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Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions

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Abstract

Objective: To critically evaluate the existing literature on acute lesions occurring in the periodontium (periodontal abscesses [PA], necrotizing periodontal diseases [NPD], and endo-periodontal lesions [EPL]) to determine the weight of evidence for the existence of specific clinical conditions that may be grouped together according to common features. The ultimate goal is to support an objective classification system.

Importance: Although PA, NPD, and EPL occur with relatively low frequency, these lesions are of clinical relevance, because they require immediate management and might severely compromise the prognosis of the tooth.

Findings: In general, the evidence available to define these three conditions was considered limited. PA and EPL are normally associated with deep periodontal pockets, bleeding on probing, suppuration, and almost invariably, with pain. EPL are also associated with endodontic pathology. NPDs have three typical features: pain, bleeding, and ulceration of the gingival interdental papilla. The available data suggested that the prognosis of PA and EPL are worse in periodontitis than in nonperiodontitis patients. Lesions associated with root damage, such as fractures and perforations, had the worst prognosis. NPD progression, extent and severity mainly depended on host-related factors predisposing to these diseases.

Conclusions: PA should be classified according to the etiological factors involved, with the most frequent being those occurring in pre-existing periodontal pockets. NPD are clearly associated with the host immune response, which should be considered in the classification system for these lesions. EPLs should be classified according to signs and symptoms that have direct impact on their prognosis and treatment, such as presence or absence of fractures and perforations, and presence or absence of periodontitis.

KEYWORDS

endo-periodontal lesions, necrotizing gingivitis, necrotizing periodontal diseases, necrotizing periodontitis, periodontal abscess

According to the American Academy of Periodontology,[1] acute periodontal diseases are rapid-onset clinical conditions that involve the periodontium or associated structures and may be characterized by pain or discomfort, tissue destruction, and infection. Among these conditions, the following diseases have been listed: gingival abscess, periodontal abscess, necrotizing periodontal diseases, herpetic gingivostomatitis, pericoronal abscess, or pericoronitis, and

<table>
<tr><td colspan="9">Acute periodontal conditions</td></tr>
<tr><td rowspan="2">Virus infection</td><td colspan="6">Abscesses in the periodontium</td><td colspan="2">Necrotizing periodontal diseases[1]</td></tr>
<tr><td colspan="2">Other abscesses</td><td colspan="4">Odontogenic or dental abscesses[7]</td><td rowspan="2">Necrotizing gingivitis</td><td rowspan="2">Necrotizing periodontitis</td></tr>
<tr><td>Herpetic gingivostomatitis[6,1]</td><td>Surgery[6]</td><td>Trauma[6]</td><td>Pericoronitis[6,5,1]</td><td>Pulp necrosis</td><td>Endo-perio lesion[6,1]</td><td colspan="2">Periodontal infections[6,5,1]</td></tr>
<tr><td rowspan="2">Out of the scope</td><td rowspan="2">Out of the scope</td><td rowspan="2">Out of the scope</td><td rowspan="2">Out of the scope</td><td>Dentoalveolar abscess</td><td>Endo-perio abscess</td><td>Gingival abscess</td><td>Periodontal abscess</td><td rowspan="2">Part 2.</td></tr>
<tr><td>Out of the scope</td><td>Part 3.</td><td colspan="2">Part 1.</td></tr>
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FIGURE 1 List of "acute periodontal conditions," according to different authors, and scope of the present review

combined periodontal-endodontic lesions. Herpetic gingivostomatitis is not included in the present review, whereas the so called gingival and periodontal abscesses were considered within a category named: abscesses in the periodontium (Figure 1).

Acute lesions in the periodontium are among the few clinical situations in periodontics in which patients may seek urgent care, mostly because of the associated pain. In addition, and in contrast to most other periodontal conditions, rapid destruction of periodontal tissues may occur during the course of these lesions, thus emphasizing the importance of prompt diagnosis and treatment. The present review and update focuses on two acute conditions (abscesses in the periodontium and necrotizing periodontal diseases); and on endo-periodontal lesions that can occur in acute or chronic forms.

Periodontal abscesses (PA) are important because they represent common dental emergencies requiring immediate management and can result in rapid destruction of the periodontium with a negative impact on the prognosis of the affected tooth. In certain circumstances, PA may have severe systemic consequences.[2,3] Although the prevalence of **necrotizing periodontal diseases (NPD)** is low, their importance is clear, because they represent the most severe conditions associated with dental biofilm, leading to very rapid tissue destruction.[3] Whereas, **endo-periodontal lesions (EPL),** in spite of being relatively rare in clinical practice, might severely compromise the prognosis of the tooth, and are considered one of the most challenging problem faced by clinicians, because they require multidisciplinary evaluation, diagnosis, and treatment.[4]

The aim of the present review was to critically evaluate the existing literature on acute lesions in the periodontium (PA and NPD) and EPL, with the purpose of determining the weight of evidence of the existence of specific clinical conditions that may be grouped together according to common features. The ultimate goal was to support an objective classification system that may help the clinician to determine the prognosis of the teeth involved, and treatment of these conditions. To achieve this objective, the three conditions were separately assessed.

METHODS

Independent electronic searches were conducted to identify relevant articles dealing with each of the three conditions addressed in this review. In total, 128 studies were included for PA, 138 for NPD and 74 for EPL. Details about the electronic search methods and studies included, flow charts showing the selection of articles for each condition evaluated in this review, and designs of the studies included are described in Appendices 1 and 2, respectively, in the online *Journal of Periodontology*.

