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Review Article

Causes of post treatment apical periodontitis

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ABSTRACT

Endodontic treatment approaches aim to achieve proper treatment and prevention of apical periodontitis to enhance the oral health status and enhance the prognosis of affected teeth. However, many complications can develop secondary to endodontic treatment. The management of post-treatment apical periodontitis might be challenging to clinicians, and the prognosis is usually lower than that of primary apical periodontitis. Therefore, identifying the potential etiology and intervening against them might be ideal for these cases. The present literature review discusses the commonest causes reported in the literature to predispose to the development of post-treatment apical periodontitis. Most of the various investigations in the literature indicate that post-treatment apical periodontitis is usually caused by either extraradicular or intraradicular infections, like primary apical periodontitis. However, it should be noted that some studies also reported that technical or procedural errors might predispose to the pathogenesis of the condition. However, it has been reported that the presence of associated bacterial infection conditions this.

Keywords: Periodontitis, Post-treatment complications, Endodontic, Etiology

INTRODUCTION

Endodontic treatment approaches aim to achieve proper treatment and prevention of apical periodontitis to enhance the oral health status and enhance the prognosis of affected teeth. However, many complications can develop secondary to endodontic treatment.[1] For instance, it has been demonstrated that the development of clinical manifestations during root canal treatment in relation to the area where treatment occurs is strongly indicative of the development of apical periodontitis. In addition, clinicians and researchers usually refer to this phenomenon as post-treatment apical periodontitis, which has been reported as recurrent (when it develops after healing occurs), persistent (when it lasts despite being treated), or emergent (when it develops after treatment).[2]

Evidence indicates that all of the events above are included under the umbrella of post-treatment apical

periodontitis, similar to the primary apical periodontitis that develops in untreated canals.[3] Accordingly, the potential difference between these conditions is related to root canal disorders. In addition, various studies in the literature have reported that the development of post-treatment apical periodontitis usually occurs following endodontic treatment that was not conducted based on standardized infection control measurements, leading to root canal infection.[4-7] In this context, different studies reported various causes for the development of post-treatment apical periodontitis. Therefore, this study will discuss the different reasons for post-treatment apical periodontitis based on evidence from studies in the literature.

LITERATURE REVIEW

This literature review is based on an extensive literature search in Medline, Cochrane, and EMBASE databases on which was performed 3rd December 2021 using the medical subject headings (MeSH) or a combination of all possible related terms, according to the database. To avoid missing poetential studies, a further manual search for papers was done through Google Scholar, while the reference lists of the initially included papers. Studies discussing causes of post treatment apical periodontitis were screened for useful information, with no limitations posed on date, language, age of participants, or publication type.

DISCUSSION

Apical periodontitis usually develops as an inflammatory response to intra-radicular infections. The immune response exerts efforts to prevent any additional spread of the infection to adjacent structures as alveolar bone and other vital tissues. It has been shown that such inflammatory responses are usually successful in containing and preventing the further spread of bacteria. However, some cases are unsuccessful, and bacterial resistance develops, leading to secondary bacterial infections. These events are usually associated with sinus and abscess formation, and patients suffer from painful swellings. Moreover, evidence indicated that chronic inflammation might be associated with these events and can increase the risk of endodontic treatment failure. Different biological and macroscopical characteristics were also reported for these events and adequately described elsewhere in the literature.[8-11]

Inadequate or improper root canal treatment is the main contributing factor for the development of post-treatment apical periodontitis. On the other hand, it has also been demonstrated that post-treatment apical periodontitis can develop among patients with healthy oral conditions and following adequate root canal treatment. In this context, previous estimates show that the condition usually develops among 5-15% of patients with preoperative apical periodontitis, although adequate root canal treatment with standard infection control measures was conducted.[12-15] Furthermore, bacterial infections have been reported as the commonest etiology attributing to the pathogenesis of post-treatment apical periodontitis. In this context, evidence shows that some cases might be owing to extra-radicular infections. However, most cases usually develop secondary to secondary intra-radicular or persistent infections. Some authors also suggested that some non-microbial factors might contribute to the pathogenesis and development of post-treatment apical periodontitis. However, evidence regarding these factors is limited by the minimal number of existing studies in the literature and needs further elaboration. Although the condition is common and might be associated with different complications, evidence shows that it can be effectively managed and eradicated. In this context, it has been indicated that conducting peri-radicular surgical approaches or applying non-surgical patterns can effectively manage these cases and enhance oral cavity health and related teeth functions.[1] The present section will discuss the different causes of post-treatment apical periodontitis.

