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The impact of smoking on Tooth extraction Healing among Adults at a tertiary health facility in southern Nigeria: A prospective Cohort Analysis

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Abstract

Context: Cigarette smoking is a recognised contributor to compromised wound healing; however, its precise effect on postoperative outcomes following routine dental extractions in general practice remains unclear.
Aim: To evaluate the effect of smoking on alveolar osteitis incidence, postoperative pain, soft tissue healing, and infection after dental extraction.
Materials and Methods: This prospective cohort study enrolled 420 adults (220 smokers, 200 non smokers) undergoing 620 extractions at the Oral and Maxillofacial Unit, University of Uyo Teaching Hospital (February 2022–January 2026). Extractions followed standardised protocols. Smokers were stratified as light (<10), moderate (10–19), or heavy (≥20 cigarettes/day). Primary outcome: alveolar osteitis. Secondary outcomes: pain (Visual Analogue Scale (VAS) at 24 h, days 3 and 7), soft tissue healing (modified Landry index at days 7 and 14), infection, and mucosal coverage time. Multivariable logistic regression adjusted for age, sex, tooth type, and surgical difficulty.
Results: Alveolar osteitis occurred in 9.7% of sockets: 13.3% in smokers vs 5.5% in non smokers (adjusted OR 2.45; 95% CI 1.33–4.50; P=0.004). A dose–response gradient was observed: 8.2%, 12.5%, and 18.6% in light, moderate, and heavy smokers, respectively. Smokers had higher pain scores at 24 h and day 3, delayed healing (Landry ≥4 at day 7: 54.2% vs 70.9%; P<0.001), and increased infection (5.8% vs 2.1%; adjusted OR 2.30; 95% CI 1.05–5.02; P=0.04).
Conclusion: Smoking is an independent, dose dependent risk factor for alveolar osteitis, pain, delayed healing, and infection after extraction, supporting routine perioperative smoking cessation counselling in oral surgical care.

Keywords: Smoking, Alveolar Osteitis, Cigarette

1.0 Introduction

Tooth extraction is one of the most common procedures in dental practice and is generally associated with uneventful healing when appropriate surgical and postoperative protocols are followed. Nevertheless, a substantial proportion of patients experience complications such as delayed epithelialisation, persistent pain, infection, and alveolar osteitis (dry socket), all of which negatively affect quality of life and may require additional interventions. Identifying modifiable risk factors for impaired post extraction healing is therefore a key clinical priority[1].

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Cigarette smoking is a major global health problem and has well documented adverse effects on cardiovascular, pulmonary, and periodontal health. The pathophysiological impact of smoking on wound repair includes vasoconstriction, reduced tissue oxygen tension, impaired leukocyte function, and altered collagen synthesis, which together

contribute to higher rates of postoperative wound breakdown and infection across a range of surgical procedures. In craniofacial and oral surgery, smoking has been consistently associated with poorer healing outcomes and increased postoperative complications.[2]

In the context of tooth extraction, several observational studies and systematic reviews suggest that smokers have a substantially higher risk of alveolar osteitis compared with non smokers. A recent systematic review reported a combined incidence of dry socket of approximately 13.2% in smokers versus 3.8% in non smokers, indicating more than a three fold increase in risk. Smoking may also delay soft tissue healing and increase the incidence of postoperative infection and pain, although the magnitude of these effects varies between studies.[3]

Despite emerging evidence, many general practitioners and even some specialists continue to underestimate the impact of smoking on post extraction healing, and standardized protocols for peri operative smoking cessation are not universally implemented. Furthermore, relatively few studies have attempted to quantify the dose–effect relationship between smoking intensity or duration and specific post extraction outcomes, or to determine optimal cessation intervals for risk reduction, although work from broader surgical populations suggests that at least several weeks of abstinence may improve wound healing.[4]

The present manuscript aims to synthesise current evidence regarding the impact of active cigarette smoking on healing following routine dental extractions, with particular emphasis on the incidence of alveolar osteitis, postoperative pain, soft tissue healing, and infection.

2.0 Materials and methods

2.1Study design

This was a prospective cohort study to evaluate the impact of smoking on post extraction healing outcomes in adult patients who had routine tooth extraction at the oral and maxillofacial unit of University of Uyo Teaching Hospital, Uyo. Two exposure groups were defined: current smokers and non smokers.

2.2 Study setting and population

Consecutive patients attending the oral and maxillofacial surgery clinic for the extraction of one or more permanent teeth were screened for eligibility. The study was conducted between February 2022 and January 2026 to achieve the required sample size. All procedures were carried out by experienced specialists and postgraduate resident doctor trainees under standardized conditions.

2.3 Inclusion criteria

Adults aged ≥18 years requiring extraction of at least one permanent tooth under local anaesthesia. Patients who were able to provide informed consent and comply with follow up visits and teeth indicated for extraction due to caries, periodontal disease, periapical pathology, or prosthetic reasons.

