

The Role of Vital Pulp Therapy in the Management of Periapical Lesions

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Dear Editor,

We have recently studied the manuscript titled, "Endodontic Periapical Lesion: An Overview on the Aetiology, Diagnosis and Current Treatment Modalities" by Karamifar et al. with great interest, and found the above-mentioned paper scientific and well-organised. We would also like to congratulate the authors on writing such a methodical article, which has been published by the prestigious "European Endodontic Journal" in 2020.

In the millennium of modern dentistry, specifically endodontics, aetiological factors and the consequent classification of pulp and periapical pathosis have acknowledged amendments and hence, related definitions have been altered (1, 2). Despite the long-standing concept of pulp tissue necrosis as the main contributing factor for periapical lesions, there has been a growing body of evidence that inflamed vital dental pulps are capable of causing apical periodontitis (AP) (3, 4). Studies have shown that AP can be associated with irreversible pulpitis (IP); a condition defined as an immunological response of dental pulp tissue (5, 6) to the inflammation caused mainly by microorganisms and/or their by-products (7, 8), which could result in local changes in pulp/periapical connective tissues (9, 10).

In the last decade, three-dimensional imaging, i.e. cone-beam computed tomography, has been used for accurate clinical diagnosis and revealing the presence of preoperative AP in teeth with IP; which has shown the radiographic signs of AP in 13.7% of teeth with symptomatic IP (11). Therefore, detection of AP in radiographic images does not necessarily indicate pulp necrosis; in other words, AP could be associated with pulpal inflammation, and pulp necrosis may not be the sole causative factor for AP (12).

Several case reports have shown that vital pulp therapy (VPT) modalities, including direct/indirect pulp cappings as well as miniature/partial/full pulpotomies, could efficiently manage permanent teeth with clinical diagnosis of IP accompanied by AP (13-16). VPT has been also used to effectively treat permanent mature molars with hyperplastic IP associated with preoperative AP (4, 12). In two randomised clinical trials conducted by our research team, minimally invasive VPT procedures were performed to successfully treat mature permanent molars with IP accompanied by preoperative AP (17, 18). A recent randomised clinical study has investigated the effect of occlusal reduction on the postoperative pain in permanent molars with symptomatic IP associated with symptomatic AP (19). Moreover, a systematic review has reported a comprehensive investigation on symptomatic IP with/without symptomatic AP; which could/could not be diagnosed radiographically (20).

Considering the aforementioned evidence, we would like to emphasise that inflamed vital dental pulp as well as necrotic/infected pulpal tissues can cause AP, as a necrotic tooth will not necessarily cause AP, e.g. following dental trauma (11, 21). It should be noted that clinicians perform an endodontic treatment in line with their clinical findings; which could differ from bleeding to necrosis. However, when bleeding occurs, pulpitis is the most possible state of the involved tooth (22), and even with AP in radiography, minimally invasive treatment modalities, e.g. VPTs employing biomaterials, can be considered as the chosen treatment. Nevertheless, the selection of biomaterials needs to be discussed separately in future studies.

The published review by Karamifar et al. has not addressed this important aspect of pulp inflammation i.e. pulpitis, next to pulpal necrosis, as an aetiological factor for AP. Vital pulp therapies, as valuable treatment modalities of inflamed vital dental pulps, are minimally invasive and could result in

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less aggression and better maintenance of tooth structure and function in the oral cavity (23). In addition, in COVID-19 pandemic outbreak, VPTs can be helpful in terms of reducing treatment time/post-endodontic pain, and consequently, lowering the risk of patient/clinician/staff exposure to the virus (24, 25).

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