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Gingival Curettage As An Adjunctive Non-Surgical Approach In Localized Chronic Periodontitis Of Tooth 43: A Case Report

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Abstract

Periodontitis is a chronic inflammatory disease that causes destruction of tooth-supporting tissues due to interactions between subgingival biofilm and the host immune response. This case report aimed to evaluate the effectiveness of gingival curettage as an adjunctive therapy in localized chronic periodontitis. A patient presented with complaints of swollen gingiva and bleeding during tooth brushing. Clinical examination revealed erythematous gingiva on tooth 43 with periodontal pocket depths of 3 mm mesiolabially, 2 mm midfacially, and 4 mm distolabially, along with an Oral Hygiene Index score of 3.3. Initial periodontal therapy consisting of scaling and root planing was performed, followed by gingival curettage due to persistent inflammation and positive bleeding on probing. Three weeks after treatment, periodontal healing was observed, characterized by negative bleeding on probing and reduced periodontal pocket depths to 2 mm at all examined sites. Gingival curettage can be considered an effective adjunctive treatment to eliminate inflammatory tissue, improve periodontal conditions, and enhance tooth prognosis when supported by optimal oral hygiene maintenance.

Keywords: Gingival Curettage, Localized Chronic Periodontitis, Periodontal Pocket, Periodontal Healing

INTRODUCTION

Periodontal health is defined as a state free from active inflammation that allows the periodontium to function optimally, both in intact tissues and in tissues that have experienced structural loss due to previous disease (Anwaristi, 2025). Periodontal disease commonly begins with untreated gingivitis and may progress to periodontitis, a condition characterized by the destruction of tooth-supporting tissues, including the periodontal ligament and alveolar bone. The progression of gingivitis and periodontitis is primarily influenced by subgingival biofilm. Under homeostatic conditions, the oral microbiota exists as a commensal community; however, when dysbiosis occurs, pathogenic bacteria such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* become dominant, triggering inflammatory responses and periodontal tissue destruction (Anwaristi and Nugrahani, 2026).

Periodontitis is a chronic inflammatory disease characterized by progressive destruction of the tooth-supporting tissues resulting from complex interactions between subgingival microbial biofilms and the host immune response (Abdulkareem et al., 2023). Currently, periodontitis is no longer regarded solely as an infection caused by bacterial accumulation, but rather as a condition associated with oral microbial dysbiosis that induces an excessive inflammatory response, leading to the destruction of the periodontal ligament, root cementum, and alveolar bone. This destructive process results in clinical attachment loss, periodontal pocket formation, alveolar bone resorption, tooth mobility, and eventually tooth loss if not properly managed (Basic and Dahlén, 2023).

Periodontitis remains one of the most prevalent oral diseases worldwide and has a significant impact on oral health and patients' quality of life. Common clinical manifestations include gingival inflammation, bleeding on probing, increased periodontal pocket depth, loss of periodontal attachment, and reduction in alveolar bone height. In localized chronic periodontitis, periodontal tissue destruction occurs in a limited area involving less than 30% of the teeth in the oral cavity. Although localized in nature, this condition may progress to more extensive periodontal destruction if etiological factors are not adequately controlled (Wei et al., 2024).

The primary objectives of periodontitis therapy are to eliminate disease-causing factors, control inflammation, prevent further periodontal tissue destruction, and maintain teeth in a functional state

for as long as possible. Non-surgical periodontal therapy through scaling and root planing is considered the initial standard treatment because of its effectiveness in reducing subgingival biofilm and calculus deposits (Purbowati and Kurniawan, 2022). However, in some cases with persistent periodontal pockets, inflammatory granulation tissue may remain and interfere with periodontal healing. Therefore, additional procedures may be required to remove pathological tissues and establish a more favorable environment for periodontal tissue repair and regeneration (Ren et al., 2023).

Gingival curettage is a periodontal procedure aimed at removing the pocket epithelium and inflamed granulation tissue from the soft tissue wall of periodontal pockets. Elimination of inflammatory tissue is expected to reduce pocket depth, improve gingival adaptation to the root surface, and support periodontal tissue healing (Ng et al., 2021). Although modern periodontal therapy increasingly emphasizes minimally invasive approaches, gingival curettage may still be considered as an adjunctive treatment in selected cases demonstrating persistent inflammation following conventional non-surgical periodontal therapy (Su et al., 2023). This case report describes gingival curettage treatment on tooth 43 diagnosed with localized chronic periodontitis. The procedure was performed as an approach to eliminate inflammatory tissue within the periodontal pocket, improve the clinical condition of the periodontal tissues, support the healing process, and maintain the function and prognosis of the affected tooth.

