



## MANAGEMENT OF INTERNAL ROOT RESORPTION USING THERMOPLASTICIZED GUTTA-PERCHA: A CASE REPORT

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### ABSTRACT

Internal root resorption is a chronic inflammatory process initiated within the pulp space with the loss of dentin and is generally found in teeth with previous history of trauma. Since tooth is usually asymptomatic, it is important to diagnose this condition and initiate treatment as early as possible to improve the prognosis of such teeth. This paper presents a case having resorptive defect at the mid root level which was treated non-surgically with thermoplastized guttapercha technique. One year follow up demonstrated clinically asymptomatic and adequately functional tooth, with radiographic signs of healing.

### INTRODUCTION

According to the Glossary of the American Association of Endodontists, resorption is defined as a condition associated with either a physiologic or a pathologic process resulting in the loss of dentin, cementum, or bone.<sup>1</sup> Andreasen has classified it as internal (Inflammatory, Replacement) and external root resorption (Surface, Inflammatory and Replacement).<sup>2</sup> External resorption begins from the external or cervical surface of the tooth and proceeds inwards and is associated with factors like periapical pathosis, pressure from orthodontic treatment, and rapidly growing tumors. Internal resorption (IR) is a rare, insidious, resorptive pathological process, beginning in the pulpal space and extending into the surrounding dentin.<sup>3</sup>

Trauma, persistent chronic pulpitis<sup>4</sup>, heat produced during cavity cutting<sup>5</sup>, orthodontic therapy<sup>6</sup> and recently the mutation in IL-1RN gene<sup>7</sup> are suggested to be associated with internal root resorption. It is caused by transformation of normal pulp tissue into granulation tissue.<sup>8</sup> Damage to the odontoblasts lead to the compromise in the integrity of predentin and exposes the intraradicular dentin.<sup>9</sup> The osteoclasts and macrophages converted from undifferentiated cells of pulp after inflammation gets activated and causes the resorption of intraradicular dentin.<sup>10</sup>

Internal resorption is usually asymptomatic and often recognized clinically through routine full mouth radiographs. Pain may occur depending on the pulpal condition or

perforation of the root resulting in a periodontal lesion.<sup>11</sup> If the internal resorption is located in the coronal part of the tooth, clinically "pinkspot" can be observed. The pink color is related to the highly vascularized connective tissue adjacent to the resorbing cells. This color turns grey/dark grey when the pulp becomes necrotic.<sup>12</sup>

Intraoral X-ray of Internal root resorption is characterized by the radiographic appearance of an oval shape enlargement within the pulp chamber or the root canal.<sup>13</sup> The classical radiographic description of internal resorption is outlined by Gartner et al. as a clearly well-defined symmetrical radiolucency of uniform density which balloons out of the pulp chamber or root canal.<sup>14</sup> The borders are in continuity with the root canal and the defect remains stationary with change in angulations of the radiographs.<sup>15</sup>

The treatment involves endodontic therapy to remove the blood supply and arrest the resorptive process.<sup>16</sup>

Various materials available for the treatment of internal root resorption include MTA, glass ionomer cement, Super EBA, hydrophilic plastic polymer (2-hydroxyethyl methacrylate with barium salts), zinc oxide eugenol and zinc acetate cement, amalgam alloy, composite resin and thermoplastized guttapercha administered either by injection or condensation techniques.<sup>17</sup>

The aim of this work is to report the management of internal resorption in the permanent maxillary central incisor, at mid-root level.

## Case Report

A 37-year-old male patient reported to the Department of Conservative dentistry and Endodontics of SudhaRustagi College of Dental sciences and Research, Faridabad with a complaint of pain and pus discharge in relation to upper front teeth region since 2 weeks. The pain was dull, intermittent, aggravated on biting and was relieved after few hours on its own or by taking analgesics occasionally. Patient did not seek any dental treatment before. He gave history of trauma 10 years back. Medical history was non-contributory. On clinical examination pus discharging sinus was present on the labial aspect of 11, and the tooth was tender on percussion. There was no mobility in the tooth. Thermal and electrical pulp testing elicited a negative response in the right maxillary central incisor.

Radiographic examination revealed a radiolucent area in the middle one third of root canal space of right maxillary central incisor. Based on clinical and radiographic findings, the lesion was diagnosed as internal resorption, and root canal therapy was initiated after obtaining the informed consent.

