Jurnal Kesehatan Gigi 12 Nomor 1 (2025) 46 -51

Jurnal Kesehatan Gigi

p-ISSN: 2407-0866
e-ISSN: 2621-3664

http://ejournal.poltekkes-smg.ac.id/ojs/index.php/jkg/index

Generalized Chronic Periodontitis in Elderly Heavy Smokers

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ABSTRACT

Periodontitis is one of the most common periodontal diseases affecting both elderly individuals and heavy smokers. This case report aims to describe the management of a geriatric patient with generalized chronic periodontitis and a history of heavy smoking. A 63-year-old male patient presented with a chief complaint of mobile teeth that had persisted for the past three months. Intraoral examination revealed necrosis, cervical caries, gingival recession, and poor oral hygiene. The treatment included extraction of the mobile teeth and socket curettage to remove necrotic tissue. The patient was prescribed antibiotics and analgesics, followed by a one-week post-operative evaluation. The patient reported no pain or swelling after extraction, and the healing process was satisfactory, with no signs of infection. The patient was also informed about a long-term treatment plan involving the fabrication of a prosthesis. Tooth extraction in patients with chronic periodontitis requires careful consideration of both clinical and non-clinical factors. This case highlights the importance of a comprehensive approach in the management of periodontitis in elderly heavy smokers.
Keyword : Generalized chronic periodontitis, elderly, nicotine, tooth extraction

Introduction

Periodontal disease remains a significant public health concern in Indonesia. According to the 2018 Basic Health Research (RISKESDAS), 74.1% of Indonesians aged over 15 years were diagnosed with periodontitis [1]. More recent data from 2022 indicate that the prevalence of periodontal disease among this population ranges from 19.6% to 27.3% [2]. Concurrently, there has been a noticeable rise in the number of active smokers, particularly those classified as heavy smokers, within the 15–64 year age group between 2019 and 2020 (Munandar, 2022) [3].

Cigarette smoke contains nicotine, tar, and more than 4,000 chemical substances that can adversely affect biological processes, including oxidation, inflammation, and carcinogenesis. In dentistry, smoking is strongly associated with a number of oral health conditions, the most common being periodontal disease, candidiasis, dental caries, and precancerous lesions [4]. These findings underscore the importance of addressing smoking as a major risk factor in the prevention and management of periodontal disease, particularly in geriatric populations with long-term exposure to tobacco.

Periodontal disease is a chronic inflammatory condition that leads to the destruction of the supporting structures of the teeth [5]. It is the leading cause of tooth loss among adults worldwide. Data collected since 2017 indicate that approximately 796 million people suffer from severe periodontitis globally [6]. Periodontitis is classified into two major types: aggressive periodontitis, which typically affects younger individuals and progresses rapidly, and chronic periodontitis, which is more prevalent but exhibits slower progression [7].

Chronic periodontitis is an inflammatory disease that damages the periodontal ligament and alveolar bone, which support the teeth [8]. Based on the extent of tissue involvement, periodontitis is further categorized into localized periodontitis—where less than 30% of teeth are affected—and generalized periodontitis, which involves more than 30% of the dentition [9].

Multiple caries, or rampant caries, refers to rapidly progressing carious lesions that affect nearly all tooth surfaces. In adults, caries are commonly found on the proximal and labial surfaces of the mandibular incisors, due to the difficulty in cleaning these areas, which makes them more susceptible to plaque and bacterial accumulation [10].

A study by Strauss et al. (2019) reported that individuals with dental caries are more likely to develop periodontitis, particularly severe periodontitis. Multiple caries are also more frequently observed in individuals with advanced periodontitis compared to those without caries. Although dental caries and periodontitis have distinct etiologies, they share common risk factors such as poor oral hygiene, unhealthy dietary habits, and low socioeconomic status, which may contribute to the concurrent development of both conditions [11].

Smoking is strongly associated with the progression of chronic periodontitis, with evidence suggesting that it can increase the severity of the disease by 2 to 7 times compared to non-smokers [12]. According to Siyanandham et al. (2023), severe periodontitis is commonly observed in heavy smokers, with clinical features including deep periodontal pockets, attachment loss, and furcation defects [13]. Several studies have also reported that heavy smoking increases the risk and progression of periodontitis while significantly reducing the success of periodontal therapy [14].

