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Desensitization Treatment for Dentin Hypersensitive in Heavy-Smoker Patient

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ABSTRACT

Introduction: Dentin hypersensitivity (DH) is the most common dental and oral complaint in the world's population, in the form of sharp and short pain as a response to exposure to stimuli on exposed dentin which can occur due to several factors, one of which is gingival recession. Smoking is a factor that causes gingival recession which can cause DH. This case report aims to demonstrate desensitization treatment in a heavy smoking patient with a case of dentin hypersensitivity.

Case report: The patient is a 35 year old male with the chief complaint of bad breath and moderate pain in tooth 43, has a bad habit of heavy smoking, up to 20 cigarettes per day. The patient's intraoral examination results had moderate OHI, gingival recession, and positive results on the pain provocation test. The patient experienced gingivitis et causa plaque and calculus accompanied by tooth 43 dentin hypersensitivity. Treatment planning for patients is to provide dental health education (DHE) regarding maintaining oral hygiene and eliminating smoking habits followed by desensitization. Before desensitization, scaling root planing was performed. Desensitization in this case is carried out to treat dentin hypersensitivity with a dual action mechanism desensitization agent (neural action and occlusive action), namely KF 2% topically on the cervical surface of tooth 43 which experienced dentin hypersensitivity due to gingival recession.

Results: Dentin hypersensitivity in the patient was successfully treated using a desensitizing agent after repeated topical application of KF 2% four times.

Conclusion: Smoking habits can worsen oral health and damage periodontal tissue in the form of gingival recession causing dentin hypersensitivity. DH treatment is desensitization accompanied by DHE to eliminate the cause of dentin hypersensitivity.

Keywords: Dentin hypersensitivity, desensitization, gingival recession, heavy-smoker

Introduction

Dentin hypersensitivity (DH) is one of the most common oral dental complaints and symptoms, widespread throughout the world's population (Fambrini et al., 2022). The incidence rate of disease in populations throughout the world regarding dentin hypersensitivity is around 35% and generally occurs between the ages of 20 and 50 years. In women, the prevalence of DH is higher than in men. The prevalence of DH can reach 98% in patients suffering from periodontal disease (Reis et al., 2023). The incidence of DH is generally found and is also associated with patients with gingival recession, around 72% to 98% (Fambrini et al., 2022).

Patients with DH can experience problems with their quality of life due to interference from pain in daily activities, such as when talking, brushing their teeth, eating, or even just drinking. This disorder can affect patients physically and psychologically (Davari et al., 2013; Liu et al., 2020). Dentin hypersensitivity has a negative effect, where DH patients usually have poor oral health and the patient's quality of life regarding their oral health is also poor (Robinson, 2015). One of the causes of dentin hypersensitivity is gingival recession. One of the factors causing gingival recession is smoking, because smoking has a bad effect on periodontal tissue (Nandhana et al., 2019). This case report discusses the treatment of dentin hypersensitivity in a patient who experienced gingival recession accompanied by a heavy smoking habit.

Gingival Recession

Gingival recession is an alteration that occurs at the gingival margin, where the gingival margin experiences a shift apically from the cementoenamel junction (CEJ) which can cause exposure of the tooth root surface. Gingival recession can also be defined as the clinical appearance of a distance between the CEJ and the gingival margin. The appearance of gingival recession indicates its occurrence of attachment loss, but does not necessarily indicate inflammation (Newman et al., 2021).

Gingival recession has several causative factors, including periodontal disease, alveolar bone dehiscence, brushing teeth with the wrong technique, traumatic and excessive brushing methods, orthodontic tooth movement, occlusion trauma, and abnormalities in muscle attachment and frenulum. Other factors that can cause gingival recession are iatrogenic factors, such as prosthodontic and orthodontic treatment (Nandhana et al., 2019; Newman et al., 2021). Another important risk factor for gingival recession is smoking, because smoking has an adverse effect on periodontal tissue (Nandhana et al., 2019). The occurrence of gingival recession can cause several conditions, including unaesthetic appearance of the gingiva, susceptibility to non-carious lesions on the cervix and tooth roots, risk of marginal gingivitis, and dentin hypersensitivity (Newman et al., 2021). Checking the severity of gingival recession can be done using a probe, the probe tip is placed on the tooth surface with the probe tip in contact with the tooth surface, then measure the distance from cementoenamel junction to gingival margin (Newman et al., 2021). There are four classifications of gingival recession, which can be seen in Table 1.

