self CDE 2 HOURS CREDIT instruction

Proposal for clinical classification of multifactorial noncarious cervical lesions

Kapil Loomba, BDS, MDS, FPFA ▪ Rhythm Bains, BDS, MDS ▪ Vivek Kumar Bains, BDS, MDS ▪ Anju Loomba, BDS, MDS

Noncarious cervical lesions often present with overlapping symptoms and have a multifactorial etiology. The dimensions of the lesion axially (depth) and occlusogingivally (width), along with the orientation of the walls of the lesion to each other (angle), are the 3 factors that can be quantified to classify the lesion and assess its progress over time. This article analyzes these factors and proposes a simple classification system based upon the clinical appearance of the lesion in order to derive simple, therapeutic, treatment-based classifications.

Received: January 22, 2013
Accepted: May 7, 2013

Keywords: abrasion, abfraction, erosion, NCCLs

A noncarious cervical lesion (NCCL) is defined as the loss of tooth structure at the cementoenamel junction (CEJ), which is unrelated to dental caries, while the terms *tooth surface loss* or *tooth wear* refer to any pathological loss of tooth tissue by a disease process other than dental caries. These lesions have a multifactorial etiology and often present diagnostic and restorative challenges for the dental profession.[1-3]

The NCCLs in human teeth have been conventionally and broadly classified according to their etiology and pathogenesis as: *abrasion, erosion,* and *tooth flexure*.[4] In 1991, Grippo described tooth flexure as *abfraction*, which is a pathological loss of dental hard tissue caused by biomechanical forces.[5] Historically, these NCCLs were classified according to their appearance as *wedge-shaped, disc-shaped, flattened, irregular,* or *smooth*.[6]

Abrasion refers to a loss of tooth structure due to friction between an exogenous agent and the tooth. This phenomenon is termed *masticatory* abrasion when the teeth are worn on the occlusal or incisal surfaces by friction from the food bolus.[7] When tooth structure is lost on the buccal and lingual surfaces due to coarse food pressing against these surfaces via the actions of the tongue, lips, and cheeks during mastication, it is known as *ablation*.[8] Various etiological factors related to abrasion are overzealous brushing, use of dentifrices that have high abrasive indices, improper use of dental floss/toothpicks, and habits such as nail biting or pipe smoking.[4]

Erosion refers to the loss of tooth surface by chemical or electrochemical actions not involving bacteria.[9] According to the *American Society for Metals,* erosion is defined as the abrasive destruction of materials by the movement of liquids or gas, with or without solid particles; whereas corrosion is defined as the physical deterioration of a material by chemical or electrochemical agents.[10] Thus, the term *corrosion* more closely applies to the tooth wear phenomenon in the oral cavity.[11]

Abfraction mainly presents at a subgingival location, with a sharp wedge-like morphology that cannot be explained by the etiological factors of toothbrush abrasion, tissue ablation, or erosion. From the engineering model proposed by Lee & Eakle and McCoy, these lesions can be attributed to lateral occlusal forces generated during mastication and bruxism, both of which can cause a flexure or bending of the tooth.[12,13] The resultant force generated at the cervical fulcrum area causes microfractures of the crystalline enamel and dentin, with a subsequent loss of tooth structure due to secondary factors such as acid or abrasion.[12]

NCCLs sometimes present with an overlapping clinical picture. Grippo & Simring described these overlapping lesions as erosion-corrosion, abrasion-corrosion, abrasion-abfraction, and biocorrosion-abfraction.[11] Various studies have found a strong correlation between occlusal and cervical pathology, thus a lesion combining cervical lesions and attrition can be considered.[5]

In some cases, the initial appearance of an abrasion lesion is that of a small, horizontal groove across the buccal (in rare cases, lingual) surface of the crown, near the CEJ. The later stages present a V-shaped lesion as the surrounding walls of this lesion meet at an acute angle axially, making the appearance similar to abfraction lesions, which are sharp and wedge-shaped. This lesion may occur on a single tooth.[14] Many cases present with concomitant occlusal facets.[15] The underlying dentin in both abrasion and erosion lesions is shiny; however, in the cases of erosion, the lesion is less demarcated from the surrounding dentin.[4]

Classification

Etiological factors are important for planning a treatment protocol, but they are more relevant in the preventive phase of the treatment plan. The real-time appearance of a lesion that the patient presents at the clinic becomes more important in deciding both the treatment procedure and which restorative material to use. The dimensions of the lesion: axially (depth, D), occlusogingivally (width, W), and the orientation of the walls of the lesion to each other (angle, A) are the factors that can be quantified to classify the lesion and check its progress over time. However, a review of the literature reveals that there is to date no orderly, scientific classification for NCCLs that can be used as a communication tool in clinics and among health personnel. Thus, there is a need for a therapeutic classification based on the clinical appearance of the lesion.

