Received: 22 May 2022 | Accepted: 25 July 2022

DOI: 10.1111/jopr.13585

AMERICAN COLLEGE OF PROSTHODONTISTS
Your smile. Our specialty.®

REVIEW

Noncarious cervical lesions: Morphology and progression, prevalence, etiology, pathophysiology, and clinical guidelines for restoration

Charles J. Goodacre DDS, MSD, FACP, FFDRCS Irel (Hon)[1] | **W. Eugene Roberts DDS, PhD, DHC (Med)**[2] | **Carlos A. Munoz DDS, MSD**[3]

[1]Advanced Education Program in Implant Dentistry, Loma Linda University School of Dentistry, Loma Linda, California

[2]Adjunct Professor Mechanical Engineering, Indiana University & Purdue University, Indianapolis, Indiana

[3]Kerr Corporation, Orange, California

Correspondence
Charles J. Goodacre, DDS, MSD, FACP, FFDRCS Irel (Hon), Loma Linda University School of Dentistry, Loma Linda, California.
Email: cgoodacre@llu.edu

Abstract

Purpose: To synthesize the literature regarding noncarious cervical lesions (NCCLs) and propose clinical guidelines when lesion restoration is indicated.

Material and methods: A PubMed search was performed related to NCCL morphology, progression, prevalence, etiology, pathophysiology, and restoration.

Results: NCCLs form as either rounded (saucerlike) depressions with smooth, featureless surfaces that progress mainly in height or as V-shaped indentations that increase in both height and depth. Prevalence ranges from less than 10% to over 90% and increases with age. Common locations are the facial surfaces of maxillary premolars. They have a multifactorial etiology due to personal habits such as excessive horizontal toothbrushing and consumption of acidic foods and drinks. Occlusal factors have been identified as contributing to the prevalence of NCCLs in some studies, whereas other studies indicate there is no relationship. The concept of abfraction has been proposed whereby mechanical stress from occlusal loading plays a role in the development and progression of NCCLs with publications supporting the concept and others indicating it lacks the required clinical documentation. Regardless of the development mechanism, demineralization occurs and they are one of the most common demineralization diseases in the body. Treatment should be managed conservatively through preventive intervention with restorative treatment delayed until it becomes necessary due to factors such as lesion progression, impact on patient's quality of life, sensitivity, poor esthetics, and food collection may necessitate restoration. Composite resins are commonly used to restore NCCLs although other materials such as glass ionomer and resin-modified glass ionomer are also used. Sclerotic dentin does not etch like normal dentin and therefore it has been recommended to texture the dentin surface with a fine rotary diamond instrument to improve restoration retention. Some clinicians use mechanical retention to increase retention. Beveling of enamel is used to increase the bonding area and retention as well as enhance the esthetic result by gradually creating a color change between the restoration and tooth. Both multistep and single-step adhesives have been used. Dentin etching should be increased to 30 seconds due to the sclerotic dentin with the adhesive agent applied using a light scrubbing motion for 20 seconds but without excessive force that induces substantial bending of a disposable applicator. Both flowable and sculptable composite resins have been successfully used with some clinicians applying and polymerizing a layer of flowable composite resin and then adding an external layer of sculptable composite resin to provide enhanced resistance to wear. When caries is

present, silver diamine fluoride has been used to arrest the caries rather than restore the lesion.

Conclusions: Noncarious cervical lesions (NCCLs) form as smooth saucerlike depressions or as V-shaped notches. Prevalence values as high as 90% and as low as 10% have been reported due to habits such as excessive toothbrushing and an acidic diet. Occlusal factors have been proposed as contributing to their presence but it remains controversial. Publications have both supported and challenged the concept of abfraction. They are one of the most common demineralization diseases in the body. Conservative treatment through prevention is recommended with restorative treatment delayed as long as possible. When treatment is needed, composite resins are commonly used with proposed restorative guidelines including texturing the sclerotic dentin, beveling the enamel, potential use of mechanical retention, 30 seconds of acid etching, and use of either multistep or single-step adhesives in conjunction with a light scrubbing motion for 20 seconds without excessive force placed on disposable applicators.

KEYWORDS
etiology, NCCL, noncarious cervical lesions, prevalence, treatment

Most teeth show signs of wear by the time individuals reach their middle age.[1] This wear has been described as "the cumulative loss of mineralized tooth substance due to physical or chemophysical processes" resulting from attrition, erosion, and abrasion.[2] The term "tooth wear" has been used to encompass all three of these causes.[1]

Enamel on occlusal surfaces may be worn away by a gritty diet, functional occlusion, or parafunction.[3] Such wear is termed attrition and occurs as a result of tooth-to-tooth contact whereas abrasion is loss of tooth structure from factors other than tooth contact.[4] One source of abrasion is excessive and vigorous horizontal tooth brushing.[5–8] Erosive tooth wear is "tooth wear with dental erosion as the primary etiological factor."[2] Erosion has been described as "probably the most common reason for tooth wear" and involves a "combination of acid-mediated and mechanical wear."[1] A review of the global prevalence of erosive tooth wear reported a mean prevalence in primary teeth between 30% and 50% and between 20% and 45% in the secondary dentition.[9] These destructive erosive processes can occur on all the coronal surfaces of a tooth as well as the root surface when it becomes exposed to the oral environment. Cervical lesions on the root surface can occur without the presence of caries and are known as noncarious cervical lesions (NCCLs).

NCCL MORPHOLOGY AND PROGRESSION

NCCLs manifest themselves as a loss of mineralized tissue along the tooth surface near the gingival margin and typically extend from the cementoenamel junction onto the root surface.[10] The lesions may form as a smooth surface, rounded (saucerlike) depression into the cervical area of a tooth, or a V-shaped indentation (Fig 1). The depth may be limited or be more pronounced (Fig 2). Sharp, V-shaped indentations can penetrate deeply into the tooth (Fig 3). There can be sensitivity,[11,12] no sensitivity, or limited sensitivity.[13] There is even the potential for loss of pulp vitality. CT scans of extracted teeth reveal the prominence of the pulp on the facial surface of many teeth in the cervical area (Fig 4) with some teeth having unique pulpal projections toward the external surface (Fig 5). Thus, there can be minimal tooth structure protecting the pulp at the facial cervical area of deep lesions even with the continued formation of secondary dentin. Overall, NCCLs are common clinical conditions that negatively impact the structural integrity and esthetics of the dentition.[14] These noncarious cervical lesions have been classified based on their shape as shallow, concave, wedge-shaped, notched, and irregular.[15]

When examined microscopically, saucer-shaped lesions were found to have a smooth, featureless surface except for craters and dimples whereas wedge-shaped lesions exhibited scratches and furrows in their surfaces that the authors attributed to toothbrush abrasion.[16] In one study of 24 extracted teeth, 54.2% had horizontal furrows varying in width from 5 to 250 μm.[10] Most saucer-shaped lesions had rounded borders whereas most wedge-shaped lesions had sharp edges.[17] In 10 mandibular anterior teeth examined microscopically, the enamel incisal to the lesions was knife-edged.[18]

In a study of lesion progression, 83 lesions from 16 participants were examined over 3 to 5 years with saucer-shaped lesions progressing mainly in height, whereas wedge-shape lesions increased in both height and depth with the annual lesion progression increasing significantly as the depth to height ratio (D/H) increased. Over half of the NCCLs with a small D/H ratio progressed 50 micrometers or more in height per year whereas none progressed more than 50 micrometers in depth. The mean volume loss was 0.36 mm^3 annually.[19] In another study of progression in 29 participants over 5 years, the volume loss was 1.50 $\pm$ 0.92 mm^3/yr.[20] Wedge-shaped lesions tended to have a greater risk of depth progression than saucer-shaped lesions.[21] It

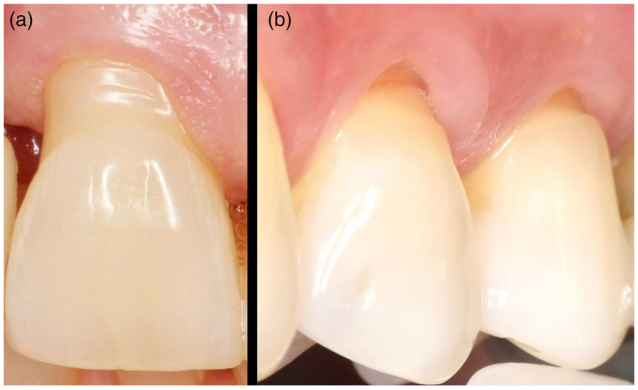

FIGURE 1 (a), The maxillary central incisor has a rounded (saucer-like) depression in the root. (b), Both the maxillary premolars have V-shaped indentations in the roots.

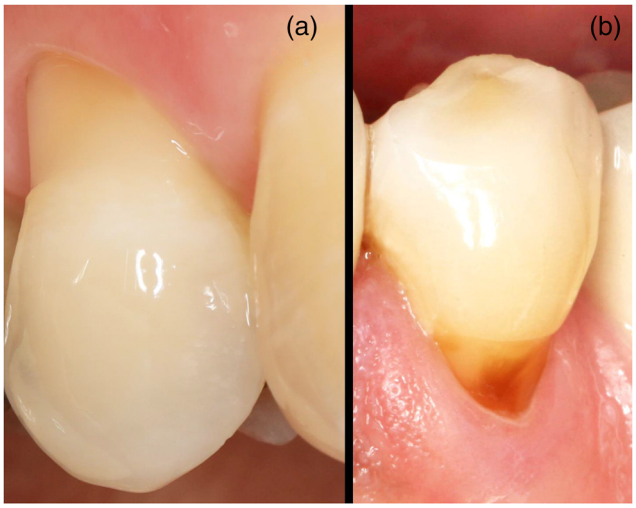

FIGURE 2 (a), The maxillary first premolar has a rounded depression of limited depth. (b), The mandibular first premolar has a substantial non-carious cervical lesion.

has been determined that individuals with a low buffering capacity of saliva were in a high-risk group for progression of lesions as were older study participants and those who consumed more dietary acids.[22]

PREVALENCE AND COMMON LOCATIONS

Prevalence percentages have varied substantially from lower percentages such as 13.1,[12] 16.6,[23] and 17.1[24] to higher percentages of 61.7%,[6] 62.5%,[25] and 63%.[8] A range between 9.1 and 93% was reported in one study.[26] The percentage of individuals who had one or more teeth with typical V-shaped NCCLs has been reported as 49.1%[27] and those having at least one lesion was 62.2%.[12] The percentage of teeth affected by NCCLs has been reported as 5.7% (259 of 4518 teeth),[27] 9.65% (280 of 2902 teeth),[11] 15% (3222 of 21,483 teeth),[6] and 17.2% (355 of 2.060 teeth).[28] In 391 randomly selected study participants, at least one severe lesion was

FIGURE 3 The maxillary first molar has a very deep V-shaped notch in the tooth.

observed in 29.9% of younger individuals (ages 26-30 years) and 42.6% of the older sample (ages 46-50 years).[29]

The prevalence increases with age,[9,30,31] but NCCLs are present in all age categories including younger individuals. As an example, an examination of 40 first-year dental students determined that 29 had at least one affected tooth. Furthermore, 129 of 1131 teeth in the sample were affected by NCCLs.[32] After 3 years, there was an increase of 57 additional lesions in the same students.[32]

