Abstract

We propose that calcification of dental pulp may have a similar pathogenesis as calcified atheromas and could lead to use of routine dental radiographs as a rapid screening method for early identification of potential cardiovascular disease (CVD). Fifty-five dental patients ages 20 to 55 were chosen because pulp stones in pulpally noninflamed teeth were not expected in this age group. They completed a questionnaire regarding their CVD status and that of their parents and siblings. Entry criteria included at least one asymptomatic, min­imally restored, noncarious molar and no history of gout, renal disease, or renal lithiasis. Patients' periapi­cal radiographs of record were viewed to determine the presence of pulp stones. There was a significant rela­tionship between pre-existing CVD and pulp stones (odds ratio of 4.4 with a 95% confidence interval of 1.1, 18.7), but no relationship was found for family history of CVD and pulp stones (odds ratio of 1.7 with a 95% confidence interval of 0.5, 5.5). Seventy-four percent (14/19) of patients with reported CVD had detectable pulp stones while only 39% (14/36) of patients without a history of CVD had pulp stones. This pilot study demonstrates that patients with CVD have an increased incidence of pulp stones in teeth with noninflamed pulps compared to patients with no history of CVD. No relationship was found between presence of pulp stones and family history of CVD. The findings suggest that dental radiographic determination of the presence or absence of pulp stones may have possibilities for use in CVD screening.

Key Words

Cardiovascular disease, dental pulp, inflammation, myocardial infarction, radiographic examination, fi­brous elements

From the ‘University of Louisville, Louisville, Kentucky; §Medical College of Georgia, Augusta, GA; and +University of Alabama at Birmingham, Birmingham, Alabama.

Address requests for reprint to Paul Eleazer, SDB 417 1530 Third Avenue, South, Birmingham, AL 35294-0007.

Copyright © 2005 by the American Association of Endodontists

Pilot Study of Correlation of Pulp Stones with Cardiovascular Disease

A.C. Edds,\* J.E. Walden,\* J.P. Scheetz,\* L.J. Goldsmith,\* C.L Drisko,\*'r and P.D. Eleazer\*

**C**alcification in the dental pulp can lead to denticles, commonly known as pulp stones (1). There are many theories as to the etiology of pulpal calcification. As a person

ages, the size of the pulp chamber is reduced, there is a decrease in vascular and cellular elements, and an increase in fibrous elements (2). There is also evidence that hyper­calcemia, gout and renal lithiasis are predisposing factors to pulpal calcification (3). Nâsström et al. (4) show that narrowing and calcification of the pulpal chamber are found with increased incidence in patients with end-stage renal disease. Others dem­onstrate that periodontal disease and caries are contributors to pulpal calcification. Carious lesions stimulate inflammatory changes within the pulp leading to secondary (reparative) dentin formation and increased calcification (5). Periodontal disease interferes with the blood supply and nutrition of the pulp causing a decrease in cellular elements and an increase in calcification (6). Other studies show an association be­tween periodontal disease and CVD, indicating that periodontal inflammation secondary to infection may have a role in systemic vascular disease via inflammatory mediators (7, 8).

Like pulp stones, calcification of carotid artery plaques is often an incidental finding on dental radiographs. Atherosclerotic fibrofatty plaque formation in the intima of arteries causes lumen narrowing and reduction in blood flow, and thus CVD (9). Carotid artery atheromas are found with increased incidence on panoramic films of patients with type 2 diabetes mellitus (10) and postmenopausal women, placing them at an increased risk of stroke and myocardial infarction because of atherosclerosis (11).

Moura and Paiva (12) confirmed increased pulpal calcifications in subjects with coronary atherosclerosis upon radiographic examination. Bernick (13) finds calcifi­cations and lumen narrowing within extirpated dental pulp vessels, both medium and small precapillary arterioles, in people as young as 40 yr of age. Ninomiya et al. (14) isolates the noncollagenous protein osteopontin, found in atherosclerotic plaques, in pulp stones by immunohistochemistry.

Yet, there is conflicting literature on this subject. Although Kreil et al. (15) dem­onstrates Ungual artery plaques in atherosclerotic monkeys, no similar changes are seen in pulpal arterioles. Oguntebi et al. (13) show pulpal arteriolar lumen narrowing and atheromatous plaque development without evidence of calicification in hypercho- lesterolemic-induced swine. The study was Umited by its short duration, which could explain the absence of calcifications.

