Endodontic-Periodontal Lesions

Evidence-Based Multidisciplinary Clinical Management

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Contents

1 Lesions of Endodontic Periodontal Origin

Igor Tsesis, Carlos E. Nemcovsky, Joseph Nissan, and Eyal Rosen

The association of the degenerative changes in the pulp tissues and periodontal disease presents a clinical and conceptual dilemma ever since it was first described in the beginning of the twentieth century by Cahn (1927) [1]. Multiple investigations on that topic were later on published. Being one of the earliest published by Simring and Goldberg in 1964 [2], claiming that pulpal and periodontal problems are responsible for more than 50% of tooth mortality [2, 3].

During the following years many possible etiologies, definitions, classifications, and management alternatives based on different paradigms have been proposed. As a consequence, the understanding of this clinical scenario is a matter for ongoing debate.

Due to the close relationship between endodontic and periodontal diseases, Weine (1972) [4] suggested that endodontics is actually "periapical periodontics." However, this term, like many others' proposed definitions, has not been widely accepted.

Regardless of the exact definition and selected characterization scheme, the etiology of these endodontic-periodontal lesions derives from the etiologies of the associated endodontic and periodontal diseases. The relative parts of the endodontic and of the periodontal associated diseases in the ensuing endodontic-periodontal lesion vary depending on the nature and pathogenesis of the endodontic-periodontal lesion. It ranges from solitary endodontic lesions, in which most, if not the entire etiology, is of endodontic origin, to solitary periodontal lesion, in which the etiology is of periodontal origin only.

Root canal space infection is the main etiology of apical periodontitis [5]. The advance of the disease involves inflammatory reaction of the peri-radicular tissues and periodontal ligamental space [6].

Periodontal disease, on the other hand, involves marginal periodontium and results in the progressive loss of the supportive tissues [7]. While the etiology of both is bacterial, their clinical presentation is different [8–11].

Endodontic disease initiates with the involvement of dental pulp and clinical signs and symptoms may include sensitivity to thermal stimuli

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and radiographic presentation of damage to the hard tissue of the tooth such as carries, trauma, or extensive restoration. If not treated, the pulp becomes progressively contaminated and periradical bone resorption becomes evident radiographically (Fig. 1.1). This process may remain asymptomatic or result in purulent inflammation, chronic or acute [12].

Infection is the main etiology for periodontal disease [13, 14]. Perio-pathogenic bacterial plaque together with calculus accumulation on the external root surfaces progress apically leading to gingival marginal inflammation that may progress to deeper supporting periodontal structures. Endotoxins from bacterial plaque together with inflammatory mediators lead to destruction of gingival connective tissue, periodontal ligament, and alveolar bone [15] (Fig. 1.2).

The transition of an endodontic disease or of a periodontal disease into a combined endodontic-periodontal disease depends on the anatomical communications between the root canal space and of the marginal periodontium.

There are multiple routes of communication between the root canal space and marginal periodontium [8, 11, 16–23]. The main root canal opening (apical foramen) is the main pathway between the infected pulp in periodontal tissues. In addition, open dentinal tubuli and lateral canals may contain bacteria and had been

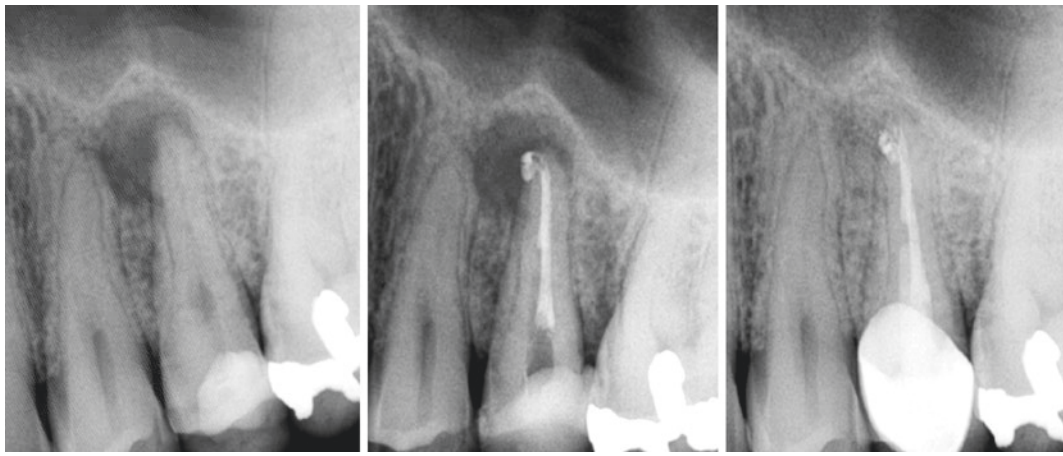

Fig. 1.1 Second maxillary premolar—the patient presented with a sensitivity to percussion: preoperative radiograph—extensive coronal restoration and radiolucent periapical area; radiograph immediately after root canal treatment, resolution of the periapical lesion at the 1 year follow-up

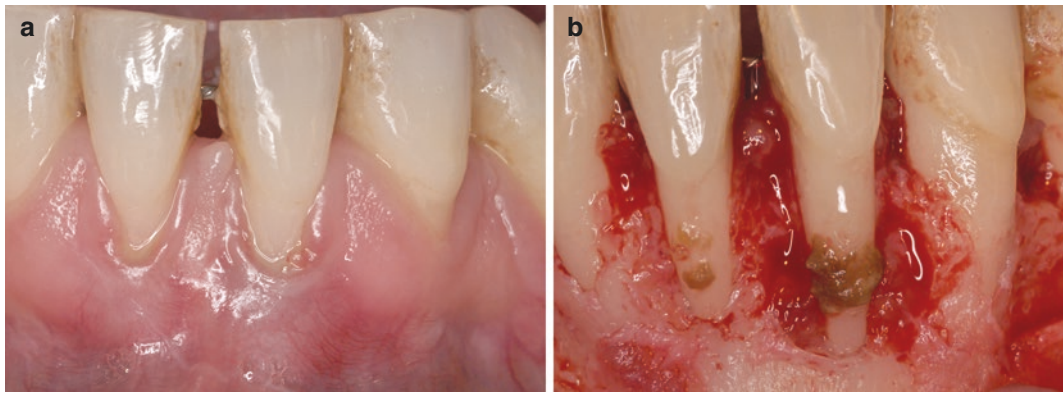

Fig. 1.2 (**a**) Anterior mandibular teeth with severe periodontal disease: gingival recession and deep periodontal pockets. (**b**) Following flap elevation, calculus on root surface with large loss of periodontal support is evident

