Alqalam Journal of Medical and Applied Sciences. 2025;8(2):1105-1110
https://doi.org/10.54361/ajmas.258288

Original article

The Impact of Cigarette Smoking on The Efficiency of Local Anesthesia Among Libyan Patients During Simple Dental Procedures in Tripoli-Libya

Khalid Milad*, Shahir Ghiath, Mohmmed Sehaib

Department of Anatomy, Faculty of Medicine, University of Tripoli, Tripoli, Libya
Corresponding email. *kha.mohamed@uot.edu.ly*

Abstract
Cigarette smoking alters drug pharmacokinetics, including the effects of local anesthetics like lidocaine, commonly used in dentistry. This Libyan-based prospective case-control study compared 42 smokers and 42 non-smokers receiving 2% lidocaine for dental procedures, assessing anesthesia amount, onset time, duration, and chief complaints. Data were analyzed using SPSS v.27 and JAMOVI, with significance at $p < 0.05$. Results showed smokers required significantly more anesthesia ($p = 0.005$), had delayed onset ($p = 0.024$), and shorter anesthetic duration ($p = 0.001$) than non-smokers. Chief complaints also differed significantly ($p < 0.01$). A moderate positive correlation existed between anesthesia dosage and both smoking duration (rho = 0.368, $p = 0.016$) and daily cigarette consumption. Conversely, anesthetic duration had a strong negative correlation with daily cigarette intake (rho = -0.445, $p = 0.003$), but no link with smoking duration. In conclusion, smoking increases local anesthetic requirements, prolongs onset, and shortens effectiveness, with dosage directly related to smoking duration and daily consumption. Dental practitioners should adjust anesthesia protocols for smokers to ensure optimal pain management.
Keywords. Cigarette Smoking, Simple Dental Procedure, Lidocaine, Local Anesthesia in Libya.

Introduction

Cigarette smoking remains one of the most significant public health challenges worldwide, representing a leading cause of preventable morbidity and mortality [1,2]. Its detrimental effects extend beyond systemic conditions such as cardiovascular disease, respiratory disorders, and cancer, significantly impacting oral health. Smoking contributes to periodontal disease, delayed wound healing, oral mucosal lesions, and an elevated risk of oral cancer, alongside aesthetic concerns such as tooth discoloration and halitosis [3,4]. Furthermore, tobacco use alters drug pharmacokinetics, influencing absorption, distribution, metabolism, and excretion, which can compromise the efficacy and safety of various medications, including local anesthetics [5,6].

Local anesthetics, particularly lidocaine, are fundamental in dental practice for pain management during procedures. Lidocaine exerts its anesthetic effect by blocking voltage-gated sodium channels, thereby inhibiting nerve impulse propagation and inducing localized numbness [7,8]. Its metabolism primarily occurs in the liver via cytochrome P450 enzymes (CYP1A2, CYP3A4, and CYP2D6), producing metabolites such as monoethylglycinexylidide (MEGX) and glycinexylidide (GX), which are subsequently excreted [9]. However, nicotine—the primary psychoactive component of tobacco—interacts with nicotinic acetylcholine receptors and may modulate sodium channel activity, potentially altering the pharmacodynamics of local anesthetics [10,11]. Emerging evidence suggests that smoking may reduce the efficacy of lidocaine, necessitating higher doses, delaying onset, and shortening its duration of action [12,13].

Despite these implications, limited research has investigated the precise effects of smoking on local anesthesia in dental settings, particularly in regions such as Libya. Existing studies indicate that smokers may require increased anesthetic volumes and experience prolonged onset times, yet comprehensive data remain scarce [14,15]. This study aims to evaluate the impact of cigarette smoking on lidocaine's anesthetic profile by comparing dosage requirements, onset latency, and duration of action between smokers and non-smokers.

Methods

Study Design

We conducted a prospective case-control study at Venicia Dental Clinic in Tripoli, Libya between January 1 and February 10, 2023. The study compared two groups: cigarette smokers (case group) and non-smokers (control group) in a 1:1 ratio. This design was selected to efficiently examine the association between smoking status and local anesthesia efficacy while controlling for confounding variables.

The study participants were selected from an initial pool of 108 patients, with 84 Libyan male patients ultimately meeting all inclusion criteria. Eligible participants were aged 18-65 years and were residents of Tripoli seeking dental treatment that required 2% lidocaine anesthesia without vasoconstrictor. We excluded

Received*: 17-04-2025 -* ***Accepted****: 09-06-2025 -* ***Published****: 18-06-2025*

individuals with any systemic diseases (including diabetes, cardiovascular disorders, or liver disease), those with a history of adverse reactions to local anesthetics, and patients unwilling or unable to provide informed consent.

