Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 9s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Unveiling the Differential Impact of Diabetes on Maxillofacial Space Infection Spread and Progression: A Comparative Analysis

Dhiral Vijayvargiya[1], Amit Kumar Sharma[2], Sunil Sharma[3], Prateek Agarwal[4], Abhishek Pandey[*5], Vaishali Pradhan[6]

[1]Post Graduate Resident, Department of Oral and Maxillofacial Surgery, NIMS Dental College and Hospital, Jaipur, Rajasthan, India.

Email ID: dhiralvijayvargiya@gmail.com

[2] Professor and Head, Department of Oral and Maxillofacial Surgery, NIMS Dental College and Hospital, Jaipur, Rajasthan, India.

Email ID: bala.dr0359@gmail.com

[3]Senior Professor, Department of Oral and Maxillofacial Surgery, NIMS Dental College and Hospital, Jaipur, Rajasthan, India.

Email ID: provcnimsuniversity@gmail.com

[4]Professor, Department of Oral and Maxillofacial Surgery, NIMS Dental College and Hospital, Jaipur, Rajasthan, India. Email ID: drprateek@gmail.com

[*5]Consultant of Oral and Maxillofacial Surgery, Kota, Rajasthan, India.

Email ID: abi2511618@gmail.com

[6]BDS, Private Practitioner, Jaipur, Rajasthan, India.

Email ID: vaishalipradhan3001@gmail.com

***Corresponding Author:**

Abhishek Pandey

Email ID: abi2511618@gmail.com

Cite this paper as: Dhiral Vijayvargiya, Amit Kumar Sharma, Sunil Sharma, Prateek Agarwal, Abhishek Pandey, Vaishali Pradhan, (2025) Unveiling the Differential Impact of Diabetes on Maxillofacial Space Infection Spread and Progression: A Comparative Analysis. *Journal of Neonatal Surgery*, 14 (9s), 560-571.

ABSTRACT

Introduction: This study compared head and neck infection characteristics between diabetic and non-diabetic patients. We investigated the spread of infection, causative pathogens, glycemic control, and comorbidities.

Methods: A study was conducted on 48 patients with maxillofacial infections, comparing those with and without diabetes. Febrile status, blood work, pathogen identification, and antibiotic sensitivities were analysed.

Results: Diabetics exhibited significantly greater spread of infection. Streptococcus, Staphylococcus, and Klebsiella were prevalent in both groups. Comorbidity rates were similar. Diabetics had higher HbA1c and random blood sugar.

Conclusion: Diabetes significantly increases the spread of head and neck infections. Prompt treatment and optimization of glycaemic control are crucial for these patients. Further research should explore comorbidity influences.

Keywords: *Diabetes, Maxillofacial Infections, Infection Spread, Glycemic Control, Microbial Pathogens*

1. INTRODUCTION

People are susceptible to a wide range of infections, varying in severity. Disease occurs when there's an imbalance among the host, the infectious agent, and the environment. The host's defence mechanisms are crucial in determining the outcome of an infection. This interaction between microorganism and host can be viewed as a balance between the microorganism's virulence and the host's defenses. Despite advances in antibiotic therapy, maxillofacial space infections persist in modern healthcare settings, with the body's host defenses playing a key role in disease progression. Several factors influence the

spread of these infections, including anatomical variations, compromised immunity, functional impairments, and underlying medical conditions[1] .

Odontogenic fascial space infections can quickly become life-threatening due to interconnected anatomical spaces, proximity to vital structures, and rapid disease progression. Serious complications can include airway obstruction, jugular vein thrombosis, and descending mediastinitis[2] .

While standard infection markers like ESR and WBC count offer a snapshot of a patient's current status, their predictive value is limited. This has driven research into serum-derived markers that could better forecast disease progression and outcomes. Consequently, various inflammatory indicators have been identified and studied[4]. Odontogenic infections are currently the most common type of infection, often arising from poor oral hygiene but also associated with conditions like pharyngitis, tonsillitis, surgical trauma, intravenous drug use, esophageal perforation, laryngopyocele, infected thyroglossal and branchial cysts, and mastoiditis. Patients with compromised immune systems, such as those with diabetes or HIV, are at increased risk of complications. Diabetes mellitus, characterized by impaired carbohydrate, lipid, and protein metabolism due to insufficient insulin secretion or reduced tissue sensitivity, leads to elevated blood glucose levels. This hyperglycemia contributes to various systemic complications, including impaired host defenses and degenerative issues like micro and macroangiopathy, retinopathy, neuropathy, and nephropathy. Micro and macroangiopathy, in particular, significantly influence infection spread and severity. Diabetic patients are generally considered more susceptible to infections, with in vitro studies suggesting impaired neutrophil activity, suppressed antioxidant mechanisms, and reduced humoral immunity.

Diabetic individuals experience reduced chemotaxis and phagocytic activity, impaired polymorphonuclear leukocyte mobility, and diminished neutrophil bactericidal function, cellular immunity, and complement activation in response to infection. Consequently, they face a higher infection rate and greater disease severity compared to non-diabetics[3].

The outcome of an infection depends on both the host's immune response and the timing and implementation of antimicrobial therapy. Diabetic patients often present with different clinical manifestations compared to non-diabetic individuals, necessitating distinct management approaches[11]. While *Streptococcus spp.* is frequently reported in odontogenic infections of non-diabetic individuals, *Klebsiella pneumoniae* is more prevalent in those with diabetes. This suggests a difference in the causative organisms between these two populations[4].

