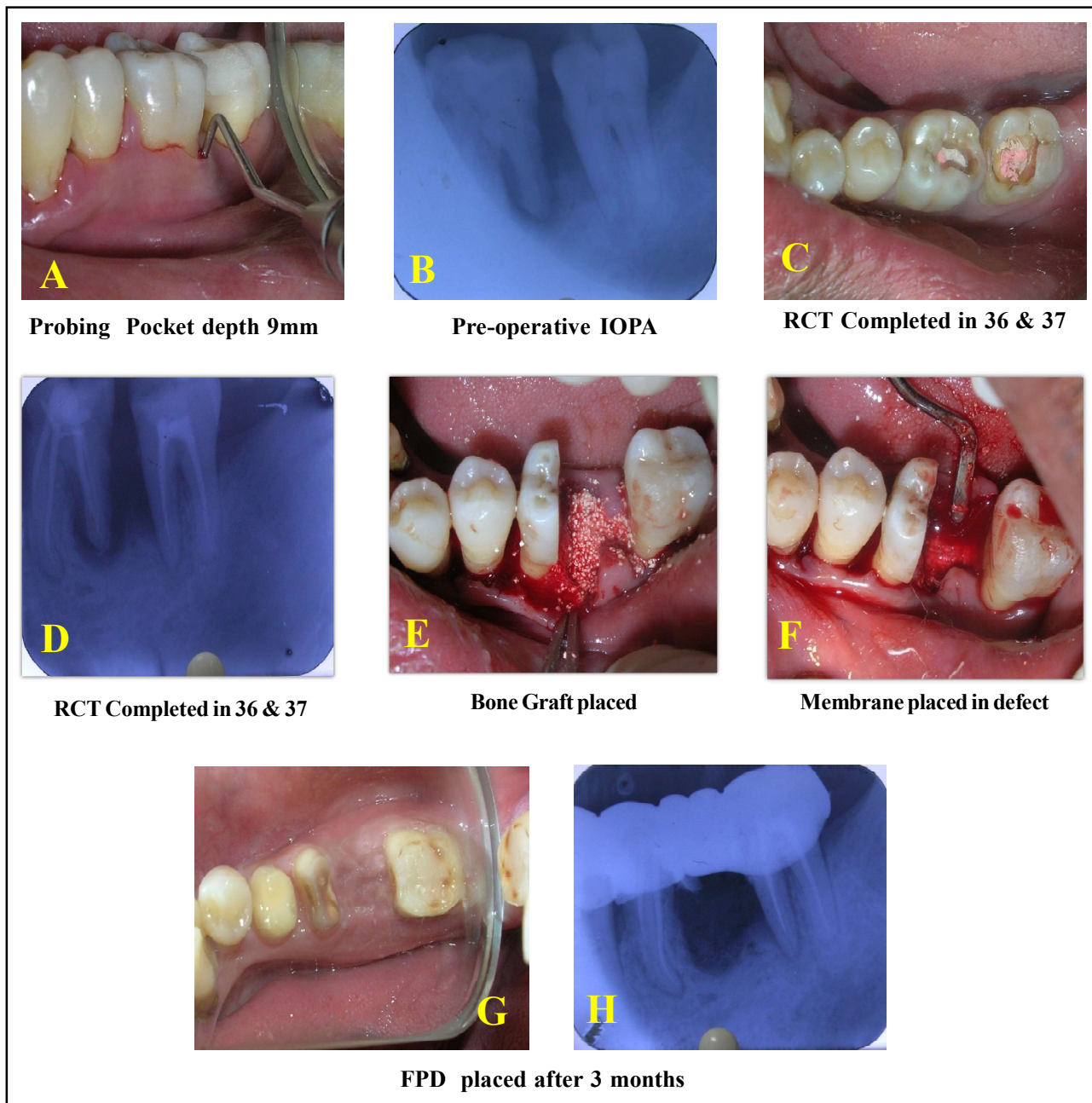


Fig 1 (A to H): Primary Periodontal with Secondary Endodontic Primary Periodontal with Secondary Endodontic Involvement

for 3 days, Tab Brufen 400mg TDS for 3 days and chlorhexidine 0.2% mouth rinse twice a day for 7 days. Recall visit was scheduled at 1 week, 1 month and at 3 months the healing was satisfactory, and no complications were observed. He was later referred to prosthodontist for restoration (Fig. 1 G to H).

