IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 13, Issue 5 Ver. IV. (May. 2014), PP 38-43
www.iosrjournals.org

Calcified Canals – A Review

[1]Bernice Thomas, [2]ManojChandak, [3]Adityavardhan Patidar, [4]Bharat Deosarkar, [5]Harshit Kothari

[1]*(Post Graduate Student, Dept. of Conservative Dentistry and Endodontics, SharadPawar Dental College,Sawangi(Meghe), Wardha*
[2]*(Prof and Head, Dept. of Conservative Dentistry and Endodontics, SharadPawar Dental College,Sawangi(Meghe), Wardha*
[3]*(Post Graduate Student, Dept. of Conservative Dentistry and Endodontics, SharadPawar Dental College,Sawangi(Meghe), Wardha*
[4]*(Post Graduate Student, Dept. of Conservative Dentistry and Endodontics, SharadPawar Dental College,Sawangi(Meghe), Wardha*
[5]*(Post Graduate Student, Dept. of Conservative Dentistry and Endodontics, SharadPawar Dental College,Sawangi(Meghe), Wardha*

Abstract: *Dental traumatic injuries may lead to several clinical complications. One of them is the pulp canal obliteration, which presents itself as an uncontrolled hard tissue deposition along the pulp chamber and root canal walls. Over time, it may partially or completely obliterate the pulp canal space. In an effort to treat a tooth in that condition, the endodontist faces a great challenge that might end up in an accident such as root perforation. This article aims to make a brief review on the causes, mode of development and management of calcified canals.*

I. Introduction:

Uncontrolled mineralization due to failure of enzyme, pyrophosphatase, reduced capillary permeability and reduced blood supply leads to calcifications. Root canal in teeth in which calcific deposits have blocked access to the canal (s), treatment efforts are often thwarted. An effort to locate the residual canal may remove large amounts of dentin and there is a risk of perforating or fracturing the root[1].

II. Review Of Literature:

Hard tissue apposition along the root canal walls is a slow, normally occurring physiological aging process. After dental trauma, the rate of hard tissue deposition may seem to be uncontrolled, resulting in Pulp Canal Obliteration (PCO)[2].

Traumatic injury to the dentition may result in pulpal necrosis, internal resorption, invasive cervical resorption, surface, inflammatory or replacement resorption[3].

The clinical signs of PCO subsequent to a traumatic injury are often a yellowish discoloration and a progressive decrease in the response to thermal and electrical pulp testing when comparing to adjacent teeth[4].

Bakland and Andreasen wrote that pulpal responses to traumatic injuries are affected by the degree of injury to the neurovascular supply, which for the most part enter through the apical foramen and also by the presence of bacteria as a significant factor in the outcome. Andreasen suggested that three possible outcomes exist: pulpal healing, pulpal necrosis, or pulp canal obliteration. They noted that all three responses can result at different times, for example, initial healing may be followed by PCO and subsequent pulpal necrosis.

Patterson and Mitchell recommended either endodontic treatment or extraction of the tooth. They reasoned that the pulp tissue involved should be regarded as a potential focus of infection and should be removed[5].

Holcomb and Gregory followed 34 patients with 41 teeth with PCO over a 4 year period and found that 7.3% developed a periapical rarefaction[6].

In a 3.4 year follow-up study by Andreasen, 7% of the patients developed pulpal necrosis following PCO[7].
In a later study, Andreasen suggested that the frequency of pulpal necrosis may increase over time[8].

III. Calcification Of The Pulp Can Occur Due To

1. Mineralization in response to various irritants
2. Aging.

Tubular / Dentinal Sclerosis:

Milder or moderately irritating agents produce tubular sclerosis. It is a condition of the primary dentinal tubules where they are ultimately occluded.[2] Sclerotic dentin, on the other hand, may be considered a defense mechanism of the pulp dentinal complex because its formation alters the permeability of the tubules, blocking the access of irritants to the pulp[9].

Certain drugs such as calcium hydroxide and corticosteroids, when placed on the dentin after cavity preparation have also been demonstrated by studies to cause sclerosis. Apparently, some remineralisation also occurs when sedative dressings, such as zinc oxide eugenol, are placed in carious cavities[9].