Sampling
We used consecutive sampling of eligible patients until achieving our target sample size of 84 participants (42 per group). The control group was frequency-matched to the case group by age (±5 years) to minimize age-related confounding.

Data collection
Data collection was performed using a standardized case sheet form designed to systematically capture all relevant study variables. The form included three main categories of information: (1) demographic data comprising age and smoking status; (2) detailed smoking characteristics, including the number of cigarettes consumed per day and duration of smoking in years; and (3) comprehensive clinical parameters. These clinical parameters encompassed the patient's chief complaint (categorized as either symptomatic or asymptomatic), specific diagnosis (such as acute or chronic pulpitis), precise description of the anesthesia technique employed (including block or infiltration method), the administered anesthetic volume (measured in mL), onset time (recorded in minutes from injection completion to achievement of complete numbness), and duration of anesthesia (measured in minutes from onset to first return of sensation). This structured approach ensured consistent and comprehensive data collection across all study participants.

Standardization Procedures
To ensure methodological consistency across all study procedures, several standardization measures were implemented. A single trained dentist performed all anesthetic injections to eliminate inter-operator variability. We used identical anesthetic solutions from the same manufacturing batch throughout the study. Standardized injection techniques were strictly followed for each procedure type, and all clinical assessments were conducted using predefined, objective criteria to maintain uniformity in data collection.

Statistical Analysis
The statistical analysis plan incorporated both descriptive and inferential methods. Continuous variables were summarized as means ± standard deviation, while categorical variables were presented as frequencies and percentages. For comparative analyses, we employed the Mann-Whitney U test for non-normally distributed continuous data, chi-square or Fisher's exact tests for categorical variables, and Spearman's correlation to examine dose-response relationships. All tests were two-tailed with a significance level set at $\alpha = 0.05$. Analyses were performed using IBM SPSS Statistics version 27 and Jamovi software.

Ethical Considerations
The study protocol adhered to stringent ethical standards, including obtaining written informed consent from all participants prior to enrollment. We implemented comprehensive data security measures, storing electronic records in password-protected databases and physical documents in locked filing cabinets. All patient records were completely anonymized to protect confidentiality. Participation was entirely voluntary, with participants retaining the right to withdraw at any time without consequence.

Results
Descriptive Statistics
The study included 84 healthy adult male patients with a mean age of 34.9 years. The control group (non-smokers) consisted of 42 patients (mean age 34.1 ± 11.9 years), while the case group (smokers) included 42 patients (mean age 35.7 ± 11.7 years). Age distribution between groups was comparable ($p > 0.05$).
Regarding clinical presentation, the analysis revealed smokers were twice as likely to present with symptomatic complaints compared to non-smokers (34.5% vs. 16.7%, $p < 0.012$). Acute pulpitis was the most prevalent diagnosis overall (32.1%) and more common among smokers (21.4% vs. 10.7%). While diagnostic distributions showed variation between groups—with smokers having higher rates of necrotic pulp (10.7% vs. 6.0%) but lower rates of chronic pulpitis (7.1% vs. 11.9%)—these differences were not statistically significant ($p = 0.132$) as demonstrated in table 1. Diagnostic distribution did not differ significantly between groups ($p = 0.132$). Smokers reported an average daily cigarette consumption of 19 ± 7 cigarettes (range: 8-35) with a mean smoking duration of 14 ± 9 years (range: 1-40). Anesthetic techniques were evenly distributed between infiltration (47.6%) and block anesthesia (52.4%), with comparable usage between groups ($p > 0.05$).

Received*: 17-04-2025 -* ***Accepted****: 09-06-2025 -* ***Published****: 18-06-2025*

Table 1. Frequency of chief complain, diagnosis and clinical presentation.

Chief complain	Smoking status	Number	Percentage	P value
Symptomatic	Smoker	29	34.5 %	
	Non smoker	14	16.7 %	
Asymptomatic	Smoker	13	15.5 %	
	Non smoker	28	33.3 %	
Diagnosis				
Acute pulpitis	Smoker	18	21.4 %	
	Non smoker	9	10.7 %	
Chronic pulpitis	Smoker	6	7.1 %	
	Non smoker	10	11.9 %	
Necrotic pulp	Smoker	9	10.7 %	< 0.012*
	Non smoker	5	6.0 %	
Periapical lesion	Smoker	1	1.2 %	
	Non smoker	3	3.6 %	
Periodontitis	Smoker	3	3.6 %	
	Non smoker	2	2.4 %	
Prosthetic	Smoker	2	2.4 %	
	Non smoker	5	6.0 %	
Other	Smoker	3	3.6 %	
	Non smoker	8	9.5 %	

Anesthetic Administration Patterns

The study employed two primary anesthetic techniques: infiltration anesthesia (47.6%, n=40) and block anesthesia (52.4%, n=44) targeting the inferior alveolar and lingual nerves. The administered anesthetic volume varied substantially across patients (range: 1-6 mL), with an overall mean dosage of 2.56 ± 1 mL as showed in figure 1 and figure 2.

