

Invasive cervical root resorption: Engineering the lost tissue by regeneration

DEXTON ANTONY JOHNS, VASUNDARA YAYATHI SHIVASHANKAR, RAMESH KUMAR MAROLI, ROSAMMA JOSEPH¹

Abstract

Invasive cervical resorption (ICR) is a localized resorptive process that commences on the surface of the root below the epithelial attachment and the coronal aspect of the supporting alveolar process, namely the zone of the connective tissue attachment. Early diagnosis, elimination of the resorption and restorative management are the keys to a successful outcome. Treatment done was a combined non-surgical root canal therapy, surgical treatment to expose the resorptive defect and the resorptive defect was filled up with reverse sandwich technique and finally the bony defect filled with platelet rich fibrin (PRF), hydroxylapatite and PRF membrane. Significant bone fill was obtained in our case after a 2 year follow-up period. This case report presents a treatment strategy that might improve the healing outcomes for patients with ICR.

Keywords: Invasive cervical resorption, platelet rich fibrin, sandwich restoration

Introduction

Number of diseases comes under the category of “silent killers” as they gradually consume one without causing any serious symptoms in the early stages. Invasive cervical resorption (ICR) is symptomless until the destruction reaches the pulp. It is defined as “a localized resorptive process that commences on the surface of the root below the epithelial attachment and the coronal aspect of the supporting alveolar process, namely the zone of the connective tissue attachment”.^[1] The permanent tooth is placed in an ecological niche with active osteoblasts and osteoclasts. There is a homeostatic balance of pulp and periodontal ligament preventing clastic insults, which is due to an intact cementoblast and odontoblast cell layer.^[2] Any breach in these layers may down-regulate osteoprotegerin and up-regulate receptor activator of nuclear factor ligand causing resorption. The severance of the protective layer may be commonly due to orthodontic treatment, dental trauma, or bleaching.^[2] A study has also revealed that an interleukin-1 β polymorphism significantly increases the risk external resorption.^[3]

Heithersay^[4] classified ICR into four classes according to the extent and severity of the lesion within the tooth. Class 1-3 has a better prognosis, while Class 4 is not amenable for treatment and might require extraction and replacement with implant retained crown restoration. ICR is usually an incidental radiographic finding. The lesion classically presents as an asymmetrical radiolucency with ragged or irregular margins in the cervical region of the tooth. Early lesions might be radiolucent; however, more advanced lesions might have a mottled appearance caused by the osseous nature of the advanced lesions.^[2] Unless proper treatment is initiated, the resorption continues unabated leading to irreversible destruction of tooth structure.

Early diagnosis, elimination of the resorption and restorative management are the keys to a successful outcome. This article discusses an ICR managed by endodontic treatment, surgical management of resorptive defect by reverse sandwich technique and periodontal regeneration using platelet rich fibrin (PRF), PRF membrane and bone graft.

Case Report

A 35-year-old non-smoking male patient presented for consultation at the Endodontic Department with a chief complaint of swelling and pus discharge in the upper left canine [Figures 1a and 2a]. Patient's medical history was non-contributory. Patient reported trauma to his upper teeth when he was about 20 years old. After performing sensitivity tests, tooth #11 was diagnosed as having pulpal necrosis. Clinical examination revealed localized gingival recession and gingival enlargement in relation to tooth #11. Probing pocket depth on the mesial surface of the tooth #11 was 10 mm with a high bleeding score. Radiographic examination showed two bowl shaped radiolucency at mesial aspect of the root. Based on the clinical and radiological finding, we came to the diagnosis of localized chronic periodontitis and pulpal necrosis with external inflammatory root resorption. The treatment

Departments of Endodontics, and ¹Periodontics, Government Dental College, Calicut, Kerala, India

Correspondence: Dr. Dexton Antony Johns,
Department of Endodontics, Government Dental College,
Calicut, Kerala, India.
E-mail: dextonjohns@gmail.com

