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Case report

COMPREHENSIVE DENTAL MANAGEMENT IN CHRONIC PERIODONTITIS STAGE II GRADE B WITH HYPERTENSION

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ABSTRACT

Background: Periodontitis in hypertensive patients receiving calcium channel blocker (CCB) therapy and with a smoking history can result in a complex clinical condition marked by concurrent pathological tooth mobility and gingival enlargement, necessitating a comprehensive management approach. **Purpose:** This case report discusses the management of FRC splinting in a chronic periodontitis with tooth mobility and gingivectomy. **Case:** A 39-year-old woman came to RSGM Soelastri with complaint of dental calculus that had been present for approximately 5 years. The patient reported no pain and had never received treatment. The patient had a history of hypertension, smoking habits, and orthodontic treatment performed by a non-dental professional. **Case Management:** Following scaling and root planing (SRP), grade 1 tooth mobility was identified in teeth 31, 41, and 42. Evaluation performed two weeks later revealed no signs of improvement, prompting the decision to proceed with splinting using Fiber Reinforced Composite (FRC) on the region of teeth 33 to 43. Three weeks after the splinting procedure, clinical examination demonstrated that mobility in teeth 31, 41, and 42 had been successfully eliminated. Treatment was subsequently continued with gingivectomy as the next phase in the comprehensive periodontal therapy sequence. **Conclusion:** The combination of these two procedures represents a staged and comprehensive periodontal therapeutic approach, ranging from conservative therapy to surgical intervention.

Keywords: Fiber Reinforce Composite (FRC), Gingivectomy, Hypertension, Periodontitis, Splinting

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INTRODUCTION

Periodontal disease is one of the most prevalent oral cavity diseases worldwide and represents a leading cause of tooth loss in the adult population. Based on data from the Riset Kesehatan Dasar (Riskesdas) in 2018, as many as 74.1% of Indonesian residents aged 15 years and above suffer from periodontal disease; however, ironically, only 11.2% seek treatment from medical professionals. Periodontitis, as the most destructive form of periodontal disease, is defined as a multifactorial inflammatory disease of the tooth-supporting tissues caused by the interaction between polymicrobial biofilm and the host's immune-inflammatory response. If not adequately managed, periodontitis can lead to the destruction of the periodontal ligament, alveolar bone, cementum, and gingival tissues[1].

Chronic periodontitis is a progressive inflammatory disease of the tooth-supporting tissues characterized by gingival inflammation accompanied by attachment loss, alveolar bone resorption, periodontal pocket formation, and tooth mobility that potentially leads to tooth loss[2]. Tooth mobility is one of the important parameters in assessing the integrity and functional status of the periodontium, as it reflects the extent of attachment loss and alveolar bone support[3]. If left untreated, this condition may progress to tooth loss[4].

The severity and progression of periodontitis, however, are rarely determined by local bacterial factors alone[5]. The host's inflammatory response to periodontitis does not occur in isolation, but is strongly influenced by the systemic conditions accompanying it[5]. One condition that has drawn increasing attention in hypertension, given its high prevalence in the population as well as its association with long-term pharmacological therapy that can potentially affect the periodontal tissues themselves[6].

Hypertension is known as "the silent killer" because it often occurs without any complaints from the sufferer, resulting in many undetected cases that give rise to serious complications[7]. An individual is considered hypertensive when the systolic blood pressure is ≥140 mmHg and/or the diastolic blood pressure is ≥90 mmHg, measured on two separate occasions under resting conditions (Riskesdas)[8]. Based on Riskesdas 2018 data, the prevalence of hypertension in Indonesia reaches 34.1%;

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however, 13.3% of diagnosed patients do not take medication and 32.3% do not routinely consume antihypertensive drugs. The increasing prevalence of hypertension is triggered by unhealthy lifestyles such as high-salt diets, lack of physical activity, and stress, with a higher prevalence found in age groups above 45 years and individuals with a family history of hypertension[9].

