



**POST-ERUPTIVE NON-CARIOUS DENTAL LESIONS: CLINICAL FEATURES AND  
DIFFERENTIAL DIAGNOSIS**

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**Abstract:** Non-cariou lesions developing after tooth eruption represent a common group of dental conditions frequently encountered in clinical practice; however, they are often underestimated or misinterpreted at early stages. These disorders are characterized by the loss of hard dental tissues not related to dental caries and are associated with the progressive destruction of enamel and dentin under the influence of mechanical, chemical, or biomechanical factors. The clinical significance of non-cariou lesions lies in their tendency to cause increased tooth sensitivity, aesthetic defects, alterations in tooth morphology, dentin exposure, and subsequent secondary complications. Moreover, non-cariou processes may clinically mimic dental caries, pulpitis, periodontitis, as well as enamel–dentin pathologies associated with endocrine or gastroenterological diseases, thereby complicating differential diagnosis. This article provides a clinical analysis of the most common post-eruptive non-cariou lesions, including enamel erosion, abfraction, abrasion, attrition, wedge-shaped defects, hypoplasia, and pathological disturbances of enamel mineralization. For each condition, etiopathogenetic factors, clinical manifestations, diagnostic criteria, and distinguishing features from dental caries and other hard tissue defects are discussed. In addition, modern diagnostic approaches used in contemporary dentistry are reviewed, with emphasis on the diagnostic value of comprehensive history taking, clinical examination, and instrumental assessment methods (transillumination, radiological evaluation, and index-based analysis), as well as evidence-based individualized therapeutic strategies. The present article is intended to support dentists, residents in restorative dentistry, and practicing clinicians in improving differential diagnostic skills and developing early detection strategies in routine clinical practice.



**Keywords:** non-carious lesions, enamel erosion, abrasion, attrition, abfraction, wedge-shaped defect, dentin hypersensitivity, differential diagnosis, enamel–dentin damage, remineralization

**Introduction:** In clinical dental practice, caries-related processes remain the leading cause of hard dental tissue damage; however, in recent years, the clinical relevance of post-eruptive non-carious tooth lesions has been steadily increasing. Non-carious tooth lesions (NCTLs) are characterized by structural loss of enamel and dentin occurring in the absence of a carious process. Due to their frequently chronic course, slow progression, and the long-term absence of pronounced clinical symptoms, these pathologies are difficult to detect in the early stages. At the same time, non-carious lesions may significantly affect patients' quality of life, as they can be associated with increased dentin hypersensitivity, pain syndrome, aesthetic defects, morphological deformation of teeth, and functional impairment.

In contemporary dentistry, non-carious lesions are considered multifactorial disorders of hard dental tissues. Their development involves the combined influence of multiple etiological factors, including improper oral hygiene habits, long-term use of highly abrasive toothpastes, toothbrush hardness, incorrect brushing technique, uneven occlusal load distribution, bruxism, psychoemotional stress, gingival recession, and systemic conditions such as dietary acid exposure and gastroesophageal reflux disease. These factors may alter the mineral composition of dental tissues, reduce the physico-mechanical strength of enamel and dentin, and lead to various clinical forms of hard tissue defects.

Among non-carious conditions, enamel erosion, abrasion, attrition, abfraction, wedge-shaped defects, pathological tooth wear, as well as disturbances in enamel mineralization represent the most clinically significant entities. Each of these conditions differs in terms of clinical presentation, localization, morphological changes on tooth surfaces, and the rate of progression. For instance, enamel erosion is primarily caused by chemical (acidic) factors and is manifested by surface smoothing, increased glossiness, and thinning of the enamel layer. Abrasion is associated with mechanical friction and most commonly results in cervical defects due to improper toothbrushing habits. Attrition develops mainly as a consequence of tooth-to-tooth contact, leading to flattening of occlusal surfaces and reduction in vertical tooth height. Abfraction is explained by biomechanical stress, resulting in microdeformation and tissue loss in the cervical region. Accurate clinical evaluation and differential diagnosis of these lesions constitute a critical component of routine dental practice. This is due to the fact that non-carious lesions may closely mimic early-stage dental caries, secondary caries, dentin hypersensitivity, early forms of pulpitis, enamel hypoplasia, fluorosis, and dentinogenesis-related anomalies. Diagnostic errors may lead to inappropriate treatment planning, unnecessary invasive procedures, excessive removal of sound tooth tissue, and an increased risk of pain and secondary complications. In clinical practice, the diagnosis of non-carious lesions requires not only visual examination but also comprehensive history taking, occlusal assessment, periodontal evaluation, sensitivity testing, transillumination, radiological assessment, and the use of clinical indices. Furthermore, evaluation of dietary habits, beverage consumption, acid exposure, gastrointestinal disease history, bruxism, and stress-related factors plays an important role in establishing an accurate differential diagnosis. Therefore, the aim of the present article is to provide an evidence-based analysis of the clinical features, etiopathogenetic factors, and differential diagnostic criteria of post-eruptive non-carious tooth lesions. The article also highlights modern diagnostic approaches applied in contemporary dentistry, methodological aspects of clinical



