Fixed Orthodontic Appliances

Subjects: Dentistry, Oral Surgery & Medicine

Contributor: Borut Poljšak

Fixed orthodontic appliances consist of archwires, brackets, and ligatures. During treatment, the archwires are ligated into the slot of the bracket. The most commonly used materials from which parts of fixed appliances are manufactured are alloys of stainless steel (SS) and nickel-nitanium (NiTi) because of their advantageous mechanical properties.

Keywords: fixed orthodontic appliances ; oxidative stress ; metal ions ; risk assessment ; antioxidant therapy

1. Introduction

Gold standard in orthodontic treatment are multibracket edgewise fixed appliances bonded to teeth, made of stainless steel (SS), combined with archwires made of nickel-titanium (NiTi) and SS. Less often, archwires made of other materials (e.g., cobalt-chromium alloy, ß-titanium (ß-Ti) alloy) are used. Orthodontic fixed appliances are usually in the oral cavity for extended time periods and are, therefore, exposed to degradation processes. The degradation process of dental metal alloys is the subject of several in vitro researches in which different parameters and their possible synergistic effects are analyzed, although a complete mapping of the oral cavity is hardly achieved due to the complex intraoral processes [1]. As a result of the ongoing temperature and pH changes, nutrition, breakdown of food and cells, varying oral flora, and the presence of plaque, various products are processed that can destroy the surface of fixed orthodontic appliances [2][3][4][5]. Metal alloys in the oral cavity are simultaneously subjected to corrosion and mechanical stress, e.g., due to masticatory forces, and in orthodontic treatments also due to the sliding of the archwire along the bracket slot. Therefore, the contact of the bracket, archwire, and ligature in the electrolytic fluid (saliva) leads to a faster and more intensive corrosion process on the metal surface [6]. Simultaneous exposure of fixed orthodontic appliances to corrosion, deformation, friction, and mechanical stress during treatment results in degradation of orthodontic brackets and archwires, which can result in elevated concentrations of metal ions in the oral cavity [7][8]. Mucosal erythema, allergic contact dermatitis, contact stomatitis, periodontitis, gingival hyperplasia, glossitis, gingivitis, and multiform erythema were all noticed during orthodontic treatment, which could be generated also by the toxic action of metal ions relieved by fixed orthodontic appliances [9][10]. As reported by the American Board of Orthodontics, treatment with fixed appliances continues for about 24 months [11]. Therefore, the safety of such appliances should be well studied as metal ions can induce either minor or major toxic effects locally (e.g., on oral cavity tissues) [12] or even systemic effects when they are absorbed and enter the systemic bloodstream [13][14].

2. Oxidative Stress and Oxidative Damage during the Treatment with Fixed Orthodontic Appliances

There are not many studies that investigated oxidative stress in the treatment with fixed orthodontic devices. Until recently, only one study determined the oxidative stress induced by orthodontic treatment at the systemic level [14], while the oxidative stress parameters were evaluated during orthodontic treatment either in saliva [15][16][17][18] or the gingival crevicular fluid [17]. Systemic oxidative stress parameters in the study of Kovac et al. [14] were evaluated in patients in the first seven-day period of treatment with fixed orthodontic appliances. The reactive oxygen species formation (ROS) as well as the antioxidant defense potential (AD) were assessed in the capillary blood samples from fifty-four male patients with malocclusion undergoing orthodontic treatment and untreated control subjects. Twenty-four hours after orthodontic treatment with fixed appliances, the ROS level was markedly elevated in the treatment group in comparison to the control group. Moreover, 24 h after archwire insertion, a significantly higher ROS/AD ratio was identified in the treatment group than in the control group. The authors concluded that the treatment with fixed orthodontic devices could cause systemic oxidative stress in the short run as the values of ROS and ROS/AD normalize during the period of one week following archwire insertion. Indeed, as revealed by Buczko et al. [15], the treatment with fixed orthodontic appliances alters the oxidant-antioxidant balance in saliva of individuals who are otherwise clinically healthy. Highest oxidative stress parameters (tiobarbituric acid reactive substance and total oxidant status) were detected in saliva within a week following the insertion of orthodontic appliance.