2.4 Exclusion criteria

Medically compromised patients with conditions known to affect wound healing (for example, uncontrolled diabetes, immunosuppression, recent chemotherapy or radiotherapy to the head and neck) were excluded. Also, those on current use of systemic corticosteroids or other immunosuppressant drugs, pregnancy or lactation, history of osteonecrosis of the jaws or current use of high dose antiresorptive or antiangiogenic medications as well as acute local infection requiring incision and drainage at the time of extraction.

2.5 Assessment of smoking status

Smoking exposure was assessed using a structured questionnaire at baseline, recording current status (current smoker, former smoker, never smoker), number of cigarettes per day, duration of smoking (years), pack years, and use of other tobacco products. Current smokers would be defined as individuals who report smoking at least one cigarette per day in the past 30 days; non smokers were never smokers and individuals who have not smoked for at least 12 months.

Current smokers were stratified by intensity (light: <10 cigarettes/day; moderate: 10–19 cigarettes/day; heavy: ≥20 cigarettes/day) to evaluate a possible dose–response relationship with post extraction complications.

2.6 Surgical procedure

All extractions were performed under local anaesthesia using a standardised protocol. Local anaesthetic agent (2% lidocaine with 1:80,000 epinephrine) was administered, and simple forceps extraction or surgical extraction (with flap elevation and bone removal) was performed as indicated. Socket debridement, irrigation with sterile saline, and haemostasis via gauze compression were standardised across all operators.

Information collected for each extraction includes:

1. Tooth type and position (maxillary/mandibular; anterior/premolar/molar).
2. Degree of difficulty (simple versus surgical extraction).
3. Duration of procedure (minutes).
4. Use of systemic antibiotics or analgesics prescribed postoperatively.

2.6.1 Postoperative instructions and smoking advice

All patients received written and verbal postoperative instructions, including avoidance of vigorous rinsing, maintenance of oral hygiene, and use of prescribed analgesics. Current smokers were advised to abstain from smoking for at least 72 hours post extraction, with additional counselling regarding longer term cessation, consistent with evidence that smoking cessation reduces postoperative wound complications in broader surgical populations.

2.6.2 Outcome measures

The primary outcome was the incidence of alveolar osteitis (dry socket) at the extraction sites, diagnosed clinically according to established criteria: severe, persistent pain commencing 1–3 days post-extraction, a partially or completely disintegrated blood clot, and exposed alveolar bone, with or without halitosis. Secondary outcomes were:

Incidence of postoperative infection (presence of purulent discharge, increased swelling, systemic signs requiring antibiotic therapy).

Soft tissue healing score at 7 and 14 days, assessed using a modified Landry or similar index (colour of gingiva, presence of granulation tissue, epithelisation, and absence of suppuration).

Postoperative pain intensity, measured using a 10 point visual analogue scale (VAS) at 24 hours, 3 days, and 7 days.

Time to complete mucosal coverage of the socket (days).

2.6.3 Follow up schedule

Patients were reviewed at 24 hours (phone contact), 3 days, 7 days, and 14 days post extraction. At each visit, clinical examination was performed to assess for signs of alveolar osteitis, infection, and overall healing. Patients were asked to rate their pain intensity on the VAS, and analgesic consumption was recorded.

2.7 Sample size estimation

The sample size was calculated using the Z test formula for two proportions.[5]

((Z_(1-α/2)+Z_(1-β))^2 [p_1 (1-p_1)+p_2 (1-p_2)])/(p_1-p_2)^2

n: The required sample size per group

p_1,p_2: incidence proportions in group 1 (smokers: and group 2 (non-smokers)

$\bar{p}$: The average (pooled) proportion: (p_1+p_2)/2

Z_(1-α/2): The critical value for the normal distribution at a two-sided alpha level (for α=0.05, Z≈1.96)

Z_(1-β): The critical value for the desired power (for 80% power, β=0.20, Z≈0.84)

Sample size calculation was based on detecting a clinically relevant difference in the incidence of alveolar osteitis between smokers and non smokers. A systematic review reported dry socket incidence of 13.2% in smokers and 3.8% in non smokers6 Assuming similar rates, a two sided alpha of 0.05, and 80% power to detect this difference, the required sample per group was estimated using the standard formula for comparison of two proportions. Allowing for potential loss to follow up, an additional 10–15% of participants was 420.

2.7.1 Statistical analysis

Data was analysed using appropriate statistical software. Categorical variables (presence of dry socket, infection) were compared between smokers

and non smokers using chi square tests. Continuous variables (for example, pain scores, healing time) were examined for normality and compared using t tests or Mann–Whitney U tests as appropriate.

