Analysis of Various Factors that Cause the Failure of Root Canal Treatment: Scoping Review

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Abstract

Endodontic treatment failure may occur due to different causes such as persistence of bacteria, root canals that are poorly cleaned and obturated, improper coronal seal (leakage), and untreated canals (missed canals). The current review aims to compile all the current studies concerning all factor that causes endodontic failure. In this scoping review, three databases, PubMed, EBSCOhost and ScienceDirect, were searched using specific inclusion and exclusion criteria. Among 428 studies, only 13 met the inclusion criteria and were included in the review for further analysis. Follow-up of 13 selected articles (6 in vivo case and 5 ex vivo case) showed variously outcomes. Root canal therapy frequently fails when it is subpar to acceptable standards. Procedure mistakes that prevent the management and prevention of intracanal endodontic infection are the main cause of the failure of root canal therapy in many teeth. The persistence of microbial infection in the root canal system and/or the periradicular area is without a doubt one of the main causes of endodontic failure.

The improper restoration that will lead to endodontic failure are root fracture, tooth fracture, and marginal leakage at tooth restoration. Those improper restoration will produce leaky channels that enabled fluids from the periradicular tissue to enter tubules and feed the remaining bacteria and causing the re-infection in that site. This is show that how crucial it is to achieve appropriate root canal system disinfection for a long-term treatment outcome.

Review (J Int Dent Med Res 2023; 16(1): 404-410)

Keywords: Endodontic treatment, infections, endodontic failure, restorations.

Received date: 15 October 2022 **Accept date:** 17 November 2022

Introduction

Predictable success rates for endodontic treatment range from 86% to 98%. Clinical symptoms and radiographic results of the treated tooth are used to determine whether the treatment was successful or not. The absence of pain, the disappearance of inflammation and fistulas, if they existed before to treatment, in addition to keeping the tooth in its alveolus preserve and functioning. Successful treatment will be determined radiographically by the complete healing of periapical bone lesion and the lamina dura's normal appearance for a minimum of six months and a maximum of twenty-four months. Histologically, the periapical tissues must be completely repaired and free of inflammatory cells.[1]

Deficient chemo-mechanical preparation and inadequate canal system filling are the most prevalent causes of endodontic treatment failure, which is predominantly brought on by the persistence of microorganisms (intra and extra radicular). All of this may happen due to poor canal preparation, fillings without adequate apical sealing, filtration during the restoration of the clinical crown, untreated canals, and iatrogenic conditions like apical transport, small access cavities, perforations, false pathways, and instrument fractures.[1]

During endodontic therapy, it is crucial to eliminate all pulpal tissues, dentinal debris, and bacteria from the root canal system. Eliminating bacteria and their metabolites is essential for a successful endodontic procedure since they are what cause pulpal and peri-radicular inflammation. Ineffective removal might result in ongoing inflammation and impaired healing. Although appropriate irrigation with sodium hypochlorite and good canal instrumentation can

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reduce the amount of bacteria, they cannot completely eradicate *Enterococcus faecalis* from the root canal.[2]

The restoration of the endodontically treated tooth is a critical factor during treatment planning because of will impact the prognosis of the tooth. The pulpless tooth is typically associated with significant loss of coronal and radicular tooth structure after the preparation of the endodontic access. As a result, restoration that are severely fractured due to poor coronal tooth structure.[3] The objective of this literature review is to compile all the current studies concerning all factors that cause endodontic failure.

Materials and methods

This review has been carried out in compliance with the guidelines of Preferred Reporting Items for Systematic Reviews (PRISMA).

The goal of this systematic review was to answer the following question:

"What is the factor on endodontics failure treatment?"

The article search was carried out by two electronic databases (PubMed and Google Scholar) with MesH words (or/and) in different combinations as the following: ("Endodontic Failure") taking into consideration the question of our review.