Orofacial infections caused by odontogenic bacteria have been a longstanding human health concern. The development of penicillin by Fleming in 1928, and its subsequent refinement into a usable powder form by Florey and Chain, revolutionized the treatment of these infections.

Antibiotic resistance emerged remarkably quickly, within just four years of penicillin's mass production in 1943. This resistance continues to increase, driven largely by evolutionary processes.

Despite the development of synthetic antibiotics to combat penicillin resistance, resistance to these newer drugs has also emerged. However, penicillin remains a preferred empirical treatment for odontogenic infections due to its efficacy, limited side effects, low cost, patient tolerability, and widespread availability[5-25].

2. MATERIALS AND METHODS

This cross-sectional study was conducted at NIMS Dental College and Hospital and the National Institute of Medical Sciences and Research Centre, Jaipur, Rajasthan, between June 2022 and April 2025. The study population comprised patients presenting to the Outpatient Department diagnosed with maxillofacial space infections. Ethical approval was obtained, and informed consent was acquired from all participants. A sample size of 48 patients was determined using power analysis with t-tests, ensuring adequate statistical power for comparisons. Patients were categorized into two groups: diabetic and non-diabetic.

Exclusion criteria included ASA IV or V classifications, infections not requiring incision and drainage, prior antibiotic use before presentation, and inability to provide informed consent[6].

Data collection employed a customized case history proforma. The following parameters were recorded for both groups:

- Demographic Data: Age, gender.
- Infection Characteristics: Involved maxillofacial spaces (buccal, mental, canine, submandibular, masticator), etiological factors (odontogenic, trauma, other), and the presence of any comorbidities.
- Clinical Parameters: Febrile status (febrile/non-febrile) assessed through body temperature measurement. Complete blood picture analysis, including white blood cell count, differential count, hemoglobin, and platelet count. Random blood sugar levels and HbA1c levels for assessing glycemic control in diabetic patients.
- Microbiological Analysis: Identification of causative organisms through culture and sensitivity testing from aspirates or swabs of the infected spaces. This involved standard microbiological techniques for isolation, identification, and antibiotic susceptibility testing.

- Treatment and Outcomes: Antibiotic regimens prescribed, any necessary changes to antibiotics based on sensitivity results, duration of hospital stay, need for re-exploration (repeat surgical intervention), and mortality rate[7-22].

Data analysis included descriptive statistics for demographic and infection characteristics. Comparative analyses were performed between the diabetic and non-diabetic groups using appropriate statistical tests (t-tests, chi-square tests) to assess differences in infection spread, clinical parameters, microbial profiles, treatment outcomes, and the influence of comorbidities. Statistical significance was set at $p < 0.05$.

Surgical procedure

Surgical management of maxillofacial space infections aims to achieve source control (e.g., removing the infected tooth) and adequate drainage of the purulent collection.

1. Pre-operative Evaluation and Preparation[8]:

- Medical History and Physical Exam: Assess the patient's overall health, including comorbidities like diabetes , which can impact healing. Evaluate the extent of the infection, including trismus, dysphagia, airway compromise, and associated lymphadenopathy(fig.– 1a, 2a, 2b).
- Imaging: CT scans are crucial for defining the extent of the infection and its relationship to vital structures.
- Airway Assessment: Evaluate the airway and secure it if necessary (e.g., via intubation) before the procedure.
- Antibiotics: Broad-spectrum antibiotics should be started empirically and then adjusted based on culture and sensitivity results.

2. Intra-operative Procedure[9]:

- Anesthesia: General anesthesia is typically preferred.
- Incision and Drainage: (fig.– 1b).
    - The incision should be placed in a cosmetically acceptable location, provide dependent drainage, and avoid vital structures (fig.– 1b,2c).
    - Blunt dissection is used to reach the infected space, minimizing trauma to surrounding tissues(fig.– 2e).
    - The purulent collection is evacuated(fig.– 2d).
    - Loculations within the abscess cavity are broken down to ensure thorough drainage.
- Source Control: If the infection originates from a tooth, extraction or other appropriate dental treatment is performed.
- Irrigation and Drainage: The wound is copiously irrigated with saline or an antiseptic solution (fig.-2).
- Drains: Placement of drains (e.g., corrugated rubber drains) facilitates continued drainage and prevents premature closure of the wound. The drains are typically removed when the drainage subsides.

3. Post-operative Care[10-16]:

- Antibiotics: Continue antibiotics post-operatively until clinical improvement is evident and inflammatory markers normalize.
- Wound Care: Regular wound checks and dressing changes are essential. Warm compresses may promote drainage (fig.– 1c,2f) .
- Pain Management: Analgesics are prescribed for pain control.
- Nutritional Support: Ensure adequate hydration and nutrition, especially in patients with difficulty swallowing.
- Follow-up: Monitor the patient for signs of complications, such as persistent swelling, fever, or spreading infection.