Table 1. Classification of Gingival Recession Defects (Newman et al., 2021).

Classification	Class I	Class II	Class III	Class IV
	Recession in marginal tissue does not reach the mucogingival junction. There was no loss of bone or soft tissue at the interdental.	Recession in the marginal tissue has reached or is apical to the mucogingival junction. There was no loss of bone or soft tissue at the interdental.	Recession in the marginal tissue has reached or is apical to the mucogingival junction. There is loss of bone or soft tissue in the interdental area or facial tooth malposition.	Recession in the marginal tissue has reached or is apical to the mucogingival junction. There is loss of bone and soft tissue in the interdental area and severe tooth malposition.

Dentin Hypersensitivity

Dentin hypersensitivity (DH) can also be known as dentinal hypersensitivity, cemental hypersensitivity, root hypersensitivity, and cervical hypersensitivity. Dentin hypersensitivity is defined as sharp, transient, rapid onset pain that occurs within a short time caused by exposed dentin in response to exposure to non-noxious stimuli such as evaporative, tactile, thermal, osmotic or chemical stimuli, not due to defects and/or pathology in the teeth (Fambrini et al., 2022; Gillam, 2015; Liu et al., 2020). An example of an evaporative stimulus is air blast from a dental three-way syringe. Thermal stimuli include temperature from cold air, water jet from a dental three-way syringe, and hot or cold food and drink. The chemical stimulus comes from acid, such as in patients who have gastric esophageal reflux disease (GERD), acid in food or drink, or acid etching dental care. Tactile stimuli are contact or touch of dental instruments on teeth during scaling, probes during examination, and when brushing teeth. The osmotic stimulus is in the form of a hypertonic solution, for example a sugar solution (Agarwal, 2019).

Dentin hypersensitivity can occur due to several factors, including a diet high in acid, gastric disorders, gingival recession, occlusion trauma, improper toothbrushing techniques or methods (Ribeiro et al., 2022). Another factor causing DH is smoking tobacco which causes damage to the gingiva and erosion of the enamel (Agarwal, 2019). Dentin hypersensitivity can occur in several teeth, one tooth, or even just one tooth surface (Longridge et al., 2019). The highest incidence of DH occurs in the cervical canines and premolars (Reis et al., 2023). The most common location of DH is on the cervical surface of the teeth, around 90% (Majeed et al., 2019).

Anatomically, dentin on the surface of the tooth crown is covered by a layer of enamel and on the surface of the tooth root the dentin is covered by a thin layer of cementum. Loss of periodontal tissue such as gingival recession is a major contributing factor that can cause loss of cementum on the cervical surface of the tooth root resulting in dentinal exposure. Dentinal exposure occurs when the enamel or cementum layer is lost, which can cause hypersensitivity (Liu et al., 2020). The exposed dentinal tubules are sensitive due to their response to external stimuli (Davari et al., 2013). Not all exposed dentin experiences hypersensitivity, due to differences in thickness of the calcified smear layer, in patients with hypersensitivity the dentin is thinner than nonsensitive dentin. The smear layer is formed from hydroxyapatite and collagen, DH can occur if this smear layer is lost (Gillam, 2015). Another factor that influences dentin sensitivity is the large diameter and number of exposed dentinal tubules. In teeth experiencing DH, the diameter of the dentinal tubules is twice as large as nonsensitive dentin (Robinson, 2015). There are eight times more dentinal tubules in hypersensitive dentin than nonsensitive dentin (Mrinalini et al., 2021).