1 | PERIODONTAL ABSCESSES

1.1 | Clinical presentation

Different etiological factors may explain the occurrence of abscesses in the periodontal tissues, such as pulp necrosis (endodontic, periapical or dentoalveolar abscesses), periodontal infections (gingival or periodontal abscess[5]), pericoronitis (pericoronal abscess), trauma, surgery,[6] or foreign body impaction. Together, they are referred to as odontogenic or dental abscesses,[7] and when they are associated with EPL, they could also be considered odontogenic abscesses. PA can specifically be defined as a localized accumulation of pus located within the gingival wall of the periodontal pocket, with an expressed periodontal breakdown occurring during a limited period of time, and with easily detectable clinical symptoms.[2]

Three different reasons could support the importance of PA:

a. *Common dental emergencies, requiring immediate management* (see Appendix 3, Table A3.1, in online journal)
PA represented approximately 7.7–14.0% of all dental emergencies, being ranked the third most prevalent infection demanding emergency treatment, after dentoalveolar abscesses and pericoronitis. In an army dental clinic, 27.5% of periodontitis patients presented with PA, with clear differences between patients undergoing active periodontal treatment (13.5%) and untreated

patients (59.7%).[8] Among patients undergoing periodontal maintenance (PeM), PAs were detected in 37% of the patients followed-up for 5–29 years.[9] In the Nebraska prospective longitudinal study, 27 PA were observed during 7 years, and 23 of them occurred in sites that received coronal scaling.[10]

b. *Rapid destruction of periodontal tissues, with a negative effect on the prognosis of the affected tooth* (see Appendix 3, Table A3.1, in online journal)
PAs may lead to tooth loss, especially if they affect teeth with previous moderate to severe attachment loss, as occur during PeM in patients with severe chronic periodontitis. Indeed, they have been considered the main cause of tooth extraction during PeM.[9,11–13] Similarly, teeth with repeated abscess formation were considered to have a "hopeless prognosis",[14] and 45% of teeth with a periodontal abscess found during PeM were extracted.[9] The main reason for tooth extraction of teeth with a questionable prognosis, which had been followed-up for 8.8 years, was the presence of periodontal abscess.[11]

c. *Severe systemic consequences*
PA may be associated with systemic dissemination of a localized infection. Numerous case reports and series have described the occurrence of systemic infections resulting from a suspected source in a periodontal abscess, either through dissemination occurring during therapy or related to an untreated abscess (see Appendix 3, Table A3.2, in online journal).

1.2 | Etiology: pathophysiology, microbiology and histological features

1.2.1 | Pathophysiology

The first step in the development of a PA is bacterial invasion of the soft tissues surrounding the periodontal pocket, which will develop into an inflammatory process through the chemotactic factors released by bacteria that attract polymorphonuclear leukocytes (PMN) and other cells. This will trigger intensive release of cytokines; lead to destruction of the connective tissues; encapsulation of the bacterial infection and the production of pus. Once the abscess is formed, the rate of destruction within the abscess will depend on the growth of bacteria inside the foci; their virulence, and the local pH (an acidic environment will favor the activity of lysosomal enzymes).[15]

1.2.2 | Microbiology

In general, microbiological reports on PA have shown a microbial composition similar to that observed in periodontitis (see Appendix 3, Table A3.3, in online journal). The most prevalent bacterial species identified in PA, by means of different techniques (see Appendix 3, Table A3.4, in online journal) were *Porphyromonas gingivalis* (50-100%), *Prevotella intermedia, Prevotella melaninogenica, Fusobacterium nucleatum, Tannerella forsythia, Treponema* species, *Campylobacter* species, *Capnocytophaga* species, *Aggregatibacter actinomycetemcomitans* or gram-negative enteric rods (see Appendix 3, Table A3.5, in online journal). Up to now, there has been limited evidence available on the role of viruses, the genetic characteristics of different strains (e.g. *P. gingivalis*), or the antimicrobial susceptibility of strains isolated from these lesions (see Appendix 3, Table A3.6, in online journal).

1.2.3 | Histopathology

The histopathology of periodontal abscess lesions was reported as follows,[15] after observing the lesion from the outside to the inside: a normal oral epithelium and lamina propria; an acute inflammatory infiltrate; intense focus of inflammation, with presence of neutrophils and lymphocytes in an area of destroyed and necrotic connective tissue; and a destroyed and ulcerated pocket epithelium.

1.3 | Etiology: risk factors

PA may develop in a pre-existing periodontal pocket (e.g., in patients with periodontitis) or in the absence of a pre-existing periodontal pocket.

1.3.1 | Periodontal abscess in periodontitis patients

In periodontitis patients, a PA could represent a period of disease exacerbation, favored by the existence of tortuous pockets, presence of furcation involvement[16] or a vertical defect,[16,17] in which the marginal closure of the pocket could lead to an extension of the infection into the surrounding periodontal tissues.[15,18,19] In addition, changes in the composition of the subgingival microbiota, with an increase in bacterial virulence, or a decrease in the host defense, could also result in an inefficient capacity to drain the increased suppuration. Different subgroups could be distinguished (see Appendix 3, Table A3.7, in online journal):

- Acute exacerbation:
  - In untreated periodontitis.[20]
  - In "refractory" periodontitis.[21]
  - In PeM, as previously described.
- After different treatments:
  - Scaling and root planing or professional prophylaxis: dislodged calculus fragments could be pushed into the tissues,[20] or inadequate scaling could allow calculus to remain in deep pocket areas, whereas the coronal part would occlude the normal drainage.[10]

- Surgical periodontal therapy: associated with the presence of foreign bodies such as membranes for regeneration or sutures.[22]
- Systemic antimicrobial intake, without subgingival debridement, in patients with severe periodontitis could also cause abscess formation,[23–25] probably related to an overgrowth of opportunistic bacteria.[23]
- Use of other drugs: e.g., nifedipine.[26]

1.3.2 | Periodontal abscess in non-periodontitis patients

PA can also occur in previously healthy sites, because of (see Appendix 3, Tables A3.8 and A3.9, in on line journal):

- Impaction of foreign bodies: dental floss, orthodontic elastic, toothpick, rubber dam, or popcorn hulls.
- Harmful habits (biting wire, nail biting, clenching) could favor abscess formation because of subgingival impaction of foreign bodies or to coronal closure of the pocket.
- Orthodontic factors, such as inadequate orthodontic forces or a cross-bite, have been reported to favor PA development.
- Gingival enlargement.[27]
- Alterations of the root surface, including:
  - Severe anatomic alterations, such as invaginated tooth, *dens evaginatus* (grooves) or odontodysplasia.
  - Minor anatomic alterations, such as cemental tears, enamel pearls or developmental grooves.
  - Iatrogenic conditions, such as perforations.
  - Severe root damage: vertical root fracture or cracked tooth syndrome extending through the root.
  - External root resorption.

