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QUESTION:

Non-carious cervical lesions: Does occlusion affect their formation and treatment?

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Dr. Abrahamsen

Scientific research begins with a question. Next, a historical search is required to find any evidence that may have already answered the question. The non-carious loss of tooth structure (NCLTS) was first mentioned in the dental literature in a text of dental history by Dr. J. Hunter in 1728. True scientific investigation began in the late 19th century after the etiology of dental decay (caries) was solved. The dental researchers then were most curious about the NCLTS at the cervix of teeth, which they named the "mysterious non-carious cervical lesion (NCCL)."

W. D. Miller, MD, PhD, ScD, an American born and dental educated scientist, spent his entire professional life at the University of Berlin trying to discover the etiology of dental decay, and upon solving that, the etiology of the NCCL. He accomplished both. His last three publications in 1907 reveal his exhaustive study regarding the etiology of the NCCL, testing every hypothesis proposed at that time. His experimentation with abrasives and acids clearly proved that the formation of the NCCL could only be reproduced with toothpaste.

His research also revealed that the toothbrush did not contribute to the loss whatsoever, merely the shape. In 1908, Dr. G.V. Black published his text on the pathology of the hard tissues of teeth, *A Work On Operative Dentistry*. In his chapter, "The Erosion of Teeth," Dr. Black tells of his trip in 1906 to Berlin to examine Dr. Miller's findings. He states: "Dr. Miller's reproduction of the NCCL with toothpaste is genuinely what I observe in the mouth (in vivo), including dish-shaped, wedge-shaped, and the flattened varieties." Even with this hands-on evidence, Dr. Black, who never conducted any research on the NCCL but freely offered his opinion, continues with: "However, I am not convinced about the toothpaste. Dentistry must expect the causes of *erosion* will eventually be found in the body fluids giving oral secretions favoring these results."

There has been no reproduction of the NCCL by any hypothesis other than from horizontal brushing with toothpaste. First accomplished by Dr. Miller in 1907, it was repeated again in 2006 by Drs. Abrahamsen and Dzakovich in their reproduction of Dr. Miller's research with modern-day materials.

The continuing promulgation of the flexure/abfraction hypothesis is simply being irresponsible or oblivious of the dental scientific literature or being self-serving by hiding it from dentistry and the public.

The scientific proof presented can only mean that occlusion does not affect the formation of NCCLs nor does it have any role in the subsequent treatment of these lesions. The popular belief that there is an occlusal component in the etiology of the NCCL is an unproven hypothesis with no bioengineering logic to ever have a chance to be reproduced. Inconsistent coincidental findings prove nothing. Yet, dentistry blindly holds fast to the validity without examining the lineage of its belief; this nonsensical hypothesis keeps our profession hostage from the correct simple treatment of this increasingly more common pathological lesion.

Dr. Grippo

The etiology of non-carious cervical lesions (NCCLs) is a combined, or more commonly, a multifactorial condition. There are three mechanisms that contribute to the etiology of NCCLs: stress, friction, and bio-corrosion. The effects of stress manifested as abfraction and occurs through the dynamics of both static and cyclic occlusal loading. Static loading occurs 1,000,000 times per year during swallowing contacts that occur about 1,500 times a day. This repeated loading—in addition to that which occurs during function and parafunction—has effects on both the teeth and their supporting structures. The effects of occlusal loading are most intense in the cervical region where the stress is concentrated, thus causing cervical dentin hypersensitivity, cementum cracking, attachment loss, gingival recession, and NCCLs.

Important co-factors in the etiology of NCCLs are bio-corrodents such as exogenous and endogenous acids as well as proteolytic agents, pepsin from the stomach, and trypsin from the pancreas, working synergistically to degrade the highly organic (30%) exposed dentin. Studies have shown that the mechanisms of both static stress and fatigue (cyclic) stress bio-corrosion increase hard tissue loss in areas of stress concentration. NCCLs are found on teeth with premature and eccentric occlusal contacts where the stress is intensified to the cervical region in the presence of acidic and proteolytic bio-corrodents.

NCCLs as well as root caries are frequently seen on the facial surface as there is 5 times more saliva buffering the lingual area than the facial vestibule. Cervical enamel is more vulnerable to tension than the elastic dentin and can readily abfract when undermined at the cementoenamel junction (CEJ). Enamel has the ability to remineralize more rapidly than dentin after acidic degradation.

