No Carious Cervical Lesions: Abfraction

Sumanth M Shetty[1], Rashmi G Shetty[2], Sudha Mattigatti[3], Noopur A Managoli[4], Surabhi G Rairam[5], Ashwini M Patil[6]

[1]Reader, Department of Pedodontics and Preventive Dentistry, SGT Dental College Hospital & Research Institute, Budhera, Gurgaon, Haryana, India; [2]Assistant Professor, Department of Conservative Dentistry and Endodontics, SGT Dental College Hospital & Research Institute, Budhera, Gurgaon, Haryana, India; [3]Associate Professor, Department of Conservative Dentistry and Endodontics, School of Dental Sciences, Krishna Institute of Medical Sciences University, Karad, Maharashtra, India; [4]Post Graduate Student, Department of Oral Pathology and Microbiology, Dr. D. Y. Patil Vidyapeeth, Dr. D. Y. Patil Dental College & Hospital, Pimpri, Pune, Maharashtra, India; [5]Lecturer, Department of Conservative Dentistry and Endodontics, HKE Society's S Nijlingappa Institute of Dental Sciences & Research, Gulbarga, Karnataka, India; [6]Lecturer, Navodaya Dental College & Hospital, Raichur, Karnataka, India.

ABSTRACT

Abfraction or Theory of Abfraction is a theory explaining the non-carious cervical lesions (NCCL). It suggests that they are caused by flexural forces, usually from cyclic loading; the enamel, especially at the cementoenamel junction (CEJ), undergoes this pattern of destruction by separating the enamel rods. Clinical aspect importance of these ineart lesions are at most important to be detected for early intervention and treatment modalities as options during the progression of the disease.

Key Words: Abfraction, cervical lesions, non-carious lesion, wear, wasting diseases.

How to cite this article: Shetty SM, Shetty RG, Mattigatti S, Managoli NA, Rairam SG, Patil AM. No Carious Cervical Lesions: Abfraction. *J Int Oral Health 2013; 5(5):142-5.*

Source of Support: Nil **Conflict of Interest:** None Declared

Received: **18th June 2013** Reviewed: **19th July 2013** Accepted: **25th August 2013**

Address for Correspondence: Dr. Sumanth M Shetty. Department of Pedodontics and Preventive Dentistry, SGT Dental College Hospital & Research Institute, Budhera, Gurgaon, Haryana, India. Phone: +91 – 8805615167. Email: drsumanthshetty@gmail.com

Introduction

Abfraction means 'to break away'[1] and the term is derived from the Latin words 'ab,' or away, and 'fractio,' or breaking by J. O. Grippo.[2-3] It is usually observed on the buccal surface at the cementoenamel junction (CEJ) of teeth, with prevalence ranging from 27 to 85%.[3] These lesions vary from shallow grooves to broad dished-out lesions or large wedge-shaped defects with sharp internal and external line angles.[4-5]

History

The term 'abfraction' evolved from the work by McCoy[6](1982), Lee and Eakle[7] (1984), and J. O. Grippo[2] (1991). It describes a theoretical process according to which occlusal forces create stresses in enamel and dentin along the cervical area and predispose it to erosion and abrasion. In the early 1980s, McCoy[6-7] questioned the role of toothbrush abrasion in the etiology of what previously had been referred to as "cervical erosion." Thus McCoy[8-9], and in the early 1990's, Grippo[10] proposed that bruxism may be the primary cause of angled notches at the CEJ.

Grippo[10] concluded that the flexure resulted in damage to the enamel rods at the CEJ resulting in their loosening and consequent flaking away of the tooth structure. He named this type of damage 'abfraction'in his paper published in 1991. He suggested that abfraction is the basic cause of all NCCLs, whereas Lee and Eakle[8] proposed a multifactorial etiology, with a combination of occlusal stress, abrasion, and erosion.

Spranger[11] supported the multifactorial etiology of the cervical lesions and suggested that the wear was related to the anatomy, the distribution of forces calculated from elastic deformation studies, development of caries, and occlusion and parafunction.

Grippo[12] has defined abfraction as the pathological loss of tooth substance caused by biomechanical loading forces that result in flexure and failure of enamel and dentin at a location away from the loading. He first used the term abfraction to refer to a process of cervical tooth structure loss, based on work completed by McCoy[7] and Lee and Eakle.[8]

Theory of Abfraction

The theory of abfraction is based primarily on engineering analyses that demonstrate theoretical stress concentration at the cervical areas of teeth[13]. Few controlled studies demonstrate the relationship between occlusal loading and abfraction lesions. The role of occlusal loading in NCCLs appears to be part of a multifactorial event that may not necessarily follow the proposed classic abfraction mechanism.

