REVIEW ARTICLE

# Invasive Cervical Resorption: A Review

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**ABSTRACT**

Invasive cervical resorption is a relatively uncommon form of external root resorption exhibiting no external signs. The resorptive condition is often detected by routine radiographic examination. The clinical features vary from a small defect at the gingival margin to a pink coronal discoloration of the tooth crown resulting in ultimate cavitation of the overlying enamel which is painless unless pulpal or periodontal infection supervenes. Radiographic features of lesions vary from well-delineated to irregularly bordered mottled radiolucencies, and these can be confused with dental caries. A characteristic radiopaque line generally separates the image of the lesion from that of the root canal, because the pulp remains protected by a thin layer of predentin until late in the process. Histopathologically, the lesions contain fibrovascular tissue with resorbing clastic cells adjacent to the dentin surface. More advanced lesions display fibro-osseous characteristics with deposition of ectopic bonelike calcifications both within the resorbing tissue and directly on the dentin surface.

**Key Words:** Cervical resorption, invasive resorption, roots resorption.

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## Introduction

Dental Resorption constitutes a challenge to dentistry due to the organic complexity of the process. The concern and curiosity of the subject is not recent. The oldest report about resorption of dental structures was described by Michael Blum in 1530 in a book 'The Science & Art of the Dental Surgery'. However the scientific studies of root resorption are considered to be recent.

Tooth resorption is the loss of hard dental tissue (i.e. cementum and dentin) as a result of odontoclastic action. Root resorption might be classified by its location in relation to the root surface i.e. internal or external resorption. External root resorption can be further classified into surface resorption, external Inflammatory resorption, external replacement resorption, external cervical resorption and transient apical breakdown.[1] External resorption may be physiological and pathological. External resorption can be classified as surface, inflammatory and replacement ankylosis resorption.[2]

Cervical external resorption also called as invasive cervical resorption is a clinical term used to describe a relatively uncommon, insidious and often aggressive form of external tooth resorption, which may occur in any tooth of permanent dentition. Invasive cervical resorption is defined as 'a localized resorptive process that commences on the surface of root below the epithelial attachment and the coronal aspect of the supporting alveolar process, namely the zone of the connective tissue attachment'.[3] It represents a special

type of pathological tooth condition that could be classified in the group of inflammatory resorptions.[4] Characterized by its cervical location and invasive nature, this resorptive process leads to the progressive and usually destructive loss of tooth structure. This resorbed structure is replaced by highly vascular tissue which may become visible through thin residual enamel as pinkish discoloured tooth.[5] The condition of Idiopathic Cervical Resorption was first studied by Mueller and Rony in 1930.[6] The terminology Invasive Cervical Resorption was used by Heithersay. Various terms used for invasive cervical resorption are odontoclastoma, idiopathic external resorption, fibrous dysplasia of teeth, burrowing resorption, late cervical resorption, cervical external resorption, extracanal invasive resorption, peripheral cervical resorption, supraosseous extracanal invasive resorption, peripheral inflammatory root resorption, subepithelial inflammatory resorption, periodontal infection resorption.[5]

**Etiology and Pathogenesis**

Since the Muller and Rony reported the cases, numerous causes have been documented but none of them had conclusively identified any specific etiologic agent.[6] The controversy remains among investigators about nature of the lesion. According to some this process is purely inflammatory while others think that the process is activated by sulcular microorganisms or alternatively it is benign proliferative fibro-vascular disorder or fibro-osseous disorder in which micro-organisms have pathogenic role but may become secondary invaders.[5] Cervical external resorption starts just beneath the epithelial attachment. The location is always related to the marginal tissues and pocket depth. Hence, some suggest that there may be changes in the composition and environment of marginal tissues which makes it less resistant to resorption.[4] From embryogenesis, dentin is protected by enamel organ, enamel, Hertwig's epithelial root sheath, cementoblasts, cementum and intermediate cementum.[7,8] They act as barrier which has to be broken to induce osteoclastic activity.[9,10] Dentin always remain protected against immune system during the development of the natural tolerance. When exposed to the immune system, a cascade of events takes place to eliminate this "Non-self" components.[7,8] After starting from its location the advancing ICR lesion characteristically stops short of root canal and pulp almost at the level of predentin. Then, the resorption extends in a circumferential and apico-coronal direction around the root canal. The exposure of pulp is prevented by the thin predentin layer.[11] The exact reason behind this is not known but studies have shown that the predentin contains an anti-invasion factor and resorption inhibitor that prevent ICR from reaching into root canal. In vitro studies by Wedenberg et al regarding the ability of macrophage spreading concluded that an inhibitor of macrophage spreading is present in organic, non-collagenous component of dentin. This inhibitor may be responsible for resistance of predentin and dentin to resorption.[12] Thus, in order for dental resorption to occur, three conditions are necessary, blood supply-for recruitment of mononuclear cells, absence of protective layer (cementum form surface and predentin from interior). After reaching predentin, lesions if extends coronally will result in cavitation of the overlying enamel. When the lesion extends in apical direction a series of channels containing active resorptive tissues are present, connecting further apically with periodontal ligament.[11]