The relationship between hypertension and periodontitis extends beyond simple co-occurrence, pointing toward a bidirectional interaction. On one hand, persistent periodontal inflammation triggers the release of pro-inflammatory mediators into systemic circulation, which may impair endothelial function and raise blood pressure. On the other hand, hypertension may compromise microvascular perfusion within periodontal tissue, thereby heightening its vulnerability to inflammatory damage[10]. In patients with hypertension, the periodontal condition can be further aggravated by the consumption of antihypertensive drugs from the calcium channel blocker (CCB)[11]. Antihypertensive drugs such as amlodipine, which belongs to the calcium channel blocker, have side effects in the form of gingival enlargement with long-term use, a condition that leads to greater plaque accumulation and more severe inflammation[12]. The mechanism of this drug involves the inhibition of intracellular calcium ion influx, resulting in decreased cellular folate absorption and impaired collagenase activity in the gingival tissue[13]. In other words, the very medication used to control a hypertensive patient's blood pressure can simultaneously accelerate the periodontal chages it was never meant to cause establishing a direct, drug-medicated pathway linking hypertension management to worsening periodontal status[12].

Smoking is one of the main risk factors for various chronic diseases, such as lung cancer, heart disease, stroke, and diabetes, which cause death. Notably, smoking is also an estebilishied risk factor for hypertension itself and its coexistence with antihypertensive therapy is common in clinical practice[14]. Harmful substances in cigarettes, such as nicotine, tar, and carbon monoxide, can damage periodontal tissues through increased oxidative stress, impairment of the body's defenses, and reduced salivary function that plays a role in protecting oral cavity tissues[15]. These toxic and carcinogenic tobacco products progressively damage the attachment of the gingiva to the alveolar bone and cementum, which ultimately contributes to the overall progression of periodontal tissue destruction[16]. Thus, the combination of risk

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factors in the form of hypertension with antihypertensive drug consumption and smoking habits in periodontitis patients creates a complex clinical condition, in which pathological tooth mobility and gingival enlargement can occur simultaneously, therefore requiring a comprehensive management approach[16].

The management of periodontitis accompanied by tooth mobility and gingival enlargement requires a comprehensive therapeutic approach. Periodontal splinting is a non-invasive and cost-effective therapy to stabilize teeth experiencing mobility, where the main goal of splinting is to distribute the occlusal load evenly, thereby improving patient comfort, aesthetic function, and oral hygiene[17]. Various splinting materials are available to manage teeth with pathological mobility due to periodontitis. The choice of splinting materials ranges from ligature wire to fiber reinforced composite[18]. Commonly used materials include ligature wire, night guard, interim fixed prostheses, and composite resin with or without wire or fiber inserts[19]. The use of Fiber Reinforced Composite (FRC) as a splinting material is increasingly popular because it is minimally invasive, has good aesthetics, and possesses a modulus of elasticity that resembles dentin, thus being able to distribute functional stress more physiologically compared to conventional splint materials that are more rigid[20].

In patients presenting gingival enlargement associated with the long-term use of calcium channel blocker antihypertensive medications, gingivectomy may be indicated as a corrective surgical procedure after the periodontal condition has been stabilized[13]. Gingivectomy is performed to eliminate excessively enlarged gingival tissue, restoring the gingival contour and preventing the recurrence of plaque accumulation around the gingival enlargement area[21]. Splinting therefore addresses the mechanical consequence of periodontal destruction (tooth mobility), while gingivectomy addresses the pharmacologically-driven consequence of hypertension management (gingival enlargement)[22]. Thus, the combination of splinting as a stabilization therapy for tooth mobility followed by gingivectomy as a corrective therapy for gingival enlargement represents the appropriate management approach in cases of periodontitis with systemic risk factors in the form of hypertension and smoking habits.