assessment, and the significance of individualized treatment and preventive strategies. Such an approach serves as an essential methodological basis for early diagnosis, slowing disease progression, and improving patients' quality of life in dental practice.

**Main part:** Post-eruptive non-carious tooth lesions (NCTLs) represent a clinically significant group of disorders characterized by progressive loss of enamel and dentin unrelated to bacterial caries. In modern dentistry, these conditions are increasingly recognized not only as local structural defects but also as complex multifactorial pathologies reflecting the interaction of mechanical, chemical, and biomechanical factors. The importance of these lesions is determined by their high prevalence, frequent association with dentin hypersensitivity, aesthetic impairment, and the risk of secondary complications, including pulpal inflammation and functional disturbances. In many cases, NCTLs progress slowly and remain undetected until irreversible hard tissue loss occurs, which emphasizes the necessity of early clinical identification and accurate differential diagnosis. From a pathophysiological standpoint, the development of non-carious lesions is closely associated with disturbances in the balance between demineralization and remineralization processes within enamel and dentin. Although the oral environment is physiologically capable of maintaining mineral homeostasis through saliva buffering systems and mineral exchange, persistent exposure to harmful factors may shift this balance toward structural weakening. Chemical erosion is one of the most common mechanisms of enamel destruction and is defined as irreversible loss of dental hard tissue caused by acids not derived from bacterial metabolism. Acidic exposure may originate from extrinsic sources, such as carbonated beverages, citrus fruits, sports drinks, and occupational chemical factors, as well as intrinsic sources including gastric acid regurgitation, gastroesophageal reflux disease, and eating disorders associated with recurrent vomiting. Clinically, enamel erosion is characterized by smooth, glossy surfaces, shallow concavities, and progressive thinning of enamel, particularly on palatal surfaces of maxillary anterior teeth in intrinsic erosion, and on occlusal surfaces of posterior teeth in extrinsic erosion. In advanced cases, dentin exposure results in hypersensitivity and accelerated tissue loss due to dentin's lower mineral density. Mechanical abrasion represents another frequent cause of post-eruptive hard tissue loss. Abrasion is associated with repeated frictional forces acting on the tooth surface, most commonly related to improper toothbrushing technique, excessive brushing pressure, and the use of highly abrasive toothpaste. Additionally, cultural and occupational habits, such as holding pins or needles between teeth, pipe smoking, or the use of abrasive cleaning powders, may contribute to abrasion. Cervical abrasion lesions typically present as well-defined, V-shaped or saucer-shaped defects at the cemento-enamel junction, often accompanied by gingival recession. A key clinical feature distinguishing abrasion from caries is the absence of soft, demineralized tissue and the presence of a hard, smooth lesion surface. In many patients, abrasion coexists with erosion, creating a combined lesion pattern that progresses more rapidly than isolated forms. Attrition is defined as tooth surface loss resulting from direct tooth-to-tooth contact and is considered a physiological phenomenon in mild forms. However, pathological attrition develops when the rate of wear exceeds the adaptive capacity of the dentition, leading to clinically relevant reduction in crown height, flattening of occlusal surfaces, and functional disturbances. Bruxism, both awake and sleep-related, is a major contributing factor. Patients with stress-related parafunctional activity often exhibit accelerated attrition, which may be accompanied by temporomandibular joint dysfunction, muscle fatigue, and pain. Clinically, attrition is typically characterized by matching wear facets on opposing teeth, shiny flattened surfaces, and, in severe cases, exposure of dentin with secondary dentin formation. Unlike erosion, attrition lesions are usually localized to



