Pulp Canal Calcification as a Sequalae of Dental Trauma; a review.

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ABSTRACT

Pulp canal calcification is a frequent sequalae of dental trauma and follows the natural process of aging. In its presence, pulp canal calcification may pose a dilemma in the less astute clinicians with regards to the prognosis and management. This article describes the incidence, presentation, mechanism, management and prognosis of pulp canal calcification. A thorough understanding of this clinical finding is not only important in avoiding misdiagnosis and subsequent overtreatment, but also for the prevention of iatrogenic errors when endodontic intervention is warranted.

Keywords:
Pulp canal calcification, pulp canal obliteration, calcific metamorphosis
INTRODUCTION

Pulpal calcification refers to hard tissue apposition along root canal walls as part of a normal physiological process\(^1,2\) and defensive response\(^3\). This response, however, can be considerably accelerated under certain conditions such as dental trauma\(^4-7\), orthodontic therapy\(^8,9\) and autotransplantation\(^10\).

Pulpal calcification arising from dental trauma is commonly known in the dental literature as pulp canal obliteration (PCO),\(^5,11-13\) or calcific metamorphosis (CM)\(^14-18\). The reason for the interchangeable use of both terms has not been well elucidated in the literature. However, the American Association of Endodontists Glossary of Endodontic Terms\(^19\) defines CM as “a pulpal response to trauma characterized by rapid deposition of hard tissue within the canal space” whereas PCO was defined as “radiographic evidence of increased dentin production primarily in response to trauma”. In essence, CM and PCO are considered as a sequela to trauma, with the former describing the physiological process, while the latter represents the radiographic signs of the process.

The dental literature is further divided with the dichotomization of PCO into partial pulp canal obliteration (PPCO) and total pulp canal obliteration (TPCO). Several studies\(^5,7,10,13,15\) assessed presence of PCO based on these two classifications, whereas the remainder did not ascertain the level of calcification in their assessment \(^4,6,11,20\). However, one study in isolation\(^21\) assessed level of pulp canal obliteration based on a different classification\(^22\). In this system, the obliteration of the pulp of tooth in question was compared with the non-injured contralateral tooth and deemed as either low, moderate and advanced. While this allows the authors to rule out degree of pulp calcification attributed to physiological phenomenon (i.e: aging), the use of this classification was not observed in further studies possibly due to the difficulty of obtaining consistent records of the contralateral teeth.

Despite the heterogeneity of the studies with regard to the classification of PCO, Yu and Abbott\(^23\) highlighted the glaring flaw of the term pulp canal obliteration, citing that “obliteration is not ideal since this term implies that there is no canal remaining” and recommended the term “calcification” instead. In fact, the term “obliteration” is defined as “to remove from existence: destroy utterly all trace, indication, or significance of”\(^24\), and in the context of a pulp canal obliteration, this would indicate the complete occlusion of the canal, which is in contrast with the findings from another study\(^25\). In the study, the authors measured canal diameters of teeth with pulp canal calcifications radiographically and compared the readings to the gold standard i.e: histological sections. They found that in spite of the radiographic absence of canal, all the sections of the root
demonstrated a canal histologically. This study is in concordance with the microscopic observations in another study\textsuperscript{14} which reported persistence of patent canals in tooth that displayed canal calcification radiographically.

Similarly, in a histological examination of the pulps of 20 maxillary permanent incisors with radiographic evidence of reduced pulp spaces, narrowing of entire pulp lumen in 18 teeth, and narrowing of apical half of root in two teeth were observed\textsuperscript{26}. Therefore, despite the radiographic appearance of an obliterated pulp space, the possibility that the canal may only be several cells wide, and therefore not be evident radiographically cannot be ruled out unless confirmed by histological sectioning. However, because the bulk of the dental literature has referred to pulp canal calcification and calcific metamorphosis as pulp canal obliteration, this article will continue to adopt the use of this term so as to not confuse the reader or amend the original content of the articles.

**ETIOLOGY AND INCIDENCE**

The incidence of PCO varies between 9-24% in the literature\textsuperscript{4,21,27}. Andreasen et al\textsuperscript{4} reported an incidence of PCO in 42 out of 189 (22.2%) luxated permanent teeth over a mean observation period of 3.4 years. Similarly, Oikarinen et al\textsuperscript{21} reported presence of PCO in 42 of 172 teeth (24%) with luxation injuries followed up over a mean period of 22 months. However, a larger data pool brought the percentage down to 15% of 637 luxated permanent teeth over a similar mean observation period in another study\textsuperscript{12}. PCO was found to be the second most frequent outcome (35%), after pulp necrosis, in 55 maxillary incisors following extrusion\textsuperscript{28}.