1.4 | Assessment and diagnosis

Data from studies with a relevant number of cases and a comprehensive description were analyzed (Tables 1A and 1B).[13,28–31]

A series of symptoms have been reported by patients suffering from a PA, such as pain, tenderness of the gingiva, swelling, or tooth "elevation." The most prominent sign during the oral examination was the presence of an ovoid elevation in the gingiva along the lateral part of the root. Suppuration on probing or sampling was a common finding (66–93%), whereas a fistula was not. A PA was usually associated with a deep periodontal pocket (7.3–9.3 mm), bleeding on probing (100%), and increased tooth mobility (56.4–100%). Bone loss was normally observed in the radiographic examination. Extraoral findings were uncommon, but could include facial swelling (3.6%), elevated body temperature, malaise, regional lymphadenopathy (7–40%) or increased blood leukocytes (31.6%). Most abscesses affected

TABLE 1A Diagnosis of periodontal abscesses: studies with series of more than 10 abscesses with comprehensive clinical description

Study			Patient sample			Periodontal status		Abscesses		Etiology			Location	
Reference	Country	Study design	*n*	Age	% female	MS	Initial	Healthy	*n*	Name	Untreated	PeM	Perio treatment	Trauma	Teeth affected	Sites affected
Smith[13]	UK	Prospective-3y	55	10-68	50.9%				62	acute lateral PA		60.0%	36.4%		LM (27.6%), UM (25.8%)	interdental (62.9%)
Hafstrom[29]	Sweden	Prospective-6 m	20	24-79	55.0%				20	PA (clear periodontal origin)						
Herrera[30]	Spain	RCT-30d	29	26-65	58.6%	93.1%	6.9%	0.0%	29	PA	62.0%		14.0%		69% M (equal U-L)	buccal (48%), interdental (38%), lingual (14%)
Jaramillo[31]	Colombia	Cross-sectional	54	48.3	53.7%	87% ChP, 9.3% AgP		3.7%	60	PA	81.6%	11.6%	6.6%	5.0%	Lant (41.6%), Uant (20%)	
Chan[28]	India	Cross-sectional	14	39.6	50.0%				14	PA	7.1%	50.0%	42.9%		86% U; equal M.PM.ant	buccal (71%), lingual (29%)

RCT, randomized clinical trial; y, year; m, month; d, day; p, patient; MS, moderate-severe
L, lower jaw; U, upper jaw; M, molar; ant, anteriors; PM, premolars; PeM, periodontal maintenance; ChP, chronic periodontitis; AgP, aggressive periodontitis

periodontitis patients (96.3–100%), either untreated (7.14–81.6%), in PeM (11.6–60%) or those undergoing active therapy (6.6–42.9%). Some studies found molars more frequently affected,[13,30] whereas others found equal distribution,[28] or predominance in anterior teeth.[31] One study reported a higher number of abscesses at the interproximal level,[13] whereas others observed more frequent abscess formation at buccal sites.[28,30]

Patient history may also provide relevant information, especially in cases of abscesses associated with previous treatments (scaling and root planing, periodontal surgery, intake of systemic antimicrobials agents, or other drugs [e.g., nifedipine] and endodontic treatment), or in abscesses related to foreign body impaction.

Differential diagnosis (see Appendix 3, Table A3.10, in online journal) is critical, because PA may be like other oral conditions:

- Other odontogenic abscesses (dento-alveolar abscesses, pericoronitis, endo-periodontal abscess), or other acute conditions (lateral periapical cyst and postoperative infection).[32]
- Tumor lesions, including metastatic tumoral lesions, odontogenic myxoma, non-Hodgkin´s lymphoma, squamous cell carcinoma, metastatic carcinoma.
- Other oral lesions: pyogenic granuloma, osteomyelitis, odontogenic keratocyst, eosinophilic granuloma.
- Self-inflicted gingival injuries.
- Sickle cell anemia.
- Abscesses after surgical procedures.

1.5 | Proposed changes to the current 1999 classification

The 1999 classification for abscesses in the periodontium included gingival, periodontal, pericoronal, and periapical abscesses.[5] Relevant problems associated with this classification system included: (1) the differentiation between gingival and PA, which could be confusing, because this differentiation was simultaneously based on location and etiology; (2) considering a PA as chronic or acute may not be adequate, because an abscess, by definition, is an acute lesion; and (3) the inclusion of pericoronitis and periapical abscesses in the classification together with PA might not be appropriate. Pericoronal abscesses were included in the 1999 classification, but no solid scientific basis for this was found in the article associated with the topic.[5] In addition, the terms "pericoronal abscess" or "pericoronitis abscess" were seldom used in the scientific literature; in the present literature search, none of the articles retrieved described a pericoronal abscess as a PA. PAs should be classified based on their etiology (see section 3.3 and Table 2).

