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Extraction of Mandibular Second Molar In A Former Drug Users, Alcohol Consumers, and Active Smokers

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ABSTRAK

Latar Belakang: Pencabutan gigi molar kedua rahang bawah pada pasien dengan riwayat penyalahgunaan obat, konsumsi alkohol, dan kebiasaan merokok aktif memiliki risiko tinggi terhadap terjadinya komplikasi pascaoperasi. **Laporan Kasus dan Tatalaksana Kasus:** Seorang pria berusia 34 tahun datang dengan keluhan lesi karies luas yang tidak dapat direstorasi pada molar kedua kanan bawah (gigi 47) dan meminta dilakukan pencabutan. Riwayat kesehatan mulut menunjukkan kebersihan mulut yang buruk, kebiasaan merokok aktif, konsumsi alcohol, serta riwayat penyalahgunaan klorpromazin di masa lalu. Prosedur pencabutan dilakukan dengan teknik anestesi blok nervus alveolar inferior menggunakan lidokain. Efek anestesi bertahan selama kurang lebih satu jam, setelah itu pasien kembali merasakan nyeri. Proses penyembuhan pasca pencabutan dipantau selama dua bulan, dimana soket gigi 47 menunjukkan keterlambatan dalam penutupan epitel. Blokade reseptor alfa-adrenergik akibat penggunaan klorpromazin, perubahan aktivitas enzim sitokrom P450 hati akibat konsumsi alkohol, kondisi asidosis sistemik, serta kebiasaan merokok pascaoperasi diketahui dapat memengaruhi efektivitas anestesi lokal dan menghambat proses penyembuhan luka. Vasokonstriksi yang diinduksi oleh nikotin dan kondisi mulut kering (xerostomia) semakin memperburuk regenerasi jaringan. Meskipun tidak ditemukan komplikasi akut, penyembuhan soket tetap tidak sempurna hingga delapan minggu setelah pencabutan. **Kesimpulan:** Kasus ini menegaskan pentingnya evaluasi terhadap faktor risiko sistemik dan perilaku pasien sebelum melakukan pencabutan gigi.
Kata Kunci: Perokok Aktif; Konsumen Alkohol; Penyembuhan Luka Tertunda; Pengguna Obat; Ekstraksi Molar Mandibula

ABSTRACT

***Background**: Extraction of Mandibular Second Molar Extraction in Former Drug Users, Alcohol Consumers, and Active Smoker has a high risk of complications. **Case report and case treatment**: A 34-year-old male presented with a non-restorable carious lesion on the lower right second molar (tooth 47) and requested extraction. The patient reported poor oral hygiene, active smoking, alcohol consumption, and a prior history of chlorpromazine abuse. The extraction was performed under Inferior Alveolar Nerve Block (IANB) using Lidocaine. Anesthesia lasted approximately one hour, after which pain resumed. Post-extraction healing was monitored over two months, during which the socket of tooth 47 showed delayed epithelial closure. **Discussions**: Alpha-adrenergic receptor blockade from chlorpromazine, hepatic cytochrome P450 enzyme alterations due to alcohol, systemic acidosis, and post-operative smoking are known to influence the efficacy of local anesthesia and impair healing. Nicotine-induced vasoconstriction and xerostomia further disrupt tissue regeneration. Despite the absence of acute complications, socket healing remained incomplete at 8 weeks post-extraction. **Conclusion**: This case emphasizes the importance of evaluating systemic and behavioral risk factors before extraction.*
***Keywords**: Active Smoker; Alcohol Consumers; Delayed Wound Healing; Drug Users; Mandibular Molar Extraction*

BACKGROUND

Modern oral surgery is increasingly confronted with complex patient profiles, particularly those with polysubstance use histories. In Indonesia, one such substance commonly misused is trihexyphenidyl—known colloquially as eximer. Chronic abuse of this anticholinergic agent contributes to systemic anticholinergic load, resulting in hyposalivation and deterioration of oral health.[1] Compounding this, the prevalence of tobacco smoking and chronic alcohol use remains high among the adult population. The coexistence of these three factors presents a unique biological environment that

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complicates even routine surgical procedures such as the extraction of the mandibular second molar.

