PULP STONES: A REVIEW

INTRODUCTION

Discrete calcifications occur in the pulp of certain teeth which might be healthy, diseased or even unerupted. These calcifications have been termed "pulp stones" or "denticles" and were first mentioned in literature by Norman and Johnston in 1921. They are seen to be fairly common and various studies have been done on their prevalence rate. Some even suggest that their prevalence can be close to 100% particularly if associated with carious or restored teeth. Their size can range from small, microscopic which are associated with arterioles to relatively large ones filling up the whole of pulp chamber. A single tooth may have from 1 to 12 or even more stones (1).

These pulp stones can be attached or embedded to the dentin of the pulp chamber or can exist freely within the pulp tissue. Attached denticles are thought to be more common than the free denticles. A denticle may appear free in one plane of section in which it is visualized it may be attached in another plane and therefore without serial sections on an entire tooth pulp, one cannot state that a given denticle is free or attached (2). They can also be divided on the basis of their structure as true or false pulp stones. True pulp stones are formed of dentin with a layer of odontoblasts lining them whereas false pulp stones appear within collagen bundles as concentric layers of calcified tissue. There is another type of pulp calcification, known as diffuse calcification, which are more irregular compared to false pulp stones, and seen to be closely connected to blood vessels. Diffuse calcifications are most commonly seen in the root canals of teeth and resemble the calcification seen in other tissues of the body following degeneration. Stones can be further subdivided into those with distinct concentric laminations and those without distinct laminations. Laminated pulp stones are not usually associated with smaller pulp stones, whereas nonlaminated stones are rougher and may have smaller stones attached to their surfaces. Pashley & Liewehr (2006) histologically recognized two types of stones: those that are round or ovoid, with smooth surfaces and concentric laminations; and those that assume no particular shape, lack laminations and have rough surfaces (3).

The formation of these calcifications is generally not well understood and various factors have been implicated such as age, systemic diseases or presence of some long-term irritants such as caries or restorative material. They have also seen to be associated with genetic diseases such as dentin dysplasia, dentinogenesis imperfecta and syndromes like Van der Woude syndrome (1).

Clinically, pulp stones pose little problem during endodontic treatments by obstructing or blocking the pathway for cleaning and shaping of the canals. Apart from this problem, it is not known whether they are of any other significance. So, their primary clinical relevance remains in the area of endodontic treatment, much in the way that secondary and tertiary dentine formations also influence the same. Hence, further investigation by reviewing contemporary studies on the same topic may shed more light on their formation.

PREVALENCE OF PULP STONES

The prevalence studies done on pulp stones report varied results, ranging anywhere from 8 to 90% (1). This difference results from the variations in sample and sample size in previous studies. Furthermore, the presentations of prevalence were also different in the literature. Some investigations presented the prevalence based on person as well as teeth numbers, and the others reported only the prevalence based on the latter. The true prevalence of pulp stones can be determined only when the teeth are subject to histological examination, since in radiologic studies, pulp stones with a diameter smaller than 200 um cannot be appreciated (1). Radiographs may also not recognize pulp stones attached to or embedded in dentine and permits superimposition of structures such as alveolar bone and metallic restorations that might obscure calcified bodies.

A study has been done which has evaluated pulp stones using the novel method of cone beam computed tomography as it overcomes many of the limitations of intra-oral radiographs by providing accurate anatomical details in three dimensions for diagnosis and treatment planning. In their study, pulp stones were identified in 31.9% of patients and 9.5% of teeth. Molars were the groups of teeth with the highest frequency of pulp stones (4). Nevertheless, usage of CBCT for checking presence or absence of pulp stones can be hardly justified and radiographs should be preferred in routine cases.

Another study was done to check the prevalence of idiopathic calcifications i.e., without any restorations, caries, history of systemic diseases or orthodontic intervention and reported the prevalence to be 6% of the total teeth assessed. The overall distribution of pulp calcifications was more in the first molars compared to the premolars and other molars (5). This finding indicates that their formation could also occur in the absence of metabolic dysfunctions, pathological changes or age-related degenerative changes.

Sener et al in 2009 did a study on Turkish population and 15,326 fully erupted posterior and anterior teeth were evaluated and found 747 teeth (4.8%) had pulp chamber calcifications. The prevalence of pulp chamber calcifications was significantly greater in women (62%) than in men (38%) and the first molars were the most commonly affected tooth (6).

Another study reported the prevalence of teeth showing pulp stones to be 18% but no association between the gender of the patient could be found. First and second molars also showed strong positive significance with presence of stones (7).

In all the studies reviewed, the pulp stones were reported taking the number of teeth into account and the prevalence ranged from 4% to 18%. Higher prevalence rates are seen when prevalence is checked on number of teeth into account and the prevalence ranged from 4% to 18%. Higher prevalence rates are seen when prevalence is checked on number of teeth.