The longevity and clinical performance of any restoration depends on the restorative material used, the clinical acumen of the practitioner, and the dimensions of the cavity. The choice of the restorative material used is largely dictated by the amount of tooth structure to be replaced, esthetic concerns, the remaining dentin thickness or proximity to the pulp, as well as the buccolingual and occlusogingival

outline of the cavity.[16] In the cervical lesions, additive factors of tooth flexure (abfraction), parafunctional occlusal habits, and the abrasive action of toothbrushes, dentifrices, or any other external agent have to be considered. The authors propose grading the clinical severity of the cervical lesion on the basis of cavity dimensions so that a treatment need-based approach can be taken. The clinical parameters taken for grading are depth, angle, and width (DAW): depth of the lesion, angle of the lesion, and the occlusogingival width of the cavity (Table).

Table. Depth, angle, and width (DAW) classification for cervical lesions.

Score	Buccolingual depth of the lesion (D)	Shape and angle of the lesion (A)	Occlusogingival width of the lesion (W)
1	≤1 mm	Wedge or V-shaped, angle <90 degrees (acute)	≤1 mm
2	>1 up to 2 mm	Saucer-shaped, angle 90 to 135 degrees (obtuse)	>1 up to 2 mm
3	>2 mm	Saucer-shaped, angle >135 degrees	>2 mm

Depth

The depth of any carious or noncarious lesion is inversely proportional to the remaining dentin thickness, and thus an important factor in determining the pulpal status. A clinical confirmation on the depth of a lesion should be made before restoring a cervical lesion. A lesion present in the cervical area near the CEJ may be shallow, moderately deep, or approaching the pulp, depending on the etiology of the lesion. The abrasion of the tooth may be the result of overzealous toothbrushing, using a dentifrice with a high abrasive index, a food bolus, etc. The lesions produced due to brushing are more prominent and deep in the incisor and premolar area than in the molar area.[4] The depth of a lesion produced due to erosive or corrosive action of an acid depends on the length of contact time of the acid with the tooth structure. The symptoms of sensitivity depend on the amount of dentin exposed to the environment, the amount of reparative or sclerotic dentin formed, and the proximity to the pulp.

Buccolingual depth may be measured by placing a calibrated periodontal probe with a rubber stopper. The tip should be placed at the deepest point of the lesion and the stopper at the outer (cavosurface) margin. The depth is then measured from the rubber stopper to the probe tip (Fig. 1). Similarly, an endodontic file with a stopper can also be used to measure the depth of a lesion. The amount of tooth surface loss can be assessed by comparing the tooth with adjacent or contralateral sound teeth. Depending on the proximity to the pulp, the lesions are graded into 3 broad categories on the ordinal scale as ≤1 mm, >1 up to 2 mm, and >2 mm.

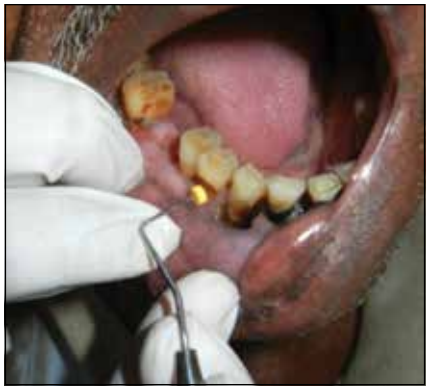

Fig. 1. Use of a calibrated probe to measure the depth of a lesion.

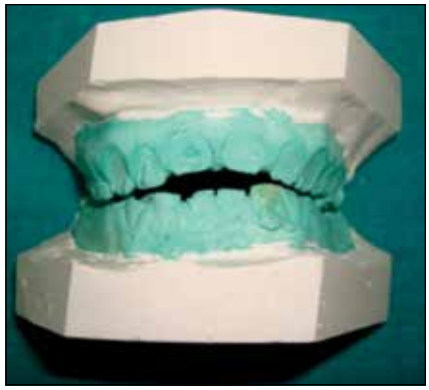

Fig. 2. A diagnostic cast showing noncarious tooth surface loss.