CASE

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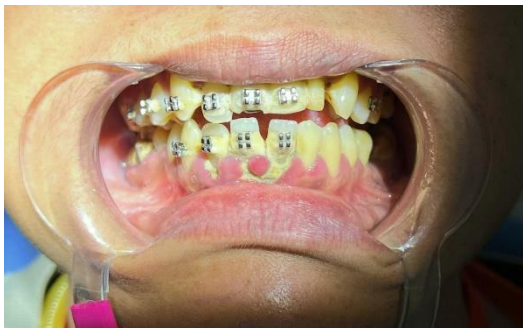
A 39-year-old woman presented to the Soelastri Dental and Oral Hospital with complaints of dental calculus on the lower anterior teeth for the past 5 years. She also reported swollen gums and frequent gingival bleeding during tooth brushing. In addition, the patient also reported that her lower anterior teeth had been mobile for the past year, causing discomfort during eating, and no treatment had been performed. The



patient reported a history of hypertension and was currently consuming antihypertensive medication (amlodipine) once daily before bedtime. The patient had previously undergone dental scaling approximately 6 years ago.

The patient was reported to have undergone fixed orthodontic treatment performed by a non-dental professional, with the brackets remaining in place and not removed in accordance with clinical procedures. The patient brushes her teeth twice daily while bathing using a horizontal brushing technique. The patient had a smoking habit of approximately five cigarettes per day, as well as a habit of unilateral chewing on the right side, adopted due to complaints of pain in the upper left posterior teeth.

On extraoral examination, no

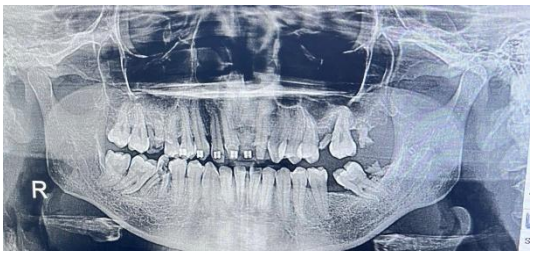


Figure 1. Panoramic radiographic view

abnormalities were found. Intraoral examination revealed the presence of supragingival and subgingival calculus across almost all teeth, particularly in the mandibular anterior segment with a poor Oral Hygiene Index (OHI) score. Edema accompanied by a shiny red discoloration was present on the labial gingival margin of teeth 32, 31, 41, 42, and 43, and teeth 32, 41, & 42 presenting with grade 1 mobility.

Radiographic examination results revealed the presence of horizontal alveolar bone loss in both the maxilla and mandible. Based on the subjective and objective examination findings, the diagnosis in this case was chronic periodontitis stage II grade B. The treatment plan to be carried out includes initial therapy consisting of scaling and root planing, followed by splinting treatment in the area of teeth 33, 32, 31, 41, 42, and 43, and subsequently gingivectomy. The prognosis in this case is

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categorized as fair due to the presence of horizontal bone loss and the patient's habit as a heavy smoker.

CASE MANAGEMENT

During first visit, probing depth examination was performed, OHI 5.2, GI 0.95, and PCR 74.25%. Scaling and root planing were then carried out in all regions. Two weeks later, splinting was performed on teeth 33, 32, 31, 41, 42, & 43.

During first visit, probing depth was performed, OHI 6.6, indicating a poor category; GI 0.95 indicating a mild gingivitis category; and PCR 74.25% indicating a poor category. Scaling and root planing were then carried out in all regions. Two weeks later, splinting was performed on teeth 33, 32, 31, 41, 42, & 43.