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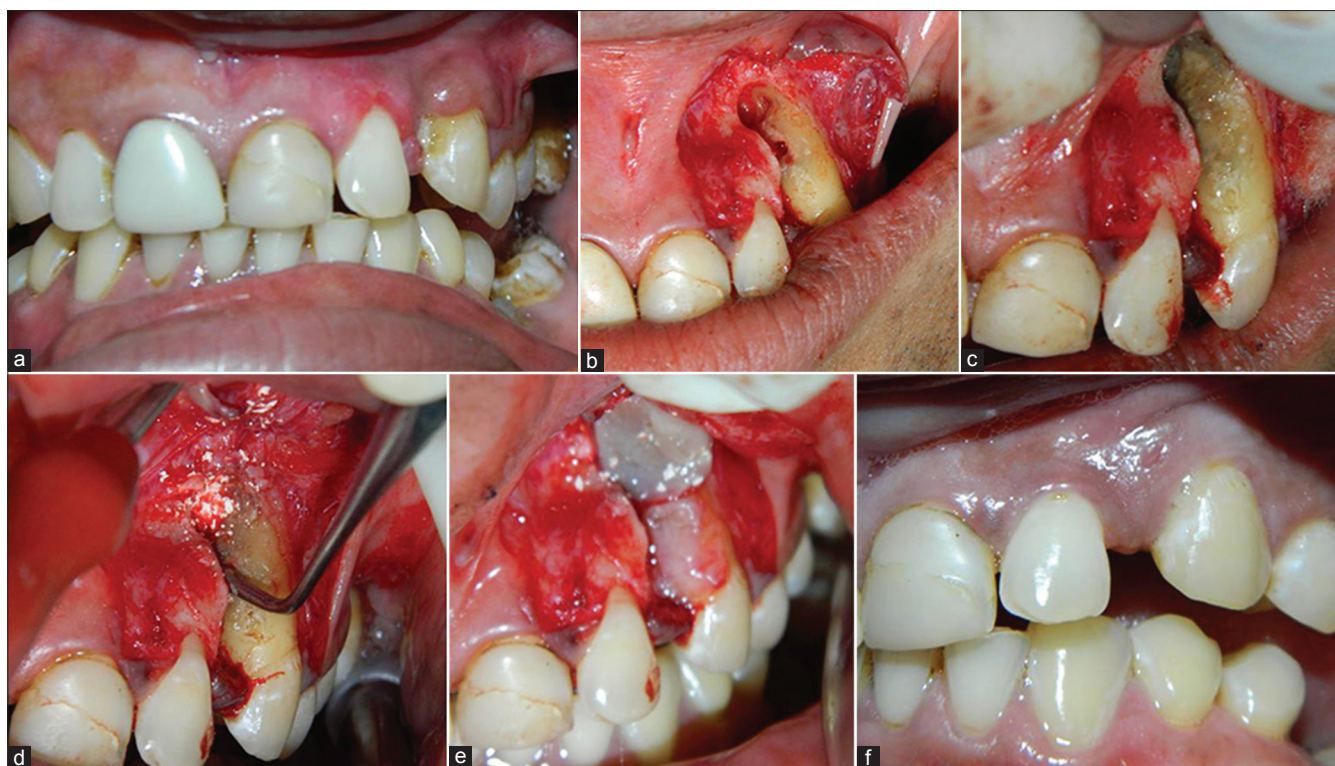


Figure 1: (a) Pre-operative clinical photograph showing swelling and pus discharge in the upper left canine. (b) Surgical exploration revealed that two lesions were present on the mesial surface of upper left canine and there was no palatal intercommunication. (c) The resorptive defects were restored with microfilled composite to a thickness of about 1 mm and light cured for 20 s. The rest of the defects were restored with Resin modified glass ionomer cement. (d) The bony defect was filled with freeze-dried bone allograft and platelet rich fibrin. (e) Autologous platelet rich fibrin membrane was placed. (f) Follow-up after 2 years shows normal probing depth, no gingival recession, and no loss of clinical attachment loss

plan was informed to the patient, which included patient motivation, oral hygiene reinforcement, scaling and root planning, endodontic procedure and lastly surgical intervention. Patient and his parents were informed of possible discomforts and potential risks concerning the planned treatment procedure and prognosis.

