Treatment of the result of chronic activation of substance P

Prof. Philippe Sleiman, Lebanon


Fig. 1

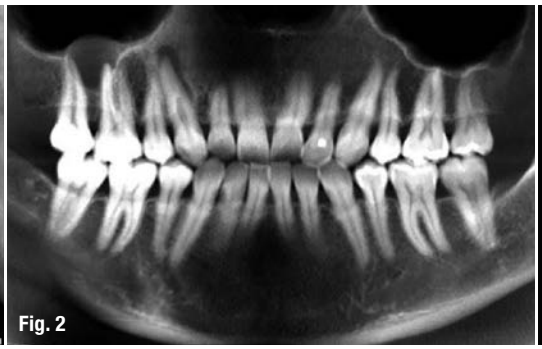
Fig. 2

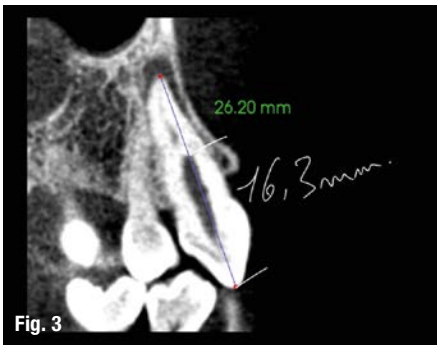

Fig. 3

Introduction

Pulp stones are primarily a physiological manifestation and may increase in number and/or size owing to local or systemic pathology. The aetiological factors involved in their formation are still not fully apparent; their formation may be associated with some local irritants that are long-standing, such as caries, dental restorations and systemic disturbance.[1] Calcific atheromas and the calcification of dental pulp have a similar pathogenesis, so routine dental radiographs may be useful. If the patient is found to have multiple pulp stones, further evaluation for associated diseases, carotid artery calcification and renal calcification is required.[2]

Calcification can occur in the dental pulp as discrete calcified stones or as a diffuse form. These pulp stones may exist freely within the pulp tissue or be embedded in the dentine.[3] Depending on their microscopic structures, pulp stones were histologically classified by Kronfeld into a true or false form.[4]

Radiographic examinations are not likely to detect pulp stones of less than 200 µm in diameter. Radiographically, pulp stones appear as radiopaque structures in the pulp cavity. The prevalence of pulp stones is higher in females than in males and occurs most commonly in molars than in other tooth types.[1,5]

Implicated factors

The formation of these calcifications is not well understood, and many aetiological factors have been proposed:

- Ageing: With age, the frequency of pulpal calcification increases because of a higher concentration of alkaline phosphatase, which could trigger calcification.[1]

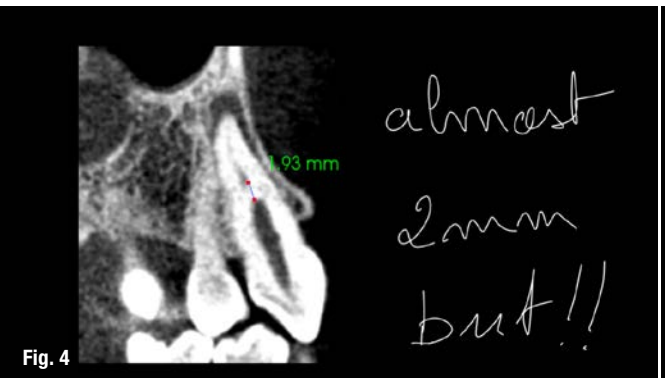

Fig. 4

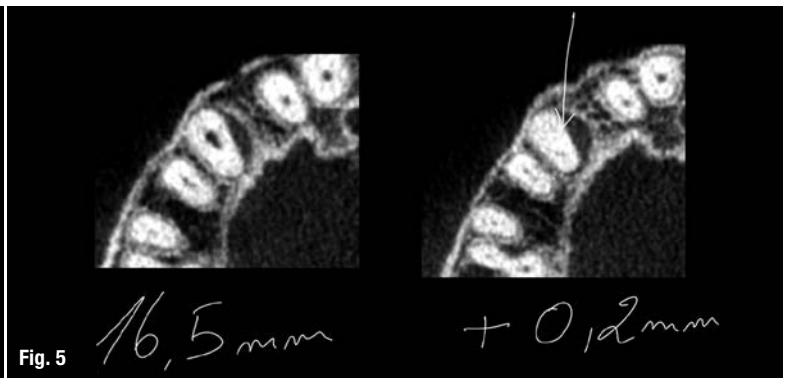

Fig. 5

- Excessive occlusal force: Such force causes changes to the pulp (hyperaemia, pulpitis, pulp necrosis, etc.). Tooth trauma causes damage to the neurovascular supply of the pulp and leads to various responses of the pulp, including tertiary dentine formation, pulp revascularisation, pulp canal calcification, chronic pulp inflammation, internal root resorption, pulp necrosis and infection of the root canal system.[6]
- Long-standing irritants, such as caries, deep fillings, chronic inflammation, abrasion and orthodontic tooth movement.