CASE NO 2

A 43-year-old male patient reported to dental OPD with the complaint of pain and pus discharge from left maxillary molar since one month. Clinical examination revealed exposure of disto-buccal root of tooth 26 (Fig.

2 A), grade-I mobility, and tenderness on percussion. Radiographic examination revealed radiolucency along the distobuccal root of maxillary first molar. Vitality test was done and was inconclusive. It was a case of Primary Periodontal with secondary endodontic Lesion. Since there was severe bone loss in relation to distobuccal root, it was decided to do root canal therapy followed by root resection. First root canal treatment was completed (Fig. 2 B), later full thickness mucoperiosteal flap was raised using crevicular incision, the distobuccal root was resected using fissure bur, the area was irrigated, bone graft placed in defect and the flap was

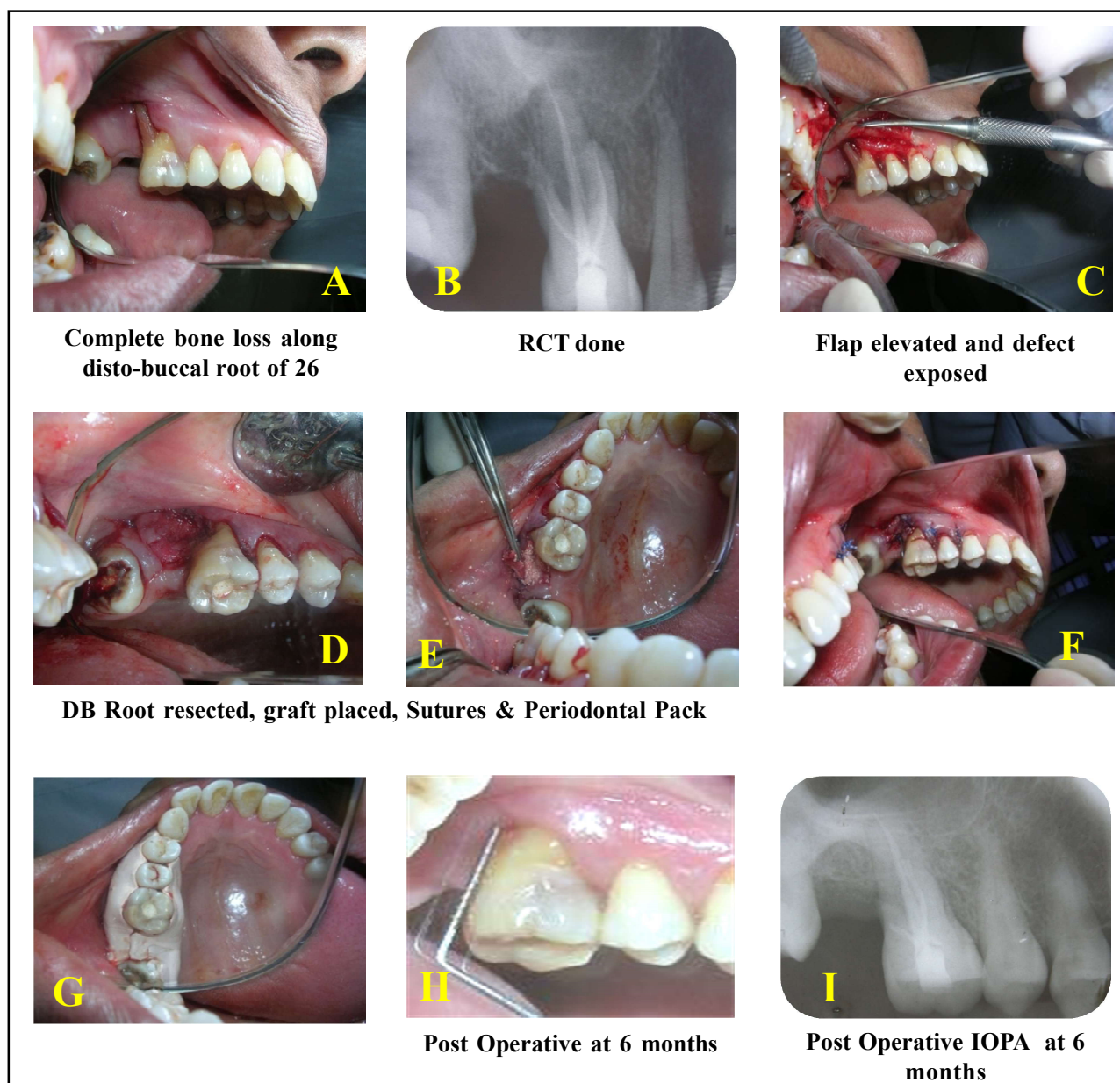


Fig 2: (A to I)

sutured (Fig. 2 C to G) . Post-operative follow up was done at 1 week, 1 month, and 3 months respectively. At end of 3 months, partial healing of the bone defect was observed (Fig. 2 H to I). The patient was subsequently referred to prosthodontist for restoration.

CASE NO 3