Decrease in superoxide dismutase (SOD1) and catalase (CAT) was observed in stimulated saliva one week after treatment, while peroxidase (Px) was increased. It was noted that the total antioxidant status (TAS) was decreased twenty-four weeks after orthodontic treatment. The oxidative status index (OSI) increased a week after the treatment. The levels of selected markers of oxidative stress (malondialdehyde, ceruloplasmin, hydrogen donors) in the saliva of the treated patients prior to and after the beginning of treatment were additionally studied by Olteanu et al. [16]. Additionally, the current research shows increased markers of oxidative stress in the 24 h period, while seven days after appliance use the concentrations were close to baseline values. In contrast, Atuğ Oezcan et al. [17] observed no increase in oxidative stress markers and damage in saliva and gingival-crevicular fluid samples of patients with fixed orthodontic appliances when comparing the pretreatment results and the results at the sixth month of orthodontic treatment. Similarly, orthodontic treatment with self-ligating metal brackets did not statistically significantly affect salivary antioxidant parameters in the period of the first ten weeks of treatment [18].

During the treatment with fixed orthodontic appliances, metal ions are released, which could potentially induce oxidative stress and have a local oxidative effect, as observed in in vitro studies. Systemically, increased oxidative stress was observed only in the first seven-day period after the implementation of fixed orthodontic appliances, most likely due to the activation of endogenous adaptive stress responses and the induction of antioxidant endogenous defenses. Numerous in vitro studies have shown that orthodontic brackets as well as archwires cause oxidative stress linked to the release of heavy metals. All types of orthodontic brackets tested, disregarding the material components, are the origin of oxidative stress in vitro, as indicated by an increased concentration of the oxidative stress marker 8-hydroxy-2′-deoxyguanosine (8-OHdG) in the DNA of murine fibroblast cells L929. The highest stress levels were observed in the all-metal and polyurethane brackets [19]. In addition, all orthodontic archwires tested induced oxidative stress in vitro by measuring (8-OHdG) in DNA of murine fibroblast cells L929. Standard NiTi archwires produced the highest oxidative stress, while TiMo and SS triggered the lowest stress [20].

Different mixtures of Cr, Fe, Co, Ni, Ti, and Mo metal ions simulating either NiTi, SS, β-Ti, or CoCr orthodontic alloys were used in testing the capacity of metal-ions as oxidative stress-inducing agents using wild-type yeast Saccharomyces cerevisiae and two mutant ΔSod1 and ΔCtt1 as model organisms, to determine whether the lack of defense system from superoxide anions and H 2O 2 contributes to metal ion-induced toxicity [21]. Indeed, mutants lacking the Sod1-Ctt1 gene should be more sensitive to transition metal ion-induced ROS cytosolic SOD (Sod1) scavenges O 2− and converts it to H 2O 2, which is further degraded to H 2O and O 2 by cytosolic catalase (Ctt1) [22]. A 1000 µM metal ion treatment with SS, Co-Cr-Ni resulted in significantly higher ROS values in all yeast strains than in the untreated control group, but the treatment using equal and lower concentrations of Ti-Mo did not significantly affect the ROS value. While SS, Co-Cr-Ni showed a significant increase in oxidative lipid damage in all yeast strains at the concentration of 1000 µM in comparison to the control group, and in the mutants (ΔSod1 and ΔCtt1) lipid oxidation presented already at 100 µM. Yeast mutants without the Sod1 gene have greater intracellular ROS levels than the Ctt1-lacking mutants although the same amount of lipid oxidation was observed in both mutants. Lipid oxidation and intracellular formation of ROS was almost twice as high in the mutants compared to the wild type.

Stainless steel bands, with (SSB) or without (NSB) silver soldered joints, were investigated for the cytostatic, cytotoxic, genotoxic, and DNA damage-inducing effects on the HepG2 and HOK cell lines [23]. After performing MTT reduction assay, alkaline and modified comet assay, cytokinesis block micronucleus assay, cytostasis assay and cytotoxicity assay, the results showed higher cytotoxicity and genotoxicity of SSB eluates in comparison to NSB samples. Ni and Fe were found in the SSB as well as NSB medium samples, while Cd, Cr, Ag, Cu, and Zn were found only in the SSB medium samples.