These changes may be induced by various predisposing factors which basically includes either physical or chemical trauma

**A) Physical**[13]

a) Orthodontic treatment
b) Segmental Orthognathic Surgery[14]
c) Transplanted teeth
d) Trauma
e) Bruxism
f) Guided tissue regeneration

**B) Chemical Agents**

a) Intracoronal bleaching
b) Secondary bone grafting in unilateral complete cleft palate patient.
c) Tetracycline conditioning of root

Pressure can be the possible etiologic factor as many cases are attributed to orthodontic tooth movement, tumors, cysts and impacted teeth. In such cases, resorption tends to cease as soon as source of pressure

is removed. Also, the excessive forces in the cervical region of tooth might result in tissue necrosis adjacent to exposed root dentin which result in stimulation of mononuclear precursor cells to different into odontoclasts, which are attracted to exposed root dentin and results in resorption.[11] Mandibular molars might be rendered prone to resorption as a result of placement of orthodontic bands that might damage the vulnerable cementoenamel region of tooth.[15] Still the basic question is why the resorptive process begins years after the initiating insult? Various researchers have proposed that it could be due to the change in the composition of cementum that makes less resistant to resorption which can be induced by minor injury later.[16,17]

Trauma could be one of the predisposing factors for ICR. According to Heitherssay, 15.1% of teeth have trauma as a major predisposing factor and when the other contributing factors are associated this increases upto 25.7%. After trauma generally the incidents which can be contributory to ICR development are bleaching due to non-vitality, orthodontic treatment, forcible repositions after trauma, trauma to cementoenamel junction region due to interdental wiring.[18]

Various reports have shown that the intracoronal bleaching results in ICR.[18,19,20,21] Reported intracoronal bleaching as a sole predisposing factor in 3.9% and along with other predisposing factor in 13.6%. Several mechanisms given for this are- hydrogen peroxide leaking through dentinal tubule on to external tooth surface which might denature dentin and provoke immunological response.[20,22] Acidic environment of root surface resulting from bleaching paste might enhance the osteoclastic activity leads to ICR.[23,24] The combination of heat, 30 H2O2 and lack of cervical barrier are related to resorption.[25] In case of walking bleaching sodium perborate when mixed with water forms H2O2 in low levels. In vivo studies have demonstrated that the H2O2 penetration can be upto 82% of total amount through the artificial defects in the cementum.[26] Thus, this unimpeded passage of H2O2 in the surrounding periodontal tissue can result in resorption.

Any surgical procedure that result in defect or damage to cementoenamel junction region can be a predisposing factor for ICR.[11] These include teeth adjacent to disimpaction, transplantation, surgical exposure of teeth for orthodontic purposes.[15] The orthognathic surgeries especially segmentalization of maxilla are also been considered as a predisposing factor for ICR. The exact association is uncertain but heat damage to bone, impairment of blood supply are important factors associated with root resorption. Apart from these, individual's genetic propensity, systemic factors, nutrition, age and sex can be the associated factors.[27]

According to Heitherssay 1999 periodontal surgeries which might result in damage to cementum can result in resorption in 1.6% of cases. Usually, the resorption is prevented after periodontal debridement as the contact of connective tissue cells with the root surface is prevented which also prevents inflammatory process.[3] But the cases of resorption have been reported after tetracycline root conditioning and after regenerative periodontal procedures.[28] Reason could be due to the damage to the root surfaces not protected by junctional epithelium are repopulated by connective tissue cells. The increased migration of connective tissue fibroblasts can enhance the risk of resorption.[28]