There are several theories that can explain the etiopathogenesis of dentin hypersensitivity, namely the hydrodynamic theory, neural theory, and odontoblastic transduction theory. The most accepted theory is the hydrodynamic theory (Fambrini et al., 2022). This hydrodynamic theory was introduced in 1964 by Brännström (Arora et al., 2021). The mechanism of DH in the hydrodynamic theory is that when the dentin tissue has open dentin tubules, exposure to stimuli in the form of chemical, mechanical, environmental or thermal changes can cause a response in the form of movement of dentinal fluid contained in the dentin tubules. This fluid movement in the dentinal tubules causes changes in pressure and ultimately triggers a response mechanoreseptor A-delta nerve fibers which are sensitive to changes in pressure in the dentin-pulp border area are activated and stimulated resulting in transient acute pain which is sharp and short (Arora et al., 2021; Fambrini et al., 2022; Gillam, 2015; Liu et al., 2020). Dentin has the capacity to respond to external stimuli from the environment that are both pathological and physiological because in the dentinal tubules there is a dentin-pulp complex which contains extensions of odontoblastic processus. Odontoblastic processus contained in the dentin tubules contain dentinal fluid originating from the pulp blood vessels (Davari et al., 2013; Liu et al., 2020).

The pain caused by the movement of dentinal fluid is also increased in hypersensitive patients because the smear layer on the dentin is thin, making it more responsive to stimulus exposure (Davari et al., 2013). Stimuli that can cause a response to the outward movement of dentinal fluid in exposed dentin include evaporative stimuli such as thermal (cold temperature), osmotic (acid and sugar) stimuli, and air blast. The touch or contact of a toothbrush or dental instrument on the exposed dentin surface is a mechanical stimulus that can put pressure on the tissue surface, so that expansion when the contact is released can trigger increased movement of dentinal fluid in the dentinal tubules (Liu et al., 2020).

The diagnosis of DH can be carried out through subjective and objective examinations. The subjective examination that needs to be asked is the characteristics, intensity, frequency, duration and stimulus that causes pain (Mrinalini et al., 2021). Objective examinations that can be carried out to confirm the diagnosis of DH are pain provocation tests, which consists of providing tactile stimulus, air stimulus and thermal stimulus. Examination by providing a tactile stimulus can be carried out by touching the instrument to the surface of the tooth using a dental explorer or probe. Examination with an air stimulus can be given with an air blast using a dental three way syringe, this air application can be done for 2 seconds. Providing a thermal stimulus with an ice cold temperature stimulus can be applied using a refrigerant spray using cotton pellets for one second (van Loveren et al., 2018).

Prevention of DH and eliminating etiological factors can be done by maintaining good oral hygiene, one of which is by improving toothbrushing techniques to reduce the risk of gingival recession, by using a soft-bristled toothbrush, brushing teeth without excessive pressure, the duration is not too long, and do not use toothpaste that contains abrasive ingredients. Other DH prevention that can be done is avoiding excessive use of toothpicks and floss, dietary modification by reducing

consumption of acidic foods and drinks, if you consume acidic foods or drinks, it is recommended to consume something neutral or alkaline (Arora et al., 2021).

Treatment to treat DH is by carrying out desensitization treatment with a desensitizing agent. Desensitization is treatment to treat hypersensitive dentin conditions due to open dentinal tubules. Indications for managing dentin hypersensitivity include teeth with Miller 1 and 2 gingival recession, dentin hypersensitivity without abrasion, abfraction or attrition of teeth without caries, without alveolar bone loss. Contraindications are Miller class 3 and 4 gingival recession, teeth with defects, alveolar bone loss characterized by tooth mobility (Soeprapto, 2017).

There are two classifications of desensitizing agents, consisting of the method of application of the material and the mechanism of action. Desensitizing agents based on the method of application of the material are divided into two, namely at-home and in-office. Application of materials at-home which is a material that can be used by the patient himself and can be used on many teeth. There are various desensitizing agent preparations in the therapy of dentin hypersensitivity at-home are chewing gums, tooth powders, mouthwashes, and tooth pastes (Davari et al., 2013). Desensitizing agent in-office applied by the dentist in the clinic, so the management is more complicated.