Results

A total of 428 articles were initially found. Excluding 51 duplicates, reaching a total of 377 articles. Then, after reading the title of each article and taking into consideration the objectives of the work, 32 articles were eliminated, reaching a total of 345 articles. After that, the summaries of the chosen articles were read and 324 were eliminated because they were not considered relevant for the review. Finally, there were 13 articles for the full text review, which 8 articles were excluded for not meeting the inclusion criteria. The articles taken into consideration were finally 12 (Figure 1. PRISMA).

Discussion

Microorganisms are the primary cause of endodontic infections, and as a result, also of endodontic treatment failure. Bacteria are protected from competing microorganisms, antimicrobial chemicals, and host defenses by biofilm, which also offers pathogens a more ideal habitat to live in and a more effective metabolic diversity. If persistent bacteria are able to remain in the canal for years after therapy and are favored by changes in the environment, they may grow once again and reactivate infection.

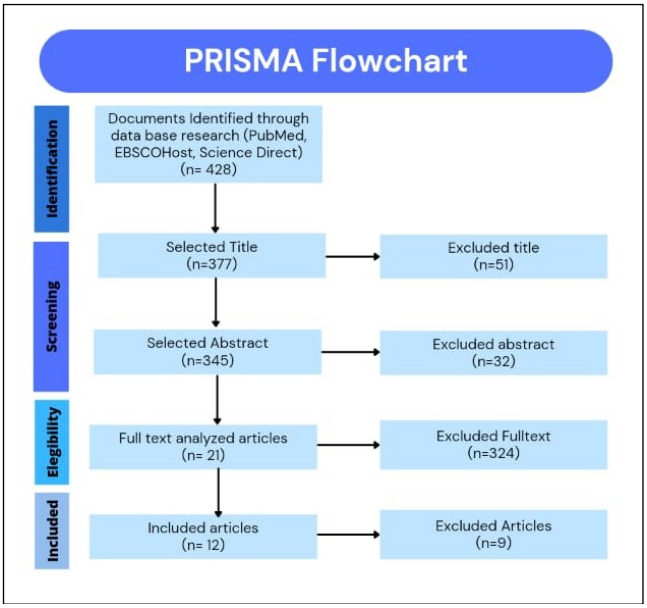

Figure 1. PRISMA flow diagram for this scoping review study.

Bacteria that cause persistent infections are typically found in places that are unaffected by instruments and antibiotic agents, such as isthmuses, lateral canals, and apical ramifications. Bacteria may persist even in the main canal, particularly on the uninstrumented dentinal canal walls. The invasion of dentinal tubules by bacteria has also been considered as a potential source of persistent infection. In the dentinal tubules of the main root canal region, a severe bacterial infection was shown.[4]

It is possible that separating off the sealant or gaps in the filling mass that were not seen will produce leaky channels that enabled fluids from the periradicular tissue to enter tubules and feed the remaining bacteria there. This fluid may have developed a significant concentration of proinflammatory bacterial products. Bacteria living inside tubular structures may use degrading odontoblastic processes, denatured collagen, bacterial cells that die while an infection, intracanal liquids that enter the

tubules through capillary, as food sources. It has been demonstrated that a number of potential endodontic infections may enter and colonize dentinal tubules. It is definitely more challenging or even impossible to treat bacterial cells that have invaded dentinal tubules. It has been demonstrated that treating intratubular infection with an intracanal medicament is efficient. For better and predictable clinical effectiveness, intratubular disinfection-based antimicrobial methods should be encouraged. This emphasizes how crucial it is to achieve appropriate root canal system disinfection for a predictable long-term treatment outcome.[4]

According to studies, endodontic secondary and chronic infections are predominantly polymicrobial and take longer to appear as clinical and radiographic symptoms along with an accumulation of bacteria. It is well known that teeth with sealed canals exhibit minimal or no nutrition, which makes it challenging for bacteria to survive in post-treatment endodontic infections.[5]

