

CLINICAL AND HISTOPATHOLOGICAL EVALUATION OF PERIO-ENDO LESIONS: ASSOCIATION WITH PERIODONTAL DISEASE SEVERITY AND ENDODONTIC TREATMENT OUTCOMES

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ABSTRACT

Background: Perio-endodontic lesions are lesions that are concurrent or communicating lesions between the periodontal tissues and the dental pulp. It is very difficult to diagnose and manage them because the clinical, radiographic and histopathological features can overlap. The severity of the periodontal disease can also affect healing from endodontic treatment. **Objective:** To evaluate the clinical and histopathological characteristics of perio-endodontic lesions and determine their association with periodontal disease severity and endodontic treatment outcomes. **Materials and Methods:** This prospective observational study was conducted at Karachi Metropolitan University, Karachi, from January to June 2025. Total 72 patients with clinically and radiographically diagnosed perio-endodontic lesions were selected by consecutive sampling. These included demographic data, periodontal evaluation, pulp status, lesion classification, radiologic data, and histopathological data. The response to treatment was classified as success or failure. Appropriate statistical tests were used for assessing associations and $p < 0.05$ was taken as significant. **Result:** The mean age of the participants was 43.6 ± 11.2 years, and 41 (56.9%) were male. True combined lesions were the most frequent type, occurring in 23 (31.9%) patients. Moderate and severe periodontal disease were observed in 28 (38.9%) and 24 (33.3%) patients, respectively. Chronic inflammatory infiltrates were identified in 39 (54.2%) lesions, while moderate and severe histopathological inflammation were present in 30 (41.7%) and 24 (33.3%) cases. Histopathological severity increased significantly with worsening periodontal disease ($p < 0.001$). Successful treatment outcomes were recorded in 53 (73.6%) patients. Success decreased from 90.0% in mild periodontal disease to 54.2% in severe disease ($p = 0.020$). Severe periodontal disease, severe histopathological inflammation, true combined lesions, and advanced tooth mobility were associated with unsuccessful outcomes. **Conclusion:** Periodontal disease severity was significantly associated with histopathological inflammation and endodontic treatment outcomes in patients with perio-endodontic lesions. Accurate diagnosis and coordinated periodontal-endodontic management are essential for improving healing and tooth retention.

INTRODUCTION

Perio-endodontic lesions are any pathological condition in which pulpal and periodontal disease(s) are associated with one another or interconnected through either anatomic or pathologic pathways. Close connections between the dental pulp and the periodontal tissues occur: through the apical foramen, lateral canals and accessory canals, through exposed dentinal tubules, through dentinal^[1,2] can then develop into the other and result in a clinical and radiographic picture that is similar. The close biological similarity of these lesions often makes it difficult to distinguish if the lesion is primarily an endodontic or periodontal lesion or if it is a combination of both.^[3-5]

Clinically, gingivitis and periodontitis can occur either as simple or complicated conditions, depending on the source, duration and extent of infection. The clinical features are pain, tender to percussion, deep pockets, bleeding on probing, tooth mobility, sinus-tract formation, furcation involvement, periapical and/or lateral radiolucency. Diagnosis is important, including pulp-vitality tests, periodontal probing, mobility, percussion, palpation and radiography. But the results obtained here may be misleading in the presence of both advanced periodontal destruction and pulpal necrosis. Therefore, the classification is important as the treatment sequence and prognosis vary between primary endodontic, primary periodontal and true combined lesions.^[6]

Histopathological examination can give further details about the type and extent of tissue damage. Inflammatory-cell infiltrate may be acute, chronic, or both, in addition to granulation tissue, fibrosis, epithelial proliferation, necrosis, vascular changes, bacterial colonies, and bone and/or cementum resorption. The magnitude of these changes could be related to the length and severity of infection. However, the histopathological evaluation should be combined with clinical and radiographic data as microscopic examination may not be able to determine the original route of the disease. To determine if any correlation exists between clinicopathological assessments and severe periodontal destruction and the extent of inflammatory changes.^[7-9]

The outcome of endodontic treatment in teeth with perio-endodontic involvement depends on several factors, including lesion origin, periodontal attachment loss, probing depth, tooth mobility, furcation involvement, pulpal status, root damage, quality of root-canal treatment, systemic health, and patient compliance. Lesions of mainly endodontic origin commonly respond well to adequate canal disinfection and obturation. In contrast, true combined lesions and teeth with advanced periodontal disease may have a less favourable prognosis because periodontal infection and structural support loss may persist after endodontic treatment. Limited local evidence is available

regarding the relationship among periodontal severity, histopathological inflammation, and treatment success. Therefore, this study was conducted to evaluate the clinical and histopathological features of perio-endodontic lesions and determine their association with periodontal disease severity and endodontic treatment outcomes.