Depth ≤1 mm (D_1)

Shallow, wide lesions ≤0.5 mm generally do not exhibit any symptoms of sensitivity and do not require any restorative treatment. Usually D_1 lesions require only enamel or cementum restorations. The margins of the lesions should be smoothed so that they become non-plaque retentive areas.[17] Application of fluoride varnish, casein phosphopeptide-amorphous calcium phosphate topical paste for remineralization of the lost enamel surface, or a desensitizing agent such as GLUMA Desensitizer (Heraeus Kulzer) are all generally indicated to reduce patient discomfort and increase caries resistance of the affected area.

If the lesion is approximately 1 mm, then it may require restoration. If there are any symptoms of sensitivity, the area should be first desensitized with repeated fluoride varnish applications, desensitizing agents, or electrophoresis. The affected area is then restored with a glass ionomer cement (GIC) or a flowable composite resin. A liner or calcium hydroxide beneath this restorative material is generally not required for shallow cavities. Historically, gold or amalgam restorations were used for these lesions, but they require a definite cavity preparation for retention and resistance, and they have lost popularity due to economic and esthetic concerns.[18] Both a GIC or a flowable composite resin restoration require minimum tooth preparation, although conditioning the enamel or cementum surface with a mild acid may be helpful in retaining the restoration.

Targeting the etiology and taking steps for the prevention of NCCLs is of the utmost importance. Prevention may include changing the toothbrush or dentifrice, changing brushing habits, dietary counseling, or treating any indication of acid reflux.[4] It is always advisable to prepare study casts and take photographs as a reference baseline to check for the progress of the disease, especially when

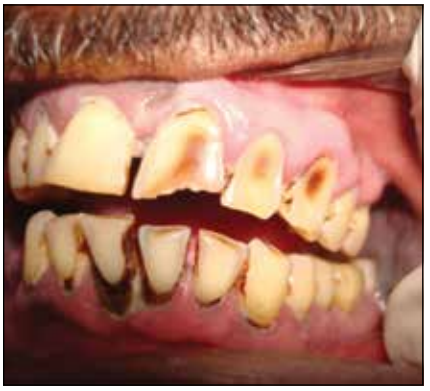

Fig. 3. D_1 A_3 W_3 classification according to the DAW system. Abbreviations: D_1, buccolingual ≤1 mm; A_3, saucer-shaped, angle >135 degrees; W_3, occlusogingival width >2 mm.

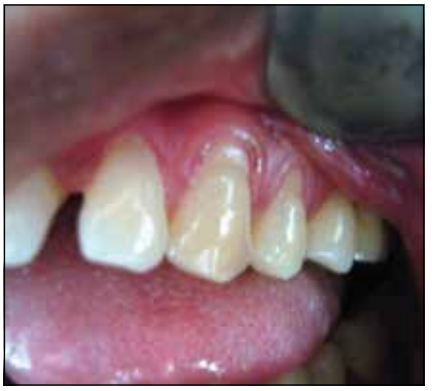

Fig. 4. D_2 A_2 W_2 classification according to the DAW system. Abbreviations: D_2, buccolingual depth >1 up to 2 mm; A_2, saucer-shaped, angle 90 to 135 degrees; W_2, occlusogingival width >1 up to 2 mm.

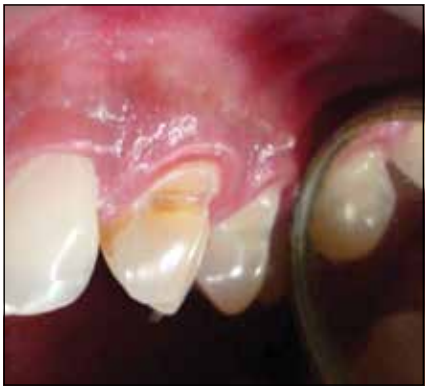

Fig. 5. D_3 A_1 W_2 classification according to the DAW system. Abbreviations: D_3, buccolingual depth >2 mm; A_1, wedge-shaped, angle <90 degrees; W_2, occlusogingival width >1 up to 2 mm.

the underlying etiology is of erosion, as even restorative materials undergo erosion (Fig. 2).[19] The D_1 lesion usually has a shallow angle (90 to >135 degrees), and the occlusogingival width usually varies from 1 to >2 mm (Fig 3).