Interocclusal loading of cusped teeth are more susceptible to cervical lesions in contradistinction to the primitive humans with severe abrasion (flattened occlusal surfaces). Worn teeth have a shorter crown-to-root ratio, thus minimizing stress to the cervical area. Their group function distributes stress uniformly over both dental arches. Only those populations with abraded occlusions which had an acidic diet had instances of NCCLs.

The mechanism of friction as a cofactor in the formation of NCCLs occurs during toothbrush/dentifrice abrasion, which removes plaque and softened dental hard tissue. If plaque is not removed, root caries will occur and rapidly progress. Friction also occurs from the swishing of acidic beverages during the action of erosion/bio-corrosion.

NCCLs are commonly treated with microhybrid resin-based composites but can debond after repeated interocclusal loading. This retentive failure lends credence to the role of stress from occlusal forces in the etiology of NCCLs. Clinicians must correct eccentric and premature contacts prior to Class V restorative treatment to enhance the retention of their composites. Establishing and monitoring occlusion to achieve maximum intercuspal position (MIP) in centric relation (CR) will increase the longevity of NCCL restorations.

Studies have shown that stress from occlusal loading forces combined with biocorrodents contribute to the etiology of NCCLs. The patient's occlusion must also be addressed prior to and in the treatment of NCCLs.

Dr. Singh

The prevalence of NCCLs on the posterior teeth is an indication of altered occlusal stress and may be evident by the wear facets. In the normal class I

occlusion, the abnormal lateral tensile and horizontal occlusal forces exerted on the teeth increase the flexure movement and break the chemical bond in the weakest part of the tooth, ie, the cervical area. Along with other synergistic factors, eg, erosion and abrasion, more loss of tooth surface results in the formation of cervical lesions, known as abfractions. Depending on the degree of occlusal stress and extent of flexural movement, the lesions will vary in axial depths, occlusal–gingival widths, angulations, and are usually smooth. Moreover, finding of V-shaped lesions adjacent to missing teeth, especially on the premolar teeth, indicate the deleterious occlusal tensile stresses. Also, if the tooth has an occlusal restoration with more resistant or different co-efficients of tensile and shear forces, the tooth will have a higher tendency to exhibit NCCLs on buccal surfaces.

Dentists restore NCCLs with resin-modified glass ionomer, resin-modified liner/base glass ionomer with composite overlay, or enamel/dentin bonding agents with resin composite to ameliorate sensitivity, protect the pulpal and tooth integrity, and, at times, correct occlusal disharmony. Those restorations often debond during the peak shear and stress at the tooth–material interface. To avoid premature failure, apart from proper moisture control, occlusal harmony has to be reestablished by restoring the missing adjacent teeth for re-distribution of the forces, occlusal adjustments including excursive contacts, and the treatment of deleterious parafunctional habits by the use of occlusal splints, etc.

Dr. Winter

For the restorative dentist, NCCLs raise a red flag as we try to ascertain whether these lesions are caused by corrosion, abrasion, attrition, occlusal traumatism (abfraction), or some combination of these individual causes. These lesions are multi-factorial in nature and are difficult to study in vitro. Tooth wear can occur at the cervical area from cyclic occlusal loading, and studies have shown that enamel fractures occur at the CEJ after 200,000 cycles or approximately two and a half months of chewing. The introduction of abrasive elements and changes in pH can contribute to further dissolution of tooth structure.

A second cause of wear can be found with eccentric forces such as premature contacts, bruxism, and clenching. These stressors can be force multipliers that add to the odontolytic effects. Thirdly, we look at abrasion as a cause of tooth structure loss and we can see how poor brushing technique, hard bristles, and parafunctional habits can lead to loss of tooth structure. Most of us have probably had patient who think that straight baking soda is better than toothpaste.

Lastly, corrosion (formerly called erosion) has been discussed as a source of tooth structure dissolution both from exogenous sources, such as sucking lemon drops, or endogenously, such as with GERD or bulimia, resulting in gastric acid reflux that can lead to NCCLs.

In my practice, I will assess these lesions by evaluating these components. I will identify a patient's habits, medications (which can be acidic or corrosive or could contribute to decreased saliva), and other corrosive exogenous sources such as soda habits and candy.

Then I will perform a thorough occlusal analysis to evaluate whether there are parafunctional habits such as nocturnal bruxism or occlusal prematurities and excursive interferences resulting in fremitus.

After treating the lesion directly, occlusal adjustment may be performed. Equilibration will centralize and equalize contacts, decrease fremitus and excursive interference, and, whenever possible, create canine-protected occlusion.

Mouthguards and splints are fabricated and used to optimize occlusal constructs and decrease force factors on the stomatognathic system prior to finalization of the patient's treatment plan.

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