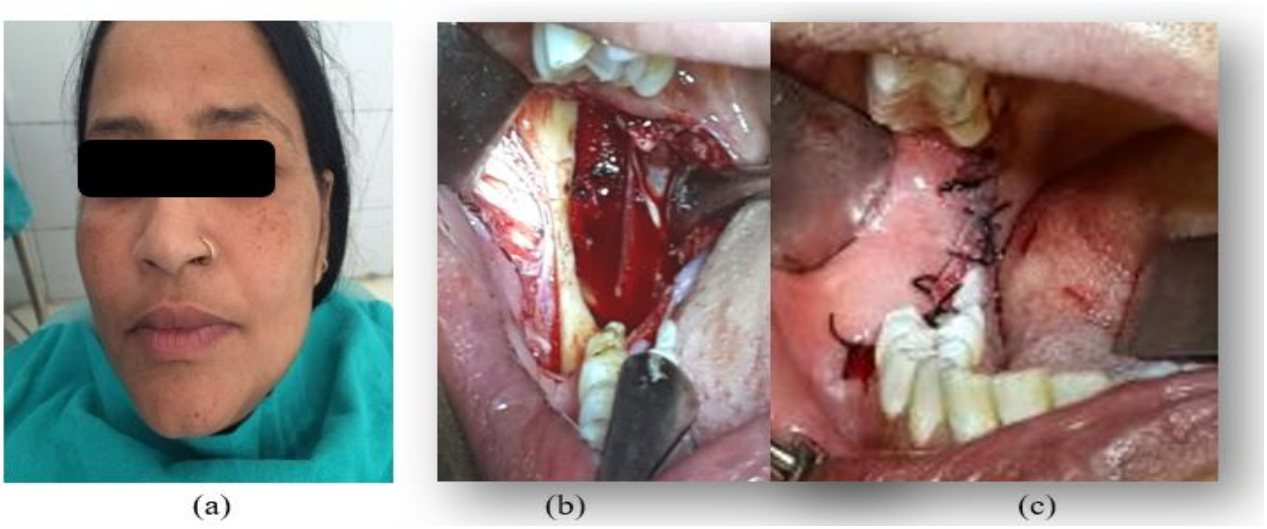
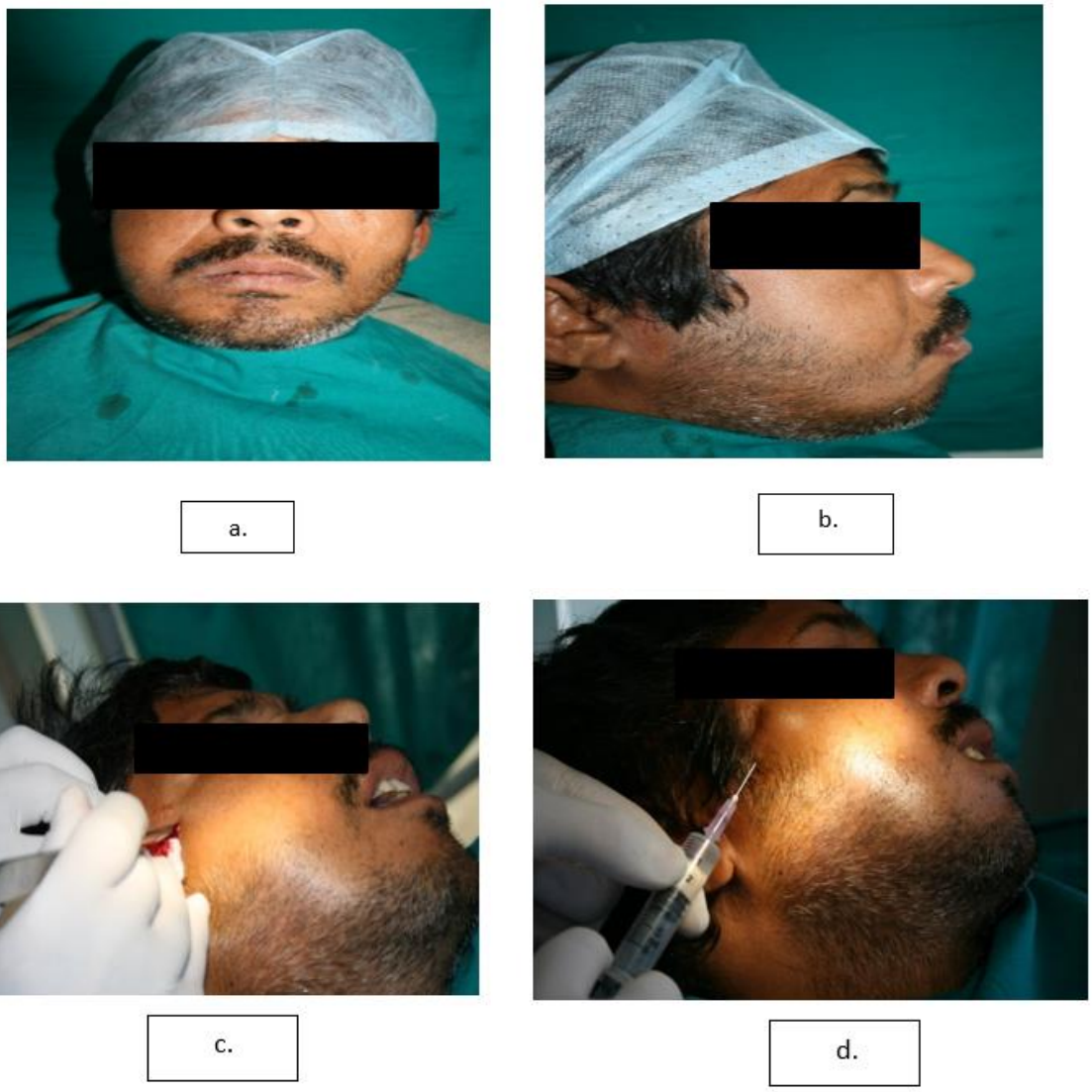
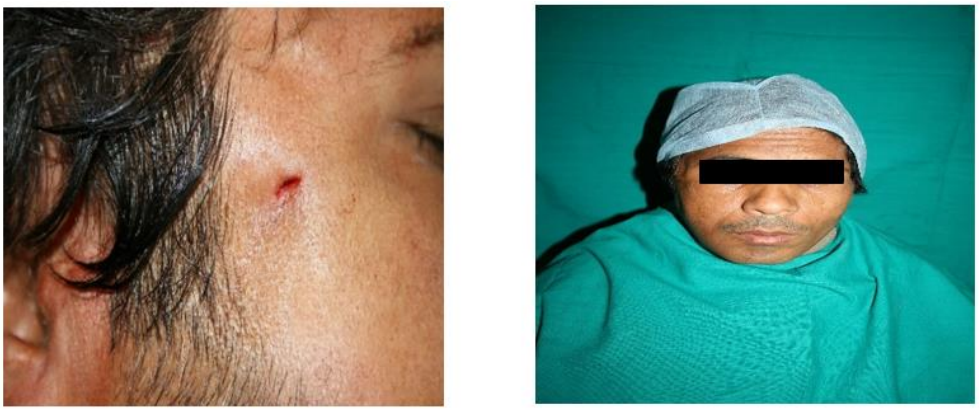


