

# Gingival Recession and its Correlation with Hypersensitivity: A Cross Sectional Study

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**Abstract:** *Aims: This study aims to investigate the risk factors and relationships between non - carious cervical lesions NCCL, cervical dentin hypersensitivity CDH, and gingival recession GR in a Jordanian population. A total of 115 patients were evaluated, and 5100 teeth were analyzed. The study found that the distributions of NCCL, CDH, and GR were 88.1, 89.1, and 59.4, respectively, with the maxillary premolars being the most affected. The study also found a positive correlation between age, NCCL, and GR NCCL and CDH CDH and GR GR and NCCL. Age, gender, oral hygiene, gastroesophageal disease, and underlying trauma were significantly associated with the presence of all three conditions. The study concludes that the distributions of NCCL and GR increase with age, and that changes in depth and morphology contribute to the high level of sensitivity and severity of recessions.*

**Keywords:** cross sectional study, gingival recession, hypersensitivity, cervical lesions, Miller classification.

## 1. Introduction

Loss of tooth structure at the interface unrelated to the presence of caries has been identified as non - carious cervical lesions (NCCL) [1], with an incidence ranging from 5 to 85% [2]. Recent research suggests that the formation and/or progression of NCCLs has a multifactorial etiology [3, 4]. A relationship between factors such as erosion (chemical or electrochemical breakdown of tooth tissue), wear, abrasion (endogenous mechanical wear) and abrasion (exogenous mechanical wear) (4 - 6), except causal limitations [7]. However, the different morphologies of the lesions are usually associated with specific etiological factors in the cervical region [5, 8], resulting in wedge - shaped or concave lesions [9]. The increasing incidence of cervical tooth wear with age suggests that NCCL is likely the result of a time - dependent process of progression [1]. Furthermore, considering the combined influence of all potential etiological factors, the presence of NCCL may contribute to exposure of dentin and accumulation of biofilm at the cervical site. Therefore, NCCLs have been associated with other conditions such as cervical dentin hypersensitivity (CDH) [10] and gingival recession (GR) [11] affecting the same tooth. However, epidemiological studies correlating the presence of NCCL, CDH, GR and risk factors are uncommon due to the difficulty of obtaining and comparing data from different populations [12]. Even within the same population, differences in the clinical features and risk factors associated with these disorders need further investigation. Therefore, the aim of this study was to evaluate the risk factors associated with NCCLs, CDH, and GR, apart from the relationship amongst these conditions in a specific Jordanian sample population.

## 2. Materials and Methods

The clinical study form and data sheets were developed to collect the data and contained the following questions: participant's name, medical history, investigator - assessed quality of hygiene, and self - reported brushing style. Each patient was then given a piece of paper to describe their eating habits for a week. A diet would be considered acidic if

the number of acidic drinks and/or frequency of food intake was greater than two. Participants were also asked about para functional habits and gastroesophageal disease. Patients previously diagnosed with gastroesophageal disease were hospitalized only under controlled conditions or after disease had been ruled out by a specialist. The clinical study individually. Causal lesions with carbon tape to identify premature patient contacts in a centric relationship across all movements. NCCLs have according to their morphological type as concave [1] or cuneiform [2]. The depth of each lesion using an NCCL cast of (PVS) elastomeric material. Calluses with a digital caliper and lesions as superficial (0.9 mm), medium (1 - 1.9 mm) or deep (more than 2 mm). Individuals reporting hypersensitivity to confirm the presence of CDH. A vaporization stimulus (a controlled puff of air) generated by an air - water syringe to determine the degree of sensitivity of the tooth. The airflow perpendicular to the buccal - cervical surface of the hypersensitive tooth at a distance of approximately 1 cm for two seconds. Adjacent teeth were fixed with polyester tape to avoid false positive results. The operator asked participants to rate their pain on a 10 - point visual analogue scale (WAS) and the score was recorded. The scores recorded were distributed according to their level: 0 - no pain; 2 - mild pain [1 - 4]; 3 - moderate pain [5 - 7]; 4 - severe pain [8 - 10]. The presence of RBC was also verified and classified as Class I, II, III, or IV according to Miller [13], taking into account the amount of keratinized tissue, the location of the junction associated with the recession, and the presence or absence of RBC interdental bone loss.