F. nucleatum, Prevotella nigrescens and *Treponema denticola* have been reported to have similar prevalence in primary and secondary infections, demonstrating that they are not completely destroyed following endodontic therapy. Asymptomatic teeth with persistent endodontic infection have been discovered to represent *Prevotella intermedia* and *P. gingivalis*. Due to its high prevalence and pathogenicity, *Dialister pneumosintes* was seen with the microbiota of primary endodontic infections and periradicular infections. The presence of certain microorganisms does not, however, guarantee that periapical illness will occur.[5]

The most frequent causes of endodontically treated teeth being extracted were prosthetic (40.8%), periodontal (15.8%), non-restorable cusp/tooth fracture (15.4%), non-restorable caries (10.0%), vertical root fracture (6.4%), endodontic failure (5.4%), and perforation/stripping (2.9%). Additionally, 3.3% of the extractions were due to the patients' insistence. Prosthetic reasons criteria are the teeth with an insufficient crown-to-root ratio, teeth that are considered not able to bear a prosthetic load as an abutment tooth, teeth that are excluded from the prosthetic treatment plan after consultation with a prosthodontist from the Department of Prosthodontics because of poor prognosis.[6]

While the distribution of coronal restorations of the extracted teeth caused by failure of endodontic treatment were 6.0% (n = 17) restored with a post and crown, 22.7% (n = 64) did not have coronal restorations, 28.4% (n = 80) had composite restorations, 18.5% (n = 52) had crown restorations, 16.3% (n = 46) had amalgam restorations, 5.0% (n = 14) had temporary restorations (glass ionomer, zinc phosphate, zinc oxide–eugenol cement, Cavit, etc.), and 2.8% (n = 8) had only post and composite restorations.[6]

It is challenging to remove *Enterococcus faecalis* from root canals since it is a Gram-positive bacterium with a variety of virulence and resistance mechanisms. The prevalence of virulence factors and the antimicrobial resistance of *E. faecalis* isolates of teeth with failure of the endodontic treatment.[7]

E. faecalis was present in 100% of the root canals investigated. All strains were susceptible to amoxicillin + clavulanate. Some strains showed intermediary susceptibility to amoxicillin (5%), azithromycin (20%), benzylpenicillin (5%), ciprofloxacin (15%), doxycycline (5%), erythromycin (75%), tetracycline (10%), and vancomycin (15%). The other antibiotics had less favorable results, including clindamycin (60%), chloramphenicol (5), gentamicin (65%), metronidazole (95%), moxifloxacin (5%), and rifampicin (10%), because strains exhibited resistance.[7]

This study has found distinct prevalence patterns of virulence genes of *Enterococcus spp.* isolates from root canals of teeth with endodontic treatment failure. The ace gene was detected in all clinical isolates. Ace is an adhesin to collagen from *E. faecalis* expressed conditionally after growth in serum or in the presence of collagen. It is an important factor for the establishment of these bacteria in the dentin of infected root canals. It was concluded that *E. faecalis* isolates from persistent endodontic infections showed various degrees of intermediate/total resistance to several antimicrobial agents, with amoxicillin + clavulanate being the most effective. The periodic evaluation of the susceptibility to antibiotics is an important practice for establishing the best drug if their use is necessary. Moreover, the strains showed different patterns of virulence genes of *E. faecalis*, and their monitoring should be encouraged in order to elucidate changes in their resistance

profiles.[7]

The cause of endodontic treatment failure was by persistent lateral canal infection, an exuberant bacterial biofilm colonizing a lateral canal in the apical root segment. One of the major problems of modern endodontic therapy; bacteria located in areas distant from the main root canal can remain unaffected by treatment procedures and maintain disease. In other instances, the tissue in the ramification may become necrotic and invaded by bacteria from the main canal. Whether infection of the lateral canal will cause a lateral periodontitis lesion with or without symptoms will mostly depend on the width of the ramification and the size of the periodontal area in contact with the ramification, which is dictated by the lateral foramen diameter. The larger the lateral canal and the associated lateral foramen, the higher the chances for the infection of the lateral canal to reach a magnitude sufficient to cause significant inflammation in the lateral periodontal ligament.[8]