occlusal and incisal surfaces, and their morphology reflects the direction of functional movements. Abfraction has gained considerable attention as a biomechanical phenomenon leading to cervical tooth tissue loss. The concept of abfraction suggests that occlusal stress and non-axial loading generate tensile and compressive forces in the cervical area, resulting in microfractures of enamel and dentin. Over time, these microfractures contribute to the formation of wedge-shaped defects, particularly on the buccal surfaces of premolars and canines. Abfraction lesions are typically narrow, deep, sharply defined, and located near the cemento-enamel junction. They are often associated with occlusal disharmony, premature contacts, and parafunctional habits. Although the exact contribution of abfraction remains debated, clinical evidence indicates that cervical lesions frequently arise from a combination of abrasion, erosion, and stress-induced biomechanical factors. Wedge-shaped defects constitute a clinically relevant category of cervical non-cariou lesions that may develop as a result of multiple interacting mechanisms. These defects are commonly detected during routine dental examination and may present as V-shaped loss of hard tissue with sharp internal angles. Patients may complain of sensitivity to cold, sweet, or mechanical stimuli. Wedge-shaped defects are frequently accompanied by gingival recession and plaque accumulation due to altered surface anatomy, which may further increase the risk of periodontal inflammation. The progression of these lesions is influenced by oral hygiene practices, occlusal stress, salivary flow, and dietary habits. Importantly, wedge-shaped defects must be differentiated from root caries, which tends to present as soft, irregular lesions with a demineralized surface and may be associated with poor oral hygiene and high cariogenic activity. Disturbances in enamel mineralization, including hypoplasia and hypomineralization, represent another clinically important group of non-cariou conditions. Unlike acquired post-eruptive lesions, these disorders are often related to developmental disturbances occurring during amelogenesis. Enamel hypoplasia is characterized by quantitative enamel defects, such as pits, grooves, or missing enamel areas, whereas hypomineralization involves qualitative defects leading to porous enamel with reduced hardness. These conditions may predispose teeth to secondary breakdown, increased sensitivity, and caries susceptibility. Clinically, hypomineralized enamel may appear as demarcated opacities, white, yellow, or brown discolorations, and may be prone to posteruptive enamel breakdown. Differential diagnosis must consider fluorosis, which typically presents as diffuse opacities and symmetrical involvement, as well as early caries lesions that manifest as chalky white spots in plaque-retentive areas.

The diagnostic approach to non-cariou lesions requires systematic evaluation based on history, clinical examination, and, when necessary, instrumental methods. Comprehensive history taking should include detailed assessment of dietary habits, consumption of acidic foods and beverages, occupational exposures, systemic diseases, medication use, gastroesophageal reflux symptoms, and parafunctional activity. In addition, psychosocial factors such as stress and anxiety should be considered, as they may contribute to bruxism and altered salivary composition. Clinical examination should assess lesion localization, shape, surface texture, hardness, and color changes. Sensitivity testing, including air stimulation and thermal tests, provides important information regarding dentin exposure and pulpal status. Transillumination may assist in detecting enamel cracks and structural changes, while radiographic evaluation helps exclude proximal caries, pulpal pathology, and other hidden lesions.

Differential diagnosis remains a critical clinical task due to overlapping symptoms between non-cariou lesions and cariou processes. Cariou lesions typically present as localized demineralization associated with bacterial plaque, often characterized by softened enamel or



dentin and irregular margins. In contrast, non-carious lesions usually have smooth, hard surfaces and are not necessarily associated with plaque accumulation, although plaque may accumulate secondarily in cervical defects. Additionally, early caries lesions frequently occur in fissures, interproximal areas, and plaque-retentive zones, whereas non-carious lesions are commonly observed on cervical, buccal, palatal, or occlusal surfaces depending on etiological factors. Another important diagnostic challenge is distinguishing dentin hypersensitivity due to exposed dentin from early pulpitis. Pulpitis is often associated with spontaneous pain, prolonged response to thermal stimuli, and sometimes nocturnal discomfort, while hypersensitivity is typically short-lasting and stimulus-dependent. Management of non-carious lesions should be individualized and based on the primary etiological factors. Preventive strategies include dietary counseling aimed at reducing acidic exposure, improving oral hygiene technique, selecting low-abrasive toothpaste, and recommending soft-bristled toothbrushes. Patients with bruxism may benefit from occlusal splints, stress management, and occlusal adjustment when clinically justified. Remineralization therapy plays a significant role, particularly in early erosive lesions, and may involve fluoride varnishes, high-fluoride toothpaste, calcium-phosphate agents, and saliva stimulation methods. In cases of significant dentin exposure and sensitivity, desensitizing agents containing potassium salts, arginine, or glutaraldehyde may be used. Restorative treatment is indicated when lesions compromise aesthetics, structural integrity, or cause persistent sensitivity. Modern restorative materials, including resin composites and glass ionomer cements, may provide effective outcomes when proper isolation and adhesive protocols are applied. Overall, post-eruptive non-carious lesions represent a multifactorial and clinically challenging group of dental disorders requiring comprehensive diagnostic and therapeutic strategies. Accurate identification of etiological factors, careful clinical evaluation, and evidence-based management approaches are essential for preventing lesion progression, preserving tooth structure, and improving patient quality of life. Furthermore, integrating preventive dentistry, patient education, and long-term monitoring into routine practice may significantly reduce the burden of non-carious lesions and improve clinical outcomes in affected individuals.