Figure 3. Clinical appearance of the patient after initial therapy

The second visit began with an explanation of the splinting procedure to the patient, who was then asked to provide written consent by signing an Informed Consent form. Following that, all instruments and materials required for the splinting procedure were prepared in advance. As an initial step before the main procedure, the teeth were first cleaned through a prophylaxis procedure using a mixture of paste and pumice applied with a

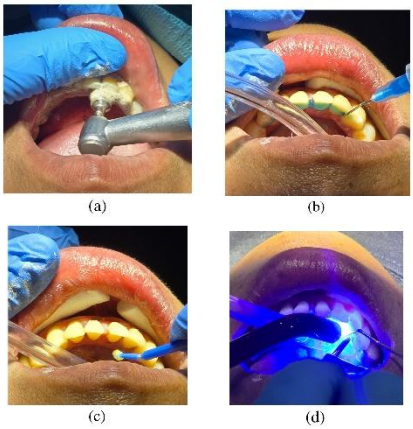

Figure 4. (a) Prophylaxis; (b) Etching application; (c) Bonding application; (d) Application of composite resin on the fiber ribbon

brush, then rinsed and dried.

The next stage was to determine the working length by measuring with dental floss stretched from the distal side of tooth 33 to the distal side of tooth 43, which was then cut to the obtained measurement. The composite fiber ribbon was then cut to match that length and soaked in bonding agent. Subsequently, 37% phosphoric acid

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was applied as an etching agent to the working area, left for 30 seconds, then thoroughly rinsed and dried. The working area was then isolated using a cotton roll, after which bonding agent was applied to the lingual surfaces of teeth 33, 32, 31, 41, 42, and 43 using a microbrush and light-cured for 10 seconds.

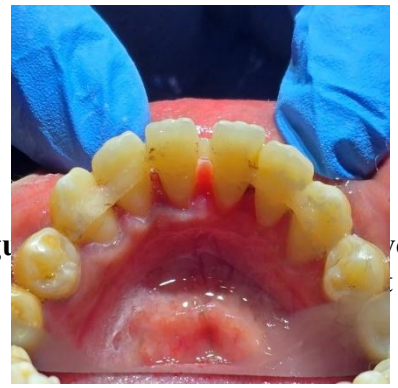
The procedure was completed by applying a thin layer of flowable resin onto the bonded surfaces, after which the composite fiber was positioned on the lingual aspect following the contour of each tooth with the aid of a plastic instrument.

Figure 5. Clinical appearance of the patient after splinting

Over the fiber, flowable resin was once again applied in a thin layer and then light-cured for 20 seconds until hardened.

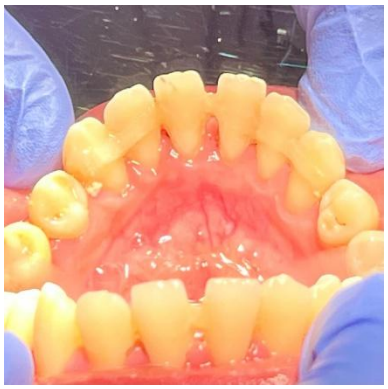
Occlusal evaluation was performed by instructing the patient to perform biting and chewing movements using articulating paper, while also asking whether there was any sensation of interference, sharpness, or discomfort in the area. The procedure was then continued with the finishing and polishing stage. The patient was instructed to avoid eating for one hour after the splinting.

The third visit was a follow up session conducted two weeks after splinting procedure. The patient reported no complaints, and the splinting remained well-seated and retentive. Intraoral



Figu eeks

examination results showed that teeth 31, 41, and 42 no longer exhibited mobility, and the FRC splinting remained perfectly adhered across the range of teeth 33 to 43. Pocket depth measurements revealed that tooth 42 had a depth of 5 mm, teeth 41 and 31 each measured 2 mm, while tooth 32 was recorded at 3 mm. The gingival index result of 0.53 indicated the presence of mild



gingivitis, whereas the plaque index score of 21.51% reflected moderate plaque

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accumulation. These overall findings reflect the success of FRC splinting treatment in stabilizing the mobility of teeth 31, 41, and 42, allowing the subsequent management in the form of gingivectomy on tooth 42 to be planned.