Secondary Dentin

Continuous deposition of the dentin, which tends to reduce the volume of the pulp takes place throughout life. Schour has claimed that 4 microns of secondary dentin is deposited daily. The deposition of dentin is slow and gradual but does increase after the age of 35-40 years. There is no firm evidence that systemic conditions such as arteriosclerosis leads to a higher incidence in pulpal calcifications[10].

In molars, deposition is seen greatest on the pulpal floor, to alesser extent on the roof, and least on the side- walls. Therefore, with age, pulp chambers decrease significantly in height i.e. in occlusoradicular direction but not extensively in width i.e. in mesiodistal direction[9,11].

As a result, the horns of the pulp in molars are often left behind. They also recede, but not as much as the rest of the pulp tissue[9].

In upper anterior teeth, the greatest dentin deposition occurs on the lingual wall of the pulp chambers, as a result of masticating forces with subsequent deposition in the incisal tip and floor of the pulp chamber.

Reparative Dentin

Localized secondary dentin laid down in areas of focal injury in response to various irritants is more haphazardly organized and is termed as reparativesecondary (irregular, irritation or tertiary) dentin[12].

In humans, the average rate of reparative dentin formation has been reported to be 2.8 microns for deciduous teeth and 1.5 microns for permanent teeth. Operations on the dentin, with resultant damage to the involved odontoblasts, cause temporary derangement in mineralization shown by the formation of a basophilic line. This has been termed as a calciotraumatic reaction. The presence of abundant amounts of reparative dentin does not appear to be correlated with pulp- test readings[9].

Retrogressive Pulp Changes

These altered pulp conditions are

1. Atrophy and fibrosis
2. Dystrophic calcification (calcific metamorphosis)[12]

P.E. Lovdahl and J.L. Gutmann have summarized pulpal responses to irritation as follows[13]:

1. Rapid death with canal patency.
2. Irritational response with pulpal demise
3. Extensive irritational response and pulp system exposure.

IV. Calcifications Of Pulp

Traditionally, calcified bodies in the dental pulp have been classified on the basis of their structural characteristics. The classification by Kronfield is most commonly used[10].

1. "true" denticles (composed of tubular (ortho)dentin),
2. "false" denticles (composed of concentric layersof calcified material not resembling dentin),
3. “diffuse calcifications” (small calcified deposits scattered throughout the pulp tissue).

Firstly, one may easily, but incorrectly, assume that the histological properties of these calcified bodies are related to their mode of development (i.e. true denticles develop as the result of epithelio-mesenchymal interactions, while false denticles form on a calcified nidus). Asshown below, such a strict relationship does not exist.

Secondly, most pulpal calcifications are conglomerates of different tissues: orthodentin, regular, and irregular calcified material, so that a strict classification becomes nearly impossible[14-16].

1. Orthodentin, tubular dentin, may be detected both in denticles and in pulp stones.

a. Denticles, formed as the result of epithelio-mesenchymal interactions, are composed of tubular dentin in the earliest stage of their development [17,18]. Odontoblasts line the periphery of the denticles, but as the diameters of these calcified bodies increase, most or all of the odontoblasts become reduced in height and apparently perish

soon thereafter. The lumina of their dentinal tubules undergo sclerosis and are no longer evident light microscopically[18].

b. Pulp stones are developed initially as an amorphous calcified nidus, but they may acquire peripheral masses of orthodentin (complete with predentin border and odontoblasts) as they increase in dimension[19,14, 20]. It has been suggested that all dental papilla cells initially undergo an induction by odontogenic epithelium and that for a limited period of time they remain capable of responding to an appropriate challenge--such as the presence of a pulp stone--by differentiating into odontoblasts and producing dentin[20]. Thus, the presence of "true dentin" might not necessarily be an indication of direct epithelio-mesenchymal interactions, although it frequently is. It could equally be an indication that healthy young pulp tissue has responded to an appropriate stimulus.