TABLE 1B Diagnosis of periodontal abscesses: signs and symptoms

	Symptoms			Signs				X-ray	Extraoral		Variety
Reference	Pain	Redness	Swelling	Mean PPD	%PPD > 6 mm	BOP	SUP	Increased mobility	Bone loss	Fever	Lymph-adenophaty	Other findings
Smith[13]	usual		usual		54.8%			56.4% > 0	most, also furcation in most molars	0.0%	40.0%	abscess pointing (69.6%), no fistula, facial swelling (3.6%)
Hafstrom[29]	100%	100%	100%	8.1		100%	68%			none	none	tenderness (100%)
Herrera[30]	62%MS; 10%none	75%MS	93%MS	7.3 (3-13)	62.1%	100%	66%	79%			10.0%	elevated leukocytes (31.6%)
Jaramillo[31]	68.3%	93.3%	95%	9.3±2.5		100%	93.3%	100%	93.3%MS			tooth elevation (23.3%)
Chan[28]				7.4 ± 1.6				71%		36.9±0.5, most afebrile	7.1%	

PPD, probing pocket depth; freq., frequency; BOP, bleeding on probing; SUP, suppuration on probing or sampling; MS, moderate-severe

TABLE 2 Proposal of classification for periodontal abscess, based on the etiological factors involved

Periodontal abscess in periodontitis patients (in a pre-existing periodontal pocket)	Acute exacerbation	Untreated periodontitis	
		Non-responsive to therapy periodontitis	
		Supportive periodontal therapy	
	After treatment	Post-scaling	
		Post-surgery	
		Post-medication	Systemic antimicrobials
			Other drugs: nifedipine
Periodontal abscess in non-periodontitis patients (not mandatory to have a pre-existing periodontal pocket)	Impaction		Dental floss, orthodontic elastic, toothpick, rubber dam, or popcorn hulls
	Harmful habits		Wire or nail biting and clenching
	Orthodontic factors		Orthodontic forces or a cross-bite
	Gingival overgrowth		
	Alteration of root surface	Severe anatomic alterations	Invaginated tooth, dens evaginatus or odontodysplasia
		Minor anatomic alterations	Cemental tears, enamel pearls or developmental grooves
		Iatrogenic conditions	Perforations
		Severe root damage	Fissure or fracture, cracked tooth syndrome
		External root resorption	

2 | NECROTIZING PERIODONTAL DISEASES

2.1 | Clinical presentation

In the 1999 classification, necrotizing ulcerative gingivitis (NUG) and necrotizing ulcerative periodontitis (NUP) were included among NDPs.[33] Studies have suggested that they may represent different stages of the same disease, because they have similar etiology, clinical characteristics, and treatment, and may even progress to more severe forms such as necrotizing stomatitis (NS) and noma.[34,35] The terminology "ulcerative" was later eliminated, because ulceration was considered to be secondary to the necrosis.[36]

NPD patients are frequently susceptible to future recurrence of disease[37,38] and NPD could also become a "chronic condition," with a slower rate of destruction.[39] In cases of severe systemic involvement, progression of NPD into other oral lesions could occur.[40,41]

Prevalence/incidence of NG has been reported for the overall population or for specific groups of individuals (for references, see Appendix 4, Tables A4.1a-e, in online journal). In general populations attending dental clinics, the prevalence of NG ranged from 0.51 to 3.3%; in military personnel, the prevalence and incidence reported was higher close to the end of the 2nd World War (3.96–20.6%) than it was in more recent studies (0.19–6.19%). In African populations, highly variable results have been reported. In students, prevalence ranged from 0.9 to 6.7%. And in HIV/AIDS patients data showed wide variations: children (2.2-5.0%), HIV adults (0.0–27.7% for NG and 0.3–9.0% for NP), and HIV/AIDS patients (10.1–11.1% for NG and 0.3–9.0% for NP).

2.2 | Etiology and risk factors

NPD are infectious conditions; however, predisposing factors, including a compromised host immune response, are critical in the pathogenesis.

a. *Microbiology* (see Appendix 4, Tables A4.2, in online journal)

The bacterial etiology of NPD, with the presence of spirochetes and fusiform bacteria, was previously demonstrated by Plaut in 1894, and Vincent in 1896 (as reviewed in[35]). Furthermore, clinical improvements observed after mechanical debridement and antimicrobial treatment further supported the bacterial etiology of these conditions.[42] Earlier studies, using electron microscopy, suggested tissue invasion by spirochetes.[43,44] Culture studies identified *P. intermedia,* and *Treponema*, *Selenomonas* and *Fusobacterium* species, which were considered "constant flora" in NPD lesions.[45] The role of spirochetes was confirmed by immuno assays[46,47] and PCR targeting 16s rRNA.[48] Recent studies by phylogenetic analysis also suggested a role of the *P. intermedia* and *Peptostreptococcus* genus in the etiology of NPD.

The microbiota associated with NPD in HIV (see Appendix 4, Tables A4.3, in online journal) was like

that of periodontitis in non-HIV patients, with some specific features, such presence and invasion of *Candida albicans,* herpes viruses or superinfecting bacterial species.

b. *Host immune response*
Although the importance of host immune response in the etiopathogenesis of NPD was indisputable, the studies available reported very heterogeneous results, as explained in Appendix 4, Tables A4.4 in online journal.

c. *Predisposing factors*
The most relevant predisposing factors for NPD were shown to be those altering the host immune response and usually more than one factor was necessary to cause onset of the disease.[49]

2.2.1 | Human immunodeficiency virus infection and acquired immune deficiency syndrome (HIV/AIDS)

NPD in HIV patients may be more frequent and show faster progression, with a higher risk of evolving into more severe lesions (NP and NS), and a higher tendency for disease recurrence and poor response to therapy (see Appendix 4, Tables A4.3, in online journal).

2.2.2 | Other systemic conditions

Different reports have found NPD lesions associated with, or because of different systemic conditions (see Appendix 4, Tables A4.5, in online journal), or mimicking NPD, in which the lesions were part of the systemic pathology (see Appendix 4, Table A4.6, in online journal).