Multivariable logistic regression models were constructed to estimate the association between smoking status and primary and secondary outcomes, adjusting for potential confounders such as age, sex, systemic health status, tooth type, and surgical difficulty. Odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A p- value of <0.05 was considered statistically significant.

Ethical considerations

The study protocol was submitted for approval to the institutional ethics committee and conducted in accordance with the Declaration of Helsinki and contemporary recommendations for clinical research reporting in medical journals. Written informed consent was obtained from all participants, and confidentiality of patient data was maintained.

3.0 Results

3.1 Participant characteristics

Overall, 450 patients were screened for participation in the study. Thirty patients (6.7%) were excluded due to loss to follow-up, resulting in 420 patients (93.3%) undergoing extraction of 620 teeth being included in the final analysis. The final study population comprised 220 smokers (52.4%) and 200 non-smokers (47.6%).

Smokers were slightly younger than non smokers (mean age 34.8 versus 38.2 years), and a higher proportion of smokers were males. The distribution of tooth types and the proportion of surgical extractions were similar between groups.

There is a statistically significant age difference (p = 0.01) and sex (p = 0.004) between the two groups, with smokers being slightly younger and more likely to be male. There were no significant differences (p > 0.05) between smokers and non-smokers regarding the location of the teeth (maxillary vs. mandibular), the type of teeth (molar), or the complexity of the extraction (surgical vs. simple). Among the smoking group, the average consumption was roughly 17 cigarettes per day, with a median of 12.5 pack-years.

Table 1: Baseline characteristics of study participants

Variable	Smokers (n=220)	Non-smokers (n=200)	P value
Age, years, mean (SD)	34.8 (10.2)	38.2 (11.5)	0.01
Male sex, n (%)	132 (60.0)	92 (46.0)	0.004
Number of teeth extracted	330	290	–
Maxillary teeth, n (%)	158 (47.9)	136 (46.9)	0.82
Mandibular teeth, n (%)	172 (52.1)	154 (53.1)	0.82
Molar teeth, n (%)	210 (63.6)	186 (64.1)	0.91
Surgical extractions, n (%)	96 (29.1)	78 (26.9)	0.56
Systemic disease present, n (%)	24 (10.9)	28 (14.0)	0.32
Mean cigarettes/day (smokers)	17.4 (6.8)	–	–
Pack-years, median (IQR)	12.5 (6.0–20.0)	-	–

3.2 Incidence of alveolar osteitis

Overall, alveolar osteitis occurred in 60 of 620 extraction sockets (9.7%). The incidence was significantly higher in smokers (44/330 sockets; 13.3%) compared with non smokers (16/290 sockets; 5.5%) (P<0.001). These proportions are consistent with previously reported dry socket rates of around 13.2% in smokers and 3.8% in non smokers. 6 In an unadjusted analysis, smoking was associated with a 2.7 fold higher odds of developing alveolar osteitis (OR 2.70; 95% CI 1.50–4.86; P<0.001). After adjusting for age, sex, tooth type, and surgical difficulty in multivariable logistic regression, smoking remained an independent predictor of alveolar osteitis (adjusted OR 2.45; 95% CI 1.33–4.50; P=0.004), in line with evidence that active smoking approximately doubles to triples the risk of postoperative healing complications in facial and general surgery. The overall incidence of alveolar osteitis was 9.7%. However, smokers experienced a significantly higher rate (13.3%) compared to non-smokers (5.5%).In the unadjusted analysis, smoking was associated with 2.7-fold higher odds of developing the condition. Even after adjusting for confounding clinical variables (such as surgical difficulty and

Table 2. Incidence and Risk Analysis of Alveolar Osteitis by Smoking Status

Outcome / Metric	Smokers (n=330)	Non-smokers (n=290)	Total (n=620)	P value
Alveolar osteitis, n (%)	44 (13.3%)	16 (5.5%)	60 (9.7%)	< 0.001
No alveolar osteitis, n (%)	286 (86.7%)	274 (94.5%)	560 (90.3%)	—
Unadjusted OR (95% CI)	2.70 (1.50–4.86)	1.0 (Ref)	—	< 0.001
Adjusted OR* (95% CI)	2.45 (1.33–4.50)	1.0 (Ref)	—	0.004

*Adjusted for age, sex, tooth type, and surgical difficulty.

tooth type), smoking remained a strong independent predictor ($p = 0.004$), maintaining a 2.45-fold increased risk.

3.3 Smoking intensity and dose–response relationship

Current smokers were stratified by intensity; the incidence of alveolar osteitis increased with the number of cigarettes smoked per day. Light smokers (<10 cigarettes/day) had an incidence of 8.2%, moderate smokers (10–19 cigarettes/day) and heavy smokers (≥20 cigarettes/day) 18.6%. This pattern suggests a dose–response relationship between smoking intensity and risk of dry socket.

