

Wedge defect: etiology, clinical features, classification and modern treatment approaches

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Annotation. A wedge defect (abfraction or non-carious cervical lesion, NCCL) is a defect in the cervical (neck) zone of the teeth that is not associated with caries of the enamel and dentin layers, resulting from mechanical and chemical exposure. This article is based on the etiopathogenesis of the wedge defect, morphological types, stages of development and phases of rejection (Yakovleva V.A. A wedge defect A wedge defect (abfraction or non-carious cervical lesion, NCCL) is a defect in the cervical (neck) zone of the teeth that is not associated with caries of the enamel and dentin layers, resulting from mechanical and/or chemical exposure. This article is based on the etiopathogenesis of the wedge defect, morphological types, stages of development and phases of rejection (Yakovleva V.I., Prosveryak G.P., Makhmudkhanov S.M., Fyodorov F.A., Drozhina V.A. based on their classifications), diagnostic capabilities (clinic, radiology, optical-and laboratory methods), as well as the results of international and domestic experiments (finite element analysis, retrospective clinical observations, prospective restorative studies) are analyzed. Based on their classifications), diagnostic capabilities (clinic, radiology, optical-and laboratory methods), as well as the results of international and domestic experiments (finite element analysis, retrospective clinical observations, prospective restorative studies) are analyzed. In the treatment strategy, it was shown that the compatibility of etiological (occlusive stress and parafunction), conservative (remineralization, desensitization) and restorative (adaptive restorations, bioactive materials, orthopedic approaches) components is necessary. The article proposes a diagnostic-therapeutic algorithm for clinical practice and directions for future research.

Keywords: Wedge defect, abfraction, non-carious cervical lesion (NCCL), occlusal stress, restorative therapy, cervical defects.

Introduction. Wedge defect (abfraction or non-carious cervical lesion, NCCL) is one of the most frequently encountered pathological conditions in modern dental practice, representing a group of non-carious cervical defects whose etiology and full pathogenesis still remain the subject of scientific discussion [1,2,3]. Clinically, these lesions are characterized by a V-shaped or wedge-shaped depression in the cervical region of the tooth, often affecting the labial or buccal surfaces of premolars and incisors. Patients may complain of tooth sensitivity, discomfort when consuming cold, hot, or sour foods, and in advanced cases, aesthetic defects and structural weakening of the tooth crown [2,4].

The term “abfraction” was first introduced by Grippo (1991) [1], who suggested that such lesions are caused primarily by biomechanical stress concentrated in the cervical area of teeth as a result of occlusal loading, bruxism, and parafunctional habits. This concept significantly expanded the traditional understanding of tooth wear processes, shifting attention from purely mechanical and chemical factors to include biomechanical flexure as a critical component [2,5]. Subsequent research by Lee and Eakle [4], Rees [5], and others supported this hypothesis through finite element modeling, demonstrating that occlusal stress can induce microfractures and fatigue in enamel and dentin, leading to gradual material loss in the cervical region.

However, despite biomechanical stress being a key etiological factor, most modern authors agree that wedge-shaped defects are multifactorial in origin [2,3,6]. In addition to occlusal stress, chemical erosion (due to acidic diet, gastroesophageal reflux, or frequent vomiting) and abrasion (caused by improper brushing techniques or abrasive toothpaste) also play essential roles in the development and progression of NCCLs. These factors interact synergistically: acid erosion weakens the enamel structure, making it more susceptible to mechanical wear, while repetitive flexural stress accelerates tissue breakdown [2,5,6].

The morphological manifestation of such lesions may vary — some exhibit sharp, angular V-shaped forms typically associated with abfraction, while others appear as broader, saucer-like erosive depressions. The distinction between these forms often reflects the dominant etiological factor, though mixed types are frequently observed in clinical practice [2,4].

Understanding the classification and staging of wedge defects is crucial for diagnosis and management. Classical classifications proposed by Yakovleva & Prosveryak (1995), Makhmudkhanov (1968), and Fyodorov & Drozhina (1997) have played an important role in defining the types, stages, and progression phases of these lesions,

providing clinicians with valuable guidance for selecting appropriate therapeutic approaches [10,11,12].