2.2.3 | Malnutrition

Malnutrition (see Appendix 4, Tables A4.5, in online journal) could also be an important predisposing factor for NPD,[50] especially in developing countries.[51–53] A marked reduction in key antioxidant nutrients and an altered acute phase response against infection ("protein energy malnutrition")[54,55] have been reported. Other consequences were an inverse proportion in the ratio of helper and suppressor T-lymphocytes, histaminemia, increased free cortisol in blood and saliva, and defects in mucosal integrity.[54,56]

2.2.4 | Psychological stress and insufficient sleep

Certain situations of acute psychological stress or stressing situations, and some personality traits or the ability to cope with a stressful situation (see Appendix 4, Tables A4.5, in online journal) may predispose individuals to NPD. During stress periods, the immune response is altered and the subject's behavior is changed. The biological plausibility of this assumption is based on the reduction of gingival microcirculation and salivary flow; increase in serum and urine levels of 17-hydroxycorticosteroid (17-OHCS)[57]; change in the function of PMN and lymphocytes, and increase in periodontal pathogen levels (*P. intermedia*).[45]

2.2.5 | Inadequate oral hygiene, pre-existing gingivitis, and previous history of NPD

Plaque accumulation has been considered a predisposing factor for NPD, which may also be aggravated by limited tooth brushing because of pain.[37,58,59] NPD usually occurred secondarily to a previously existing periodontal disease (chronic gingivitis,[39,60] previous NPD[58]) (see Appendix 4, Tables A4.5, in online journal).

2.2.6 | Tobacco and alcohol consumption

Most adult patients with NPD are smokers.[39,61–65] Alcohol consumption has also been associated with the physiological and psychological factors favoring NPD[58,66] (see Appendix 4, Tables A4.5, in online journal).

2.2.7 | Young age and ethnicity

Young people (15-34 years old) in the developed world are at a higher risk of suffering from NPD, frequently in combination with other predisposing factors.[58,64,67,68] Children are at a higher risk in developing countries, and this is normally associated with malnutrition and other infections.[52,53,56,69] Some studies suggested that Caucasians suffered from NPD more frequently[58,64,70] than other ethnic groups, however, this finding needs to be confirmed (see Appendix 4, Tables A4.5, in online journal).

2.2.8 | Seasonal variations

Different studies (see Appendix 4, Table A4.5e in online journal) have evaluated the hypothesis of the effect of seasonal variations on the prevalence of NPD: in central Africa, NPD peaked in the rainy season; less clear patterns were observed in military personnel, students or general populations, although winter months were normally peak periods, except in South Africa.

2.2.9 | Other factors

Local factors (see Appendix 4, Table A4.5d, in online journal), including decorative crowns[71] or orthodontic therapy[72] may favor the onset of NG. Body geometry,[73] thermoregulatory abnormalities,[74] allelic variants for complement factors, and properdin factor B [75] or erythrocyte catalase activity,[76] have also been studied with inconclusive results.

2.3 | Pathophysiology and histological features

NG lesions observed with light microscopy[44] showed the presence of an ulcer within the stratified squamous epithelium and the superficial layer of the gingival connective tissue, surrounded by a nonspecific acute inflammatory reaction. Four regions have been described: the (1) superficial bacterial area; (2) neutrophil-rich zone; (3) necrotic zone; (4) spirochetal infiltration zone. Additional findings included plasma cells in the deeper parts and IgG and C3 between epithelial cells.[77] These observations have been confirmed by electron microscopy, adding areas of transition to a chronic stage of inflammation.[43]

2.4 | Assessment and diagnosis

Diagnosis of NPD should be primarily based on clinical findings.[35,78] Microbiological or biopsy assessment may be recommended in cases of atypical presentations or nonresponding cases.

The most relevant clinical findings in NG (Table 3) reported in relevant studies (with 35 or more patients[58,64,67,70]) were: necrosis and ulcer in the interdental papilla (94–100%), gingival bleeding (95–100%), pain (86–100%), pseudomembrane formation (73–88%), and halitosis (84–97%). Extraoral signs included adenopathy (44–61%) or fever (20-39%). In children,[52] pain and halitosis were less frequent, whereas fever, adenopathy, and sialorrhea were more frequent.

For NP,[79] in addition to the previous signs and symptoms, periodontal attachment and bone destruction were observed, together with more frequent extraoral signs. In severely immune-compromised patients, bone sequestrum could occur.[80] NP could be the result of one or various episodes of NG (less frequent pocket formation), or of NG occurring at a site previously affected by periodontitis (periodontal pocketing would be found).[34,81]

In NS, bone denudation extended through the alveolar mucosa, with larger areas of osteitis and bone sequestrum, in severely compromised systemic patients (HIV/AIDS patients, severe malnutrition). Atypical cases have also been reported, in which NS developed without the appearance of previous NPD lesions.[82–85]

Clinical criteria for identifying NG, NP, NS and Noma, according to the studies included in the present review, are summarized in Appendix 4, Tables A4.7,8,9, in online journal.

2.4.1 | Differential diagnosis

It is mandatory to establish a differential diagnosis with vesicular-bullous diseases, primary or recurrent herpetic gingivostomatitis,[86,87] oral manifestation mimicking NPD lesions (see Appendix 4, Table A4.6, in online journal) and toothbrush abrasion.[88]

2.5 | Proposed changes to the current 1999 classification

In the present 1999 classification, the consensus report established "that necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis should be collectively referred to as Necrotizing Periodontal Diseases." The group agreed that both diseases were associated with a diminished systemic resistance to bacterial infection. This rather simplistic approach did not consider the huge differences in prevalence, risk of progression, and extent and severity of NPD among patients with different predisposing conditions. NPD in HIV/AIDS patients or in malnourished children in developing countries may represent a severe and even life-threating condition (in the latter case). Conversely, NPD in smokers and stress adult patients in developed countries represented a relevant but normally non-threatening condition. Therefore, patients continuously exposed to a severe systemic compromise (see previous examples) have a higher risk of suffering from NPD and of faster and more severe progression (from NG to NP, and even to NS and Noma). Conversely, in patients with a systemic compromise of limited duration (e.g. stressful situation in students or militaries), NG may not progress, although the lesions would be different if they affected a gingivitis or a periodontitis patient. A proposal for a new classification system is presented in Table 4.

3 | ENDO-PERIODONTAL LESIONS

3.1 | Clinical presentation

EPL are clinical conditions involving both the pulp and periodontal tissues and may occur in acute or chronic forms. When they are associated with a recent traumatic or iatrogenic event (e.g. root fracture or perforation), the most common manifestation is an abscess accompanied by pain. However, EPL, in subjects with periodontitis, normally present slow and chronic progression without evident symptoms.

