World Academy of Science, Engineering and Technology
International Journal of Medical and Health Sciences
Vol:18, No:12, 2024

Antibiotic Treatment of Apical Periodontitis

Ilma Robo, Saimir Heta, Gerhard Nokaj

Abstract**—**The method of treatment and the treatment protocols of apical periodontitis are now known, but the ongoing debate remains on whether or not prescription antibiotics should be given to patients suffering from this type of pathology. In fact, as an indication for prescribing antibiotics, this type of pathology remains between clinical indication and contraindication. This article is of the short-communication type and the sole purpose of analyzing the clinical picture of apical periodontitis and the fact that the appearance and extent of this pathology in the periapex area passes the stage when the host or the immune cells of the organism of the affected individual, react against bacterial factors. It is possible to avoid making decisions on the prescription of systemic antibiotics based on literary sources. In some cases, research in this field about this pathology even indicates endodontic rinsers or irrigants, such as chlorhexidine in typical cases, mainly in persistent apical periodontitis. In times when bacterial resistance is a hot topic in some fields of scientific research, it is important to divide dental pathologies of bacterial origin into those when systemic antibiotic prescriptions must be given and those when every clinical issue is resolved only with endodontic root canal treatment. Even certain sources of published literature show the specifics of the most effective antibiotics against the bacterial flora causing the pathology of apical periodontitis.

Keywords**—**Endodontic treatment, apical periodontitis, antibiotics, chlorhexidine.

I. Introduction

THE vitality test is applied to understand the origin of the pathology, which can be pulpitis or necrosis. Categorically, symptomatic apical periodontitis originates from pulpitis and necrosis, while asymptomatic apical periodontitis originates only from pulpal necrosis. The tooth vitality test can be performed with both warm and cold stimuli [1]-[3]. Diagnosis by palpation can serve for asymptomatic apical periodontitis since the latter in clinical manifestations can soften the cortex of the jaw area where the affected tooth is positioned. Symptomatic apical periodontitis has no clinical signs that can be distinguished by palpation of the affected tooth area [4]-[6]. Percussion diagnosis is the most effective diagnosis in cases of symptomatic apical periodontitis since the touch of the periodontal ligament is associated with the compression of nerve fibers which give the sensitive pain in the case of pathology. On percussion, asymptomatic apical periodontitis may not present pain, or minimal pain may be present [1], [7].

Caution, pulpal symptoms and periradicular symptoms often combine with each other, thus making pulp control difficult. The final diagnosis is made after collecting the data from the pain anamnesis, clinical examination and radiographic examination [7]-[9]. Chronic pulpalgia is the most accurate term used to describe the pain that patients endure, which lasts for several weeks, months or even years, and for which patients are forced to take a pill or two in order to control it; analgesics, two or three times a day. After this phenomenon, real toothache can appear, which does not let you sleep at night [10], [11]. Chronic pulpalgia does not have a target area where the patient can put his finger and can point to the affected tooth in this way, but it has the nature of undefined pain, it can also be irradiating in nature. Chronic pulpalgia is triggered by heat and is not diagnosed by the application of cold. The sensation as if the tooth is higher than the others and the feeling of irritation when the affected tooth is contacted by the antagonist are the most characteristic sensations. The onset of pain when the patient feels tired or when the body falls into a relaxed position in the lying position, are two advertisements of the patient that orient us to the presence of chronic pulpalgia [5], [12].

If the tooth is necrotic, the patient feels throbbing pain in the descent of the plane, or in the case of an abscess or periradicular cyst, the patient feels strong persistent pain both in the ascent and descent of the plane [13]-[16]. In chronic pulpalgia, radiographic images may show thickening of the periodontal ligament, which is an indication that the inflammation is not only within the hard structures of the tooth, i.e. in the pulp, but has also gone outside beyond the apical foramen of the tooth. Root apexes can also present internal resorption as a result of pulpal necrosis and complete periradicular involvement of the tooth root. The tooth with periradicular lesions is referred to as different from others by patients during the percussion test and is painfully induced by the application of heat [17]-[20]. Treatment for chronic pulpalgia is pulp extirpation and endodontic treatment.

Periradicular pain can be as severe as pain originating from the pulp of a tooth. Periradicular pain can occur in both symptomatic apical periodontitis and asymptomatic apical periodontitis [1]-[3]. Symptoms of symptomatic apical periodontitis include very severe periradicular pain that lasts for several days, persistent pain that lasts 24 hours, and the tooth is very painful even to the touch. Throbbing, stabbing pain that in most cases patients ask for the tooth to be extracted, but even if it is decided by the dentist, the patient should know that the pain will continue for another 48 hours post extraction, as a result of the osteitis that caused the infection. There is no presence of edema, but it is simply a tooth protruding from the alveolu [21]-[23].

