Pulp Stones as a Warning Sign for Coronary Artery Disease- A Narrative Review

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ABSTRACT

Globally, Coronary Artery Disease (CAD) has been the main factor in both morbidity and mortality. However, Pulp Stones (PS) that span the entire dentition are uncommon and require additional analysis to determine the likelihood of other related disorders. PS formation is more likely to occur in individuals with systemic illnesses including diabetes mellitus and cardiovascular disease. Calcifications of dental pulp may have a similar pathogenesis as calcified atheromas. When compared to diabetic and healthy people, patients with systemic disease in the cardiovascular group had a higher probability of developing PS. More than half of the teeth in early adolescents have localised pulp calcifications that may be seen under the microscope. The detection of PS in a patient with unidentified systemic disorders can serve as an early diagnostic sign.

Keywords: Cardiovascular diseases, Dental pulp, Pulp calcification, Systemic diseases

INTRODUCTION

Denticles, also known as PS, are nodular, calcified masses that can develop within the pulp of healthy, damaged, or even developing teeth. PS come in a variety of sizes, from tiny particles to huge masses that almost destroy the pulp chamber [1]. PS are normally asymptomatic unless any nerve fiber bundles are invaded. Especially in the elderly, PS was often seen as a sign of aging. However, it can also manifest in genetic or systemic conditions like diabetes, renal disorders, autoimmune conditions, and cardiac diseases [2]. Calcium phosphate crystals are the primary component of all calcifications, including atherosclerotic plaques, PS joint calcifications, and renal calculi. These crystals cause an immediate immune reaction, which has the potential to cause significant morbidity and mortality [1]. PS was related to CAD in patients older than 40-year-old compared to younger patients (40-year-old) [3]. Worldwide, CAD has been identified as the primary factor in morbidity and mortality. Those with CAD have an increased risk of developing PS [4]. Dentists are the first, who can diagnose the presence of PS in the oral cavity and hence, can refer the patient to a physician for early detection of the systemic disorders and thereby, can contribute to reducing the morbidity and mortality brought on by these illnesses [5].

Epidemiology

The PS is thick, calcified tissue masses, found in the tooth pulp. These PS may be located in the coronal or radicular region of the pulp. Coronal PS is more common than radicular PS. In a single tooth, PS can range in size and number from 1 to 12 or more, with some PS being extremely small and others being large enough to obstruct the pulp chamber [6]. These PSs could be free, embedded, or attached to the pulp space wall. They may be true PS or false PS. False PS is formed from degenerative cells that get calcified, whereas true PS is formed from dentin and lined by odontoblast. PS can be seen in primary and permanent dentition [7]. PS prevalence is more common in females than males and in the first molar than the second molar in both maxillary and mandibular teeth [8]. It is plausible that the first molar's early eruption will expose it to more deteriorating changes over time, proving that the calcification rate of pulp rises over time [9]. The age-related changes in the normal pulp structure could be the most significant factor. Age-related changes are more common in the coronal pulp than in the radicular pulp. The majority of PS was found in decayed teeth and restored teeth, suggesting that the PS may be a response to long-term irritation [10].

Types of Pulp Stones (PS)

Pulpal calcifications/PS can be categorised into different types according to their size, composition, and location. Depending on their size, they might be classified as diffuse calcifications and denticles. Denticles are comparatively large, clearly defined, and hard structures within the pulp cavity [11]. According to Ashley DH and Liewehr FR (2006), PS can be divided into two groups based on histologic features: those with laminations, smooth surfaces, and concentrical shapes, and those with smooth surfaces, no laminations, and no particular form [12]. PS were histologically classified by Kronfeld R and Boyle PE into true or false forms [13]. True denticles have irregular dentin with remnants of odontoblasts and dentinal tubules. True denticles are very rare. True denticles develop when Hertwig's epithelial root sheath fragments become trapped in the pulp as a result of a local perturbation. These remains may then cause pulp cells to develop into odontoblasts and start the development of dentin.

False denticles are calcified formations that do not display the structure of dentin in the pulp cavity. False denticles are composed of calcified material in the form of concentric layers (lamellae) that encircle a core nidus of necrotic and calcified cells. The cells come from hyalinised pulp tissue that has deteriorated. The size of the stone grows as a result of the continual addition of new layers once calcification has started. Denticles can also be categorised based on their position as interstitial (embedded), adherent (attached), or free. In the pulp tissue, free denticles are found lying there without attachment to the walls. False denticles make-up the majority of free ones. Adherent denticles that are attached to the pulp cavity's wall tend to be false as well. Denticles that have integrated themselves into dentin are known as interstitial denticles. Fine, irregular, fibrillar calcific deposits are known as diffuse calcifications. The structure of diffuse calcifications is uncertain [14].