The most common signs and symptoms associated with a tooth affected by an EPL are deep periodontal pockets reaching or close to the apex and negative or altered response to pulp vitality tests. The other signs and symptoms reported, in order of prevalence, are: bone resorption in the apical or furcation region, spontaneous pain or pain on palpation and percussion, purulent exudate, tooth mobility, sinus tract, crown, and gingival color alterations (Table 5).

3.2 | Etiology and risk factors

3.2.1 | Primary etiology

An established EPL is always associated with varying degrees of microbial contamination of the dental pulp and the

TABLE 3 Diagnosis of necrotizing periodontal diseases: frequent clinical findings

					Primary symptoms and signs			Other symptoms and signs				
Reference	**Country**	**Study design**	**Population**	**Patients, condition**	**Gingival necrosis**	**Gingival bleeding**	**Pain**	**Pseudo-membranous formation**	**Halitosis**	**Adenopathies**	**Fever**	**Other features**
Barnes[70]	USA	Case-control	Military	218 ANUG	94.0%	95.5%	86.2%	73.4%	84.4%	na	No frequent	Cratering (79.8%); wooden or wedge-like (40.4%); bad taste (39.4%)
Stevens[64]	USA	Epidemiological (1y) and case and control	General population	51 ANUG	100%	100%	100%	na	na	na	20%	na
Falkler[67]	USA	Case and control	Clinic at urban dental school	35 ANUG	100%	100%	100%	85%	97%	61%	39%	na
Horning[58]	USA	5y epidemiological study	Military (10 HIV)	68 NG	100%	100%	100%	88%	87%	44%	24%	Interdental gingival craters (previous NG) (21%)
Jimenez[52]	Colombia	Prospective case series	Children	28 NUG, 9 Noma	100%	96.43%	53.57%	Acute cases	50%	Submaxilar (57.1%), Submaxilar & cervical (21.4%)	67.9%	Tooth mobility (46.43%); sialorrhea (42.86%)
Cobb[79]	USA	Case series	HIV	16 NUP	100%	100%	100%	81.3%	100%	68.8%	43.8%	Advanced generalized alveolar bone loss (100%)

y, years; na, not available; HIV, human immunodeficiency virus; ANUG, acute necrotizing ulcerative gingivitis; NUG, necrotizing ulcerative gingivitis; NG, necrotizing gingivitis; NUP, necrotizing ulcerative periodontitis

TABLE 4 Proposal of classification for necrotizing periodontal diseases (NPD)

Category	Patients	Predisposing conditions	Clinical condition
Necrotizing periodontal diseases in chronically, severely compromised patients	In adults	HIV+/AIDS with CD4 counts < 200 and detectable viral load	NG, NP, NS, Noma. Possible progression
		Other severe systemic conditions (immunosuppression)	
	In children	Severe malnourishments[a]	
		Extreme living conditions[b]	
		Severe (viral) infections[c]	
Necrotizing periodontal diseases in temporarily and/or moderately compromised patients	In gingivitis patients	Uncontrolled factors: stress, nutrition, smoking, habits	Generalized NG. Possible progression to NP
		Previous NPD: residual craters	
		Local factors: root proximity, tooth malposition	Localized NG. Possible progression to NP
		Common predisposing factors for NPD (see paper)	NG. Infrequent progression
	In periodontitis patients		NP. Infrequent progression

NG, necrotizing gingivitis; NP, necrotizing periodontitis; NS, necrotizing stomatitis

[a]Mean plasma and serum concentrations of retinol, total ascorbic acid, zinc, and albumin markedly reduced, or very marked depletion of plasma retinol, zinc, and ascorbate; and saliva levels of albumin and cortisol, as well as plasma cortisol concentrations, significantly increased

[b]Living in substandard accommodations, exposure to debilitating childhood diseases, living near livestock, poor oral hygiene, limited access to potable water and poor sanitary disposal of human and animal fecal waste

[c]Measles, herpes viruses (cytomegalovirus, Epstein-Barr virus-1, herpes simplex virus) chicken pox, malaria, febrile illness

supporting periodontal tissues. Nonetheless, the primary etiology of these lesions might be associated with (1) endodontic and/or periodontal infections or (2) trauma and/or iatrogenic factors.

Endo-periodontal lesions associated with endodontic and periodontal infections

They might be triggered: (1) by a carious lesion that affects the pulp and, secondarily, affects the periodontium; (2) by periodontal destruction that secondarily affects the root canal; (3) or by both events concomitantly. The latter type occurs less frequently and is usually referred to as a "true-combined" or "combined" lesion.[89,90] These lesions may develop in subjects with periodontal health[91–93] or disease[94,95] (Table 5). The periodontal condition has an important impact in the prognosis of the EPL because of the striking changes in the oral ecology of subjects with periodontal diseases. Converting this ecology back into a healthy state is challenging,[96,97] especially in patients with severe periodontitis and in teeth with deep pockets, as in the case of EPL. Therefore, a detailed periodontal examination is a very important step for the accurate diagnosis and treatment plan of EPL.

Endo-periodontal lesions associated with trauma and iatrogenic factors

These conditions usually have a poor prognosis as they affect the tooth structure. The most common lesions in this category were: (1) root/pulp chamber/furcation perforation (e.g. because of root canal instrumentation or to tooth preparation for post-retained restorations)[98]; (2) root fracture or cracking (e.g., because of trauma or tooth preparation for post-retained restorations)[98]; (iii) external root resorption (e.g., because of trauma)[99]; or (iv) pulp necrosis (e.g., because of trauma) draining through the periodontium[100] (see Appendix 5, Table A5.1, in online journal).