Pulp canal calcification also appears to follow transient apical breakdown (TAB). Andreasen\textsuperscript{29} reported that 22 out of 27 (81.5%) teeth demonstrating TAB were followed by pulp canal obliteration. Partial or complete obliteration of the pulp canal also presents a common finding after root fractures\textsuperscript{30}. In clinical studies of root-fractured permanent incisors, pulp canal obliteration was found in 69–73% teeth\textsuperscript{31,32}. In contrast, a lower number of PCO in root fractured teeth has been reported, with 21 out of 49 (42%) teeth exhibiting partial or complete PCO in the apical fragment\textsuperscript{27}.
**CLINICAL PRESENTATION**

**Color**

Teeth with pulpal obliteration typically present with yellow discolouration and reduction in transparency of the crown. Jacobsen and Kerekes\(^5\) reported reduced transparency or yellowish discoloration in 78.7% (96/122) of teeth with radiographic appearance of PCO, with only six being severely discoloured. The mild degree of discoloration in the bulk of the sample is reflected by only a small number of patients (n=4) who were bothered by the appearance. Similarly, Robertson et al\(^13\) found that the majority (69%) of the teeth in their study exhibited yellow discoloration, while 3 out of 82 (2.5%) teeth demonstrated greyish discoloration. This is in concordance with a more recent study which reported yellow discoloration in 67% of the teeth (186 of 276 teeth), and greyish discoloration in further 12% of the teeth\(^7\). Although a higher percentage of teeth with grey discoloration developed periapical lesions than teeth with yellow discoloration, the difference was not statistically significant. Therefore, the authors advised against relying on presence of greyish discoloration as a definite indication of pulpal breakdown.

While yellow discoloration appears common following PCO, it is important to note that not all teeth with PCO demonstrated any discoloration. In fact, Andreasen\(^4\) reported only 21% (9 of 42) teeth displayed crown discoloration, whilst a later study reported one and two teeth exhibiting grey and yellow discoloration respectively in the total of 96 teeth that developed partial PCO\(^6\). This further highlights the lack of diagnostic value of crown discoloration in teeth with PCO.

**Pulp sensibility**

The assessment of pulpal condition in teeth displaying PCO is riddled by uncertainty from the perspective that threshold values for electric pulp test (EPT) often elicit various and reversible response\(^6\). Such conflicting findings provide difficulties in interpretation because they may or may not imply pulp necrosis. In fact, 27% (22/82 teeth) of teeth with PCO showed no response to EPT after an average follow up of 16 years\(^13\), while only 32.1% (18/56) teeth with PCO and normal colour responded normally to EPT\(^7\). This further demonstrates that sensibility tests are unreliable in the presence of PCO. Although, Andreasen et al\(^12\) noted that teeth that elicited negative pulp sensibility response at the time of injury was significantly more likely to develop PCO after concussion and subluxation injury.
A general trend of increasing threshold to thermal and electric pulp stimuli with PCO progression has also been observed\(^7,14\). This trend becomes more obvious in cases with total PCO observed in long observation periods (10-23 years)\(^11\), and possibly explains why Andreasen et al\(^12\) reported only 1 out of 9 teeth with total PCO did not respond to sensibility testing at the final examination.

A difference between EPT response in total and partial PCO has also been observed, whereby more teeth with partial PCO responded positively to EPT than complete PCO at follow up\(^5\). This similar trend is also reported by Oginni et al\(^7\) with a statistically significant difference. Based on these various observations, it is therefore vital that clinicians exercise judicious interpretation of investigations results to avoid making the error of interpreting absence of pulp response to EPT as absolute pulp necrosis.

**Radiographic**

PCO presents radiographically as either partial or total obliteration of the pulp canal space. The literature in general agrees that partial PCO is represented by visible, but reduced contours of pulp space, whereas teeth with no visible pulp space represents total PCO\(^13\). However, Oikarinen et al\(^21\) deviated from the convention and defined degree of pulp canal obliteration based on a system that was used to describe degree of pulp canal calcification following autotransplantation\(^22\). The reason for this choice was not made clear by the authors. This creates heterogeneity in the study, rendering it difficult for inclusion in future systematic reviews, perhaps explaining why no future studies have followed suit in adopting this assessment method.