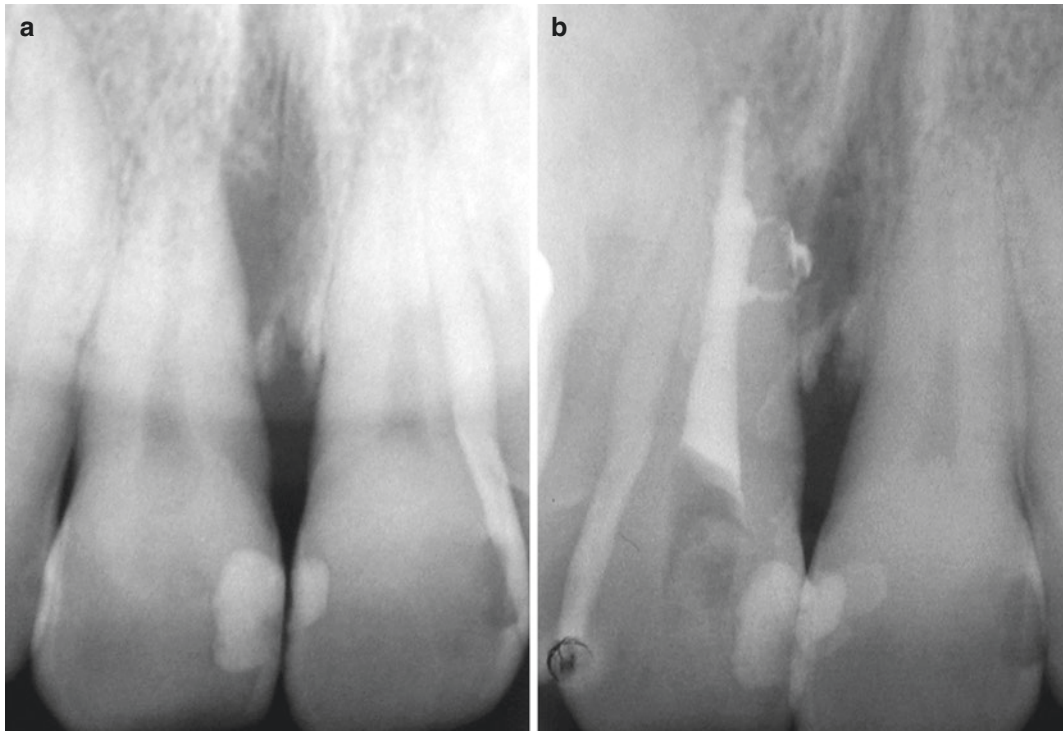

Fig. 1.3 Central maxillary incisor with pulp necrosis and periapical lesion (**a**). Following root canal filling: lateral canals communicating between the main root canal and periapical lesion are clearly seen (**b**)

reported as possible communication routes for bacteria [8, 11, 16–23] (Fig. 1.3). In addition, various pathological conditions, such as root fractures, perforations, resorption, or anatomical anomalies, may present a pathway for the bacteria [24]. By these communications the bacteria from the root canal space may contaminate and infect the marginal periodontium and vice versa [2, 5, 10, 15, 25].

The unique etiology and pathogenesis of the endodontic-periodontal disease dictates the required management plan of these challenging clinical cases and the prognosis of the affected teeth.

The management of the pulpal disease is almost exclusively based on the elimination of the bacteria from the infected root canal space and reinfection prevention [26].

Unlike in endodontic disease, in periodontally affected teeth, bacteria reside on the exposed root surfaces in the gingival sulcus and periodontal pockets [8, 9, 14, 15, 25]. Accordingly, the management of the periodontal disease is different, consisting on plaque and calculus elimination to render the root surface biocompatible that may be combined with periodontal reconstructive procedures to enhance periodontal support [27] (Fig. 1.4).

The diagnosis of endodontic-periodontal lesions may be intriguing, since both periodontal and endodontic diseases have similar clinical and radiographic symptoms and may mimic each other. Moreover, the simultaneous occurrence of the pulpal and periodontal pathology can complicate diagnosis and treatment and compromise the prognosis of the involved teeth.

While in most cases the manifestation of the periodontal and endodontic diseases is clearly distinct, there are certain clinical scenarios when the signs and symptoms may be confusing, making the final diagnosis complicated and subsequently result in the wrong treatment choice [8, 23, 28, 29] (Fig. 1.5).

Misdiagnosis and subsequent wrong treatment choice may ultimately result in tooth extraction

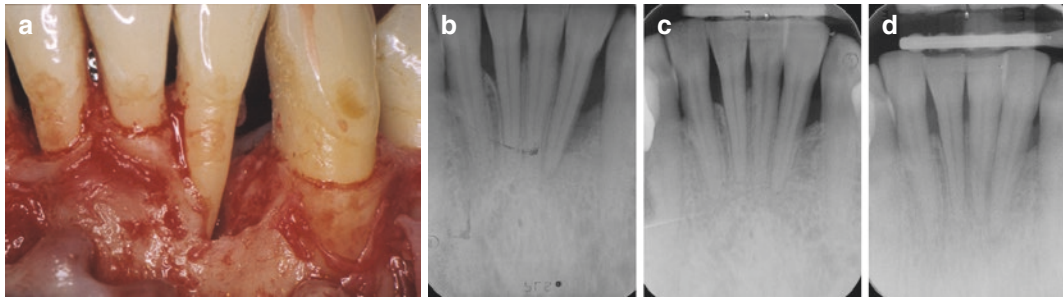

Fig. 1.4 (**a**, **b**) Clinical and radiographic (respectively) aspect of lower anterior teeth shows generalized loss of periodontal support, especially on distal aspect of lateral left incisor. (**c**) Radiograph taken 1 year following reconstructive periodontal treatment with use of enamel matrix proteins derivative, enhanced periodontal support may be appreciated in most involved teeth, note large bone fill on distal aspect of lateral left incisor. (**d**) Radiograph taken 3 years following periodontal surgical treatment, further enhancement of periodontal support may be appreciated in most involved teeth