NCCLs are commonly found on the facial surfaces[4] but slight lingual erosion has been found in 3.6% of a younger age group and 6.1% of older individuals with severe lingual erosion being rare.[29] Multiple studies have identified maxillary premolars as the most commonly affected teeth[6,8,27,33,34] whereas another study determined mandibular first premolars were most commonly affected (Fig 2B) followed by mandibular second premolars and then canines.[23] It also was reported that first premolars in all the quadrants were most frequently involved with NCCLs.[33] The next most common site after premolars were determined to be maxillary molars in two studies.[6,8] While these studies indicate the lesions are most commonly found on premolars and molars, they can also be found on canines and anterior teeth in both arches.[8,13,35] The teeth affected by the most severe lesions were reported as being the first premolars.[35] The least common site was the lingual surface of mandibular molars.[36]

ETIOLOGY

Numerous causes for NCCLs have been proposed in the literature but it is generally recognized they have a

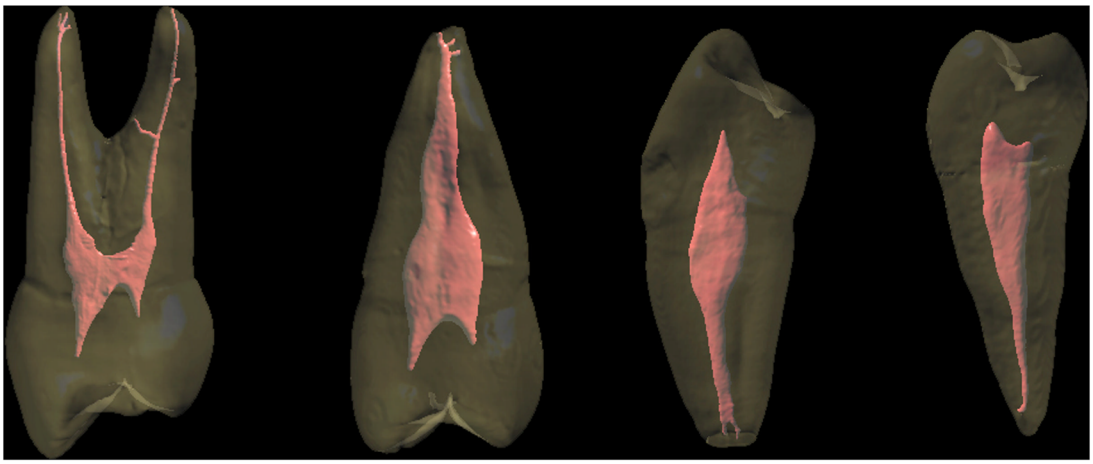

FIGURE 4 The 4 CT scan images of extracted teeth show the facial prominence of the pulp in the following teeth from left to right: maxillary first premolar, maxillary second premolar, mandibular first premolar, and mandibular second premolar.

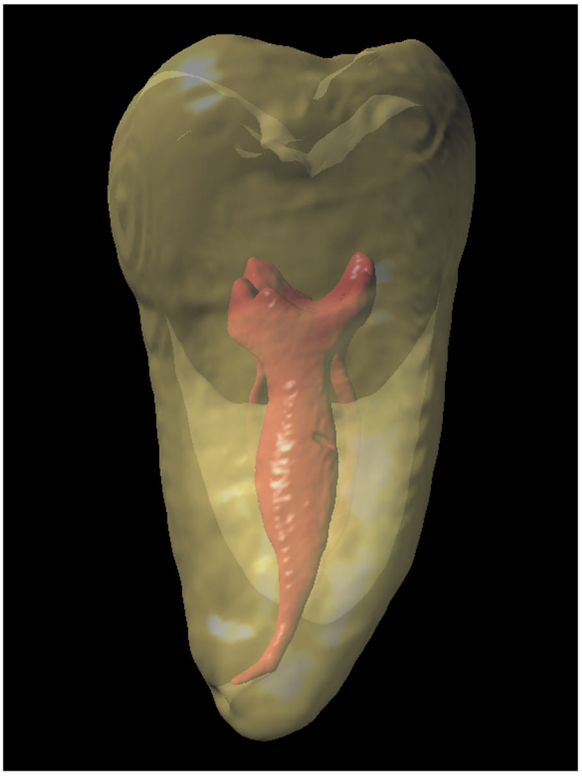

FIGURE 5 This CT scan of a mandibular second premolar shows a substantial extension of the pulp toward the facial surface.

multifactorial etiology.[37] Contributing factors include excessive horizontal toothbrushing,[4] consumption of acidic foods and drinks,[1,9,25,38] gastroesophageal reflux disease,[9,38,39] being asthmatic,[40] and medications that inhibit salivary flow.[38] There is a preference for acidic beverages in the western diet because they are refreshing, particularly after physical exertion and they "clear the palate" for better appreciation of food. Beverages with high acid content such as carbonated soda, citrus-based drinks, and wine demineralize enamel surfaces. Natural fruit tannins (tannic acid) are prevalent in higher quality wine which may have a pH of 3.5 or less. The European culture of wine and cheese consumed together is a wise social strategy since the cheese buffers the acidity of the wine, tends to adhere to enamel surfaces, and supplies both calcium and phosphate ions for remineralization.[3]

The linkage between erosive tooth wear and an acidic diet was examined in a randomized clinical trial, determining that participants who reduced their dietary intake between meals (snacks) had substantially less volume loss of tooth structure.[41] Other acidic items have been identified and they include acidic lozenges, tablets, and mouth rinses that may potentiate demineralization.[42,43] Most foods, including those with phytoliths (minute mineral particles) in plants, are known to be abrasive.[3] Superimposing an acidic environment with mechanical factors such as toothbrush abrasion and a coarse diet increases the prevalence of cervical lesions. However, despite knowing the effect of dietary and oral hygiene habits, one study found that even after specific counseling sessions, no behavioral changes were observed regarding acid ingestion and toothbrushing. The authors concluded, "dietary and oral hygiene habits are not easily changed, not even with better knowledge after detailed instruction."[22]

In addition to abrasion and erosion, occlusal factors have been identified as potentially contributing to the prevalence of NCCLs. The factors have included occlusal wear facets,[8,28,30,32,44,45] heavy occlusal forces,[20] increased occlusal contact area,[27] premature contacts,[46,47] extended occlusion and disocclusion time,[48] group function occlusion,[49] lateral excursive contact of teeth and bruxism,[31] irregular lateral excursions,[50] and lack of canine disclusion.[45] Using clinical data and genotypical analyses, a significant association was identified between attrition, malocclusion, and NCCLs.[51] However, other NCCL etiology studies indicate occlusal wear is not related to their formation,[7] occlusal factors alone are not sufficient to explain their presence,[52] and there is no association with wear facets.[33]

Furthermore, a systematic review of clinical studies concluded that "the role of occlusion in the pathogenesis of noncarious cervical lesions seems as yet undetermined."[53] The above publications present differing views on the effect of occlusal factors and that leads to the controversial topic of abfraction.

Abfraction

Abfraction is considered as a potential etiology for NCCLs.[38] The term "abfraction" was introduced to explain cervical wear resulting from mechanical stress related to loading.[54] Abfraction is thought to result from tensile stress caused by mastication and malocclusion.[55] It has been proposed that high occlusal loads[56] and tooth bending (flexure) has a fundamental role in the development of NCCLs due to stress parallel or oblique to the occlusal load.[57] Multiple studies using finite element modeling analysis (FEA) have defined the potential relationship between occlusal stress and the development of NCCLs.[58–61] The clinical scenario for the progression of a cervical lesion was modeled and analyzed using finite element analysis (FEA), indicating lesions can occur when heavy function and parafunction overload cervical areas in tension and compression that exceed the material strength (fatigue resistance) of mineralized dental structures. It was concluded that overloading of teeth may initiate a cervical lesion.[62] In another study, 3D FEA of a mandibular second premolar revealed an asymmetric pattern of strains in the cervical enamel of the facial surface in response to oblique occlusal loads that were consistent with the clinical picture for asymmetric NCCLs.[63] The same authors used 3D models to conclude that stress was concentrated at the cementoenamel junction was consistent with the common clinical manifestations of NCCLs.[64] In addition, a systematic review concluded there is an association between occlusal stress and NCCLs but the authors also stated there are no clinical studies demonstrating that NCCLs were caused by mechanical stress alone.[65]

This lack of clinical studies showing a direct relationship between tooth stress and the formation of NCCLs has resulted in questions regarding the concept of abfraction. It has been stated that abfraction lacks the scientific documentation of more established causes,[1] is not consistent with appropriate clinical evidence,[66] and fails to establish the validity of abfraction as an etiologic entity.[4] Also, the theory of abfraction may be flawed.[67] "In addition, applying a load to the buccal cusps of extracted premolars failed to document the progression of cervical tooth wear commonly referred to as abfraction."[68] Another assessment of 299 casts from dental students suggested there was no relationship between NCCLs and occlusal/incisal wear.[69] As a result of the above concerns, the term 'abfraction lesion' has been judged to be misleading.[14] Also, a consensus report has discouraged the use of the term abfraction since the level of evidence was judged to be too weak to justify it as an independent etiologic process.[2]

The lack of clinical studies relating NCCLs to occlusal forces and cervical stresses is due in part to no "smoking gun" being evident such as cracks or particulate material near the surface of a cervical lesion being observed clinically or with histology. Should such small mineral particles be avulsed as a result of surface fatigue failure, they would be readily washed away by saliva and would not be available for examination. Also, any fragments of the collagenous matrix due to dentin and cementum failure are rapidly destroyed by enzymes in saliva and the gastrointestinal tract. In effect, abfraction is difficult to study clinically because lesion progression is slow, and the evidence is destroyed rapidly. Therefore, clinical confirmation of the actual abfraction process clinically is not tenable, so modeling of the phenomenon with FEA has been the most realistic approach,[56,70,71] along with the examination of tooth flexure and associated stress during loading.

Tooth flexure (deformation) of a mandibular first premolar using FEA revealed that occlusal loads of 200 N are 10-fold greater for nonaxial loading compared to axial loads.[72] Also, the deepest aspect of a wedge lesion is the stress riser so it is the most susceptible to the progressive fatigue failure that advances the lesion[73] when the tooth is flexed.[3] These data suggest the mineralized tissue at the base of a NCCL is failing under surface compression.[3] It is also thought that delamination of enamel from dentin at the dentin-enamel junction (DEJ) occurs in the cervical area as a result of undermining the enamel and may occur from a repeated oblique load of 100 N which is well within the range of normal function.[70] In a finite element study of simulated molars, overloading resulted in enamel damage at the CEJ and led to the initiation of a cervical lesion with subsequent overloading resulting in enamel destruction along the DEJ.[62] Thus, the thin enamel in the cervical region may be susceptible to fatigue failure when exposed to tensile, compressive, and shear loads that exceed its material strength.[71]

As mentioned previously, clinical studies to either prove or disprove the concept of abfraction are not possible to perform due to the slow progress of the lesions and the rapid loss of surface material. However, regardless of whether the concept of abfraction can be proved or disproved, there is demineralization of tooth structure occurring with NCCLs.