Desensitizing agents based on their mechanism of action are divided into two, namely neural action and occlusive action. Mechanism of action of desensitizing agents neural action namely by interfering with the neural response to a painful stimulus, whereas occlusive action by blocking the dentinal tubules (tubule occlusion) to prevent movement of dentinal fluid. One of the mechanisms of action of desensitizing agents is occlusive action with blocking the dentinal tubules or also known as tubule occlusion. Blocked dentinal tubules can prevent and reduce fluid movement from hydrodynamic effect so that pain due to nerve sensitivity can be reduced (Davari et al., 2013; Ribeiro et al., 2022; van Loveren et al., 2018). Mechanism of tubule occlusion can be done through the topical application of materials on the tooth surface that form precipitation in the dentinal tubules which have the properties of insoluble (van Loveren et al., 2018). The ingredients that have this desensitization mechanism are *fluorides, strontiums, oxalates, stannous, arginine, nanohydroxyapatite,* varnishes, and adhesive resin. *Fluoride* reduces the level of permeability in dentin by forming precipitates of calcium fluoride crystals into the dentinal tubules. Available preparation materials include *sodium fluoride, stannous fluoride, potassium oxalate, fluoride* varnishes, and others (Davari et al., 2013; Ribeiro et al., 2022; van Loveren et al., 2018). The ideal desensitizing agent does not cause pain, is easy to apply, does not cause staining on teeth, has a long-term effect, is not harmful to the pulp, and the effect is felt quickly (Davari et al., 2013).

The mechanism of action of other desensitizing agents is neural action or nerve desensitization, where these ingredients have a direct desensitizing action or directly affect innervation pulpal side or below the dentin surface in the dentinal tubules. This mechanism of action disrupts the transmission of nerve impulses. Materials that have a working mechanism nerve desensitization is *potassium* salts (*potassium nitrate*) (Davari et al., 2013; van Loveren et al., 2018).

Cigarettes on Periodontal Tissue

Cigarette content and its relationship with periodontal tissue

Cigarette smoke contains various types of chemicals, including *carbon monoxide*, *hydrogen cyanide and nitrogen oxides*, *formaldehyde*, *acrolein*, *benzene and N-nitrosamines*, *nicotine*, *phenol*, *polyaromatic hydrocarbons*, and tobacco-specific *nitrosamines*, which are chemicals that are dangerous to humans health and oral health, in particular, influence the severity of periodontal disease. Smoking activities can be categorized as follows, light smokers ((≤10 cigarettes/day), and heavy-smokers (≥10 cigarettes/day) (Kanmaz et al., 2022). Smokers have a tendency to have more plaque and calculus, as well as increased occurrence of alveolar bone loss, periodontal pockets, and clinical attachment loss compared to non-smokers (Ahad et al., 2021).

The incidence and prevalence of localized and generalized gingival recession are strongly associated with the presence of supragingival calculus and smoking. Smoking is associated with periodontal disease due to the immunosuppressive effects of smoking, this is known to increase the level of tissue damage and also the level of susceptibility to periodontal pathogens. The chemical content in cigarettes affects the immune response, so that the elimination of periodontal pathogens is disrupted and causes the mechanism of gingival recession to continue (Nandhana et al., 2019). Smoking causes changes in the body in the form of changes in gene expressions in the respiratory tract epithelium, damage and reduces the level of immune resistance in the oral mucosa. In smokers, fewer defense cells can migrate to the gingival pocket, thereby reducing the immune system. Attachment loss and pocket depth are higher in smokers compared to non-smokers even with similar plaque conditions (Kanmaz et al., 2022).

Ingredients of cigarettes and their relationship to saliva secretion and tissue damage

Nicotine is one of the substances found in cigarettes and is a pharmacologically active ingredient contained in cigarettes, where this substance has a peripheral vasoconstrictive effect due to the nature of *nicotine* which can mimic the action of *acetylcholine* because of its structural similarity to *acetylcholine* (Newman et al., 2021). In smokers, worsening circulation in periodontal tissue causes the function and shape of vascular structures to be adversely affected (Kanmaz et al., 2022).