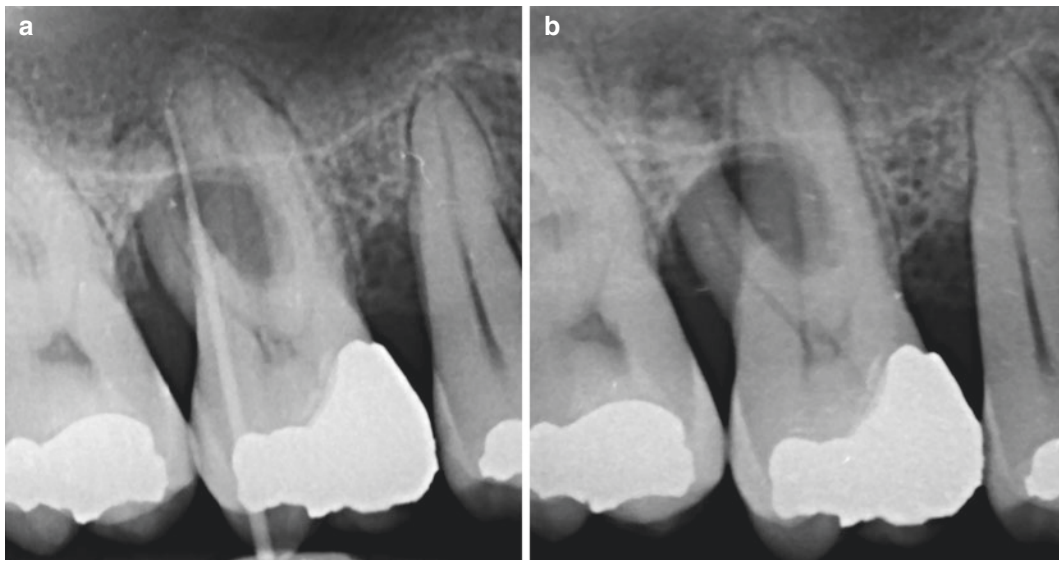

Fig. 1.5 First maxillary molar: the tooth was diagnosed as having a necrotic and infected pulp, chronic apical abscess with a sinus tract traced to the disto-buccal root using a gutta-percha cone (**a**), peri-radicular bone resorption, and advanced periodontal disease (**b**)

[28, 30, 31]. Numerous reports in the literature have presented possible options for the diagnosis and treatment of this condition [32].

Following treatment of teeth with endodontic-periodontal lesions, appropriate restorative plan is crucial for the prognosis of the teeth.

Endodontic as well as periodontal pathologies are closely related to the restorative aspects of dentistry. Any restorative procedure may cause some degree of pulp damage, and at the same time faulty restoration may result in periodontal involvement [4]. Besides, all root canal treated teeth require some type of coronal restoration, and in cases of severe damage to the tooth hard tissues, there may be even needs for surgical treatment. In consequence, restoration of teeth with endo-perio lesion is challenging due to uncertain prognosis while tooth structure preservation and proper restorative materials and techniques are essential for long-term success. Permanent restoration, direct or indirect, should be placed as soon as possible after the completion

of root canal therapy due to the fact that coronal leakage is considered as one of the important factors that influence tooth survival during and after endo-perio treatment.

From the above mentioned it is clear that the topic of endodontic- periodontal lesion is ultimately relevant to all areas of dentistry.

The comprehensive multidisciplinary approach is of outmost importance in the diagnosis and management of the endodontic-periodontal lesions in order to provide the best chance of providing an optimal treatment.

A simple and clinically relevant classification and appropriate treatment alternatives and considerations together with biological perspectives of the endodontic periodontal lesions are presented in the following book chapters.

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2 Etiology and Classification of Endodontic-Periodontal Lesions

Eyal Rosen, Carlos E. Nemcovsky, Joseph Nissan, and Igor Tsesis

2.1 Introduction

The periodontium and the dental pulp are closely associated, sharing embryonic, functional, and anatomical interrelationships. A century ago, Turner and Drew [1] described for the first time the effect of periodontal diseases on the pulp tissue. Then, in 1964 Simiring and Goldberg [2] described a disease of the periodontium caused by a pulpal disease, termed "Retrograde periodontitis." They stated that unlike marginal periodontitis, in which the disease proceeds from the gingival margin as the source of infection toward the tooth apex, in "retrograde periodontitis" the pulp is the source of the pathogens affecting the periodontium, potentially causing a periodontal disease, contributing to a periodontal disease, or preventing healing of a periodontal disease [2]. Simiring and Goldberg [2] also explained that these two processes generally exist side by side, and may have the same signs and symptoms. Thus, they may be difficult to distinguish [2].

The traditional classifications of endodontic-periodontal lesions are usually based on the origin of the infection, i.e., primary endodontic lesions, primary periodontal lesions, and different combinations of the above. However, due to the interrelationships of these two entities it had been claimed that these classifications are too academic and theoretical and may not be clinically practical.

This chapter will review the etiological factors of endodontic-periodontal lesions, the common classifications of these pathologies, and will suggest a novel and clinically practical classification for these intriguing clinical scenarios.