PATHOPHYSIOLOGY OF DEMINERALIZATION

Demineralization is the most prevalent chronic disease in the world, resulting in osteoporosis (OP) with a worldwide prevalence >10%,[74] NCCLs with a higher prevalence than osteoporosis,[6,8,25] and dental caries being one of the most prevalent chronic diseases worldwide.[75] In fact, the prevalence of untreated dental caries in children aged 5 to 19 years has been reported as 13.2% and in adults aged 20 to 44 years, it is 25.9%.[76] The above data indicates NCCLs are the second most common manifestation of demineralization disease in the body.

Environmental acidity, mechanical loading, and percolation of endemic fluoride regulate resistance to demineralization such as osteopenia, NCCLs, and dental caries.[3,77] In general, the etiology of demineralization involves fluid percolation, metabolism, homeostasis, biomechanics, mechanical wear (attrition or abrasion), and biofilm-related infections.[3,77] An NCCL has a multifaceted etiology that draws on the same mechanisms for demineralization, but in a unique pattern that is specific for each tooth. Patients may have multiple NCCLs but each one is not quite the same manifestation as the others.[78,79] Only one tooth may be affected and bilateral symmetry is rare.

Demineralization is a broad-spectrum disease that has both an infectious and noninfectious etiology.[3,77] Cariogenic bacteria do not contribute to NCCLs, but other oral microorganisms such as *Fusobacteriales* may protect a cervical lesion from acidic degradation via bacterial interaction (interference) and pH regulation.[78] Equilibrium of demineralization and remineralization in the presence of optimal fluoride decreases caries prevalence,[3,77] but the effect on NCCLs is unknown. Fluoride is an essential trace element with multiple health benefits, but fluorosis disrupts cell physiology via modifications of apoptosis, stress, and signaling.[80] Optimal physiologic levels of fluoride ion (F^-) during mineralization and remineralization result in the formation of fluorapatite (FA). Compared to hydroxyapatite (HA), FA has a more compact (dense) crystalline structure rendering it less susceptible to acidic attack[3,77] and mechanical degradation.[81] However, the relationship of FA to NCCL prevention, prevalence, and progression is unknown.[79]

ORTHODONTIC TREATMENT AND NCCLS: IS THERE A RELATIONSHIP?

NCCLs are a potential concern for any elective dental treatment because little is known about the progression of the lesions during clinical interventions. Despite transient occlusal trauma inherent in tooth movement, orthodontic treatment has no known effect on NCCL incidence or progression. However, the plaque load associated particularly with fixed appliance treatment is a concern since deep cervical lesions inoculated with cariogenic pathogens in plaque may result in a rapid progression of caries that could result in serious bone infections.[77] In a seeming paradox, "orthodontics treatment" was found to be a significant contributing factor for NCCLs in male athletes (footballers).[82] However, examination of the data revealed that most of the study participants did not receive orthodontic treatment which suggests that the presence of uncorrected malocclusion may play a role in the etiology. Since elite athletes frequently clench their teeth during demanding training sessions and games, it was interesting to note that footballers who trained up to one hour daily had a significantly higher prevalence of NCCLs than those who trained for more than an hour. The authors of the study proposed the reason was related to poorer physical fitness in those who trained only up to one hour and therefore they were not as capable of withstanding the intensity of the loads applied to the teeth. Similar findings were reported in a study of dental erosion in amateur runners where those who trained more than one hour had lower erosive lesions.[83]

Mouthguards are designed to help control occlusal forces from clenching,[84] but their effectiveness is limited by compliance and the type of occlusal challenge. Attrition due to bruxism is effectively controlled by the mouthguard material, but sustained clenching may still deliver a traumatic load to individual teeth. Maintaining a static load across a mouthguard compresses the compliant material and transfers the entire load to the resisting dentition. A powerful static load is spread to some degree throughout the arch but is concentrated on teeth that are out of alignment (malocclusion). There is no evidence that NCCLs are associated with the transient occlusal trauma of routine tooth movement, but some malocclusions and compromised outcomes like excessive lingual inclination of maxillary premolars may be a predisposition to NCCLs based on finite element analysis.[85] However, the authors indicated that excessive stress concentration at the buccal neck may be the cause, indicating malocclusion rather than orthodontic treatment is associated with the incidence and progression of NCCLs.

PARAFUNCTION

Parafunction in dentistry is commonly designated as bruxism, clenching, and nail biting. Bruxism is dynamic motion in intermaxillary occlusion that manifests as dental attrition.[3] Clenching and nail biting are defined as static and sustained loads generated by powerful contraction of the mandibular elevator muscles. Clenching, nail biting, and a diagnosis of TMD were significantly associated with the presence of NCCLs and these habits along with the presence of TMD should be considered in the diagnosis and treatment of NCCLs.[34] Thus, there may be a relationship between the proposed mechanical etiology of NCCLs and TMJ degeneration.[86,87] Sustained loads of clenching and nail biting are more damaging to mineralized tissues than the dynamic (shifting) loads of bruxism. Nail biting is typically a habit that requires conscious control. Similar to thumb sucking, the wearing of cloth gloves during the day and/or night can serve as a helpful reminder to curtail the destructive habit. In comparison, occlusal devices known as disocclusion orthotics are useful for controlling bruxism and clenching.[3]

NEUROLOGIC ORTHOTIC

Protecting oral structures from fatigue-related demineralization is an important clinical priority.[3,77] Teeth, jaws, and TMJ are damaged by both cyclic and sustained loads that result in flexure that exceeds the fatigue resistance of mineralized tissues.[88] The dynamic intermaxillary motion of bruxism is manifest as dental attrition, but sustained clenching is

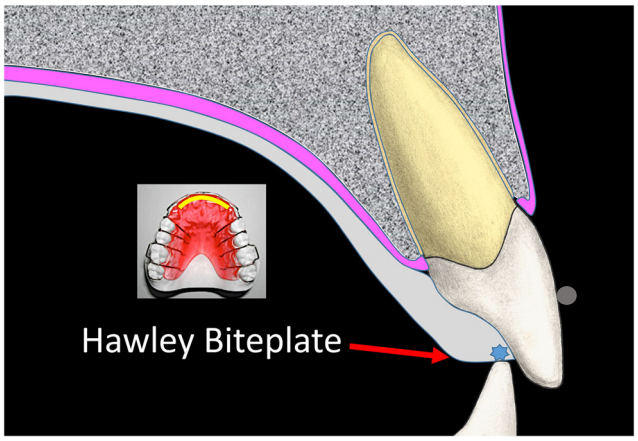

FIGURE 6 Diagram showing a cross-sectional view of a Hawley Biteplate with the mandibular incisor contact and the resin plate located on the lingual surface of the maxillary incisor. The insert shows an occlusal view of the device on a cast.

more commonly associated with TMJ degeneration.[87] Habitual clenchers are difficult to diagnose because there are no wear facets on the teeth. However, there may be a history of tired jaws or muscle hypertrophy.

Parafunctional loads may contribute to tooth fracture and root resorption[89] as well as induce tooth flexure with its proposed relationship to NCCLs,[34,77,82,90] Parafunction duration and frequency are difficult to control in a dental setting since the involuntary contractions that contribute to parafunction are of central nervous system origin. The secondary author of this paper has determined from multiple decades of clinical practice that the magnitude of parafunctional contractions can be reduced with a Hawley biteplate that prevents contact of the posterior occlusion. The power of the mandibular elevator muscles is reduced by a polysynaptic reflex that inhibits the strength of contraction when the posterior teeth are not in occlusion.[87,91] Full coverage occlusal devices transmit parafunctional loads to the jaws and teeth, but a Hawley biteplate (Fig 6) can decrease the magnitude of the load by reducing the strength of muscle contraction. It is proposed that this device can help control parafunctional loading but it should not be worn 24 hours a day to prevent extrusion of the molars that would negate the neurologic effect of the orthotic nor at mealtime to avoid overloading the anterior teeth when attempting to chew with the posterior teeth.

TREATMENT OF NCCLS

Much has been written about the management of NCCLs and the purpose of this section is to provide a synopsis of factors related to their treatment that are important to understand.

When to monitor and when to restore?

Since NCCLs are typically noncarious, they present a perplexing condition for practitioners as to whether they should

FIGURE 7 The maxillary first molar has a particularly deep V-shaped notch that extends well into the secondary dentin of the tooth that has extended into the area where the pulp was located. Such lesions should be restored as they are compromising the strength of the tooth. Also, plaque has been accumulating in the depth of the notch and restoration should be initiated before caries occurs.

be restored and at what degree of wear should a restorative intervention be considered. Due to the lack of clinical trials, systematic reviews, or meta-analyses regarding when and how the treatment should be initiated,[1] one has to depend on published recommendations such as "restorative intervention is typically best delayed as long as possible"[92] and "preventive intervention may be preferable to extensive restorative care and high maintenance needs."[93] It has also been stated that "the decision to monitor NCCLs rather than intervene should be based on the progression of the lesions and how they compromise tooth vitality, function, and esthetics."[94] Recommendations regarding when a restoration should be placed have included a negative impact the patient's quality of life[1] and when there is sensitivity, poor esthetics, and food stagnation.[95]

There are some patients with very deep NCCLs that require restorations for esthetic reasons as well as preserving structural integrity and preventing tooth fracture (Fig 7). A small number may be sensitive, a condition also making restoration a reasonable treatment. There also are patients who present challenging treatment decisions due to extensive erosion-abrasion that encompasses the roots of multiple teeth in such a way that little root structure remains (Fig 8). Should one tooth fracture (Fig 9), a decision needs to be made as to whether to restore the tooth, extract the tooth and replace it with an implant, or extract adjacent teeth with substantial lesions, and replace multiple teeth via implants.

It is interesting to review the results of a survey of attendees at a meeting of the Greater New York Academy of

FIGURE 8 This patient has substantial non-carious cervical lesions on the facial and proximal surfaces of the mandibular anterior teeth, placing them at severe risk of fracture. Lesions are located on the facial surfaces of all the other mandibular teeth.

FIGURE 9 The mandibular left central incisor has fractured due to weakening from the cervical lesion. There is now a challenging decision as to what treatment is best for this patient. Should the root be retained through endodontic treatment and a post and core, with the associated risks due to lack of tooth structure or should the tooth be extracted and an implant placed? Should the adjacent teeth be restored to prevent further deepening of the lesions and weakening of the teeth or are they sufficiently weakened that they should be extracted and implants placed to support a fixed partial denture?

Prosthodontics regarding their approach to management and restoration of NCCLs. Digital images were projected with 1.0, 2.0, and 3.0 mm deep lesions. As the restoration depth increased more clinicians indicated they would restore the area. Also, when sensitivity was present, clinicians were more likely to restore the tooth. It was interesting to note that clinicians who do not use mechanical retention had a lower estimate of their restoration longevity.[96]

The effect of sclerotic dentin

NCCLs typically have sclerotic dentin walls that consist of hypermineralized intertubular dentin.[97,98] In other words, the tubules are occluded with crystalline deposits.[99,100] There is a lack of intact, banded collagen[101] resulting in the sporadic absence of the hybrid layer[98,102] required for optimal bonding, and no resin tags or only short tags are present.[97,101] Therefore, the bond strength of composite resins is significantly lower to sclerotic dentin than to normal dentin.[102,103] Due to the tubule occlusion with minerals, the dentin etches more slowly[99,102.104] and longer etching times are needed than when bonding to normal dentin.[98,105]

Should occlusal adjustment be performed?