Smoking alone is a well-established risk factor for delayed wound healing and alveolar osteitis. Smokers have been found to have over three times the risk of developing dry socket compared to non-smokers (13.2% vs 3.8%).[2] Nicotine causes vasoconstriction, disrupts platelet aggregation, and alters local cytokine expression, all of which impair blood clot formation and stabilization post-extraction. Moreover, a meta-analysis reported that smokers have a 2.5-fold increased risk of bone non-union, a complication that can be mitigated by cessation of smoking at least four weeks before surgery.[3]

Alcohol adds another layer of physiological compromise. Chronic alcohol intake is associated with thrombocytopathy, immune suppression, and dehydration, which all contribute to increased risk of infection and delayed epithelialization.(4) A recent narrative review on postoperative dental complications found that alcohol misuse extends postoperative pain duration and increases the likelihood of secondary hemorrhage, even in the absence of direct systemic involvement.[4] Clinical recommendations now advocate for complete abstinence from alcohol for at least two weeks following intraoral surgery to reduce systemic inflammation and lower the risk of alveolitis.

Complicating this further is the prolonged misuse of benzodiazepines and anticholinergics like trihexyphenidyl. A large cohort study involving 10,896 general surgery patients found that 21.6% of hospitalized individuals received oral benzodiazepines during their stay, and this subgroup showed significantly higher 30-day readmission rates (OR 1.37) and increased postoperative opioid requirements.[5] While the short-term use of benzodiazepines is considered safe in dental practice as an anxiolytic, there is a stark absence of research addressing the long-term implications of dependence on oral tissue healing and bone regeneration.[6]

From an oral pathophysiology standpoint, the anticholinergic burden caused by trihexyphenidyl linearly reduces unstimulated salivary flow—one study quantifies this as a decrease of 0.058 mL/min for every 1 pmol/mL increase in serum anticholinergic activity.[7] Hyposalivation contributes to higher caries incidence, accelerated periodontitis, and greater colonization by opportunistic pathogens—all of which are independently associated with an increased risk of infection and alveolitis post-extraction. When combined with the hypoxic microenvironment induced by smoking and nutritional compromise due to alcohol, this triad creates a "high-risk biological milieu" that challenges surgical predictability and postoperative recovery.

However, between 2019 and 2025, the literature remains fragmented, with most studies focusing on these risk factors in isolation. A scoping review on the oral health needs of individuals undergoing treatment for substance use disorders noted the lack of integrated clinical guidelines for managing patients with overlapping risks.[8] To date, no documented case report specifically addresses the extraction of a mandibular second molar in a patient with a history of trihexyphenidyl abuse, chronic alcoholism, and active smoking.

This clinical case report seeks to address this critical gap by outlining a comprehensive approach to preoperative risk screening, intraoperative management, and postoperative care tailored for a medically complex patient. We aim to contribute novel clinical insights that may inform future interdisciplinary guidelines for oral surgery in patients with poly-substance use histories. Emphasis is placed on strategies such as anticholinergic burden evaluation, smoking cessation counseling, nutritional rehabilitation, and enhanced socket irrigation protocols to mitigate complications, improve healing outcomes, and enhance quality of life for such high-risk populations.

CASE PRESENTATION

First Visit (January 3th, 2025)

A 34-year-old male patient presented to UMY Dental Hospital (RSGM UMY) with a chief complaint of a discolored, extensively decayed lower right posterior tooth that had been causing discomfort. The patient expressed a desire to have the tooth extracted for subsequent prosthetic rehabilitation. He was unable to recall when the discoloration had begun. The tooth had previously caused pain and gingival swelling,

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although no current pain was reported at the time of the visit.

The patient's oral hygiene history revealed infrequent toothbrushing and a high consumption of sugary snacks. Intake of fruits and vegetables was minimal. The patient is an active smoker (approximately 12 cigarettes per day), a regular alcoholic consumer (once a week, with the last consumption occurring one week prior), and a former user of eximer(trihexyphenidyl), a narcotic substance he had used for approximately five years before discontinuing use one year ago. Daily water intake was reported to be less than one liter. He reported no known allergies to food or medications, was not on any routine medications, and had no signs or history of systemic disease. His last meal was consumed two hours before the visit, and his sleep pattern was adequate (approximately 11:00 p.m. to 9:00 a.m.).