II. Materials and Methods

The term "apical periodontitis" is generally used to describe

Ilma Robo and Gerhard Nokaj are with University of Medicine, Faculty of Dental Medicine, Department of Therapy, Tiranë, Albania (e-mail: ilmarobo@yshoo.com).

Saimir Heta is with University Hospital Hospital, Pediatric Surgery, Pediatric Surgeon, Tiranë, Albania.

and group together various periapical periodontitis, conditions that originate from pulp disease, but there are a number of different pathological conditions that form this group of disorders. The processes involved are dynamic in nature and involve many complex tissue interactions [1], [28]. Therefore, it is essential that dentists understand the progressive nature of periapical disease, the process, and how and why the various stages occur, so that they can be properly diagnosed and managed. The diagnosis will usually be based on clinical and radiographic manifestations and the results of various tests that may be performed as part of a routine dental examination [24]-[27].

It is also important to recognize that there are many other lesions out there that mimic "apical periodontitis" because of their position and radiographic appearance [28]-[30]. Most cases of apical periodontitis are associated with some form of pulp disease. A number of factors are implicated, which can be grouped as short-term edema, trauma or long-term irritation. Typical short-term edemas are those initiated by dentists during treatment - such as during drying of a cavity. They generally cause an acute inflammatory response, which spontaneously resolves or heals if there is no further irritation. On the other hand, if the edema lasts for a longer period or recurs, then such long-term edema can lead to chronic inflammation and necrosis of the pulp [31]-[33].

Trauma can damage a dental pulp by severing blood vessels at the apex – for example from luxation and avulsion injuries – or by rupturing intrapulpal blood vessels, which will lead to intrapulpal haemorrhage. The pulp may recover and repair or may become necrotic. If the vessels are severed, revascularization may occur depending on the time to repositioning and the stage of development of the apical foramen [34]-[36]. Basically, any break in the integrity of the tooth's outer surface from dental caries, chemical erosion or fissures can result in long-term pulp irritation. In these situations, the dentin is exposed, so bacterial elements can reach the pulp and cause a tissue response. The pulp has a great capacity to withstand bacterial attack and initiate repair as long as there is no direct invasion of the tissue by bacteria. The outcome of any dental treatment will be greatly influenced by the pulp's ability to withstand irritation of bacterial or iatrogenic origin. Without proper treatment, the inflammatory processes of the pulp can spread and eventually necrosis of the pulp will occur. In connection with this spread of inflammation through the apical foramina, apical periodontitis can occur. This process can occur with or without any symptoms. After necrosis, there is no blood supply within the tooth to transport all the important protective cells that would normally be activated by the body in response to an infection [36]-[39].

The result of any dental treatment will be very high. Torabinejad et al. also mention a study in which the pulps were lacerated apically in monkeys, the pulp tissue was left inside the canals, the canals were infected with plaques, and the animals were left exposed in the oral cavity for 10–14 days [14]. One set of teeth was then closed with a zinc oxide-eugenol temporary filling while the other set was left open. Radiographs were taken and the teeth were examined histologically after different time intervals. In the 'closed' group, the root canals were void of pulp tissue (i.e., 'pulp-free') after only 1 month, while the 'open' group took 2 months to become pulp-free. The different time interval was most likely related to different growth conditions in "closed" channels favoring anaerobes, which are more virulent and destructive [39]-[41].

III. Results

When considering the progression of pulp diseases through these similar stages, the stimulus is usually bacteria, as described above. This is the body's defense response to the irritation created by the bacteria themselves as well as the irritation caused by their products such as exotoxins and lipopolysaccharides [11]-[13]. Studies have shown that in both germ-free and conventional (germ-fed) mice, pulpal degeneration and necrosis following non-preserved exposures to the oral environment only occurred when germs were present and might contaminate the tooth and pulp [11]-[13]. Later research revealed a direct correlation between bacteria and periapical tissue reactions [21]-[23].

Invasion of the root canal and periapical tissues will generally not become inflamed until the root canal becomes infected. When microorganisms were found throughout the length of the root canal, the periapical inflammatory reaction was severe. Bhangdia et al. confirmed that periapical lesions were found in 18 of 19 cases where microbes were present in the canal, and the size of the periapical radiolucency was directly related to the number of strains that could be isolated from the affected tooth [42]. Haapasalo et al. [11] also confirmed in a study that no periapical inflammatory reaction occurred in the absence of bacteria inside the root canals and John et al. reported no inflammatory reaction associated with noninfected devitalized teeth, except in two cases where canals were instrumented beyond the apical foramen while the pulp was being removed [15].