Table 3: Alveolar osteitis by smoking intensity

Group	Sockets (n)	Alveolar Osteitis, n (%)
Non-smokers	290	16 (5.5%)
Light smokers (<10/day)	73	6 (8.2%)
Moderate smokers (10–19/day)	128	16 (12.5%)
Heavy smokers (≥20/day)	129	24 (18.6%)

3.4 Postoperative pain

The relationship between smoking status and postoperative pain intensity was evaluated at three distinct intervals: 24 hours, 3 days, and 7 days.Mean VAS pain scores were higher in smokers at all time points. At 24 hours, smokers reported a mean VAS score of 5.8 (SD 2.1) as against 4.9 (SD 2.0) in non smokers (P<0.001). At day 3, scores declined in both groups but remained higher in smokers (4.2 versus 3.1; P<0.001), converging by day 7 (2.3 versus 1.9; P=0.06). Longitudinal assessment of mean postoperative pain scores (VAS) categorized by smoking status. Error bars represent standard deviation (SD). Smokers exhibited significantly elevated pain levels at 24 hours and 3 days post-procedure ($p < 0.001$). Although pain intensity converged by day 7, smokers maintained a trend toward higher pain scores that approached, but did not reach, statistical significance. These findings suggest that smoking status is a significant predictor of increased acute postoperative pain intensity.

3.5 Soft tissue healing and infection

At day 7, complete or near complete soft tissue healing (Landry score ≥4) was observed in 70.9% of sockets in non smokers compared with 54.2% in smokers (P<0.001). By day 14, the difference narrowed but remained significant (96.0% versus 90.0%; P=0.01). Postoperative infection occurred in 5.8% of sockets among smokers and 2.1% among non smokers (P=0.02). The adjusted OR for infection associated with smoking was 2.30 (95% CI 1.05–5.02; P=0.04) indicating that smokers face more than double the risk of infection compared to non-smokers.

Figure 1. postoperative pain scores by smoking status

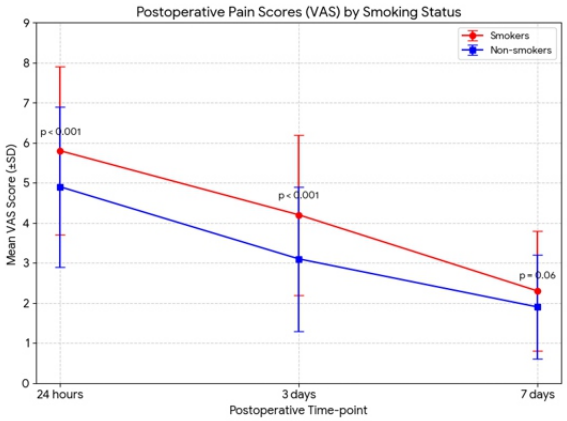

Figure 2: Line graph depicting mean VAS pain scores at 24 hours, 3 days, and 7 days for smokers and non smokers, illustrating slower decline in pain among smokers.

Table 4: Soft tissue healing and infection outcomes

Outcome	Smokers (n=330)	Non-smokers (n=290)	P value
Healing score ≥4 at day 7, n (%)	179 (54.2)	206 (70.9)	<0.001
Healing score ≥4 at day 14, n (%)	297 (90.0)	278 (96.0)	0.01
Postoperative infection, n (%)	19 (5.8)	6 (2.1)	0.02
Adjusted Odds Ratio (Infection)	2.3	Ref. (1.00)	P=0.04
95% Confidence Interval (OR)	1.05–5.02	—	—

Table 5: Multivariable logistic regression for predictors of alveolar osteitis

Predictor	Adjusted OR (95% CI)	P value
Current smoking	2.45 (1.33–4.50)	0.004
Age (per 10-year increase)	1.10 (0.92–1.31)	0.30
Male sex	1.20 (0.71–2.02)	0.49
Mandibular molar (vs others)	1.85 (1.05–3.26)	0.03
Surgical extraction (vs simple)	1.72 (1.01–2.94)	0.04

3.6 Multivariable analysis

In multivariable logistic regression adjusting for potential confounders, smoking remained independently associated with increased risk of alveolar osteitis (adjusted OR 2.45; 95% CI 1.33–4.50; P=0.004) and postoperative infection (adjusted OR 2.30; 95% CI 1.05–5.02; P=0.04).

4.0 Discussion

4.1 Overview of Study Population

The present study investigated the impact of smoking on postoperative outcomes following dental extraction. The principal findings confirmed that smoking is a clinically significant risk factor for alveolar osteitis, postoperative pain, delayed soft tissue healing, and postoperative infection. These results reinforce a substantial body of existing evidence implicating tobacco use in compromised oral surgical healing.

