

# Pulp Canal Obliteration: A Review

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## ABSTRACT

Pulp canal obliteration (PCO) is a condition characterized by the pronounced deposition of hard tissue along the internal walls of the root canal that fills most of the pulp system leaving it narrowed and restricted. Teeth with PCO usually present with a yellowish or greyish discoloration. Different factors have been attributed to cause PCO. However, the exact pathogenesis of pulp obliteration still remains unclear. When the tooth is asymptomatic, the clinician is usually confronted with a dilemma of whether endodontic treatment should be initiated in such teeth or it should be delayed until clinical symptoms or radiographic signs of periapical disease develop. Endodontic treatment of teeth with PCO is often challenging. The possibility of iatrogenic errors during conventional approach using ultrasonics under dental operating microscope (DOM) are relatively high. Guided endodontics has recently gained popularity as a successful treatment option for PCO as it overcomes the drawbacks of the conventional approach. Additionally, the use of cone-beam computed tomography (CBCT) helps in the assessment of the extent and degree of obliteration three dimensionally and also aids in early identification of periapical lesion.

**Keywords:** Calcific metamorphosis, Cone-beam computed tomography, Endodontics, Guided endodontics, Pulp canal obliteration.

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## INTRODUCTION

The dental practitioner is often faced with a formidable challenge while dealing with teeth with calcified pulp canals. Calcific metamorphosis (CM) or pulp canal obliteration (PCO) is defined by American Association of Endodontics (AAE) as the rapid deposition of hard tissue within the pulp space often in response to trauma.<sup>1</sup> It is most often reported in teeth with concussion and luxation injuries.<sup>2</sup> Pulp canal obliteration can be recognized clinically as early as 3 months post-injury; however, it may take up to a year for its detection in majority of the cases.

A characteristic clinical feature of PCO is a tooth that is darker in hue than the adjacent teeth, exhibiting a yellow discoloration and loss of translucency of the clinical crown, due to a greater thickness of dentin deposition.<sup>3,4</sup> On the contrary, greyish discoloration following trauma is more frequently associated with pulp necrosis.<sup>5</sup> The pulp space appears partially or totally obliterated on radiographs, even though some portion of the pulp space remains in histological sections.<sup>1</sup> Pulp canal obliteration may be associated with either a normal or thickened periodontal membrane space or periradicular radiolucency with or without subjective symptoms.<sup>3,6</sup> It is generally accepted that the absence of a positive response to the electric pulp test does not automatically imply loss of vitality of pulpal tissues, as these tests are uncertain in such cases.<sup>7,8</sup>

Pulp canal obliteration is often an incidental finding and the patient is usually asymptomatic.<sup>3</sup> Hence, the clinician is confronted with a dilemma of whether or not to initiate root canal treatment (Figs 1 and 2). This article gives an overview with regards to PCO.

## ETIOLOGY OF PULP CANAL OBLITERATION

Based on previous dental literature, numerous local and systemic factors have been reported to be associated with PCO.

### Local Factors

#### Trauma

Dental trauma has been most often reported to result in PCO with 3.8–24% of traumatized teeth developing varying degrees