In these two cases, periapical inflammation was considered as a direct result of mechanical damage during the devitalization procedure. Although bacteria are the most common cause of pulp disease, dental pulps can also become necrotic for other reasons. A typical example is after trauma when the blood vessels at the apex are severed, as described above. In such cases, the presence of a necrotic pulp does not in itself imply that a chronic periapical inflammatory reaction will occur. Necrotic debris alone, although stimulating phagocytosis and tissue repair, will not produce sufficient irritation to sustain an inflammatory response in the periapical region. Periapical inflammation is a direct result of interactions between bacteria in an untreated infected root canal system. and the host's defense or immune system. It begins as an acute inflammatory response, but is a dynamic situation that can change spontaneously throughout the disease process. Since there is no longer a blood supply to a necrotic pulp or to the root canal system in a pulpless tooth, host defense cells cannot reach the source of irritation (i.e., the bacteria in the canal) and therefore the body is unable to eliminate the infection. Therefore, a chronic inflammatory response develops in the periapical region and intra-canal bacteria survive on nutrients obtained

from tissue fluid and inflammatory exudate that penetrate the root-canal system through the apex [41]-[43].

Once an infection is established within the root canal system, the number of bacteria will gradually increase through the mechanisms of normal cell reproduction and proliferation [1]-[3]. Nutrient conditions in each canal may vary over time, which may explain the different rates of development of periapical responses and may also explain why there are different numbers of bacteria that can be recovered from root canals during sampling procedures. Therefore, during the evaluation of the teeth, it is mandatory to examine and diagnose the status of the pulp and periapical tissues. In addition, clinicians must also determine the cause of the disease. Apical periodontitis can develop without an infected root canal. Traumatic occlusion is an example where inflammation is caused by continuous irritation of the periodontal ligament during function as a result of premature occlusal contact or occlusal interference during lateral and/or protrusive movements of the mandible [31]-[33].

Trauma is a long-term irritation that causes a bone resorbing process. Other forms of trauma can cause apical periodontitis – such as luxation injuries and avulsion when the tooth is displaced by a violent force. In these situations, the periodontal ligament is directly damaged and is likely to be torn or completely detached. Acute inflammation is the first response to injury and then repair will usually follow provided the tooth is correctly repositioned and stabilized [21]-[23].

If the treatment is not adequate, then chronic inflammation may result. Periapical radiographs may falsely suggest an area of apical periodontitis without root system infection when there is extensive periodontal disease and the pocket has extended beyond the level of the root apex. In this situation, significant breakdown of the supporting tissues and bone loss creates a radiolucency, which overlaps the periapical region giving the appearance of apical periodontitis. This situation is one that can occur when the periodontal pocket actually encompasses the apical foramen and the blood supply to the pulp is interrupted - this situation leads to pulp necrosis and infection of the root canal system [41]-[43]. Pulpitis can also cause symptoms of apical periodontitis typically. It is more common with irreversible pulpitis, but can also occur with reversible pulpitis. It should be noted that both forms of pulpitis are inflammatory conditions of the pulp. However, pulpitis is usually caused by the presence of microorganisms somewhere within the tooth structure, but not necessarily within the pulp tissue itself, although in cases of irreversible pulpitis a bacterial invasion of the tissue is expected. The presence of bacteria can restart the onset of apical periodontitis infection as it is shown by Paterson et al. [43]. However, a more likely scenario is that the apical periodontitis is simply an 'extension' of an inflammatory pulpal process, which is contained by the confines of the root canal walls and has only one direction to follow as it spreads - that is through the foramen apical [43]-[45].

IV. Discussions

The terms "acute" and "chronic" are used in this classification as indicators of clinical conditions based on the patient's perception of their pain; thus, they were not used from a histological perspective as it is impossible to know the true histological state of the tissues and this is likely to vary from one case to another, or even from time to time with the same tooth [1]-[3]. Therefore, in this classification, "acute" is used to describe a case with moderate to severe symptoms, while "chronic" indicates either no symptoms or only mild symptoms. Some other suggested classifications use terms such as "symptomatic apical periodontitis" and "asymptomatic apical periodontitis", but these can be confusing and many conditions can be symptomatic and asymptomatic at different stages of the disease process [15]-[18].