2. Regular calcified material may be found in the peripheral

Pulp stones increase in size by gradual depositionof layers of regular calcified material on the surface of the irregular calcified nidal core. The most striking feature of the resulting calcified body is that it appears laminated light microscopically. A laminated pulp stone is composed of layers of concentrically arranged collagen fibers and electron-dense interfibrillar material into which hydroxyapatite mineral has been deposited [21,22].The regular structure of this material as well as the presence of an uncalcified peripheral border which gradually becomes calcified suggest that it has been deposited by some kind of hard tissue-forming cell. However, thus far such cells have not been identified. The tissue has been described as non tubular fibrodentin ("osteodentin'). It resembles dentin formed in older teeth, particularly dentin found near the root apex. Similar layers of non tubular fibrodentin are also frequently found on the surface of any denticle that remains free in the pulp tissue rather than becoming attached or embedded, as is more usual.

Thus, the deposition of regular layers of non tubular fibrodentin may just reflect the particular ability of a somewhat older pulp tissue to respond to the challenge which the presence of a pulp stone or denticle represents.

3. Irregular calcified material

These may be found at the core of most pulp stones but also occasionally on the surface of a laminated pulp stone or even on the surface of a denticle.

Diffuse calcifications are composed entirely of this material. Irregular calcified deposits consist of an irregular matrix of collagen fibers and electron-dense inter fibrillar material into which hydroxyapatite crystallites have been deposited. The collagen fibers appear to be part of the normal intercellular matrix of the pulp tissue. These calcified bodies, which may become quite large, have an irregular periphery. They grow by addition of mineral to adjacent matrix fibers[21, 23, 24].

Calcific Metamorphosis

Calcific metamorphosis is defined as a pulpal response to trauma that is characterized by deposition of hard tissue within the root canal space and is commonly found in young adults in the anterior region of the mouth[14].

V. Possible Modes Of Development

Generally, two different modes of development of pulpal calcifications have been proposed:

a) Initial calcification of isolated pulp tissue components; and
b) Epithelio-mesenchymal interactions.

A) Calcification Of Tissue Components

Many researchers considered only a single mechanism: the initial calcification of a component of pulp tissue (ground substance, necrotic cell remnants, collagen fibril), which is believed to serve as a nidus upon which calcified material is eventually deposited, either in a concentric lamellar or in a radial fashion[25, 14]. Bab et al. and Appleton et al. have provided ultrastructural descriptions of this process[21, 26].

Several researchers' suggests that a substantial number of pulpal calcifications develops in similar manner, even in preeruptive teeth[18]. This mode of development is mostly involved in the formation of all of the calcified structures in the coronal pulp and all of the calcified structures that form either as part of normal age-related changes or as the result of local pathological changes of the pulp tissue (coronal and radicular).

In any connective tissue various calcification promotors and inhibitors are present.Calcification may occur in a normally noncalcifying tissue,such as the pulp, when the balance between the two is disturbed;for example, by the local breakdown of inhibitors (e.g.proteoglycan complexes).

Sundell et al. have stated that vascular damage following trauma or a "local metabolic dysfunction" is the precipitating factor for the development of a nidus[25].There exists a close spatial relationship between calcified structures and the blood vessels and/or nerves of the pulp [27, 25, 28].

Occasionally these nidi (or foci) are located inside vascular lumina or in neurovascular bundles, mostly in the pulps of deciduous teeth during shedding. While in the latter the calcificationis probably associated with the degeneration of the nerves themselves, in most other instances the observed close spatial relationship between calcifications and neurovascular bundles may be coincidental due to the richness of the neurovascular supply of the pulp[29] which makes it nearly impossible for the calcified structure not to be close to a neurovascular bundle.

Certain investigators have suggested that pulpal calcifications may be associated with certain systemic conditions such as arteriosclerosis. In a radiographic study, Stafne et al. reported the incidence of calcifications in pulp chambers was correlated with the documented existence of some non dental systemic conditions[30]. While the incidence in normal individuals was 46%, it was somewhat, but not significantly, higher in patients with arteriosclerosis (53%), osteitis deformans (55.7%), and acromegaly(57.1%).