Intraoral examination revealed a large occlusal cavity on tooth 47, extending to the pulpal floor and deemed non-restorable. Pulpal testing including probing, percussion, palpation, and vitality testing yielded negative responses. The patient's blood pressure was 107/77 mmHg, with a pulse rate of 93 beats per minute and respiratory rate of 20 breaths per minute. Based on clinical and subjective findings, a diagnosis of pulpal necrosis on tooth 47 was established.

The treatment plan included comprehensive patient education and communication (KIE) to explain the dental condition, the planned procedure, and potential risks associated with the treatment. Following this, the extraction of tooth 47 was scheduled to be performed under local block anesthesia to ensure adequate pain control. After the procedure, postoperative evaluation and follow-up were arranged to monitor healing progress and manage any possible complications.

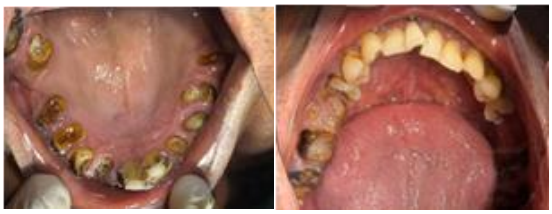

Figure 1. Intraoral condition at initial visit (pre-extraction, Day 0)

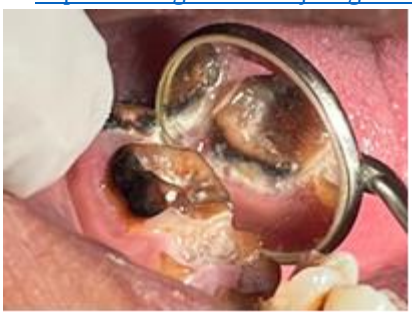

Figure 2. Tooth 47 before extraction (Day 0, preoperative view)

Following the KIE session, the patient was informed of the tooth condition, treatment procedures, and potential complications. After providing clear verbal and written consent, the patient signed the informed consent form for both the extraction and the use of clinical data for case reporting in accordance with institutional regulations.

Extraction Procedure (January 3th, 2025)

The procedure began with the application of topical benzocaine anesthetic using a cotton ball to reduce discomfort during injection. Aseptic preparation was performed on both the injection site and the area surrounding tooth 47. An inferior alveolar nerve block was administered using the indirect Fischer technique with 1 cc of Pehacaine delivered to the inferior alveolar nerve, 0.5 cc to the lingual nerve, and an additional infiltration at the target tooth site.

Anesthesia effectiveness was confirmed subjectively (numbness reported by the patient) and objectively (light tactile stimulation of surrounding soft tissue). Gingival separation was performed using an excavator, followed by luxation using a Bein elevator and a luxator. The tooth was extracted using mandibular molar forceps. After successful extraction, granulation tissue was debrided with a curette, and any sharp bony edges were smoothed with a bone file. The socket was irrigated using saline and povidone-iodine solutions, and a resorbable hemostatic agent (Spongostan) was placed.

The patient was provided with detailed post-extraction instructions to promote optimal healing and prevent complications. These included biting firmly on sterile gauze for 30 minutes to control bleeding, avoiding hot food or beverages for at least one hour, and chewing on

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the left (non-extraction) side to reduce trauma to the surgical site. The patient was also advised to refrain from frequent or forceful rinsing, to avoid touching the extraction area with the tongue or fingers, and to prevent negative intraoral pressure by not sucking on the wound or spitting. Additionally, the patient was instructed to abstain from smoking and alcohol consumption for at least one week, as these behaviors are known to impair wound healing and increase the risk of postoperative complications.

Pharmacological management included the prescription of amoxicillin 500 mg, 15 tablets, to be taken three times daily at eight-hour intervals, as a prophylactic measure against infection. For pain management, the patient was prescribed mefenamic acid 500 mg, 10 tablets, to be taken as needed for discomfort, with a maximum of three doses per day. These measures were aimed at ensuring both antimicrobial coverage and adequate analgesia during the immediate postoperative recovery phase.