Nonetheless, it must be borne in mind that although radiographic and histological often overall correlate\(^25\), this is not always the case. A non-discriminable outline of the pulp space radiographically does not imply complete absence of the pulp canal space, as endodontic intervention often reveals pulp canal space with pulp tissue in these cases\(^18,33,34\).

**MECHANISM**

The exact mechanism of pulp canal obliteration is unknown, but is linked to damaged neurovascular supply of the pulp at the time of the injury\(^13,35\). Andreasen\(^30\) proposed that PCO occurs via two possible mechanisms: i.e: a reflection of the neural or neurovascular repair in the pulpal tissue, or as an aftermath of intrapulpal hemorrhage following displacement injury. Results from several animal studies\(^36-38\) suggest that PCO may be an uncontrolled
sympathetic nervous response to trauma, subsequent to the loss of parasympathetic inhibition. Diminished blood flow in pulpal vessels observed during sympathetic nerve stimulation could lead to pulpal respiratory depression, ultimately leading to a pathological calcification. While the evidence for sympathetic vasoconstrictor nerves in the pulp is robust, the role of blood-borne mediators in intrapulpal vasodilation remains a subject of controversy. Nonetheless, the previous hypothesis that intrapulpal vasodilation is controlled by these blood-borne substances formed the basis of the assumption that inhibitory control of odontoblastic secretion, and therefore dentine formation could only be re-initiated after pulpal revascularization is complete.

As PCO is observed more frequently in teeth that has undergone luxation injuries with moderate degree of displacement (extrusion and lateral luxation) as opposed to those with no displacement (concussion and subluxation), the link of PCO to intrapulpal hemorrhage following displacement cannot be dismissed. Following rupture of the blood vessels in a severe luxation injury, apical pathosis may occur in presence of bacteria. However, development of apical pathosis is unlikely in the first three years post-trauma, and often manifests as a late complication after some uneventful years. The percentage of traumatised teeth that eventually developed periapical pathosis as defined by a negative response to electrical pulp test and confirmed with presence of periapical radiolucency has been reported to be at approximately 27% (75/276 teeth).

PCO can be clinically recognised as early as three months post-injury but is usually undetected until the first year after injury, and is significantly related to three factors: type of injury, stages of root development, and type of fixation.

**Type of Injury**

The association between PCO and type of luxation injuries appear to reflect a similar trend in that, PCO more frequent in teeth following moderate displacement such as extrusion and lateral luxation as opposed to those with mild displacement (concussion and subluxation). This suggests that PCO is elicited by a certain magnitude of injury, and that a more severe displacement injury predisposes the teeth to pulp necrosis.

In a retrospective study of 55 maxillary incisors with history of extrusion, Lee et al reported that the degree of extrusion was significantly associated with the development of PCO. Teeth that sustained mild extrusion were nearly three
times as likely to be diagnosed with PCO than teeth with severe extrusions. In a mild extrusion scenario, the degree of damage to the vascular supply was deemed likely not to be adequate to induce pulp necrosis.

**Stages of Root Development**

PCO has also been found to be significantly associated to the stages of root development\(^6,13,21,27\). Andreasen et al\(^6\) reported that PCO was dependent upon the stage of root development at the time of injury, with PCO seen less frequently after luxation of teeth with closed apices than open apices. This is in concordance with other studies\(^6,13,21,27\). Hecova et al\(^27\) reported that the effects of stages of root development were statistically significant following luxation and extrusion injuries. This observed trend reflects the importance of vital pulp for development of PCO.

**Type of Fixation**

While Andreasen et al\(^6\) found that there was no significant difference in the frequency of PCO between different types of resin splint, the historically used orthodontic bands/resin type of splint appeared to have resulted in significant increase of PCO in luxated teeth. Rock and Grundy\(^20\) also reported that splint placement did not significantly prevent occurrence of pulp calcifications in luxated permanent teeth. However, the type of splint used in the study was not mentioned. The adverse effect of the orthodontic bands/resin type of fixation is presumably due to the added trauma inflicted during manipulation of the teeth during fixation. As this type of fixation is no longer in use, the effect of this type of splint has not been further discussed in the literature.

**HISTOLOGY**

Lundberg and Cvek\(^34\) histologically examined the pulp tissue of 20 endodontically treated maxillary incisors that were subjected to dental trauma. 18 teeth exhibited narrowing of the entire pulpal lumen, and two teeth demonstrated narrowing only in the apical half of the root. The pulp tissue varied from being rich in cells with only a slight increase in collagen content, to being rich in collagen with a marked decrease in the number of cells. Microorganisms were not detected in any of the 20 specimens, whilst a moderate inflammatory reaction was only identified in only one pulp.