Since occlusal factors have been identified as potentially contributing to the prevalence of NCCLs,[8,20,28,30–32,44–50] occlusal adjustment would be a possible treatment option for NCCLs. However, a study examined the effect of occlusal adjustment on the rate of cervical tooth wear and determined occlusal adjustment does not appear to halt the progression of NCCLs and therefore cannot be recommended.[106] In addition, a systematic review indicated evidence does not support an intervention that alters occlusal factors for the purpose of preventing or controlling the progression of NCCLs.[107] A conservative option to reduce the force applied to teeth and therefore reduce wear on the teeth is the use of a Hawley biteplate, as described previously.

RESTORATION OF NCCLS

There has been a plethora of publications related to the restoration of noncarious cervical lesions and they have included studies of various procedural steps as well as materials used.

Material selection

Regarding the material to be used, it has been stated that composite resin restorations are a general indication based on their good esthetic properties and clinical performance[94] and such restorations are an appropriate method for preventing further deterioration.[108] A meta-analysis of the clinical performance of Class V restorations determined that composite resins were preferred over glass ionomer cement and polyacid-modified composite resins.[109] Another systematic review and meta-analysis determined that glass ionomer has better retention than composite resin and was similar in the other properties evaluated.[110] Yet another systematic review and meta-analysis indicated glass ionomers had better retention than composite resin but inferior surface roughness and color matching.[111] It has been proposed that placing a glass ionomer cement underneath a composite resin, the so-called sandwich or mixed technique, combines the good characteristics of glass ionomer with that of composite resin.[112] An 18-month study of a glass hybrid (glass particles dispersed in a glass ionomer) indicated it may be a suitable alternative to composite resins.[113]

Resin-modified glass ionomers have been compared with other materials for the restoration of NCCLs. A 1-year study compared resin-modified glass ionomer, flowable composite resin, and polyacid-modified composite resin and the authors concluded all three materials were clinically acceptable but the resin-modified glass ionomer had superior

marginal adaptation and esthetics.[114] A 5-year study comparing composite resins and resin-modified glass ionomer determined the clinical performance of resin-modified glass ionomer was superior to composite resin.[115] A 7-year study reported resin-modified glass ionomers as being superior to the adhesive system and composite resin used in the study.[116] Conversely, a 3-year clinical evaluation determined that composite resin restorations were superior to resin-modified glass ionomer.[117] A 5-year evaluation of resin-modified glass ionomer cement and polyacid-modified composite resin restorations in a general dental practice resulted in both types of restorations being judged as having unsatisfactory long-term performance.[118] Similarly, a 5-year evaluation of resin-modified glass ionomer and polyacid-modified composite resin resulted in a high and similar failure rate for both materials when restoring NCCLs.[119]

After reading the above studies and systematic reviews, it is easy to be confused as to which material is best for restoring NCCLs. There was no material identified in the selected literature as being the best. It appears that different clinicians have obtained different results with the same materials. Therefore, it becomes a matter of personal experience and preference regarding the material to be used. The authors of this paper prefer composite resin for the restoration of NCCLs due to its good bonding, surface smoothness, color matching, and established clinical performance.

PROPOSED CLINICAL GUIDELINES FOR RESTORING NCCLS

The purpose of this section is to present procedural guidelines based on available evidence as well as the perspective of the authors of this publication.

Isolation of the lesion

Use of rubber dam isolation was determined to produce significantly higher restoration retention,[109] but one of the challenges with the use of a rubber dam is that clamp may promote gingival recession when restoring NCCLs.[120] Use of retraction cord is an alternative method of isolation with less risk of gingival recession.

Clinical recommendation

Rubber dam isolation is very effective when the risk of gingival recession is not likely to occur as with a thick healthy gingival phenotype and the lesion is in a location that is not esthetically critical should some recession occur. In the presence of unhealthy gingiva where bleeding occurs easily, the use of a rubber dam is beneficial. Retraction cord can also be effectively used to obtain isolation in most situations and is easier to manage than the use of a rubber dam for many patients. Placing a small, gingival retraction cord in the sulcus

FIGURE 10 Retraction cord has been placed in the sulcus to provide gingival retraction and isolation of the lesion in preparation for restoration.

such as #000 Ultrapak™ (Ultradent, South Jordan, UT) that has been soaked in a hemostatic agent and then blotted on a gauze square to remove excess agent will provide gingival retraction and control any bleeding that may occur (Fig 10). However, it is important not to have hemostatic agent present over the dentin surface. While there have been studies that determined the presence of hemostatic agents on dentin did not affect composite resin bond strength,[121,122] there have been multiple reviews and studies that determined the presence of such agents negatively affects bond strength.[123–131] One study determined the tooth surfaces contaminated with hemostatic agents can be cleaned using airborne particle abrasion using low-pressure aluminum oxide or phosphoric acid etching and thereby restoring bond strengths to precontamination levels.[132] In addition to the above bonding issues, the authors of this paper have found that leaving retraction cords in the sulcus for extensive time periods or using overly large diameter cords may induce gingival recession.

Surface preparation of the lesion

It has been stated that "mechanical removal of surface dentin improved retention rates of composite resins in NCCLs"[133] and "hybrid layer thickness was increased with all adhesives when superficial dentin was removed."[134] It has also been stated there is little evidence directly comparing roughening the surface with nonroughening.[135] In addition, the removal of surface dentin may not be completely effective,[98] meaning it still may not provide a bonding surface comparable to normal dentin but will be better than no surface preparation. Another recommendation is to extend the preparation of the lesion to include peripheral sound dentin to improve the

bond strength[102] but one has to weigh this proposal versus the additional removal of sound tooth structure and the likely subgingival extension of the restoration that makes isolation more difficult.

Clinical recommendation

While there have been different perspectives regarding the potential benefit or lack thereof with surface texturing, it has not been determined to be detrimental. Therefore, it is recommended to slightly remove surface dentin using either a fine grit rotary diamond instrument or carbide bur. A fine grit diamond instrument is preferred rather than a carbide bur because a bur can result in more aggressive dentin removal then is desirable.

Use of mechanical retention

To test the adhesive characteristics of different materials, clinical studies have been performed without the use of mechanical retention and have reported successful results.[135–137] They have reported favorable restoration retention without the aid of mechanical retention.[135] However, there is a lack of studies specifically comparing the use of mechanical retention with no mechanical retention and there is a lack of evidence to indicate mechanical retention is detrimental to the longevity of restorations. In the previously discussed survey of members of a prosthodontic organization, it was interesting to note that clinicians who did not use mechanical retention had a lower estimate of their restoration longevity.[96]

Clinical recommendation

Since there is a lack of evidence documenting a negative effect of using conservative mechanical retention, the procedure below is recommended for those clinicians who choose to use mechanical retention in addition to adhesive treatment of the tooth. It is proposed that two rounded depressions, about 1.0 mm deep, be prepared into the cervical floor of the lesion and two into the occlusal wall of posterior tooth using the tip of a number 330 pear carbide bur with a diameter of 0.8 mm or a number 2 round bur with a diameter of 1.0 mm (Fig 11). The depressions are located about 1.0 inside the perimeter of the root (Fig 12). With smaller teeth such as mandibular incisors, only one depression is placed into each surface.

Enamel beveling

Beveling of enamel is used because enamel is more effectively etched than dentin and extending the restoration of beveled enamel improves the esthetic result by gradually creating a color change between the restorative material and the tooth.[138] It also increases the surface area for bonding by creating additional micromechanical retention.[102,138] In a systematic review of enamel beveling, it was concluded there is not sufficient evidence to support the benefits of enamel beveling over nonbeveling[139] but it was pointed out that the conclusion was based on only two low risk of bias randomized controlled trials and a subsequent analysis of the paper indicated there is not enough evidence to support this conclusion.[140]

Clinical recommendation

Since there is no evidence indicating a negative effect of beveling the enamel and it increases the bonding area, provides a better substrate for etching, and improves the esthetic result, it is recommended to bevel the enamel.

Selecting adhesive agent

There have been studies examining the long-term effectiveness of adhesives used with NCCLs. A13-year randomized clinical trial of two different three-step adhesives used with two composite resins concluded the clinical effectiveness of the tested systems was clinically acceptable.[141] Another study of a three-step adhesive (etch, primer, and adhesive) after 12 years showed long-term durability with an overall retention rate of 89%.[142] Other studies have compared the effectiveness of adhesives when using single-step (self-etch) and multistep systems for the restoration of NCCLs. In one study using resin-modified glass ionomer restorations, the three-step adhesive system provided better retention then the one-step adhesive[143] whereas other studies have determined there was no difference in retention between one-step and multistep adhesives.[138,144,145–147] Regarding marginal discoloration, it has been reported to be higher with one-step systems.[139,147,148] A systematic review of one-step (self-etch) and multistep (etch-and-rinse) adhesives determined there was not sufficient evidence to support one system over the other.[149] Another systematic review indicated there was no significant difference between three-step, two-step, and one-step adhesive systems.[150]

Clinical recommendation

Both multistep and single-step adhesives can be effectively used for restoration of NCCLs as long as total etching is incorporated into the procedure to improve the clinical longevity of the restoration.

Enamel etching

A 3-year clinical evaluation determined that acid-etching of enamel margins "enhanced the performance of the two-step

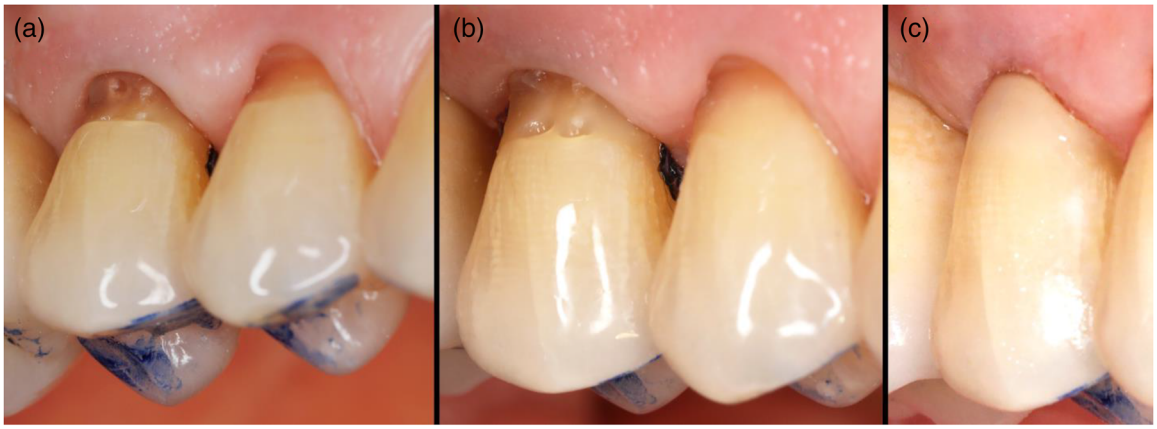

FIGURE 11 (a), Two depressions, about 1.0 mm deep have been placed in the cervical floor of the lesion to provide mechanical retention for a restoration. (b), Two additional retentive depressions have also been placed into the occlusal wall of the lesion. (c), The lesion has been restored with a composite resin restoration.

FIGURE 12 Two depressions, approximately 1.0 mm deep, have been formed about 1.0 mm inside the perimeter of the root and two additional depressions in the occlusal surface (not visible) for retention of a restoration in this very deep cervical lesion.

self-etch adhesive in terms of marginal discoloration and marginal adaptation at the enamel side."[151] In addition, a systematic review stated "selective enamel etching prior to application of self-etch adhesive systems in NCCLs can produce composite restorations with higher longevity" with less marginal discoloration and loss of retention.[152] Another 3-year clinical evaluation of a two-step self-etch adhesive stated "additional etching of the enamel cavity margins was not critical for its clinical performance."[153]

Clinical recommendation

It is suggested the enamel be etched in accordance with the etchant manufacturer's recommended time for enamel.