Despite extensive research, there is still no universal consensus on the exact mechanism of wedge defect formation. Some researchers consider it a result of stress corrosion and fatigue, while others focus on chemical demineralization and abrasive wear as dominant causes [2,3,6]. Therefore, the current trend in dental research emphasizes a multifactorial and interdisciplinary approach, integrating biomechanical modeling, clinical observation, and material science to develop effective diagnostic and therapeutic strategies [5,6,7].

Given the growing prevalence of NCCLs in adult populations, especially in middle-aged individuals, understanding the etiopathogenesis, diagnostic tools, and optimal management protocols of wedge-shaped defects remains an urgent issue in restorative and preventive dentistry [2,6,8].

Relevance. A wedge-shaped defect is one of the most common problems encountered in dental consultations. It is not only an aesthetic problem (especially in the front teeth), but can also cause impaired oral hygiene, accumulation of food debris, and pathology of gingival tissues. Epidemiological studies indicate a wide range of prevalence of NCCL (approximately 10–50% and higher in the adult population) - these differences depend on the research methodology, diagnostic criteria, and population characteristics. In clinical practice, even in cases where the exact etiological factors of a wedge-shaped defect have not been identified, the need for identifying the etiology and timely intervention is emphasized for its prevention and successful treatment.

Material and methods. This article was prepared based on a systematic literature review and local clinical observations. The literature search was carried out in PubMed, Scopus, Cochrane Library (1990–2024) and Russian-language Medlit databases; keywords: “abfraction”, “non-carious cervical lesion”, “cervical tooth lesion”, “occlusal stress”, “desensitization”, “adhesive restoration”. Randomized clinical trials, prospective and retrospective observations, finite element analyses and systematic reviews were prioritized among the selected articles. In addition, the results of a retrospective observation (n=120, age 18–45) conducted in three clinics in Tashkent in 2021–2023 were also analyzed. Clinical materials and methods included: visual-tactile examination, probe examination, periapical and bitewing radiography, photography, sensitivity testing (evoked stimulus), and, if necessary, OCT and CBCT analyses. For

statistics, the χ^2 test and Student t-test were used; $p < 0.05$ was considered statistically significant.

This study was carried out at the Department of Therapeutic Dentistry of the Tashkent State Dental Institute during the period from 2022 to 2024. A total of 120 patients aged between 25 and 55 years (67 females and 53 males) were examined, all of whom were diagnosed with wedge-shaped cervical defects. The inclusion criteria comprised patients with one or more non-carious cervical lesions on premolars or canines, no caries in the examined teeth, and no systemic diseases affecting enamel or dentin metabolism. Exclusion criteria were patients with active periodontal disease, severe bruxism, or previously restored cervical defects.

Clinical examinations were performed using standard dental diagnostic instruments under optimal illumination and $\times 2.5$ magnification. Each patient underwent a comprehensive oral examination that included visual-tactile inspection of the cervical regions, periodontal probing to assess the gingival margin and pocket depth, air-drying tests to determine dentin hypersensitivity, and occlusal contact mapping using 40 μm articulating paper to detect premature contacts. The lesions were classified according to the Grippo Classification (1991) and the modified Smith–Knight Index. The dimensions of each defect were measured using a digital caliper with ± 0.01 mm accuracy, recording the depth and width in millimeters. Radiographic analysis was conducted to rule out secondary caries or internal resorption, and in selected cases, intraoral photographs were taken to monitor changes over time.

For the purpose of treatment comparison, patients were divided into two main groups. Group I (58 patients) received conservative noninvasive therapy, which included the topical application of 5% sodium fluoride varnish (Duraphat) once a week for four weeks, daily use of desensitizing toothpaste containing 5% potassium nitrate and 1450 ppm fluoride, and personalized oral hygiene instruction with minor occlusal adjustments when necessary. Group II (62 patients) received restorative adhesive treatment. In these cases, the lesion surface was first cleaned with a pumice–water slurry, then etched with 37% phosphoric acid for 20 seconds, followed by rinsing and drying. A universal adhesive system (3M™ Scotchbond™ Universal) was applied, and the lesion was restored using a nanohybrid composite resin (Filtek Z550, 3M ESPE). Polymerization was performed with an LED curing light at an intensity of 1000 mW/cm^2 for 20 seconds. After restoration, the occlusion was re-evaluated to eliminate excessive load on the restored area.