Fig 1.0: Case of pytergomasseteric space infection.





e. f.

Fig 2.0: Case of multiple space infection.

Observation and Results

Demographic and Baseline Characteristics:

Out of 48 participants, 24 were diabetic and 24 were non-diabetic. The mean age was 45.46 years, with an equal distribution of males and females in both groups (p=1.00). There was no significant difference in age between the two groups (p=0.717). (B & D, n.d.) (table-1) (GRAPH- 1,2)

Infection Source and Location:

The majority of infections were odontogenic (79.2%), followed by trauma (14.6%), and other causes (6.2%). The buccal space was the most frequently involved (35.4%), followed by the mental (25%), canine (18.8%), submandibular (14.6%), and masticator (6.2%) spaces. (table-2,9) (GRAPH- 3,4)

Microbiology: (table-7) (GRAPH- 5)

- Streptococcus (41.7%)
- Staphylococcus (35.4%)
- Klebsiella (25%)
- Enterococcus faecalis (10.4%)
- Pseudomonas (4.2%)
- Fusobacterium nucleatum (2.1%)
- Escherichia coli (4.2%)
- Acinetobacter (2.1%)

Comorbidities: Comorbidities were present in 35.4% of the total patient population. (B & D, n.d.) Further details regarding the specific types of comorbidities were not provided in the initial information. (table-3,8) (GRAPH- 6,7)

Glycemic Control: Mean HbA1c was significantly higher in the diabetic group (8.15%) compared to the non-diabetic group (5.32%). Random blood sugar levels were also higher in the diabetic group. Specific values for random blood sugar were not provided in the initial data. Larger studies are warranted to validate these findings and investigate specific comorbidity types. Optimizing glycemic control in diabetic individuals is essential for mitigating infection risks. (table-4,5,6) (GRAPH- 8,9,10)

TABLE 1: DEMOGRAPHIC DATA

	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	Total (n=48)	P value
Age (in years) (Mean±SD)	44.63±15.66	46.29±16.03	45.46±15.70	0.717 NS

Gender - Male Female	 12 (50%) 12 (50%)	 12 (50%) 12 (50%)	 24 (50%) 24 (50%)	 1.00 NS
Source of infection Odontogenic Trauma Others	 14 (58.3%) 6 (25%) 4 (16.7%)	 14 (58.3%) 9 (37.5%) 1 (4.2%)	 28 (58.3%) 15 (31.2%) 5 (10.5%)	 0.301 NS

TABLE 2: INVOLVEMENT OF MAXILLOFACIAL SPACE IN BOTH THE GROUPS

Maxillofacial space involved	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	Total (n=48)	P value
Buccal	6 (25%)	8 (33.3%)	14 (29.2%)	0.972 NS
Mental	6 (25%)	5 (20.8%)	11 (22.9%)	
Canine	4 (16.7%)	3 (12.5%)	7 (14.6%)	
Submandibular	5 (20.8%)	5 (20.8%)	10 (20.8%)	
Masticator	3 (12.5%)	3 (12.5%)	6 (12.5%)	

TABLE 3: NEED FOR REEXPLORATION IN BOTH THE GROUPS

Reexploration	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	P value
Required	22 (91.7%)	11 (45.8%)	<0.001*
Not Required	2 (8.3%)	13 (54.2%)	

TABLE 4 : LABORATORY PARAMETERS

	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	Total (n=48)	P value
HbA1c (%) (Mean±SD)	6.63±0.25	5.07±0.45	5.85±0.87	<0.001*
RBS (mg/dL) (Mean±SD)	238.63±47.57	89.71±5.63	164.17±82.37	<0.001*

TABLE 5: ANTIBIOTIC CHANGES

Antibiotic changes	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	P value
Required	22 (91.7%)	11 (45.8%)	<0.001*