## 3. Results

The study included 115 people (aged 19 to 7 years, mean: 41.9 years). The male/female ratio was 0.68: 1. After clinical examination, 163 of 115 teeth examined were diagnosed with NCCL, 165 with CDH and 110 with GR, a distribution of 88.1%, 89.1% and 59.4%, respectively. Of the 163 people with NCCL, 161 (98.7%) also had CDH and 106 (57th percentile) also had CDH. 2% had all three diseases at the same time. 5, 100 teeth were examined. NCCL was diagnosed in 1308 (25.2%), CDH in 1613 (31.1%) and

erythrocytes in 1334 (257%). ECCL, CDH and GR showed a similar increase in distribution with age. More patients with NCCL, CDH, or GR were in Sgt; in the 50 - year age group, while lower concentrations were found in the 19 to 30 year age group. NCCL showed faster growth than CDH or GR with increasing age. In contrast, combinations of NCCL and CDH were more common in the 31 - 40 age group. These three diseases converge in the age range of 41 to 50 years. In the age group over 50 years, CDH seems to decrease and the association of NCCL and GR is more frequent. Maxillary teeth were more affected than mandibular teeth when all three conditions were considered. The presence of NCCL, CDH and GR depending on the tooth type indicated that the premolars were the most commonly affected teeth, followed by the first molars and the canines. The second molars were least affected. Risk factors The most important risk factors for NCCL were age, gender and underlying trauma; for CDH, venereal and gastric diseases; by GR, age and causal damage. Brushing, acidic diets, and para functional habits did not show statistically significant differences in changes that could be considered a significant risk factor.

#### 4. Discussion

The present study used a standardized questionnaire to assess risk factors associated with NCCL, CDH and GR in a defined patient population. The distribution of NCCL, CDH and GR was 88.1%, 89.1% and 59.4%, respectively. These values are higher than the ranges reported in previous studies [14, 15] which could be due to the fact that the subjects were patients of a specific clinic dealing with the treatment of these specific diseases. The results of this study confirm the results of previous studies which found that the incidence of NCCL and RBC increases with age [1, 12, 16 - 18], probably because older people are exposed to more common etiological factors than teenagers. On the other hand, CDH levels appear to decrease with age, which could be due to continued dentin deposition and subsequent lifelong pulp atrophy [19]. The teeth most sensitive to NCCL and CDH [1, 2, 12, 17, 20] were the maxillary premolars. These teeth have a smaller crown volume, a much thinner buccal plate, and receive excessive lateral loads during mandibular movement. This can result in increased buccal flexion of the tooth and increase deformity in the cervical area [21, 22], which could explain the more frequent occurrence and distribution of NCCLs. In this regard, some studies have shown that eccentric causal loading is associated with the presence of NCCL [23 - 26], supporting the results of this study. However, data are still insufficient and/or inconclusive, as most studies supporting this association lack a strong evidence base. This was examined in two systematic reviews that found no evidence for this correlation [22, 27]. Conversely, the main association between occlusion and NCCL found in most studies is the presence of causal wear veneers [28 - 31], often performed by a single blinded investigator, leading to a possible bias in the results, affecting their reliability reduced. [27]. Therefore, more robust, evidence - based and standardized studies need to be conducted to obtain more meaningful results.