Depth >1 up to 2 mm (D_2)

Moderately deep D_2 lesions in the cervical area may have symptoms of sensitivity to sweets, cold, or air. However, the lesion may be symptom-free due to sclerotic dentin. Such sclerotic dentin may have a dark yellow-brown color. Dentin from NCCLs is pathologically changed and differs in composition and morphology from unaffected dentin.[20] These D_2 lesions are restored with a GIC or a composite resin after applying resin-modified GIC or flowable composite resin as a liner. Sclerotic dentin is generally resistant to bonding, thus conditioning of the surface with mild acids or roughening the enamel might be necessary before applying the GIC. The flowable composite resin or GIC are more flexible materials and act as a stress breaker beneath the less flexible composite resins. The GIC is the preferred liner for slightly deeper lesions as it is less harsh to the sensitive pulp.[21] Prevention of the underlying etiology by counseling and change in habits is a must in these cases also. The D_2 lesion usually has a shallow angle (90-135 degrees); the occlusogingival width usually varies from >1 up to 2 mm (Fig. 4).

Depth >2 mm (D_3)

These lesions may cause pain or sensitivity that lingers after removal of stimulus. In such cases, if the lesion is determined to be near the pulp, a calcium hydroxide liner is advocated, followed by a resin-modified GI or a flowable composite resin and then a definite composite resin restoration. These deeper lesions generally have an acute angle of ≤90 degrees. The etiology of these wedge-shaped deep lesions may be related to severe abrasive action, but sometimes these lesions are isolated, without any attributable etiology, and may be associated with signs of abfraction (Fig. 5).[6] The occlusal adjustment and correction of any parafunctional habits are of primary importance in these cases. Root canal treatment (RCT) is required in cases of acute or chronic irreversible pulpitis. Care has to be taken while preparing the access cavity and shaping the canals so that no inadvertent perforation of the pulp chamber or canal may occur. A full coverage crown is advocated after an RCT. If any signs of occlusal wear are present, care should be taken not to place ceramic on the occlusal surface; instead, a metal crown with buccal veneer is recommended. In cases where the lesion is very deep, the remaining tooth structure at the CEJ area will be very thin, thus making it prone to fracture at the neck. In these cases, the restorability of the tooth becomes questionable, and this prognosis should be explained to the patient.

Angle

The angle refers to the junction formed between the occlusal and cervical walls of the lesion. A deeper lesion will generally have an acute angle, compared to flatter or saucer-shaped lesions which have an obtuse angle. The wear associated with the phenomenon of abfraction and abrasion will have an acute angle, while erosive lesions will exhibit an obtuse angle.

The angle can be measured clinically by first placing silver cones or thin orthodontic wires adapted to the inner surface of cervical and incisal walls, and then measuring the angle (Fig. 6 and 7). A stone replica of the area can also be used. After visually inspecting the vertical buccolingual cross section, the angle can be categorized on an ordinal scale as <90 degrees, 90-135 degrees, or >135 degrees. Impressions taken in rubber-based materials and study casts can verify the precision of the angle measured.

Wedge-shaped, acute angle <90 degrees (A_1)

In cases of deep lesions with an acute angle, extra attention should be given to any occlusal wear patterns and chipped-off occlusal surfaces of old restorations or crowns. Corrective measures for occlusal adjustment and parafunctional habits such as bruxism are prerequisites before any definitive therapy, as the continuous fatigue caused by these flexural forces can cause debonding of the restoration at the dentin-cement joint.[22] A resin-modified GI

or flowable composite resin liner should be placed beneath the composite resin, as both these liners have a low modulus of elasticity and act as a stress breaker, thus preventing the debonding of the restoration.[23] If the lesion is moderately deep (1-2 mm) and in proximity to the pulp, a calcium hydroxide liner is recommended. In cases where the pulp is exposed (lesion is >2 mm) and there are signs of irreversible pulpitis, an RCT is recommended. The occlusogingival width of the lesions usually varies from >1 up to 2 mm (Fig. 5).

Fig. 6. Use of a silver cone to measure the angle of the lesion in an extracted tooth.

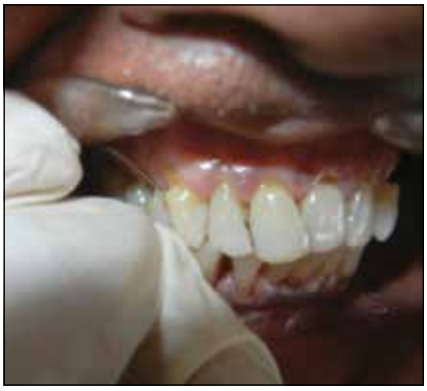

Fig. 7. Clinical use of a silver cone to measure the angle of the lesion.