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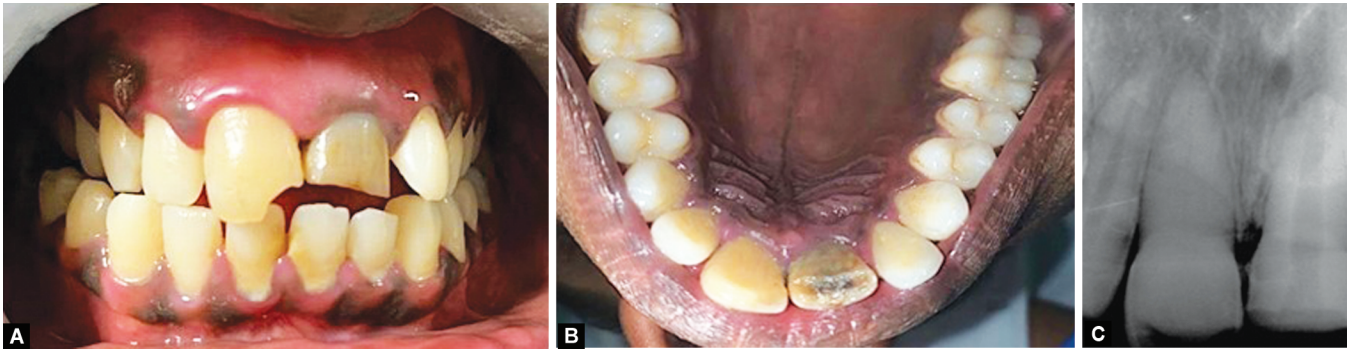
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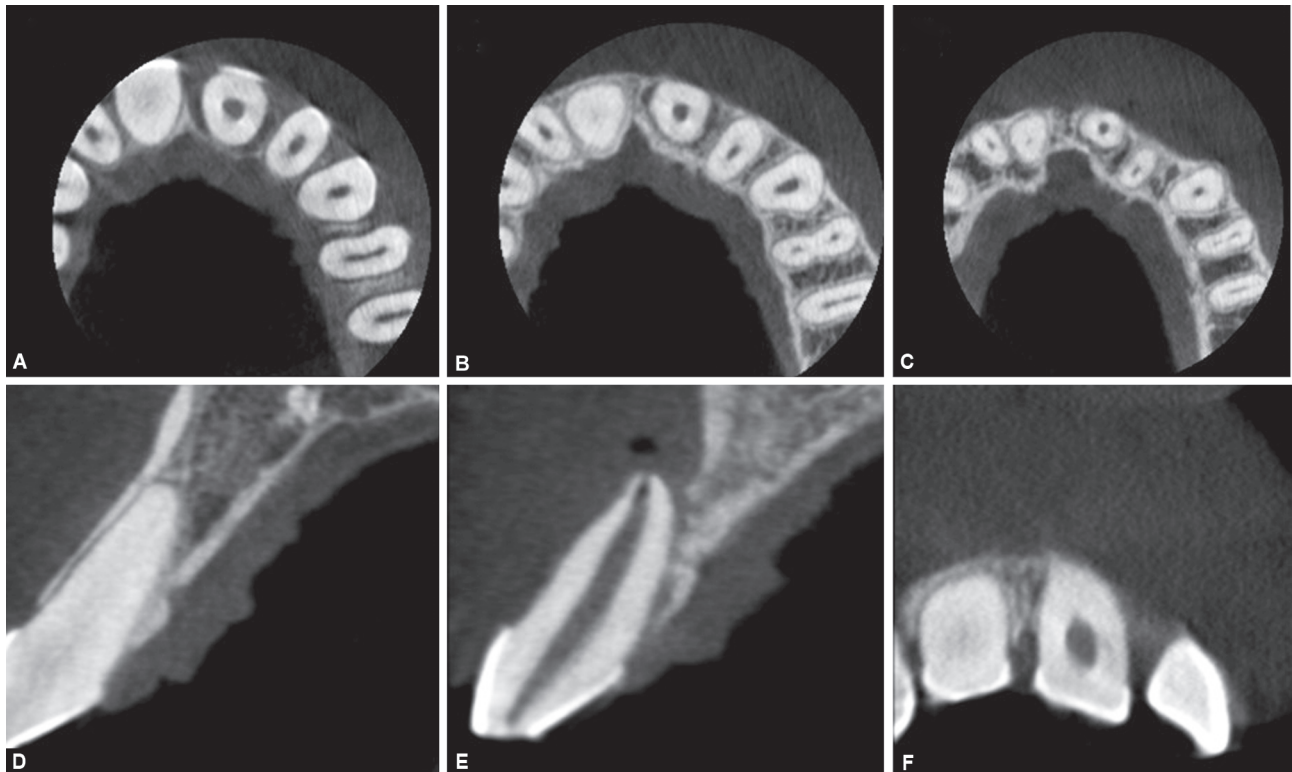
of PCO.<sup>5</sup> The incidence of pulpal necrosis in teeth with PCO ranges between 1% and 27.2%.<sup>5</sup> Recent systematic reviews on the complications following lateral luxation injury in immature and mature teeth reported the frequency of PCO to be 31.3%<sup>9</sup> and 8.1%,<sup>10</sup> respectively. With respect to replanted avulsed immature teeth, a recent systematic review reported PCO in 96% of cases with healed pulps.<sup>11</sup> A longitudinal outcome study reported the incidence of PCO in extruded permanent maxillary central incisors to be 35%.<sup>12</sup> Additionally, a recent rat model study reported a positive correlation between the incidence and extent of PCO following extrusive luxation injuries.<sup>13</sup>