Many authors have indicated that the development of post-treatment apical periodontitis is typically attributed to microbiological issues. This is because evidence from different studies shows that almost all patients usually have a microbiological profile related to the condition, even among teeth that have been adequately treated. As previously mentioned, infections can be intra-radicular (located within the root canal systems) or extra-radicular (which usually extends to the peri-radicular tissues). Besides, bacteria infection can either be secondary or persistent based on when bacteria had access to the root canal systems.[16,17] Evidence shows that non-successful control and elimination of bacteria when endodontic treatment is conducted leads to persistent infection. This occurs when the first treatment approach is usually conducted. Therefore, studies show persistent bacteria infections are the main etiology for developing post-treatment apical periodontitis.[13] On the other hand, secondary infection is not usually introduced during the first treatment approach. It is usually introduced to the canal system by manipulating the septic status during endodontic treatment or secondary to failure within the coronal seal following treatment conclusion. Recurrent and emergent diseases usually result from secondary bacterial infections. However, it was also reported that recurrent diseases might develop secondary to persistent infections.[18] Previous molecular, cultural, and microscopy-based investigations reported that intra-radicular infections usually contribute to post-treatment apical periodontitis development.[19-26]

These studies show that such complications usually occur secondary to resistant bacteria that are not effectively eradicated by inducing a remarkable state of peri-radicular inflammation. Furthermore, these bacteria are usually in close contact with a source of nutrition from the peri-radicular tissues and areas that are hard to reach with routine instrumentation and eradication approaches.

In this context, studies demonstrated different areas for these events. These include dentinal tubules, isthmuses, apical ramifications, lateral canals, and the very apical part of the root canal. In addition, studies demonstrate that bacterial biofilms are commonly found in these areas.[19-21,27-29] Finally, studies indicate that the presence of bacterial infections during filling time can negatively deteriorate the treatment outcomes.[30] Accordingly, it has been shown that residual bacteria can survive within root canal systems for a long period and induce remarkable tissue inflammation and periodontitis.

The literature proposes some reasons to justify that post-treatment periodontal diseases are most commonly caused by persistent and not secondary infections.[31,32] Although prior evidence shows that secondary bacteria infections (coronary leakage) are the commonest cause of post-treatment apical periodontitis, recent evidence shows that persistent infections occurring during the initial treatment approaches are the main causes of developing these conditions after persisting within the root canal systems despite applying adequate disinfection measures.[14,33] These suggestions are mainly based on previous findings from published investigations. For instance, bacterial infections are usually detected within the apical third of the canal after biopsy specimens that follow endodontic treatment. It should be noted that these bacterial biofilms are not furtherly detected along the entire canal walls (which suggests coronal leakage and secondary bacterial infections). The second lead is the poor outcomes of obtaining positive cultures during root canal filling.[21] This indicates persistent infections responsible for initiating tissue inflammation and apical periodontitis.[14] Finally, if secondary bacterial infections were the commonest etiology for developing post-treatment apical periodontitis, the treatment-related failure rates for necrotic and vital tissues would be similar. However, they are not.[5,7,12,15,34] On the other hand, it has been well-evidenced that the presence of preoperative apical periodontitis increases the risk of developing post-treatment apical periodontitis than in cases when no underlying etiologies are present. However, it should be noted that although persistent infections represent the commonest causes for developing post-treatment apical periodontitis, secondary infections are still a remarkable etiology for developing these conditions. Accordingly, based on data from previous cross-sectional investigations, it is advisable that clinicians install a permanent coronal restoration following root canal treatment to enhance the outcomes.[4,6,35]