The tooth was isolated under rubber dam and the access opening was made using round bur (Mani, Inc) and de – roofing was done by Endo Z bur (DentsplyMaillefer, North America). Working length was determined using an apex locator (Romipex™ A-15) and was calculated to be 23mm and reconfirmed with radiograph (Ingle's method). Apical preparation was done up to ISO size #55 K-files (Mani, Inc) followed by step back technique upto#80 K-file. Irrigation between each instrument was done using 1ml of normal saline (0.9% w/v, Lifusion™) and 1ml of 5.25% NaOCl solution (Bharat Chemical, India). The resorption defect was circumferentially filed with H-files (Mani, Inc) along with copious NaOCl irrigation to ensure complete removal of the necrotic tissue remnants. After preparation the root canal was dried and Ca(OH)<sub>2</sub> paste (Ultracal, Ultradent, Inc.) as an intracanal medicament was placed for 7 days. In the second visit Metapex paste (calcium hydroxide and iodoform, Biomed) was placed for 1 week. In the third visit, the resorptive cavity was completely free of pulpal tissue and the patient was relieved of his pain and was asymptomatic. Pulp space was obturated with thermoplastic gutta-percha and AH plus sealer (Dentsply) using E & O plus system (Meta Biomed co., Ltd, Korea). First the apical filling was done up to the resorption defect and backfilling was completed using the extruder.

At the one year follow up, patient was asymptomatic and with no clinical signs of inflammation. The follow up radiograph showed arrest of resorption process and successful healing.



Figure 1 Radiograph showing internal resorption at mid-root level



Figure 2 Radiograph showing working length

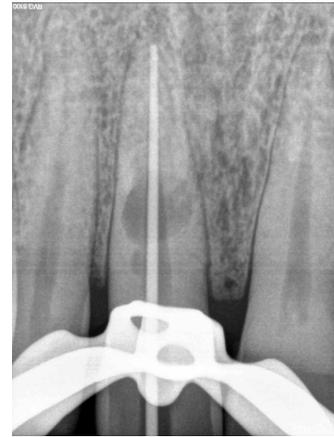


Figure 3 Radiograph showing master cone gutta-percha



Figure 4 Radiograph showing apical seal with 5mm gutta-percha

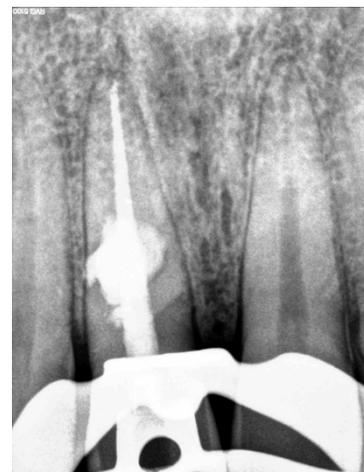


Figure 5 Radiograph showing obturation with thermoplasticized gutta-percha



Figure 6 Post-operative radiograph



Figure 7 Post-operative Radiograph at one year follow-up

## DISCUSSION

Dentin is lined internally from the pulpal surface by the odontoblastic layer and predentin. The two layers together form a barrier and prevent its resorption. Many studies have proven that, similar to the osteoclasts, the odontoclasts do not adhere to or resorb unmineralized matrix.<sup>18</sup> Wedenberg and Lindskog reported that damage to both these layers result in exposure of the underlying mineralized dentin to odontoclasts making it vulnerable to resorption.<sup>18</sup> The uncommon occurrence of dentin resorption can furthermore be explained by the dominance of odontoclast inhibitory substances such as OPG (osteoprotegerin) over activators like RANKL (receptor activator of nuclear factor kappa B ligand).<sup>19</sup>

Genetic factors have also been implicated in the development of internal resorption. The link between interleukin- (IL-) 1 gene polymorphism and root resorption has been reported in a study of monozygotic twins.<sup>21</sup>

In the present case, trauma lead to the damage to predentin and initiated inflammatory internal root resorption. The origin of clastic cells is related to the viable blood supply and the necrotic tissue acts as a stimulus for the formation of these cells. This probably explains as to why IR is stated as a rare occurrence compared to external root resorption.<sup>20</sup> The vascular changes in the pulp produce hyperaemia, causing an increased oxygen tension resulting in low pH levels, thus attracting numerous macrophages to the site, thereby piloting the onset of resorptive process. The connective tissue, following the resorptive activity, may undergo metaplasia to form granulation tissue.<sup>21</sup> Predominance of a progressing infection causes necrosis of the entire pulp tissue and limits the

resorptive process and thus acts as a protective mechanism. The presence of a collateral blood supply through an accessory canal from the periodontal ligament to the resorption site can add to maintaining the resorptive process. Internal inflammatory root resorption in its most classical forms spreads symmetrically in all directions into the dentin surrounding the pulp.<sup>19</sup> Presence of vital pulp tissue is essential for internal root resorption to take place and the pulp vitality tests generally show a positive response.<sup>16</sup> The negative vitality response can be seen after necrosis of coronal pulp or complete necrosis of pulp after active resorption process has taken place.<sup>22</sup>

The concave resorbed area demands cleaning with chemical substances that have an appropriate organic solvent capacity as it is impossible to achieve complete pulpal eradication with mechanical methods only. Sodium hypochlorite, because of its antibacterial and tissue dissolving abilities was used to remove the inflamed tissue that can't be removed with instrumentation alone. Calcium hydroxide dressing was used to control the infection in between the appointments and for disinfection of root canal.