Not Required	2 (8.3%)	13 (54.2%)	

TABLE 6: HOSPITAL STAY AND MORTAITY RATE

	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	Total (n=48)	P value
Hospital stay (No of days) (Mean±SD)	9.25±1.42	7.33±1.24	8.29±1.64	<0.001*
Mortality rates	0	0	0	-

TABLE 7 : PATHOGEN CONFIRMED

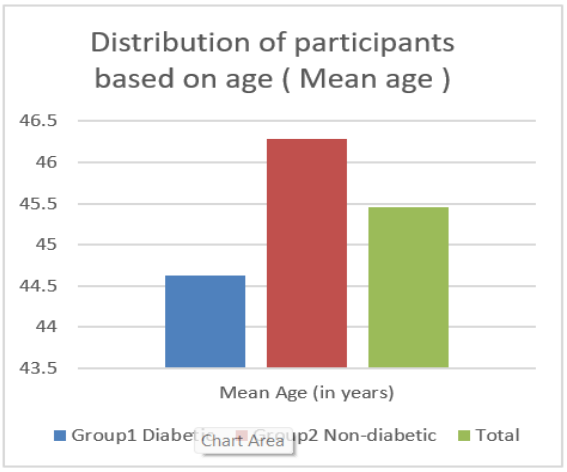
Pathogen confirmed	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	Total (n=48)	P value
Streptococcus	5 (20.8%)	7 (29.2%)	12 (25%)	0.707 NS
Staphylococcus	7 (29.2%)	6 (25%)	13 (27.1%)	
Klebsiella	6 (25%)	6 (25%)	12 (25%)	
Enterococcus fecalis	2 (8.3%)	3 (12.5%)	5 (10.4%)	
Pseudomonas	1 (4.2%)	1 (4.2%)	2 (4.2%)	
Fusobacterium nucleatum	0 (0%)	1 (4.2%)	1 (2.1%)	
Escherichia coli	2 (8.3%)	0 ()%)	2 (4.2%)	
Acinetobacter	1 (4.2%)	0 (0%)	1 (2.1%)	

TABLE 8: COMORBIDITIES ASSOCIATED

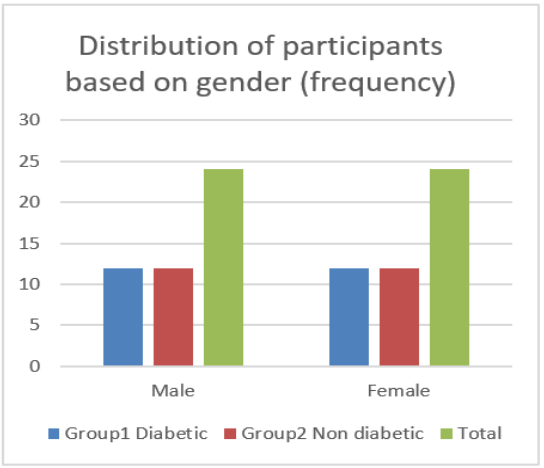
Comorbidities	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	P value
Present	7 (29.2%)	7 (29.2%)	1.00 NS
Absent	17 (70.8%)	17 (70.8%)	

TABLE 9 : SPREAD OF INFECTION

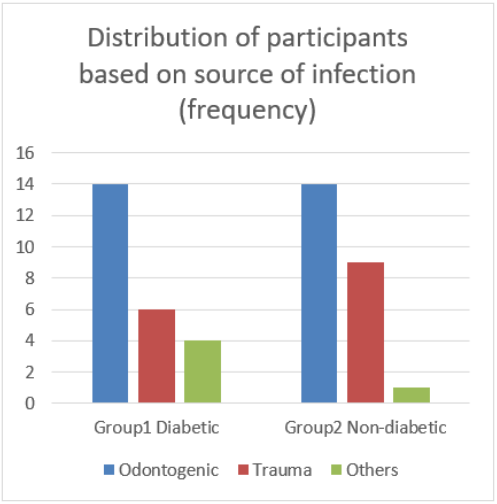
Spread of infection	Group 1 (Diabetic) (n=24)	Group 2 (Non-diabetic) (n=24)	P value
Present	21 (87.5%)	6 (25%)	<0.001*
Absent	3 (12.5%)	18 (75%)	