Moreover, these terms do not take into account the different pathological entities that occur during the progression of periapical disease in its various stages. Each stage of periapical disease should be considered as part of a continuum of stages that occur during the development and progression of disease processes. Thus, the use of the terms "symptomatic apical periodontitis" and "asymptomatic apical periodontitis" do not help clinicians distinguish between different stages of the disease. The periapical disease process is dynamic in nature and therefore each condition may progress to some other condition [7]-[13]. Pulpal and periapical diseases are progressive in nature and therefore, signs and symptoms along with clinical and radiographic findings will vary according to the stage of the disease at the time of examination. If no interventional treatment is offered, periapical disease will usually follow a sequence of events. An infected root canal system is a reservoir for the bacteria that cause apical periodontitis and will persist until appropriate treatment is provided. The first goal is unattainable due to the lack of blood supply in the canal, while the second is generally successful, but only until local or systemic changes occur to alter the "equilibrium" situation described above [41]-[43].

Such a reaction can also be caused by trauma, or by endodontic instrumentation procedures and irrigating materials. Acute apical periodontitis occurs with abscess formation (i.e., primary apical abscess), development of a sinus tract (i.e., chronic apical abscess), spread of infection through bone and/or soft tissue (i.e., cellulitis), cyst formation, or may become chronic (i.e., chronic apical periodontitis). Healing will only occur if no further irritation occurs to maintain the reaction and if there are no microorganisms inside the canal - such as after some traumatic incident or after endodontic treatment is completed. Acute secondary apical periodontitis is an acute exacerbation of an existing lesion of chronic apical periodontitis. This can occur in the form of an abscess (secondary apical abscess) when bacteria migrate from the root canal to infect the periapical tissues, although other local or systemic changes can also cause an acute exacerbation of the inflammation [41]-[45].

If treatment is still not provided, then there will be a constant presence of irritants in the apical part of the root canal system, so the initial acute inflammation gradually turns into a chronic inflammatory reaction, known histologically as periapical granuloma. Clinically, this is usually seen as an asymptomatic radiolucency and reflects a state of quiescence, or 'equilibrium'

with the microbes confined to the canal. A periapical granuloma can remain dormant for long periods of time, but the balance can be disturbed at any time by any factor that favors the growth and/or migration of the microbial flora. Bacteria can then migrate from the canal to the periapical tissues and the chronic inflammation will become acute, presenting as secondary acute apical periodontitis or as a secondary apical abscess with clinical signs and symptoms of varying intensity [1]-[3]. At this stage of the disease process, microorganisms can be found in extra-radicular tissues and bone resorption occurs with rapid expansion of the radiolucency. Some patients may not experience symptoms that are severe enough to require treatment, so the acute reaction may again take on one of several possible options, such as: further intensification, abscess formation (secondary apical abscess), development of a sinus tract (chronic apical abscess), spread through bone and/or soft tissue (cellulitis), cyst formation, or it may become chronic apical periodontitis again. As described above, periapical disease is usually the result of pulp disease and therefore signs and symptoms of periapical disease will be present in association with signs and symptoms of concurrent pulp disease. Therefore, the diagnosis should include an assessment of the condition of the pulp (or root canal) and the periapical region, together with an assessment of the cause of these diseases. The following discussion is limited to signs and symptoms of periapical conditions only, and readers should assume that the relevant pulpal (or root canal) condition and its cause have also been diagnosed [41]-[43].

V. Conclusions

The size of the radiolucency is also irrelevant to the histological status of the tissue as both small and large lesions can be granulomas, abscesses, or cysts. Since granulomatous and radicular cysts are difficult to differentially diagnose, and since the initial treatment for each is identical (i.e., orthograde endodontic treatment), they can be classified clinically under the general term "chronic apical periodontitis." These disease processes are dynamic and pathologic entities are interchangeable, so this more generalized diagnostic term is often more valid for clinical use. Unfortunately, further confusion can arise if other conditions are considered, such as apical abscesses, extra-radicular infections, and periapical wounds, as these conditions also have similar radiographic and clinical appearances.

List of Abbreviations

Not applicable.

Declarations

Ethics Declarations

Ethics Approval and Consent to Participate

As the authors of the article, we state that there is no violation of the code of ethics during the realization of this article. Consent in the participation of patients in the study was performed with the signature of the patients themselves, procedures based on national protocols.

Availability of Data and Materials

The datasets analyzed during the current study are available from the corresponding author.

Competing Interests

The authors declare that they have no competing interests.

Funding

Not applicable.

Authors' Contributions

IR and GN collected the scientific data and wrote the manuscript. SH revised and edited the manuscript. Literature research was conducted by SH. IR and GN collected the scientific data. All authors read and approved the final manuscript.

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