MATERIALS AND METHODS

This prospective observational study was conducted at the Department of Periodontology and Department of Operative Dentistry/Endodontics, Karachi Metropolitan University, Karachi, from January 2025 to June 2025. A total of 72 patients presenting with clinically and radiographically diagnosed perio-endodontic lesions were enrolled through non-probability consecutive sampling. Patients of either sex, aged 18 years or above, with at least one tooth showing combined periodontal and endodontic involvement were considered eligible. The diagnosis was based on periodontal probing, pulp-vitality testing, percussion and palpation findings, tooth mobility, and radiographic evidence of periodontal or periapical destruction. Patients with incomplete clinical records, recent periodontal or endodontic treatment, severe systemic illness, ongoing immunosuppressive therapy, pregnancy, non-restorable teeth without obtainable tissue samples, or unwillingness to participate were excluded.

The demographic characteristics, medical history, oral hygiene practices, tobacco use, presence of diabetes mellitus, previous dental treatment and presenting complaints were recorded on a structured data-collection form. The clinical examination was carried out under standard illumination and with the help of a mouth mirror, periodontal probe, explorer and suitable endodontic diagnostic instruments. The following clinical findings were recorded: affected tooth, the type of the tooth, the presence of dental caries, the presence of restorations, pain, swelling, sinus tract, pus discharge, tenderness to percussion, and mobility grade. Periodontal assessment involved assessment of plaque accumulation, BOP, probing pocket depth, gingival recession, clinical attachment loss and furcation involvement. Measurements were made at six locations around the involved tooth using a calibrated periodontal probe (in millimetres). Severe periodontal disease was defined as mild, moderate, or severe, based on the amount of clinical attachment loss and the amount of radiographic bone loss.

Endodontic examination involved thermal pulp testing, electric pulp testing as needed, percussion, palpation and past history of root canal therapy. The clinical findings of the teeth were classified as vital, irreversibly inflamed, necrotic, previously initiated, or previously treated. Paralleling technique was used for standardised intraoral periapical radiographs. Radiographic parameters included periapical

radiolucency, widening of the periodontal ligament space, loss of lamina dura, horizontal or vertical bone destruction, furcation radiolucency, root resorption and suspected communication between the periodontal and periapical lesions. On the basis of combined clinical and radiographic evaluation, the lesions were classified as primary endodontic lesion, primary periodontal lesion, primary endodontic lesion with secondary periodontal lesion, primary periodontal lesion with secondary endodontic lesion, or true combined lesion of the periodontal and endodontic disease.

Histopathological samples were only retrieved if excising tissue was part of the clinically indicated procedure, e.g. curettage, periodontal surgery, apical surgery, extraction of a non-restorable tooth. No tissues were removed for the purpose of research. Collected tissues were then placed immediately in 10% neutral buffered formalin and sent to the pathology laboratory. Specimens were fixed and processed routinely, embedded in paraffin wax, sectioned at about 4–5 µm and stained with hematoxylin and eosin. An experienced histopathologist blind to the clinical severity and outcome of treatment examined the slides. Histopathological features were acute inflammatory infiltrates, chronic inflammatory infiltrates, mixed inflammatory infiltrates, granulation tissue, fibrosis, epithelial proliferation, necrosis, vascular proliferation, microabscess formation, bacterial colonies and bone or cementum resorption. Severity of inflammation was assessed as mild, moderate, or severe and based upon inflammatory cell density, distribution, and tissue destruction.