Other miscellaneous factors are developmental defects like hypoplasia or hypomineralization of cementum, bruxism.[11]

Various systemic factors have also been reported to be associated with ICR though the association is still not confirmed. Moskow et al reported a case of ICR in patient with hyperoxaluria and oxalosis. Increased concentration of oxalates caused by kidney failure results in precipitation of crystals in pulp, bone marrow, gingival connective tissue, periodontal ligament. These crystalline deposits provoked a granulomatous foreign body reaction which might have resulted in aggressive ICR.[29] Neely and Gordon have reported multiple ICR in father and son which describes the possible familial pattern of idiopathic ICR. Hence the close relatives should be examined for the similar lesions for early diagnosis and treatment.[30] Liang et al reported five patients of ICR that were associated with hormonal abnormalities; although, it is still unclear whether these abnormalities were initiating or contributing factors. The periodontal tissues are sensitive to hormonal fluctuations during puberty and pregnancy but still there is no clear

evidence of role of hormonal changes in pathogenesis of ICR.[31]

Recently, the ICR's have also been related with viral etiology. Thomes et al studied the relationship of multiple ICR (mICR) and neutralization tests for feline Herpes Virus Type I. This virus is associated with the felin odontoclastic resorptive lesions in domestic cats. They have advised future studies to find out the exact and possible role of feline virus in mICR.[32] Kjar et al made an attempt to explain the regional association of ICR when multiple lesions are seen. He reported cases with idiopathic ICR running regionally. In these patients anamnestic information revealed severe viral diseases during childhood. This neuronal virus spreading along the nerve paths could be possible explanation for the unexpected resorptions. The extent of resorption process in the dentition followed the virus infected nerve paths and resorption process stopped reaching regions that were innervated differently and not infected by virus.[33]

So it is not possible to find out the exact etiology of ICR rather it is considered as multifactorial resulting from variety of predisposing factors which ultimately leads to an aggressive lesion of ICR.

Heithersay G.S. (Table 1) has proposed a clinical classification of invasive cervical resorption depending on the amount of destruction.[11]

invasion of pulp by the lesion.[34] Hence, the chances of majority of lesions detected in the later stages i.e. class 3 type of lesions are more. Clinically, the lesion may present as painless pinkish discoloration of the crown indicating resorptive process.[5] Pulp vitality tests are within normal limits unless deep resorptive cavity due to proximity to pulp results in sensitivity to temperature. The reason for this is that the resorption starts on the root surface, but when the predentin is reached, it proceeds laterally and in an apical and coronal direction, progressively enveloping the root canal.[4] The pinkish discoloration is due to deep red, granulomatous tissue showing through the thin translucent enamel. Probing the cavity results in bleeding and gives spongy texture of granulomatous tissue. At times, the resorption cavity can also give feeling of hard, mineralized tissue.[13] This is accompanied by sharp, knife edged cavity borders these two features are important in the differential diagnosis with root caries. Carious lesions are softer due to disintegration of organic component of dentin.[4] carious lesions are generally associated with gingival recession.[31] Radiographically, the lesion shows radiopaque mineralized outline of canal through the radiolucency of the external resorptive defect. If the lesion is long standing, then mottled appearance may be seen due to deposition of calcified reparative tissue

**Table 1: Clinical Classification of Invasive Cervical Resorption**

| | |
|---|---|
| Class 1 | a small invasive resorptive lesion near the cervical area with shallow penetration into dentin |
| Class 2 | a well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into the radicular dentin |
| Class 3 | a deeper invasion of dentin by resorbing tissue, not only involving the coronal dentin but also extending into the coronal third of the root |
| Class 4 | a large, invasive resorptive process that has extended beyond the coronal third of the root |

**Clinical Presentation**

The extent of resorptive process dictates the clinical manifestations of the lesion. Clinically the lesion can be asymptomatic and could be an incidental finding on routine dental radiographic examination. The lesion poses diagnostic difficulties as it may be insidious in onset without any visual signs and symptoms.[5] The lesions may become symptomatic when the pulpal or periodontal infection supervenes secondarily on

within areas of cavity surface.[35] The lesion can be misdiagnosed as an internal resorption on radiograph which can be prevented by taking radiographs of varying angulations. If the lesion is internal resorption the radiolucency will remain static; if lesion is ICR then the lesion will move according to Clarks rule or SLOB rule.[36] Multiple lesions can occur so the full mouth radiography should be done to rule out the multiple lesions.[5] In severe cases if resorptive process extends in

coronal direction ultimately results in cavitation of tooth. If resorptive process extends in radicular dentin it forms canals containing resorptive tissue which have connections further apically with periodontal ligament.[11]