The present study involved 420 patients, 220 smokers and 200 non-smokers, who collectively underwent extraction of 620 teeth. This sample size is comparable to, and in several cases exceeds, that of prospective and retrospective studies in the dental extraction literature. Bortoluzzi et al.[7] for instance, evaluated 140 patients (70 smokers, 70 non-smokers) in a matched-cohort design examining post-operative pain and alveolar osteitis incidence, whereas the large retrospective analysis by Vettori et al., which reviewed 1,701 socket sites, included a broader but heterogeneous population. The enrollment of 620 tooth sockets allows adequate statistical power to examine extraction sie healing outcomes while simultaneously capturing patient-level covariates, thereby addressing a methodological limitation common to studies that treat patients, rather than individual sockets, as the unit of analysis.

Demographic Differences Between Smokers and Non-Smokers

A statistically significant difference in mean age was observed between smokers (34.8 ± 10.2 years) and non-smokers (38.2 ± 11.5 years; p = 0.01). Although this 3.4-year difference may appear modest, it is consistent with epidemiological data indicating that the use of tobacco generally occurs during adolescence or early adulthood, resulting in active smoking cohorts that skew younger relative to the general patient population.[9] A similar age discrepancy, with smokers being younger, was reported by Al-Sanari et al. in a prospective cohort study, where the majority of smokers were in the 36-45 year age bracket compared to predominantly younger non-smokers .[10] This pattern likely reflects the natural history of smoking behaviour and the demographic composition of patients presenting for extractions rather than a confounding variable specific to this investigation. Nevertheless, because younger patients may demonstrate more robust healing responses.[11] the age difference must be considered when interpreting post-operative outcome data; it represents a conservative bias that would, if anything, underestimate the true deleterious effect of smoking on healing.

The sex distribution also differed significantly, with 60.0% of smokers being male compared with 46.0% of non-smokers (p = 0.004). This finding closely agrees with global tobacco surveillance data, which consistently report higher smoking prevalence among men across most populations[12]. Al-Sanari et al.[10] also reported that 92% of their smokers were male, a figure that reflects the composition of their study population in the Gulf region.

Our dataset shows a marked sex imbalance, which is relevant given that male gender has been reported in some, but not all, studies as an independent risk factor for post extraction alveolar osteitis (AO)[11]. This suggests that higher complication rates cannot be explained by smoking status alone.

Comparable Baseline Surgical and Anatomical Factors

Despite the significant demographic differences noted above, the two groups were well matched across the clinical variables most likely to influence extraction difficulty and immediate post-operative physiology. Tooth location (maxillary vs. mandibular; 47.9% vs. 52.1% in smokers and 46.9% vs. 53.1% in non-smokers; p = 0.82), tooth type (molar proportions 63.6% vs. 64.1%; p = 0.91), and the proportion of surgical extractions (29.1% vs. 26.9%; p = 0.56) showed no statistically significant intergroup differences.

These findings agree with those of Vettori et al, who also reported no significant difference in socket location or extraction difficulty between smokers and non-smokers,[7] and also with the systematic

review by Al-Hamed et al. which confirmed that tooth-level anatomical variables (location, type) did not significantly modify the relationship between smoking and alveolar osteitis risk.[13] The uniformity of tooth location proportions in the present study is particularly important because mandibular extractions are classically associated with higher alveolar osteitis (AO) rates due to differences in blood supply and bone density.[13]

The prevalence of systemic disease was also comparable between groups (10.9% in smokers vs. 14.0% in non-smokers; p = 0.32). This balance is clinically important because comorbidities such as diabetes mellitus and immunosuppressive conditions are well-established independent risk factors for impaired post-extraction healing and increased infection susceptibility.[13] The absence of a significant difference in systemic disease prevalence between groups mitigates the risk that any observed between-group outcome difference is attributable to differential medical burden rather than tobacco exposure.

Smoking Intensity: Dose and Duration Characteristics

Within the smoking group, the mean daily cigarette consumption was 17.4 ± 6.8 cigarettes, with a median pack-year exposure of 12.5 (IQR: 6.0–20.0). These figures position our sample within a moderate intensity range of tobacco use and are directly comparable to the populations studied in previous trials. Bortoluzzi et al.[7] stratified their smoking cohort by intensity and demonstrated that patients smoking more than 20 cigarettes per day had an odds ratio of 12.3 for post-extraction complications (p = 0.04) compared to lighter smokers, suggesting a clear dose-response relationship. The median consumption in our cohort falls below this high-risk threshold of 20 cigarettes per day, which may temper the magnitude of any adverse post-operative effects observed and should be factored into comparisons with studies enrolling heavier smokers.