The different distribution of NCCLs between men and women can be explained by the higher chewing force (higher causal loads generate higher stress concentrations), which makes the tooth structure more sensitive to other risk factors [4]. Interestingly, women were more likely to develop CDH, possibly related to healthy oral habits or frequent consumption of acidic foods, and a lower pain threshold than men [33, 34]. There was no statistical difference in the incidence of GR by gender. As for the specific risk factors, according to some authors, the acidity of the biofilm, which is thought to trigger GR, acts as an endogenous factor [3] and may also contribute to the progression of NCCL. However, this differs from the results of this study; Therefore, this study does not provide evidence for a conclusive relationship between biofilms and NCCL. In this context, the effect of tooth brushing, which is considered a wearing event, on the impact of NCCL progression remains controversial. According to some authors, brushing with toothpaste would cause only minimal abrasion of the dentin over a lifetime if used normally and appropriately [35]. Other studies note that the frequent occurrence of lesions on the buccal surface of the teeth automatically implies the effect of tooth brushing on NCCL formation [36], consistent with a Chinese study [ ] that found that the effectiveness of tooth brushing a Predictor for the presence of NCCL [37]. On the other hand, a recent systematic review [38] suggests that the evidence for an association between brushing and NCCL/GR remains largely inconclusive and that long - term projects should be undertaken to determine with certainty whether this factor is predisposing or simple related to the above changes. Other reports have also shown that there are patients with NCCL in populations that do not brush their teeth [39, 40], which may indicate that tooth brushing is not a trigger for NCCL but rather amplifies or accelerates the impact of this process. However, none of these studies showed a clear or standardized method of assessment. Because methods are limited, and the results obtained in this population are insufficient to draw conclusions, further research is needed to fully elucidate the role of tooth brushing in lesion progression. Likewise, the association and clinical significance of RBC development after tooth brushing remain uncertain and unproven [38, 41, 42].

#### 5. Conclusions

The study concludes that the incidence of non - carious cervical lesions NCCL, cervical dentin hypersensitivity CDH, and gingival recession GR is closely related to lifestyle factors. Recognizing the main etiological factors and their interrelationships is crucial for clinicians to prevent and control these changes, thereby improving the populations quality of life.

#### References

- [1] T. C. Aw, X. Lepe, G. H. Johnson, L. Mancl, Characteristics of noncarious cervical lesions: a clinical investigation, J. Am. Dent. Assoc.133 (6) (2002) 725-733.
- [2] D. W. Bartlett, P. Shah, A critical review of non - carious cervical (wear) lesions and the role of