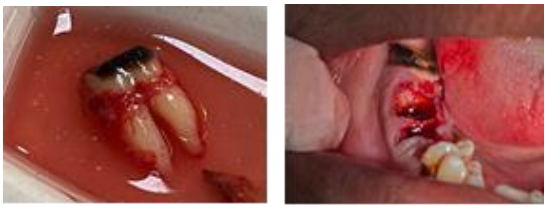

Figure 3. Extraction socket immediately after tooth removal (Day 0, postoperative)

Second Visit (January 15th, 2025)

On 12th Day postoperative, the patient returned for a follow-up evaluation. Subjectively, the patient reported no significant complaints. He stated that mild pain started approximately 30 minutes after the extraction and persisted as a light discomfort when the area was touched, with a pain rating of 2 out of 10. The prescribed antibiotic was taken regularly and completed as instructed. Analgesic medication was taken only twice—once on the night of the extraction and once the following morning. The patient admitted to resuming smoking after the extraction and reported consuming alcohol on day 7 postoperatively, contrary to the postoperative instructions.

Clinical examination revealed mild tenderness upon palpation at the extraction site of tooth 47 (pain score 2/10). The socket was irrigated using povidone iodine and normal saline to promote local debridement and healing.

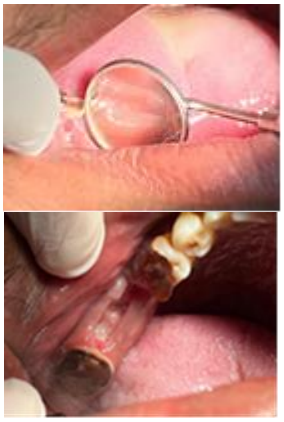

Figure 4. Extraction socket at first follow-up (Day 12 postoperative)

Subsequent Visits (March 4th and March 11th, 2025)

On 47th postoperative, clinical examination of the extraction site of tooth 47 revealed delayed healing, with the socket still partially open and showing incomplete tissue closure. A follow-up evaluation on 54th postoperative, showed similar findings: the socket of tooth 47 remained unhealed, with persistent soft tissue deficiency. This delayed healing was likely influenced by the patient's systemic and local risk factors, including active smoking, prior substance abuse (trihexyphenidyl), and poor nutritional status.

Figure 5. Extraction socket at second follow-up (Day 54 postoperative, delayed healing observed)

DISCUSSION

In this case, the extraction of tooth 47 was performed under an inferior alveolar nerve block (IANB) using one ampule of Pehacaine 1:50,000. The anesthetic effect lasted approximately one hour, after which the patient reported pain, likely due to the expiration of the

anesthetic's effective duration. Typically, Pehacaine has an onset time of 2–3 minutes, with pulpal anesthesia lasting 60–75 minutes and soft tissue anesthesia lasting 3–5 hours.[9]The patient had a history of substance abuse involving eximer (chlorpromazine), an antipsychotic with known alpha-adrenergic receptor blocking effects. In contrast, epinephrine acts on both alpha- and beta-adrenergic receptors; therefore, the interaction between chlorpromazine and epinephrine can potentially induce severe hypotension and tachycardia due to reduced peripheral resistance caused by alpha-receptor blockade.[10]

Interestingly, an experimental study on rats involving intrathecal spinal anesthesia found that chlorpromazine produced a longer duration of nerve blockade than bupivacaine, a long-acting local anesthetic. These findings contradict the assumption that alpha-blockade from chlorpromazine would shorten anesthetic duration. Another study indicated that local anesthetics and phenothiazines (including chlorpromazine) could inhibit glucose uptake in yeast cells, suggesting a potential mechanism of cellular glucose deprivation. Although this may reflect biochemical interactions, its clinical implications for local anesthesia duration in human dental procedures remain unclear.[11] Moreover, patients taking antipsychotic medications, including chlorpromazine, often present with significantly poorer oral health, characterized by higher caries prevalence and more severe periodontal disease. One common side effect of these medications is xerostomia, particularly those with strong anticholinergic activity such as quetiapine, olanzapine, and chlorpromazine. Xerostomia increases the risk of caries and periodontal disease and may impair salivary gland function and oral mucosal integrity, ultimately delaying wound healing.[12]