Moreover, even if the irrigant is driven into ramifications by ultrasonics, it remains to be determined whether it succeeds in exerting optimal cleaning and disinfection effects therein. Effectiveness will depend on the volume, concentration, renewal rate, and time of action of the irrigant in the lateral canal. Because it remains in the canal for longer periods than the irrigating solution, intracanal dressing with calcium hydroxide has the potential to reach these distant areas to exert disinfecting effects.[8]

Most advances in endodontic technology and investment from industry rely on the development of better or safer instruments to shape root canals. The challenge for researchers and clinicians that arises from this problem is to develop strategies, instruments, or substances that can reach those areas and achieve sufficient reduction in the infectious bioburden to permit predictable periradicular healing.[8]

Approximately one-third of endodontically treated teeth are susceptible to the occurrence of periapical lesions in radiographic assessment, which are significantly associated with females, molars, and poor RCT quality, indicating a challenge for endodontists to improve their root canal assessment accuracy.[9]

The majority of root canal fillings were executed poorly. The technical quality of endodontic treatment, which is a major factor in the healing and prevention of apical pathosis, is generally inadequate. The low quality is due to the lack of rubber dam use, inadequate aseptic techniques, and poor root canal cleaning and shaping. All of these factors contribute to the introduction or persistence of microbes and their metabolic products within root canal systems, which results in the development of periradicular disease. This indicate a strong correlation between the technical quality of root canal treatment and apical health.[10]

Adequate root canal fillings (within 2 mm of the radiographic apex) were associated with lower incidence of AP and better success rate when compared to root canal filling limited to the pulp chamber or extruding from the radiographic apex. These findings indicate the great need for improvement of endodontic treatment through emphasis on undergraduate endodontic teaching, more training of general practitioners and the great need for endodontic specialists.[10]

The ultimate goal of the endodontic treatment is either to prevent the development of apical periodontitis or in cases where the disease is already present, to create adequate conditions for periradicular healing. Because apical periodontitis is an infectious disease, the rationale for endodontic treatment is unarguably to eradicate the occurring infection and or to prevent microorganisms from infecting or reinfecting the root canal or the periradicular tissues. [11]

Deficient chemomechanical preparation and insufficient canal system filling are the most prevalent causes of endodontic treatment failure, which is mostly brought on by the persistence of microorganisms (intra and extra radicular). All of this could be caused by inadequate canal preparation, fillings without apical sealing, filtration during the restoration of the clinical crown, untreated canals, as well as iatrogenic factors like apical transport, small access cavities, perforations, false pathways, instrument fractures, etc.[12]

In rare instances, despite the therapy adhering to the highest technical requirements, failure occurs. The disappointing outcome of instances that have been properly treated may be caused by a number of circumstances, according to scientific research. They consist of intrinsic or extrinsic nonmicrobial causes as well as microbial factors, such as extra radicular and/or intraradicular infections.

A significant component in the

pathophysiology of periradicular lesions is played by microorganisms populating the root canal system. If infection is thoroughly eliminated before the root canal system is sealed off, the likelihood of a successful root canal procedure is noticeably increased. However, there is a greater chance that the procedure will not be successful if bacteria are present in the root canal at the time of root filling or if they enter the canal after filling.

Even in teeth that have undergone successful endodontic therapy, bacteria frequently continue to grow in the apical region of the root canal system. Regardless the method and tools used, studies have shown that a portion of the root canal space frequently remains undisturbed during chemomechanical preparation.

During root canal therapy, environmental forces work in the root canal system, allowing some microorganisms to thrive and, depending on a number of conditions, causing failure. A well-treated root canal's reduced nutrient availability and intracanal disinfection methods (chemomechanical preparation and medicine) have an impact on these factors. Therefore, the failure of root canal therapy may be related to the few bacteria species that possess such a capability.