All affected teeth were treated based on the clinical diagnosis and restorability of the teeth. All nonsurgical root-canal and retreatment procedures used the usual isolation, preparing the canal, flushing the interior with irrigating solutions, introducing intracanal medication when necessary, and sealing

the canal with temporary cement. Oral hygiene was taught, scaling and root planing was provided, and surgical therapy was done as clinically indicated for periodontal management. Patients were re-evaluated at predetermined intervals to determine pain, swelling, sinus tract, tenderness, probing pocket depth, clinical attachment level, tooth mobility and radiographically to assess healing. The success of treatment was defined as the maintenance of tooth function and lack of clinical inflammation and/or radiographic signs of healing. A failure to resolve, progressive loss of radiolucency, recurrent swelling, continued deep periodontal pockets, requiring retreatment, or extraction were defined as "unsuccessful outcomes. SPSS Statistics version 26 was used to enter and analyze the data. Categorical data were analyzed by computing frequencies and percentages, and continuous data were analyzed by computing means and standard deviations. Associations between categorical variables were evaluated by the chi-square test or Fisher's exact test; numerical comparisons were evaluated by independent-samples t-tests, ANOVA tests or their non-parametric alternatives. A logistic regression model was used to identify the predictors of treatment failure. The p value was considered statistically significant when < 0.05.

RESULTS

The total number of patients with perio-endodontic lesions included in the study were 72. The average age of the subjects was 43.6 ± 11.2 years, ranging from 21 to 68 years. The majority of patients were in the age range 41–50 years. There were 41 (56.9%) males and 31 (43.1%) females. There were 15 (20.8%) patients with diabetes mellitus and 17 (23.6%) current smokers. Thirty-one (43.1%) participants had poor oral hygiene.

Table 1. Demographic and baseline characteristics of the study participants (n = 72)

Variable	Frequency, n (%) or Mean $\pm$ SD
Age, years	43.6 $\pm$ 11.2
20–30 years	10 (13.9)
31–40 years	18 (25.0)
41–50 years	24 (33.3)
>50 years	20 (27.8)
Male	41 (56.9)
Female	31 (43.1)
Current smoker	17 (23.6)
Diabetes mellitus	15 (20.8)
Poor oral hygiene	31 (43.1)
Previous periodontal treatment	19 (26.4)
Previous endodontic treatment	22 (30.6)

Most common tooth involvement was found in the mandibular molars with 28 cases (38.9%) and maxillary molars, 19 cases (26.4%). Pain was reported by 51 (70.8%) patients, tenderness on

percussion by 46 (63.9%), and swelling by 24 (33.3%). In 17 (23.6%) cases a sinus tract was detected. 29 (40.3%) patients showed Grade II or III mobility of their teeth.

Table 2. Clinical and tooth-related characteristics of perio-endodontic lesions

Variable	Frequency, n (%)
Maxillary anterior tooth	8 (11.1)
Maxillary premolar	7 (9.7)
Maxillary molar	19 (26.4)
Mandibular anterior tooth	3 (4.2)
Mandibular premolar	7 (9.7)
Mandibular molar	28 (38.9)
Pain	51 (70.8)
Tenderness on percussion	46 (63.9)
Swelling	24 (33.3)
Sinus tract	17 (23.6)
Pus discharge	20 (27.8)
Grade II-III tooth mobility	29 (40.3)
Furcation involvement	27 (37.5)
Periapical radiolucency	58 (80.6)

The most prevalent type of lesion was true combined perio-endodontic lesions (23, 31.9%) patients. Nineteen (26.4%) primary endodontic lesions were associated with secondary periodontal involvement

while 16 (22.2%) had primary periodontal involvement and secondary endodontic involvement. Developing primary endodontic and primary periodontal lesions were rather uncommon.

Table 3. Distribution of perio-endodontic lesions according to clinical classification

Type of lesion	Frequency, n (%)
Primary endodontic lesion	8 (11.1)
Primary periodontal lesion	6 (8.3)
Primary endodontic lesion with secondary periodontal involvement	19 (26.4)
Primary periodontal lesion with secondary endodontic involvement	16 (22.2)
True combined perio-endodontic lesion	23 (31.9)
Total	72 (100.0)

Periodontal disease was determined to be mild in 20 (27.8%), moderate in 28 (38.9%) and severe in 24 (33.3%) patients, based on clinical attachment loss. The mean probing depth and mean clinical attachment loss were 6.2 ± 1.8 mm and 5.4 ± 2.1 mm,

respectively, and 59 (81.9%) patients had bleeding on probing. Vertical bone loss was noted in 44 (61.1%) cases and radiographic furcation involvement was seen in 27 (37.5%) cases.