It can be concluded that oxidative stress during orthodontic treatment can be caused by exposure to heavy metals (e.g., Cr, Fe, Co, Ni, and Ti), both locally and systemically. It can be induced also by many other factors, including aseptic inflammation in the periodontal ligament by cause of mechanical force and inflammation of periodontal tissues as a result of improper oral hygiene [14]. We would also like to draw attention to the occurrence of a synergistic effect when cells are simultaneously exposed to two or more metal ions. This is because their toxic effect not only adds up with simultaneous exposure, but it can even be multiplied or potentiated. According to Rinčić Mlinarić et al. (2019), the collective concentration of Ni and Ti in concentrations of at least 162 µg/L is necessary for synergism, provoking moderate to strong cytotoxicity and a notable induction of free radicals [24]. Terpilovska and Siwicki reported that Cr 3+ and Fe 3+ show synergistic effects in cytotoxicity, genotoxicity, and mutagenicity assays, but in some other combinations metal ions can act antagonistically—Cr 3+ and Mo 3+ or Cr 3+ and Ni 2+ show antagonistic effect— Cr 3+ protects from Ni 2+ or Mo 3+ toxicity [25].

3. Conclusions

To summarize: The results of in vitro and in vivo studies that examined the release of metal ions show differences in the amount of metal ions released due to different study designs. Metal ions released from corroded orthodontic appliances could be exposed to redox cycling reactions and cause oxidative stress. Therefore, the safety of such appliances should be investigated, the risk assessed, and the justification and appropriateness of additional antioxidant intake confirmed by the results of future human studies to demonstrate the efficacy of antioxidant therapy in reducing oxidative stress during dental treatment. The consumer growing awareness, interest and knowledge impels the manufacturing industry to confront the challenges of choosing adequate orthodontic appliance materials to minimize their impact on health.

Although Žukowski et al. [26] reported some beneficial effects of generating ROS in the oral cavity during dental treatment (e.g., stimulation of immune response and wound healing, cytotoxic effect on pathogenic bacteria), the metal ions released into the oral cavity from fixed orthodontic appliances due to corrosion and biodegradation pose a health risk to patients. This risk is, however, a minor one, as only very high metal concentrations induce cytotoxicity and oxidative stress, which was found in the studies on cell cultures. On the other hand, several studies reported that orthodontic treatment can induce transient imbalance in the ratio between oxidants and antioxidants in saliva as well as at the systemic level of clinically healthy subjects.

Nevertheless, the increased ROS levels can occur at a local level in the oral cavity, which could pose a problem to patients with lower efficiency of endogenous antioxidant defense systems, metal ions hypersensitive individuals, or patients with decreased salivary antioxidant power [27]. This topic should be further investigated, since there is currently only scarce scientific research on the effect of orthodontic materials on the formation of oxidative stress.

References

1. Wichelhaus, A.; Geserick, M.; Hibst, R.; Sander, F.G. The effect of surface treatment and clinical use on friction in NiTi orthodontic wires. Dent. Mater. 2005, 21, 938–945.
2. Eliades, T.; Bourauel, C. Intraoral aging of orthodontic materials: The picture we miss and its clinical relevance. Am. J. Orthod. Dentofac. Orthop. 2005, 127, 403–412.
3. Eliades, T.; Athanasiou, A.E. In Vivo Aging of Orthodontic Alloys: Implications for Corrosion Potential, Nickel Release, and Biocompatibility. Angle Orthod. 2002, 72, 222–237.
4. Cortizo, M.C.; De Mele, M.F.L.; Cortizo, A.M. Metallic dental material biocompatibility in osteoblastlike cells: Correlation with metal ion release. Biol. Trace Elem. Res. 2004, 100, 151–168.
5. Kao, C.T.; Ding, S.J.; Min, Y.; Hsu, T.C.; Chou, M.Y.; Huang, T.H. The cytotoxicity of orthodontic metal bracket immersion media. Eur. J. Orthod. 2007, 29, 198–203.
6. Drescher, D.; Bourauel, C.; Schumacher, H.A. Frictional forces between bracket and arch wire. Am. J. Orthod. Dentofac. Orthop. 1989.
7. Landolt, D.; Mischler, S.; Stemp, M. Electrochemical methods in tribocorrosion: A critical appraisal. Electrochim. Acta 2001, 46, 3913–3929.
8. Močnik, P.; Kosec, T.; Kovač, J.; Bizjak, M. The effect of pH, fluoride and tribocorrosion on the surface properties of dental archwires. Mater. Sci. Eng. C 2017, 78, 682–689.
9. Ortiz, A.J.; Fernández, E.; Vicente, A.; Calvo, J.L.; Ortiz, C. Metallic ions released from stainless steel, nickel-free, and titanium orthodontic alloys: Toxicity and DNA damage. Am. J. Orthod. Dentofac. Orthop. 2011, 140, 115–122.
10. Keinan, D.; Mass, E.; Zilberman, U. Absorption of Nickel, Chromium, and Iron by the Root Surface of Primary Molars Covered with Stainless Steel Crowns. Int. J. Dent. 2010, 2010, 326124.
11. Moresca, R. Orthodontic treatment time: Can it be shortened? Dent. Press J. Orthod. 2018, 23, 90–105.
12. Wataha, J.C. Biocompatibility of dental casting alloys: A review. J. Prosthet. Dent. 2000, 83, 223–234.
13. Amini, F.; Jafari, A.; Amini, P.; Sepasi, S. Metal ion release from fixed orthodontic appliances—An in vivo study. Eur. J. Orthod. 2012, 34, 126–130.
14. Kovac, V.; Poljsak, B.; Perinetti, G.; Primozic, J.; Reis, F.S. Systemic Level of Oxidative Stress during Orthodontic Treatment with Fixed Appliances. Biomed. Res. Int. 2019, 2019, 5063565.
15. Buczko, P.; Knaś, M.; Grycz, M.; Szarmach, I.; Zalewska, A. Orthodontic treatment modifies the oxidant–antioxidant balance in saliva of clinically healthy subjects. Adv. Med. Sci. 2017, 62, 129–135.