- abfraction, erosion, and abrasion, *J. Dent. Res.*85 (4) (2006) 306–312.
- [3] J. O. Grippo, M. Simring, T. A. Coleman, Abfraction, abrasion, biocorrosion, and the enigma of noncarious cervical lesions: a 20 - year perspective, *J. EsthetRestor. Dent.*24 (1) (2012) 10–23.
- [4] J. O. Grippo, M. Simring, S. Schreiner, Attrition, abrasion, corrosion and abfraction revisited: a new perspective on tooth surface lesions, *J. Am. Dent. Assoc.*135 (8) (2004) 1109–1118 quiz 63 - 5.
- [5] J. A. Michael, G. C. Townsend, L. F. Greenwood, J. A. Kaidonis, Abfraction: separating fact from fiction, *Aust. Dent. J.*54 (1) (2009) 2–8.
- [6] A. Scherman, P. L. Jacobsen, Managing dentin hypersensitivity: what treatment to recommend to patients, *J. Am. Dent. Assoc.*123 (4) (1992) 57–61.
- [7] W. C. Lee, W. S. Eakle, Stress - induced cervical lesions: review of advances in the past 10 years, *J. Prosthet. Dent.*75 (5) (1996) 487–494.
- [8] B. Hur, H. C. Kim, J. K. Park, A. Versluis, Characteristics of non - carious cervical lesions—an ex vivo study using micro computed tomography, *J. Oral Rehabil.*38 (6) (2011) 469–474.
- [9] C. Walter, E. Kress, H. Gotz, K. Taylor, I. Willershausen, A. Zampelis, The anatomy of non - carious cervical lesions, *Clin. Oral Investig.*18 (1) (2014) 139–146.
- [10] J. O. Grippo, Noncarious cervical lesions: the decision to ignore or restore, *J. Esthet Dent.* (4 Suppl) (1992) 55–64.
- [11] G. Sangnes, P. Gjermo, Prevalence of oral soft and hard tissue lesions related to mechanical toothcleansing procedures, *Commun. Dent. Oral Epidemiol.*4 (2) (1976) 77–83.
- [12] J. Borcic, I. Anic, M. M. Urek, S. Ferreri, The prevalence of non - carious cervical lesions in permanent dentition, *J. Oral Rehabil.*31 (2) (2004) 117–123.
- [13] P. D. Miller Jr., A classification of marginal tissue recession, *Int. J. Periodontics Restor. Dent.*5 (2) (1985) 8–13.
- [14] B. Faye, M. Sarr, A. W. Kane, B. Toure, F. Leye, F. Gaye, et al., Prevalence and etiologic factors of non - carious cervical lesions. a study in a Senegalese population, *Odontostomatol. Trop.*28 (112) (2005) 15–18.
- [15] D. Telles, L. F. Pegoraro, J. C. Pereira, Incidence of noncarious cervical lesions and their relation to the presence of wear facets, *J. EsthetRestor. Dent.*18 (4) (2006) 178–183 discussion 84.
- [16] K. Que, B. Guo, Z. Jia, Z. Chen, J. Yang, P. Gao, A cross - sectional study: non - carious cervical lesions, cervical dentine hypersensitivity and related risk factors, *J. Oral Rehabil.*40 (1) (2013) 24–32.
- [17] K. Que, J. Ruan, X. Fan, X. Liang, D. Hu, A multi - centre and cross - sectional study of dentine hypersensitivity in China, *J. Clin. Periodontol.*37 (7) (2010) 631–637.
- [18] B. G. Smith, N. D. Robb, The prevalence of toothwear in 1007 dental patients, *J. Oral Rehabil.*23 (4) (1996) 232–239.
- [19] J. Cunha - Cruz, J. C. Wataha, L. J. Heaton, M. Rothen, M. Sobieraj, J. Scott, et al., The prevalence of dentin hypersensitivity in general dental practices in the northwest United States, *J. Am. Dent. Assoc.*144 (3) (2013) 288–296.
- [20] Q. Kehua, F. Yingying, S. Hong, W. Menghong, H. Deyu, F. Xu, A cross - sectional study of dentine hypersensitivity in China, *Int. Dent. J.*59 (6) (2009) 376–380.
- [21] D. A. Brandini, D. Pedrini, S. R. Panzarini, I. M. Benete, C. L. Trevisan, Clinical evaluation of the association of noncarious cervical lesions, parafunctional habits, and TMD diagnosis, *Quintessence Int.*43 (3) (2012) 255–262.
- [22] P. Senna, A. Del Bel Cury, C. Rosing, Non - carious cervical lesions and occlusion: a systematic review of clinical studies, *J. Oral Rehabil.*39 (6) (2012) 450–462.
- [23] O. Bernhardt, D. Gesch, C. Schwahn, F. Mack, G. Meyer, U. John, et al., Epidemiological evaluation of the multifactorial aetiology of abfractions, *J. Oral Rehabil.*33 (1) (2006) 17–25.
- [24] J. Borcic, I. Anic, I. Smojver, A. Catic, I. Miletic, S. P. Ribaric, 3D finite element model and cervical lesion formation in normal occlusion and in malocclusion, *J Oral Rehabil.*32 (7) (2005) 504–510.
- [25] L. C. Levitch, J. D. Bader, D. A. Shugars, H. O. Heymann, Non - carious cervical lesions, *J. Dent.*22 (4) (1994) 195–207.
- [26] L. F. Pegoraro, J. M. Sclaro, P. C. Conti, D. Telles, T. A. Pegoraro, Noncarious cervical lesions in adults: prevalence and occlusal aspects, *J. Am. Dent. Assoc.*136 (12) (2005) 1694–1700.
- [27] A. G. Silva, C. C. Martins, L. G. Zina, A. N. Moreira, S. M. Paiva, I. A. Pordeus, et al., The association between occlusal factors and noncarious cervical lesions: a systematic review, *J. Dent.*41 (1) (2013) 9–16.
- [28] L. Pikdoken, E. Akca, B. Gurbuzer, B. Aydil, B. Tasdelen, Cervical wear and occlusal wear from a periodontal perspective, *J. Oral Rehabil.*38 (2) (2011) 95–100.
- [29] H. Ahmed, E. S. Durr, M. Rahman, Factors associated with Non - carious cervical lesions (NCCLs) in teeth, *J. Coll. Phys. Surg. Pak.*19 (5) (2009) 279–282.
- [30] A. Estafan, P. C. Furnari, G. Goldstein, E. L. Hittelman, In vivo correlation of noncarious cervical lesions and occlusal wear, *J. Prosthet. Dent.*93 (3) (2005) 221–226.
- [31] B. T. Piotrowski, W. B. Gillette, E. B. Hancock, Examining the prevalence and characteristics of abfractionlike cervical lesions in a population of U. S. Veterans, *J. Am. Dent. Assoc.*132 (12) (2001) 1694–1701 quiz 726 - 7.
- [32] D. H. Pashley, How can sensitive dentine become hypersensitive and can it be reversed? *J. Dent.*41 (Suppl.4) (2013) S49–S55.
- [33] R. Miyazaki, T. Yamamoto, [Sex and/or gender differences in pain], *Masui* 58 (1) (2009) 34–39.
- [34] Z. Wiesenfeld - Hallin, Sex differences in pain perception, *Gend. Med.*2 (3) (2005) 137–145.
- [35] R. P. Shellis, M. Addy, The interactions between attrition, abrasion and erosion in tooth wear, *Monogr. Oral Sci.*25 (2014) 32–45.