A 35-year-old female patient reported with complaint of mild pain and swelling in her upper front teeth since 10 days. The patient gave history of trauma 10 years back, on clinical examination, there was swelling in relation to tooth 11 which was extending beyond the mucogingival junction, probing pocket depth of 10 mm (Fig. 3 A to B) with grade II mobility in tooth 11 and mild tenderness on percussion. IOPA revealed extensive

bone in relation to tooth 11. Vitality test was carried out and it was found to be nonvital. Diagnosis: Primary periodontal lesion with secondary endodontic involvement. Root canal treatment was carried out, abscess was allowed to drain by giving open dressing on first appointment. Two days later there was reduction of swelling, and reduction in probing pocket depth. The root canal was irrigated and closed dressing with calcium hydroxide was given. The patient was recalled after 1 week for obturation (Fig. 3 C). After one month full thickness flap was raised, extending from 12 to 21 region, the granulation tissue were removed from the defect (Fig. 3 D to E), later Platelet Rich Plasma (PRP) was mixed with the calcium sulfate α -hemihydrate graft and placed into the defect, the flap was sutured and

**Fig 3: A to J**

periodontal dressing was placed (Fig. 3 F to I). Post-operative instructions were given, and patient was placed on antibiotics and anti-inflammatory for 05 days. Pack and suture were removed after 2 weeks. The patient was reviewed at one month, three month and six months respectively. At the end of six months there was partial resolution of the bone defect (Fig. 3 J).

