Dentistry 3000

Vol 13, No 1 (2025)
ISSN 2167-8677 (online)
DOI 10.5195/d3000.2025.975

Impact of Fixed Orthodontic Appliances on *Staphylococcus aureus* and *Candida albicans*

Maryam Aljbouri, Nebras Al-Dabbagh, Thaer Jaber Al-Khafaji

College of Dentistry, University of Babylon, Babylon, Iraq

Abstract

Objective: This study investigated the prevalence of salivary *S. aureus* and *C. albicans* in patients with fixed orthodontic appliances and assessed their resistance profiles to selected antibacterial and antifungal agents over three-time intervals. **Material and Methods**: A prospective cohort of 40 patients (20 males, 20 females) undergoing orthodontic treatment was followed across three time points: baseline (T0), two weeks post-application (T1), and four weeks post-application (T2). Unstimulated saliva samples were collected and cultured for microbial identification. Organisms were confirmed via Vitek biochemical testing. Antimicrobial susceptibility was determined using the disc diffusion method against six antibacterial and six antifungal agents. Data were analyzed using two way-ANOVA and chi-square tests. **Results**: Among the 80 total samples analyzed, 73.75% were positive for *S. aureus* and/or *C. albicans*. *S. aureus* was most prevalent, with Clindamycin and Vancomycin showing the strongest antibacterial activity. Resistance to Amoxicillin, Ampicillin, and Erythromycin was notably high. For *C. albicans*, Fluconazole and Amphotericin B demonstrated the highest efficacy, while Clotrimazole and Nystatin showed poor inhibition. Resistance patterns might suggest a biofilm-associated microbial adaptation and reduced susceptibility over time. **Conclusion**: Fixed orthodontic appliances significantly influence oral microbial ecology by facilitating colonization of resistant strains of *S. aureus* and *C. albicans*. The findings highlight the importance of routine microbial surveillance and personalized antimicrobial strategies in orthodontic care to mitigate infection risks and manage emerging resistance.

Open Access

Citation: Aljbouri M, et al. (2025) Impact of Fixed Orthohontic Appliances on Staphylococcus aureus and Candida albicans. Dentistry 3000. 1:a001 doi:10.5195/d3000.2025.975
Received: June 30, 2025
Accepted: July 7, 2025
Published: August 21, 2025

Email: mariamaljbori1999@gmail.com

Introduction

Orthodontic appliances significantly modify the physiological environment of the oral cavity by hindering natural cleaning mechanisms, leading to increased plaque accumulation and impaired oral hygiene. These devices create retention sites that facilitate biofilm formation composed of diverse bacteria and fungi, which colonize various oral surfaces. Consequently, orthodontic biofilms contribute to microbial overgrowth and increase the risk of infection if hygiene is not properly maintained [1].

Fixed orthodontic components such as bands, wires, and brackets complicate oral hygiene practices. Their complex structure and the extended surface area they cover hinder effective cleaning, encouraging plaque accumulation. Inadequate oral hygiene in orthodontic patients can lead to gingival inflammation and progressive periodontal disease. Therefore, maintaining high standards of cleanliness during orthodontic treatment is crucial to preventing long-term oral health complications [2].

The oral cavity hosts the second most diverse microbial community in the human body, following the gut. It comprises numerous bacteria, fungi, and viruses that coexist in a balanced microbiome under normal conditions. This ecosystem plays a vital role in oral health but is vulnerable to disruption through external influences such as orthodontic treatment [3].

Orthodontic therapy has been shown to alter the oral microbiota composition, increasing the prevalence of streptococci, staphylococci, lactobacilli, and yeasts. The physical presence of orthodontic appliances fosters bacterial plaque accumulation, exacerbates gingival inflammation, and deepens probing depth. These conditions create an ideal niche for pathogenic bacteria, potentially escalating to periodontitis if left unchecked [4,5].

Staphylococcus aureus is commonly found on the skin and mucosal surfaces, including the

oropharynx. Though typically harmless in carriers, it can become pathogenic and cause a wide range of infections, from minor skin conditions to severe systemic diseases like osteomyelitis and endocarditis. Around 30% of individuals are asymptomatic carriers, but the spread of antibiotic-resistant strains such as methicillin-resistant *S. aureus* (MRSA) poses a significant clinical challenge [6-8].