- [36] F. Khan, W. G. Young, S. Shahabi, T. J. Daley, Dental cervical lesions associated with occlusal erosion and attrition, *Aust. Dent. J.*44 (3) (1999) 176–186.
- [37] J. Yang, D. Cai, F. Wang, D. He, L. Ma, Y. Jin, et al., Non - carious cervical lesions (NCCLs) in a random sampling community population and the association of NCCLs with occlusive wear, *J. Oral Rehabil.*43 (12) (2016) 960–966.
- [38] P. A. Heasman, R. Holliday, A. Bryant, P. M. Preshaw, Evidence for the occurrence of gingival recession and non - carious cervical lesions as a consequence of traumatic toothbrushing, *J. Clin. Periodontol.*42 (Suppl.16) (2015) S237–S255.
- [39] B. Faye, A. W. Kane, M. Sarr, C. Lo, A. V. Ritter, J. O. Grippo, Noncarious cervical lesions among a non - toothbrushing population with hansen's disease (leprosy): initial findings, *Quintessence Int.*37 (8) (2006) 613–619.
- [40] A. V. Ritter, J. O. Grippo, T. A. Coleman, M. E. Morgan, Prevalence of carious and non - carious cervical lesions in archaeological populations from North America and Europe, *J. EsthetRestor. Dent.*21 (5) (2009) 324–334.
- [41] M. Addy, M. L. Hunter, Can tooth brushing damage your health? Effects on oral and dental tissues, *Int. Dent. J.*53 (Suppl.3) (2003) 177–186.
- [42] N. A. Rosema, R. Adam, J. M. Grender, E. Van der Sluijs, S. C. Supranoto, G. A. Van der Weijden, Gingival abrasion and recession in manual and oscillating - rotating power brush users, *Int. J. Dent. Hyg.*12 (4) (2014) 257–266.
- [43] W. A. Smith, S. Marchan, R. N. Rafeek, The prevalence and severity of non - carious cervical lesions in a group of patients attending a university hospital in Trinidad, *J. Oral Rehabil.*35 (2) (2008) 128–134.
- [44] D. W. Bartlett, A. Lussi, N. X. West, P. Bouchard, M. Sanz, D. Bourgeois, Prevalence of tooth wear on buccal and lingual surfaces and possible risk factors in young European adults, *J. Dent.*41 (11) (2013) 1007–1013.
- [45] K. T. Yoshizaki, L. F. Francisconi - Dos - Rios, M. A. Sobral, A. C. Aranha, F. M. Mendes, T. Scaramucci, Clinical features and factors associated with non - carious cervical lesions and dentin hypersensitivity, *J. Oral Rehabil.*44 (2) (2017) 112–118.