Dentin etching time

Since sclerotic dentin, as noted above, has occluded tubules and is more resistant to etching, it has been recommended to increase the etching time to improve the bonding.[98,154] A laboratory study of human canines and premolars with saucer-shaped NCCLs determined that extending the etching time to 30 seconds rather than 15 seconds resulted in a more predictable bond.[105] Another laboratory study of extracted premolars determined that increasing the etching time to 30 seconds produced more effective demineralization of NCCL sclerotic dentin and was comparable to normal dentin etched for 15 seconds.[155] Yet another laboratory study of extracted premolars reported that increased etching time can enhance bond strength.[156] To protect the pulp in the presence of very deep lesions, a small amount of a light polymerized base can be applied (Ultra-Blend™ plus, Ultradent, South Jordan, Utah, USA (Fig 13). Ultra-Blend™ is a radiopaque calcium hydroxide material in a urethane dimethacrylate base used to help protect the pulp during the adhesive treatment procedures.

Clinical recommendation

It is proposed that a 30-second dentin etching time with 37% phosphoric acid be used when restoring NCCLs. Since both the enamel and dentin need to be etched, if clinically possible, the etching can be started with the dentin surface and after 15 seconds the application can be extended to enamel surface for the remaining 15 seconds.

Adhesive agent application technique

A systematic review indicated application in a "frictional mode" improved composite resin retention rates.[133] In addition, studies have used terms such as "agitation,"[157]

FIGURE 13 A light-polymerized radiopaque calcium hydroxide base has been placed in the deep portions of these lesions to help protect the pulp.

FIGURE 14 A disposable applicator with a constriction at the end of the handle is shown here with very slight bending of the tip when an application force of 29 grams is applied.

FIGURE 15 This disposable applicator without a constriction is applying a force of 27 grams with no bending of the tip.

"active application,"[158] "application with agitation,"[159] "continuous agitation,"[160] "vigorously rubbed,"[161] "vigorously agitated,"[162] and a "vigorous rubbing action."[163] Use of such terms can imply different actions and it is not clear what exactly is meant by these terms during application of an adhesive agent. Therefore, it is important to understand the amount of force to be used in applying an adhesive to dentin. Multiple studies have reported using a force of 34.5 ± 6.9 grams during adhesive application.[158,161–163]

The authors of this paper have studied the amount of force applied during use of several types of disposable applicators for the purpose of providing clinical guidance as to what is meant by active application of adhesive agents. It was determined that disposable applicators vary somewhat in their tip flexibility. With applicators that have a constriction at the end of the handle, there will be slight flexion of the tip when rubbing the dentin surface using a force between 20 and 30 grams (Fig 14). Disposable applicators without a constriction in the tip are slightly stiffer and there should not be any bending of the tip when actively rubbing the dentin surface (Fig 15).

Clinical recommendation

Adhesive agents should be applied with a disposable microbrush applicator using a light scrubbing motion (force between 20 and 30 grams) for 20 seconds or as indicated by the adhesive manufacturer. When using the more flexible disposable applicators there should only be slight flexion of the applicator tip and no flexion of the tip with stiffer applicators. When using a self-adhesive bonding agent, it is recommended that fresh adhesive be applied during the application time to prevent a rapid rise in pH as the calcium is dissolved which reduces the effectiveness of the acidic monomer at etching the surface.

Type of composite resin

There have been clinical studies that compared flowable and sculptable composite resins. Two of these studies reported the flowable materials produced similar results to the sculptable materials.[164,165] Another study also reported similar clinical performance of flowable and sculptable composite resins but there was better marginal adaptation with the flowable material.[166] A systematic review reported better marginal adaptation for flowable compared with sculptable composite resin but the authors questioned the quality of evidence.[167] One of the reasons why the current studies have reported comparable performance is due to the newer flowable

composite resins having higher filler content and better physical and mechanical properties.

Clinical recommendation

Both flowable and sculptable materials can be effectively used but it is the opinion of the authors that flowable composite resins are easier to manipulate into deep areas and move around the surfaces of NCCLs. Flowable composite resins are also easier to place in small increments and manipulate into retentive areas when retention is used. With a large cervical lesion, some dentists apply and polymerize a layer of flowable composite resin and then add an external layer of sculptable composite resin to provide enhanced resistance to wear.

CONSERVATIVE TREATMENT WHEN CARIES ARE PRESENT IN CERVICAL LESIONS

When cervical caries lesions are present, they can be restored as described above but another option is available. Silver diamine fluoride has been effectively used to arrest caries and has been determined to be more effective than sodium fluoride.[168] An umbrella review, summarizing systematic reviews on the effectiveness of silver diamine fluoride, reported consistent support for its effectiveness in arresting coronal caries in the primary dentition as well as arresting and preventing root caries in older adults.[169] A protocol has been proposed for use of silver diamine fluoride for root caries where the solution is applied and then a few days or a week later a second treatment is applied.[170] The rationale for the two-appointment protocol is that the carious dentin will become very dark after a few days, making it easier to distinguish between carious and sound dentin, a process that helps prevent the removal of excess tooth structure should a restoration subsequently be placed for esthetic reasons.[170]

The primary author of this publication has effectively used silver diamine fluoride to arrest cervical caries in adults while recognizing there will be substantial darkening of the area following treatment (Fig 16). Due to the tooth discoloration produced by the treatment, it is important to inform the patient so they understand the benefit of the treatment as well as the discoloration that will be present. They also need to know the area can be restored with a tooth-colored material should they desire after the caries is arrested. It is proposed to dry the carious area using compressed air, dispense one drop of silver diamine fluoride solution into a dappen dish, and then apply the solution to the area using a small disposable applicator (Fig 17) along with 10 to 20 seconds of scrubbing action. Excess solution is then removed using a cotton pellet and the lesion is maintained saliva-free for 3 to 5 minutes. The surface is then gently dried using compressed air if moisture from the solution is still present on the surface of the lesion. More than one treatment may be needed so it is recommended to check the area after a few days or week to

FIGURE 16 Silver diameter fluoride was applied to the carious cervical lesions in the mandibular second premolar and first molar and the caries has been arrested. The treated areas are now dark and were left that way for this patient who was not concerned about the discoloration. For patients who want to eliminate the discoloration, the dark surface can be restored using a tooth-colored material.

FIGURE 17 A bottle of 38% silver diamine fluoride (Advantage Arrest, Elevate Oral Care LLC., West Palm Beach, FL) is shown along with a dappen dish into which one drop has been dispensed. The solution will be applied with the disposable applicator shown here.

determine if there is still some soft material present that warrants another treatment. The solution will invariably contact the gingiva and produce a temporary stain on the gingiva so it is good to advise the patient that there will be some soft tissue discoloration that will disappear whereas the tooth discoloration will not disappear. To avoid the temporary gingival discoloration, a protective coating such as petroleum jelly can be applied but care must be exercised to not allow the jelly onto the surface of the lesion as it will interfere with the effectiveness of the silver diamine fluoride solution in that area. Drying the gingiva and carefully applying a layer of PermaSeal™ resin glaze (Ultradent, South Jordan, UT)

with a small disposable applicator, followed by light polymerization, is another effective means of preventing gingival discoloration. Following application of the fluoride solution, the layer of resin glaze can be peeled free from the gingiva.

CONCLUSIONS

NCCLs have a prevalence that ranges between 10% and 90% and they increase with age. They can be shallow depressions or deep V-shaped notches commonly caused by factors such as excessive horizontal toothbrushing and consumption of acidic foods and drinks. Occlusal factors have been identified as contributing to the prevalence of NCCLs in some studies whereas other studies indicate there is no relationship. Treatment of NCCLs should be managed conservatively through preventive intervention with restorative treatment delayed as long as possible. However, lesion progression, impact on patient's quality of life, sensitivity, poor esthetics, and food collection may necessitate restoration. Composite resins are commonly used to restore NCCLs but the presence of sclerotic dentin with tubules occluded with crystalline deposits makes etching and bonding more challenging. Therefore, it is recommended to texture the surface of the dentin with a rotary instrument to improve restoration retention with some clinicians adding mechanical retention. Beveling the enamel is used to increase the bonding area and retention as well as enhance the esthetic result. Both multistep and single-step adhesives have been used with dentin etching increased to 30 seconds due to the sclerotic dentin. The adhesive agent should be applied with a light scrubbing motion for 20 seconds but without excessive force that induces substantial bending of a disposable applicator. Both flowable and sculptable composite resins have been successfully used with some clinicians applying and polymerizing a layer of flowable composite resin and then adding an external layer of sculptable composite resin to provide enhanced resistance to wear. When caries is present, silver diamine fluoride has been used to arrest the caries rather than restore the lesion.

CONFLICT OF INTEREST

No conflict of interest to report for any of the authors.

ORCID

Charles J. Goodacre DDS, MSD, FACP, FFDRCS Irel (Hon)
https://orcid.org/0000-0002-0626-3624