Antibiotic resistance in *S. aureus*, particularly MRSA, has made treatment increasingly difficult. These strains have developed resistance to several frontline antibiotics, including penicillin, methicillin, and in some cases, daptomycin and vancomycin. This resistance complicates therapeutic options and demands the development of novel approaches to bacterial infection control [9,10].

As bacterial resistance accelerates faster than antibiotic development, the potential for a post-antibiotic era looms. Understanding bacterial resistance mechanisms such as changes in cell wall composition and virulence is vital for designing new treatments. The peptidoglycan-rich cell wall of *S. aureus* is a critical target due to its role in maintaining bacterial integrity and pathogenicity [11].

Candida species, particularly *C. albicans*, naturally colonize the skin, mucosa, and gastrointestinal tract without causing harm. However, under certain conditions like immunosuppression or poor oral hygiene, they can overgrow and lead to candidiasis. Symptoms vary depending on the infection site [12-14]. *Candida albicans* predominates in the oral cavity, with colonization reported in up to 60% of young adults. However, non-albicans species such as *C. glabrata* and *C. krusei* are becoming more common. These species often display resistance to commonly used antifungals like azoles and polyenes, emphasizing the importance of targeted antifungal therapy [15-17].

This study aimed to evaluate the impact of fixed orthodontic appliances on the salivary prevalence of *Staphylococcus aureus* and *Candida albicans*, as well as their resistance to various antimicrobial agents. The objectives included isolating and identifying both microorganisms using culture media, quantifying their presence at three different treatment intervals, and assessing their resistance patterns to antibacterial and antifungal drugs over time.

Subjects and Methods

A prospective cohort study was followed in which 40 individuals (20: females, 20: males) have undergone orthodontic treatment in orthodontic department, Nasser Almousawi specialized center, Najaf, Iraq.

Eligible study participants: included individuals aged 18 to 35 years with Class I malocclusion and mild to moderate dental crowding, characterized by an ANB angle between 0º and 4º. Subjects also exhibited a normal vertical relationship (SN-MP angle of 32º ± 2), a straight facial profile, and a full set of teeth, with or without third molars.

Excluding criteria: Participants were excluded if they had a history of orthodontic treatment, orthognathic surgery, systemic diseases, craniofacial anomalies, or traumatic injuries. Prior to treatment, all necessary patient information was recorded. Saliva samples were collected at three time points: before the application of fixed orthodontic appliances (T0), two weeks after (T1), and four weeks after treatment began (T2). Each participant underwent comprehensive intraoral and extraoral examinations, including panoramic and lateral cephalometric radiographs, to establish a diagnosis and determine the appropriate treatment plan.

Sample Collection: Unstimulated saliva samples were collected from participants between 9 and 11 a.m. using the passive drooling method.[18] Subjects abstained from food, drink, and oral hygiene for at least one hour prior. After rinsing with 150 ml distilled water and resting, they allowed approximately 3 ml of saliva to collect in labeled containers. Samples were immediately stored on ice and transported to the microbiology laboratory in Najaf Governorate [19].

Ethical approval: This study was conducted in accordance with the ethical principles of the Declaration of Helsinki and the scientific committee in College of Dentistry/ University of Babylon was approved under Protocol Number 123, dated 03/10/2024.

Isolation and Identification of S*. aureus:* The specimens were transferred directly to the laboratory. At 37°C for 24 hours, blood agar media was used then Mannitol salt agar (MSA) for the cultivation of *S. aureus*. The grown bacterial colonies were subjected to various biochemical tests by API system (API 20 -Vitek apparatus) to ensure their identity [20].

Antibiotic Sensitivity Test: The antibiotic sensitivity test was done following the disk diffusion method.[21] Using a sterile loop, 4-5 colonies were suspended in 2 mL of sterile normal saline. The bacterial suspension's turbidity was adjusted to a McFarland standard of 0.5. The bacteria were then inoculated into the mueller-hinton agar plate by using a sterile swab. The plates were left at room temperature for about 5 minutes. The following antibiotics were used: Clindamycin (2µg), Erythromycin (15µg), Azithromycin (15µg), Ampicillin (10µg), Amoxicillin (10µg) and Vancomycin (30µg). Using sterile forceps, the antibiotic discs were placed and fixed on the surface of the cultivated agar. Finally, the plates were inverted and incubated at 37°C for 18 h.

Preparation of Specialized Culture Media for *Candida albicans*

Sabouraud's Dextrose Agar Medium (SDA)

This medium (65 g) was dissolved in (1000 mL) distilled water and heated to dissolve in a glass beaker, after which the medium was sterilized in an autoclave, distributed to Petri dishes, and (0.05 g) anti-chloramphenicol was added after cooling the medium to (45°C) to prevent bacterial growth [22].