RESULTS AND DISCUSSION

The patient presented with a chief complaint of swollen gingiva and easy bleeding. The patient reported that these symptoms had been experienced since school age. The patient frequently noticed reddish gingiva and discomfort during tooth brushing, particularly on the right side. The patient also complained of dental crowding, which caused plaque accumulation and made oral hygiene maintenance more difficult. The patient had a history of professional dental scaling approximately one month prior. Based on the medical history, no systemic diseases were reported.

Intraoral examination revealed erythematous gingiva accompanied by gingival enlargement around tooth 43. The periodontal pocket depths of tooth 43 were recorded as 3 mm at the mesiolabial site, 2 mm at the midfacial site, and 4 mm at the distolabial site. The Oral Hygiene Index (OHI) score was 3.3, indicating a moderate level of oral hygiene status (Figure 1). Based on the clinical findings, the patient was diagnosed with localized chronic periodontitis. The prognosis was considered favorable due to the absence of systemic diseases, high patient motivation in maintaining oral hygiene, and good cooperation throughout the treatment.

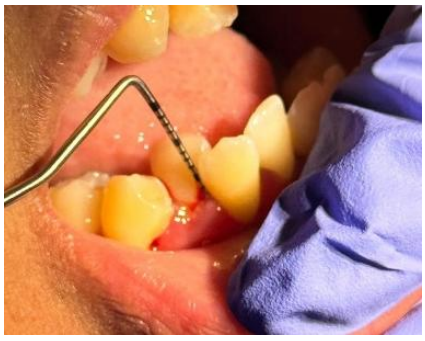

Figure 1. Initial condition of tooth 43

Based on the anamnesis and clinical examination, the initial treatment performed was periodontal initial therapy consisting of scaling and root planing. The patient was instructed to return after one week for post-treatment evaluation. During the second visit, subjective examination revealed that the patient still experienced gingival bleeding during tooth brushing. Intraoral examination showed persistent gingival erythema, slight gingival enlargement, and positive bleeding on probing (BOP). Periodontal pocket measurements of tooth 43 remained at 3 mm mesiolabially, 2 mm

midfacially, and 4 mm distolabially. Therefore, gingival curettage was planned as an adjunctive periodontal treatment.

Prior to the procedure, the treatment area was anesthetized using local infiltration anesthesia with Pehacain solution. Gingival curettage in the anterior region of tooth 43 was performed using a Gracey curette number 1–2. The curette was inserted parallel to the long axis of the tooth, with the cutting edge facing the gingival wall of the pocket, and advanced until reaching the base of the periodontal pocket. The instrument was then moved in an occlusal direction to remove inflamed tissue and granulation tissue. The procedure was performed systematically on both the mesial and distal periodontal pocket areas.

After completion of the curettage procedure, the surgical area was irrigated with 0.9% saline solution and distilled water until clean. The treated area was subsequently cleaned and dried using sterile gauze. Gingival adaptation to the tooth surface was achieved by applying gentle pressure toward the tooth surface using a finger for approximately 1–3 minutes. Metronidazole gel was then applied to the post-operative area.

A periodontal pack in the form of two paste tubes, such as Coe-Pak®, was prepared by mixing equal amounts of the base paste and accelerator paste until a homogeneous color was achieved. Prior to application, the periodontal pack was shaped into a rod-like form according to the length of the surgical area requiring coverage. The material was then placed over the wound area and adapted according to the gingival contour and interproximal areas. On the vestibular surface, adaptation was performed with assistance from the patient's lips or cheeks to prevent the material from adhering to the operator's fingers. The periodontal pack was extended to cover part of the tooth surface and surrounding gingiva.