REFERENCES

1. Bartlett D, O'toole S. Tooth wear: best evidence consensus statement. J Prosthodont. 2021;30:20–5.
2. Schlueter N, Amaechi BT, Bartlett D, Buzalaf MAR, Carvalho TS, Ganss C, et al. Terminology of erosive tooth wear: consensus report of a workshop organized by the ORCA and the cariology research group of the IADR. Caries Res. 2020;54:2–6.
3. Roberts WE, Mangum JE, Schneider PM. Pathophysiology of demineralization, Part 1: attrition, erosion, abfraction, and noncarious cervical lesions. Curr Osteoporos Rep. 2022;20:90–105.
4. Bartlett DW, Shah P. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. J Dent Res. 2006;85:306–12.
5. Piotrowski BT, Gillette WB, Hancock EB. Examining the prevalence and characteristics of abfraction like cervical lesions in a population of U.S. veterans. J Am Dent Assoc. 2001;132:1694–701.
6. Que K, Guo B, Jia Z, Chen Z, Yang J, Gao P. A cross-sectional study: non-carious cervical lesions, cervical dentin hypersensitivity and related risk factors. J Oral Rehabil. 2013;40:24–32.
7. Sadaf D, Ahmad Z. Role of brushing and occlusal factors in non-carious cervical lesions (NCCL). Int J Biomed Sci. 2014;10:265–8.
8. Yang J, Cai D, Wang F, He D, Ma L, Jin Y, Que K. Non-carious cervical lesions (NCCLs) in a random sampling community population and the association of NCCLs with occlusive wear. J Oral Rehabil. 2016;43:960–6.
9. Schlueter N, Luka B. Erosive tooth wear – a review of global prevalence and its prevalence in risk groups. Br Dent J. 2018;224:364–70.
10. Nguyen C, Ranjitkar S, Kaidonis Ja, Townsend G. A qualitative assessment of non-carious cervical lesions in extracted human teeth. Aust Dent J. 2008;53:46–51.
11. Yoshizaki KT, Francisconi-Dos-Rios LF, Sobral MAP, Aranha ACC, Mendes FM, Scaramucci T. Clinical features and factors associated with non-carious cervical lesions and dentin hypersensitivity. J Oral Rehabil. 2017;44:112–8.
12. Smith WAJ, Marchan S, Rafeek RN. The prevalence and severity of non-carious cervical lesions in a group of patients attending a university hospital in Trinidad. J Oral Rehabil. 2008;35:128–34.
13. Aw TC, Lepe X, Johnson GH, Mancl L. Characteristics of noncarious cervical lesions. A clinical investigation. J Am Dent Assoc. 2002;133:725–733.
14. Bhundia S, Bartlett D, O'Toole S. Non-carious cervical lesions – can terminology influence our clinical assessment? Br Dent J. 2019;227:985–8.
15. Michael JA, Kaidonis JA, Townsend GC. Non-carious cervical lesions on permanent anterior teeth: a new morphological classification. Aust Dent J. 2010;55:134–7.
16. Abdalla R, Mitchell RJ, Ren YF. Non-carious cervical lesions imaged by focus variation microscopy. J Dent. 2017;63:14–20.
17. Walter C, Kress E, Götz H, Taylor K, Willershausen I, Zampelis A. The anatomy of non-carious cervical lesions. Clin Oral Investig. 2014;18:139–46.
18. Daley TJ, Harbrow DJ, Kahler B, Young WG. The cervical wedge-shaped lesion in teeth: a light and electron microscopic study. Aust Dent J. 2009;54:212–9.
19. Hayashi M, Kubo S, Pereira PNR, Ikeda M, Takagaki T, Nikaido T, Tagami J. Progression of non-carious cervical lesions: 3D morphological analysis. Clin Oral Investig. 2022;26:575–83.
20. Sawlani K, Lawson NC, Burgess JO, Lemons JE, Kinderknecht KE, Givan DA, et al. Factors influencing the progression of noncarious cervical lesions: a 5-year prospective clinical evaluation. J Prosthet Dent. 2016;115:571–7.
21. Sugita I, Nakashima S, Ikeda A, Burrow MF, Nikaido T, Kubo S, et al. A pilot study to assess the morphology and progression of non-carious cervical lesions. J Dent. 2017;57:51–6.
22. Lucci A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. Caries Res. 2000;34:182–7.
23. Borcic J, Anic I, Urek MM, Ferreri S. The prevalence of non-carious lesions in permanent dentition. J Oral Rehabil. 2004;31:117–23.
24. Faye B, Sarr M, Kane AW, et al. Prévalence et facteurs etiologiques des lesions cervicales non carieuses. Etude dans une population sénégalaise [Prevalence and etiologic factors of non-carious cervical lesions. A study in a Senegalese population]. Odontostomatol Trop. 2005;28:15–8. French. Erratum in: Odontostomatol Trop 2006
25. Penoni DC, Gomes Miranda M, Sader F, Vettore MV, Leão ATT. Factors associated with noncarious cervical lesions in different age ranges: a cross-sectional study. Eur J Dent. 2021;15:325–31.

26. Teixeira DNR, Thomas RZ, Soares PV, Cune MS, Gresnigt MMM, Slot DE. Prevalence of noncarious cervical lesions among adults: a systematic review. J Dent. 2020;95:103285. https://doi.org/10.1016/j.jdent.2020.103285
27. Takehara J, Takano T, Akhter R, Morita M. Correlations of noncarious cervical lesions and occlusal factors determined by using pressure-detecting sheet. J Dent. 2008;36:774–9.
28. Pegoraro LF, Scolaro JM, Conti PC, Telles D, Pegoraro TA. Noncarious cervical lesion in adults: prevalence and occlusal aspects. J Am Dent Assoc. 2005;136:1694–700.
29. Lussi A, Schaffner M, Hotz P, Suter P. Dental erosion in a population of Swiss adults. Commun Dent Oral Epidemiol. 1991;19:286–90.
30. Bernhardt O, Gesch D, Schwahn C, Mack F, Meyer G, John U, Kocher T. Epidemiological evaluation of the multifactorial aetiology of abfractions. J Oral Rehabil. 2006;33:17–25.
31. Tomasik M. Analiza czynników etiologicznych ubytków przyszyjkowych niepróchnicowego pochodzenia [Analysis of etiological factors involved in noncarious cervical lesions]. Ann Acad Med Stetin. 2006;52:125–36. Polish. PMID: 17385359
32. Telles D, Pegoraro LF, Pereira JC. Incidence of noncarious cervical lesions and their relation to the presence of wear facets. J Esthet Restor Dent. 2006;18:178–83; discussion 184
33. Ahmed H, Durr-E-Sadaf RM. Factors associated with non-carious cervical lesions (NCCLs) in teeth. J Coll Physicians Surg Pak. 2009;19:279–82.
34. Brandini DA, Pedrini D, Panzarini SR, Benete IM, Trevisan CL. Clinical evaluation of the association of noncarious cervical lesions, parafunctional habits, and TMD diagnosis. Quintessence Int. 2012;43:255–62.
35. Brady JM, Woody RD. Scanning microcopy of cervical erosion. J Am Dent Assoc. 1977;94:726–29.
36. Young WG, Khan F. Sites of dental erosion are saliva-dependent. J Oral Rehabil. 2002;29:35–43.
37. Alvarez-Arenal A, Alvarez-Menendez L, Gonzalez-Gonzalez I, Alvarez-Riesgo JA, Brizuela-Velasco A, Dellanos-Lanchares H. Non-carious cervical lesions and risk factors: a case-control study. J Oral Rehabil. 2019;46:65–75.
38. Warreth A, Abuhijleh E, Almaghribi MA, Mahwal G, Ashawish A. Tooth surface loss: a review of literature. Saudi Dent J. 2020;32:53–60.
39. Ranjitkar S, Kaidonis JA, Smales RJ. Gastroesophageal reflux disease and tooth erosion. Int J Dent. 2012; 2012:1. https://doi.org/10.1155/2012/479850
40. Sivasitamparam K, Young W, Jirattanasopa V, Priest J, Khan F, Harbrow D, et al. Dental erosion in asthma: a case-control study from south east Queensland. Aust Dent J. 2002;47:298–303.
41. O'Toole S, Newton T, Moazzez R, Hasan A, Bartlett D. Randomised controlled clinical trial investigating the impact of implementation planning on behaviour related to the diet. Sci Rep. 2018;8:8024. https://doi.org/10.1038/s41598-018-26418-0
42. Delgado AJ, Dias Ribeiro AP, Quesada A, et al. Potential erosive effect of mouthrinses on enamel and dentin. Gen Dent. 2018;66:75–9.
43. Delgado A, Dias Ribeiro AP, Aslam M, Olafsson VG, Pereira PN. Erosive assessment of dry mouth lozenges and tablets on enamel and dentin. J Dent. 2021;105:103496. https://doi.org/10.1016/j.jdent.2020.103496
44. Pintado MR, Delong R, Ko C-C, Sakaguchi RL, Douglas WH. Correlation of noncarious cervical lesion size and occlusal wear in a single adult over a 14-year time span. J Prosthet Dent. 2000;84:436–43.
45. Miller N, Penaud J, Ambrosini P, Bisson-Boutelliez C, Brianã§On S. Analysis of etiologic factors and periodontal conditions involved with 309 abfractions. J Clin Periodontol. 2003;30:828–32.
46. Yoshizaki KT, Francisconi-Dos-Rios LF, Sobral MAP, Aranha ACC, Mendes FM, Scaramucci T. Clinical features and factors associated with non-carious cervical lesions and dentin hypersensitivity. J Oral Rehabil. 2017;44:112–8.
47. Madani A-Ol-S, Ahmadian-Yazdi A. An investigation into the relationship between noncarious cervical lesions and premature contacts. Cranio. 2005;23:10–5.
48. Haralur, Alqahtani, Almazni, Alqahtani Association of non-carious cervical lesions with oral hygiene habits and dynamic occlusal parameter. Diagnostics (Basel). 2019;9:43.
49. Mayhew RB, Jessee SA, Martin RE. Association of occlusal, periodontal, and dietary factors with the presence of non-carious cervical dental lesions. Am J Dent. 1998;11:29–32.
50. Bevenius J, 'Estrange P, Karlsson S, Carlsson GE, Idiopathic cervical lesions: in vivo investigation by oral microscopy and scanning electron microscopy. A pilot study. J Oral Rehabil. 1993;20:1–9.
51. Nascimento BL, Vieira AR, Bezamat M, Ignã¡Cio SA, Souza EM. Occlusal problems, mental health issues and non-carious cervical lesions. Odontology. 2022;110:349–55.
52. Alvarez-Arenal A, Alvarez-Menendez L, Gonzalez-Gonzalez I, Jiménez-Castellanos E, Garcia-Gonzalez M, Dellanos-Lanchares H. The role of occlusal factors in the presence of noncarious cervical lesions in young people: A case-control study. Oper Dent. 2019;44:E12–22.
53. Senna P, Del Bel Cury A, Rösing C. Non-carious cervical lesions and occlusion: a systematic review of clinical studies. J Oral Rehabil. 2012;39:450–62.
54. Grippo JO. Abfractions: a new classification of hard tissue lesions of teeth. J Esthet Dent. 1991;3:14–9.
55. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. J Prosthet Dent. 1984;52:374–80.
56. Rees JS, Jagger DC. Abfraction lesions: myth or reality? J Esthet Restor Dent. 2003;15:263–71.
57. Ceruti P, Menicucci G, Mariani GD, et al. Non carious cervical lesions: a review. Minerva Stomatol. 2006;55:43–57.
58. Tanaka M, Naito T, Yokota M, Kohno M. Finite element analysis of the possible mechanism of cervical lesion formation by occlusal force. J Oral Rehabil. 2003;30:60–7.
59. Jakupovic S, Cerjakovic E, Topcic A, Ajanovic M, Konjhodzicprcic A, Vukovic A. Analysis of the abfraction lesions formation mechanism by the finite element method. Acta Inform Med. 2014;22:241–5.
60. Vandana K, Deepti M, Shaimaa M, Naveen K, Rajendra D. A finite element study to determine the occurrence of abfraction and displacement due to various occlusal factors and with different alveolar bone height. J Indian Soc Periodontol. 2016;20:12–6.
61. Borcic J, Anic I, Smojver I, Catic A, Miletic I, Ribaric SP. 3D finite element model and cervical lesion formation in normal occlusion and in malocclusion. J Oral Rehabil. 2005;32:504–10.
62. Dejak B, Mlotkowski A, Romanowicz M. Finite element analysis of mechanism of cervical lesion formation in simulated molars during mastication and parafunction. J Prosthet Dent. 2005;94:520–9.
63. Palamara D, Palamara JEA, Tyas MJ, Messer HH. Strain patterns in cervical enamel of teeth subjected to occlusal loading. Dent Mater. 2000;16:412–9.
64. Palamara JEA, Palamara D, Messer HH, Tyas MJ. Tooth morphology and characteristics of non-carious cervical lesions. J Dent. 2006;34:185–94.
65. Duangthip D, Man A, Poon PH, et al. Occlusal stress is involved in the formation of non-carious cervical lesions: a systematic review of abfraction. Am J Dent. 2017;30:212–20.
66. Michael Ja, Townsend Gc, Greenwood Lf, Kaidonis Ja. Abfraction: separating fact from fiction. Aust Dent J. 2009;54:2–8.
67. Litonjua LA, Andreana S, Patra AK, et al. An assessment of stress analysis in the theory of abfraction. Biomed Mater Eng. 2004;14:311–21.
68. Litonjua LA, Bush PJ, Andreana S, Tobias TS, Cohen RE. Effects of occlusal load on cervical lesions. J Oral Rehabil. 2004;31:225–32.
69. Estafan A, Furnari PC, Goldstein G, Hittelman EL. In vivo correlation of noncarious cervical lesions and occlusal wear. J Prosthet Dent. 2005;93:221–6.