Nearly all the research on the relationship of occlusal forces (bruxing) to cervical lesions shows that teeth do, indeed flex in the cervical region under bruxing loads, but none seems to cite actual damage caused by this deformation without an abrasive or erosive component applied as well. Nevertheless, the abfraction theory argues that bruxing forces alone can cause the erosion of the tooth structure on buccal surface, especially in the cervical region.

Many dispute the theory of abfraction, blaming this type of damage on what is commonly called "toothbrush abrasion".[8] This harks back to the early work of W.D. Miller in 1917, however it has been confirmed by more recent studies by T.C. Abrahamsen[14] which have shown that toothpaste (not the toothbrush) is abrasive enough to cause this type of damage if the patient is too aggressive in brushing the teeth in a very hard and vigorous "sawing" motion. Abrahamson suggests that the term "toothbrush abrasion" be replaced with the term "toothpaste abuse".[8,14]

His studies using mechanical "tooth brushing" machines have shown that the toothbrush alone does not cause this type of tooth damage, but the addition of toothpaste to the bristles does. Toothbrushes without toothpaste do cause soft tissue damage and indeed, overly vigorous tooth brushing without toothpaste leads to gingival recession.[14]

Grippo[2] has suggested that abfraction is the basic cause of all NCCLs. There is some evidence supporting the tooth flexure theory: presence of class V non-carious lesions in some teeth but adjacent teeth (not subjected to lateral forces) are unaffected;[12-13] the lesions progress around restorations that remain intact and under the margins of complete crowns;[12] the lesions are rarely seen on the lingual aspect of mandibular teeth.[13] However, other studies have proposed a combination of occlusal stress, parafunction, abrasion, and erosion in the development of lesions, leading to a conclusion that the progression of abfraction may be multifactorial.[11,15] Thus the theory of abfraction is not yet proven.

Clinical Features

Abfraction lesions present primarily at the cervical region of the dentition and are typically wedge-shaped, with sharp internal and external line angles. Subgingival lesions have also been observed.In theory, the shape and size of the lesion are dictated by the direction, magnitude, frequency, duration and location of forces that arise when teeth come in contact.[16]

Lee and Eakle[8] first described the characteristics of the lesions resulting from tensile stresses. They concluded that an abfraction lesion should be located at or near the fulcrum in the region of greatest tensile stress concentration, be wedge-shaped, and display a size proportional to the magnitude and frequency of tensile force application.

They proposed that the direction of the lateral forces acting on a tooth determines the location of the lesion. Two or more lateral forces result in an NCCL composed of two or more overlapping wedge-shaped NCCLs. Abfraction is postulated to be responsible for chronic sensitivity of the teeth to cold foods and liquids.[8,17]

Tooth Wear Index proposed by Smith and Knight[18] is the most accepted index to categorize tooth wear in the cervical region and it is as follows:

The classifications on this index are as follows:

0 = no change in contour;
1 = minimal loss of contour;
2 = defect < 1 mm deep;
3 = defect 1 mm to 2 mm deep;
4 = defect > 2 mm deep, or pulp exposure, or exposure of secondary dentin

Treatment

Determination of activity of abfraction lesion can be done by using 12 scalpel blade. Loss of scratch made by the blade signifies active abfraction lesion. In an attempt to reproduce the phenomenon of stress distribution in teeth and their anatomic support structures, a variety of methodologies have been used.The engineering studies cited by McCoy[19] and Lee and Eakle[15] employed finite elemental analysis (FEA), or photoelastic methods. They used computerized geometric or plastic models, respectively. By using FEA, each factor can be rapidly modified and the stress distribution can be investigated in two-dimensional (2D) or three-dimensional (3D) models.[19-20]

When abfraction lesion is less than 1mm in depth[17], only monitoring at regular intervals is enough. Restoring NCCLs improves the maintenance of oral hygiene by the patient. It also helps in decreasing thermal sensitivity, improving esthetics and strengthening the teeth. Along with restoration, a variety of treatment strategies have also been proposed like occlusal adjustments, occlusal splints, elimination of parafunctional habits,[21] altering toothbrushing techniques etc.