3.2.2 | Microbiology

Only a few studies to date have evaluated the microbiota of EPL using culture,[101–103] "targeted" molecular techniques (polymerase chain reaction [PCR][90,94,103,104] real time PCR[105] and checkerboard DNA-DNA hybridization[90]), or "open-ended" molecular techniques (Next Generation Sequencing [NGS][95] and Denaturing Gradient Gel Electrophoresis [DGGE] or cloning and sequencing[94,104]) (see Appendix 5, Table A5.2, in online journal). Overall, these studies showed a great similarity between the microbiota found in the root canals and periodontal pockets. Most of the bacterial species identified were recognized periodontal pathogens from the so called "red" and "orange" complexes,[106] such as *P. gingivalis*, *T. forsythia*, or *Parvimonas micra*, and species from the genera *Fusobacterium*, *Prevotella* and *Treponema.*[90,103,105,107–109] Studies using "open-ended" molecular techniques[94,95,104] observed a higher

TABLE 5 Main characteristic of the studies included in the endo-periodontal lesion review, stratified by the periodontal condition

				Percentage (%) of studies according to each study design								
				Signs						Signs and symptoms		
Periodontal condition	Study design	References	Number of teeth included	Deep periodontal pocket (≥5 mm)	Altered pulp response	Purulent exudate	Apical bone resorption	Sinus tract	Tooth mobility	Gingival color alteration	Crow color alteration	Pain
Periodontitis patients	CR	Aksel[124]; Blanchard[125]	5	100	100	50	100	50	50	0	0	100
	CS	Didilescu[90]; Fatemi[126]; Gomes[95]; Kipioti [101]; Kobayashi[102]; Rupf[105]; Pereira[103]; Li[94]	190	100	100	0	75	0	12.5	0	0	25
	RCT	Cortellini [127]; Gupta [128]	62	100	100	0	0	0	0	0	0	0
		TOTAL	**257**	**100**	**100**	**8.3**	**83.3**	**8.3**	**16.6**	**0**	**0**	**33.3**
Non-periodontitis patients	CR	Asgary[129]; Attam[120]; Ballal[130]; Castelo-Baz[131]; Coraini[132]; Floratos[133]; Fujii[134]; Gandhi[135]; Goyal[136]; Haueisen[137]; Jivoinovici[138]; Kambale[139]; Karabucak[98]; Keceli[140]; Kerezoudis[141]; Kishan[142]; Koyess[143]; Mali[144]; Miao[91]; Nagaveni[145]; Oh[146]; Oh[147]; Pickel[148]; Sharma[92]; Singh[149]; Sooratgar[93]; White[99]	39	100	100	33.3	70.3	33.3	29.6	3.7	7.4	55.5
	CS	Xia[104]	13	100	100	0	100	0	0	0	0	0
		TOTAL	**52**	**100**	**100**	**32.1**	**71.4**	**32.1**	**28.5**	**3.5**	**7.1**	**53.5**
Unclear	CR	Karunakar[150]; Narang[151]; Solomon[152]; Tobón-Arroyave[100]; Tseng[153]; Varughese[154]	8	100	100	83.3	100	33.3	66.6	0	0	50
	CrS	Rhee [155]	168	100	100	0	100	0	0	0	0	0
	CS	Li[94]; Nicopoulou-Karayianni[156]; Pereira[103]	69	100	100	0	100	0	0	0	0	0
		TOTAL	**245**	**100**	**100**	**50**	**100**	**20**	**40**	**0**	**0**	**30**
FINAL TOTAL		**Number of studies: 50**	**554**	**100**	**100**	**30**	**80**	**24**	**28**	**5**	**4**	**44**

CR: Case report; CS: Clinical study; RCT: Randomized clinical trial; CrS: Cross-sectional

microbial diversity and identified less common taxa, such as *Filifactor alocis, Enterococcus faecalis,* and species from the genera *Desulfobulbus*, *Dialister*, *Fretibacterium*, or *Rothia*. Incidentally, most of these species and genera have recently also been associated with chronic or aggressive periodontitis.[110,111]

Taken together, the above-mentioned data suggest that there are no major differences between the microorganisms found in the endodontic and periodontal lesions, or a specific microbial profile associated with the EPL. This was somehow expected, as both sites of infection (root canal and periodontal pockets) are anaerobic environments exposed to similar nutrients.

3.2.3 | Risk factors

The main risk factors for the occurrence of EPL were advanced periodontitis, trauma, and iatrogenic events. Other reported risk factors were the presence of grooves, furcation involvement, porcelain-fused-to-metal crowns and active carious lesions (see Appendix 5, Table A5.1, in online journal). Furcation involvement, high level of bone destruction around the affected tooth, and anatomic problems (e.g. the presence of grooves), could worsen the prognosis of EPL. Most of the single EPL in non-periodontitis patients reported in the literature were associated with palatal grooves.

3.3 | Pathophysiology and histological features

The dental pulp and the periodontium have different communication pathways, such as the apical radicular foramina, accessory (or lateral) canals, and dentinal tubules.[112] Accessory canals are more prevalent at the apical third of the roots, but they may be found in high numbers in other areas, such as in the furcation regions.[112,113] Pathological communication between these structures, which includes the migration of microorganisms and inflammatory mediators between the root canal and the periodontium, may lead to the EPL.[89,112–116]

3.4 | Assessment and diagnosis

The classification system most commonly used for the diagnosis of EPL was published in 1972 by Simon et al.[89] and included the following categories: (1) primary endodontic lesions; (2) primary endodontic lesions with secondary periodontal involvement; (3) primary periodontal lesions; (4) primary periodontal lesions with secondary endodontic involvement; and (5) "true" combined lesions. The main drawback of this classification and a recent proposed amendment[117] was to base their categories on the primary source of infection (root canal or periodontal pocket). This seemed to be a suitable approach, as lesions of periodontal origin might have a worse prognosis than those of endodontic origin. Nonetheless, using "history of the disease" as the main criteria for diagnosis was not practical, because in the majority of cases the complete history is unavailable to the clinician. In addition, determining the primary source of infection is not relevant for the treatment of EPL, as both the root canal and the periodontal tissues would require treatment.[118,119] Thus, ideally, the diagnosis and classification of EPL should be based on the present disease status and on the prognosis of the tooth involved, which would determine the first step of the treatment planning that would be whether to maintain or extract the tooth.