Saucer-shaped, obtuse angle 90-135 degrees (A_2)

In cases in which the lesion is caused by abrasion that is less in severity, or due to erosive action of any acid of exogenous (such as overconsumption of citrus fruits or aerated drinks, or the long-term chewing of vitamin C lozenges) or endogenous origin, the appearance is shallow, saucer-shaped, and shiny.[4] These usually have a buccolingual depth of >1 up to 2 mm (Fig. 4). The acidic environment may be due to endogenous factors such as any eating disorders or gastroesophageal reflux disorder (GERD).[24] Treatment includes dietary counseling, antacids, and psychological counseling of the patients. Restorative treatment includes a GIC or composite resin restoration. Sometimes conditioning of the surface is required with a mild acid or with the help of burs before GIC placement, as the sclerotic dentin is very smooth and shiny, and does not provide adequate retention to the GIC or composite resin.[25] The occlusogingival width usually varies from >1 up to 2 mm.

Saucer-shaped, obtuse angle >135 degrees (A_3)

Such lesions are mostly associated with erosive action of any acid of exogenous or endogenous origin. These lesions are very shallow; and the buccolingual depth is usually ≤1 mm (Fig. 8). They present with a smooth and shiny surface, and if <0.5 mm, may not require any restoration. A complete history of any eating disorder, GERD, or erratic food habits must be evaluated. Treatment generally includes antacid therapy, psychological counseling, and dietary control to prevent progression of the lesions. If there is any symptom of sensitivity and the lesion is >0.5 mm, desensitizing agents or topical fluoride varnishes and gels may be prescribed. If the lesion is ≤1 mm, a GIC or composite resin restoration may be required.

Width

The occlusogingival width of the lesion depends on the contact area of the abrasive or external agent with the tooth surface and the location of the affected tooth. If the etiology is toothbrushing, the anterior teeth are more severely affected in terms of both area and depth, compared to the posterior teeth. If there is GERD, the palatal surface of anterior teeth and occlusal surfaces of posterior teeth are affected more and over a larger area. It is the maximum vertical distance between the occlusal and gingival walls of the lesion and measured on ordinal scale (≤1 mm, >1 up to 2 mm, and >2 mm).

Width ≤1 mm (W_1)

Lesions of small occlusogingival dimension may be related to any of the etiologies of tooth wear. The treatment is dictated by the depth and angle of the lesion, and a liner may be indicated beneath the GIC or composite resin material (Fig. 9). A small lesion of minimum width and depth may just require desensitizing or remineralizing agents and/or a GIC or composite resin restoration.

Width >1 up to 2 mm (W_2)

The area may be restored using a GIC or composite resin. The use of a liner depends on the depth of the lesion. If the lesion is involving the pulp, RCT may be required. The angle in such lesions is usually 90-135 degrees.

Width >2 mm (W_3)

If the width is >2 mm, secondary retention involving the roughening of the tooth or dentinal grooves may be required for retaining the restoration (Fig. 9). If the depth and occlusogingival width are >2 mm, and the pulpal condition is compromised, the treatment will consist of an RCT and full coverage crown. In cases of increased width, the floor of the lesion might be subgingival and compression of the gingival tissue with the help of vasoconstrictors, astringents, heavy rubber dam, cervical rubber dam clamp, or gingivectomy may be required for a proper restorative procedure. Sometimes open-flap surgery may be required to place a restoration. GIC restorations are generally preferred in subgingival lesions as they are more compatible with soft tissues.

Discussion

NCCLs have a multifactorial etiology and no single mechanism can explain their various characteristics.[26] Various factors associated with these lesions are occlusion, age, sex, occupation, diet, personal habits, salivary flow, systemic health, and parafunctional habits.[4] The clinical presentation of a lesion, its characteristics, and identification of susceptible individuals can serve as a useful guide as to whether intervention is needed, and help predict the progress of the lesion if left untreated.[1]

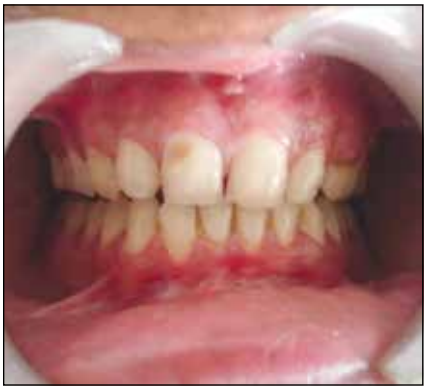

Fig. 8. D_1 A_3 W_2 classification according to the DAW system. Abbreviations: D_1, buccolingual ≤1 mm; A_3, saucer-shaped, angle >135 degrees; W_2, occlusogingival width >1 up to 2 mm.