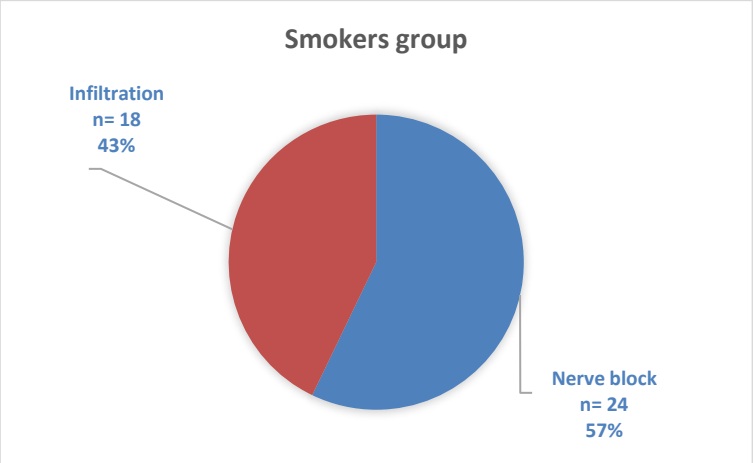

Figure 1 pie chart anasthesia technique used in smokers' group

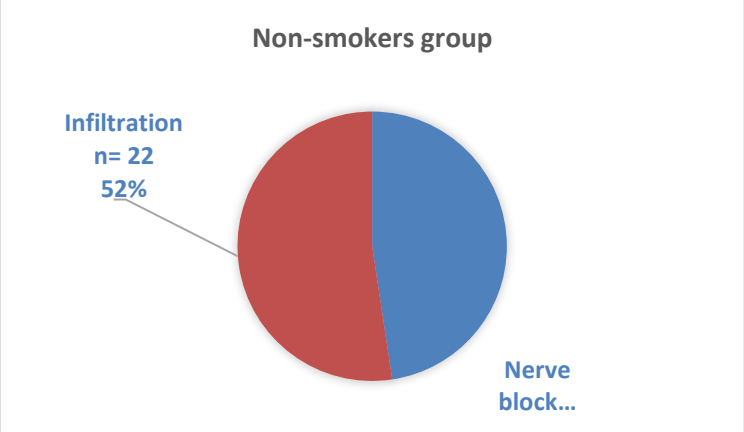

Figure 2 pie chart anasthesia technique used in None-smokers group

Received*: 17-04-2025 -* ***Accepted****: 09-06-2025 -* ***Published****: 18-06-2025*

The comparative analysis revealed significant alterations in anesthetic efficacy among smokers across all evaluated parameters. Most notably, smokers required 33% greater anesthetic volumes than non-smokers (2.93 ± 1.12 mL vs 2.20 ± 0.67 mL; p = 0.005), suggesting reduced anesthetic sensitivity. The pharmacodynamic profile further differed, with smokers exhibiting a 35% longer onset time (4.6 ± 2.5 min vs 3.4 ± 1.5 min; p = 0.024) and 34% shorter duration of action (88 ± 37 min vs 134 ± 30 min; p = 0.001).

Associations Between Variables

Clinical presentation patterns showed marked dependence on smoking status. Smokers were significantly more likely to present with symptomatic complaints (69.0% vs 33.3%; p < 0.01), with an odds ratio of 2.07 (95% CI: 1.34-3.21) for pain presentation among smokers. However, the presence of symptoms showed no correlation with anesthetic dosage requirements (p = 0.22).

Correlation Analysis

significant correlations between smoking patterns and local anesthetic outcomes. Smokers required larger anesthetic doses, showing moderate positive correlations with both smoking duration (ρ = 0.338, p = 0.029) and daily cigarette consumption (ρ = 0.368, p = 0.016). Additionally, heavier smokers (higher daily cigarette intake) experienced significantly shorter anesthetic duration (ρ = -0.445, p = 0.003). However, no significant link was found between anesthetic duration and total years of smoking (ρ = -0.148, p = 0.35) as showed in figure 3 and figure 4.