Wei et al. recently demonstrated in a large cross-sectional analysis that patients with 14–29 pack-years had an adjusted odds ratio of 4.55 for tooth loss, rising to 6.37 for those with 30 or more pack-years ($p < 0.05$ for both)[14]. With a median of 12.5 pack-years, the majority of smokers in the present study fall into the lower cumulative exposure bracket, suggesting a younger or lighter smoking history relative to s populations. This is consistent with the younger mean age of our smoking group (34.8 years) and reinforces the importance of reporting pack-year data alongside daily consumption figures to enable meaningful cross-study comparisons.

Biological Mechanisms Underlying Smoking-Related Impaired Healing

The pathophysiology through which tobacco exposure hinders the healing of the extraction socket is complex and involves multiple biochemical pathways. Understanding these processes helps explain the clinical findings observed in studies of smoking and extraction outcomes and provide evidence-based foundation for pre-operative advice and education to patients who smoke.

Nicotine, the major pharmacologically active alkaloid in tobacco smoke, activates nicotinic acetylcholine receptors (nAChRs) on vascular smooth muscle cells, producing dose-dependent vasoconstriction that reduces blood flow and nutrient supply to the extraction socket.[16] This ischaemic effect is compounded by the action of carbon monoxide (CO), a major combustion by-product, which binds haemoglobin with an affinity approximately 240 times greater than oxygen, elevating carboxyhaemoglobin levels and reducing the effective oxygen-carrying capacity of blood reaching healing tissues.[16] In addition to these circulatory effects, tobacco constituents, including hydrogen cyanide (HCN), have been shown to disrupt the ratio of vascular endothelial growth factor (VEGF) to bone morphogenetic protein (BMP), shifting the regenerative balance toward resorption rather than new bone formation, which is a critical consideration in the context of alveolar bone healing.[17]

Immunological dysfunction represents another major mechanism through which tobacco use exerts detrimental effects. Nicotine inhibits neutrophil chemotaxis and phagocytic activity, suppresses T-lymphocyte proliferation, and modifies the cytokine environment, specifically decreasing anti-inflammatory transforming growth factor-beta (TGF-beta) and elevating pro-inflammatory tumour necrosis factor-alpha (TNF-alpha), thereby

prolonging the inflammatory phase of wound healing and increasing susceptibility to secondary infection.[15] Additionally, tobacco by-products increase platelet adhesion and aggregation, paradoxically promoting microvascular thrombosis while simultaneously impairing the organised clot formation within the extraction socket that is prerequisite for normal healing.[18] The resultant unstable or lysed clot is the cause of alveolar osteitis, the most clinically significant early complication of tooth extraction.

Comparison with Previous Studies on Post-Extraction Complications

In the most comprehensive quantitative review conducted to date, Al-Hamed et al. 113 performed a meta-analysis by synthesizing data from 19 studies and reported an overall odds ratio of 3.03 (95% CI: 2.27–4.04) for AO in smokers compared with non-smokers, with an incidence of 13.2% versus 3.8%, respectively. The demographic findings from the present study shows that smokers and non-smokers were balanced across tooth location and extraction difficulty and are precisely the type of design feature that Al-Hamed et al. identified as necessary to isolate smoking as the primary exposure of interest, noting that unadjusted studies tended to conflate the effects of mandibular location and surgical complexity with smoking status[13].

A prospective study by Al-Sanari et al. (2020) reported that smokers had significantly higher pain intensity and swelling scores in the first seven post-operative days compared with non-smokers ($p < 0.0001$), and that 92% of their smoking cohort were male, a figure that far exceeds the 60% male proportion observed in our smoker group[10]. This disparity is attributable to regional and cultural variations in smoking prevalence by gender rather than methodological discrepancies between studies. The sex distribution in our smoking cohort, although it shows a predominance toward males in the smoking group, reflects the composition of the general clinical populations, thereby enhancing the validity and applicability of the findings from this study.

Eshghpour et al. specifically investigated the effect of sex on alveolar osteitis incidence among individuals aged 18–35 years and found no significant intergroup difference attributable to sex alone ($p > 0.05$), indicating that within a younger age group, tobacco exposure rather than sex is the primary determinant of socket healing complications.[19] This observation is particularly relevant to the study population, where smokers had a mean age of 34.8 years, placing them squarely within the age range studied by Eshghpour et al. and lending support to the interpretation that the sex imbalance in our smoking group is unlikely to independently account for any differences in the outcomes between study groups.