Chromo Agar Candida Medium

The medium was prepared according to the manufacturer's instructions by suspending 42.72 g of powder in 1000 mL of distilled water, and the solution was heated to boiling point to completely dissolve the medium.

Isolation and Identification of *Candida* Diagnosis using biochemical test (Vitek apparatus)

It is a selective and differential chromogenic medium intended for the identification of Candida species and to determine color and colony morphology well when chromium was used for *Candida* isolate directly and filamentous fungi and of clinical specimens. *C. albicans* grow as green colonies after incubation for 48 hours at 37°C. This medium does not require sterilization in an autoclave.[23] Diagnosed by genus and species using biochemical tests, the Vitek System consists of cassette and reagent Cards that contained 64 pits (every pit represents the substrate to conduct the test), and plastic pipes, as well as the Densi Chek device and the unity of the input and output of information.[24] Diagnosis of Candida species isolates using biochemical tests Diagnosis using the Vitek® apparatus (VITEK® 2 DensiChek; bioMérieux, France). Yeast isolates were diagnosed as genus and species using biochemical tests.

Study the resistance of oral *C. albicans* to the antifungal drugs: A clinical isolates of *Candida* species were tested for antifungal susceptibility against six antifungal drugs [Clotrimazole (10µg), Fluconazole (10µg), Nystatin (50µg), 5-Flucytosine (1µg), Caspofungin (5µg), and Amphotericin B (100µg)]; the antifungal susceptibility of oral *C. albicans* was tested by disc diffusion.

Statistical Analysis: Data were analyzed using SPSS (version 26, SPSS Inc. Chicago, Illinois, USA). Descriptive statistics (mean, standard Error), as the results was expressed as %. Statistical analysis was carried out using chi-square. Differences were compared by two-way ANOVA at $p \leq 0.05$ using Duncan's Multiple Range test [25].

Results

Out of the total 40 participants enrolled in this study, the culture results showed a significant presence of *Staphylococcus aureus* (80%) and *Candida albicans* (68%) among individuals undergoing fixed orthodontic treatment. Among male participants (n = 20) in three period times (T0, T1 and T2), *Staphylococcus aureus* was detected in 16 cases in each period time (40% vs total No.) while *Candida albicans* was found in 13 cases in each period time (32.5% vs total No.). In the female group (n = 20) in three period times (T0, T1 and T2), 16 participants (40% vs total No.) tested positive for *Staphylococcus aureus* in each period time, while only 14 cases (35% vs total No.) were positive for *Candida albicans* in each period time. Overall, 59 out of 80 samples (73.75%) were culture positive for either *S. aureus* or *C. albicans*, whereas 21 samples (26.25%) were negative for both. The prevalence appeared consistent across genders (Table 1).

Antibiotics Susceptibility test for *Staphylococcus aureus Isolates*

Vancomycin and Clindamycin showed the largest inhibition zones (14–15 mm), suggesting strong antibacterial activity against *S. aureus*. The difference is statistically significant (*p < 0.001) compared to all other antibiotics while Azithromycin showed intermediate inhibition (10–11 mm), which is statistically less than Clindamycin but still more effective than Ampicillin and Erythromycin. The weakest Activity were Amoxicillin, Ampicillin, and Erythromycin that demonstrated smaller inhibition zones (5–8 mm), indicating poor antimicrobial effectiveness. These results may reflect the emergence of resistance to β-lactam and macrolide antibiotics among oral *S. aureus* isolates (Figure 1).

Antifungals Susceptibility test for *Candida albicans Isolates*

Fluconazole and Amphotericin B produced the largest inhibition zones (13–14 mm), indicating the strongest antifungal activity against *C. albicans*. Their effect is statistically significant ($p < 0.001$) compared to all other agents while 5-Flucytosine showed moderate inhibition (11 mm) and was significantly better than Nystatin and Clotrimazole but slightly less effective than Fluconazole and Amphotericin. Clotrimazole and Nystatin had the smallest inhibition zones (5–6 mm), suggesting limited antifungal effectiveness against oral *C. albicans* isolates. The statistical differences between Clotrimazole, Nystatin, and Caspofungin were not significant (Figure 2).