B) Epithelio-Mesenchymal Interactions

This mode of development has been suggested for a second, much smaller group of calcified bodies in the dental pulp. Baume et al.[14] reported this theory of development suggests that epithelial strands are detached from the enamel organ during tooth development. Later, these strands become isolated in the dental papilla where they interact with the papilla mesenchyme, resulting in the physiologically normal differentiation of odontoblasts around the strands.

Detached epithelial strands or cell nests have been observed in the papilla mesenchyme[32,33] and studies[31, 17, 18] have shown what can be interpreted to be developmental stages of the resulting calcified bodies. Typically, an epithelial strand is found at the core of each of these bodies during the initial stages of development. Odontoblasts deposit dentin as they move away from this epithelium. The resulting structures are initially hollow, not solid. They may be thimble-shaped, with their concavities facing apically, or approximately spherical, with a central cavity surrounded on all sides by dentin.

Since, in addition, these structures are initially composed of true dentin; the use of the term denticles (small teeth) for them is most suited. These distinctively shaped denticles are found only in those locations where a root sheath is present[31, 17, 18] or in the furcation areas of multirooted teeth where epithelial extensions subdivide the cervical opening of the enamel organ.

VI. Management

A) Orifice Recognition

An important fact to remember is that the canal space in normal root canal anatomy is always in the cross- sectional center of the root. Similarly the pulp chamber is (or was, before calcification) located in the cross sectional center of the crown[13]**.** In a tooth with a calcified pulp chamber, the distance from the occlusal surface to the projected pulp chamber is measured from the preoperative periradicular film, or preferably from a bite-wing film, which maximizes accuracy. They applied the buccal object rule for the determination of calcified root canals as follows:

After the initial access opening, the bur is left in place and three radiographs are taken:

1. Straight – on to the bucco- lingual dimension to determine the position of the head of the bur in the root canal in the mesio- distal dimension
2. Radiograph taken with a 20^0 horizontal angulation with the cone shifted distally.
3. Radiograph taken with a 20^0 horizontal angulation with the cone directed mesially.

The last two radiographs give information regarding the relation of the bur to the canal lumen in the bucco- lingual dimension[34]. Krasner and Rankow have given certain laws which are particularly useful in locating calcified canal orifices. These are:

1. Law of symmetry 1: Except for maxillary molars, the orifices of the canal are equidistant from a line drawn in a mesiodistal direction through the pulp- chamber floor.
2. Law of symmetry 2: Except for maxillary molars, the orifices of the canals lie on a line perpendicular to a line drawn in a mesiodistal direction across the center of the floor of the pulp chamber.
3. Law of color change: The color of the pulp chamber is always darker than the walls.
4. Law of orifices location 1: The orifices of the root canals are always located at the junction of the walls and the floor.
5. Law of orifices location 2: The orifices of the root canals are always located at the angles in the floor- wall junction.
6. Law of orifices location 3: The orifices of the root canals are located at the terminus of the root development fusion lines.

The LN bur (Caulk/ Denstply, Tulsa, OK, USA), the Mueller bur (Brasseler, Savannah, GA, USA) and thin ultrasonic tips are especially useful for locating calcified canals. Another important instrument for orifice location is the DG-16 explorer. At this point a fine instrument, usually a No. 8 or No. 10 K –file is placed into the orifice, and an attempt is made to negotiate the canal. An alternative option is to use instruments with reduced flute, such as a Canal Pathfinder (JS Dental, Ridgefield. Conn.) or instruments with greater shaft strength such as the Pathfinder CS (Kerr Manufacturing Co.), which are more likely to penetrate even highly calcified canals. Remove the cervical ledge or bulge. If the orifice still cannot be negotiated with a fine instrument, drill 1-2 mm into the center of the orifice with a No.2 round bur on slow speed and use the explorer to re-establish the canal orifice. When counter- sinking or troughing in an area where an orifice is located, be sure the pulp chamber is dry. The bur rotating at a slow speed will remove whitish chips that then accumulate in the orifice. After a light stream of air is blown into the chamber, these chips appear as white spots on the dark floor of the chamber and serve as markers for exploration or further countersinking. This approach can be used if the fourth canal of the maxillary molar or a separation of the mesio-buccal and mesiolingual canals is anticipated in mandibular second molars. Recently a combination of access refinement ultrasonic tips and magnification has revolutionized the basic concept of access cavity preparation. The uncovering of the floor of the pulp chamber can be accomplished with the help of the CPR 2D or BUC 1 tips. The pulp stones sometimes can be vibrated or teased out by the CPR 2D or BUC 1 tips; at other times, they can be planed with the help of a BUC 2 tip- a process similar to planning the root surface. Grind the floor until the dark- colored dentine becomes visible. It is of critical importance because it dictates and guides the extension of access cavity[35]**.** Locating canals and initial penetration under the microscope is also aided by fine instruments like the Micro-Orifice Opener (DenstplyMaillefer, Ballaigues, Switzerland.).