**Conclusion:** Post-eruptive non-carious tooth lesions constitute a highly relevant clinical problem in contemporary dentistry due to their increasing prevalence, multifactorial etiology, and significant impact on oral health-related quality of life. Unlike caries, these lesions develop independently of bacterial biofilm activity and are primarily associated with mechanical abrasion, chemical erosion, tooth-to-tooth wear (attrition), and biomechanical stress-related tissue loss (abfraction). The progressive destruction of enamel and dentin may result in dentin hypersensitivity, aesthetic impairment, alteration of tooth morphology, occlusal dysfunction, and, in advanced cases, secondary pulpal involvement.

The clinical evaluation of non-carious lesions requires a comprehensive and systematic approach, as their manifestations may mimic early carious lesions, root caries, initial pulpitis, enamel developmental defects, fluorosis, and other enamel–dentin pathologies. Accurate differential diagnosis is therefore essential to avoid inappropriate invasive interventions, unnecessary removal of sound dental tissues, and incorrect treatment planning. The diagnostic process must include thorough history taking, assessment of dietary acid exposure, evaluation of oral hygiene practices, identification of parafunctional habits such as bruxism, and detection of systemic conditions including gastroesophageal reflux disease. In addition, clinical examination should focus on lesion morphology, localization, surface hardness, sensitivity testing, periodontal status, and occlusal analysis. The findings emphasize that non-carious lesions should be considered not only as isolated defects of hard dental tissues but also as markers of behavioral,



functional, and systemic risk factors. Consequently, effective clinical management requires etiological control, individualized preventive strategies, and long-term monitoring. Modern minimally invasive dentistry prioritizes early detection, remineralization therapy, patient education, and conservative restorative interventions when indicated. An integrated approach combining preventive dentistry, occlusal management, and patient-centered care is essential for reducing lesion progression, maintaining tooth vitality, and ensuring stable clinical outcomes.

#### **Practical recommendations:**

1. Early clinical detection and risk assessment: Routine dental examinations should include targeted screening for non-carious lesions, with emphasis on early erosive changes, cervical defects, and occlusal wear facets. Risk assessment should be performed based on patient history, clinical findings, and lifestyle factors.

2. Comprehensive history taking as a diagnostic standard: Dentists should systematically evaluate dietary habits, frequency of acidic beverage intake, occupational exposure, oral hygiene techniques, and systemic conditions such as gastroesophageal reflux disease. Identification of psychosocial stress and bruxism is strongly recommended.

3. Differential diagnosis to prevent overtreatment: Non-carious lesions must be carefully differentiated from dental caries, root caries, early pulpitis, enamel hypoplasia, fluorosis, and other developmental defects. Lesion surface hardness, morphology, and localization should be considered key diagnostic criteria.

4. Implementation of preventive strategies and patient education: Patients should receive individualized education on correct toothbrushing techniques, selection of low-abrasive toothpaste, and the use of soft-bristled toothbrushes. Dietary counseling should aim to reduce acidic exposure and improve oral environmental stability.

5. Remineralization and desensitization protocols: For early-stage lesions, non-invasive treatment should include fluoride varnish applications, high-fluoride toothpaste, calcium-phosphate remineralizing agents, and saliva stimulation methods. Dentin hypersensitivity may be managed using potassium-based, arginine-based, or glutaraldehyde-containing desensitizers.

6. Occlusal management and bruxism control: In patients with pathological attrition or suspected abfraction, occlusal evaluation should be performed. Occlusal splints, stress reduction strategies, and, where clinically justified, occlusal correction may be indicated to minimize biomechanical overload.

7. Indications for restorative intervention: Restorative treatment should be considered when lesions compromise aesthetics, structural integrity, or cause persistent sensitivity. Adhesive restorative materials, including resin composites and glass ionomer cements, should be selected according to lesion characteristics, moisture control feasibility, and long-term prognosis.

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