Smoking has the effect of decreasing saliva secretion, resulting in increased plaque build up on the surface of the teeth. This condition is caused by the *nicotine* content in cigarettes which has a pharmacokinetic effect, namely stimulating the

sympathetic nervous system so that saliva secretion decreases (Kanmaz et al., 2022). Saliva has antioxidant properties that protect the body against free radicals. Cigarette use causes high levels of free radicals, thus affecting antioxidant levels and smokers have low antioxidant levels. Cigarette smoke is a large source of free radicals which can cause cell damage (Bakhtiari et al., 2015). Tissue damage can also occur because smoking causes oxidative stress-mediated tissue damage because of free radicals in cigarettes (Nandhana et al., 2019).

Case Report

A 35 year old patient came to the Soelastri Dental and Oral Hospital (RSGM Soelastri) complaining of discomfort because he felt bad breath and wanted his teeth cleaned. The results of the patient's history showed that he had tartar cleaned 4 years ago. The patient admitted that he had no history of systemic disease. The patient has bad habits, namely smoking 20 cigarettes per day, drinking 2-3 glasses of black coffee per day, and brushing his teeth irregularly. The patient also admitted to having complaints of sharp pain of short duration in the lower jaw tooth region when consuming cold food or drinks.

On objective extraoral examination, the patient's lips appeared black. On objective intraoral examination, the clinical picture appears to be blackish brown spots that are widespread on the labial and buccal surfaces of the attached gingiva with unclear boundaries, there is edentulous on teeth 37, 36, 46, and 47, there is abrasion on the cervical teeth 44 and 45, then on The upper and lower jaw teeth contain supragingival and subgingival calculus, debris, and blackish brown stains, see Figure 1.



Figure 1. Clinical features of the patient before treatment.

Periodontal tissue examination is carried out to determine the level patient's oral hygiene, after the examination Oral Hygiene Index (OHI) obtained a score of 3.9 (medium), Gingival Index (GI) obtained a score of 1.3 (medium), Plaque Control Record O'Leary (PCR) obtained a score of 95.8% (bad) and on Periodontal Chart it was found that there was no mobility, there was gingival recession on tooth 43 of 3 mm with a normal pocket depth (0-2 mm), and on tooth 43 there was BOP (bleeding on probing) positive. Based on the results of subjective and objective examinations, the diagnosis was determined to be gingivitis *et causa* plaque and calculus. Treatment planning for this case is communication, information and education (DHE), scaling root planning with ultra-sonic scaler, desensitization, and post-treatment control.

At the first visit, phase 1 periodontal treatment is carried out, scaling root planning with ultra-sonic scaler. Treatment begins by providing DHE regarding the patient's diagnosis, namely gingivitis *et causa* plaque and calculus, are caused by the buildup of tartar (calculus) and cause complaints of bad breath as well as inflammation of the gums, tooth staining (stain) which appears blackish brown on the surface of the teeth is caused by the bad habit of smoking and frequent consumption of colored drinks such as coffee. Education also conveys the goals of care scaling root planning is to eliminate tartar. Patients are provided with information regarding risk when no action is taken scaling, among other things, it can make the disease worse and the bad breath that the patient complains about cannot disappear, then regarding the effects that can occur due to the scaling, namely pain and slight bleeding when cleaning tartar. Education on how to maintain oral hygiene and health, namely eliminating the bad habits of smoking and drinking coffee, brushing your teeth properly and having your teeth checked by a dentist regularly, as well as check-ups 1 week after treatment. Scaling root planing carried out after obtaining written informed consent from the patient on the informed consent. Scaling root planing use ultra-sonic scaler to clean supragingival and subgingival stains and calculus, then polishing used a rotary brush lowspeed with pasta and pumice on the tooth surface.

At the second visit, subjective and objective examinations were performed for post-treatment evaluation scaling root planning, it was found that patients felt more confident because bad breath was reduced and there were still complaints of disturbances in comfort due to sharp pain of short duration when consuming cold food or drinks. The results of the objective intraoral examination showed OHI 0.6 (good), GI score 0.08 (mild), PCR 23.9% (good) and Periodontal Chart was found that there was no mobility, there was still gingival recession in tooth 43 of 3 mm with a normal pocket depth (0-2 mm) and

there was no BOP. Examination of dentin hypersensitivity in tooth 43 which experienced recession was carried out using pain provocation test in the form of providing an air stimulus (dehydration test) and providing a tactile stimulus (sondation test). A dehydration test is carried out on with air blast on dental three way syringe with a distance of 1 cm from the tooth being examined, and carried out for 1 to 2 seconds by covering the surface of the neighboring tooth using cotton roll, Look Figure 2.