16. Olteanu, C.; Muresan, A.; Daicoviciu, D.; Tarmure, V.; Olteanu, I.; Irene, K.M.L.W. Variations of some saliva markers of the oxidative stress in patients with orthodontic appliances. Fiziologia 2009, 19, 27–29.
17. Atuğ Özcan, S.S.; Ceylan, I.; Özcan, E.; Kurt, N.; Dağsuyu, I.M.; Çanakçi, C.F. Evaluation of oxidative stress biomarkers in patients with fixed orthodontic appliances. Dis. Markers 2014, 2014, 1–7.
18. Portelli, M.; Militi, A.; Cervino, G.; Lauritano, F.; Sambataro, S.; Mainardi, A.; Nucera, R. Oxidative Stress Evaluation in Patients Treated with Orthodontic Self-ligating Multibracket Appliances: An Case-Control Study. Open Dent. J. 2017, 11, 257–265.
19. Buljan, Z.I.; Ribaric, S.P.; Abram, M.; Ivankovic, A.; Spalj, S. In vitro oxidative stress induced by conventional and self-ligating brackets. Angle Orthod. 2012, 82, 340–345.
20. Spalj, S.; Mlacovic Zrinski, M.; Tudor Spalj, V.; Ivankovic Buljan, Z. In-vitro assessment of oxidative stress generated by orthodontic archwires. Am. J. Orthod. Dentofac. Orthop. 2012, 141, 583–589.
21. Kovač, V.; Poljšak, B.; Primožič, J.; Jamnik, P. Are metal ions that make up orthodontic alloys cytotoxic, and do they induce oxidative stress in a yeast cell model? Int. J. Mol. Sci. 2020, 21, 7993.
22. Farrugia, G.; Balzan, R.; Madeo, F.; Breitenbach, M. Oxidative Stress and Programmed Cell Death in Yeast; Frontiers Media SA: Lausanne, Switzerland, 2012; pp. 1–21.
23. Gonçalves, T.S.; de Menezes, L.M.; Trindade, C.; Machado, M. da S.; Thomas, P.; Fenech, M.; Henriques, J.A.P. Cytotoxicity and genotoxicity of orthodontic bands with or without silver soldered joints. Mutat. Res. Genet. Toxicol. Environ. Mutagen. 2014, 762, 1–8.
24. Rincic Mlinaric, M.; Durgo, K.; Katic, V.; Spalj, S. Cytotoxicity and oxidative stress induced by nickel and titanium ions from dental alloys on cells of gastrointestinal tract. Toxicol. Appl. Pharmacol. 2019, 383.
25. Terpilowska, S.; Siwicki, A.K. Interactions between chromium(III) and iron(III), molybdenum(III) or nickel(II): Cytotoxicity, genotoxicity and mutagenicity studies. Chemosphere 2018, 201, 780–789.
26. Żukowski, P.; Maciejczyk, M.; Waszkiel, D. Sources of free radicals and oxidative stress in the oral cavity. Arch. Oral Biol. 2018, 92, 8–17.
27. Tartaglia, G.M.; Gagliano, N.; Zarbin, L.; Tolomeo, G.; Sforza, C. Antioxidant capacity of human saliva and periodontal screening assessment in healthy adults. Arch. Oral Biol. 2017, 78, 34–38.