The three main prognostic groups for a tooth with an EPL are: (1) hopeless, (2) poor, and (3) favorable. The hopeless prognosis is normally associated with EPL caused by trauma or iatrogenic factors, whereas the prognosis of a tooth with an EPL associated with endodontic and periodontal infections may range from favorable to hopeless, depending on the extension of the periodontal destruction around the affected tooth, and the presence and severity of the periodontal disease affecting the patient's oral health.

The first steps in diagnosis should be to assess patient's history and clinical or radiographic examination. Patient history is important for identifying the occurrence of trauma, endodontic instrumentation or post preparation. If one or more of these events are identified, detailed clinical and radiographic examinations should be conducted to seek the presence of perforations, fractures, and cracking or external root resorption. Careful radiographic evaluation and clinical examination of the root anatomy is of great importance at this stage, to assess the integrity of the root and to help with differential diagnosis. A radicular groove, for example, might mimic a vertical root fracture in the radiograph.[120]

If perforations and fractures are not identified, the diagnosis should proceed to a second phase consisting of full-mouth periodontal assessment, including probing depth, attachment level, bleeding on probing, suppuration and mobility, as well as tooth vitality and percussion tests. The presence of a periodontal pocket reaching or close to the apex combined with absence of pulp vitality would indicate the presence of an EPL.

3.5 | Proposed changes to the current 1999 classification

For the first time, the 1999 classification system for Periodontal Diseases and Conditions[118,121] included the EPLs, which were described under Section VII - Periodontitis Associated with Endodontic Lesion, as a single category entitled "Combined Periodontal-Endodontic Lesions." An advantage of this classification over the previous ones[89,117] was that it

TABLE 6 Proposal for endo-periodontal lesions classification

Endo-periodontal lesion with root damage	Root fracture or cracking	
	Root canal or pulp chamber perforation	
	External root resorption	
Endo-periodontal lesion without root damage	Endo-periodontal lesion in periodontitis patients	*Grade 1* – narrow deep periodontal pocket in 1 tooth surface
		Grade 2 – wide deep periodontal pocket in 1 tooth surface
		Grade 3 – deep periodontal pockets in more than 1 tooth surface
	Endo-periodontal lesion in non-periodontitis patients	*Grade 1* – narrow deep periodontal pocket in 1 tooth surface
		Grade 2 – wide deep periodontal pocket in 1 tooth surface
		Grade 3 – deep periodontal pockets in more than 1 tooth surface

reflected the current clinical condition of the lesion, thereby overcoming the problem of using "history of the disease" as the main criteria. Nonetheless, the following problems were associated with this classification system: (1) grouping all EPL under a single section entitled "Periodontitis Associated with Endodontic Lesion" was not ideal, as these lesions may occur in subjects with or without periodontitis; (2) the single category presented, "Combined Periodontal-Endodontic Lesions", was too generic and not sufficiently discriminative to help the clinician to determine the most effective treatment for a particular lesion. Finally, EPL should be classified according to signs and symptoms feasible to be assessed at the time that the lesion is detected and that have direct impact on their treatment, such as presence or absence of fractures and perforations, presence or absence of periodontitis, and the extent of the periodontal destruction around the affected teeth (Table 6).

OBSERVATIONS AND DISCUSSION

The present literature review focused on three conditions that have in common a possible acute onset and severe destruction. A comprehensive analysis of the available scientific literature (336 studies were included) allowed for a description of the importance, etiology, pathogenesis and diagnosis, together with the proposal of new classifications.

Quality of the available evidence

In general, the evidence to define the etiology, diagnosis, prognosis and treatment of the teeth affected by the three conditions studied was considered limited. Most of the included studies were case reports with small sample sizes. Very few clinical studies with a reasonable number of cases were found, and no robust epidemiological studies were identified (see Appendix 1 in online journal). To enable solid evidence on these lesions to be made available, additional studies with adequate designs and sample sizes are needed, specifically on the topics with less information available (e.g. PA in non-periodontitis patients, and EPL).

Pending topics for the proposed classification

The topic of whether the lesions associated with root alterations and damage (e.g. fracture, perforation, root resorption), should be classified in a different category, is debatable. However, because these lesions are EPL in nature (i.e., invariably affect both the periodontium and the pulp-root canal complex, irrespective of being associated with abscess formation, or not), we understand that they should be classified as such. Thus, in the classification system suggested for EPL, these conditions are grouped as "EPL associated with trauma and iatrogenic factors."

Pericoronal abscesses have been excluded from the category of PA.[121] However, pericoronitis may still be considered an acute periodontal condition, but in a separate category.

"Periodontal" abscesses around implant sites have also been described.[122,123] Considering that from a histological point of view the lesions may be similar, it is also debatable whether they should be given a different name should be given to these lesions (e.g. "periimplant" abscesses) and whether they should be classified together with the other abscesses in the periodontium.

In this manuscript, the term "risk factor" was used, however, in some cases, the available literature was insufficient to support the use of this term.

CONCLUSIONS

PAs can present different aetiologies, and they should be classified according to the aetiological factors involved. These lesions are commonly associated with reduced drainage of a deep periodontal pocket. They normally cause rapid tissue destruction, which may compromise the prognosis of teeth, and represent one of the most frequent reasons for tooth extraction during PeM. PAs are also associated with evident systemic risks.

NPD present three typical clinical features: papilla necrosis, bleeding, and pain. They represent the most severe biofilm-related periodontal condition. The onset, severity, extent, and progression of NPD are clearly associated with the host immune response, giving credit to a classification based on this response.

An EPL is a pathological communication between the endodontic and periodontal tissues of a given tooth. It may occur in acute or chronic forms and should be classified according to signs and symptoms that have direct impact on their prognosis and treatment, such as presence or absence of fractures and perforations, presence or absence of periodontitis and the extent of periodontal destruction around the affected teeth.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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