After completion of the procedure, the surgical area was cleaned, and the patient received post-curettage instructions. Medications prescribed included amoxicillin 500 mg three times daily and sodium diclofenac 50 mg twice daily. The first evaluation was performed one week after gingival curettage. Subjectively, the patient reported no pain in the treated area, and the periodontal pack remained intact. Objective examination revealed that the gingiva was still slightly erythematous, with the presence of debris and calculus. The OHI-S score was 1.4, categorized as good oral hygiene status. Complete healing was observed during the third follow-up visit, performed three weeks after gingival curettage. Clinical examination showed negative bleeding on probing (BOP), with periodontal pocket depths of tooth 43 measuring 2 mm at the mesiolabial, midfacial, and distolabial sites.

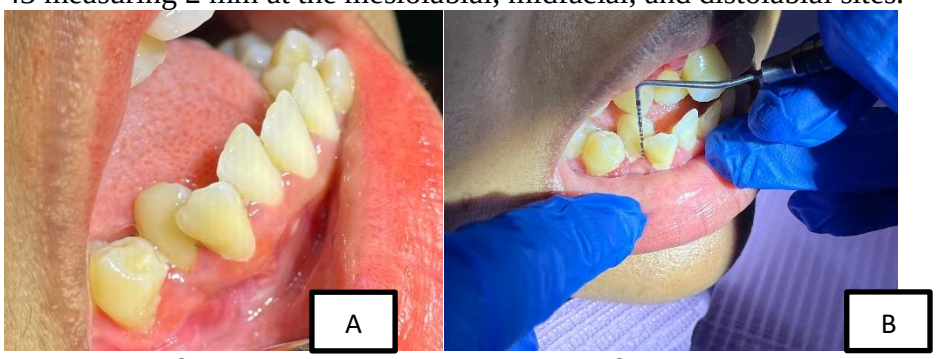

Figure 2. Post-curettage follow-up results; A) one week after surgery, B) three weeks after surgery

Discussion

Periodontitis is characterized by inflammation of the periodontal tissues, resulting in periodontal pocket formation and loss of attachment of the tooth-supporting structures. This condition develops due to the accumulation of subgingival bacterial biofilm, which triggers immune responses and chronic inflammation within the periodontal tissues (Putri et al., 2024). Bacterial products, such as lipopolysaccharides (LPS), activate immune cells, leading to the release of various inflammatory mediators, including interleukin-1β (IL-1β), interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and matrix metalloproteinases (MMPs). These mediators contribute to the degradation of periodontal

ligament collagen and stimulate osteoclast activity, resulting in alveolar bone resorption. Consequently, periodontal pocket depth increases, providing a favorable environment for bacterial retention and accelerating disease progression if adequate intervention is not performed (El Mobadder et al., 2022)

The treatment provided in this case involved gingival curettage, which aims to remove inflammatory granulation tissue and infected pocket epithelium from the soft tissue wall of the periodontal pocket. Granulation tissue in periodontitis consists of chronic inflammatory cell infiltration, abnormal newly formed blood vessels, and degraded collagen resulting from proteolytic enzyme activity (Wang et al., 2025). The persistence of this pathological tissue may maintain the inflammatory process and interfere with periodontal healing. Therefore, removal of granulation tissue through curettage is expected to reduce the local inflammatory burden and create a more favorable periodontal environment for tissue repair. Furthermore, elimination of the pocket epithelium aims to minimize the recurrence of inflamed epithelial tissue and facilitate gingival adaptation to the cleaned root surface (Walther et al., 2024).

The success of gingival curettage is strongly influenced by the periodontal wound healing process following the procedure. Healing begins with the hemostatic phase, which occurs immediately after tissue curettage (Fraser et al., 2022) During this phase, vasoconstriction and blood clot formation occur, serving as a temporary matrix for the migration of healing cells. The blood clot contains platelets that release various growth factors, including platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), and vascular endothelial growth factor (VEGF). These growth factors play important roles in recruiting fibroblasts, endothelial cells, and progenitor cells to the wound area. This phase is crucial as it provides the foundation for the formation of new tissue in damaged periodontal areas (Graziani et al., 2024).

Following the hemostatic phase, the healing process proceeds to the inflammatory phase, which occurs during the first few days after treatment. During this phase, neutrophils and macrophages migrate to the wound site to phagocytose bacteria, necrotic tissue, and remaining debris. Macrophages also function as key regulators of wound healing by producing various cytokines and growth factors that initiate the formation of healthy granulation tissue. Reduction of microorganisms and inflammatory tissue after gingival curettage allows the inflammatory response to become more controlled compared with the pre-treatment condition, thereby facilitating a more optimal tissue regeneration process (Wibowo et al., 2024).