AETIOLOGICAL FACTORS

Recently, PS has been shown to be associated with numerous systemic disorders, including CAD, autoimmune diseases, diabetes mellitus, and renal diseases. Thus, the presence of PS may serve as one of the diagnostics in systemic disease [15]. The few causes of PS formation include pulp degeneration, inductive interactions between epithelium and pulp tissue, orthodontic tooth movements, aging, circulatory disturbances in the pulp, genetic predisposition,

fluoride supplementation, and Marfan syndrome [10]. Their development may be influenced by long-lasting irritants including cavities, substantial fillings, and persistent inflammation. Carious lesions cause the pulp to become inflamed, which produces secondary (reparative) dentin and increases calcification. The pulp's blood flow and nourishment are hampered by periodontal disease, which results in a reduction in cellular components and an increase in calcification [16]. Dental pulp diseases are either inflammatory or infectious in nature. In either scenario, the dental pulp's healthy microcirculation begins an inflammatory reaction as part of a complex defensive system to preserve the dental pulp's structural integrity [17]. Additionally, it has been demonstrated that risk factors such as smoking, poor oral hygiene, diabetes, medication, age, hereditary, and stress accelerate the development of periodontal diseases by changing the microbiota and inflammatory responses [18]. Typically, this causes a steady decrease in the number of pulp cells as well as an increase in mucopolysaccharides and fibrous components, which results in calcification.

Periodontal illness interferes with the blood supply and nutrients in pulp, leading to a loss of cellular components and an increase in calcification. The high rate of attrition is physically related to the abrasive nature of the food and the highly developed masticatory muscles. Attrition-based irritation disrupts the circulatory system and results in thrombosis, which mineralises and forms PSs. Carious lesions cause the pulp to become inflamed, which causes the formation of secondary dentin and further calcification. It is well-recognised that stress in the form of a restorative procedure can damage the vascular wall and/or create capillary thrombosis, which, upon mineralisation, can result in PS formation [19].

Calcifying Nanoparticles (CNPs)

It has been unclear how PS develops. Aging, biological factors, physical factors, and chemical variables are some of the aetiological factors that have been associated. The identification of Calcifying Nanoparticles (CNPs), also known as nanobacteria, resulted in the development of newly discovered hypothesis regarding pathological calcification. Bovine and human blood and blood products include self-replicating calcifying macromolecular complexes, which are the precursors of CNPs. CNPs have been recognised as a potential aetiological factor for pathological calcification, including PS, kidney stones, gallstones, atherosclerosis, and dental calculus in periodontitis, due to their capacity to produce nucleate hydroxyapatite. The typical result of physiological or pathological calcification of the human body as seen in the tooth pulp tissue is the formation of PS.

A possible hypothesis is that CNPs contribute to the calcification of the tooth pulp tissue [20]. One of the most contentious issues in modern biology is the existence of nanobacteria, also known as CNPs, nanobes, or nanobacterium [21]. CNPs were once known as nanobacteria and are self-replicating, cultivable macromolecular complexes. The ability of CNPs to collect calcium and phosphate on their outer envelope at physiological concentrations and circumstances is one of their most distinctive characteristics. As a result of this, it precipitates CNPs, which may be a possible aetiological factor in a variety of pathological calcification conditions in people. Dental PS could potentially be formed as a result of CNPs [22].

Age Changes and Pulpal Calcifications/Pulp Stones (PS)

Vascular, lymphatic, and nerve supply diminish in the aging pulp. Fibroblasts get smaller and fewer in number. The secretory activity was reduced, indicating that the potential for repair was impaired. An increased number of collagen cross-linkages, more collagen fibers, lipid infiltration, and calcifications are further age-related alterations [23]. According to several reports, the pulp's histologic composition changes with increasing age. The aged pulp exhibits nerve fiber degeneration and calcification in both the coronal and root regions

of the pulp and has more collagenous fibers and fewer blood vessels [24]. With increasing age, secondary and tertiary dentin deposits cause shrinkage in the size of the pulp spaces. Fibroblasts, odontoblasts, and mesenchymal cells are among the cells that significant decline in number as pulp ages, with cell density declining by half between twenty and seventy years. At the same time, fibrous tissue builds up to the point where it is almost the only thing left. This is referred to as pulp atrophy or fibrous degeneration [25].