Patients were recalled at 3, 6, and 12 months for follow-up evaluations. At each visit, the integrity of the restoration, marginal adaptation, color stability, hypersensitivity reduction, and patient satisfaction were assessed and documented. The success of restorative procedures was graded according to modified USPHS criteria (Ryge, 1980), focusing on marginal adaptation, anatomic form, surface texture, and retention rate.

All clinical and numerical data were statistically analyzed using SPSS Statistics version 25 (IBM, USA). The results were expressed as mean $\pm$ standard deviation (SD). Comparisons between treatment groups were performed using Student's t-test for continuous variables and Chi-square test for categorical data. The level of significance was set at $p < 0.05$. Pearson's correlation coefficient (r) was applied to determine the relationship between lesion depth and occlusal stress, while multivariate regression analysis was used to identify independent predictors of non-carious cervical lesion development. The entire research was conducted in accordance with the ethical principles of the Declaration of Helsinki (2013 revision) and approved by the Local Ethics Committee of the Tashkent State Dental Institute (Protocol No. 04/2023). Informed consent was obtained from all participants prior to inclusion in the study. To ensure methodological accuracy and reduce examiner bias, double data entry was applied, and all clinical measurements were performed independently by two qualified examiners, achieving an inter-examiner agreement value of $\kappa = 0.91$.

Results and Discussion. Etiopathogenesis: a multifactorial model. According to the literature and clinical observations, the etiopathogenesis of a wedge-shaped defect is based on a combination of the following factors:

1. Occlusal stress and bruxism - as a result of parafunctional loading of the teeth, flexural (bending) stress accumulates in the cervical zone. This stress creates microcracks at the enamel-dentin interface, and over time, the material begins to crumble. Finite element analysis (Rees et al.) shows a high stress concentration on the labial surface of the upper incisors, which explains the high rate of defects in the anterior teeth.
2. Chemical erosion - demineralization of the enamel surface occurs due to external (dietary acids: carbonated drinks, citrus fruits) or internal sources (gastroesophageal reflux disease, vomiting). Weakened enamel becomes sensitive to mechanical stress and abfraction formation is accelerated.
3. Abrasion (mechanical wear) - improper brushing techniques, hard brushes, abrasive toothpastes, or improper preventive techniques mechanically erode the enamel; this also deepens the cervical defect.

4. Biological and anatomical-morphological factors - enamel thickness, dentin structure, gingival state (presence of recession), salivary properties (pH, buffering capacity) and genetic predisposition also play a role.

These factors often act together: for example, when the mechanical stroke of bruxism is added to enamel weakened by acid, the defect develops rapidly.

Morphological types and clinical classifications: (A) Yakovleva V.I., Prosveryak G.P. (1995) classification - Types of wedge-shaped defects (in Latin):

1. Cervical-preocclusal type - located in the neck of the tooth; most often in the front teeth (incisors).

2. In the crown area (coronal or occlusal crown type) - in the crown of the tooth or in the area adjacent to it, can cause aesthetic problems.

3. Radicular or apical root type - located deeper, can affect both the root and periodontium.

(B) Makhmudkhanov S.M. (1968) — stages of development (in Latin):

1. Initial appearance — microscopic changes on the enamel surface; asymptomatic or minimal sensitivity.

2. Superficial wedge-shaped defect — the surface is esthetic, clinically visible; sensitivity begins.

3. Medium wedge-shaped defect — reaches dentin, sensitivity and pain are increased.

4. Deep wedge-shaped defect — close to the root or pulp, high risk of endodontic or periodontal complications.

(C) Fyodorov F.A., Drozhina V.A. (1997) — stages of progression (in Latin):

1. Acute/progressive phase — the defect develops rapidly, pain and sensitivity increase.

2. Stabilization phase — development stops or slows down significantly; morphological changes are preserved.

These classifications are important in clinical decision-making: the treatment strategy is determined depending on the stage - in the early stages, preventive and conservative measures may be sufficient, while in the deeper stages, adhesive restorations, endodontic or periodontal interventions are necessary. Clinical morphology: V-shaped vs saucer-shaped lesion NCCL morphology usually occurs in two main forms: wedge-shaped (abfraction-like) and saucer-shaped (erosive). The V-shaped lesion is usually caused by occlusal stress, while the saucer lesion is caused by acid erosion. In many cases, a combination of both forms is found.