The subsequent phase is the proliferative phase, which generally begins on the third day and continues for several weeks after the procedure. During this phase, fibroblast proliferation, new collagen fiber formation, angiogenesis, and epithelial migration occur to cover the wound surface. Fibroblasts derived from periodontal tissues synthesize extracellular matrix components and type I collagen, which contribute to strengthening the gingival connective tissue. The formation of new blood vessels is essential to provide oxygen and nutrients required for tissue repair. Clinically, this phase is characterized by reduced signs of inflammation, decreased bleeding on probing, and reduction of periodontal pocket depth due to tissue contraction and new connective tissue formation (Windakhrisma et al., 2024).

The maturation or remodeling phase may continue for several months, during which newly formed collagen fibers undergo reorganization and increased tensile strength. The newly established junctional epithelium adapts to the root surface, resulting in more stable tissue attachment. In most gingival curettage cases, healing commonly results in the formation of a long junctional epithelium and connective tissue repair, although complete regeneration involving the formation of new cementum, periodontal ligament, and alveolar bone does not always occur. Nevertheless, successful elimination of inflammatory tissue provides clinical benefits, including reduced periodontal pocket depth, decreased bleeding on probing, improved clinical attachment, and establishment of a healthier periodontal condition that can be maintained by the patient (Herawati and Olivia, 2022).

The application of metronidazole gel may be used as an adjunctive therapy following gingival curettage to enhance the elimination of periodontal pathogenic bacteria. Metronidazole is a nitroimidazole-class antibiotic with bactericidal activity, particularly against Gram-negative anaerobic bacteria commonly found in periodontal pockets, including *Porphyromonas gingivalis*, *Prevotella intermedia*, *Fusobacterium nucleatum*, and *Tannerella forsythia*. These microorganisms play important roles in periodontitis pathogenesis through the production of virulence factors that induce periodontal tissue destruction and sustain chronic inflammatory responses (Zussman and Zilberman, 2022).

The use of metronidazole in gel form provides advantages by delivering high local antibiotic concentrations within periodontal pockets while minimizing the risk of systemic adverse effects associated with oral antibiotic administration. When applied into periodontal pockets, the gel provides sustained drug release, maintaining effective antimicrobial concentrations for a longer duration. The antibacterial mechanism of metronidazole involves the reduction of its nitro group by anaerobic bacteria, producing cytotoxic metabolites. These metabolites interact with bacterial DNA and inhibit nucleic acid synthesis, ultimately resulting in bacterial cell death (Gandhi et al., 2025).

The treatment outcome in this case demonstrated improvement in periodontal tissue conditions, characterized by reduced gingival inflammation, decreased periodontal pocket depth, and improved periodontal health surrounding the affected tooth. The success of therapy was not solely determined by the gingival curettage procedure but was also influenced by effective plaque control, patient compliance in maintaining oral hygiene, and regular follow-up visits for evaluation of healing and prevention of periodontal disease recurrence. Through comprehensive elimination of etiological factors and pathological tissues, the prognosis of the affected tooth can be maintained, and the progression of periodontitis can be optimally controlled.

CONCLUSION

Gingival curettage on tooth 43 with localized chronic periodontitis was effective as an adjunctive therapy for removing inflammatory tissue and supporting periodontal healing. This procedure contributed to the reduction of periodontal pocket depth, improvement of periodontal tissue health, and enhancement of the tooth prognosis when accompanied by adequate plaque control and proper periodontal maintenance.

REFERENCES

Abdulkareem, A. A., Al-Taweel, F. B., Al-Sharqi, A. J. B., Gul, S. S., Sha, A., and Chapple, I. L. C. 2023. Current concepts in the pathogenesis of periodontitis: from symbiosis to dysbiosis. *Journal of Oral Microbiology*. *15*(1). 2197779.

Anwaristi, A. Y. 2025. Efektivitas Ekstrak Kulit Salak Pondoh Berbagai Konsentrasi dalam Menghambat Bakteri Penyebab Periodontitis (Kajian In Vitro). *Jurnal Bidang Ilmu Kesehatan*. *15*(4). 312–323.

Anwaristi, A. Y., and Nugrahani, N. A. 2026. *Diagnosis Klinis dan Imunologi Penyakit Periodontal*. UMS Press.