Discussion

This study highlighted a significant increase in the prevalence of *Staphylococcus aureus* and *Candida albicans* in the saliva of patients undergoing treatment with fixed orthodontic appliances. The components of these appliances, such as brackets, wires, bands, and ligatures, create multiple niches that favour the accumulation of dental plaque and biofilm. These structures hinder natural self-cleansing mechanisms and complicate routine oral hygiene, fostering a micro-environment that promotes microbial colonization and growth. Consequently, the oral microbial load and diversity are notably altered, creating conditions conducive to the proliferation of pathogenic organisms, especially during the early phases of orthodontic treatment [1,2,4,5].

This observation is in line with existing research indicating that fixed appliances function as reservoirs for microbial biofilms. These devices hinder effective plaque removal and increase the oral burden of opportunistic pathogens. Both *S. aureus* and *C. albicans* are part of the normal oral flora but can become pathogenic when the microbial balance is disrupted such as by reduced immunity, increased substrate availability, or structural changes in the oral environment. This microbial shift can lead to localized infections, including oral candidiasis and gingival inflammation, as the pathogens exploit the altered oral ecology [3,6,26-28].

Antibiotic and antifungal susceptibility tests revealed patterns of resistance that pose a clinical concern. Methicillin-resistant *S. aureus* (MRSA) and antifungal-resistant *C. albicans* are increasingly identified in orthodontic patients, raising treatment challenges. Resistance mechanisms such as gene mutations, efflux pumps, and biofilm-related protection contribute to diminished therapeutic outcomes. This highlights the need for ongoing microbiological surveillance and tailored antimicrobial strategies in orthodontic practice to prevent complications arising from resistant infections [8,9,29-32].

The antibiotic sensitivity analysis in this study showed limited efficacy of commonly used agents such as Amoxicillin, Ampicillin, and Erythromycin, likely due to β-lactamase production and other resistance mechanisms. In contrast, Clindamycin and Vancomycin demonstrated strong inhibitory effects, reflecting their distinct modes of action. Clindamycin by targeting the 50S ribosomal subunit and Vancomycin by inhibiting cell wall synthesis with binding to the terminal D-alanyl-D-alanine residues of peptidogly can precursors, specifically lipid II [9,29,33].

Likewise, antifungal testing demonstrated varied efficacy. Clotrimazole and Nystatin showed minimal inhibition zones, suggesting possible resistance due to frequent topical use. Conversely, Fluconazole and Amphotericin B were highly effective against *C. albicans*, supporting their continued use in oral candidiasis. Amphotericin B's mechanism of binding to ergosterol and disrupting fungal membranes underpins its potency, especially in drug-resistant strains [16,28,3034,35]. 5-Flucytosine, though moderately effective, remains valuable in combination therapies due to its rapid resistance development when used alone [36,37].

Overall, these findings emphasize the importance of incorporating routine susceptibility testing and individualized treatment approaches into orthodontic care. As fixed appliances significantly alter the oral microenvironment, they increase the risk of harboring resistant microbial species. Regular microbial assessments and targeted antimicrobial interventions are essential for preventing and managing persistent or recurrent oral infections in orthodontic patients [1].

Acknowledgements

We would like to extend our heartfelt thanks to the orthodontic department, Nasser Almousawi specialized center, Najaf, Iraq, and special thanks to Orthodontist Dr. Almustafa Alhumadi for their support and the assistance provided.

Conflict of Interest Statement

None to declare.

References

1. Lucchese A, Bondemark L. The influence of orthodontic treatment on oral microbiology. Biological mechanisms of tooth movement, 2021: 139–158.
2. Mazin H, Salman SA, Salah R. The effect of fixed orthodontic appliances on gingival health diode laser versus scalpel gingivectomy view project periodontology view project. IOSR Journal of dental and medical sciences (IOSR-JDMS), 15(11), 2016: 82–88. Available at: https://doi.Org/10.9790/0853-1517078288.
3. Deo PN, Deshmukh R. Oral microbiome: unveiling the fundamentals. journal of oral and maxillofacial pathology. JOMFP. 2019; 23(1), p. 122.
4. Kado I, Hisatsune J, Tsuruda K, Tanimoto K, Sugai M. The impact of fixed orthodontic appliances on oral microbiome dynamics in Japanese patients. Sci Rep. 2020;10(1):21989. doi: 10.1038/s41598-020-78971-2.
5. Kim SH, Choi DS, Jang I, Cha BK, Jost-Brinkmann PG, Song JS. Microbiologic changes in subgingival plaque before and during the early period of orthodontic treatment. Angle Orthod. 2012 ;82(2):254-60. doi: 10.2319/030311-156.1.
6. Rojo P et al. (2010). Community-associated S. Aureus infections in children. Expert Rev Anti Infect Ther, 8, 541–554.
7. Ryu,S.; Song, P.I.; Seo, C.H.; Cheong, H.; Park, Y. Colonization and infection of the skin by S.