Figure 2. Dehydration test.

The results of the dehydration test showed that the patient admitted that he felt sharp and brief pain, the pain disappeared as soon as the air stimulation used an air blast removed. Then, a sondation test is also carried out by scratching the tip of the sonde without pressure on the cervical surface of the tooth cementoenamel junction (CEJ) and the tooth root surface is exposed due to recession, see Figure 3. The results of the sondation test showed that the patient felt sharp and brief pain.



Figure 3. Sondation test.

The patient's subjective and objective examination results show that patient's oral hygiene improved, but the patient experienced dentin hypersensitivity in tooth 43 due to classification class 2 gingival recession Miller, so that the patient is indicated for further treatment, namely desensitization to eliminate pain complaints. The patient was given DHE again before the desensitization treatment, that dentin hypersensitivity can occur as a result of gingival recession, then the risk if no treatment is carried out, where the patient's complaint of pain in the teeth will appear when consuming cold food or drinks or when exposed to cold air, has been informed that desensitization treatment is temporary, and the treatment must be repeated periodically. Patients have also been educated on how to maintain oral hygiene and health, and not to brush their teeth too hard.

Follow-up desensitization treatment is carried out after receiving informed consent written by the patient. Desensitization treatment on tooth 43 begins with carrying out prophylaxis using rotary brush lowspeed with pasta and pumice on the tooth surface 43 with the aim of keeping the tooth surface clean of debris so as not to interfere with the working mechanism of the desensitization agent, see Figure 4.



Figure 4. Prophylaxis with paste and pumice.

The work area is isolated using cotton roll on the labial and lingual, and continued by drying the tooth surface with air blast from dental three way syringe. Desensitizing agent *potassium fluoride* (Desensibilize KF 2% - FGM Dental Product) is applied using microbrush on the cervical tooth from the CEJ to the entire exposed surface of the tooth root, then left for 10 minutes, see Figure 5.



Figure 5. Application of KF% on the tooth surface with microbrush.

Evaluation after completion of treatment is carried out by means of dehydration and sondation tests to see whether there is pain or not to evaluate the success of the treatment. The application of the desensitization agent is carried out 4 times until the patient no longer feels pain when the dehydration test and sound test are carried out. Post-treatment instructions are that patients should not rinse their mouths for 30 minutes after treatment, should not eat for 1 hour after treatment, and control 1 week after treatment, then patients are given education to maintain oral health and cleanliness and eliminate the bad habit of smoking.

At the third visit, a post-desensitization treatment evaluation was carried out on tooth 43, with subjective and objective examination. Based on subjective examination results, the patient admitted that he no longer felt pain when eating and drinking cold food or when exposed to cold air. The results of the objective examination of dehydration and sondation tests showed that the patient had no pain.

Discussion

The case in this treatment is a patient whose subjective examination results have a habit of smoking up to 20 cigarettes per day and are categorized as heavy smokers (Kanmaz et al., 2022). The results of the subjective examination also showed that the patient lacked awareness in maintaining the health and cleanliness of his oral cavity, where the patient admitted that he did not brush his teeth regularly every day. The patient's bad habits in the form of not brushing his teeth regularly and smoking had an impact on the patient's oral health. The results of the patient's objective intraoral examination showed that there was melanosis due to smoking on the gingiva, visible stains on the surface of the teeth, decreased saliva secretion in the patient so that the patient also complained that the mouth felt dry, there was a large buildup of calculus and debris both supragingival and subgingival, and there was a clinical picture of gingival recession, dentin hypersensitivity, and abrasion. Supragingival calculus is mostly yellowish white in color, but in patients there appears to be a brownish coloring in the supragingival calculus which can be influenced by tobacco smoking and food and drink color pigments (Newman et al., 2021). Apart from calculus, on the surface of the patient's teeth there is also blackish brown external staining (stain) caused by the patient's smoking habit.