Additionally, a high prevalence of pulp stones has been reported in individuals with cardiovascular disease, kidney stones, gallstones and salivary gland stones. Also, pulp stones have been observed in females more than in males because they experience sleep bruxism more frequently.[1,5] First molars have a maximum number of calcifications compared with any other teeth because they are the first teeth to erupt and have a greater surface area and occlusal forces, perhaps leading to early degenerative changes.[1,5]

Classification of pulp stones

Pulpal calcifications have been classified by Kronfeld as: (1) true denticles (composed of orthodentine); (2) false denticles (composed of concentric layers of calcified material not resembling dentine); and (3) diffuse calcification (small calcified deposits scattered throughout the pulp tissue). However, most pulpal calcifications are conglomerates of different tissues—orthodentine, and regular and irregular calcified material—so applying this classification becomes nearly impossible.[4] In a calcified pulp, a slight increase in collagen content is present, but there is no evidence of an inflammatory response or the presence of microorganisms.[7]

In occlusal trauma and masticatory function, the dental pulps respond with a neurogenic inflammatory process in which substance P plays an important role in the direct and indirect mechanisms of angiogenesis by activating the neurokinin-1 receptors of cells such as fibroblasts, and endothelial and inflammatory cells, leading to the formation of new blood vessels, which are needed as a defence mechanism, by stimulating the formation of mineralised tissue.[8] These neuropeptides, substance P, neurokinin A and calcitonin gene-related peptide, once released by the sensory fibres, have effects on the blood circulation, inflammatory process, immune responses and connective tissue cells, and therefore they have an important role in inflammation and pulp pain.[9] Further research is needed on the association between these neuropeptides and the pulp state for understanding more about pulp inflammation and dental pain.

Fig. 6


Fig. 7


Fig. 8

Types of calcifications

There are two main morphological forms of pulpal calcifications: discrete pulp stones and diffuse calcification. The discrete form appears radiographically as a round or ovoid opacity, and it is more common in the pulp chamber than in the root canal. The diffuse form is a generalised calcification in a large area of the pulp chamber and canals. It is called calcific degeneration, and it is amorphous and unorganised.[3]

Clinical signs

The clinical signs are (1) a yellowish discoloration or reduced transparency of the crown or even grey discoloration, although not all teeth with pulp obliteration undergo a colour change;[7] (2) a progressive decrease in the response to thermal and electrical pulp testing compared with adjacent teeth;[4] and (3) generally asymptomatic.[4]

Radiographic findings

Pulp stones are calcified masses present in any portion of the pulp. Coronal pulp stones are more common than radicular ones. Radiographically, they appear as dense radiopaque masses.[5] To identify pulpal calcifications, previous studies have performed research on the prevalence of pulp stones using panoramic, periapical and bitewing radiographs, as well as cone beam computed tomography (CBCT).[1]

Complete or partial radiographic obliteration of the pulp cavity does not necessarily mean the absence of the pulp canal. In the majority of these cases, a pulp cavity with pulp tissue is present.[7]

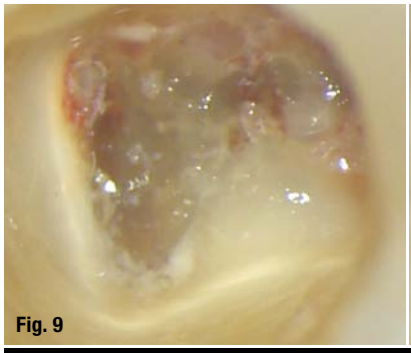
Fig. 9

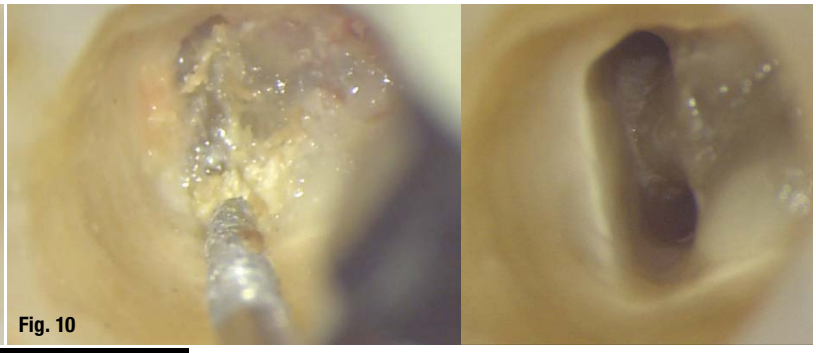
Fig. 10


Fig. 11

Treatment

Teeth with a negative response to sensitivity and tenderness to percussion should be treated in cases of pulpal calcification, and any sign of a periapical infection is an indication for root canal therapy. In 2010, the American Association of Endodontists (AAE) advised clinicians to treat teeth with obliteration of the pulp in a careful way through clinical observation and periodic radiographic examination in view of the difficulties envisaged during this treatment.

CBCT presents the tooth in all spatial planes, allowing exploration of the anatomy of the root canal, and can help in the assessment of pulp canal calcification. It is a non-invasive, reliable imaging modality with low radiation doses and has the advantage of reducing processing time. In 2015, the AAE and the American Academy of Oral and Maxillofacial Radiology updated their guidelines on the use of CBCT imaging in endodontics.[10]

When initiating the treatment, the use of a dental microscope is recommended, and during creation of the access cavity, straight-line access to the pulp is better than traditional access using different ultrasonic tips.