Table 4. Periodontal and radiographic findings among the participants

Finding	Frequency, n (%) or Mean $\pm$ SD
Mean probing pocket depth, mm	6.2 ± 1.8
Mean clinical attachment loss, mm	5.4 ± 2.1
Bleeding on probing	59 (81.9)
Mild periodontal disease	20 (27.8)
Moderate periodontal disease	28 (38.9)
Severe periodontal disease	24 (33.3)
Horizontal bone loss	28 (38.9)
Vertical bone loss	44 (61.1)
Furcation involvement	27 (37.5)
Periapical radiolucency	58 (80.6)
Root resorption	9 (12.5)
Loss of lamina dura	42 (58.3)

Histopathological examination revealed that 39 (54.2%) lesions had chronic inflammatory infiltrates, 21 (29.2%) lesions had mixed inflammatory infiltrates, and 12 (16.7%) lesions had predominantly acute inflammatory infiltrates. Thirty (41.7%) specimens showed moderate inflammation and 24

(33.3%) specimens showed severe inflammation. Focal tissue necrosis, epithelial proliferation, fibrosis and granulation tissue were observed in 19 (26.4%), 28 (38.9%), 31 (43.1%) and 50 (69.4%) specimens respectively.

Table 5. Histopathological findings in perio-endodontic lesions

Histopathological finding	Frequency, n (%)
Predominantly acute inflammatory infiltrate	12 (16.7)
Predominantly chronic inflammatory infiltrate	39 (54.2)
Mixed inflammatory infiltrate	21 (29.2)
Mild inflammation	18 (25.0)
Moderate inflammation	30 (41.7)
Severe inflammation	24 (33.3)

Granulation tissue	50 (69.4)
Fibrosis	31 (43.1)
Epithelial proliferation	28 (38.9)
Tissue necrosis	19 (26.4)
Microabscess formation	14 (19.4)
Vascular proliferation	35 (48.6)
Bacterial colonies	16 (22.2)
Bone or cementum resorption	20 (27.8)

The correlation between histopathological severity and degree of periodontal disease showed that the higher the grade of periodontal disease, the more severe the histopathological severity. The majority of patients with mild periodontal disease, 8 (28.6%) and 14 (58.3%) patients with moderate and severe

periodontal disease, respectively, had severe histological inflammation, while 2 (10.0%) patients with mild periodontal disease had severe histological inflammation. A correlation was found between the severity of periodontal disease and the histopathological grade of inflammation ($p < 0.001$).

Table 6. Association between periodontal disease severity and histopathological inflammation

Periodontal severity	Mild inflammation n (%)	Moderate inflammation n (%)	Severe inflammation n (%)	Total	p-value
Mild	11 (55.0)	7 (35.0)	2 (10.0)	20	
Moderate	6 (21.4)	14 (50.0)	8 (28.6)	28	
Severe	1 (4.2)	9 (37.5)	14 (58.3)	24	<0.001
Total	18	30	24	72	

All the patients were treated with non-surgical endodontics (NSE) and the clinical indication of the patient was treated with the periodontal treatment. Successful treatment outcomes were noted in 53 (73.6%) patients while unsuccessful treatment outcomes were noted in 19 (26.4%). Successful clinical outcomes included the absence of pain, swelling, sinus tract and tenderness, and

improvement in periodontal and radiographic parameters. The success rates declined in severity of periodontal disease. In 18 out of 20 (90.0%) patients with mild PD, 22 out of 28 (78.6%) patients with moderate PD, and 13 out of 24 (54.2%) patients with severe PD were successfully treated. This association was statistically significant ($\chi^2 = 7.79$ $p = 0.020$).

Table 7. Association between periodontal disease severity and endodontic treatment outcome

Periodontal disease severity	Successful outcome n (%)	Unsuccessful outcome n (%)	Total	p-value
Mild	18 (90.0)	2 (10.0)	20	
Moderate	22 (78.6)	6 (21.4)	28	
Severe	13 (54.2)	11 (45.8)	24	0.020
Total	53 (73.6)	19 (26.4)	72	

The type of PERL also influenced the treatment outcome. The success rates were highest for primary endodontic lesions and lowest for true combined lesions. Out of 23 patients with true combined

lesions, 13 (56.5%) were successfully treated while 10 (43.5%) were not. The strength of the association between the classification of lesions and treatment outcome was statistically significant ($p = 0.041$).