2.2 Pulpal-Periodontal Routes of Communication

Although it may seem that the dental pulp and the periodontium are two distinct tissues, there are many potential routs in which these tissues can communicate [3–7], such as the apical foramen [2, 8]; exposed dentinal tubules [3]; lateral and accessory canals [4]; certain anatomical

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variations [9, 10]; or pathological conditions such as root perforations and fractures [11, 12].

The apical foramen is the main route of communication between the pulp and the periodontal tissues. In case of pulp infection, the bacteria and their by-products may exit through the apical foramen causing periapical inflammation. In certain cases, the associated periapical tissue destruction can spread coronally and involve the marginal periodontium. On the other hand, in case of severe periodontal disease with deep periodontal pockets the vice versa may happen [2, 8].

Dentinal tubuli are another possible route for the communication between the root canal system and periodontium. Between 13,700 and 32,300 dentinal tubules per square millimeter may be present in the cervical dentin [5]. Therefore, periodontal disease and procedures such as scaling and root planning may lead to exposed dentin [3], and allow the pulp tissue to communicate with the external root surface and the periodontium.

Lateral and accessory canals can be present along the root including the cervical areas of the tooth. Gutmann [4] studied the external root surface of molars to determine patent accessory canals, and reported that accessory canals were demonstrated in the furcation region in 28% of the teeth. The presence of such patent accessory canals is a potential pathway for the spread of bacteria and toxic substances resulting in inflammatory process in the periodontal tissues [6].

Anatomical variations such as palatogingival groove [9], a relatively common developmental anomaly in maxillary incisors, or the presence of gaps between the enamel and cementum with exposed dentin [10], may provide favorable conditions for communication between the periodontal and the pulpal tissues, for plaque retention, and for periodontal disease progression toward the apical areas of the root that may eventually involve the pulp [9, 10].

Treatment complications such as root perforations or root originated fractures open up a significant communication passage between the root canal system and the periodontal tissues. In case of infection, these complications can lead to the formation of endodontic-periodontal lesions [11, 12].

2.3 The Etiology of Endodontic-Periodontal Lesions

Both endodontic and periodontal diseases are multifactorial with many demographic [13, 14], anatomical [4, 5, 9, 10, 15], genetic [16, 17], systemic [18, 19], behavioral [20, 21], and other potential contributing factors. However, since both endodontic [22] and periodontal diseases [23] are primarily associated with infection, even in the presence of these contributing factors, a disease will develop mainly in the presence of infection [22, 23].

In 1965, Kakehashi et al. [24] evaluated the pathological changes resulting from untreated experimental pulp exposures in germ-free rats as compared with conventional rats with normal oral flora. In the normal rats, pulp necrosis and abscess formation occurred in all specimens. In contrast, no devitalized pulps or abscesses were found in the germ-free animals, thus demonstrating that bacterial infection is necessary for the development of an endodontic periapical disease [24]. In accordance, numerous studies have demonstrated that the basic etiology of periodontal diseases is also a bacterial infection [25, 26].

However, even when the conditions developed allow the progression of an endodontic-periodontal disease, for example, following root perforation or development of root originated fracture, it may take time until bacteria colonize the pulpal-periodontal communication site, and additional time until an associated pathology develops [27].

Traditionally, one of the most intriguing questions has been how endodontic and periodontal microorganisms link together to form an endodontic-periodontal disease. Zehnder et al. [28] claimed that although the periodontal pocket presents a greater variety of microorganisms than the infected pulp, when an endodontic infection is caused by severe periodontitis, all bacterial species found within the root canals are also present in the periodontal pocket. These similarities in the microflora of these two niches were also reported by Kerekes and Olsen [29], supporting the concept that infection may spread from one niche to the other.

However, other reports suggested that there are fundamental differences between the microflora recovered from infected root canals and from periodontal pockets, perhaps because coccus and rods predominate within infected root canals while spirochetes and rods predominate within periodontal pockets [30, 31].

Rôças et al. [32] assessed the occurrence of the so-called "red complex bacteria" (Porphyromonas gingivalis, Bacteroides forsythus, and Treponema denticola) that may be associated with severe periodontal diseases, in root canal infections. They found that at least one member of the red complex was found in 33 of 50 cases, and concluded that since the "red complex" bacteria are known oral pathogens, their manifestation in root canal infections suggests that they may play a role in the pathogenesis of periradicular diseases [32].

Nevertheless, in recent years as our understanding of the ecology of biofilms improved, these traditional controversies seem to become redundant. Despite the commonly held perception of oral bacteria as solitary surviving microorganisms, in the different oral niches, bacteria form complex biofilm communities. These biofilms are specialized ecological communities, where the bacteria use different mechanisms to align their activity within the community in order to adopt to the constantly changing environmental conditions. These adaptations include dynamic changes in the biofilm species compositions and proportions within the community [33–36]. Thus, exposure of a specific biofilm to a different ecological niche, like exposure of endodontic biofilm to the periodontium and vice versa, would initiate these adaptation processes, altering these two communities to align together and to spread from one niche to the other.

2.4 Traditional Classifications of Endodontic-Periodontal Lesions

Many classifications were suggested along the years to describe the versatility of these clinical scenarios. Each of these was based on different characteristics of the pathological process, such as: classifications that were based on the diagnosis, prognosis, and treatment of these lesions [7]; classifications that were based on pathologic relationship [37]; or classifications that were based on treatment [38].

Simon et al. [7] were the first to suggest a classification of endodontic-periodontal lesions that was mainly based on diagnosis, prognosis, and treatment. This classification included primary endodontic lesions, primary periodontal lesions, primary endodontic lesions with secondary periodontal involvement, primary periodontal lesions with secondary endodontic involvement, and true combined lesions.

According to Simon et al. [7], *Primary endodontic lesions* clinically manifest with a possible drainage from the gingival sulcus, swelling in attached gingiva, and some discomfort. The necrotic pulp may be associated with a sinus tract extending from the root apex along the root surface, to exit at the cervical line. The radiographic examination would usually show bone loss, appearing as a radiolucency along the entire root length. Other clinical presentations are also possible such as in multi-rooted teeth, were the sinus tract may drain into the bifurcation area with an associated radiographic appearance of periodontal involvement [39].