Before the gingivectomy procedure was initiated, the operator first explained the treatment procedure to the patient and requested the patient to sign the medical consent form (Informed Consent). Subsequently, all instruments and materials required for the gingivectomy procedure were prepared.

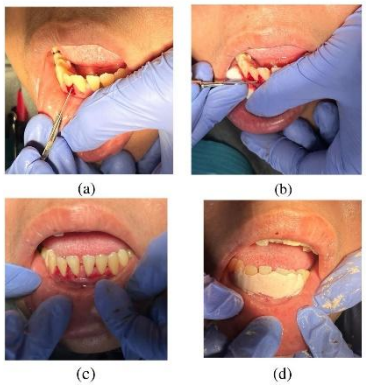
The working area was then dried and asepsis was performed using povidone iodine, followed by the application of topical anesthesia in the form of benzocaine. Thereafter, infiltration anesthesia technique at a 45-degree angle was administered at the mucolabial fold area of teeth 42, 41, 31, and 32. Bleeding points were determined using a pocket marker as an incision guide. Incision of the hyperplastic tissue was performed using blade number 15 or a Kirkland knife on the facial side, and an Orban knife on the interdental side. The hyperplastic tissue was then excised using blade number 15.

The next stage was root planing, which aimed to remove residual calculus and necrotic tissue using Gracey curette number 1-2, designated for anterior teeth. The curette was inserted parallel to the axial

Figure 7. (a) Gingivectomy; (b) Gingivoplasty; (c) Post-gingivectomy; (d) Coepack placement

axis of the tooth, with the cutting edge facing the tooth surface at a 45-degree angle. Curette strokes were performed in vertical, oblique, and horizontal directions to ensure that the entire root surface was thoroughly clean.

Gingivoplasty to reshape the gingival contour according to anatomy was performed using a number 15 blade. The



gingival surface in the working area was then scraped to remove tissue debris. Irrigation was carried out using saline solution, followed by bleeding control and tissue adaptation using sterile gauze pressed for 2–5 minutes. The working area was then dried with sterile gauze and subsequently covered with a periodontal pack to prevent infection.

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Figure 5. Clinical appearance of the patient after gingivectomy

The medications prescribed consisted of amoxicillin and mefenamic acid, accompanied by post-gingivectomy care education. The patient was instructed to take the medications as prescribe, to avoid eating and drinking for one hour after the procedure, and to keep the periodontal pack in place for one week. In addition, the patient was advised to maintain oral hygiene by brushing teeth twice daily, while avoiding brushing in the surgical area. The patient was also asked to refrain from consuming hot food and beverages for 24 hours, to avoid vigorous rinsing, and to attend follow-up visits as scheduled.

At the fourth visit, two weeks after the procedure, a second follow-up examination was conducted. Based on the subjective assessment, the patient reported no complaints. The objective examination revealed that the gingiva had healed well, the gingival contour was anatomically appropriate in the anterior mandibular region, the gingival margin was beginning to appear well-defined, and no edema or bleeding was detected.

DISCUSSION

This case report presents a patient diagnosed with chronic periodontitis stage II grade B based on anamnesis, clinical examination, and radiographic findings. Periodontitis is a multifactorial inflammatory disease of the tooth-supporting tissues that arises from the interaction between polymicrobial biofilm and the host's immune-inflammatory response, which if inadequately managed leads to duestruction of the periodontal ligament, alveolar bone, cementum, and gingival tissue[23].

Calculus, horizontal tooth-brushing technique, and orthodontic brackets that weere not removed in accordance with clinical procedures served as local risk factors that promoted biofilm accumulation, impeded adequate interdental cleaning, and increased the risk of periodontitis progression. Among systemic risk factors, smoking is the most significant. Smoking contributes to the progression of periodontal disease through immunomodulatory mechanisms, including

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reduced chemotaxis and phagocytosis of polymorphonuclear neutrophils, impaired gingival vascularization, and dysregulation of cytokine profiles through increased expression of interleukin-1β and TNF-α[24]. All of these mechanisms synergistically drive the progressive destruction of periodontal tissues[25].