Nicotine, a major component of tobacco, contributes to a decrease in both the quantity and quality of saliva [4]. Saliva plays a protective role against dental caries by clearing food debris and promoting remineralization. However, in smokers, saliva tends to be thicker and more mucous-like compared to the more fluid saliva in non-smokers [15]. Nicotine not only promotes the growth of cariogenic bacteria such as *Streptococcus mutans* and *Lactobacillus*, but also reduces salivary buffering capacity, ultimately creating an acidic environment that predisposes teeth to caries. Furthermore, nicotine enhances the ability of *Streptococcus mutans* to adhere to tooth surfaces, exacerbating the development of caries. A study by Javed et al. (2023) also found that men are more susceptible to dental caries than women, largely due to the higher prevalence of smoking among men [16].

The aim of this case report is to describe the clinical management of generalized chronic periodontitis in a geriatric heavy smoker.

Methods

A 63-year-old male patient presented to Soelastri Dental and Oral Hospital with a chief complaint of tooth mobility, which caused discomfort while eating. According to the patient, the teeth had been mobile for the past three months, and he requested extraction. The patient had no prior history of dental treatment and denied any known allergies or systemic diseases. He reported a smoking habit of eight cigarettes per day.

Vital signs on examination were as follows: blood pressure 155/91 mmHg, pulse rate 77 beats per minute, respiratory rate 18 breaths per minute, and body temperature 36.3°C. The patient was in good general health and cooperative. Extraoral examination revealed no abnormalities. Intraoral examination (Figure 1) showed retained roots in teeth 27, 36, and 46; edentulous areas at teeth 17, 16, 26, 37, and 47; necrosis in teeth 13, 12, 11, and 22; cervical caries on teeth 15, 14, 21, 23, 24, 25, 36, 35, 34, 33, 42, 43, 44, and 45; and Class III gingival recession on teeth 42, 31, and 32. Poor oral hygiene was noted, with an oral hygiene index score of 7 (Figure 1).

Figure 1. Intraoral examination revealed multiple carious lesions on both the maxillary and mandibular teeth, with dark discoloration and gingival recession.

The patient underwent laboratory investigations (Table 1), including routine hematological tests such as leukocyte count, erythrocyte count, hemoglobin (Hb), and hematocrit, using the automated hematology analyzer Mindray BC-3000 Plus. All results were within normal limits. HbA1c testing was performed

using the ion-exchange high performance liquid chromatography (HPLC) method with the Bio-Rad D-10 Hemoglobin Testing System, and the HbA1c result was within the normal range (<5.7%).

Panoramic radiographic examination was performed with the patient in an upright position, chin slightly lowered, and the head adjusted to ensure that the occlusal plane was parallel to the floor [17]. The radiograph revealed severe alveolar bone resorption in the anterior mandibular region (Figure 2). Based on these findings, the patient was diagnosed with generalized chronic periodontitis with heavy smoking as a predisposing factor. The attending dentist decided to extract teeth 42, 31, and 32 under local anesthesia, scheduled over multiple visits.

Pemeriksaan *Examination*	Hasil *Result*	Nilai Rujukan *Reference Value*	Satuan *Unit*	Metode *Method*
HEMATOLOGI				
Hematologi Rutin				
Hemoglobin	14,6	13.2 - 17.3	g/dL	
Lekosit	7.3	3.8 - 10.6	10^3/uL	
Basofil	0	0 - 1	%	
Eosinofil	1	1 - 3	%	
Netrofil Batang	3	2 - 6	%	
Netrofil Segmen	46*	50 - 70	%	
Limfosit	44*	20 - 40	%	
Monosit	6	2 - 8	%	
Trombosit	176	150 - 440	10^3/uL	
KIMIA				
Glukosa Sewaktu	104	Glukosa darah sewaktu (Plasma Vena) Bukan DM : < 100 Belum pasti DM : 100 - 199 DM >= 200 Glukosa darah sewaktu (Darah Kapiler) Bukan DM : < 90 Belum Pasti DM : 90 - 199 DM : >= 200	mg/dL	
HbA1c (NGSP)	5,1	Metode yang digunakan : Boronate Affinity dan telah distandarisasi NGSP Normal : < 5,7 Prediabetes : 5,7 - 6,4 Diabetes : >= 6,5	%	
Kreatinin	0.98	0.67 - 1.17	mg/dL	
Ureum	21.1	16.6 - 48.5	mg/dL	