After some time plaque accumulates at the gingival margin which could result in marginal periodontitis, and then this primary endodontic disease may become secondarily involved with periodontal destruction. Simon termed this condition as *Primary endodontic lesions with secondary periodontal involvement* [7, 39]. When this occurs, both endodontic and periodontal therapy are required and the tooth prognosis depends mainly on the success of the periodontal treatment, assuming that the endodontic procedures are usually more predictable [7, 39].

Simon et al. [7, 39] classified *Primary periodontal lesions* as lesions that are caused by a periodontal disease that gradually progresses along the root surface toward the apical region. The diagnosis is based on common periodontal examinations such as probing depth measurement.

Pulp vitality examination should confirm that the pulp is vital. Thus, since the pulp in still vital, the prognosis in this scenario primarily depends upon the efficacy of the periodontal treatment [7, 39]. According to Simon et al. [7, 39], as the periodontal pocket progresses toward the apical areas of the root, lateral canals and eventually the apical foramen may become exposed to the periodontal microflora which can lead to pulp necrosis. This condition was termed *Primary periodontal lesions with secondary endodontic involvement* [7, 39]. Simon et al. pointed out that diagnostically, these lesions may cause a dilemma as they may be indistinguishable from primary endodontic lesions with secondary periodontic involvement. It should be noted that the exact association between the progression of a periodontal disease and its effect on the condition of the dental pulp is a matter of long-lasting debate [40, 41]. However, modern studies reveled that in the presence of a significant chronic periodontal disease, pulp inflammation and necrosis do occur [41].

According to Simon's classification [39] *True combined lesions* may develop when an endodontic periapical lesion progresses in a tooth that is also periodontally involved, until these two pathologies merge along the root surface. Again, this condition may also pose a significant diagnostic dilemma as its clinical and radiographic presentations are indistinguishable from other previously mentioned lesion types. From the treatment and prognosis aspects, periapical healing is probable following endodontic treatment. However, the periodontal disease may or may not respond to periodontal treatment, depending on the severity of the periodontal disease [39].

Following the publication of Simon's classification, in 1982 Guldener and Langeland [37] suggested a new classification that was based on the pathologic relationship: endodontic-periodontal lesion, periodontal-endodontic lesion, and combined lesions.

In 1990 Belk and Gutmann [42] suggested to add to the previously presented Simons's classification an additional classification, termed *Concomitant pulpal-periodontal lesion.* In this clinical scenario, both endodontic and periodontal diseases coexist in the same tooth, with no evidence that either disease has influenced the other [42].

Then in 1996 Torabinejad and Trope [38] suggested another classification that was based on the treatment point of view: endodontic origin, periodontal origin, combined endo-perio lesions, separate endodontic and periodontal lesions, lesions with communication, lesions with no communication.

Most of these classifications agreed on the possible origins of these lesions as some of these are of endodontic origin, some are of periodontal origin, and some are different combinations of the above [7, 37, 38]. However, there are significant disagreements among the traditional classification schemes as to how these pathologies should be further subdivided into additional subgroups as the pathology progresses.

Accurate diagnosis of the exact nature of the lesion is crucial for an effective treatment, and to assess the tooth prognosis [8, 43, 44]. Generally, when it is a lesion of purely endodontic origin, the treatment of choice would be endodontic, and the prognosis would mainly depend on the ability to endodontically treat the disease. When the lesion is purely of periodontal etiology, a periodontal treatment is the main treatment of choice and the feasibility of this periodontal treatment would determine the tooth prognosis. In all other cases, both endodontic and periodontal treatments are required and the ability to control and treat both diseases would determine the tooth prognosis [8, 43, 44].

In this context, the diagnosis of primary endodontic lesions without periodontal involvement and primary periodontal lesions without endodontic involvement is usually straightforward and feasible. In primary endodontic lesions, the pulp is non-vital and infected, and on the other hand, in a tooth with primary periodontal lesion, the pulp is vital. However, a combined disease such as primary endodontic lesion with secondary periodontal involvement, primary periodontal disease with secondary endodontic involvement, concomitant lesions, or true combined lesions may all radiographically and clinically look alike, especially in advanced stages of the disease [43, 44]. Thus, it seems that from the treatment

and prognosis aspects it is not practical to use the traditional categorization schemes.

Two major groups of endodontic-periodontal lesions may be identified according to the etiological origin: pathological endo-perio lesions—resulting from the disease of the pulp or periodontium—and iatrogenic endo-perio lesions—representing a complication of the treatment that results in an artificial communication between the root canal space and marginal periodontium. Classical example of iatrogenic endo-perio lesion can be iatrogenic root perforation or iatrogenic root fractures.

Thus, we suggest to use a three-component categorization scheme of endodontic-periodontal lesions:

1. Purely endodontic lesion: when the pulp is necrotic and infected, and there is a draining sinus tract coronally through the periodontal ligament into the gingival sulcus.
2. Purely periodontal lesion: when a deep periodontal lesion involves most of the root surface, and the dental pulp is vital.
3. Endodontic-periodontal lesion: when the pulp is necrotic and infected, and there is a deep periodontal pocket.

For lesions of purely endodontic origin, the clinical manifestation and the diagnosis is usually consistent with chronic or acute apical abscess. The proper management of the disease will include eradication of the bacterial infection by a root canal treatment, and the tooth prognosis will depend mainly on the efficacy of the endodontic treatment.

Purely periodontal lesions are clinically consistent with severe periodontal disease, involving a great part of the root/s surface. The management of these lesions is by periodontal treatment and there is no need for endodontic treatment. Tooth prognosis mainly depends on the efficacy of the periodontal treatment.