CASE 4

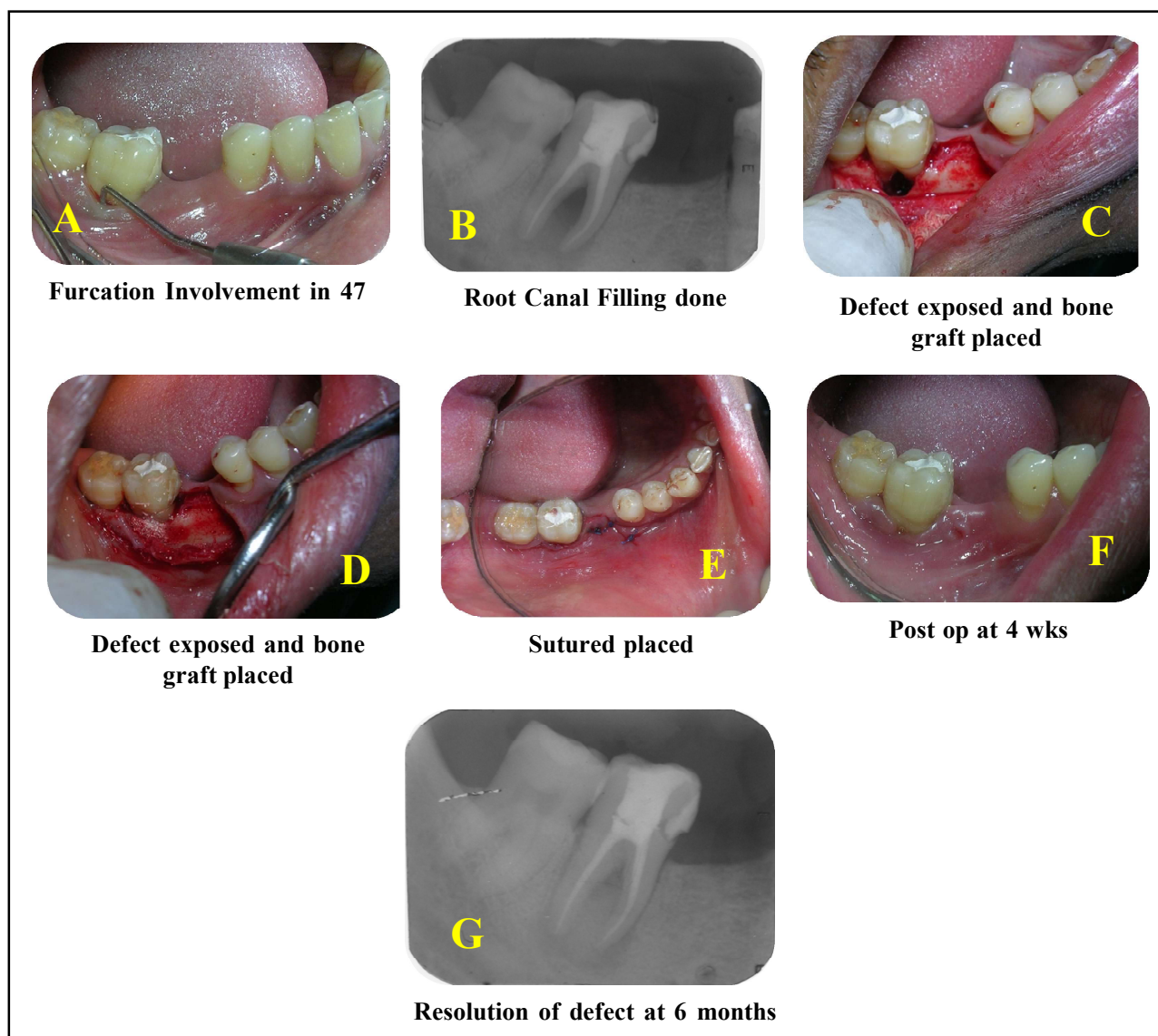
A 44-year-old male patient was referred to OPD with a chief complaint of pain in lower left back tooth region since 3 months. Patient was systemically healthy. On intra oral examination there was a temporary restoration in tooth 47, probing pocket depth was 10 mm in relation to Tooth 47 (Fig. 4 A) with Grade I mobility and the tooth was tender on percussion. Radiographic findings revealed bone loss in the furcation area. Diagnosis: Primary endodontic with secondary

periodontal lesion in relation to tooth 47. On vitality test it was found to be non-vital, endodontic treatment was done first and the patient was recalled after 4 weeks (Fig. 4 B). At four weeks the patient was asymptomatic, but the probing pocket depth was 8 mm. Periodontal flap was elevated the defect was degranulated, the defect was filled with bioglass, the flap was sutured (Fig. 4 C to F).

The patient was reviewed at one week, one month and six-month period complete closure of furcation defect was observed (Fig. 4 G). Subsequently the patient was referred to the Prosthodontist for prosthesis.

DISCUSSION

Endo-Perio lesions have shown to affect the attachment apparatus; before therapy can be instituted a determination must be made as to whether a pulpal, periodontal or combined lesion is present. The therapy

**Fig 4: A to G**

is indicated towards the removal of etiological factor responsible for tissue destruction. Depending on the etiology, the lesion may respond to endodontic or periodontal therapy alone. In some instance more complicated measures are required to treat the coexisting endo-perio lesion indicating a close relationship between them^{7,8}.

Periapical lesion respond well to endodontic therapy and repair of the attachment apparatus is predictable. Once the etiological factor is removed and root canal is adequately cleaned the result is usually predictable and complete regeneration is possible.