This patient also had a history of chronic alcohol consumption. Several studies have demonstrated that alcohol-dependent individuals often require higher doses of anesthetic and experience a shorter anesthetic duration. Chronic alcohol intake significantly alters hepatic cytochrome P450 enzyme activity, particularly affecting the metabolism of amide-type local anesthetics such as Lidocaine, Pehacaine®, and bupivacaine®. Alcohol-induced modulation of CYP2E1, CYP1A2, and CYP3A1 enzymes accelerates the metabolic breakdown of anesthetic agents and other drugs.[13] Additionally, prolonged alcohol consumption may lead to systemic acidosis by lowering blood pH, thereby creating an acidic environment that reduces the efficacy of local anesthetics. Local anesthetics must remain in their non-ionized form to penetrate nerve membranes and block sodium channels effectively. This acidic shift could delay the onset time of the anesthetic.[14] Furthermore, long-term alcohol use suppresses immune function, making the extraction socket more susceptible to infection due to impaired immune response.[15]

Wound healing after tooth extraction typically involves three distinct phases. The first is the inflammatory and coagulation phase (0–3 days), during which neutrophils, macrophages, and other immune cells migrate into the socket to clear debris and release cytokines and growth factors—such as PDGF, TGF-β, FGF, and VEGF—to initiate vascularization and granulation. The second phase, occurring between days 3 and 14, is characterized by granulation tissue formation, angiogenesis, and fibroplasia, mediated by factors such as TGF-β1 and FGF-2. Early bone deposition (woven bone) generally begins around day 14. The third phase, known as the remodeling phase, spans from two weeks to several months post-extraction. By weeks 6–8, woven bone replaces the granulation tissue, and bone remodeling continues for 2–6 months.[16,17]

In the present case, the patient resumed smoking two days after the extraction, despite having received postoperative instructions to abstain. Two months post-extraction, clinical evaluation revealed incomplete socket closure, suggesting delayed healing. Smoking significantly impairs the post-extraction healing process. Nicotine induces vasoconstriction in the oral vasculature, which restricts blood flow and oxygen delivery to the surgical site—both essential for effective healing. Blood circulation transports vital nutrients and immune cells necessary for tissue repair.[18] A 2022 study concluded that smokers are three times more likely to develop dry socket compared to non-smokers, with an incidence of 13.2% among smokers versus 3.8% in non-smokers.[19] Current

clinical guidelines recommend that smokers abstain from smoking for at least 48 to 72 hours before and after dental extraction to support optimal healing.[20]

Mandibular molar extraction is a common dental procedure typically performed under local anesthesia and requires effective pain control through appropriate anesthetic techniques, such as the Inferior Alveolar Nerve Block (IANB). In this case, the patient presented with multiple systemic and behavioral factors that potentially influenced both the efficacy of local anesthesia and the post-extraction healing process. A history of substance abuse involving eximer (chlorpromazine), chronic alcohol dependence, and active smoking are known to alter the duration and effectiveness of local anesthetics, as well as compromise wound healing quality. The relatively short duration of anesthesia—approximately one hour—may have been influenced by antagonistic pharmacological interactions between chlorpromazine and epinephrine, hepatic metabolic alterations due to chronic alcohol use (particularly involving cytochrome P450 enzymes), and possible systemic pH changes that impair anesthetic diffusion across nerve membranes.

Additionally, the patient's continued smoking immediately after the extraction procedure likely hindered the healing process. Nicotine-induced vasoconstriction reduces blood flow to the surgical site, thereby limiting the delivery of essential nutrients and immune cells necessary for tissue regeneration. Although no severe postoperative complications were observed during follow-up visits, this case highlights the critical importance of thorough preoperative evaluation of the patient's medical history and lifestyle factors in planning minor oral surgeries such as tooth extractions. Comprehensive patient education and behavior modification—including cessation of smoking and alcohol consumption before and after surgery—are essential to enhance the success and healing outcomes of the extraction procedure.