### Pulp Canal Obliteration Related to Dental Treatments

Pulp canal obliteration may also occur as a result of pulpal response to carious lesions,<sup>14,15</sup> after vital pulp therapy procedures,<sup>16–18</sup> after coronal restorations,<sup>19</sup> after avulsed tooth replantation/autotransplantation,<sup>20</sup> after crown-root fracture,<sup>21</sup> and after root fracture.<sup>22</sup> Majority of root-fractured teeth appeared to develop PCO and its prevalence is known to range from 29.4 to 95.2%.<sup>23</sup> In case of hard tissue union of fragments or rarely in non-union cases, PCO was seen to occur in the apical fragment alone. However, in cases of connective tissue union of fragments, it has been reported to occur in both apical and coronal fragments.<sup>22</sup> This can be ascribed to the ability of the pulp and periodontal



**Figs 1A to C:** (A and B) Intraoral picture showing yellowish discoloration in 11 and grayish discoloration in 21; (C) Intraoral periapical image showing obliteration of root canal in 11 and wide root canal space with periapical lesion in 21



**Figs 2A to F:** Cone-beam computed tomography images of 11 and 21. (A to C) Axial section at coronal, middle, and apical third, respectively, showing extent of pulp canal obliteration in 11; (D) 11-Sagittal section; (E) 21-Sagittal section; (F) 11- and 21-Coronal section

tissues to reestablish the hindered blood supply at the fracture line.<sup>23</sup> In case of mild stretching of the pulp, devoid of bacterial contamination, cells originating from the pulp are responsible for initial callus formation, which stabilize the fracture. This is followed by tissue ingrowth from periodontal ligament (PDL) cells which form cementum.<sup>21,24</sup>

Orthodontic forces have been reported to increase the prevalence of pulp stones by 2.2–4%.<sup>25–27</sup> However, contrasting views have stated that concussion injuries that occur prior to orthodontic treatment could play a major role in obliteration of the root canal.<sup>28–30</sup> The authors opined that orthodontic forces are mild to moderate dissipating forces which promote a completely different tissue change; as compared to heavy traumatic forces, that cause sudden movement of teeth in the socket and possibly damage the pulpal vascular and neural supply, resulting in PCO.<sup>29</sup>

Secondary dentin depositions over time may also lead to obliteration of root canal in elderly patients due to aging process.<sup>31–33</sup>

### Systemic Factors

Systemic conditions like progressive systemic sclerosis,<sup>34</sup> tumoral calcinosis,<sup>35</sup> dentin dysplasia type I<sup>36–38</sup> and II,<sup>39</sup> dentinogenesis imperfecta,<sup>40,41</sup> nonsyndromic dentine defect,<sup>42</sup> osteogenesis imperfecta type I,<sup>43</sup> familial expansile osteolysis,<sup>44</sup> and also with chronic graft-vs-host disease (cGVHD)<sup>45</sup> have been shown to be linked with calcifications in the pulp. Increased predisposition to PCO is also seen in patients with hypercalcemia (excess vitamin D),<sup>46</sup> hyperparathyroidism,<sup>47</sup> gout,<sup>48</sup> end-stage renal diseases,<sup>49,50</sup> cardiovascular disease,<sup>51–54</sup> type II diabetes mellitus,<sup>52</sup> and autoimmune disorder.<sup>52</sup> A recent meta-analysis reported that

patients with pulp stones have a two-fold chance of presenting kidney stones.<sup>55</sup>

It has been reported that PCO may also have a genetic predisposition.<sup>56</sup>

### Pulp Canal Obliteration Associated with Syndromes

Saethre–Chotzen syndrome,<sup>57</sup> Elfin facies syndrome,<sup>58</sup> Ehlers–Danlos syndrome type I,<sup>59</sup> otodental syndrome,<sup>60</sup> Van der Woude syndrome,<sup>61</sup> Marfan syndrome,<sup>62,63</sup> and syndromes associated with gingival fibromatosis<sup>64</sup> have shown to have pulp calcifications as an associated clinical and radiographic finding.

### Drugs and Pulp Canal Obliteration

Systemic intake of drugs like statins<sup>65</sup> and long-term use of glucocorticoids<sup>66,67</sup> have also been implicated in obliteration of pulp.

### PATHOGENESIS OF PULP CANAL OBLITERATION

The mechanism of calcific tissue deposition during PCO remains largely unclear. However, there have been few theories that have been put forward to explain this phenomenon.