The seepage of tissue fluids can serve as a growing medium for germs if the root canal filling does not completely seal the tooth. Growing bacteria can continue to inflame the periradicular tissues if they reach a significant number and are able to access the periradicular lesion. Anaerobes are more likely to prevail in the microbiota associated with poorly treated teeth, which is more likely to be similar to the microbiota found in untreated teeth and contain a greater number of microbial species (primary infections). This presumably happens because after inadequate cleaning of the root canal system, the germs that caused the initial infection lingered in the canal.

The discovery of *E. faecalis* in infected root-filled canals suggests that *E. faecalis* may be more common in the oral cavity than previously thought. It is highly speculative as to whether the persistent bacteria found in root-filled canals originated from the initial infection and were able to withstand endodontic disinfection procedures, or if they were introduced from the saliva through faulty restorative material and were able to enter the root canal system, where they remained due to the environment-specific conditions that featured a secondary infection.[13]

E. faecalis was not present in the majority of the saliva samples, but this does not rule out the potential that it was present at this site and caused root filling reinfection by coronal microleakage. Microorganisms must have time to enter the root canal and cause a periapical response. In conclusion, Rep-PCR and AP-PCR revealed that *E. faecalis* genotypes isolated from saliva, pulp chambers, and root canals were comparable. These results imply that coronal microleakage may be a potential reason for endodontic failure.[13]

Coronal leakage may be a significant factor in endodontic treatment failure, according to certain reports. Obturated root canals may become polluted from the oral cavity under certain circumstances, including: leakage through the temporary or permanent restorative material, breakdown, fracture, or loss of the temporary or permanent restoration, fracturing of the tooth structure, recurring decay exposing the root canal filling material, or a delay in placing permanent restorations.

The restoration part of endodontic treatment can lead to failure. The amount of leftover dentin following endodontic treatment appears to be a critical component for the tooth's prognosis as evidenced by the direct correlation between the resistance of the tooth and the amount of remaining dentin. The findings of the current study demonstrate that the fracture resistance of 3-wall ferrule designs with coronal heights of 3 to 4 mm is greater than that of 3-wall ferrule designs with coronal heights of 2 mm. This implies that the lingual wall has a significant role in the resistance to fracture in endodontically treated teeth and that a partial ferrule can be more successful if 3 to 4 mm in height of the residual dentin walls can be preserved.[14] If a fracture occurs in a tooth that has had endodontic treatment, it will cause microleakage and cause the endodontic procedure to fail.

Because of the associated qualitative and quantitative changes to the dental substrate, endodontically treated teeth represent a unique scenario. According to recent research, the effectiveness of a treatment depends on two factors: an appropriate restoration that can withstand functional loads placed on the remaining tooth structure, and an efficient

coronal seal that can prevent canal reinfection.[11]

The ideal rehabilitation of posterior teeth that have had endodontic treatment would increase their mechanical resistance and guard against damaging fractures, restoring anatomy and function. According to reports, mandibular molars are the teeth that require endodontic treatment the most and are usually extracted due to secondary caries, cusps, or radicular fractures. In order to lessen the likelihood of fractures that could result in endodontic failure, a restoration procedure that can strengthen the compromised remaining tooth structure is essential. Additionally, conventional MOD cavities were made in the chosen teeth because this clinical situation significantly lowers resistance to cuspal fractures because mesial and distal walls are absent, putting a lot of load on the vestibular and lingual cusps.[15]

The endodontically treated molars' fracture resistance was dramatically enhanced by the addition of glass fibers to direct composite restorations. It is crucial to note that none of the direct restoration methods examined in this study were able to duplicate a sound mandibular molar's fracture resistance. This discovery highlights how crucial it is to preserve as much healthy tissue as possible throughout endodontic and restorative procedures because the structural strength of a tooth is solely dependent on the amount and integrity of its anatomical form. Cuspal covering, preceded by custom cast post and cores, has traditionally been utilized as a solution to improve teeth resistance and load distribution in order to prevent tooth fracture. Currently, good tissue preservation is advocated using a conservative strategy, which is directly related to the fracture resistance of a tooth.[15]