In addition to smoking, the patient's history of hypertension managed with amlodipine as a calcium channel blocker (CCB) represented the second significant systemic risk factor in this case. Amlodipine has been consistently associated with drug-influenced gingival fibroblast, resulting in reduced collegenase activity, excessive extracelluler matrix accumulation, and upregulated expression of IL-13 and procollagen that promotes fibrosis of the gingival connective tissue[26]. The clinical presentation observed in this case—edema accompanied by shiny, erythematous discoloration of the gingival margin, is more characteristic of plaque-induce inflammatory enlargement secondary to local irritants, such as calculus and retained orthodontics brackets, than of the classically described CCB-induced overgrowth, which typically presents as a firm, pale pink, lobulated, and less hemorrhagic tissue enlargement[12]. This suggests a combined etiology, in which pre-existing local inflammation resulting from poor oral hygene was amplified by the fibrotic effect of amlodipine on the gingival connective tissue[27]. Furthermore, the relationship between hypertension and periodontitis is bidirectional in nature: persistent periodontal infection reduces nitric oxide bioavailability in impairs endothelial funtion, while hypertension-associated vascular changes compromise gingival microcirculation and increase tissue susceptibility to plaque-induced inflammation. These findings indicate that the patient's hypertensive status was not merely an incidental comorbidity, but independently contributes to the severity of periodontal inflammation.

Initial phase therapy through scaling and root planing (SRP) was performed to eliminate calculus deposit and minimize the inflammatory response of the periodontal tissue[28]. As adjunctive therapy, splinting was performed on teeth 33, 32, 31, 41, 42, and 43 using Fiber Reinforce Composite (FRC) applied to the lingual surfaces. This material was selected due to its minimally invasive and aesthetic nature, its requirement for no extensive hard tissue preparation, and its modulus of elasticity that closely approximates dentinal structure, thereby enabling a more physiological distribution of functional

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stress[20]. Although splinting has been hown to improve stability and funtional comfort, it does not eliminate the primary etiology of periodontal disease and may complicate plaque control in the vicinity of the splint[29]. This concern was particularly relevant in the present case, given that amlodipine induced gingival enlargement indenpendently facilitated plaque retention, contributing to the persistence of periodontal pocketing on tooth 42 despite adequate non-surgical therapy. Therefore, comprehensive oral hygiene instruction and scheduled routine follow up apointments are essential to prevent recurrence of periodontal inflammation[30].

Gingivectomy on tooth 42 was performed as a corrective procedure following post-splinting evaluation, which revealed a residual pocket depth of 5 mm that had not responded to non-surgical therapy. In this case, gingivectomy aimed to eliminate pathologically enlarged gingival tissue, reduce periodontal pocket depth, and restore physiological gingival contour[21]. Considering that the gingival enlargement was in all likelihood influenced by the ongoing amlodipine therapy, gingivectomy addressed the consequences rather than the underlying causative factor[31]. Without reassessment of the antihypertensive regimen in coordination with the treating physician, or without rigorous plaque control, reccurence of gingival enlargement remains an anticipated risk. This underscores the improtance of interdiciplinary collaboration between dental and medical practioners in the long-term management of hypertensive patient receiving CCB therapy[32].

In conclusion, the periodontal condition in this case represents the convergence of multiple contributing factors: local irritants that initiated and sustained inflammation, smoking that impaired periodontal tissue defense and healing capacity, and long term amlodipine therapy that amplified gingival enlargement through its fibrotic effect on the connetive tissue. Accordingly, the combination of FRC splinting and gingivectomy in this case represents a staged and comprehensive periodontal therapeutic approach that look into account the patient's hypertensive status and pharmacological therapy, with the goal of achieving a healthy, stable, and long term maintainable periodontal condition.

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