Robertson et al\(^48\) found dentine and bone like tissue, along with fibrotic tissues in the pulpal lumen of 123 traumatized primary teeth. 50% of the traumatised teeth had pulp tissue with a normal appearance, the pulp remnants looked normal in most cases even in the presence of obliterating hard tissue. No correlation could be made between the various tissue observed and the clinical
diagnosis. As there is no similar study in the literature that assessed permanent teeth instead, it is not known if the pulp calcification process seen in primary teeth is comparable to that in the permanent successors.

Piattelli and Trisi observed presence of two types of calcific tissue in light microscopic assessment of a tooth with radiographic evidence of complete pulp obliteration and found two types of calcific tissue in the pulp lumen; reparative dentine, and calcified tissue with no tubular pattern and with a concentric layer structure. However, the tooth studied was a permanent molar with caries, and all teeth in this particular patient exhibited similar pattern of radiographically obliterated pulp chamber. A possible systemic predisposing factor to the pulp calcification cannot be ruled out in this particular case, and the significant difference between pulp calcification of this etiology and trauma is unknown.

Fonseca et al presented a morphohistometric evaluation of two permanent teeth that presented with calcific metamorphosis with a history of trauma. They found that the tissue occluding the coronal pulp demonstrated three different patterns of collagen structures resembling collagenous connective tissue, cortical bone and cancellous bone. They concluded that the differing patterns of the tissue represent different stages of an abnormal collagen organization and calcification.

**MANAGEMENT**

Conflicting views regarding management of PCO exists in the literature with earlier studies advocating endodontic intervention. This was based on the rationale that traumatically induced pulp calcified teeth present a potential focus for infection, and that when endodontic intervention is indicated, guidance afforded by the pulpal space will be lost, rendering treatment more difficult. For that reason, teeth with partial and total PCO have been categorised in moderate and high difficulty groups respectively in the Endodontic Case Difficulty Assessment form.

The lack of agreement regarding decision making of teeth with pulp calcification amongst clinicians has been demonstrated in a survey. 147 endodontists and 112 general dentists who participated in a questionnaire were provided with a case of calcific metamorphosis of two maxillary central incisors, which were vital and asymptomatic six months following trauma. Results of the survey indicated that the endodontist cohort was split with regard to their choice of treatment, with 54.1% opting to do nothing, and 45.9% favouring endodontic treatment. Surprisingly, the general dentists had a more uniform consensus with 85.7% choosing not to intervene. More alarmingly, the result of this study
highlights the apparent discord amongst the supposed specialists in the discipline.

The emergence of more follow-up studies, however, sheds more light on the management of teeth with pulp calcification. As only 1-27% of teeth with PCO will develop pulp necrosis\textsuperscript{4,5,6,11,13}, the management has moved away from prophylactic endodontic treatment on routine basis. It is no longer deemed justified to initiate endodontic treatment in absence of radiographic and clinical signs of periapical pathosis.

While there is no published protocol regarding decision making of teeth with pulp calcification, McCabe and Dummer\textsuperscript{53} suggested a treatment decision flowchart based on clinical and radiographic indications and aesthetic concern. This flowchart merely provides guidance to the clinician and should not be heralded as an ultimatum. The importance of clinical prudence is highlighted in light of the flow chart’s suggestion of partial coverage restoration of teeth with discolouration despite endodontic intervention.

In a study that investigated correlation between radiographic appearance of the periapical tissue of teeth with PCO for treatment decision making, Oginni et al\textsuperscript{7} found that all teeth that were non responsive to sensibility testing with a periapical lesion (PAI 3 or more) were tender to percussion. Based on this observation, the authors recommended endodontic intervention in this group of teeth. In contrast, the monitoring approach of teeth with PCO was recommended in absence of clinical and radiographic evidence of pathosis.

In the event that endodontic treatment in a tooth with PCO is indicated\textsuperscript{54}, the challenge of identifying the obliterated pulp canal space presents a concern for any clinicians. Cvek et al\textsuperscript{26} reported that technical failures during endodontic treatment of teeth with calcified canals were high (20%), with an even higher failure rate in mandibular incisors (71%). The failures reported in this study include file fracture, root perforation and inability to locate the canal. Similarly, Kvinnsland et al\textsuperscript{55} indicated that 20% of perforation detected in the study were due to failed attempts to locate calcified canals. Unsurprisingly, endodontic treatment was performed without the aid of a dental operating microscope in these studies.