Fig. 9: Multiple lesions having noncarious tooth surface loss.

Knowledge about an individual's history is crucial for the prevention of these lesions progressing to an advanced stage.[27] Patients with a known history of overzealous brushing or of using a dentifrice with a high abrasive index should be advised about making alterations in their daily oral hygiene routine, such as switching to a more sensitive toothpaste, modifying their brushing habits, and using a soft toothbrush. Unilateral NCCLs found on the left side of the mouth often indicate that the patient is a right-handed individual.[4] Patients with a history of GERD or those who are anorexic or bulemic exhibit shallow, shiny, erosive lesions, especially on the palatal surfaces of maxillary incisors (perimylosis) and buccal surfaces of mandibular molars. The severity of the acid reflux and time of contact of the vomitus with the tooth structure determines the depth or extent of the lesion.[28] Apart from these intrinsic factors, extrinsic factors that can cause dental erosion include consumption of citrus foods, acidic beverages, and effervescent or chewable vitamin C tablets; exposure to acids in chemical and metal industries; and frequent swimming in chlorinated swimming pools.[29-31] Patients with reduced salivary flow and buffering capacity are also more prone to erosive lesions on the teeth.[32] A patient with GERD should follow a modified diet regimen, and antacids may be recommended after consultation with a physician or dietician. Patients suffering from eating disorders frequently need a psychiatric consultation along with dietary counseling. Persons exposed to occupational hazards, such as acidic fumes, should be warned about their adverse effects, and proper ventilation in the working area should be recommended.

Another important factor in the development of wedge-shaped, subgingival, and sometimes isolated lesions is occlusion. Patients with parafunctional habits, bruxism, or high biting forces often exhibit NCCLs in the absence of any other obvious cause.[33] The restorative treatment may fail if occlusal adjustment is not done prior to restoration. Parafunctional habits and bruxism should be treated by occlusal equilibration, an occlusal nightguard, or a bite plane whenever necessary. Stress is an important predisposing factor for bruxism, and antidepressant therapy and psychiatric consultation are other options.[4]

Lesions should be evaluated for dentinal sensitivity before starting the restorative phase. Restorative treatment of the NCCLs generally consists of placement of an adhesive tooth-colored restorative material. Surface treatment or conditioning is a prerequisite for successful adhesion. As reported by Mjor, the surface of these lesions is hypermineralized and thus need an alteration in the surface conditioning protocols.[34,35] In a Raman spectroscopic study of NCCLs, it was noted that a demineralized NCCL has a higher phosphate ion (PO4) to silicon ratio than normal dentin, which indicates that there is more mineral remaining on the surface of an NCCL after conditioning. The surface characteristics of an NCCL after conditioning with a weak acid, such as 20% polyacrylic acid, results in a higher residual mineral content than after using a strong acid etchant such as 37% phosphoric acid. This also implies that using a resin-based adhesive, which depends on hybrid layer formation, would not be able to expose enough of the collagen fibers required for producing a reliable micromechanical adhesion. The most commonly used restorative material for restoration of NCCLs is a GIC. Thus, using a milder acid for surface conditioning and GIC as a restorative material is the preferred treatment.[20,36]

Over the years, a number of reviews and studies have reported the challenges of the restoration of NCCLs, indicating marginal shrinkage, contraction stresses, cavity dimensions, insertion technique, and adhesive systems as the main reasons for failure.[16] Apart from these, abfraction and stress at the cervical area, the sclerotic nature of dentin in these areas, occlusion, and the barreling effect on the tooth from masticatory forces are also major factors that have been proposed as reasons for loss of retention in these areas.[37-40]

The restoration of an NCCL is required in order to strengthen the tooth, decrease stress concentration, check the progress of the lesion, and prevent hypersensitivity. The material used to restore such lesions should have enough elasticity to accommodate bending, as a lower modulus allows the material to flex with the tooth, rather than debond.[12] Some authors have suggested the use of an elastic resin layer between the bonding agent and composite resin to alleviate stresses due to thermal changes, water absorption, and occlusal forces.[23] Care should be taken to restore a cervical abrasion with no visible occlusal facet or coaxial strain, and to use a restoration material that possesses enough abrasive resistance. Besides having ionic (conventional) and micromechanical (resin-modified) bonding, GICs also have the desirable modulus of elasticity and flexibility to endure the squeezing effect experienced by the restorative materials at the cervical area.[12,41] However, because of low abrasive resistance and high solubility, many authors have suggested the use of the sandwich technique

instead of GICs or composite resins alone. This is advantageous for deep cervical lesions, or for lesions that lack enamel in the cervical margins.