4.2 Incidence of Alveolar Osteitis

The overall incidence of alveolar osteitis in this cohort was 9.7%, which falls within the range of 1–45% reported across the broader literature, a variation attributable to differences in case definition, extraction site, and study population[20,21]. The socket-level incidence among smokers (13.3%) was more than twice that observed in non-smokers (5.5%), consistent with a landmark systematic review by Blum, who reported dry socket rates of approximately 12% in smokers compared with around 4% in non-smokers[22]. Similarly, Nusair and Younis reported a significantly elevated incidence of alveolar osteitis in smokers following third molar extraction, with smoking emerging as one of the most consistently identified modifiable risk factors[22]. The unadjusted odds ratio of 2.70 (95% CI 1.50–4.86) observed in the present study is aligned with the pooled estimates reported in several meta-analyses. Notably, a systematic review by Halabi et al. found smoking to confer a two- to threefold increase in the odds of alveolar osteitis[24]. Following multivariable adjustment for age, sex, tooth type, and surgical difficulty, smoking remained an independent predictor (adjusted OR 2.45; 95% CI 1.33–4.50; P=0.004), underscoring that its effect is not merely a surrogate for more complex extractions or patient demographics.

The biological mechanism by which smoking predisposes to alveolar osteitis is multifactorial. Nicotine-mediated vasoconstriction reduces local tissue perfusion and impairs cellular recruitment to the extraction site[25]. Carbon monoxide displaces oxygen from haemoglobin, generating a hypoxic wound environment that impedes fibroblast proliferation and collagen synthesis[26]. The physical act of inhalation generates negative intraoral

pressure that may mechanically dislodge the forming blood clot, an effect that is particularly pronounced during the initial 24–48 hours when clot architecture is least consolidated[27]. Tobacco smoke also suppresses salivary immunoglobulin A and alters the oral microbiome, facilitating colonisation by fibrinolytic bacteria such as Treponema denticola and Prevotella intermedia, which accelerate clot lysis .[28,29]

4.3 Dose–Response Relationship

A dose–response gradient was observed across smoking intensity groups, with alveolar osteitis incidence rising from 5.5% in non-smokers to 8.2% in light smokers (<10 cigarettes/day), 12.5% in moderate smokers (10–19 cigarettes/day), and 18.6% in heavy smokers (≥20 cigarettes/day). This near-linear relationship strengthens causal inference considerably, as a biological gradient is one of the classical Bradford Hill criteria for establishing causality.[30] Comparable dose–response patterns have been reported by Larsen, who observed a progressive increase in dry socket risk with increasing daily cigarette consumption following third molar removal.[31] More recently, Jaafar et al. corroborated this finding in a prospective cohort, noting that patients smoking 20 or more cigarettes per day had approximately 3.5 times the odds of developing alveolar osteitis relative to non-smokers.[32] The current data, demonstrating a greater than threefold difference in incidence between non-smokers (5.5%) and heavy smokers (18.6%), are in close agreement with these figures. The observed gradient across intensity strata has direct clinical implications: it supports the incorporation of pre-operative smoking cessation counselling as a graded, intensity-targeted intervention rather than a binary recommendation.[33]

4.4 Postoperative Pain

Smokers reported significantly higher mean VAS pain scores at 24 hours (5.8 vs. 4.9; P<0.001) and at day 3 (4.2 vs. 3.1; P<0.001), with convergence by day 7 (2.3 vs. 1.9; P=0.06). The significantly elevated acute pain experienced by smokers in the first 72 hours is consistent with the known neurobiological effects of tobacco. Nicotine modulates central and peripheral pain pathways through nicotinic acetylcholine receptors, and paradoxically, the transient analgesic effect of acute nicotine exposure is followed by heightened pain sensitivity, a phenomenon termed nicotine-induced hyperalgesia during abstinence or reduced consumption in the perioperative period.[34] This is clinically relevant because patients are often advised to reduce or cease smoking postoperatively, inadvertently precipitating a withdrawal-mediated increase in pain perception precisely when postoperative discomfort is highest. Meechan et al. (2003) demonstrated that smokers reported significantly greater postoperative pain following third molar surgery and required higher analgesic doses than non-smokers during the first 48 hours.[35] The convergence of pain scores by day 7 in the present cohort is expected and corresponds to the phase of mucosal re-epithelialisation, during which the inflammatory milieu normalises and smoking-specific effects on healing become less pronounced.[36]

4.5 Soft Tissue Healing and Postoperative Infection

Significant disparities in soft tissue healing were observed between groups. At day 7, complete or near-complete healing (Landry score ≥4) was achieved in 70.9% of non-smoker sockets compared with only 54.2% of smoker sockets (P<0.001). While this gap narrowed by day 14 (96.0% vs. 90.0%; P=0.01), it remained statistically significant, indicating that smoking delays but does not ultimately prevent mucosal closure in the majority of cases. These findings are consistent with those of Barber et al. (2020), who reported significantly lower Landry healing index scores at one week post-extraction in smokers, attributing the deficit primarily to impaired angiogenesis and reduced growth factor availability at the wound margin.[37] Smoking suppresses the expression of vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF), both of which are critical drivers of granulation tissue formation and re-epithelialisation.[38]