Secondary bacterial infections might develop secondary to intra-radicular infection or not.[36] However, it should be noted the evidence regarding the existence and development of dependant secondary bacterial infections is poorly proven among the current studies in the literature. This indicates that the incidence of these etiologies, if present, is very low and attributed to a minimal number of post-treatment apical periodontitis.[28] In this context, previous histological analysis by Ricucci et al reported that none of the analyzed cases of post-treatment apical periodontitis was associated with an apparent cause of independent bacterial infections.[21] Moreover, it has been reported that persistent infections were also detected. On the other hand, extra-radicular infections were more common among symptomatic teeth. Accordingly, it can be concluded that most of the current molecular-based studies indicate that the etiology of post-treatment apical periodontitis is attributed to bacterial infections, whether intra-or extra-radicular.[23,37,38] Furthermore, these investigations also indicate that most apical periodontitis events are attributed to persistent bacterial infections.[19,21] However, some previous studies reported that other etiologies might contribute to the pathogenesis of post-treatment apical periodontitis. These might be exogenous or endogenous non-bacterial factors.[39,40]

The most common exogenous factors might include foreign body abnormal reactions to apically extruded food, paper points, or filling materials. On the other hand, the commonest endogenous factors might include cholesterol true cysts and crystals, and the associated complications. In addition, it has been demonstrated that clinicians usually find it challenging to locate any source of infection as the primary cause of post-treatment apical periodontitis in these cases.[21,41] Accordingly, these studies concluded that non-microbial causes could contribute to post-treatment apical periodontitis. However, further evidence is still needed from larger studies with better evidence.[42,43]

It has been further shown that apical periodontitis's development might be predisposed by procedural errors when the treatment process is conducted. It has been shown that overfilling, perforation, and fractured instruments are various events that might be considered procedural errors and lead to developing post-treatment apical periodontitis.[44,45] In addition, the persistence of the associated inflammation is also usually accompanied during these events secondary to the potential presence of infections. Following technical errors, the main issue contributing to the pathogenesis of post-treatment apical periodontitis is the difficulties and presenting challenges that clinicians face when approaching root canal treatment. Accordingly, the presence of infected root canals secondary to these events can significantly contribute to treatment failure secondary to these technical errors. This occurs secondary to the inability of clinicians to impede other instruments to the apical part of the canal, being hindered by the fractured instruments. Accordingly, this will hinder the process of disinfection and leave root canal bacteria unaffected. Moreover, past evidence shows that overfilling might be associated with endodontic treatment failure and developing post-treatment apical periodontitis.[46] However, evidence from recent studies indicates that root canal inflammation is not associated with the apical extent of root canal fillings.[14,47] It should be noted that this is conditioned by the absence of bacterial infections in this region.

In addition, studies indicate that most of the currently used materials for root canal treatment might only show cytotoxicity before settings and are usually biocompatible.[48,49] Accordingly, researchers showed that the association between root canal inflammation and using extruded sealers-induced tissue injury is not permanent. Moreover, studies show that an associated infection is usually the primary etiology of treatment failure and tissue injury in disorders that develop after root canal overfilling. In this context, it has been reported that some predisposing factors are usually involved in this process. These include favoring a source of nutrition for residual bacteria that exist within the canal system, not performing a proper canal seal, and a previous over instrumentation which subsequently leads to the extraradicular projection of infected dentinal debris. Accordingly, these factors should be considered when reporting that technical errors are responsible for the development of post-treatment apical periodontitis.

CONCLUSION

The management of post-treatment apical periodontitis might be challenging to clinicians, and the prognosis is usually lower than that of primary apical periodontitis. Therefore, identifying the potential etiology and intervening against them might be ideal for these cases. The present literature review discusses the commonest causes reported in the literature to predispose to the development of post-treatment apical periodontitis. Most of the various investigations in the literature indicate that post-treatment apical periodontitis is usually caused by either extraradicular or intraradicular infections, like primary apical periodontitis. However, it should be noted that some studies also reported that technical or procedural errors might predispose to the pathogenesis of the condition. However, it has been reported that the presence of associated bacterial infection conditions this.

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