Table 1. Results of Routine Hematology and HbA1c Examination

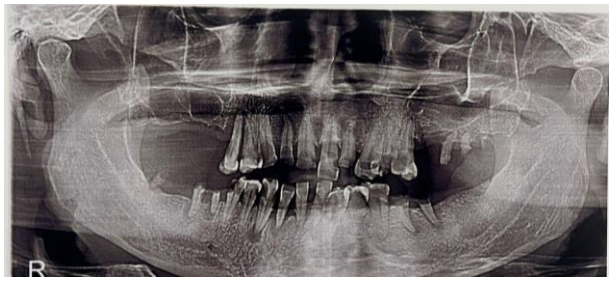

Figure 2. Panoramic Radiographic

The treatment began with communication, information, and education (CIE) to inform the patient about the diagnosis and the proposed treatment plan. The patient consented to the extraction of teeth 42 and 31, as documented through a signed informed consent form. The extraction procedure started with aseptic preparation using 3% povidone iodine applied to the buccal and lingual mucosa with a cotton pellet, followed by local anesthesia using 2% Pehacaine administered via infiltration technique.

The extraction sockets were irrigated using a spooling method with a solution of 0.9% NaCl mixed with 3% povidone iodine in a 1:1 ratio (Rondhianto, Wantiyah, & Widyaputri, 2015), followed by surgical curettage to remove necrotic tissue from the socket. No significant post-extraction bleeding was observed (Figure 3).

In cases of chronic periodontitis, subgingival plaque and calculus harbor pathogenic bacteria that contribute to chronic inflammation. Curettage in this context aims to remove infected granulation tissue and residual plaque and calculus from the pocket walls, thereby reducing local inflammation and preventing further spread of infection to surrounding healthy tissues (Khalifah, 2021).

Spongostan was applied to the socket to promote healing and reduce the risk of post-extraction complications such as dry socket (Vordemvenne et al., 2020). The patient was prescribed medication including amoxicillin 500 mg to be taken three times daily every 8 hours, and diclofenac sodium 50 mg to be taken twice daily every 12 hours.

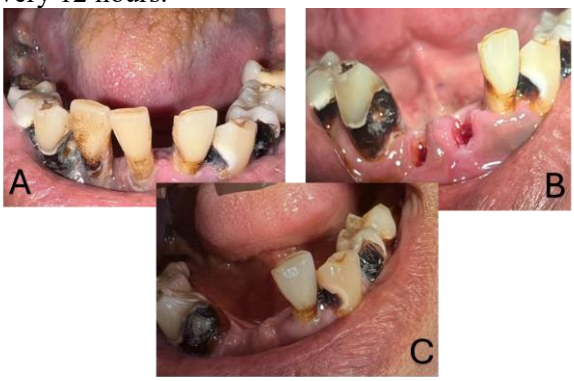

Figure 3. (A) Teeth 42 and 31 prior to extraction, (B) Post-extraction view of teeth 42 and 31, (C) Follow-up condition of the extraction sites of teeth 42 and 31

A follow-up evaluation was conducted one week after the extraction. Subjective assessment revealed no pain, swelling, or discomfort in the extraction area. Intraoral clinical examination showed that the extraction sockets of teeth 42 and 31 had healed well, with no signs of infection or complications such as dry socket. The patient reported satisfaction with the treatment outcome and no longer experienced discomfort from the previously mobile teeth. The patient was also informed about the long-term treatment plan, including the fabrication of a dental prosthesis to restore masticatory function.

Result and Discussion

Tooth extraction in patients with chronic periodontitis is a complex clinical decision influenced by multiple factors. According to Lim et al., in patients diagnosed with generalized periodontitis stage IV, grade C, both clinical and

non-clinical factors play a role in determining whether extraction should be pursued over more conservative treatments such as root canal therapy [18].

Clinical factors include measurable indicators that can be directly evaluated by the dentist. These encompass the severity of periodontal disease, often assessed by periodontal pocket depth, which reflects the extent of inflammation and alveolar bone loss. Tooth mobility is another crucial consideration; severely mobile teeth often indicate advanced destruction of supporting structures and poor prognosis for retention. Furcation involvement is also a significant indicator, as it represents advanced periodontal breakdown that is difficult to manage with non-surgical therapy [19]. Panoramic radiographs showing substantial alveolar bone loss further support the decision for extraction, as the remaining bone may no longer provide adequate support.

Non-clinical factors, on the other hand, relate to aspects not directly tied to the patient's oral health status, such as socioeconomic conditions. Patients with financial constraints may prefer tooth extraction as a more affordable solution compared to long-term periodontal therapy, which often requires repeated visits and substantial costs [20].