Basic, A., and Dahlén, G. 2023. Microbial metabolites in the pathogenesis of periodontal diseases: a narrative review. *Frontiers in Oral Health*. *4*. 1210200.

El Mobadder, M., Nammour, S., Matys, J., and Grzech-Leśniak, K. 2022. Sodium hypochlorite and diode laser in non-surgical treatment of periodontitis: Clinical and bacteriological study with real time polymerase chain reaction (PCR). *Life*. *12*(10). 1637.

Fraser, D., Caton, J., and Benoit, D. S. W. 2022. Periodontal wound healing and regeneration: insights for engineering new therapeutic approaches. *Frontiers in Dental Medicine*. *3*. 815810.

Gandhi, U. H., Vyas, S. D., Mane, V., Patel, S. N., Patadiya, H. H., Kumar, S., and Haque, M. 2025.

The effectiveness of metronidazole as a localized drug delivery system in the treatment of periodontal diseases: a narrative review. *Cureus*. *17*(3).

Graziani, F., Izzetti, R., Perić, M., Marhl, U., Nisi, M., and Gennai, S. 2024. Early periodontal wound healing after chlorhexidine rinsing: a randomized clinical trial. *Clinical Oral Investigations*. *28*(6). 354.

Herawati, D., and Olivia, N. 2022. Gingival Curettage for the Management of Chronic Periodontitis: A Case Report. *KnE Medicine*. 370–376.

Ng, E., Tay, J. R. H., and Ong, M. M. A. 2021. Minimally invasive periodontology: a treatment philosophy and suggested approach. *International Journal of Dentistry*. *2021*(1). 2810264.

Purbowati, B., and Kurniawan, A. A. 2022. Periodontitis kronis pada pasien dengan penyakit diabetes melitus. *Majalah Kedokteran Gigi Klinik*. *7*(2). 71. https://doi.org/10.22146/mkgk.37775

Putri, S. A., Astuti, L., Komala, O. N., Kedokteran, F., and Universitas, G. 2024. *Periodontitis pada lansia*. *6*(2). 36–38.

Ren, J., Fok, M. R., Zhang, Y., Han, B., and Lin, Y. 2023. The role of non-steroidal anti-inflammatory drugs as adjuncts to periodontal treatment and in periodontal regeneration. *Journal of Translational Medicine*. *21*(1). 149.

Su, Y., Ye, L., Hu, C., Zhang, Y., Liu, J., and Shao, L. 2023. Periodontitis as a promoting factor of T2D: current evidence and mechanisms. *International Journal of Oral Science*. *15*(1). 25.

Walther, K., Gröger, S., Vogler, J. A. H., Wöstmann, B., and Meyle, J. 2024. Inflammation indices in association with periodontitis and cancer. *Periodontology 2000*. *96*(1). 281–315.

Wang, Z., Saxena, A., Yan, W., Uriarte, S. M., Siqueira, R., and Li, X. 2025. The impact of aging on neutrophil functions and the contribution to periodontitis. *International Journal of Oral Science*. *17*(1). 10.

Wei, X., Zhang, X., Chen, R., Li, Y., Yang, Y., Deng, K., Cai, Z., Lai, H., and Shi, J. 2024. Impact of periodontitis on type 2 diabetes: a bioinformatic analysis. *BMC Oral Health*. *24*(1). 635.

Wibowo, R., Lastianny, S. P., Ardhani, R., Handajani, J., and Syaify, A. 2024. The Effect of Local Administration of Metronidazole from Carbonate Apatite-gelatin Film on the Healing of Chronic Periodontitis Post-Curettage. *Malaysian Journal of Medicine & Health Sciences*. *20*.

Windakhrisma, A. A., Ardhani, R., Koes Soesilowati, A. S., Handajani, J., and Syaify, A. 2024. The Healing of Post-curettage Chronic Periodontitis on the Implantation of Carbonate Apatite-gelatin Film as a Chlorhexidine Delivery System (A Randomized-Controlled Trial). *Malaysian Journal of Medicine & Health Sciences*. *20*.

Zussman, M., and Zilberman, M. 2022. Injectable metronidazole-eluting gelatin-alginate hydrogels for local treatment of periodontitis. *Journal of Biomaterials Applications*. *37*(1). 166–179.