Clinical Case 1

A fistula between the two maxillary molars was seen by the hygienist during a routine cleaning session of the patient. When the patient was transferred to my room, I inserted a gutta-percha cone into the fistula and took a radiograph (Fig. 1). Upon studying the radiograph (we will not discuss the molar in this article), I saw that the canine had a problem. Legally, we are responsible for all the information revealed by the radiograph or on the area shown by the radiograph, which is why we always need to interpret very carefully the entirety of the radiograph and not focus only on what is considered the main problem. A CBCT scan was taken in order to understand better what was going on (Fig. 2). Looking at one sagittal cross section (Fig. 3), we can see clearly that the canine indeed had an apical infection and that there was a calcification in the middle of the root canal. The calcification was located at almost 16.3 mm, and the thickness of the

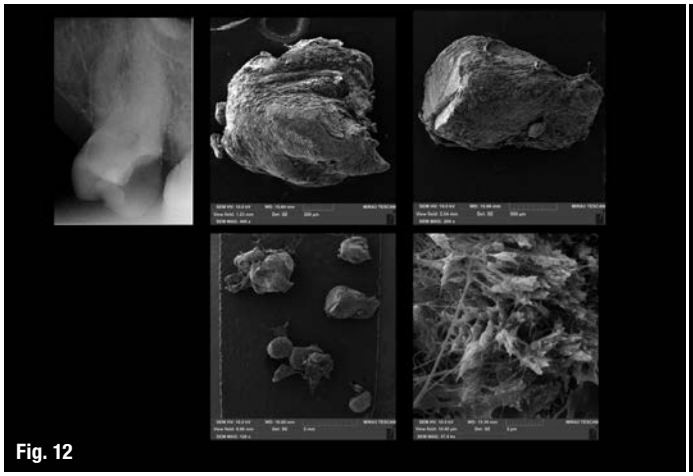
Fig. 12

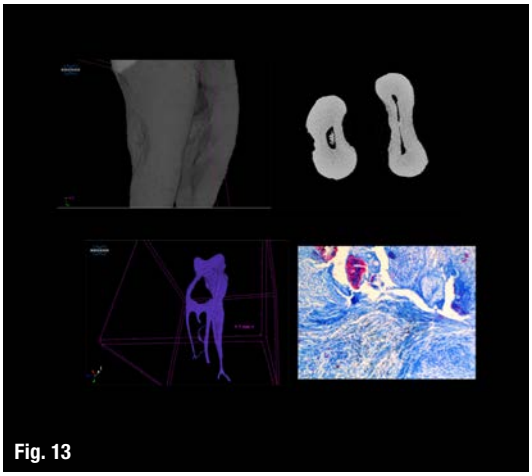
Fig. 13

calcification was almost 2.0 mm (Fig. 4). But this was not all the information that we could gain from the scan: at almost 16.5 mm and a bit beyond, by 0.2 mm, the canal was completely obliterated (Fig. 5). In Figure 6, it can be seen that the canal reopened after 2 mm but from the palatal angulation. This is crucial information, as when we opened the canal, we would need to choose a palatal angulation in order to access the original part of the canal and to avoid any perforation in this area. Ultrasonic tips were used in order to open the canal and remove the internal calcification, allowing for the root canal therapy to be performed in a single session (Fig. 7).

Clinical Case 2

A patient was referred to the office with signs of irreversible pulpitis of a mandibular molar. From the preoperative radiograph, we could see a deep filling, a very calcified pulp chamber and calcification extending to the roots (Fig. 8). After opening the access cavity under the microscope, we could see the amount of calcification (Fig. 9). Using the diamond ultrasonic tip, we were able to cut the calcification very carefully and create a second access cavity inside the first one, where, with the help of ultrasonic tips and a clinical microscope, we were able to go as deep as we could inside the canals. Figure 10 shows complete access to both distal canals and a bit of calcification left inside the isthmus between the canals. The treatment was achieved in a single session (Fig. 11). Figures 12 and 13 show the structure of the pulp stones under a scanning electron microscope and the pulp stone blocking the distal root beyond the middle part of the root (courtesy of Dr Alexey Volokitin).

Editorial note: A list of references is available from the publisher.

about

Prof. Sleiman received his DDS in 1990, his DUA in 1995, and his PhD in 2006 at the Lebanese University School of Dentistry. He also received his CESE in Endodontics at St-Joseph University in 1999. He lectures internationally and is currently Assistant Professor at the Endodontic Department of the Lebanese University Dental School.

Sleiman teaches at the Lebanese University and University of North Carolina (USA). He achieved fellowship with the International College of Dentists and the American Association of Endodontists. Prof. Sleiman maintains private practices in Beirut, Lebanon, Dubai, UAE; and Oman.

Prof. Sleiman is an international speaker in endodontics, collaborates in several undergoing research endodontic studies, and has a private practice limited to endodontics.