Table 8. Endodontic treatment outcomes according to lesion type

Lesion type	Successful n (%)	Unsuccessful n (%)	Total
Primary endodontic lesion	8 (100.0)	0 (0.0)	8
Primary periodontal lesion	5 (83.3)	1 (16.7)	6
Primary endodontic with secondary periodontal involvement	16 (84.2)	3 (15.8)	19
Primary periodontal with secondary endodontic involvement	11 (68.8)	5 (31.3)	16
True combined lesion	13 (56.5)	10 (43.5)	23
Total	53 (73.6)	19 (26.4)	72

Patients who had successful outcomes had more reduction in probing pocket depth and periapical lesion size. The mean probing depth at follow-up was 6.0 ± 1.6 mm at baseline and 3.5 ± 1.1 mm at follow-up in the successful-outcome group. For the patients

who had unsuccessful outcomes, it dropped from 6.8 ± 1.9 mm to 5.4 ± 1.6 mm. The difference in the reduction of probing depth between the two groups was statistically significant ($p < 0.001$).

Table 9. Comparison of clinical and radiographic changes according to treatment outcome

Variable	Successful outcome (n = 53)	Unsuccessful outcome (n = 19)	p-value
Baseline probing depth, mm	6.0 ± 1.6	6.8 ± 1.9	0.081
Follow-up probing depth, mm	3.5 ± 1.1	5.4 ± 1.6	<0.001
Reduction in probing depth, mm	2.5 ± 1.2	1.4 ± 1.0	<0.001
Baseline clinical attachment loss, mm	5.0 ± 1.8	6.5 ± 2.2	0.005

Follow-up clinical attachment loss, mm	3.3 ± 1.3	5.7 ± 1.8	<0.001
Reduction in periapical lesion size, mm	3.8 ± 1.5	1.4 ± 1.1	<0.001

Severe periodontal disease, severe histopathological inflammation, true combined lesions and baseline tooth mobility of Grade II or III were significant independent predictors of unsuccessful treatment on multivariable logistic regression analysis. More than

a fourfold increase in risk of treatment failure was found for people with severe periodontal disease. The odds of unsuccessful outcome were increased for diabetes mellitus and smoking but were not statistically significant.

Table 10. Factors associated with unsuccessful endodontic treatment outcomes

Predictor	Adjusted odds ratio	95% confidence interval	p-value
Severe periodontal disease	4.38	1.32–14.55	0.016
Severe histopathological inflammation	3.72	1.18–11.74	0.025
True combined perio-endodontic lesion	3.41	1.09–10.68	0.035
Grade II–III tooth mobility	3.09	1.01–9.46	0.048
Diabetes mellitus	2.07	0.65–6.58	0.217
Current smoking	1.84	0.60–5.64	0.286

Overall, the findings indicated that perio-endodontic lesions were characterized by substantial clinical, radiographic, and histopathological inflammation. Treatment was successful in nearly three-quarters of the patients. However, severe periodontal destruction, intense histopathological inflammation, advanced tooth mobility, and true combined lesions were associated with poorer endodontic treatment outcomes.

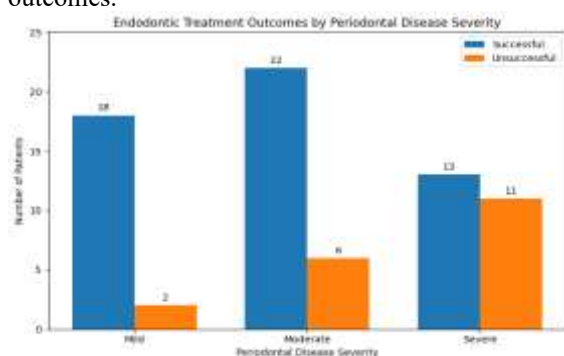


Figure 1. Endodontic treatment outcomes according to periodontal disease severity.

Treatment success decreased as periodontal disease severity increased, from 90.0% in mild disease to 54.2% in severe disease.

DISCUSSION

The present study evaluated the clinical, radiographic, and histopathological characteristics of perio-endodontic lesions and examined their association with periodontal disease severity and endodontic treatment outcomes. True combined perio-endodontic lesions were the most frequent lesion type, followed by primary endodontic lesions with secondary periodontal involvement. Most affected teeth showed pain, percussion tenderness, deep periodontal pockets, clinical attachment loss, bleeding on probing, and periapical radiolucency. These findings reflect the complex communication between pulpal and periodontal tissues through the apical foramen, lateral and accessory canals, dentinal tubules, developmental defects, root fractures, and

iatrogenic pathways. Current expert guidance emphasizes that the origin and extent of both infections must be identified carefully because incorrect classification can lead to inappropriate treatment and an inaccurate prognosis.^[10-13]