Aureus: immune system evasion and the response to cationic antimicrobial peptides. Int. J. Mol. Sci. 2014, 15, 8753–8772.
8. Kale, P., Dhawan, B. (2016). The changing face of community-acquired methicillin-resistant staphylococcus aureus. Indian J. Med. Microbiol., 34, 275–285.
9. Casanova, G.N.; Ruiz, S.M.; Bellido, M.J.L. Mechanisms of resistance to daptomycin in staphylococcus aureus. Rev. Española quimioter. (2017), 30, 391–396.
10. Sirichoat, A.; Lulitanond, A.; Kanlaya, R.; Tavichakorntrakool, R.; Chanawong, A.; Wongthong, S.; Thongboonkerd, V. Phenotypic characteristics and comparative proteomics of staphylococcus aureus strains with different vancomycin-resistance levels. Diagn. Microbiol. Infect. Dis. 2016, 86, 340–344.
11. Romaniuk, J.A.; Cegelski, L. Bacterial cell wall composition and the influence of antibiotics by cell-wall and whole-cell NMR. Philos. Trans. R. Soc. B biol. Sci. 2015, 370.
12. Al-Tekreeti AR, Al-Halbosiy MMF, Dheeb BI, Hashim AJ, Al-Zuhairi AFH. Molecular identification of clinical candida isolates by simple and randomly amplified polymorphic DNA-PCR. Arab J. Sci. Eng. (2017); doi 10.1007/s13369-017-2762-1.
13. Hussain AF, Sulaiman GM, Dheeb BI, Hashim AJ, Seddiq SH. Improving conditions for gliotoxin production by local isolates of aspergillus fumigatus. Journal of biotechnology research center. (2017); 11(2):14-24.
14. Ayesha A. Prevalence, antifungal susceptibility and biofilm characterization of candida species isolated from tertiary care hospitals, 2016; Quaid-I-Azam University, Islamabad, Pakistan (2018); 1-22.
15. Khanpayeh E, Jafari AA, Tabatabaei Z. Comparison of salivary Candida profile in patients with fixed and removable orthodontic appliances therapy. Iran J Microbiol. 2014 Aug;6(4):263-8.
16. Al-Harba, Huda Reyad; Nasir-Alla, Nebrass; Jabir, Azher Abdulhafidh. The Correlation between Oral Candida albicans and Interlukine-23 in Diabetic Patients in Hilla City, Iraq. Medical Journal of Babylon 21(4):p 915-920, October-December 2024. | DOI: 10.4103/MJBL.MJBL_517_23.
17. Alhamadi W, Al-Saigh RJ, Al-Dabagh NN, Al-Humadi HW. Oral Candida in Patients with Fixed Orthodontic Appliance: In Vitro Combination Therapy. Biomed Res Int. (2017); 2017:1802875. doi: 10.1155/2017/1802875.
18. Navazesh, M., Kumar, S.K. 2008. Measuring salivary flow: challenges and opportunities. The journal of the american dental association, 139, 35S-40S.
19. Kaushik, R., Yeltiwar, R. K., Pushpanshu, K. 2011. Salivary interleukin-1β levels in patients with chronic periodontitis before and after periodontal phase I therapy and healthy controls: A case-control study. Journal of periodontology, 82, 1353-1359
20. James C, Natalie S. Microbiology. A laboratory manual. Pearson education, 2014. Available from: https://lib.Hpu.Edu.Vn/handle/123456789/28998
21. Hudzicki J. Kirby-bauer disk diffusion susceptibility test protocol. American society for microbiology. 2009;15:55-63.
22. Scognamiglio T, Zinchuk R, Gumpeni P, Larone DH. Comparison of inhibitory mold agar to Sabouraud dextrose agar as a primary medium for isolation of fungi. J Clin Microbiol. 2010 May;48(5):1924-5. doi: 10.1128/JCM.01814-09.
23. Nadeem, S.G., Hakim, S.T., Kazmi, S.U. (2010). Use of chromagar candida for the presumptive identification of candida species directly from clinical specimens in resource-limited settings, Libyan Journal of Medicine, 5(1).