Endodontic-periodontal lesions are cases with long-standing severe infection that involves both the root canal space and the marginal periodontium. In these cases, although the prognosis depends primarily on the severity of the periodontal disease, it is usually impossible to initially assess the contribution of the endodontic infection to clinical manifestation of this combined disease. On the other hand, the endodontic treatment is considered more predictable than the periodontal. Thus, it is advised to initially perform a root canal treatment, and only initial, nonsurgical periodontal procedures such as scaling and root planing. Following, it is advised to control healing for 3–4 months to monitor resolution of the endodontic infection and its effect on the tooth periodontal status. Provided endodontic improvement, based on the more specific and accurate understanding of the periodontal status of the tooth, a comprehensive periodontal treatment strategy may be planned.

In cases involving teeth with previous endodontic treatment, the diagnosis and classification can be challenging. In these cases, since pulp vitality tests cannot be performed, it is more difficult to clinically assess the condition of the pulp space and its involvement in the disease. Therefore, in case of a doubt, when it is suspected that the root canal treated pulp space is infected, the cases should be endodontically retreated.

2.5 Conclusions

- A close anatomical association between endodontic and periodontal tissues may lead to spread of the infection between the root canal and marginal periodontium.
- Classification of the endodontic-periodontal lesions should be based on the primary etiological factor of the pathology and clinical presentation as purely endodontic, purely periodontal or endodontic-periodontal lesions.

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3 Endodontic Considerations in the Management of Endodontic-Periodontal Lesions

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Objectives

1. Understand endodontic diagnosis and how it relates to periodontal lesions.
2. Understand the pulpodentinal complex and how its dynamics can influence the development of endo-perio lesions.
3. Become familiar with a variety of endo-perio related lesions and their clinical presentation.

3.1 Endodontic Diagnosis: Getting to the "Root" of the Problem

3.1.1 Medical and Dental History

Medical History: The age of the patient and current medical conditions can influence both the diagnosis and course of treatment. Younger patients often present with good health and are taking few oral medications that may affect their teeth. However, older patients can present with multiple health conditions that may influence their oral health, such as diabetes, cardiovascular disease, and cancer. Patients with diabetes mellitus have been associated with increased risk to periodontal disease and may also be at greater risk of developing apical periodontitis [1]. Recent evidence has also been presented suggesting patients with periodontal disease may have delayed healing after endodontic therapy [2]. The presence of cardiovascular disease, from a clinical study in Sweden, was found to increase the odds of having apical periodontitis by a factor of 3.8 [3]. Many patients being treated for cardiovascular disease also have hypercholesterolemia and are most likely taking a statin drug. As a result, they may be at risk of developing pulp canal obliteration over time [4], which may make the tooth more susceptible to developing apical periodontitis. Even cancer, such as lymphomas, may mimic periodontal and periapical conditions [5]. So it is important to be aware of these possibilities when assessing the patients' medical history and any possible link to their current chief complaint.

Dental History: The dental history as reported by the patient is a critical element. It is a detailed review of the history of the patient's chief complaint and can be a key to the diagnosis, although it is a subjective history, and is influenced by the patient's memory and current emotional status or stress level. At times patients are very poor historians of their oral health! Questions to the patient

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should include: How long has the condition been present? Does the area feel swollen? What is the pain like? What brings on the pain? Does the pain linger? What does not affect the pain? Has the condition prevented sleep? These questions are designed to determine the nature of the problem, as endodontic symptoms (history of spontaneous pain, lingering pain to cold, pain to biting) usually develop over a period of weeks or months, but periodontal related symptoms (sore gums, bleeding gums, foul odor) may linger for months to years. Another important question to consider pertains to the possibility of a history of trauma. Were there any events with the patient that may have led to this current condition? This last question may be important to ask of the younger patients (or their guardian). Dental trauma, although not the scope of this chapter, is another possible etiology of gingival and dental conditions. The reader is referred to the publications of the International Association of Dental Traumatology for further information regarding the topic of dental trauma [6].

Another important question to ask as part of the dental history involves previous endodontic treatments. Could the current condition be related to a recently completed root canal procedure? Or has the patient had root canal therapy years ago, but currently periodontal disease has flared up, and now an issue has developed around one of these previously treated root canals. In a retrospective cohort study, Ruiz et al. has shown that the risk of developing apical periodontitis in endodontically treated teeth is 5.19 times greater for patients with periodontal disease compared to patients without the disease [7].

3.1.2 Clinical Exam

Radiographs: In order to determine an accurate diagnosis, three areas must be considered: the history of the problem, current radiographs (and historical ones if available), and a thorough clinical exam. The first part of this, the medical and dental history, was presented above. The next step is to obtain radiographs of the affected area and complete the clinical exam. Although this is a problem-focused exam, do not ignore the overall presentation of the patient's mouth. What is the level of oral hygiene? Is there generalized gingivitis, perhaps even hyperplastic tissue? This may point to a periodontal verses endodontic assessment; consider the side effect of calcium channel blocking agents causing gingival hyperplasia [8]. Radiographs should include two periapical and one bitewing projection, as it has been shown that radiographs exposed from multiple angulations are more diagnostic [9]. It also may be prudent to consider a 3D CBCT scan. Depending on the results of the periapical radiographs, CBCT scans may be indicated, as they are more accurate in revealing apical pathologies and root morphological anomalies as compared to 2D periapical images [10, 11]. In reviewing the radiographs, special attention is given to cortical bone height and bone loss associated with the roots of the tooth in the area of interest pointed out by the patient, as well as the condition of the root canals. The clinician must be aware of possible indications of horizontal or vertical bone defects that may suggest periodontal disease and will need to be probed in the mouth. Other questions the clinician must consider regarding the radiographs are whether canals are visible in the roots, do the canals appear calcified, are there areas of resorption, and has the tooth had endodontic therapy, as well as, what is the condition and type of any present restorations. Lastly, what is the condition of the PDL space and is it traceable on the radiograph next to the lamina dura? These are all questions to be considered when viewing the radiographs.