Warm vertical condensation may cause root fracture in the weekend root structure hence in the present case, the hybrid technique was used for root canal filling where thermo plasticized guttapercha passively filled the entire resorbed defect.<sup>23</sup>

The outcome of treatment of teeth with internal root resorption depends primarily on the size of the lesion.<sup>15</sup> Therefore, access cavity preparation was kept conservative preserving as much tooth structure as possible. Large lesions cause a reduction in the resistance of the tooth to shear forces that may lead to tooth fracture.<sup>24</sup> Therefore, it is imperative to initiate endodontic treatment as soon as possible to arrest the progression of the resorptive process and to prevent root or cervical crown fracture.<sup>15</sup> Once perforation occurs, therapy becomes more difficult leading to poor prognosis. However, the present case was non-perforating one, therefore, successful management was possible with fair prognosis.

## CONCLUSION

Internal root resorption can be successfully managed by thorough debridement of root canal and disinfection by sodium hypochlorite and calcium hydroxide. Thermoplasticized guttapercha provides a good option for three dimensional obturation of a non perforating internal root resorption defect.

## References

1. Ne RF, Witherspoon DE, Gutmann JL. Tooth resorption. *Quint Int* 1999; 30(1):9-25.
2. Tronstad L. Root resorption-etiology, terminology and clinical manifestations. *Endod Dent Traumatol* 1988;4:241-52.
3. Thomas P, Pillai RK, Ramakrishnan BP. An Insight into Internal Resorption. *ISRN Dent* 2014;759326.
4. Weine FS. Endodontic therapy, 4th edn. St. Louis, MO: Mosby; 1989. p. 150.
5. Kinomoto Y, Noro T, Ebisu S. Internal root resorption associated with inadequate caries removal and orthodontic therapy. *J Endod* 2002;8:405-7.
6. Weine FS, Potashnick SR. Endodontic-orthodontic relationships. In: Weine FS, editor. Endodontic therapy, 5th edn. St. Louis, MO: Mosby; 1996:674-8.

7. Urban D and Mincik A. Monozygotic twins with idiopathic internal root resorption: A case report. *AustEndod J* 2010; 36:79–82.
8. Meire M, Moor RD. Mineral Trioxide Aggregate Repair of a Perforating Internal Resorption in a Mandibular Molar. *J Endod* 2008;34:220-3.
9. Trope M. Root Resorption due to Dental Trauma. *Endod Topics* 2002;1:79–100.
10. Singhal A, Gurtu A, Dua K. Endodontic management of internal resorptive defect in maxillary central incisor: A case report. *Annal Essen Dent* 2010;2:82-4.
11. Caliskan MK, Turkun M. Prognosis of permanent teeth with internal resorption: A clinical review. *DentTraumatol* 1997; 13(2):75–81.
12. Silveira FF, Nunes E, Soares JA, Ferreira CL, Rotstein I. Double “pink tooth” associated with extensive internal root resorption after orthodontic treatment: A case report. *Dent Traumatol* 2009; 25(3):e43-7.
13. Patel S, Dawood A, Wilson R, Horner K, Mannocci F. The detection and management of root resorption lesions using intraoral radiography and cone beam computed tomography—An in vivo investigation. *Int Endod J* 2009;42(9): 831-8.
14. Gartner AH, Mack T, Somerlott RG, Walsh LC. Differential diagnosis of internal and external root resorption. *J Endod* 1976;2(11):329–34.
15. Keinan D, Heling I, Stabholtz A, Moshonov J. Rapidly progressive internal root resorption : A case report *Dent Traumatol* 2008;24:546–9.
16. Ne RF, Witherspoon DE, Gutmann JL. Tooth resorption. *Quint Int* 1999;30:9 –25.
17. Mittal S, Kumar T, Mittal S, Sharma J. Internal root resorption: An endodontic challenge: A case series. *J Conserv Dent*. 2014;17(6):590-3.
18. Wedenberg CLindskog S. Evidence for a resorption inhibitor in dentin. *Scand J Dent Res* 1987;95(3);205–11.
19. Haapasalo M, Endal U. Internal inflammatory root resorption: the unknown resorption of the tooth. *Endod Topics* 2006;14(1):60–79.
20. Urban D, Mincik J. Monozygotic twins with idiopathic internal root resorption: A case report. *AustEndod J* 2010;36(2),79–82.
21. Sikri VK. Root resorption an enigma. *Ind J Comprehensive Dental Care* 2011;1(1);15.
22. Nigurdsson A. The role of endodontics after dental traumatic injury. In: Cohen ST, Hargreaves KM editors. *Pathways of the pulp*, 10th edn. St. Louis, MO: Mosby;2011. p.620-654.
23. Koh ET, McDonald F, Pitt Ford TR, Torabinejad M. Cellular response to mineral trioxide aggregate. *J Endod* 1998;24:543–7.
24. Friedland B, Faiella RA, Bianchi J. Use of rotational tomography for assessing internal resorption. *J Endod* 2001;27:797–9.