The results of the current study unmistakably shown how a direct composite restoration might significantly increase the fracture resistance of endodontically treated molars with MOD cavities. According to some reports, adhesive restorations have the ability to strengthen the compromised tooth structure because they are better able to transmit and distribute functional stresses through the restorative material-tooth contact. However, by inserting a glass fiber post inside the direct composite restoration, a significant reinforcement effect was attained. Inserting a fiber post within a composite restoration can increase the tooth-restoration complex's capacity to withstand occlusal loads along the major axis of the tooth, could strengthen the endodontically treated tooth's resistance to occlusal loads, and may reduce cuspal deflection, thereby lowering occlusal loads on the tooth thus reducing the possibility of marginal leakage that creates a gap at the tooth-restoration interface with consequent marginal infiltration.[15]

The mechanical and physical characteristics of direct restorative materials should be taken into account to maximize the performance of the restoration-root complex as direct adhesive restorations strengthen the tooth that has been compromised. According to recent studies with posterior teeth, retention can be adequately maintained without dowels by bonding to the coronal root dentin and the interior walls of the pulp chamber dentin. Dowels have been linked to an increase in catastrophic failures. In addition to failing to strengthen the structure, adding a glass-fiber dowel also causes 100% catastrophic failures. Due to its characteristics resembling those of dentin, it has been stated that the glass-fiber reinforced dowel can avoid catastrophic root fractures.[16] To avoid fracture, teeth with lingual ferrule have a better fracture resistance than teeth with buccal ferrule.[17]

All things considered, it would seem that using a dowel is not always advantageous and should be questioned even in severely damaged endodontically treated implants. The patient often is unaware of and undetected by the initial failures of foundation restorations with dowels, such as cyclic opening, infiltration gap, and bacterial leakage, which progress to mobility or complete fracture of the restoration. However, restorations without dowel generally fail without warning (patient will probably consult right away), and they are probably repairable. [16]

Successful root canal therapy depends on the integrity of the canal fillings and thorough canal disinfection. A sufficient amount of root canal filling material must be removed in order to prepare the post-space in order to minimize root canal contamination during post fabrication and installation, which could indirectly impair the stability or retention of ensuing prosthetic crowns. The manner of filling material removal, the quantity of leftover root canal filling, the kind and setting time of the root canal sealer, and the timing of filling material removal are just a few of the variables that might affect the integration or sealability of the remaining obturation material.

Due to the importance of the post space preparation operations and the need for adequate maintenance of the acquired aseptic conditions, the root canal seal must be maintained. In order to sustain the integrity of fillings and a proper apical seal, post spaces should be prepared only when crown fabrications are ready to be installed.[18]

Conclusions

Root canal therapy frequently fails when it is subpar to acceptable standards. Procedure mistakes that prevent the management and prevention of intracanal endodontic infection are the main cause of the failure of root canal therapy in many teeth. The persistence of microbial infection in the root canal system and/or the periradicular area is without a doubt one of the main causes of endodontic failure. The improper restoration that will lead to endodontic failure are root fracture, tooth fracture, and marginal leakage at tooth restoration. Those improper restoration will produce leaky channels that enabled fluids from the periradicular tissue to enter tubules and feed the remaining bacteria and causing the re-infection in that site. This is show that how crucial it is to achieve appropriate root canal system disinfection for a long-term treatment outcome.

Acknowledgements

The authors would like to thank Universitas Padjadjaran to support this work (2203/UN6.3.1/PT.00/2022). All authors contributed significantly to this manuscript.

Declaration of Interest

The authors declare that there is no conflict of interest.

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