Extraoral Exam: The purpose of the extraoral exam is twofold. First, it should be done as an oral cancer screening, checking lymph nodes, thyroid gland, and muscles of mastication for signs of abnormalities and asymmetry; second, as a means to see any evidence of odontogenic swelling of the face. Depending on the information derived from the dental history, the clinician might suspect temporomandibular disease (TMD) as part of the differential diagnosis, especially if no direct soft tissue or endodontic lesion is found to explain the chief complaint. TMD has been shown to be one of the most common causes of non-odontogenic pain that is mistaken for toothache [12].

Intraoral Exam: It is during this portion of the examination process that most causes of the chief complaint will be revealed. Periodontal probing, palpation, percussion, and sensibility testing (Cold test and Electric Pulp Test (EPT)) of the suspected area will all need to be carefully considered. Most likely the patient will direct you to the area of concern, but before exploring that area, the clinician must do an intraoral sweep of the mouth as part of the oral cancer screening process, and to gauge the overall periodontal health (and oral hygiene) of the patient. Then, a periodontal probing survey of the mouth can be done, ending in the suspected problem area.

Periodontal Probing: With the completion of the periodontal probing in multiple areas of the mouth, the clinician should be aware of the general periodontal health of the patient. With this knowledge, careful probing of the affected tooth is completed, paying particular attention to the pattern of probing depths around the tooth. A gingival abscess of periodontal origin would commonly have wide areas of pocketing compared to those from an endodontic origin, which tend to be narrower. Harrington published a classic illustration of this in 1979 [13] and a similar illustration based on it is shown in Fig. 3.1.

Palpation: Documentation of the sensitivity of the alveolar gingival tissues, both buccal and lingual, is an important part of the examination process. Areas of palpation sensitivity and or swelling should be noted and recorded.

Percussion: This test often identifies the offending tooth, especially if there is an endodontic component responsible. However, complications to this test exist. It is important to discern whether the percussion sensitivity is coming from an inflamed periodontal ligament (PDL), or is it from dentinal sensitivity due to caries or a cuspal fracture. Percussion sensitivity that is present no matter where the tooth is tapped (buccal, occlusal, or lingual) is most probably from an inflamed PDL and apical periodontitis. Isolated areas of percussion sensitivity on the same tooth suggest a dentinal issue, such as a fracture, caries, or possible occlusal trauma. Endodontic etiologies tend to be more percussion sensitive than periodontal ones [14, 15].

Sensibility Testing: Testing a tooth's response to cold or heat has often been called vitality testing, but this is actually an inaccurate use of the term. Vitality testing measures the level of vascularity of a tissue, and is more of a histological term. Sensibility testing measures the neural response of a tissue, and how the subject responds. The level of the response can be defined as the sensitivity of the test. Thus, when a cold or heat test is conducted on a tooth, the sensibility is tested, with the level of response being the sensitivity [14]. Endodontically involved teeth that have not become necrotic will usually have an exaggerated and delayed and/or lingering response. The clinician should not be surprised by this response if the patient reported lingering and spontaneous pain as part of their dental history. A negative response to the thermal tests would indicate a necrotic pulp, especially if it also tested negative (no response, i.e., 80 reading) to an electric pulp test (EPT). The combination of these negative responses to both tests has a high sensitivity and specificity in providing an accurate diagnosis of pulpal necrosis [16, 17]. Regarding the concept of sensitivity and specificity, terms that are sometimes confusing to the average clinician, consider this simple illustration as an example. Figure 3.2 shows a photo of a

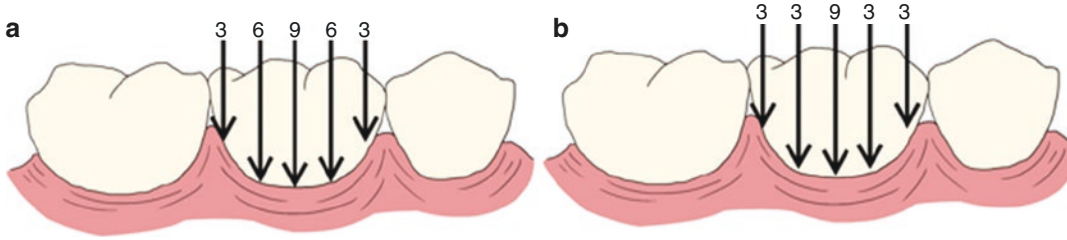

Fig. 3.1 (**a**) The probing depths of a wide periodontal pocket. (**b**) The probing depths of a narrow periodontal pocket (Illustration courtesy of Molly S Kaz Frick, 2018)

Fig. 3.2 Sign on door not intended to be used as an example of a specificity test

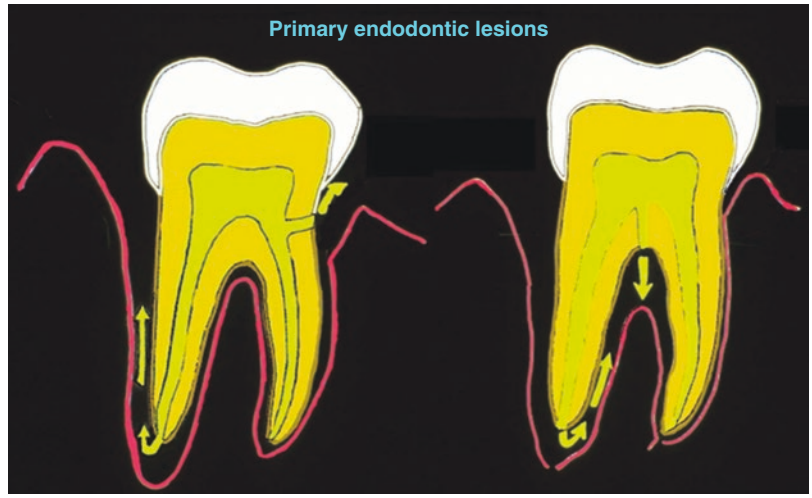

Fig. 3.3 Routes of endodontic infection through the apex or lateral canals of a tooth (Courtesy Dr. Riley, UMKC School of Dentistry)

doorway with two doors. One of the doors is marked with a sign that says, "use ***other*** door." So in this example, if the presence of disease would be identified by going through the correct door, the sign on the door identifying where you should not go, i.e., no disease, would be the specificity test. If instead, however, a sign was on the door intended to be opened said "use ***this*** door," that sign would be the sensitivity test. So sensitivity are tests that identify a condition, response, or disease, and specificity tests identify the lack of the presence of a condition, response, or disease.