**Histopathology**

Microscopically this lesion is similar to any other inflammatory root resorption. The resorptive cavity contains granulomatous fibrovascular tissue. It consists of mass of fibrous tissue, inflammatory cell infiltrate consisting of lymphocytes, plasma cells, histiocytes, macrophages, numerous blood vessels and clastic resorbing cells adjacent to the dentin surface. A thin layer of dentin and predentin is always present separating inflammation free pulp tissue. The resorbing tissue is also free of acute inflammatory cells which rules out the possibility of infection as a primary etiology. Clastic resorbing cells are attached to dentinal surface in resorption bays or Howship's lacunae and are derived from blood macrophages.In advanced lesions ectopic calcification can also be observed within the invading fibrous tissue and on the surface of resorbed dentin.[35] This calcified poorly organised bonelike tissue indicates attempt of healing of the resorbed tooth surface.[11]

**Treatment**

The Incisive cervical root resorption begin and progress asymptomatically. When multiple teeth are involved resorption may not occur simultaneously or progress at same rate. Separate lesions can begin at different time. The basic treatment modalities are directed towards the complete removal of resorptive tissue with spoon shaped excavator or with bur at slow speed. Once sound dentinal margins are achieved dentinal walls are conditioned by some chelating agent to destroy remnants of resorptive tissue. Then defect might be restored by appropriate restorative material like glass-inomer cement, composite resin[35], amalgam[25] or MTA[37] etc. MTA is generally used if defect is in radicular region because it is most biologically acceptable material and periodontal re-attachment can be expected on it but it can be polished. Amalgam and composite resins are generally used in defects in coronal region as they can be polished well but not biologically acceptable in periodontal tissues.

If defect is inaccessible in oral cavity but present in cervical third of tooth then orthodontic extrusion of tooth or apically positioned flap can be used but this might gives esthetically compromised results.[5] An alternative treatment option is intentional replantation of tooth. This should be attempted only when no other treatment modality is feasible. When resorptive portal of entry is within the bone and cannot be accessible, the affected tooth is endodontically treated, extracted, the defect is debrided, restored and then replanted into its socket.[38]

Endodontic treatment may be necessary with some class 2 and class 3 lesions where defect is very close to pulp and remaining thin dentin layer is at risk of perforation during removal of granulation tissue.[39]

Thus teeth with cervical resorption fall into difficult category in achieving predictable outcome. It may be challenging for even experienced practitioners. This review will present information necessary to aid practitioners in proper diagnosis and clinical management of this type of teeth.[5]

**References**

1. Patel SS, Pitt Ford T. Is the resorption external or internal? Dent Update 2007;34:218-29.
2. Andeasen JO. External root resorption: its implications in dental traumatology, pedodontics, periodontics, orthodontics and endodontics. Int J Endod 1985;8:109-18.
3. Tronstad L. Root resorption: etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241-52.
4. Bergmans L, Van Cleynenbreugel J, Verbeken E, Wevers M, Van Meerbeek B, Lambrechts P. Cervical external root resorption in vital teeth: X-ray microfocus-tomographical and histopathological case study. J Clin Periodontal 2002;29:580-85.
5. Heithersay G. Invasive cervical resorption. Endod Topics 2004;7:73-92.
6. Mueller E, Rony HR. Laboratory studies of an unusual case of resorption. J Am Dent Assoc 1930;17:326-34.