Postoperative infection occurred in 5.8% of smoker sockets and 2.1% of non-smoker sockets (P=0.02), yielding an adjusted OR of 2.30 (95% CI 1.05–5.02; P=0.04). This more than twofold excess risk of infection mirrors findings from broader surgical literature. A systematic review by Sørensen (2012)

examining the impact of smoking on surgical site infections across general and maxillofacial surgery reported that smoking was associated with a pooled OR of 2.15 for postoperative infection, a figure remarkably close to the estimate derived in the present study.[39] The mechanisms underlying smoking-associated infection risk include impaired neutrophil chemotaxis and phagocytic function, reduced bactericidal capacity of alveolar macrophages, and disruption of the oral mucosal barrier, all of which are well established in the immunological literature.[40,41]

4.6 Multivariable Analysis and Independent Predictors

The multivariable logistic regression model identified four independent predictors of alveolar osteitis: current smoking (adjusted OR 2.45; P=0.004), mandibular molar tooth type (adjusted OR 1.85; P=0.03), and surgical extraction as opposed to simple extraction (adjusted OR 1.72; P=0.04). Age and sex did not reach statistical significance after adjustment. The association with mandibular molar extractions is well established and relates to the anatomically unfavourable blood supply, deeper socket morphology, and the greater surgical trauma frequently required for these teeth.[42] The independent contribution of extraction complexity (surgical vs. simple) aligns with evidence that periosteal reflection, bone removal, and prolonged operative time each independently elevate inflammatory burden and fibrinolytic activity at the wound site.[43] Critically, even after accounting for these procedural and anatomical confounders, smoking retained a robust, independent effect, affirming that the biological impact of tobacco is not merely mediated through case complexity.

4.7 Clinical Implications

The cumulative evidence from this study supports the integration of structured pre-operative tobacco cessation advice into routine oral surgical assessment and management. At least two weeks of preoperative abstinence has been shown to meaningfully reduce postoperative complication rates.[44] A randomised controlled trial by Thomsen et al. (2010) demonstrated that intensive perioperative smoking cessation intervention reduced overall surgical complications by 49%, a magnitude of effect that is clinically transformative.[45] While the feasibility of achieving complete cessation before urgent or semi-elective extractions may be limited, even a reduction in cigarette consumption, particularly in heavy smokers, may confer a measurable risk reduction given the dose–response relationship demonstrated in this study. Practically, clinicians should advise patients to avoid smoking for at least 24–72 hours postoperatively to protect the forming clot, use chlorhexidine-impregnated dressings in high-risk sockets, and schedule earlier review appointments for smokers to detect alveolar osteitis at an earlier, more manageable stage.[46]

4.8 Limitations

Several limitations of the present study warrant acknowledgement. First, smoking status was determined by self-report, introducing the possibility of misclassification bias, as some participants may have under-reported tobacco use due to social desirability. Biochemical verification of smoking status using cotinine assays was not performed.[47] Second, post-extraction smoking behaviour including the timing, quantity, and method of tobacco use relative to the procedure was not captured, precluding analysis of perioperative smoking patterns as a modifying variable. Third, the study did not account for the use of e-cigarettes or other tobacco products, which may carry different risks given their distinct delivery of nicotine and combustion by-products.[48] Fourth, while multivariable regression adjusted for several key confounders, unmeasured variables such as oral hygiene status, hormonal contraceptive use, and pre-existing periodontal disease may have contributed to residual confounding.[49] Finally, the generalisation of findings may be limited by the single-centre design and the specific population studied.

4.9 Conclusion

This study provides robust, multivariable-adjusted evidence that smoking is an independent and dose-dependent risk factor for alveolar osteitis, postoperative pain, delayed soft tissue healing, and postoperative infection following dental extraction. The observed biological gradient across smoking intensity groups strengthens the causal inference

and underscores the potential clinical benefit of targeted cessation counselling in oral surgical patients. Future research using biochemical verification of smoking status, including e-cigarette users, and incorporating detailed perioperative smoking behaviour data would further refine risk stratification and inform the design of preventive interventions.

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Healing Grade	Clinical Criteria
Very poor 1	Tissue color: > 50% of gingivae red Response to palpation: bleeding Granulation tissue: present Incision margin: not epithelialized, with loss of epithelium beyond margins Suppuration: present
Poor 2	Tissue color: > 50% of gingivae red Response to palpation: bleeding Granulation tissue: present Incision margin: not epithelialized, with connective tissue exposed
Good 3	Tissue color: < 50% of gingivae red Response to palpation: no bleeding Granulation tissue: none Incision margin: no connective tissue exposed
Very good 4	Tissue color: < 25% of gingivae red Response to palpation: no bleeding Granulation tissue: none Incision margin: no connective tissue exposed
Excellent 5	Tissue color: all gingivae pink Response to palpation: no bleeding Granulation tissue: none Incision margin: no connective tissue exposed

APPENDIX 1