The purpose of this study was to determine if a relationship exists between a patient’s history of CVD, or a family history of CVD, and the presence of pulp stones. The presence of the calcifications within waUs of pulp vessels suggests a possible correlation between pulp stones and atherosclerosis in other vessels. Our hypothesis was that the same factors leading to atherosclerosis and calcified atheromas in large vessels simi­larly cause atherosclerosis and calcification of smaU vessels, and thus pulp stones. Assuming this premise, the identification of pulp stones could have some diagnostic value for CVD.

Materials and Methods

Fifty-five dental patients from the University of LouisviUe School of Dentistry were consecutively invited to participate. The patients were between the ages of 20 to 55. Ages above 55 were excluded as the incidence of pulp stones increases with increasing age. They agreed to participate via a university human studies committee-approved recruit­ment and consent procedure. To be eligible to participate, the patient had to have at

**TABLE 1.** Questionnaire

|  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Disease/Condition** | **Yourself** | | **Father** | | **Mother** | | | **Brother** | | **Sister** |
|  | Yes | No Do not | Yes | No Do not | Yes | No Do not | Yes | No Do not | Yes | No Do not |
|  |  | know |  | know |  | know |  | know |  | know |

Chest pain (angina)

Heart attack Heart Surgery

High blood pressure (Hypertension)

Congestive Heart Failure (CHF)

Transient ischemic attack (TIA)

Stroke

Arrhythmia Aneurysm

Anemia

Vascular diseases (vasculitis, Kawasaki, Wegener) Hypercholesterolemia

Hyperlipidemia

Kidney disease

Diabetes

Gout

Metabolic diseases (kidney or gallstones,hyperparathyroid)

Connective tissue disorders (lupus,scleroderma)

Other Diseases/Conditions

Anemia

Vasculitis

Kidney Disease

Gout

Metabolic Diseases\*

Connective Tissue Disorders1

|  | **CVD1** | **Family CVD** |
| --- | --- | --- |
| Sensitivity | 74% (14/19) | 57% (16/28) |
| Specificity | 61% (22/36) | 56% (15/27) |
| Odds Ratio | 4.4 Cl (1.1, 18.7) | 1.7 Cl (0.5, 5.5) |

**TABLE 3.** Results of Pilot Study

CVD1 = Cardiovascular Disease.

**TABLE 2.** Case Definitions for Cardiovascular Disease and Other Diseases

Cardiovascular Disease

Angina Pectoris

Myocardial Infarction

Heart Surgery

Hypertension

Congestive Heart Failure

Cerebrovascular Accident

Hypercholesterolemia

Arrhythmia

‘includes gallstones, kidney stones, and hyperparathyroid disease. •{•Includes systemic lupus erythematosus and systemic sclerosis.

least one fully erupted, minimally restored, noncarious molar, free from radiographically observable periodontal disease. Restorations limited to enamel only or shallow dentin; no class V restorations reviewed. Exclusion criteria included history of gout, renal lithiasis, or renal dis­ease.

Patients were assisted by an author in completing a questionnaire regarding their cardiovascular disease status, as well as that of their parents and siblings (Table 1). Presence or absence of CVD was deter­mined by the information in Table 2. Patients’ periapical radiographs from full mouth series of record were viewed over a light source with a magnifier in blinded fashion by the first author to determine the pres­ence or absence of pulp stones. The last author was available for con­sultation, also blinded to the results of the questionnaire.

Results

Sensitivity, specificity, and statistical odds ratios were determined using computer aided systems and summarized in Table 3.

Seventy-four percent of patients with reported CVD had detectable pulp stones, while 39% of patients without a history of CVD had pulp stones. The odds ratios disclosed a significant relationship between pre-existing CVD and pulp stones. No correlation was found for family history of CVD and pulp stones. The incidence of pulp stones was greater in the CVD history group. Those without a history of CVD showed a lesser incidence of stones in comparable teeth.