Treatment procedure

In phase one therapy oral prophylaxis was performed. Endodontic access cavity was prepared after placing a rubber dam and disinfecting the area with 2% of chlorhexidine digluconate (Calypso, Septodont, India). Working length was determined by using the electronic apex locator Root ZX (J. Morita MFG. Corporation, Kyoto, Japan) and file in-radiograph with 15 number K-files. The root canal was cleaned and shaped by Rotary Ni-Ti Protaper System along with GLYDE (Dentsply Maillefer Company, USA) using the crown down technique. The root canal was copiously irrigated with 2.5% sodium hypochlorite (Novo Dental Product, India). Access cavity was temporized with calcium hydroxide and temporary endodontic restorative material (TERM). Patient was recalled after 1 week; the tooth was asymptomatic. Root canal was irrigated again with normal saline and dried using paper points. Before obturation, master points were seated to test their suitability to canals and radiograph was taken. The canals were

obtained with selected master gutta-percha cone (variable taper) and AH-Plus sealer (Dentsply Maillefer Company, USA). The coronal gutta-percha cones were sheared off using heated instrument and vertical compaction was done using the heated pluggers at the canal orifices. The access cavity was restored with a composite (Clearfil Majesty; Kuraray, Osaka, Japan).

The surgical procedure was performed under local infiltration anesthesia (2% of lidocaine with 1:100,000 adrenaline) on vestibular mucosa. An intrasulcular incision was made from the distal surface of the maxillary left central incisor to the distal surface of the maxillary left canine and full-thickness mucoperiosteal flap was elevated. Surgical exploration revealed that two lesions were present on the mesial surface of upper left canine and there was no palatal intercommunication [Figure 1b]. Granulomatous tissue within the defect area was removed and 90% of trichloroacetic acid was applied to the resorptive defect for coagulation necrosis. The resorptive defects were restored with microfilled composite (Durafill VS; Heraeus Kulzer GmbH, Dormagen, Germany) to a thickness of about 1 mm and light cured for 20 s. The rest of the defects were restored with Resin modified glass ionomer cement (RMGIC) (Fuji II LC; GC Corporation, Tokyo, Japan) to a smooth finish with a cervical matrix and light cured for 20 s [Figure 1c].

Preparation of PRF membrane

A test dose of 12 ml sample of whole blood was drawn intravenously from the patient's right antecubital vein and centrifuged (REMI Model R-8c with 12 ml × 15 ml swing out head) under 3000 rpm for 10 min to obtain the PRF, which was jelly like in consistency. PRF clot started to release its serum (PRF-clot exudates) and was ready for compression into the membrane. The bony defect was filled with freeze-dried bone allograft (LifeNet, Virginia Beach, VA) and PRF [Figure 1d]. Without delay autologous PRF membrane was placed [Figure 1e] and the flaps were secured with 4-0 polyglactin 910 sutures (Vicryl, Ethicon, Inc., Piscataway, NJ). After the surgery, the patient was prescribed amoxicillin 1 g twice a day for 1 week and 0.2% of chlorhexidine mouthwash 15 ml twice a day for 2 weeks. The desired gingival contour was achieved; the patient was asymptomatic 1 week after the surgery and the sutures were removed. Patient was followed-up for 6 months, [Figure 2b] 1 year [Figure 2c] and 2 year. At 2-year follow-up period, periodontal status of related tooth demonstrated mild mobility with normal probing depth, no gingival recession and no loss of clinical attachment [Figures 1f and 2d].

Discussion

Treatment depends on the severity, location, whether the defect has perforated the root canal system and the restorability of the tooth. As smaller lesions offer the most favorable long-term outcome, endodontic treatment followed by surgical treatment was initiated in our case. The true nature of the defect could be assessed only after surgical exploration. To treat the ICR lesions, it is usually necessary, to curette away the granulomatous tissue from the adjacent periodontium to sever the blood supply to the resorbing cells and fills the defect with an inert filling material. Periodontal regeneration is the next aim, which includes reconstruction of lost or injured tissue so that the form and function of the lost structures are restored.