Conclusion

These multifactorial lesions with a wide variety of clinical presentations need a very balanced and thorough approach in their treatment. Their clinical management would become easier if a relatively standardized, yet simple and stable restorative approach were adopted. Furthermore, a communicable clinical classification would be a covenient tool by which the general dentist can communicate clear-cut terminologies to express the clinical lesions to their staff and patients.

Author information

Dr. K. Loomba is a professor and head, Department of Conservative Dentistry & Endodontics, Career Post Graduate Institute of Dental Sciences & Hospital, Lucknow, India. Dr. R. Bains is an assistant professor, Department of Conservative Dentistry & Endodontics, Faculty of Dental Sciences, King George's Medical University, Lucknow. Dr. V. K. Bains is a reader, Department of Periodontology, Saraswati Dental College & Hospital, Lucknow. Dr. A. Loomba is a private practitioner, Dental Care Clinic, Lucknow.

References

1. Aw TC, Lepe X, Johnson GH, Mancl L. Characteristics of noncarious cervical lesions: a clinical investigation. *J Am Dent Assoc.* 2002;133(6):725-733.
2. Khan F, Young WG, Shahabi S, Daley TJ. Dental cervical lesions associated with occlusal erosion and attrition. *Aust Dent J.* 1999;44(3):176-186.
3. Osborne Smith KL, Burke FJ, Wilson NH. The aetiology of the noncarious cervical lesion. *Int Dent J.* 1999; 49(3):139-143.
4. Bader JD, Levitch LC, Shugars DA, Haymann HO, McClure F. How dentists classified and treated noncarious cervical lesions. *J Am Dent Assoc.* 1993;124(5):46-54.
5. Grippo JO. Abfractions: a new classification of hard tissue lesions of teeth. *J Esthet Dent.* 1991;3(1):14-19.
6. Bartlett DW, Shah P. A critical review of noncarious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. *J Dent Res.* 2006;85(4):306-312.
7. Grippo JO, Simring M, Schreiner S. Attrition, abrasion and abfraction revisited. *J Am Dent Assoc.* 2004; 135(8):1109-1118; quiz 1163-1165.
8. Solberg WK, Seligman DA. Dental occlusion. In: Carrranza FA & Newman MG, eds. *Clinical Periodontology.* Philedalphia PA: WB Saunders; 1996:173-184.
9. Larsen MJ, Nyvad B. Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effects and contents of calcium phosphate. *Caries Res.* 1999;33(1):81-87.
10. Levy, AV. *Solid Particle Erosion and Erosion-Corrosion of Materials.* Materials Park, OH: ASM International; 2007.
11. Grippo JO, Simring M. Dental 'erosion' revisited. *J Am Dent Assoc.* 1995;126(5):619-620, 623-624, 627-630.
12. Lee WC, Eakle WS. Stress-induced cervical lesions: review of advances in the past 10 years. *J Prosthet Dent.* 1996;75(5):487-494.
13. McCoy G. The etiology of gingival erosion. *J Oral Implantol.* 1982;10(3):361-362.
14. Grippo JO, Masi JV. Role of biodental engineering factors (BEF) in the etiology of root caries. *J Esthet Dent.* 1991;3(2):71-76.
15. Estafan A, Furnari PC, Goldstein G, Hittleman EL. In vivo correlation of noncarious cervical lesions and occlusal wear. *J Prosthet Dent.* 2005;93(3):221-226.
16. Costa Pfeifer CS, Braga RR, Cardoso PE. Influence of cavity dimensions, insertion technique and adhesive system on microleakage of Class V restorations. *J Am Dent Assoc.* 2006;137(2):197-202.
17. Marzuok MA. *Operative Dentistry-Modern Theory and Practice.* Tokyo: Ishiyaku EuroAmerica, Inc; 1997.
18. Blackwell RE. *GV Black's Operative Dentistry.* South Milwaukee, WI: Medico Dental Publishing Company; 1955.