For restoring abfractions, many materials and techniques have been tried till date. The following materials are indicated for restoring the lesions: Glassionomer cements (GICs), Resin-Modified GICs (RMGICs), Polyacid-modified resin-based composites (compomers), composites resins and a combination of the techniques.[22-24] According to Tay[25], RMGIC should be the first preference. RMGIC/ GIC liner or base with resin composite should be used wherever aesthetics is concerned. Matis et al[26] found that retention was same for GIC and microfilled resin. GICs have been found to perform better than the composites because of their greater resilience allowing the material to flex with the tooth. RMGICs give better esthetic results than conventional GIC.

References

1. Braem M, Lambrechts P, Vanherle G. Stress-induced cervical lesions. J Prosthet Dent 1992;67(5):718-22.
2. Grippo JO. Abfractions: A New Classification of Hard Tissue Lesions of Teeth. J Esthet Dent 1991;3(1):14-9
3. Litonjua LA, S Andreana, Bush PJ, Tobias TS, Cohen RE. Non carious cervical lesions and Abfractions: A Re-evalution. J Am Dent Assoc 2003;134(7);845-50.
4. Levitch LC, Bader JD, Shugars DA, Heymann HO. Non-carious cervical lesions. J Dent 1994;22(4):195-207.
5. Barttlet 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(4):306-12.
6. Pereira AV, Poiate IA, Poiate-Junior E, Miranda-Junior WG. Abfraction lesions reviewed: current concepts. RGO 2008;56(3);321-6.
7. McCoy G. The etiology of gingival erosion. J Oral Implantol 1982;10(3):361–2.
8. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. J Prosthet Dent 1984;52(3):374–80.
9. McCoy G. On the longevity of teeth. J Oral Implantol 1983;11(2);248-67.
10. Grippo JO. Tooth flexure. J Am Dent Assoc 1991;122(7):13.
11. Spranger H. Investigation into the genesis of angular lesions at the cervical region of teeth. Quintessence Int 1995;26(2):149-54.
12. Grippo JO. Noncarious cervical lesion the decision to ignore or restore. J Esthet Dent 1992;4(Suppl):55-64.
13. Vasudeva G, Bogra P. The effect of occlusal restoration and loading on the development of abfraction lesions: A finite element study. J Conserv Dent 2008;11(3):117-20.
14. TC Abrahamsen. The worn dentition – pathognomonic patterns of abrasion and erosion. Int Dent J 2005; 55(4):268-76.

15. Lee WC, Eakle WS. Stress-induced cervical lesions: review of advances in the past 10 years. J Prosthet Dent 1996;75(5):487-94.
16. BT Piotrowski, Gillette WB, Hancock EB. Examining the prevalence and characteristics of abfraction like cervical lesions in a population of US veterans. J Am Dent Assoc 2001;132(12):1694-701.
17. Michael JA, Townsend GC, Greenwood LF, Kaidonis JA. Abfraction: separating fact from fiction. Aust Dent J 2009;54(1):2-8.
18. Smith BG, Knight JK. An index for measuring the wear of teeth. Br Dent J 1984;156(12):435-8.
19. McCoy G. On the longevity of teeth. J Oral Implantol 1983;11(2):248-67.
20. Ichim I, Schmidlin PR, Kieser JA, Swain MV. Mechanical evaluation of cervical glass-ionomer restorations: 3D finite element study. J Dent 2007;35(1):28-35.
21. Lyttle HA, Sidhu N, Smyth B. A study of the classification and treatment of noncarious cervical lesions by general practitioners. J Prosthet Dent 1998;79(3):342-6.
22. Fruits TJ, VanBrunt CL, Khajotia SS, Duncanson Jr MG. Effect of cyclical lateral forces on microleakage in cervical resin composite restorations. Quintessence Int 2002;33(3):205-12.
23. Li Q, Jepsen S, Albers HK, Eberhard J. Flowable materials as an intermediate layer could improve the marginal and internal adaptation of composite restorations in Class-V-cavities. Dent Mater 2006;22(3):250-7.
24. Peaumans M, De Munck J, Landuyt V, Kanumilli P, Yoshida Y, Inoue S. Restoring cervical lesions with flexible composites. Dent Mater 2007;23(6):749-54.
25. Tay FR, Gwinnett AJ , Pang KM, Wei SH. Structural evidence of a sealed tissue interface with a total etch wet bonding technique in vivo. J Dent Res 1994;73(3):629-36.
26. Matis BA, Cochran MA, Platt JA, Oshida Y, Choi K. Microtensile bond strength of GIC to artificially created carious dentin. Oper Dent 2006;31(5):590-7.