Worldwide, atherosclerosis is one of the main causes of death. It frequently shows up as cerebrovascular disease, peripheral vascular disease, or CAD leading to Myocardial Infarction (MI) [26]. The most frequent cause of CAD and IHD includes angina pectoris, MI, and cerebrovascular diseases including stroke and peripheral artery problems. Therefore, it is essential to identify and diagnose IHDs as soon as possible, before they develop into clinical illnesses [27]. The mechanism of the formation of atheromatous plaques in blood vessels, PS, renal calculi, and joint calcifications is similar to one another [28]. Vascular matrix proteins are proteolytically broken down under pathological circumstances, producing bioactive fragments that can affect vascular wall matrix remodeling. Vascular cell or blood-borne leukocyte accumulation, proliferation, and neointima formation are all consequences of the destruction of vascular matrix proteins. Blood vessels are also more likely to grow out of control during circulatory diastole, develop convoluted veins, and undergo neovascularisation from pre-existing diseased tissue microvessels [29]. Cardiologists have determined that calcium phosphate crystals play a part in acute MI, which cause the early death of patients. Calcium phosphate crystals make up the majority of all calcifications including PS, joint calcifications, renal calculi, and atherosclerotic plaques. These crystals immediately activate an immunological response that has the potential to significantly increase morbidity and mortality [30].

Osteopontin, a recently discovered component of atherosclerotic plaque, has also been demonstrated to appear to contribute to plaque calcification [25]. Systemic diseases like arteriosclerosis and renal lithiasis could be viewed as risk factors for pulpal calcification after the study in 591 human teeth by Sayegh FS and Reed AJ in the year 1968 [31]. This was later validated by Maranhao de Moura AA and de Paiva JG in their radiographic investigation [32]. Sener S et al., evaluated the relationship between pulp chamber calcification, dental health, gender, age, and cardiovascular disease in Turkey. A total of 15,326 radiographs of teeth from 536 dental patients, including 270 males and 266 women, ranging in age from 13 to 65, have been examined. In 204 of them (38%) there was calcification in the pulp chambers. On 15,326 teeth, 747 (4.8%) had calcified pulp chambers [33]. Horsley SH et al., carried out a carotid calcification screening process, where they found pulp calcification efficiency was 66.4%. Participants above the age of 60 were found to have a considerably greater frequency of pulp and carotid calcification than participants in younger age groups [34]. According to Zeng J et al., CNPs, also known as nanobacteria are found in blood and blood products and may lead to pathological calcifications. It has been proposed that the formation of nucleate hydroxyapatite crystals by CNPs is one of the primary causes of these pathological calcifications, including PS, atherosclerotic plaques, gallstones, joint calcifications, and renal calculi. Additionally, they reached the conclusion that pathological calcifications are ultimately caused by two unique CNP characteristics, satellite-like aggregations, and concentric circles [20].

Prevalence and Association of Pulp Stones (PS) with Cardiovascular Disease

The prevalence of PS varies depending on the study type and design [Table/Fig-1] [1,5,8,35-39].

Several studies suggest a correlation between the presence of Pulp Stones (PS) and cardiovascular disease [Table/Fig-2] [1,10, 16,34,40-42].

Name of the author and year of the study	Age	Site	Gender	Prevalence
Panwar PS et al., 2019 [1]	20-55 years	Arch: Maxilla >Mandible	-	51.92%
	-	Intraoral location: Posterior teeth > Anterior teeth	-	87.42%
Alsweed A et al., 2019 [5]	18-25 years	-	-	6.9 %
	-	-	Male >Female	5%
Ranjitkar S et al., 2002 [8]	17-35 years	Intraoral location: 1st molar >2nd molar	-	19.7 %
	-	Arch: Maxilla	-	23.8 %
	-	Laterality: Bilateral >Unilateral	-	22 %
Jawahar G et al., 2021 [35]	More than 40 years	-	-	51.4%
	-	Maxilla >Mandible	-	40%
	-	-	Male >Female	54%
Ravanshad S et al., 2015 [36]	18 to 70 years	-	Female >Male	51%
	-	Maxillary molars >Mandibular molars	-	26 %
Kumar DS et al., 2020 [37]	31-60 years	-	-	54%
	31-60 years	-	Female >Male	52%
Jannati R et al., 2019 [38]	-	-	Female >Male	39.23%
Turkal M et al., 2013 [39]	15 to 50 years	Maxilla >Mandible	-	3%
	-	Intraoral location: Premolar >Molar	-	4.12%

[Table/Fig-1]: Difference in the prevalence of PS according to various studies [1,5,8,35-39].