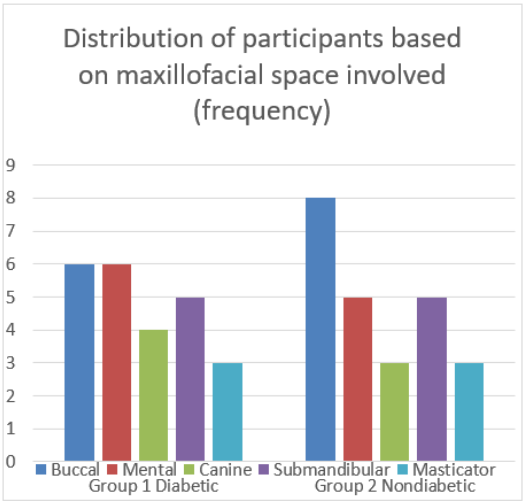
GRAPH- 1



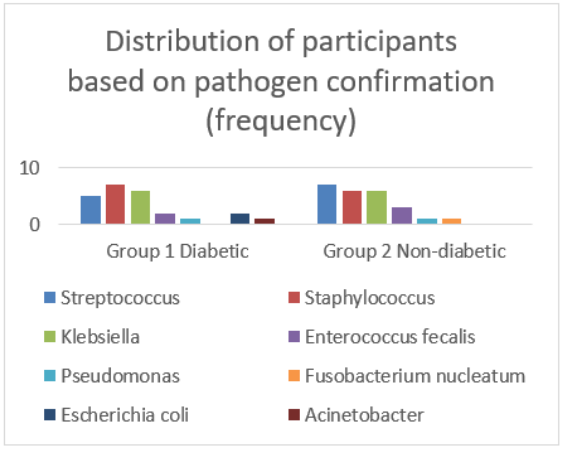
GRAPH- 2



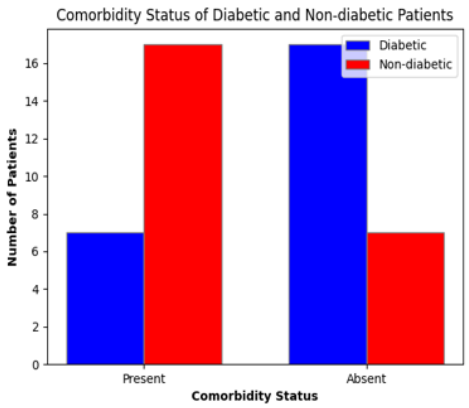
GRAPH- 3



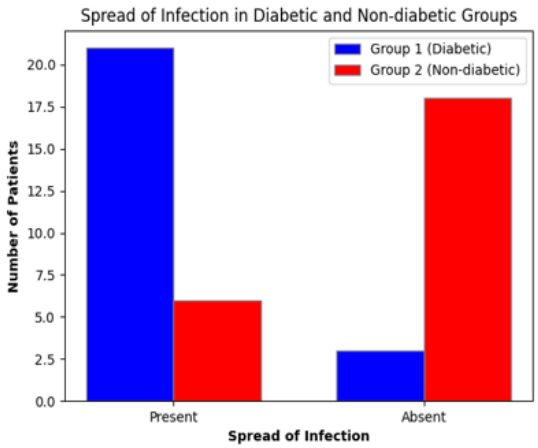
GRAPH- 4



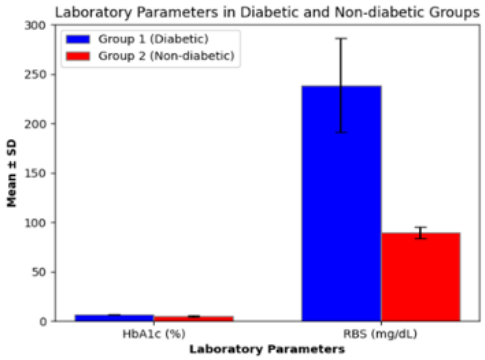
GRAPH- 5



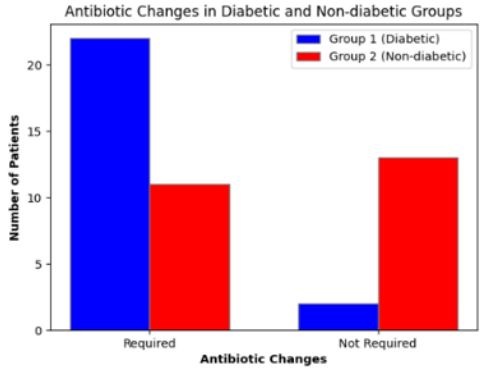
GRAPH- 6



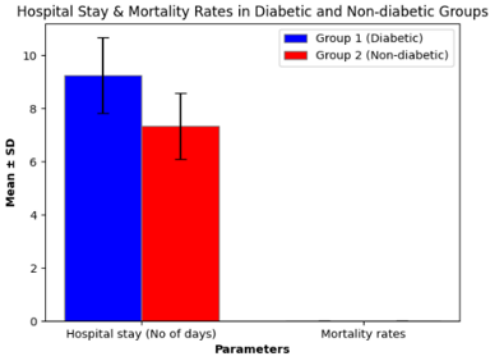
GRAPH- 7



GRAPH- 8



GRAPH- 9



GRAPH- 10

3. DISCUSSION

This study investigated the impact of diabetes on head and neck infections, focusing on infection spread, microbiology, comorbidities, and glycemic control. Our findings demonstrate that diabetes significantly increases the likelihood of infection spread within the maxillofacial spaces. This observation aligns with existing literature, which attributes increased infection severity in diabetics to factors such as impaired immune response, microvascular damage, and altered collagen synthesis. The higher prevalence of multi-space involvement in diabetic patients underscores the importance of early diagnosis and aggressive management in this population[11].

While Streptococcus, Staphylococcus, and Klebsiella species were the most prevalent pathogens in both groups, no statistically significant difference was observed in their distribution. This suggests that while diabetes may not significantly alter the *type* of bacteria causing these infections, it influences the *severity* and *spread* due to systemic factors[12]. The observed microbial profile is consistent with the typical flora found in head and neck infections, reinforcing the importance of broad-spectrum antibiotic coverage initially, followed by targeted therapy based on culture and sensitivity results.

Interestingly, our study did not reveal a significant difference in comorbidity prevalence between diabetic and non-diabetic patients. However, the *types* of comorbidities present, which were not detailed in this study, could still influence infection outcomes. Future research should explore the specific comorbidity profiles within these groups to better understand their impact on infection management.