70. Rees JS, Hammadeh M. Undermining of enamel as a mechanism of abfraction lesion formation: a finite element study. Eur J Oral Sci. 2004;112:347–52.
71. Lee HE, Lin CL, Wang CH, Cheng CH, Chang CH. Stresses at the cervical lesion of maxillary premolar—a finite element investigation. J Dent. 2002;283–90.
72. Ahmić Vuković A. Occlusal stress distribution on the mandibular first premolar – FEM analysis. Acta Medica Academica. 2019;48:255–61.
73. Machado AC, Soares CJ, Reis BR, Bicalho AA, Raposo L, Soares PV. Stress-strain analysis of premolars with non-carious cervical lesions: influence of restorative material, loading direction and mechanical fatigue. Oper Dent. 2017;42:253–65.
74. Salari N, Ghasemi H, Mohammadi L, Behzadi MH, Rabieenia E, Shohaimi S, et al. The global prevalence of osteoporosis in the world: a comprehensive systematic review and meta-analysis. J Orthop Surg Res. 2021;16:609.
75. Selwitz RH, Ismail AI, Pitts NB. Dental caries. Lancet. 2007;369: 51–9.
76. Fleming E, Afful J. Prevalence of total and untreated dental caries among youth: United Stated, 2015–2016. NCHS Data Brief. 2018;307:1–8.
77. Roberts WE, Mangum JE, Schneider PM. Pathophysiology of demineralization, Part II: enamel white spots, cavitated caries, and bone infection. Curr Osteoporos Rep. 2022;20:106–19.
78. Huang X, She L, Liu H, Liu P, Chen J, Chen Y, et al. Study of oral microorganisms contributing to non-carious cervical lesions via bacterial interaction and pH regulation. J Cell Mol Med. 2021;25:3103–12.
79. Pecie R, Krejci I, Garcia-Godoy F, Bortolotto T. Noncarious cervical lesions–a clinical concept based on the literature review. Part 1: prevention. Am J Dent. 2011;24:49–56.
80. Wei W, Pang S, Sun D: The pathogenesis of endemic fluorosis: research progress in the last 5 years. J Cell Mol Med. 2019;23:2333–42.
81. Bhadang KA, Gross KA. Influence of fluorapatite on the properties of thermally sprayed hydroxylapatite coatings. Biomaterials. 2004;25:4935–45.
82. Medeiros TLM, Mutran SCAN, Espinosa DG, Do Carmo Freitas Faial K, Pinheiro HHC, D'Almeida Couto RS. Prevalence and risk indicators of non-carious cervical lesions in male footballers. BMC Oral Health. 2020;20:215.
83. Antunes LS, Veiga L, Nery VS, Nery CC, Antunes LA. Sports drink consumption and dental erosion among amateur runners. J Oral Sci. 2017;59:639–43.
84. Kinjo R, Wada T, Churei H, Ohmi T, Hayashi K, Yagishita K, et al. development of a wearable mouth guard device for monitoring teeth clenching during exercise. Sensors (Basel). 2021;21:1503.
85. Yan J, Xie W, Zhang L, et al. Low torque is a risk factor for non-carious cervical lesions (NCCLs) in maxillary premolars. Am J Dent. 2021;34:245–9.
86. Roberts WE, Stocum DL. Part II: temporomandibular joint (tmj)-regeneration, degeneration, and adaptation. Curr Osteoporos Rep. 2018;16:369–79.
87. Roberts WE, Goodacre CJ. The temporomandibular joint: a critical review of life-support functions, development, articular surfaces, biomechanics and degeneration. J Prosthodont. 2020;29:772–9.
88. Carter DR, Hayes WC. Compact bone fatigue damage: a microscopic examination. Clin Orthop Relat Res. 1977;(127):265–74.
89. Hartsfield Jr J. Pathways in external apical root resorption associated with orthodontia. Orthod Craniofac Res. 2009;12:236–42.
90. Veitz-Keenan A, Barna JA, Strober B, Matthews AG, Collie D, Vena D, et al. Treatments for hypersensitive noncarious cervical lesions: a practitioners engaged in applied research and learning (PEARL) network randomized clinical effectiveness study. J Am Dent Assoc. 2013;144:495–506.
91. Roberts WE, Stocum DL. Part II: tempormandibular joint (TMJ)-Regeneration, degeneration, and adaptation. Curr Osteoporos Rep. 2018;16:369–79.
92. Loomans B, Opdam N, Attin T, et al. Severe tooth wear: European consensus statement on management guidelines. J Adhes Dent. 2017;19:111–9.
93. Bartlett D, O'toole S. Tooth wear and aging. Aust Dent J. 2019;64(1 Suppl):S59–62.
94. Peumans M, Politano G, Meerbeek BV. Treatment of noncarious cervical lesions: when, why, and how. Int J Esthet Dent. 2020;15:16–42.
95. Osborne-Smith KL, Burke FJT, Wilson NHF. The aetiology of the non-carious cervical lesion. Int Dent J. 1999;49:139–43.
96. Estafan A, Bartlett D, Goldstein G. A survey of management strategies for noncarious cervical lesions. Int J Prosthodont. 2014;27:87–90.
97. Van Meerbeek B, Braem M, Lambrechts P, Vanherle G. Morphological characterization of the interface between resin and sclerotic dentin. J Dent. 1994;22:141–6.
98. Kwong S-M, Tay FR, Yip H-K, Kei L-H, Pashley DH. An ultrastructural study of the application of dentine adhesives to acid-conditioned sclerotic dentin. J Dent. 2000;28:515–28.
99. Marshall GW, Chang YJ, Saeki K, Gansky SA, Marshall SJ. Citric acid etching of sclerotic dentin lesions: an AFM study. J Biomed Mater Res. 2000;49:338–344.
100. Daley Tj, Harbrow Dj, Kahler B, Young Wg. The cervical wedge-shaped lesions in teeth: a light and electron microscopic study. Aust Dent J. 2009;54:212–19.
101. Tay FR, Kwong SM, Itthagarun A, King NM, Yip HK, Moulding KM, et al. Bonding of self-etching primer to non-carious cervical sclerotic dentin: interfacial ultrastructure and microtensile bond strength evaluation. J Adhes Dent. 2000;2:9–28.
102. Tay FR, Pashley DH. Resin bonding to cervical sclerotic dentin: a review. J Dent. 2004;32:173–96.
103. Kwong S. Micro-tensile bond strengths to sclerotic dentin using a self-etching and a total-etching technique. Dent Mater. 2002;18:359–69.
104. Duke ES, Lindemuth J. Variability of clinical dentin substrates. Am J Dent. 1991;4:241–6.
105. Lopes GC, Baratieri CM, Baratieri LN, Monteiro S Jr, Cardoso Vieira LC. Bonding to cervical sclerotic dentin: effect of acid etching time. J Adhes Dent. 2004;6:19–23.
106. Wood ID, Kassir ASA, Brunton PA. Effect of lateral excursive movements on the progression of abfraction lesions. Oper Dent. 2009;34:273–9.
107. Silva AG, Martins CC, Zina LA, Moreira AN, Paiva SM, Pordeus IA, et al. The association between occlusal factors and noncarious cervical lesions: a systematic review. J Dent. 2013;41:9–16.
108. Du J-K, Wu J-H, Chen P-H, Ho P-S, Chen K-K. Influence of cavity depth and restoration of non-carious cervical root lesions on strain distribution from various loading sites. BMC Oral Health. 2020;20:98.
109. Mahn E, Rousson V, Heintze S. Meta-analysis of the influence of bonding parameters on the clinical outcome of tooth-colored cervical restorations. J Adhes Dent. 2015;17:391–403.
110. Bezerra IM, Brito ACM, De Sousa SA, Santiago BM, Cavalcanti YW, De Almeida LFD. Glass ionomer cements compared with composite resin in restoration of noncarious cervical lesions: a systematic review and meta-analysis. Heliyon. 2020;6:e03969.
111. Boing TF, de Geus JL, Wambier LM, et al. Are glass-ionomer cement restorations in cervical lesions more long-lasting than resin-based composite resins? A systematic review and meta-analysis. J Adhes Dent. 2018;20:435–52.
112. Francisconi LF, Scaffa PM, de Barros VR, Coutinho M, Francisconi PAS. Glass ionomer cements and their role in the restoration of non-carious cervical lesions. J Appl Oral Sci. 2009;17:364–9.
113. Göstemeyer G, Seifert T, Jeggle-Engbert L-M, et al. Glass hybrid versus nanocomposite for restoration of sclerotic non-carious cervical lesions: 18-month results of a randomized controlled trial. J Adhes Dent. 2021;23:487–96.
114. Hussainy SN, Nasim I, Thomas T, et al. Clinical performance of resin-modified glass ionomer, flowable composite, and polyacid-modified resin composite in noncarious cervical lesions: one-year follow-up. J Conserv Dent. 2018;21:510–5.