Smoking is a major risk factor that exacerbates periodontal tissue destruction. Nicotine, the primary component of tobacco, contributes to periodontal breakdown through various mechanisms. First, nicotine stimulates the production of pro-inflammatory cytokines such as interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), and prostaglandin E2 (PGE2) (Zhou et al., 2020), which amplify the inflammatory response in periodontal tissues, ultimately leading to tissue damage and alveolar bone resorption. Nicotine also enhances osteoclast activity while inhibiting osteoblast function, disrupting bone remodeling and resulting in progressive bone loss [21].

Furthermore, nicotine affects endothelial vasoreactivity. It increases the expression of endothelin-1 (ET-1), which binds to the endothelin type A receptor (ETA) on vascular smooth muscle cells, causing vasoconstriction [22]. This vascular narrowing reduces blood flow to the periodontal tissues [23], thereby limiting oxygen and nutrient supply necessary for tissue maintenance [24]. Hypoxia in periodontal cells impairs cellular energy production and regenerative capacity. Reduced blood flow also weakens immune cell delivery to the infection site, allowing persistent inflammation [25]. Additionally, nicotine-induced vasoconstriction masks clinical signs of inflammation, potentially delaying diagnosis and treatment, which may further worsen periodontal damage [26]

Smoking adversely affects both innate and adaptive immune responses. In smokers, the numbers and functions of Natural Killer (NK) cells and T cells are significantly reduced compared to non-smokers, weakening the body's ability to combat bacterial infections that cause periodontitis [12]. Nicotine also suppresses the osteogenic differentiation of periodontal ligament stem cells (PDLSCs) and reduces prostaglandin E2 (PGE2) secretion, thereby inhibiting periodontal tissue proliferation [27]. Zhang Lu et al. demonstrated that nicotine significantly impairs PDLSC proliferation, negatively impacting their regenerative potential [25].

Aging is another contributing factor in the progression and severity of periodontitis. It compromises immune function, leading to a chronic low-grade inflammatory state known as *inflammaging*. Neutrophils in elderly individuals exhibit reduced migration and phagocytic capabilities, while macrophages produce lower levels of pro-inflammatory cytokines such as TNF-α, IL-1β, IL-6, IL-12, and IFN-γ, resulting in diminished immune responses [28]. Delayed healing and metabolic changes in older adults further increase the risk of periodontal disease.

Multiple caries in elderly individuals are often associated with reduced salivary function. Xerostomia, or dry mouth, is common with aging and impairs the natural cleansing and buffering capacity of saliva, making teeth more vulnerable to acid attacks from cariogenic bacteria [29]. Caries often develop on hard-to-clean surfaces such as the proximal and labial surfaces of mandibular incisors [10]. Additionally, reduced dexterity may hinder proper oral hygiene, further increasing the risk of rampant caries [29].

Untreated periodontal disease leads to progressive destruction of the tooth-supporting structures, including the formation of deep periodontal pockets and eventual alveolar bone resorption. Clinically, this manifests as tooth mobility, a hallmark of advanced chronic periodontitis [30]. In such cases, extraction is indicated when there is severe mobility, deep pockets, significant attachment loss, and poor response to previous periodontal therapy [20].

Management of multiple caries in adults aims to restore function and esthetics while preserving healthy tooth structure. Initial steps include

comprehensive oral examination and severity assessment. Non-restorable teeth should be extracted to prevent further infection, while salvageable teeth can be treated with root canal therapy and fiber post placement. Replacing missing teeth with partial dentures restores mastication. This pragmatic esthetic approach balances esthetic outcomes with structural preservation. Patient education on oral hygiene and dietary habits is essential to prevent future caries [31].

In this case, the patient was diagnosed with generalized chronic periodontitis stage IV, grade C, presenting with discomfort due to tooth mobility. Prior to treatment, comprehensive subjective, objective, and radiographic examinations were performed. Panoramic imaging revealed severe alveolar bone resorption, indicating compromised tooth support and instability. Intraoral findings included multiple carious lesions, heavy plaque and calculus deposits, and nicotine stains—signs of chronic heavy smoking. This risk factor not only worsens periodontal prognosis but also delays healing, ultimately influencing the decision to proceed with tooth extraction.

Conclusion

Smokers are at significantly higher risk for severe periodontal destruction and often require tooth extraction as part of their treatment plan. Clinical and radiographic assessments consistently show that heavy smokers exhibit greater loss of periodontal support, with deeper periodontal pockets and more extensive alveolar bone resorption compared to non-smokers. The decision to extract a tooth is frequently based on the severity of the disease, the response to non-surgical periodontal therapy, and non-clinical factors such as the patient's socioeconomic status. Integrating both clinical and non-clinical considerations ensures that treatment decisions are appropriate and tailored to each patient's specific condition.

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