Furthermore, the chronic anticholinergic burden associated with trihexyphenidyl misuse may have a long-lasting impact on salivary gland physiology, even after cessation. Prolonged receptor desensitization and neuronal adaptation can persist for months, resulting in reduced baseline salivary secretion and altered mucosal defense mechanisms.[21] Saliva plays a vital role in maintaining oral homeostasis, acting as a natural lubricant, buffer, and antimicrobial medium. A deficiency in salivary flow reduces mucosal resilience and increases susceptibility to opportunistic infections, such as *Candida albicans*, which can further delay socket epithelialization and secondary intention healing.[22] Additionally, anticholinergic-induced xerostomia can lead to changes in oral microbiota composition, increasing anaerobic bacterial colonization that contributes to local inflammation and alveolitis.[23]

Recent evidence also suggests that patients with a history of polysubstance use may exhibit subclinical nutritional deficiencies, particularly in vitamins A, C, D, and zinc—all of which are essential for fibroblast activity, collagen synthesis, and immune modulation during wound healing.[24] This metabolic impairment could further explain the slow epithelial regeneration observed in this case. In similar contexts, interdisciplinary approaches involving collaboration between oral surgeons, physicians, and nutritionists are recommended to optimize the perioperative condition of patients with a history of substance use.[25]

From a broader surgical perspective, the integration of risk stratification tools—such as the American Society of Anesthesiologists (ASA) physical status classification and assessment of anticholinergic cognitive burden (ACB) scores—may enhance clinical decision-making for oral surgery candidates with complex medication or substance histories.[26] Tailored preoperative protocols, including salivary substitutes, topical sialogogues, and antioxidant supplementation, can support mucosal recovery and improve overall healing quality.[27]

This case thus reinforces the importance of adopting a holistic and preventive approach when managing patients with overlapping behavioral and pharmacological risk factors. Oral health professionals should prioritize comprehensive counseling, behavioral modification programs, and early postoperative follow-up to identify potential complications at their onset. The findings presented here contribute to the emerging discourse on the interplay between substance use,

pharmacological burden, and oral wound healing. Future research should aim to develop standardized clinical guidelines for dental management of patients with polysubstance use, ensuring safer, evidence-based practice across diverse patient populations.[28]

This case report presents several limitations. As it involved a single patient, the observations cannot be generalized or utilized to infer causal relationships. Certain clinical data were obtained through the patient's self-reported history of smoking, alcohol consumption, and previous drug use, which may have introduced recall bias. Furthermore, objective parameters such as salivary flow rate assessment, laboratory investigations, and radiographic monitoring were not performed. The pharmacological mechanisms proposed remain theoretical, as they were not empirically validated within this case. Additionally, the follow-up period of eight weeks may have been insufficient to capture the full process of bone remodeling. Future research should address these constraints through studies involving larger sample sizes, objective clinical measurements, and standardized outcome evaluations.

CONCLUSION AND SUGGESTION

The extraction of the mandibular second molar in a patient with a history of eximer (chlorpromazine) use, chronic alcohol consumption, and active smoking presents distinct challenges in terms of anesthetic effectiveness and postoperative healing. The combination of chlorpromazine's pharmacological interactions, hepatic metabolic alterations from alcohol use, and biological effects of smoking contributed to the shortened duration of anesthesia and delayed wound healing. Although the extraction procedure was completed without acute complications, the prolonged socket healing highlights the significant impact of systemic and behavioral factors on post-extraction tissue regeneration.

This case underscores the importance of a holistic approach in the planning and execution of minor oral surgeries in dentistry, with careful consideration of the patient's medical history and lifestyle habits. Comprehensive patient education, screening for systemic conditions, and strong recommendations for behavioral modifications—such as cessation of smoking and alcohol consumption before and after surgery—should be integrated into the standard care protocol. Such measures are essential to optimize clinical outcomes and promote more effective healing, even in high-risk patients**.**

CONFLICT OF INTEREST

The author declares that there is no conflict of interest regarding this article.

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