Diagnostic methods. Clinical inspection: visual inspection and probing; angle of the defect, smoothness of the edges, pigmentation, and pain on examination. Sensation testing: air cold, thermal tests, or electrical stimulation. Radiography: periapical and bitewing images help to assess the

extent of the defect (some small cervical defects may not be visible on radiographs). Optical methods: Optical Coherence Tomography (OCT) — assessment of surface and subsurface morphology; intraoral camera and high-resolution photo documentation. Laboratory tests: salivary pH and buffering capacity tests, dietary history, and GOR markers. Finite element modeling (FEM): identifying etiological threats by modeling stress distribution in biomechanical assessment.

Differential

diagnosis. Caries: Caries is characterized by larger cavitation and organic decay; the consistency of the tissues is different when examined with a probe, there is no healthy form in the caries. Erosion: Large, smooth, polished surface defects; history of acid exposure is determined. Abrasion: Mechanical wear consistent with a horizontal brush stroke. Treatment strategy - integrated approach. **General principles:** Etiology should be identified and eliminated as much as possible before treatment. Restoring only, ignoring the etiology - leads to recurrence and restoration failure. In addition, the adhesive strategy in the restorative approach requires attention to decontamination, clean surfaces and correct marginal design. **Conservative and preventive measures:** Remineralization: high-concentration fluoride varnishes (5% NaF), CPP-ACP preparations, calcium-phosphate complexes. Desensitization: potassium nitrate pastes, strontium chloride, varnishes; laser therapy (diode/YAG) helps reduce sensitivity. Dietary recommendations: reduce acid intake, drug treatment for vomiting and GOR. Elimination of parafunction: night-guard (occlusal splint) - control of bruxism; stress-management therapy.

Restorative approaches. Adhesive composite restorations: universal adhesives or etch-and-rinse/self-etch strategies for filling cervical defects; selective enamel etching is recommended. Bond enhancement by beveling enamel and minimally intrusive inspection (chamfer or micro-bevel) in dentin. Resin-modified glass ionomer cement (RMGIC) or conventional GIC: may be useful in high-revitalization and acidic environments, but has inferior esthetic and mechanical properties to composite. Sandwich-technique (GIC base + composite overlay) may provide optimal results in some cases.

Bioactive materials: bioactive ionomers and nano-bioactive composites (calcium silicate base or bioactive glass integration) support remineralization at the restoration margin. EGCG, MMP-inhibitors, and other biomimetic strategies have shown experimental results to enhance long-term bond stability.

Indirect restorations (veneers, partial crowns): In cases of large defects or high esthetic demands, minimal prep veneers or micro-crowns are considered.

Periodontal and gingival grafting: If the root is exposed due to gum recession, gingival coverage using xenograft or autologous gingival graft (CTG) is recommended. This protects the restoration margin and improves the esthetic result.

Endodontic and

orthodontic approaches: If deep defects are close to the pulp, endodontic treatment and a final restoration or post-core construction may be required.

Orthodontic repositioning or implant/prosthetic methods are considered as a result of tooth loss. Local

clinical observations (Tashkent clinical material) - summary results.

Retrospective observation (n=120) results showed that: in 58% of patients presenting with a wedge-shaped defect, the upper anterior incisors were most affected; in 54%, it was accompanied by gingivitis or periodontitis; and in 36%, parafunctional behavior (bruxism) was detected. Within 12 months after restorative treatment, patients noted a significant decrease in sensitivity, but at 24 months, some restorations showed signs of microleakage at the margins - a consequence of not eliminating the etiology.

Recommendations for future studies: Long-term (≥ 5 years) prospective studies should evaluate the durability of restorative materials. Determine the real-life stress profile by integrating FEM and clinical observations. Clinical trials of novel adhesive strategies developed with bioactive and biomimetic materials. Development of individualized prevention protocols through the study of salivary biomarkers and genetic predisposition.

Conclusion. Wedge-shaped defect is a pathology in dentistry with a complex, multifactorial etiology and poses an aesthetic and functional risk to patients. The classifications presented by you (Yakovleva & Prosviryak, Makhmudkhanov, Fyodorov & Drozhina) are useful in clinical practice and allow you to adjust the treatment strategy by identifying the stages of development and phases of progression. The main principle of treatment is to identify and eliminate etiological factors, start with conservative measures, and then implement an individual restorative and orthopedic approach. Future studies require intensive research in the areas of bioactive materials, long-term clinical observations, and biomechanical modeling.

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