Most of the patients in the present study had moderate to severe periodontal disease, and the most common sites were the mandibular molars. Molars may be especially prone to failure due to their complicated root canal anatomy, furcation area, accessory canals, and the challenge of maintaining adequate plaque control. The high prevalence of vertical bone loss, furcation involvement, tooth mobility and periapical radiolucency in the present study suggested that many of the lesions were diagnosed following significant destruction of the supporting tissues. Modern publications also refer to endo-periodontal lesions as diagnostic dilemmas in which, in addition to microbial biofilm, anatomical communication, root injury, and pre-existing periodontitis, may all play a role. The significance of the presence of periodontal destruction is that, even if technically sound, the prognosis is poor if there is extensive attachment loss, deep probing depths, furcation involvement, or poor restorability.^[14-16]

The most frequent findings in the present study using the histopathological examination were the presence of chronic inflammatory infiltration, granulation tissue, vascular proliferation, fibrosis and epithelial proliferation. There was a significant clinicopathological association between severe histopathological inflammation and the severity of mild, moderate and severe periodontal disease. The finding confirms the biologic link between the extent of clinical periodontal destruction and the extent of the microscopic inflammatory change. Ongoing stimulation by microbes can encourage infiltration of lymphocytes, plasma cells, macrophages and neutrophils, along with granulation tissue formation, tissue necrosis, fibrosis and resorption of mineralized tissues. Histological findings also have shown that in advanced periodontitis, the disease process may also be responsible for pulpal inflammation, fibrosis, calcification and necrosis, especially when periodontal destruction extends into the region of the

root-canal system's lateral and apical communications. However, the histological characteristics alone will not identify the site of original infection and should be used in conjunction with pulp-vitality tests, periodontal probing, radiographs, and the clinical history of the patient.^[17,18]

With regard to overall treatment success, this study showed a success rate of 73.6%, with this rate decreasing in severe periodontal disease (54.2%) compared with mild periodontal disease (90.0%). The true combined lesions also showed inferior results compared to lesions that are primarily pulpal. The results are clinically plausible because there is generally a good prognosis for a mainly endodontic lesion after appropriate canal disinfection, while combined lesions may have deep periodontal pockets, significant attachment loss, furcation involvement and residual periodontal infection after root-canal therapy. Studies also concluded that the prognosis of endodontic-periodontal lesions was moderated by periodontal and tooth-related factors and that the complexity of the diseases was related to the chances of success and survival. Combined endodontic and periodontal therapy has been reported to provide acceptable clinical results, and isolated endodontic therapy has been reported to be less effective when combined with periodontal disease.^[19,20]

In the present study, the severity of periodontal disease, severe histopathological inflammation, true combined lesions and Grade II–III tooth mobility were found to be predictive of failed treatment. The results underscore the importance of early diagnosis, proper diagnosis of primary lesion, evaluation of restorability and endodontic-periodontal co-ordinated treatment. With pulpal infection, endodontic treatment is usually performed first, and then a period of reassessment before definitive periodontal or regenerative treatment is planned. There is general agreement and clinical reviews that an individual, multi-specialty approach taking into consideration the pulp status, periodontal attachment, lesion anatomy, root damage, tooth mobility, patient-related risk factors, and response to initial therapy is recommended. However, the present results must be recognized with a number of restrictions. The study took place in one institution, and a small sample of 72 patients was used, which may limit the generalizability and multivariable estimates. Selection bias may have occurred in consecutive sampling and a relatively small follow-up may not have adequately identified late failures and complete radiographic healing. Only lesions for which tissue removal is required were available for histopathological examination, which may have favoured more advanced lesions. Multicentre trials with more patients, systematic classification of the lesions, standardisation of periodontal measurements, blinded evaluation of the radiographs, microbiological analysis and extended follow-up are suggested.

CONCLUSION

Perio-endodontic lesions demonstrated substantial clinical, radiographic, and histopathological variation. Chronic inflammatory infiltration and granulation tissue were the predominant microscopic findings, and the severity of histopathological inflammation increased significantly with worsening periodontal disease. Endodontic treatment produced favourable outcomes in most patients, but success was considerably lower among those with severe periodontal destruction, advanced tooth mobility, severe microscopic inflammation, and true combined lesions. Comprehensive clinical examination, pulp-vitality testing, periodontal assessment, radiographic evaluation, and appropriate histopathological examination are therefore important for establishing the diagnosis and estimating prognosis. Early, coordinated endodontic and periodontal treatment may improve tooth retention and reduce the risk of treatment failure.

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