The removal of granulomatous tissue the surgical defect was filled by the reverse sandwich technique^[5] microfilled

composite as a liner followed by lamination with RMGIC. Microfilled resin was used because they tend to flex with the tooth rather than debond and RMGIC was biocompatible to periodontal tissues. The development of subgingival plaque over RMGIC is prevented by achieving a smooth finish using the cervical matrix.^[5]

Periodontal regeneration includes regeneration of alveolar bone, cementum, periodontal ligament and gingiva. The regenerative process is a complex biologic process in itself, requiring intricately regulated interaction between cells, signaling molecules (local and systemic) and extra cellular matrices in which these entities interact. PRF has been used as an ideal scaffold material for periodontal^[6] and pulpal regeneration.^[7] PRF is a scaffold that not only acts as carriers for growth factors and proteins, but also allow cellular infiltration and subsequent integration of the newly formed tissue within the native one. It is biocompatible, non-cytotoxic and non-immunogenic to prevent adverse effects on recruited cells and neighboring tissue.^[8] This scaffold material along with growth factors and cells pave the way to accelerate bone and periodontal regeneration. PRF can up-regulate phosphorylated extracellular signal-regulated protein kinase expression and suppress osteoclastogenesis by promoting the secretion of osteoprotegerin in osteoblasts cultures.^[9] PRF membrane has a very significant slow sustained release of key growth factors for at least 1 week and up to 28 days,^[10] which means that the membrane stimulates its environment for a significant time during wound healing. Hydroxyapatite (HA) was added to PRF in our study, this was because it has been demonstrated that the treatment of intrabony defects with PRF results in significant improvements of pocket depth, clinical attachment level and bone fill compared with baseline and secondly, that HA increases the clinical effects observed with PRF in the treatment periapical defects.^[11] Significant bone fill was obtained in our case after a 2-year follow-up period; thanks to the synergistic combination of PRF and HA.

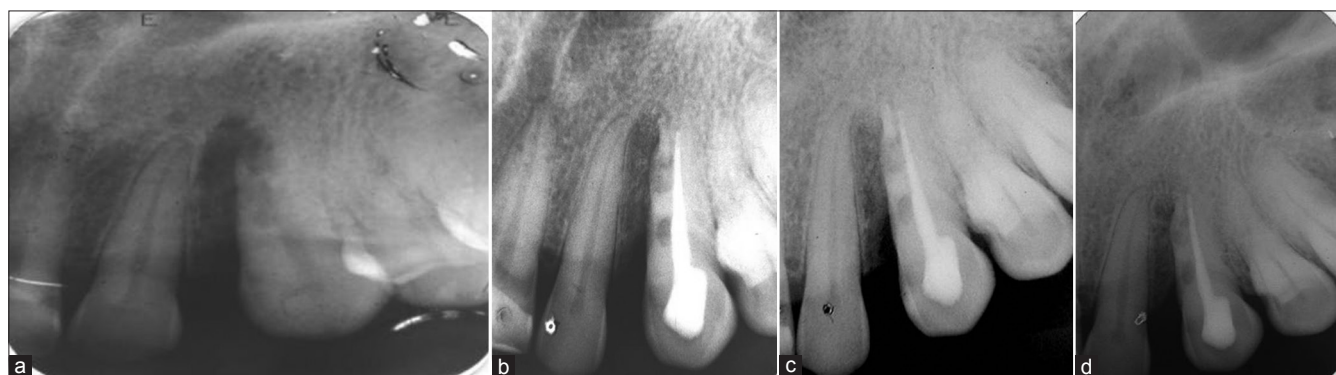


Figure 2: (a) Pre-operative intraoral periapical showing bone loss in the mesial aspect of upper left canine with two resorptive regions. (b) At 6 months follow-up. (c) At 1 year follow-up. (d) At 2 years follow-up showing significant bone fill

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