7. Hidalgo MM, Itano EN, Consolaro A. Humoral immune response of patients with dental trauma and consequent replacement resorption. Dental Traumatol 2005;21:218-21.
8. Ng KT, King GJ, Courts FJ. Humoral immune response to active root resorption with a murine model. Am J Orthod Dentofac Orthop 1990;98:456-62.
9. Lindskog S, Pierce A, Blomlof L, Hammarstrom LE. The role of the necrotic periodontal membrane in cementum resorption and ankylosis. Endod Dent Traumatol 1985;1:96-101.
10. Chambers TJ. Phagocytic recognition of bone by macrophages. J Pathol 1981;135:1-7.
11. Heithersay GS. Clinical, radiologic and histopathologic features of invasive cervical resorption. Quintessence Int 1999;30:27–37.
12. Wedenberg C, Lindskog S. Evidence for a resorption inhibitor in dentin. Scand J Dent Res 1987;95:270-1.
13. Royzenblat LA, Tordik CP, Goodell CG. Cervical Resorption. Clin Update 2005;27(6):1-2.
14. Hokett SD, Hoen MM. Inflammatory cervical root resorption following segmental orthognathic survey: a case report. J Periodontol 1998;69:219-26.
15. Patel S, Kanagasingam S, Pitts Ford T. External Cervical Resorption: A Review. J Endod 2009;35(5):616-25.
16. Coyle M, Toner M, Barry H. Multiple teeth showing invasive cervical resorption–an entity with little known histologic features. J Oral Pathol Med 2006;35:55-7.
17. Jensen AL. External invasive resorption in a three-rooted lower first molar. Aust Endod J 2006;32:70-4.
18. Heithersay GS. Invasive cervical resorption following trauma. Aust Endod J 1999;25:79–85.
19. Goon WW, Cohen S, Borer RF. External cervical root resorption following bleaching. J Endod 1986;12:414–8.
20. Cvek M, Lindvall AM. External root resorption following bleaching of pulpless teeth with oxygen peroxide. Endod Dent Traumatol 1985;1:56–60.
21. Friedman S, Rotstein I, Libfield H, Stabholz A, Heling I. Incidence of external root resorption and esthetic results in 58 bleached pulpless teeth. Endod Dent Traumatol 1988;4:23–6.
22. Lado AE, Stanley HR, Weisman MI. Cervical resorption in bleached teeth. Oral Surg Oral Med Oral Pathol 1983;55:78–80.
23. Kehoe JC. pH reversal following in vitro bleaching of pulpless teeth. J Endod 1987; 13:6–9.
24. Mc Cormick JE, Weine FS, Maggio JD. Tissue pH of developing periapical lesions in dogs. J Endod 1983;9:47–51.
25. Frank AL. External-internal progressive resorption and its non-surgical correction. J Endod 1981;7:473–6.
26. Trope M. Root resorption due to dental trauma. Endod Topics 2002;1:79–100.
27. O'Ryan F, Silva A. Complications with maxillary orthognathic surgery. Sel Read Oral Maxillofac Surg 2008;16(5):1-36.
28. George G, Darbar U, Thomas G. inflmaatory external root resorption following surgical treatment for intrabony defects: a report of two cases. J Clin Periodontal 2006;33:449-54.
29. Moskow BS. Periodontal manifestations of hyperoxalouria and oxalosis. J Periodontol 1989;60:271–8.
30. Neely AL, Gordon SC. A familial pattern of multiple idiopathic cervical root resorption in a father and son: a 22-year follow-up. J Periodontol 2007;78:367–71.
31. Liang H, EJ Burkes, NL Frederiksen. Systematic review: Multiple idiopathic cervical root resorption: systematic review and report of four cases. Dentomaxillofac Radiol 2003;32:150–5.
32. von Arx T, Schawalder P, Ackermann M, Bosshardt DD. Human and Feline Invasive Cervical Resorptions: The Missing Link?—Presentation of Four Cases. J Endod 2009;35:904-13
33. Kjaer I, Strøm C, Worsaae N. Case Report Regional Aggressive Root Resorption Caused by Neuronal Virus Infection. Case Rep Dent 2012; doi:10.1155/2012/693240.
34. Umer F, Adnan S, Khan FR. Conservative Management of Invasive Cervical Resorption: A Case Report. J Dent Traumatol 2013;10(3):289-95.

35. Goldman HM. Spontaneous intermittent resorption of the teeth. J Am Dent Assoc 1954;49:522–32.
36. Seward GR. Periodontal disease and resorption of teeth. British Dent J 1963;34:443–49.
37. Koh ET, Torabinejad M, Pitt Ford TR, Brady K. MTA Stimulates a Biological Response in Human Osteoblasts. J Biomed Mater Res 1997;37:432-9.
38. Frank AL, Torabinejad M. Diagnosis and Treatment Of Extra- canal Invasive Resorption. J Endod 1998;24(7):500-4.
39. Kim Y, Lee CY, Kim E, Roh BD. Invasive cervical root resorption: treatment challenges. Restor Dent Endod 2012;37(4):228-31.

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