According to Heithersay,<sup>68</sup> traumatic injuries result in temporary disruption of blood supply. This causes a transient decrease in the pulpal cellularity due to destruction of the native odontoblasts in the blood-deprived region. These odontoblasts are then replaced by undifferentiated mesenchymal cells, which even in the absence of epithelial influence, rapidly deposit reparative dentin in an uncontrolled fashion.<sup>69</sup> It also results in an increased deposition of calcium ions in the capillaries, reducing their permeability, ultimately decreasing the serum flow within the pulp. The reduced supply of serum leads to a low concentration of a self-limiting enzyme—pyrophosphatase which is involved in regulating the rate of mineralization.<sup>68</sup>

Andreasen described PCO as a response to severe injury to the neurovascular supply to the pulp, which after healing leads to accelerated dentin deposition and is closely related to the loss and reestablishment of the pulpal neural supply. They also postulated that PCO may be caused by an uncontrolled sympathetic nervous response to trauma (a result of loss of parasympathetic inhibition). This causes a subsequent decrease in the blood supply to the pulp leading to pulpal respiratory depression, ultimately culminating in pathological calcification and obliteration of the root canal.<sup>70</sup>

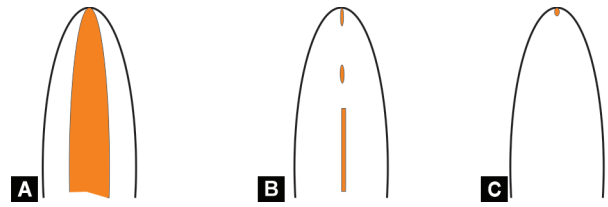
Torneck attributed the uncontrolled deposition of hard tissue as a result of the stimulation of or the loss of regulatory mechanism in the preexisting odontoblasts.<sup>71</sup>

According to Cohen and Hargreaves, trauma may result in bleeding in the canal, resulting in a blood clot which serves as a nidus for the initiation of calcification in vital pulp, eventually leading to obliteration of the root canal<sup>72</sup> (Fig. 3).

### CLINICAL MANAGEMENT

The clinician is usually confronted with a dilemma of whether root canal treatment should be initiated in teeth with PCO, which are asymptomatic. Few authors recommend delaying the root canal treatment until clinical symptoms or radiographic signs of periapical disease develop.<sup>3,773–75</sup>

According to Oginni et al., teeth with mild clinical symptoms, such as normal/high normal or a negative response to electric pulp testing (EPT) with a peri apical index (PAI) scores  $\leq 2$ , should be continuously evaluated without endodontic intervention. However, endodontic treatment should be initiated in teeth experiencing



**Figs 3A to C:** Progressive obliteration of pulp canal space: (A) Normal root canal; (B) Partially obliterated root canal; (C) Completely obliterated root canal

tenderness to percussion with a PAI scores  $\geq 3$  and displaying a negative response to sensibility testing.<sup>76</sup>

### Conventional/Non-guided Approach for Negotiation of Obliterated Canals

American Association of Endodontics classifies negotiation of PCO cases in the moderate to high risk category.<sup>77</sup> Access in anterior teeth with PCO is often initiated incisally instead of the traditional cingulum access, as the former approach allows for a more centered and straight line access which increases the possibility of locating the sclerosed canal.<sup>78</sup> With the use of dental operating microscope (DOM) and availability of long shank burs and ultrasonic instruments, the negotiation of these canals has become predictable. Repeated radiographs are taken to verify whether the path of negotiation is correct and adjustments are made accordingly. However, this method is very time-consuming, laborious, and needs patient cooperation. Additionally, the chances of instrument separation, perforation, and excessive dentin removal are very high with this method of negotiation.<sup>5</sup> Nonetheless, some of these problems can be overcome using the guided endodontics.

### Guided Approach for Negotiation of Obliterated Canals

Guided endodontics is a new technique wherein patient-specific templates are designed for canal negotiation using the cone-beam computed tomography (CBCT) images of the involved tooth.<sup>79–81</sup> The CBCT images and the surface scan of the arch are uploaded and integrated in a virtual imaging software. This software superimposes the planned path of negotiation on the scanned images of the tooth and using these details a template is made for access opening.<sup>80</sup> Numerous case reports and case series of successful negotiation of sclerosed canals using the guided approach have been published recently. This approach is faster and easier compared to the conventional procedure. This new method also results in less dentin removal and is highly successful.<sup>82–85</sup>

### CONCLUSION

Pulp canal obliteration most often occurs after traumatic dental injuries. The use of 3D imaging in such cases allows for better understanding of the extent of obliteration, early identification of the periapical lesion, and it helps in the fabrication of patient-specific templates for access opening. Negotiation of these canals has become definitely possible and simpler with the use of the guided access approach.

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