B) Biomechanical Preparation

Coronal flaring in a crown- down fashion is preferred. Incremental instrumentation is achieved by creating new increments between the established widths by cutting off a portion of the file tip, thus making it slightly wider in diameter. For example, if a 1 mm segment is clipped from a size 10 file, the instrument becomes a size 12, by trimming sizes 15, 20 and 25, instruments of sizes 17, 22 and 27 respectively can be created. In extremely sclerotic canals, only 0.5 mm segments are trimmed, increasing the instrument width by 0.01mm and making a size 10 into a size 11, etc. because cutting the shaft imparts a flat tip, a metal nail file is used to smooth the end and reestablish a bevel after the removal of any segment[12]**.**

C) Chelating Agents-Is There Any Role

Chelator preparations have been advocated frequently as adjuncts for root canal preparation, especially in narrow and calcified root canals. Apical dentin is more frequently sclerosed, and is more mineralized. Several authors recommend liquid EDTA solution be introduced into the pulp chamber (pipette, cotton pellet) to identify the entrance to calcified canals[36]**.**

VII. Conclusion:

In the treatment of calcified canals it is common to find a total occlusion of the canal space at any level[13]**.** Smith reviewed the literature concerned with calcific metamorphosis and collected the following data:

- Calcified teeth that were not treated endodontically developed radiographic or clinical symptoms in upto 16% of the cases.
- Studies of the success and failure of conventional endodontic therapy reported failure rates of 10% to 19%.

When these failure rates are compared, it appears that a calcified tooth treated endodontically would have no better chance of success than if it were left alone[37].

VIII. References:

[1]. Selden S. The role of dental operating microscope in improved nonsurgical treatment of "calcified" canals. Oral Surg Oral Med Oral Pathol 68:93-8, 1989
[2]. (Ngeow WC & Thong YL. 1998. Gaining access through a calcified pulp chamber: a clinical challenge. IntEndod J, 31: 367–71.)
[3]. The Editorial Board of the Journal of Endodontics. 2008. Traumatic injuries: an online study guide. J Endod, May; 5S.
[4]. Schindler WG &Gullickson DC. 1988. Rationale for the management of calcific metamorphosis secondary to traumatic injuries. J Endod, Aug; 14(8):408–12.
[5]. Patterson SS & Mitchell DR. 1965. Calcific metamorphosis of the dental pulp. Oral Surg Oral Med Oral Pathol, 20: 94–101.
[6]. Holcomb JB & Gregory WB. 1967. Calcific metamorphosis of the pulp: Its incidence and treatment. Oral Surg Oral Med Oral Pathol, 24: 825–30.
[7]. Andreasen JO. 1970. Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up study of 189 injured teeth. Scand J Dent Res, 78:273–86.