115. Franco EB, Benetti AR, Ishikiriama SK, Santiago SL, Lauris JRP, Jorge MFF, et al. 5-year clinical performance of resin composite versus resin modified glass ionomer restorative system in non-carious cervical lesions. Oper Dent. 2006;31:403–8.
116. Fagundes Tc, Barata T, Bresciani E, Santiago Sl, Franco Eb, Lauris J, Navarro MF. Seven-year clinical performance of resin composite versus resin-modified glass ionomer restorations in noncarious cervical lesions. Oper Dent. 2014;39:578–87.
117. Folwaczny M, Loher C, Mehl A, Kunzelmann K-H, Hickel R. Class V lesions restored with four different tooth-colored materials –3-year results. Clin Oral Investig. 2001;5:31–9.
118. Smales RJ, Ng K. Longevity of a resin-modified glass ionomer cements and a polyacid-modified resin composite restoring non-carious cervical lesions in a general dental practice. Aust Dent J. 2004;49:196–200.
119. Folwaczny M, Mehl A, Kunzelmann KH, Hickel R. Clinical performance of a resin-modified glass ionomer and a compomer in restoring non-carious cervical lesions: 5-year results. Am J Dent. 2001;14:153–6.
120. Favetti M, Montagner AM, Fontes ST, Martins TM, Masotti AS, Jardim PDS, et al. Effects of cervical restorations on the periodontal tissues: 5-year follow-up results of a randomized clinical trial. J Dent. 2021 Mar:106:103571. https://doi.org/10.1016/j.dent.2020.103571
121. Kimmes NS, Olson TL, Shaddy RS, Latta MA. Effect of ViscoStat and ViscoStat Plus on composite shear bond strength in the presence and absence of blood. J Adhes Dent. 2006;8:363–6.
122. Groddeck S, Attin T. Effect of cavity contamination by blood and hemostatic agents on marginal adaptation of composite restorations. J Adhes Dent. 2017;19:259–64.
123. De Oliveira Bernades K, Hilgert LA, Ribeiro APD, Garcia FCP, Pereira PNR. The influence of hemostatic agents on dentin and enamel surfaces and dental bonding: a systematic review. J Am Dent Assoc. 2014;145:1120–7.
124. Maischberger C, Stawarczyk B, von Hajmasy A, Liebermann A. Hemostatic gingival retraction agents and their impact on prosthodontic treatment steps: a narrative review. Quintessence Int. 2018;49:719–32.
125. O'Keefe KL, Pinzon LM, Rivera B, Powers JM. Bond strength of composite to astringent-contaminated dentin using self-etching adhesives. Am J Dent. 2005;18:168–72.
126. Kuphasuk W, Harnirattisai C, Senawongse P, Tagami J. Bond strengths of two adhesive systems to dentin contaminated with a hemostatic agent. Oper Dent. 2007;32:399–405.
127. Mohammadi N, Kimyai S, Bahari M, Pournaghi-Azar F, Mozafari A. Effect of aluminum chloride hemostatic agent on microleakage of class V composite resin restorations bonded with an all-in-one adhesive. Med Oral Patol Oral Cir Bucal. 2012;17:e841–4. https://doi.org/10.4317/medoral.17683
128. Ajami AA, Kahnamoli MA, Kimyai S, Oskoee SS, Pournaghi-Azar F, Bahari M, et al. Effect of three different contamination methods on bond strength of a self-etching adhesive to dentin contaminated with an aluminum chloride hemostatic agent. J Contemp Dent Pract. 2013;14:26–33.
129. Ebrahimi SF, Shadman N, Abrishami A. Effect of ferric sulfate contamination on the bonding effectiveness of etch-and-rinse and self-etch adhesives to superficial dentin. J Conserv Dent. 2013;16:126–30.
130. Saad A, Inoue Go, Nikaido T, Abdou AMA, Sayed M, Burrow MF, Tagami J. Effect of dentin contamination with two hemostatic agents on bond strength of resin-modified glass ionomer cement with different conditioning. Dent Mater J. 2019;38:257–63.
131. Hoorizad M, Heshmat H, Hosseini TA, Kazemi SS, Tabatabaei SF. Effect of hemostatic agent on microshear bond strength of total-etch and self-etch adhesive systems. Dent Res J (Isfahan). 2019;16:361–5.
132. Chaiyabutr Y, Kois JC. The effect of tooth-preparation cleansing protocol on the bond strength of self-adhesive resin cement to dentin contaminated with a hemostatic agent. Oper Dent. 2011;36:18–26.
133. Rocha AC, Da Rosa W, Cocco AR, Da Silva AF, Piva E, Lund R-G, Influence of surface treatment on composite adhesion in noncarious cervical lesions: systematic review and meta-analysis. Oper Dent. 2018;43:508–19.
134. Eliguzeloglu E, Omurlu H, Eskitascioglu G, Belli S. Effect of surface treatments and different adhesives on the hybrid layer thickness of non-carious cervical lesions. Oper Dent. 2008;33:338–45.
135. Ritter AV, Swift EJ Jr, Heymann HO, Sturdevant JR, Wilder AD Jr. An eight-year clinical evaluation of filled and unfilled one-bottle dental adhesives. J Am Dent Assoc. 2009;140:28037; quiz 111–12.
136. Oz FD, Kutuk ZB, Ozturk C, Soleimani R, Gurgan S. An 18-month clinical evaluation of three different universal adhesives used with a universal flowable composite resin in the restoration of non-carious cervical lesions. Clin Oral Investig. 2019;23:1443–52.
137. Schwendicke F, Müller A, Seifert T, Jeggle-Engbert L-M, Paris S, Göstemeye G. Glass hybrid versus composite for non-carious cervical lesions: survival, restoration quality and costs in randomized controlled trial after 3 years. J Dent. 2021:110:103689. https://doi.org/10.1016/j.jdent.2021.103689
138. Aw TC, Lepe X, Johnson GH, Mancl LA. A three-year clinical evaluation of two-bottle versus one-bottle dentin adhesives. J Am Dent Assoc. 2005;136:311–22.
139. Schroeder M, Reis A, Luque-Martinez I, Loguercio AD, Masterson D, Maia LC. Effect of enamel bevel on retention of cervical composite resin restorations: a systematic review and meta-analysis. J Dent. 2015;43:777–88.
140. Ahmed K. Enamel bevelling and retention of composite restorations in non-carious cervical lesions. Evid Based Dent. 2016;17:88–9.
141. Peumans M, De Munck J, Van Landuyt KL, Poitevin A, Lambrechts P, Van Meerbeek B. A 13-year clinical evaluation of two three-step etch-and-rinse adhesives in non-carious class-V lesions. Clin Oral Investig. 2012;16:129–37.
142. Wilder AD, Swift EJ, Heymann HO, Ritter A©V, Sturdevant JR, Bayne SC. A 12-year clinical evaluation of a three-step dentin adhesive in noncarious cervical lesions. J Am Dent Assoc. 2009;140:526–35.
143. Van Dijken JWV. Clinical evaluation of three adhesive systems in class V non-carious lesions. Dent Mater. 2000;16:285–91.
144. Schroeder M, Correa IC, Bauer J, Loguercio AD, Reis A. Influence of adhesive strategy on clinical parameters in cervical restorations: a systematic review and meta-analysis. J Dent. 2017;62:36–53.
145. Swift EJ, Perdigão J, Wilder AD, Heymann HO, Sturdevant JR, Bayne SC. Clinical evaluation of two one-bottle dentin adhesives at three years. J Am Dent Assoc. 2001;132:1117–23.
146. Boushell LW, Heymann HO, Ritter AV, Sturdevant JR, Swift EJ, Wilder AD, et al. Six-year clinical performance of etch-and-rinse and self-etch adhesives. Dent Mater. 2016;32:1065–72.
147. Atalay C, Ozgunaltay G, Yazici AR. Thirty-six-month clinical evaluation of different adhesive strategies of a universal adhesive. Clin Oral Investig. 2020;24:1569–78.
148. Ruschel VC, Shibata S, Stolf SC, Chung Y, Baratieri LN, Heymann HO, Walter R. Eighteen-month clinical study of universal adhesives in noncarious cervical lesions. Oper Dent. 2018;43:241–9.
149. Chee B, Rickman LJ, Satterthwaite JD. Adhesives for the restoration of non-carious cervical lesions: a systematic review. J Dent. 2012;40:443–52.
150. Santos MJMC, Ari N, Steele S, Costella J, Banting D. Retention of tooth-colored restorations in non-carious lesions—a systematic review. Clin Oral Investig. 2014;18:1369–81.
151. Say EC, Özel E, Yurdagüven H, Soyman M. Three-year clinical evaluation of a two-step self-etch adhesive with or without selective enamel etching in non-carious cervical sclerotic lesions. Clin Oral Investig. 2014;18:1435–5.
152. Szesz A, Parreiras S, Reis A, Loguercio A. Selective enamel etching in cervical lesions for self-etch adhesives: a systematic review and meta-analysis. J Dent. 2016;53:1–11. https://doi.org/10.1016/j.jdent.2016.05.009
153. Peumans M, Munck J, Landuyt K, Lambrechts P, Meerbeek B. Three-year clinical effectiveness of a two-step self-etch adhesive in cervical lesions. Eur J Oral Sci. 2005;113:512–18.

154. Badavannavar AN, Ajari S, Nayak KUS, Khijmatgar S. Abfraction: etiopathogenesis, clinical aspect, and diagnostic-treatment modalities: a review. Indian J Dent Res. 2020;31:305–11.
155. Karan K, Yao X, Xu C, Wang Y. Chemical characterization of etched dentin in non-carious cervical lesions. J Adhes Dent. 2012;14: 315–22.
156. Liu KL, Zhang XF, Wei X. [Influence of different acid etching modes on bond strengths to non-carious sclerotic dentin]. Shanghai Kou Qiang Yi Xue. 2016;25:38–41. Chinese. PMID:27063306
157. Velasquez LM, Sergent RS, Burgess JO, Mercante DE. Effect of placement agitation and placement time on the shear bond strength of 3 self-etching adhesives. Oper Dent. 2006;31:426–30.
158. Zander-Grande C, Amaral RC, Loguercio Ad, Barroso Lp, Reis A. Clinical performance of one-step self-etch adhesives applied actively to cervical lesions: 24-month trial. Oper Dent. 2014;39: 228–38.
159. Amaral RC, Stanislawczuk R, Zander-Grande C, Gagler D, Reis A, Loguercio AD. Bond strength and quality of the hybrid layer of one-step self-etch adhesives applied with agitation on dentin. Oper Dent. 2010;35:211–9.
160. Chan K-M, Tay FR, King NM, Imazato S, Pashley DH. Bonding of mild self-etching primers/adhesives to dentin with thick smear layers. Am J Dent. 2003;16:340–6.
161. Reis A, Pellizzaro A, Dal-Bianco K, Gomes OM, Patzlaff R, Loguercio AD. Impact of adhesive application to wet and dry dentin on long-term resin-dentin bond strengths. Oper Dent. 2007;32: 380–7.
162. Loguercio AD, Raffo J, Bassani F, Balestrini H, Santo D, Do Amaral RC, et al. 24-month clinical evaluation in non-carious cervical lesions of a two-step etch-and-rinse adhesive applied using a rubbing motion. Clin Oral Investig. 2011;15:589–96.
163. Dal-Bianco K, Pellizzaro A, Patzlaft R, De Oliveira Bauer JR, Loguercio AD, Reis A. Effects of moisture degree and rubbing action on the immediate resin-dentin bond strength. Dent Mater. 2006;22:1150–6.
164. Cieplik F, Scholz KJ, Tabenski I, May S, Hiller K-A, Schmalz G, et al. Flowable composites for restoration of non-carious cervical lesions: results after five years. Dent Mater. 2017;33:e428–37.
165. May S, Cieplik F, Hiller K-A, Buchalla W, Federlin M, Schmalz G. Flowable composites for restoration of non-carious cervical lesions: three-year results. Dent Mater. 2017;33:e136–45.
166. Zhang H, Wang L, Hua L, Guan R, Hou B. Randomized controlled clinical trial of a highly filled flowable composite in non-carious cervical lesions: 3-year results. Clin Oral Investig. 2021;25:5955–65.
167. Szesz A, Parreiras S, Martini E, Reis A, Loguercio A. Effect of flowable composites on the clinical performance of non-carious cervical lesions: a systematic review and meta-analysis. J Dent. 2017;65:11–21.
168. Trieu A, Mohamed A, Lynch E. Silver diamine fluoride versus sodium fluoride for arresting dentine caries in children: a systematic review and meta-analysis. Sci Rep. 2019;9:2115. https://doi.org/10.1038/s41598-019-38569-9
169. Seifo N, Robertson M, Maclean J, Blain K, Grosse S, Milne R, et al. The use of silver diamine fluoride (SDF) in dental practice. Br Dent J. 2020;228:75–81.
170. Hiraishi N, Sayed M, Takahashi M, Nikaido T, Tagami J. Clinical and primary evidence of silver diamine fluoride on root caries management. Jpn Dent Sci Rev. 2022;58:1–8.

How to cite this article: Goodacre CJ, Eugene Roberts W, Munoz CA. Noncarious cervical lesions: Morphology and progression, prevalence, etiology, pathophysiology, and clinical guidelines for restoration. J Prosthodont. 2023;32:e1–e18. https://doi.org/10.1111/jopr.13585