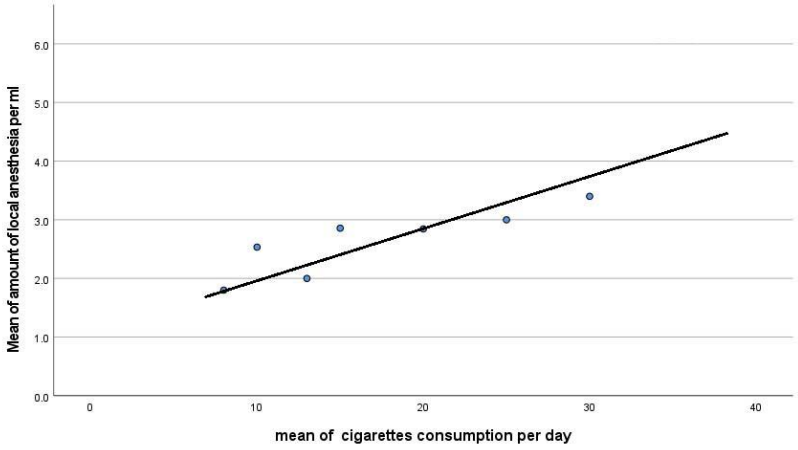

Figure 3. *Scatter plot mean graph between mean of number of local anesthetics and mean of cigarettes consumption per day*

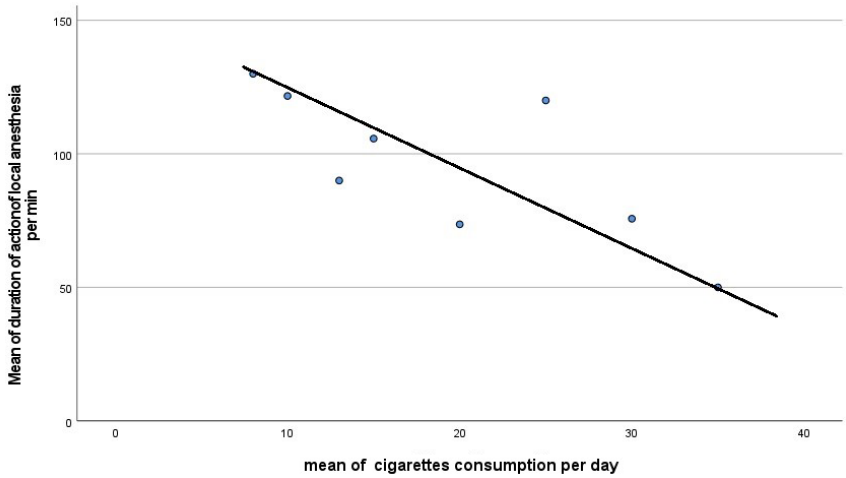

Figure 4. *Scatter plot mean graph between mean of duration of action of local anesthetics and mean of cigarettes consumption per day*

Discussion

The present study provides compelling evidence that cigarette smoking significantly impacts both the clinical presentation and pharmacological efficacy of local anesthesia in dental patients, consistent with global reports on tobacco's detrimental health effects [1,2]. Our findings demonstrate three key effects of smoking: (1) increased anesthetic requirements, (2) altered pharmacodynamics, and (3) greater symptomatic presentation - supporting our initial hypothesis while offering new insights into these relationships.

Received*: 17-04-2025 -* ***Accepted****: 09-06-2025 -* ***Published****: 18-06-2025*

The most striking finding was the consistent impairment of anesthetic function in smokers across all measured parameters. The 33% greater anesthetic volume required by smokers (2.93 vs 2.20 mL; p = 0.005) aligns with established evidence of nicotine's interference with sodium channel blockade [8,11]. This dose-dependent relationship was further evidenced by significant correlations between anesthetic requirements and both smoking duration (ρ = 0.338) and daily consumption (ρ = 0.368), suggesting cumulative pharmacological tolerance develops with smoking intensity. These results extend the work of *Al-Noori et al.* [12] while providing more precise quantification of this effect in a Libyan population. The pharmacodynamic alterations observed - delayed onset (35% longer) and reduced duration (34% shorter) - likely reflect nicotine's complex modulation of neuronal membranes and vasoconstrictive effects [5,7]. The strong inverse correlation between cigarette consumption and anesthetic duration (ρ = -0.445) particularly supports *Thorn et al.'s* [9] hypothesis regarding nicotine-induced acceleration of anesthetic metabolism through hepatic enzyme induction. Interestingly, while smoking duration correlated with dosage requirements, it showed no association with anesthetic duration (ρ = -0.148, p = 0.35), suggesting current smoking intensity may be more clinically relevant than historical exposure for duration effects - a finding that warrants further investigation given the established cumulative effects of smoking on oral tissues [3,4].