[8]. Andreasen FM, Yu Z, Thomsen BL & Andersen PK. 1987. Occurrence of pulp canal obliteration after luxation injuries in the permanent dentition.Endod Dent Traumatol, 3: 103–5.
[9]. Seltzer S, Bender.I.B. The Dental Pulp .Third edition 2000.MDMI inc.Schafer.Textbook of oral pathology.
[10]. Moss- Salentijn L, Hendricks- Klyvert M. Calcified structures in human dental pulps. Journal of Endodontics 14(4):184-189, 1988.
[11]. Neville .Oral and Maxillofacial Pathology. Second edition 2002. Saunders.
[12]. Weine F. Endodontic Therapy. Fifth edition.1996. Mosby.
[13]. Gutmann J.L, Dumsha T.C, Lovdahl P.E, Hovland E.J. Problem Solving in Endodontics: prevention, identification and management. Third edition.1997 Mosby.
[14]. Baume LJ. The biology of pulp and dentine.A historic, terminologictaxonomic, histologic-biochemical, embryonic and clinical survey.Monographsin oral science.Vol. 8. Basel: S Karger, 1980:174-8.
[15]. Miller WA. Pulp calcifications in a taurodont tooth. Br Dent J 1969;126:456-9.
[16]. Weinreb M, Michaeli Y. Possible mechanisms of induction of dentinogenesis. Med Hypotheses 1984;13:163-9.
[17]. Stenvik A, Mlor IA. Epithelial remnants and denticle formation in the human dental pulp.ActaOdontolScand 1970;28:721-8.
[18]. Moss-Salentijn L, Klyvert MH. Epithelially induced denticles in the pulps of recently erupted, noncarious human premolars. J Endedon 1983;9:554-60.
[19]. Johnson PL, Bevelander G. Histogenesis and histochemistry of pulpal calcification. J Dent Res 1956;35:714-22.
[20]. Weinreb M, Michaeli Y. Possible mechanisms of induction of dentinogenesis. Med Hypotheses 1984;13:163-9.
[21]. Bab I, Lustmann J, Azaz B, Gazit D, Garfunkel A. Calcification of noncollagenous matrix in human gingiva: a light and electron microscopic study. JOral Pathol 1985; 14:573-80.
[22]. Harrop JT, Mackay B. Electron microscopic observations on healing dental pulp in the rat. Arch Oral Bio11968;13:365-85.
[23]. Plackova A, Vahl J. Ultrastructure of mineralizations in the human pulp. Caries Res 1974;8:172-80.
[24]. Aoba T, Ebisu S, Yagi T. A study of the mineral phase of pulp calcification. J Oral Patho11980;9:129-36.
[25]. Sundell JR, Stanley HR, White CL The relationship of coronal pulp stone formation to experimental operative procedures. Oral Surg 1968;25:579- 89.
[26]. Appleton J, Williams MJR. Ultrastructural observations on the calcification of human dental pulp.Calcif Tissue Res 1973;11:222-37.
[27]. Willman W. Numerical incidence of calcification in human pulps [Abstract]. J Dent Res 1934;1:160-1.
[28]. 28. Stella A, Sisco RG, Hernandez E, Osorio S, Di Piramo S. Contribucion al estudio de lasformacionescalcicas del organopulpar y periodontal. Origen, evotucion y significacionclinica. An FacOdontol 1966;12:55-68.
[29]. Johnson PL, Bevelander G. Histogenesis and histochemistry of pulpal calcification. J Dent Res 1956;35:714-22.
[30]. Stafne EC, Szabo SE.The significance of pulp nodules. Dent Cosmos 1933;75:160-4.
[31]. Stenvik A. Pulp and dentin reactions to experimental tooth intrusion [Academic Thesis]. Oslo: University of Oslo, 1969.
[32]. Fridrichovsky H. ZurHistologie der Dentikel. Z Stomato11927;25:124- 57.
[33]. Orban B. Epithelial rests in the teeth and their supporting structures. Proc Am Assoc Dent Schools 1929;121:133.
[34]. Khabbaz MG, Serefoglou MH. The application of buccal object rule for the determination of calcified root canals.International Endodontic Journal 29:284-287, 1996.
[35]. Iqbal M K. Nonsurgical ultrasonic endodontic instruments. Dent Clin N Am 48 :19- 34. 2004.
[36]. Hulsmann M, Heckendorff M, Lennon A. Chelating agents in root canal treatment: mode of action and indications for their use. International Endodontic Journal, 36, 10- 830,2 003
[37]. Amir F, Gutmann JL, Witherspoon D.E. Calcific metamorphosis: A challenge in endodontic treatment and diagnosis. Quintessence International 32:447-455, 2001.