Discussion

Within the limitations of this pilot study, it appears that patients with cardiovascular disease have an increased prevalence of pulp stones when compared to patients with no reported history of CVD. No rela­tionship was found between presence of pulp stones and family history of CVD. The positive presence of pulp stones in patients with cardiovas­cular disease contributes to our theory that large and small vessel cal­cifications have a similar etiology. By limiting the reviewed radiographs to noncarious, minimally restored molars with previously stated exclu­sion criteria, we are able to rule out other potential causes for the presence of pulp stones. The self-reported CVD questionnaire although reliable for this pilot study will need to be medically confirmed in future studies.

The findings of Kreil et al. (15) are in contradiction to our results, but may not be germane to this study. It is unclear how to relate the ages or risk factors of the monkeys studied by Kreil to the patients studied here. In Kreil’s study, atherosclerosis was induced by diet alone. The presence of pulp stones was noted in posterior teeth that would other­wise not be expected to have these calcifications.

The association between pulp stones and cardiovascular disease is of significance. Our study suggests that routine dental radiographs could pos­sibly have prognostic significance or even theoretically be used as a rapid screening method for early identification of potential cardiovascular dis­ease. Dental radiographs require minimal radiation, especially the newer digital imaging techniques available. Such a screening method could easily be employed on a large scale as a public health measure, perhaps many years before vascular disease symptoms occur.

Yet, this study did not attempt to determine the temporal relation­ship between atherosclerosis and pulp stones (i.e., which comes first). Future research will entail a case-control prospective study. This will attempt to further delineate the temporal relationship between the onset of development of pulp stones versus CVD. Additional research will involve biopsies of dental pulp stones in patients with atherosclerotic disease to further compare the biochemical composition of the pulp stones to the patient’s atherosclerotic plaques.

Acknowledgment

*Supported by the ULSD Office of the Dean.*

References

1. Tamsc H, Koffcc I, Lottner MM, Shani R. Statistical evaluation of radiologic survey of pulp stones. J Endod 1982;8:455-458.
2. Bernick S, Nedelman C. Effect of aging on the human pulp. J Endod 1975;1:88-94.
3. Sayegh FS, Reed AJ. Calcification in the dental pulp. Oral Surg Oral Med Oral Pathol 1968;25:873-82.
4. Nasström K, Forsberg B, Petersson A, Westesson P. Narrowing of the dental pulp chamber in patients with renal diseases. Oral Surg Oral Med Oral Pathol 1985;59- 242-6.
5. Seltzer S, Bender IB. The dental pulp; biologic considerations in dental procedures, 2nd ed. Philadelphia: Lippincott, 1975;199-227.
6. Seltzer S, Bender IB, Ziontz M. The interrelationship of pulp and periodontal disease. Oral Surg Oral Med Oral Pathol 1963;16:1474-90.
7. Beck JD, Pankow J, Tyroler HA, Offenbacher S. Dental infections and atherosclerosis. AmHeartJ1999;138:S528-33.
8. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. J Periodontol 1996;67:S1123-37.
9. Kumar V, Cotran R, Robbins S. Basic pathology, 6th ed. Philadelphia: WB Saunders Co., 1997;282-9.
10. Friedlander AH, Maeder LA. The prevalence of calcified carotid artery atheromas on the panoramic radiographs of patients with type 2 diabetes mellitus. Oral Surg Oral Med Oral Pathol 2000;89:420-4.
11. Friedlander AH, Altman L. Carotid artery atheromas in post-menopausal women. J Am Dent Assoc 2001;132:1130-6.
12. Moura AAM, PaivaJG. Pulpal calcifications in patients with coronary atherosclerosis. Endod Dent Traumatol 1987;3:307-9-
13. Bernick S. Age changes in the blood supply to human teeth. J Dent Res 1967;46:544 - 50.
14. Ninomiya M, Ohishi M, Kido J, Ohsaki Y, Nagata T. Immunohistochemical localization of osteopontin in human pulp stones. J Endod 2001;27:269-72.
15. Kreil KV, McMurtrey LG, Walton RE. Vasculature of the dental pulp of atherosclerotic monkeys: light and electron microscopic findings. J Endod 1994;20:469-73-
16. Oguntebi BR, Stafford DS, Cerda J, Robbins F. Vascular changes in the dental pulp in the hypercholesterolemic miniature swine. Oral Surg Oral Med Oral Pathol 1992 ;74: 351-6.
17. Am Heart Association. 2001 Heart and Stroke Statistical Update, [www.american-](http://www.american-heart.org) heart.org.