As expected, diabetic patients exhibited significantly higher HbA1c and random blood sugar levels. (B & D, n.d.) Strict glycemic control is paramount in managing infections in diabetic individuals. Poorly controlled diabetes impairs immune function and wound healing, increasing the risk of severe infections and complications[13]. Our results emphasize the need for optimizing glycemic control both as a preventative measure and as a crucial component of infection management.

This study has limitations, including its relatively small sample size, which may limit the generalizability of the findings. Further research with a larger cohort is warranted to confirm these results and investigate the influence of specific

comorbidity types on infection outcomes.

Diabetes significantly increases the risk and severity of head and neck infections, primarily by promoting the spread of infection rather than altering the causative pathogens. Prompt and aggressive surgical intervention combined with meticulous glycemic control is crucial for successful outcomes in this patient population. Further studies are needed to explore the complex interplay of diabetes, comorbidities, and infection outcomes in the head and neck region[14].

4. CONCLUSION

Spread of Infection: A significantly higher proportion of diabetic individuals experienced the spread of infection compared to their non-diabetic counterparts ($p < 0.001$). This highlights the vulnerability of diabetics to more extensive infections, likely due to factors like impaired immune function, neuropathy, and vascular disease.

Pathogen Distribution: While *Streptococcus*, *Staphylococcus*, and *Klebsiella* were the most common pathogens in both groups, there was no statistically significant difference in the distribution of specific pathogens between the groups. This suggests that the diabetic state may not significantly alter the types of bacteria causing these infections, although it can influence the severity and progression due to the aforementioned systemic factors[15].

Comorbidities: The prevalence of comorbidities was similar between the diabetic and non-diabetic groups, indicating that in this specific study population, diabetes did not significantly influence the likelihood of having other medical conditions. However, the *types* of comorbidities, not specified in the data, could still differ and potentially influence outcomes.

Glycemic Control: Diabetic individuals exhibited significantly higher HbA1c and random blood sugar levels compared to non-diabetics ($p < 0.001$ for both). This underscores the importance of optimizing glycemic control in managing infections and overall health in diabetic patients[17-21].

Antibiotic Changes: A higher percentage of diabetic patients required antibiotic changes compared to non-diabetics, possibly reflecting the challenges in managing infections in the presence of diabetes-related complications[18].

Clinical Implications: These findings emphasize the need for heightened vigilance in managing infections in diabetic individuals. Early detection, aggressive treatment, and optimization of glycemic control are crucial to prevent complications and improve outcomes. The similar distribution of pathogens suggests that standard antibiotic regimens may be appropriate initially, but the increased risk of spread and need for antibiotic changes necessitates close monitoring and adjustment of treatment as needed.

Limitations: The relatively small sample size of the study may limit the generalizability of the findings. Larger, more comprehensive studies are needed to confirm these observations and explore the complex interplay between diabetes and infection management, particularly in the context of dental implants[19]. Further research should also investigate the specific types of comorbidities present and their impact on outcomes, as well as the factors contributing to the need for antibiotic changes.

Summary

Higher Infection Spread in Diabetics: Diabetic individuals showed a significant higher rate of infection spread compared to non-diabetics (87.5% vs. 25%, $p < 0.001$). This highlights the increased vulnerability of diabetics to more severe and extensive infections.

Similar Pathogen Distribution: While *Streptococcus*, *Staphylococcus*, and *Klebsiella* were the most common pathogens identified in both groups, there was no statistically significant difference in their distribution. This suggests that the type of bacteria causing the infection may not be significantly influenced by diabetic status, although the body's response and the infection's progression differ significantly[20].

Comparable Comorbidity Prevalence: The prevalence of comorbidities was similar in both groups, indicating that in this specific study population, diabetes did not significantly increase the likelihood of having other medical conditions. However, the *specific types* of comorbidities present, which could influence outcomes, were not detailed in the provided data[21].

Poorer Glycemic Control in Diabetics: As expected, diabetic individuals had significantly higher HbA1c and random blood sugar levels compared to non-diabetics ($p < 0.001$ for both)[23]. This reinforces the importance of glycemic control in managing infections and overall health in diabetic patients.

Increased Need for Antibiotic Changes in Diabetics: A larger proportion of diabetic patients required changes in their antibiotic regimen compared to non-diabetics, likely reflecting the challenges in managing infections effectively in the presence of diabetes-related complications. These findings underscore the need for heightened awareness and proactive management of infections in diabetic individuals. Early detection, aggressive treatment strategies, and optimization of glycemic control are crucial to prevent complications and improve outcomes.

Further research with larger cohorts is needed to confirm these observations and explore the complex interplay between diabetes, infection, and treatment response[24]. Additional investigation into the specific types of comorbidities and their

impact, as well as the factors contributing to antibiotic changes, would provide a more comprehensive understanding.

Declaration of patient consent

The authors declare that they have obtained consent from patients. Patients have given their consent for their images and other clinical information to be reported in the journal. Patients understand that their names will not be published and due efforts will be made to conceal their identity but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

[1] Topazian RG, Goldberg MH, Hupp JR. Oral and maxillofacial infections: Elsevier Health Sciences; 2002.