19. Xhonga FA, Wolkott RB, Sognnaes RF. Dental erosion II. Clinical measurement of dental erosion progress. *J Am Dent Assoc.* 1972; 84(3):577-582.
20. Sakoolnamarka R, Burrow MF, Prawer S, Tyas MJ. Raman spectroscopic study of noncarious cervical lesions. *Odontology.* 2005;93(1):35-40.
21. Anusavice KJ. *Phillip's Science of Dental Materials.* St. Louis, MO: Saunders; 2003.
22. Shore NA. *Temporomandibular Joint Dysfunction and Occlusal Equilibration.* Philidelphia, PA: Lippincott; 1976.
23. Van Meerbeek B, Peumans M, Verchueren M, et al. Clinical status of ten dentin adhesive systems. *J Dent Res.* 1994;73(11):1690-1702.
24. Milosevic A, Brodie DA, Slade PD. Dental erosion, oral hygiene, and nutrition in eating disorders. *Int J Eat Disord.* 1997;21(2):195-199.
25. Powis DR, Folleras T, Merson SA, Wilson AD. Improved adhesion of a glass ionomer cement to dentin and enamel. *J Dent Res.* 1982;61(12):1416-1422.
26. Levitch LC, Bader JD, Shugars DA, Heymann HO. Noncarious cervical lesions. *J Dent.* 1994;22(4):195-207.
27. Pecie R. Noncarious cervical lesions - a clinical concept based on the literature review. Part 1: prevention. *Am J Dent.* 2011;24(1):49-56.
28. Ali DA, Brown RS, Rodriguez LO, Moody EL, Nasr MF. Dental erosion caused by silent gastroesophageal reflux disease. *J Am Dent Assoc.* 2002;133(6):734-737, quiz 768-769.
29. Linkosalo E, Markkanen H. Dental erosions in relation to lactovegetarian diet. *Scand J Dent Res.* 1985;93(5): 436-441.
30. Lynch JB, Bell J. Dental erosion in workers exposed to inorganic acid fumes. *Br J Ind Med.* 1947;4(2):84-86.
31. Centerwall BS, Armstrong CW, Funkhouser LS, Elzay RP. Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. *Am J Epidemiol.* 1986;123(4):641-647.
32. He LH, Xu Y, Purton DG. In vitro demineralisation of the cervical region of human teeth. *Arch Oral Biol.* 2011;56(5):512-519.
33. Wood ID, Kassir AS, Brunton PA. Effect of lateral excursive movements on the progression of abfraction lesions. *Oper Dent.* 2009:34(3):273-279.
34. Mjor IA. *Dentin and Dentin Reactions in the Oral Cavity.* Oxford: IRL Press Ltd; 1987.
35. Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The surface effects of erosion and abrasion on dentine and without a protective layer. *Br Dent J.* 2004;196(6): 351-354, discussion 339.
36. de-Melo MA, Passos VE, Alves JI, Barros EB, Santiago SL, Rodrigues LK. The effect of diode laser irradiation on dentin as a preventive measure against dental erosion: an in vitro study. *Lasers Med Sci.* 2011;26(5): 615-621.
37. Vandewalle KS, Vigil G. Guidelines for the restoration of class V lesions. *Gen Dent.* 1997;45(3):254-260, quiz 265-266.
38. Marshall GW Jr, Chang YJ, Saeki K, Gansky SA, Marshall SJ. Citric acid etching of cervical sclerotic dentin lesions: an AFM study. *J Biomed Mater Res.* 2000; 49(3):338-344.
39. Lambrechts P, Braem M, Vanherle G. Buonocore memorial lecture. Evaluation of clinical performance for posterior composite resins and dentin adhesives. *Oper Dent.* 1987;2(2):53-78.
40. Heymann HO, Sturdevant JR, Brunson WD, Wilder AD, Sluder TB, Bayne SC. Twelve-month clinical study of dentinal adhesives in class V cervical lesions. *J Am Dent Assoc.* 1988;116(2):179-183.
41. Hanoaka K, Nagao D, Mitusi K, Mitsuhashi A, Sugizaki S, Teranaka T. A biomechanical approach to the etiology and treatment of noncarious dental cervical lesions. *Bull Kanagawa Dent Coll.* 1998;26(2):103-111.

Manufacturer

Heraeus Kulzer, South Bend, IN
800.435.1785, www.heraeus-dental-us.com