24. Abdulbaqi NJ, Dheeb BI, Irshad R. Expression of biotransformation and antioxidant genes in the liver of albino mice after exposure to aflatoxin B1 and an antioxidant sourced from turmeric (curcuma longa). Jordan Journal of Biological Sciences. (2018); 11(2) 89 – 93.
25. Cohen, J. (2013). Statistical power analysis for the behavioral sciences. Routledge.
26. Wade, W.G. The oral microbiome in health and disease, Pharmacological Research, (2013); 69(1), pp. 137–143.
27. Salman, S.A., Salah, R. (2016). The effect of fixed orthodontic appliances on gingival health diode laser versus scalpel gingivectomy view project periodontology view project, IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), 15(11), pp. 82–88. Available at: https://doi.Org/10.9790/0853- 1517078288.
28. Moyes DL, Wilson D, Richardson JP, Mogavero S, Tang SX, Wernecke J, Höfs S, Gratacap RL, Robbins J, Runglall M, Murciano C, Blagojevic M, Thavaraj S, Förster TM, Hebecker B, Kasper L, Vizcay G, Iancu SI, Kichik N, Häder A, Kurzai O, Luo T, Krüger T, Kniemeyer O, Cota E, Bader O, Wheeler RT, Gutsmann T, Hube B, Naglik JR. Candidalys in is a fungal peptide toxin critical for mucosal infection. Nature. 2016 Apr 7;532(7597):64-8. doi: 10.1038/nature17625.
29. Ali T, Basit A, Karim AM, Lee J-H, Jeon J-H, Lee SH. Mutation-based antibiotic resistance mechanism in methicillin-resistant staphylococcus aureus clinical isolates. Pharmaceuticals. Https://doi.Org/10.3390/ph14050420 18. (2021);14(5):420.
30. Nobile CJ, jJhnson AD. (2015). Candida albicans biofilms and human disease. Annu rev microbiol, 69:71–92.
31. Parastan R, Kargar M, Solhjoo K, Kafilzadeh F. A synergistic association between adhesion-related genes and multidrug resistance patterns of staphylococcus aureus isolates from different patients and healthy individuals. Journal of global antimicrobial resistance. 2020;22:379-85. Https://doi.Org/10.1016/j.Jgar.2020.02.025
32. Abbasi Montazeri, E., Khosravi, A.D., Khazaei, S. et al. Prevalence of methicillin resistance and superantigenic toxins in Staphylococcus aureus strains isolated from patients with cancer. BMC Microbiol 21, 262 (2021). https://doi.org/10.1186/s12866-021-02319-7
33. Gardete S, Tomasz A. Mechanisms of vancomycin resistance in Staphylococcus aureus. J Clin Invest. 2014 Jul;124(7):2836-40. doi: 10.1172/JCI68834.
34. Achkar JM, Fries BC. Candida infections of the genitourinary tract. Clinical microbiology reviews. (2010); 23(2), 253–273.
35. Abdalwahd N., Al-Saigh R.J., Al-Humadi H.W. Assessment of antifungal drugs' activity against some *Candida albicans* isolates in the presence or absence of human albumin: a study employing an *in vitro* pharmacokinetics / pharmacodynamics model. Rev. Clin. Pharmacol. Pharmacokinet. Int. Ed. 38(Sup2): 39-42 (2024). https://doi.org/10.61873/SEXH5182
36. Bhavan, P., Rajkumar, R, Subramanian, Radhakrishnan, Seenivasan, Dr. Chandrasekar & Kannan, Soundarapandian. Culture and Identification of Candida Albicans from Vaginal Ulcer and Separation of Enolase on SDS-PAGE. International Journal of Biology. (2010); Vol 2. 10.5539/ijb.v2n1p84.
37. Abbas, Nada Fadhil. Comparison between Conventional Methods and Molecular Diagnosis for Candida albicans and Candida dubliniensis isolated from Cancer Patients Infected with Oral Candidiasis. Medical Journal of Babylon 21(Suppl 2):p S276-S281, November 2024. | DOI: 10.4103/MJBL.MJBL_1079_23