There are various bad effects of smoking on periodontal tissue, one of which is causing damage to the gingival attachment resulting in changes to the gingival margin in the form of gingival recession. This gingival recession can cause dentin hypersensitivity in the teeth due to exposure of the cervical surface and tooth roots. The exposure of the tooth root surface due to gingival recession can cause loss of the thin layer of cementum on the root surface, resulting in dentinal exposure (Fambrini et al., 2022). Exposed dentin causes the dentinal tubules to be open, making them more susceptible to external non-noxious stimuli. This external stimulus causes a sensitivity response in the form of movement of dentinal fluid in the dentinal tubules, which can activate terminal nerves and cause sharp and short pain known as dentin hypersensitivity. The canines and first premolars are the teeth that have the highest level of gingival recession and dentin sensitivity (Davari et al., 2013). In this case, the patient experienced a recession of 3 mm and dentin hypersensitivity in the right lower canine (tooth 43). Dentin hypersensitivity in this case was suffered by the patient due to gingival recession.

The Dental Health Education (DHE) that needs to be provided is regarding the main cause of patient complaints, where complaints of bad breath and pain in the teeth felt by patients are caused by irregular toothbrushing and heavy smoking, then explained about the treatment that needs to be carried out, and continued education about how to maintain oral hygiene and advice on stopping the patient's bad smoking habit. It is hoped that this education can create behavioural changes on oral hygiene The patient will get better, namely brushing his teeth regularly and with the correct technique, using toothpaste containing a desensitizing agent, and stopping the smoking habit, in order to prevent the worsening of periodontal disease and prevent DH from recurring.

Dental Health Education given to patients after initial periodontal therapy phase 1, namely scaling root planning is an effort to remove calculus and plaque that cause gingival recession. The action to remove plaque, calculus, and stain on the surface of the crown and root of the tooth is scaling (Asykarie et al., 2017). Root planning is the act of cleaning the root surface from necrotic tissue and bacteria on the root surface (Prihandini et al., 2022). Scaling root planning has been given, but it shows maximum results, so that gingival recession is still visible on the cervical surface of the teeth. Further treatment is needed for cases of gingival recession that are still not corrected, namely desensitization.

Dentin hypersensitivity in patients is also treated in the form of desensitization treatment using desensitization agents in the form of potassium fluoride 2% by using microbrush, so that the material can enter and close the open dentinal tubules. Potassium fluoride (Desensibilize KF 2% - FGM Dental Product) which is used for desensitization treatment in cases has a dual action desensitization mechanism from its ingredients consisting of potassium nitrate and sodium fluoride. The potassium nitrate content has a desensitization mechanism neural action and the sodium fluoride content has a desensitization mechanism occlusive action. This desensitization needs to be carried out so that the pain complaints felt by the patient can disappear, in order to improve the patient's quality of life in daily activities.

Desensitization treatment must also be accompanied by patient education, where this treatment is temporary, and needs to be repeated if pain complaints reappear. Preventing hypersensitivity from occurring again must also be done, namely by educating patients to increase awareness in maintaining the health and cleanliness of their oral cavity, namely by brushing their teeth regularly in the correct way, and not brushing too hard, brushing their teeth using a soft-bristled toothbrush and toothpaste. containing desensitizing agents. The patient's choice of food (diet) must also be considered, where if the patient consumes acidic drinks, it is best to use a straw and can drink something neutral or alkaline such as mineral water or milk after consuming acid.

Conclusion

Smoking habits can worsen oral health and damage periodontal tissue. Periodontal tissue damage in the form of gingival recession causes dentin hypersensitivity. Dentin hypersensitivity treatment is desensitization to overcome aches and pains and is accompanied by dental health education. Patient awareness in maintaining oral hygiene so that behavioral changes occur and continuing professional treatment at the dentist is important to prevent and eliminate the causes of dentin hypersensitivity.

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