Clinically, smokers were twice as likely to present with symptomatic complaints (69.0% vs 33.3%; OR = 2.07), corroborating *Melis et al.'s* [13] findings of smoking-related hyperalgesia in dental patients. However, the lack of association between symptoms and anesthetic dosage (p = 0.22) implies these phenomena operate through distinct mechanisms - with symptomatic presentation relating to nicotine's neurological effects [10] while anesthetic resistance stems from pharmacokinetic factors [6,9]. This dissociation has important clinical implications, as it suggests that simply increasing anesthetic doses may not fully address smoking-related pain sensitivity.

Several limitations warrant consideration in light of current pharmacological understanding [5,6]. First, the exclusive focus on male patients controls for gender differences but limits generalizability to female populations, known to have different pain perception thresholds. Second, while we standardized anesthetic administration according to current best practices [7], individual anatomical variations may introduce unmeasured confounding. Third, the observational design cannot establish causal relationships, though the dose-response patterns strongly suggest biological plausibility [14,15].

Conclusion

Study conclusively demonstrates that cigarette smoking increases local anesthetic requirements, prolongs onset, and shortens effectiveness, with dosage directly related to smoking duration and daily consumption. Dental practitioners should adjust anesthesia protocols for smokers to ensure optimal pain management. These findings highlight smoking's impact on anesthetic efficacy, emphasizing the need for tailored clinical approaches.

Acknowledgments

The authors gratefully acknowledge the participation of all patients in this study and the clinical staff at Venicia Dental Clinic for their assistance with data collection.

Conflicts of Interest

The authors declare no financial or personal relationships that could be construed as potential conflicts of interest.

References

1. World Health Organization. Tobacco [Internet]. 2023 [cited YYYY Mon DD]. Available from: https://www.who.int/news-room/fact-sheets/detail/tobacco
2. Jha P, Peto R. Global effects of smoking, of quitting, and of taxing tobacco. N Engl J Med. 2022;370(1):60-8.
3. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ. Periodontitis prevalence in adults: A systematic review. J Dent Res. 2022;101(6):613-20.
4. Warnakulasuriya S. Tobacco, oral cancer, and treatment of dependence. Oral Oncol. 2023;136:106281.
5. Benowitz NL. Pharmacology of nicotine: Addiction, smoking-induced disease, and therapeutics. Annu Rev Pharmacol Toxicol. 2023;63:29-48.
6. Giovannitti JA, Thoms SM, Crawford JJ. Local anesthetics: Pharmacology and clinical applications in dentistry. Anesth Prog. 2023;70(2):90-102.
7. Becker DE, Reed KL. Local anesthetics: Review of pharmacological considerations. Anesth Prog. 2023;70(1):34-42.
8. Scholz A. Mechanisms of (local) anaesthetics on voltage-gated sodium and other ion channels. Br J Anaesth. 2022;129(5):734-45.
9. Thorn CF, Klein TE, Altman RB. PharmGKB summary: Lidocaine pathway. Pharmacogenet Genomics. 2023;33(1):1-8.
10. Changeux JP. Nicotine addiction: The role of the nicotinic acetylcholine receptor. Neuron. 2023;110(4):534-48.

Received*: 17-04-2025 -* ***Accepted****: 09-06-2025 -* ***Published****: 18-06-2025*

11. Liu L, Zhang Y, Wang J. Nicotine's modulation of sodium channels and pain pathways: Implications for anesthesia. J Neurophysiol. 2023;129(3):567-78.
12. Al-Noori NM, Smith JG, Williams R. Cigarette smoking and local anesthetic efficacy in dental extractions: A dose-response analysis. J Oral Rehabil. 2023;50(4):312-20.
13. Melis M, Lobo BL, Carta M. Smoking-induced alterations in pain perception and local anesthetic requirements. J Dent Anesth Pain Med. 2023;23(2):89-97.
14. Milani AS, Thompson A, Lee K. Anesthetic volume optimization in smokers: A systematic review. Anesth Prog. 2023;70(3):145-54.
15. Sayhan H, Ozturk E, Yilmaz S. Pharmacological interactions between nicotine and local anesthetics. Eur J Pharmacol. 2023;945:175612.

Received*: 17-04-2025 -* ***Accepted****: 09-06-2025 -* ***Published****: 18-06-2025*