Results of sensibility tests are a critical element in determining whether the diseased condition of the tooth is periodontal or endodontic origin. An etiology of endodontic origin is easily ruled out if the offending tooth responds normally to those tests. Figure 3.3 presents an illustration of the typical routes of infection of endodontic lesion, such as from apical foramina or lateral and furcal canals.

3.1.3 Endodontic Only or Periodontic Only Lesions

In this next section several cases representing either only endodontic or only periodontic lesions are shown. Figure 3.4 shows an example of a purely endodontic in orgin lesion. Figures 3.5, 3.6, and 3.7 show an example of a case that tested normal to pulp testing and was diagnosed as a

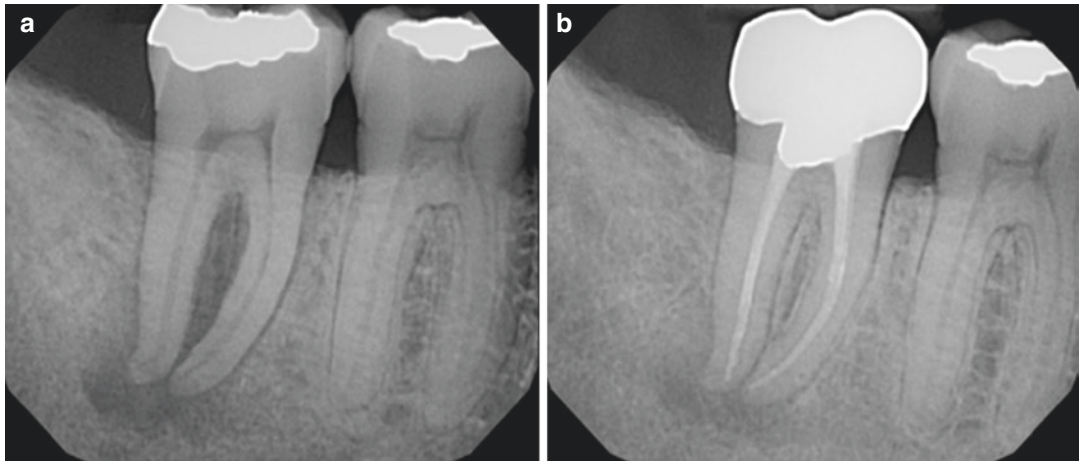

Fig. 3.4 (**a**) shows tooth #31, initially referred to a periodontist for treatment of a periodontal abscess. Deep pocketing (9 mm +) was found on the buccal furcation, but all other areas around the tooth had normal probings (3 mm or less). The patient was not in pain but had some minor buccal swelling of the gingival tissue near the furcation. Sensibility testing revealed no responses from both cold and EPT. A diagnosis of pulpal necrosis and chronic apical abscess were made and the tooth was treated endodontically. (**b**) shows osseous healing 9 months after endodontic treatment and restoration with a crown (Radiographs courtesy Dr. Stephanie Mullins)

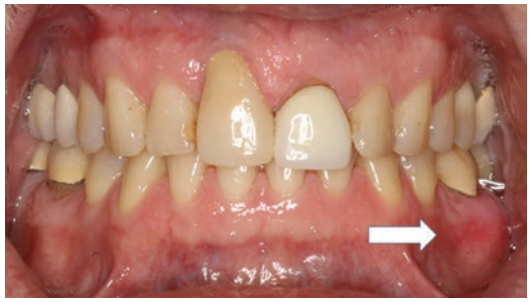

Fig. 3.5 Clinical photograph of symptomatic gingival abscess buccal to tooth #19. Note the swelling on the lower right side as indicated by the arrow. Gentle palpation of the swelling was sensitive and produced suppuration from a broad 6 mm pocket, mesial buccal and midbuccal areas of the tooth. Subgingival calculus was clinically detectable. Sensibility testing with cold was normal (responded, no lingering) on this and all control teeth (Image courtesy Dr. Rex Livingston, UMKC School of Dentistry)

periodontal abscess. Another case illustrating this is shown in Fig. 3.8, where the patient was initially referred for endodontic therapy on tooth #14 due to chewing pain and buccal swelling. Pulp sensibility tests indicated the tooth was vital, so periodontal therapy with osseous surgery and grafting was completed.

Regarding sensibility testing, it is important to note, however, that the actual histological condition of the pulp is not always accurately predictable, as discussed in Seltzer and Bender's classic paper [18]. The terminology used at the time of Seltzer's paper included terms such hyperemia, acute serous pulpitis, and acute suppurative pulpitis, and it were these diagnostic terms that were not correlated to the histological status of the tooth in their paper. The study at the time called into question the accuracy of pulpal sensibility testing for diagnostic purposes. However, the validity of clinical sensibility testing has been more recently demonstrated. In an evaluation of 150 patients receiving endodontic therapy, Weisleder et al. compared the clinical ability of cold and electric pulp testing (EPT) to predict tooth vitality or necrosis via direct observation of the status of the pulp after initiation of endodontic therapy. Ninety-seven percent of the teeth responding positively to both cold and EPT were found to be vital, and 90 percent of the teeth responding negatively to both were found to be necrotic [17]. In another study, Ricucci et al. evaluated 95 human extracted teeth and compared their clinical diagnosis to the histological presentation of the tooth. Using current American Board of Endodontics terminology of normal pulp, reversible pulpitis, and irreversible pulpitis,