As the accurate determination of the canal orifice location proves a daunting task for the clinician despite histological evidence of a persistent narrow root canal in a tooth with PCO\textsuperscript{56}, the use of a dental operating microscope is therefore strongly recommended\textsuperscript{57}. Should the repercussion of a misguided access to the canal space involves perforation, this may ultimately result in tooth loss\textsuperscript{58,59}.
As such, a novel method that employs three-dimensional (3D) printing technology to produce a template to plan for minimal access to the root canal has been presented in 2015\textsuperscript{60}. ‘Guided Endodontics’ utilizes data from CBCT and intra-oral scans to design and fabricate templates with corresponding sleeves that would subsequently indicate when to stop drilling. This technique has reported a low mean angle deviation (1.59 - 1.81°) and high accuracy regardless of the operator difference\textsuperscript{60,61}. Since then, this technique has gained traction in the endodontic literature, with various published studies\textsuperscript{62-69}. Guided endodontics has demonstrated its superior performance compared with conventional method of access cavity preparation in terms of amount of tooth structure removal, success of canal detection and treatment duration\textsuperscript{70}. However, despite the promising outcomes of these measured parameters, Guided Endodontics are not without their limitations. This includes limited accessibility to posterior teeth, as a straight path to the apical target area is required\textsuperscript{60}. In addition, issues regarding dentinal microcrack formation\textsuperscript{63} and increase in temperature\textsuperscript{64} have been raised as has the presence of beam hardening artifacts in CBCT images\textsuperscript{71} which may pose some difficulty during alignment of the scans. Perhaps more importantly, the cost factor of this technique may ultimately hinder the patient or the clinician from pursuing this treatment approach.

**PROGNOSIS**

The assessment of pulp status post-trauma presents an issue to the attending clinicians due to difficulties of interpreting EPT results which may or may not yield a response. For that reason, incidence of pulp necrosis in post-traumatic teeth have been reported separately. In a long-term observation study\textsuperscript{13}, the authors acknowledged this difficulty by reporting pulp survival based on radiographic examination and EPT respectively. However, this study demonstrated a downward trend of pulp survival as measured by both radiographs and EPT, subsequently reporting an increasing trend of pulp necrosis with time. At the end of the 20-year observation period, an additional 20 teeth became pulp necrosed to the original 66 teeth that were diagnosed within the same year of trauma incident.

Studies that investigated prognosis of endodontically-treated teeth after PCO has reported a success rate of between 62.5 - 97.9\%\textsuperscript{26,54}. Cvek et al\textsuperscript{26} found that 80\% of the teeth re-examined 4 years after endodontic treatment were free from radiographic signs of periapical disease. Whereas Hasselgren\textsuperscript{72} reported that the success rate of endodontic treatment of roots with radiographic evidence of pulp canal calcification reduces from 97.9\% to 62.5\% when pre-
operative periapical radiolucency is present. However, this study was riddled with several flaws. The inclusion criteria for teeth undergoing endodontic treatment merely included radiographic evidence of pulp obliteration with complete disregard of neither presence of symptoms nor response to EPT. Thus, suggesting that in some teeth, perhaps no root canal treatment may have been indicated after all. In addition, the methodology only vaguely described the endodontic procedure, but failed to mention not only the percentage of roots where canals could not be negotiated, but also the consequent management. The authors followed this with a glaringly obvious statement that the lower success rate of roots with periapical lesion was likely due to presence of intracanal bacteria.

There are no other studies that has assessed the outcome of endodontic treatment of teeth with pulpal obliteration, but it is reasonable to conclude that where indicated, endodontic treatment of teeth with pulpal obliteration has a reasonable prognosis once a technically adequate treatment has been carried out with minimal technical complications.

SUMMARY

Pulp canal calcification is a sequela of dental trauma and is reported to develop in 9-35% of traumatically injured teeth. Despite conflicting opinions on the management of pulps exhibiting canal obliteration, the reported incidence of pulpal necrosis in these teeth of between 1-27% should lend some credence to decision making. Although endodontic treatment of these cases is considered challenging, in presence of the skilled hands and the appropriate armamentarium, the endodontic prognosis should not differ from that of a tooth in absence of pulp calcification. However, as histological examination in pulps of teeth with pulp calcification has shown no signs of inflammation where clinical and radiographic signs of diseases was absent, it would seem reasonable that endodontic intervention is only initiated in teeth with sound evidence of pulp necrosis.
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