Table 1. Culture positive and culture negative for *Staphylococcus aureus* and *Candida albicans.*

Type of Microorganisms	Gender	Culture Positive		Culture Negative		Total
Staphylococcus aureus	Male	16	32	4	8	**40**
	Female	16		4		
Candida albicans	Male	13	27	7	13	**40**
	Female	14		6		
Total		**59**		**21**		**80**

Vol 13, No 1 (2025) DOI 10.5195/d3000.2025.975

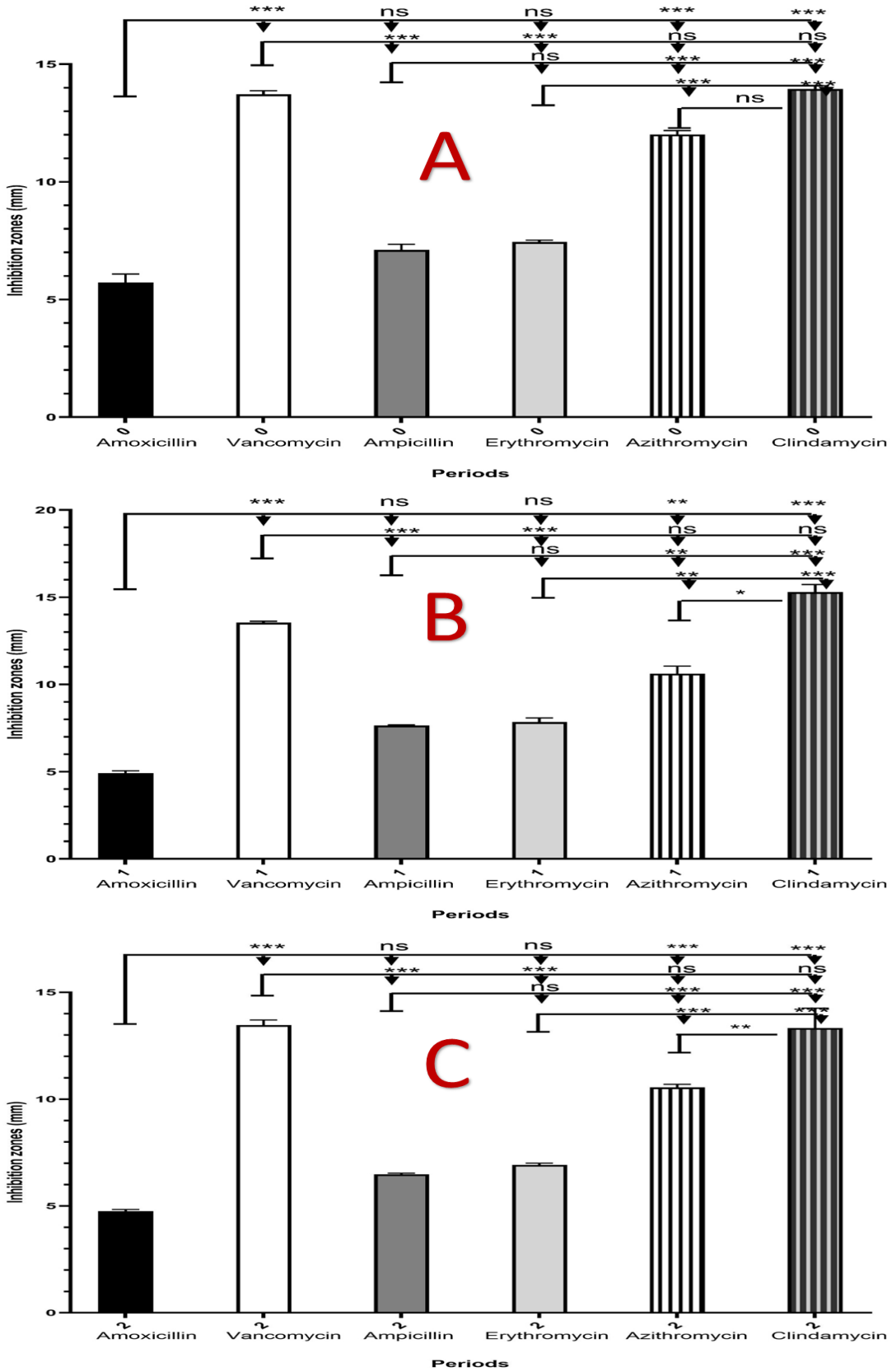

Figure 1. The bar graph illustrates the inhibition zones (mm) for Staphylococcus aureus isolates at three-time intervals (T0 (A), T1 (B), T2 (C)), when exposed to six different antibiotics: Amoxicillin, Vancomycin, Ampicillin, Erythromycin, Azithromycin, and Clindamycin. Statistical comparisons between groups are indicated with asterisks (* for $p < 0.05$, ** for $p < 0.01$, *** for $p < 0.001$) and "ns" for non-significant differences.