The most important factor to be considered when treating the combined lesion is to establish the vitality of involved tooth or teeth. The therapy depends upon the form and extent of bony destruction, location of tooth in dental arch and the vitality of pulp. Once lack of vitality

of pulp is determined root canal therapy may be instituted. The required periodontal therapy may vary from attempts at regeneration to root amputation/hemisection with endodontic therapy on remaining roots. If the tooth is vital and bony configuration meets the requirement of regeneration, then regenerative technique is indicated. If the bony topography precludes the regenerative attempt, then root amputation with root canal treatment is indicated. With the use of these combined techniques the remaining tooth may be utilized as a unit or abutment for prosthesis^{5,8}. In our first and second cases, there was severe bone loss around roots and in both the cases we went for resective therapy as periodontal regeneration was not possible due to complete bone loss around one root. In the third case we did osseous grafting and could achieve limited resolution of osseous defect.

The potential for repair of apical periodontal tissue associated with pulpal infection is greater even in face of extensive destruction, on other hand prognosis of repair of identical defect caused by periodontal disease is not predictable and is generally considered poor⁹. In our fourth case we could get complete resolution of furcation defect which was basically due to endodontic lesion.

Regeneration has been stated goal of periodontal treatment, but studies have casted doubt on exact structure of clinically healed periodontal lesion. There is little doubt that even in face of extensive destruction of periodontium, healing will restore the structure destroyed by pulpal disease with new attachment, the same is not true in case of bacterial infection of periodontal tissue. Nyman has suggested that if cells repopulating the healing wound can be restricted to connective tissue cells derived from the periodontal ligament the potential of new attachment will be greater¹⁰.

CONCLUSION

It is important to identify the etiological factor responsible for endo-perio lesions so that correct therapy can be instituted. Depending on the etiology, the lesion may respond to either endodontic or periodontal therapy alone. In certain cases when these lesions coexist a more complex treatment may be required indicating their close relationship. The prognosis of an endodontic lesion is highly predictable, but the other hand, the prognosis for repair or regeneration of periodontal tissues is questionable if associated with it. Root canal therapy should be done before periodontal procedures in the case of a primary endo and secondary periodontal

involvement; however, endodontic therapy would result only in resolution of the endodontic component of involvement and would have a little effect on the periodontal lesion. Prognosis of Primary Periodontal lesion with secondary endodontic involvement depends on outcome of periodontal therapy and is not predictable.

REFERENCES

1. **Simring M, Goldberg M.** The pulpal pocket approach: Retrograde periodontitis. *J Periodontol* 1964; 35:22- 8.
2. **Simon JH, Glick DH, Frank AL.** The relationship of endodontic-periodontic lesions. *J Endod.* 2013; 39:41-6.
3. **Rotstein I, Simon JH.** Diagnosis, prognosis and decision-making in the treatment of combined periodontal-endodontic lesions. *Periodontol* 2000. 2004; 34:165- 8.
4. **Mandel E, Machtou P, Torabinejad M.** Clinical diagnosis and treatment of endodontic and periodontal lesions. *Quintessence Int.* 1993;24:135-139.
5. American Academy of Periodontology. Guidelines for periodontal therapy. *J Periodontol* 1998; 69: 405–8.
6. **Shenoy N, Shenoy A.** Endo-perio lesions: Diagnosis and clinical considerations. *Indian Journal of Dental Research.* 2010; 21(4):579-5.
7. American Academy of Periodontology. Guidelines for periodontal therapy. *J Periodontol* 1998; 69: 405–8.
8. **Lindhe J.** Clinical periodontology and implant dentistry. 4th ed. Blackwell Munksgaard; 2003. p. 339-5.
9. **Aksel H, Serper A.** A case series associated with different kinds of endo-perio lesions. *Journal of clinical and experimental dentistry.* 2014; 6(1):91-7.
10. **Nyman S, Lindhe J, Karring T, Rylander H.** New attachment following surgical treatment of human periodontal disease. *Journal of clinical periodontology.* 1982 Aug;9(4):290-6.