[2] Nagendrababu, V., Pulikkotil, S. J., Suresh, A., Veettil, S. K., Bhatia, S., & Setzer, F. C. (2019). Efficacy of local anaesthetic solutions on the success of inferior alveolar nerve block in patients with irreversible pulpitis: a systematic review and network meta-analysis of randomized clinical trials. *International Endodontic Journal*, *52*(6), 779-789.

[3] Ylijoki S, Suuronen R, Jousimies-Somer H. Meurman JH, Lindqvist C. Differences between patients with or without the need for intensive care due to severe odontogenic infections. Journal of oral and maxillofacial surgery. 2001;59(8):867-72.

[4] Ren Y-F, Malmstrom HS. Rapid quantitative determination of C-reactive protein at chair side in dental emergency patients. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology.2007;104(1):49-55.

[5] Kumar, A., Pasha, Z., Salam, S., Aruna, V., Kashwani, R., Sachdeva, K., & Bhagat, N. (2024). Innovative use of a double Y-shaped miniplate in complex mandibular fracture: A rare case report. *Community Practitioner*, *21*(7), 1-8.

[6] Shah BR, Hux JE. Quantifying the risk of infectious diseases for people with diabetes. Diabetes care. 2003;26(2):510-3.

[7] Zheng L, Yang C, Kim E, Zhang W, Cai X, Jiang B, et al. The clinical features of severe multi-space infections of the head and neck in patients with diabetes mellitus compared to non-diabetic patients. British Journal of Oral and Maxillofacial Surgery. 2012;50(8):757-61.

[8] Huang T-T, Tseng F-Y, Liu T-C, Hsu C-J, Chen Y-8, Deep neck infection in diabetic patients: comparison of clinical picture and outcomes with a non-diabetic patients. Otolaryngology-Head and Neck Surgery2005;132(6):943-7.

[9] Kamat RD, Dhupar V, Akkara F, Shetye O. A comparative analysis of9.odontogenic maxillofacial infections in diabetic and nondiabetic patients: an institutional study. Journal of the Korean Association of Oral and Maxillofacial Surgeons. 2015;41(4):176.

[10] Hawkey P. Mechanisms of resistance to antibiotics. Intensive care medicine. 2000;26:S9.

[11] Rega AJ, Aziz SR, Ziccardi VB. Microbiology and antibiotic sensitivities of head and neck space infections of odontogenic origin. Journal of oral and maxillofacial surgery. 2006;64(9):1377-80.

[12] Bryan CS, Reynolds KL, Metzger WT. Bacteremia in diabetic patients: comparison of incidence and mortality with nondiabetic patients. Diabetes care. 1985;8(3):244-9.

[13] Krishnan V, Johnson JV, Helfrick JF. Management of maxillofacial infections: a review of 50 cases. Journal of oral and maxillofacial surgery.1993;51(8):868-73.

[14] Stephenson Jr E, Haug RH, Murphy TA. Management of the diabetic oral and maxillofacial surgery patient. Journal of oral and maxillofacial surgery. 1995;53(2):175-82.

[15] Peters ES, Fong B, Wormuth DW, Sonis ST. Risk factors affecting hospital length of stay in patients with odontogenic maxillofacial infections. Journal of Oral and Maxillofacial surgery. 1996;54(12):1386-91.

[16] Chen M-K, Wen Y-S, Chang C-C, Lee H-S, Huang M-T, Hsiao H-C. Deep neck infections in diabetic patients. American journal of otolaryngology2000;21(3):169-73.

[17] McManus LM, Bloodworth RC, Prihoda TJ, Blodgett JL, Pinckard RN.Agonist-dependent failure of neutrophil function in diabetes correlates with extent of hyper-glycemia. Journal of Leukocyte Biology.

2001;70(3):395404.

[18] Storoe W, Haug RH, Lillich TT. The changing face of odontogenic infections. Journal of Oral and Maxillofacial surgery. 2001;59(7):739-48.

[19] Brook I, editor Antibiotic resistance of oral anaerobic bacteria and their effect on the management of upper respiratory tract and head and neck infections. Seminars in respiratory infections; 2002.

[20] Wang J, Ahani A, Pogrel M. A five-year retrospective study of odontogenic maxillofacial infections in a large urban public hospital. International journal of oral and maxillofacial surgery. 2005;34(6):646-9.

[21] Chang C-M, Lu F-H, Guo H-R, Ko W-C. Klebsiella pneumoniae fascial space infections of the head and neck in Taiwan: emphasis on diabetic patients and repetitive infections. Journal of Infection. 2005;50(1):34-40.

[22] Boscolo-Rizzo P, Marchiori C, Montolli F. Vaglia A, Da Mosto M neck infections: a constant challenge. ENT. 2006:68(5):259-65.

[23] Marioni G, Rinaldi R, Staffieri C. Marchese-Ragona R. Saia G, StramaR, et al. Deep neck infection with dental origin: analysis of 85 consecutive cases (2000-2006). Acta otolaryngological. 2008;128(2):201-6.24.

[24] Stoeckle M, Kaech C. Trampuz A. Zimmerli W. The role of diabetes mellitus in patients with bloodstream infections. Swiss medical weekly.2008;138(35):512.

[25] Youssef W, D'Innocenzo R. Mehra P. Antibiotic therapy in the management of severe odontogenic infections: a comparison of two treatment regimens. Oral Surgery, Oral Medicine, Oral Pathology. Oral Radiology and Endodontology, 2008;4(106):505.