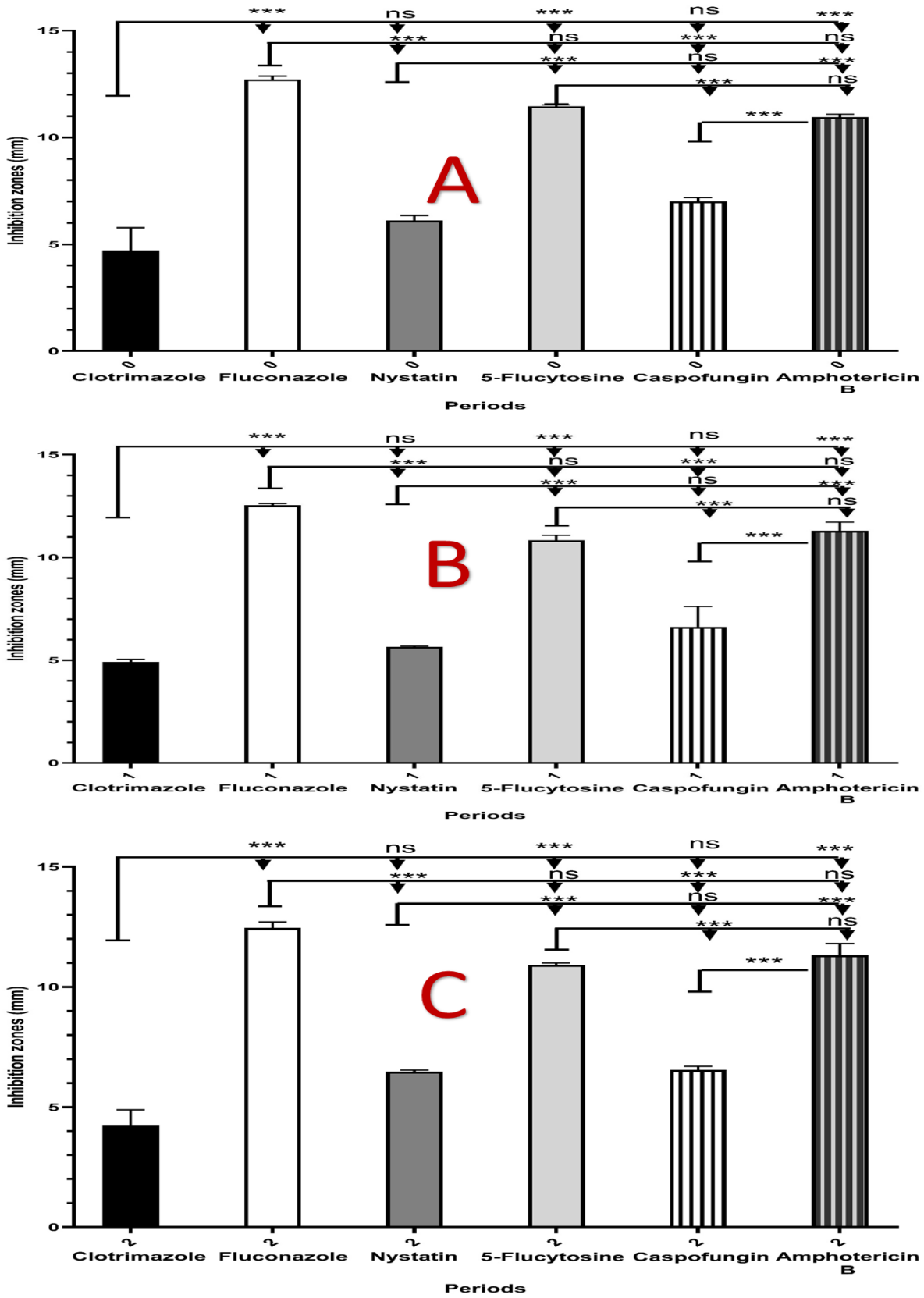

Figure 2. The bar graph illustrates the inhibition zones (mm) for *Candida albicans* isolates at three-time intervals (T0 (A), T1 (B), T2 (C)), when exposed to six antifungal agents: **Clotrimazole**, **Fluconazole**, **Nystatin**, **5-Flucytosine**, **Caspofungin**, and **Amphotericin B**. Statistical comparisons between groups are annotated with *** for $p < 0.001$ and "ns" for non-significant differences.