Name of the author and year of the study	Study population with cardiovascular disease	Age of subjects (years) Or mean age (years)	Prevalence % (Teeth with Pulp Stone (PS) n (%)	Radiographic technique used
Panwar PS et al., 2019 [1]	300	20-55 years	100%	Bitewing
Bains SK et al., 2014 [10]	500	18- 67 years	38.88%	Bitewing
Edd AC et al., 2005 [16]	55	20 to 55 years	74%	Periapical
Horsley SH et al,, 2009 [34]	262	10->60 years	66.4%.	Panoramic
Yeluri G et al., 2015 [40]	50	30-60 years	91%	Panoramic and ultrasonography
Khojastepour L et al, 2013 [41]	122	<55 years	68.2%	Panoramic
Nayak M et al., 2010 [42]	28	39 to 45 years	15.86%	Bitewing

[Table/Fig-2]: Correlation of PS with CAD according to various studies [1,10,16,34,40-42].

In a study by Panwar PS et al., they included 150 participants with a history of CAD who participated and ranged in age from 20 to 55. They detected PS radiographically using a Bitewing radiograph and a paralleling approach. PS were present in 100% of subjects. When compared to the mandible, the maxilla and posterior teeth are where PS is most frequently found. The researchers concluded participants in CAD have a significant risk of developing PSs [1]. According to Nayak M et al., patients with established systemic disorders, especially cardiovascular disease, had PS in 15.86%

of their teeth [42]. According to Edds AC et al., routine dental radiographs could be used as a quick screening approach for early detection of probable cardiovascular disease because dental pulp calcification may have a similar aetiology to calcified atheromas. They also suggested that only 39% of patients without a history of CVD had PS, compared to 74% of individuals with a history of CVD [16]. One of the most frequently used radiograph to diagnose PS are Panoramic Radiographs (PRs). Because of its broad coverage of the head and neck region, the short amount of time required to obtain these images, and the huge amount of data, it is the preferred way of diagnosing PS. Friedlander AH and Lande A in the year 1981, published the first study in the dental literature describing the capability of PRs to identify Carotid Artery Calcifications (CACs). In addition, high-resolution PRs and bitewing and periapical radiographs are the two primary methods for identifying idiopathic PSs [43]. According to Maranhao de Moura AA and de Paiva JG, individuals with coronary atherosclerosis had more teeth with pulpal calcifications than patients in the control group. Additionally, the incidence of free pulpal calcifications in the pulp chambers of these patients' teeth was higher [32]. PS, CAC, and kidney calcifications were found to be significantly correlated, according to Yeluri G et al., [40]. They carried out a panoramic examination of the 50 patients, which revealed that 88.28% of the teeth had PSs and 91% carotid arteries with calcification.

A total of 46 individuals underwent ultrasound examinations out of 50 patients who had CACs and PS evidence on panoramic pictures, confirming the existence of CAC. Due to the high shared risk factors for atherosclerosis, such as increased levels of low-density lipoprotein cholesterol, triglyceride levels, diabetes, hypertension, smoking, and body mass index, it was discovered that males had a higher incidence of CAC than females [40]. Some studies even reported lower prevalence rates. Alsweed A et al., (6.9%), Ranjitkar S et al., (19.7%), Turkal M et al., (3%) [5,8,39]. The detection of PSs can be observed by a dental radiograph. To take the radiographs, many research used the paralleling technique. Others used periapical and bitewing radiographs, and other studies even used PRs to assess the frequency of PSs. So far, no specific radiographic modality has been identified [44]. Bitewing and periapical radiographs were employed in the literature, and it was stated that there was no significant difference between the two radiographic techniques for diagnosing pulpal calcification [45]. The most favoured method is bitewing paralleling rather than periapical radiography because the former could be distorted while the latter can produce a more uniform image by placing the central beam perpendicular to the long axis of the tooth [46].

CONCLUSION(S)

Multiple teeth PS prevalence may be discounted as a risk factor for CAD and other possible connected disorders. It is advised that dentists check the teeth for the existence of PS in addition to doing routine oral hard and soft tissue examinations. They should refer patients to cardiologists for advice if there are many PS present. Early detection of disease helps a physician to carry out the most conservative management methods, reducing potential consequences, as well as expenses that could arise if, a disease is kept undetected and untreated for a long duration of time. In order to prevent further complications screening should be done at an early stage to prevent further life-threatening complications.

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