Most of the contemporary studies reported a higher incidence of pulp stones in posterior teeth compared to anterior teeth.
stones in first molars which is in accordance with earlier studies (8). The reason for this could be first molars are the first definitive teeth that show up in the secondary dentition, and therefore, they endure more stress. Stress is cumulative, and the first molars are the oldest teeth in secondary dentition. Having the largest pulp space volume in the oral cavity, molar teeth present a considerable amount of pulp tissues and a superior supply of blood that may assist and increase precipitation of calcification. Higher prevalence of caries and restorations in molars may affect the formation of pulp chamber calcifications.

To sum up, prevalence of pulp stones is affected by a number of factors. Continuous secondary and tertiary dentine deposition may envelop existing pulp stones and guise their true prevalence.

**ETIOLOGICAL FACTORS**

Few factors that have been implicated in the formation of pulp stones are:

- **Caries and restorative procedures**
  The status of the tooth is considered to be significant in pulp stone development according to several studies (6),(8). The reason for this has been attributed to chronic irritation to pulp tissue because of exposure to long-standing irritants. The incidence of calcification in carious teeth is seen to be nearly five times than that in non-carious teeth in young adults (1).

- **Age**
  With age, there occurs changes in the pulp tissue of the tooth. Many authors link these changes to the formation of calcifications. Pulp spaces present in the teeth decrease due to deposition of secondary and tertiary dentin. There is also a decrease in number of cells and increase in the number of fibres present in the pulp. These degenerative changes might lead to an environment which favours incidence of calcification in elderly patients. Bernick et al in 1967 stated that calcification has been seen in 90% of the teeth from people more than 40 years. These calcifications either involve nerve tissue or apically located blood vessels. The various age-related changes that occur in the dentin pulp complex act as modulatory mechanisms for mineralization and differentiation events. Senescent fibroblasts can serve as nucleation foci for pulp stone formation because of their low-level cytokine release (9). There is evidence that there is up-regulation of growth factors including transforming growth factor beta-1 and fibroblast growth factor-1, which are family molecules that mediate the signaling of odontoblast differentiation (10). Older age groups are also reported to have increased expression of alkaline phosphatase activity in their pulps when they were irreversibly inflamed (11). These findings corroborate that ageing has a significant effect on cell physiology that may lead to increased incidence of pulp stones. Along with these findings, calcifications have also been seen in young patients with or without any irritant as a factor (12). It has been shown that tooth wear such as attrition, abrasion, erosion and trauma could hasten ageing in dentin-pulp complex. The vascular supply and innervations present in this complex can rapidly degenerate leading to increased rate of reparative dentin formation and finally fibrosis and calcification (2). Nevertheless, the studies determining the relation of age to pulp calcification are contrasting and whether there exists a correlation still remains a question.

- **Systemic disorders and genetic predisposition**
  Researchers have tried to correlate the presence of pulp stones, which are a form of calcification, to other types of calcifications in the body i.e., gall stones, renal stones and carotid artery calcifications.

  In a study done in 2015, presence of pulp stones was affirmed with OPGs and USG examination was done to check for renal and carotid artery calcification. According to the study, presence of pulp stones and CAC’s on panoramic radiograph can be suggestive of renal artery calcification in the patient (13).

  Another study reported higher incidence of pulp stones in patients with cardiovascular disorders and type II diabetes mellitus (14).

  One study reported that the chance of having kidney stone is 5.78 times higher in the subjects having pulp stone in three teeth or more (≥ 3 teeth) (15).

  A study found negative correlation between pulp stones and gall bladder stones was found concluding they are not related to each other (16).

**Orthodontic intervention**

Presence of pulp stones was evaluated before and after orthodontic treatment using panoramic radiographs. Dental pulp stones were detected in 3% of the teeth in conventional panoramic radiographs and 5.2% of the teeth at post-treatment panoramic radiographs. Pulp stone prevalence increased pointedly (2.2%) hence indicating orthodontic treatment may trigger their formation (17).

**Implicated molecular factors**

Calcifying nanoparticles are self-propagating calcifying molecules. Their complexes found in bovine and human blood and blood products. These nanoparticles produce biogenic carbonate apatite on their cell envelope at all growth phases, which resulted in white biofilm and mineral aggregates closely resembling those found in tissue calcification in the human body. These CNPs have been found in human dental pulp stones and can be instrumental in their formation. (18)

Dental pulp cells produce osteopontin (OPN) and it has been localized in the human pulp stone matrix. OPN has been shown to be associated with urinary stones, atherosclerosis, cardiovascular diseases, and obesity. It enhanced the formation and aggregation of calcium oxalate crystals as components of urinary stones in vitro, and OPN mRNA levels were seen to be significantly increased in urinary stone-forming rats in vivo. Pulp calcifications also expressed OPN with high concentration of glucose along with increased alkaline phosphatase activity. OPN might be a key molecule involved in the increase of pathologic pulp calcifications when seen in diabetic patients (19).

Advanced glycation end products (AGE) which accumulate slowly during a person's life span and can contribute to age-associated physiological changes were seen to enhance the calcification potentials of rat dental pulp cells, suggesting that it may stimulate pathologic calcification of diabetic dental pulp tissues (20).

Statins, 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors, are the first-line pharmaceuticals for the prevention and treatment of dyslipidemia. Patients on statin medication indicated a possible increased odontoblastic activity. Therefore, systemic statins could be a contributing factor for pulp chamber calcification. Significant increase of calcification and loss of vertical height of the pulp chamber was observed in mandibular molars in patients on statin medication (21).

LIM mineralization protein (LMP-1) is an intracellular signaling molecule that stimulates the synthesis and secretion of other osteo-inductive factors. The expression pattern of LMP-1 in human pulp-dentin complex shown in the study by Wang et al indicates a role of LMP-1 in the odontoblast differentiation and dentin matrix mineralization during dentin formation. Wang et al reported presence of this protein in pulp stone surface and suggested its involvement in pulp stone mineralization process (22).

Presence of trace elements such as Zn and Cu has also been implicated in development of calcifications (23). According to the authors, the increased concentration of Cu and that were observed in the pulp calcifications might reflect the increased antioxidative action of Cu/Zn SOD (superoxide dismutase), which was probably secreted by odontoblasts or other local cells to control the oxidative state in the inflamed area. This oxidative state can be a cause of calcification of the pulp tissue. Pulp stones composition also reported presence of lead, arsenic etc. suggesting they can trap environmental pollutants and that can lead to accelerated mineralization (24).

**CLINICAL SIGNIFICANCE**

The clinical significance of pulpal calcification is not very clear. Several authors have stated that pulp stones may be a reason for pain which can range from mild to severe. Pulp stones are associated with necrotic tissue, may be the development of pulp stones around the tissue or entrapment of the nerve within it during formation. Both can cause pain that is idiopathic in origin. Though denticiles might seem to impinge on the nerves of the pulp, they most probably do not. Therefore, the treatment of teeth which radiographically demonstrate pulp stones in the hope of relief of pain is not a sensible option in most cases.

With regard to pain of the patient, pulp stones have been likened to urinary and gall bladder stones, but a much higher incidence of
unexplained dental pain would be expected when correlating with these types of calcifications. Pulp stones are more likely to be the indicators of changes in the pulp tissue rather than being a reason for those changes. The presence of pulp stones or diffuse calcifications has not been seen to affect the threshold of pulp testing. However, endodontists usually struggle in patients with calcifications to properly extirpate the pulp tissue. Attached pulp stones can pose problems by deflecting or blocking the tip of the instrument and preventing its way down the canal. A large pulp stone present in the pulp chamber can also be removed by the help of ultrasonic instrumentation or by using burs. Should a stone be attached to the canal wall and a file can be passed alongside the stone, it may be removed by careful instrumentation. By and large, pulp stones present minute problems during endodontic treatment when appropriate magnification, good access and proper instruments are employed.

CONCLUSION

Despite their ubiquitous presence in human teeth, the formation of pulp stones remains an enigma. Their true prevalence has also not yet been affirmed due to limitations of the radiographic method of assessment as well as the histological method. Various studies have correlated the prevalence of pulp stones to the age of the patient but the increased incidence of pulp stones in young patients' questions that finding.

Presence of underlying inflammatory disease has been postulated as a reason for their formation, but they have also been noticed in healthy teeth free of caries and restorative material, i.e., of idiopathic origin.

Increased incidence of pulp stones was seen in patients presenting with systemic disorders but the underlying mechanism seems to be unclear. Various studies have been done trying to link pulp stones with other pathological calcifications but they are inconclusive.

Another aspect is the relevance of calcifying nanoparticles as the causative agent for these pulp stones. In a study done by Zeng et al., CNPs have been implicated as the reason for calcification in pulp tissue but it is possible that they are simply a marker of pathological state. CNPs can be studied further for their role.

LMP-1 has been found on the surface of pulp stones but no significant correlation has been made. Further studies need to be done to affirm the finding and to establish correlation between the two.

Future research can be done studying the prevalence of pulp stones with increased sample size or a multi-institutional study can be done to ascertain any association. Further studies need to be done to affirm the correlation has been made. Further studies need to be done to affirm the association.

In summary, pulp stones seem to be physiological manifestation which gets accelerated due to pathological conditions in the body. Their etiological factors do not seem to be fully apparent. Their area of relevance is mainly endodontic treatment. Further studies are needed to shed light on their formation.

REFERENCES