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Original Article

Expression of Microbial Isolates, Sensitivity Profile, and Clinical Aspects in Maxillofacial Infections with or without Diabetes Mellitus

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Abstract

Aim: The aim of the study was to assess and compare the clinical presentation, microbial profile, antibiotic susceptibility of maxillofacial infections, and in diabetic and non-diabetic patients.

Materials and Methods: All patients presenting with maxillofacial infection were initially screened through a complete case history including any known medical comorbidities patients with either a known history of diabetes or sustained hyperglycemia were assigned to Group I and other to Group II. Clinical presentation and laboratory profile were monitored for patient in both the groups. Exudates obtained either through sterile close aspiration technique or during incision and drainage were subjected to Gram staining microbial culture and antibiotic sensitivity.

Results: There was a significant difference between the clinical presentation, age, and antibiotic susceptibility in both the groups although there was no significant deference in the microbial expression in both the groups.

Conclusion: Because of the ever-changing nature of microorganisms due to contentious adaptation, reassessment to improve our knowledge, we have performed a prospective study. After the evaluation of the results of the study, we were able to state that; diabetic males are at higher risk of getting infections. Diabetic patients are at a higher risk of bacteremia, the spread of infection is higher in diabetic patients. The response to empirical antibiotic therapy (clindamycin) in both the groups provides satisfactory results. There were no significant differences in organism isolated and antibiotic susceptibility between the groups. *Kleibsiella* (facultative anaerobe) isolated in higher numbers in diabetic population.

Key words: Culture Sensitivity, Maxillofacial Infections, Microbial Isolates

INTRODUCTION

Evidence of maxillofacial infections troubling mankind has been found in the remains of early Egyptians with signs of dental abscess and osteomyelitis.[1] Establishment of any infection is a consequence of disturbed equation between pathogenicity of the microorganism and host defense mechanisms. Factors which govern the spread of infection are, the mode of entry of the organism, production of toxins, virulence, physical and chemical barriers of host resistance, and immune defense mechanisms of the host.[2]

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Infections can be autogenous, caused by body's resident flora that becomes pathogenic due to some reason as in case of odontogenic infection or may be the result of cross infection. Cross infection leading to sepsis may be a responsible factor if unsterile injections and contaminated needles have been used.

Odontogenic infections are the most common type of orofacial infections oral and maxillofacial surgeons come across.[3] It has been suggested that immunocompromised patients are more susceptible to these infections, in whom their course is often unpredictable.[4] Outcome depends on the host defense, anatomic location and abnormality, virulence of micro-organisms, as well as the timing and choice of anti-microbial treatment.[5]

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Diabetes mellitus (DM) is becoming much more prevalent with advancement of civilization and elongation of lifespan. Angiopathy and susceptibility to infection constitute two major problems in DM. Correlations have been shown between mean plasma glucose levels and the frequency of acute bacterial infections.[6] The coexistence of DM may complicate orofacial infections, and treatment in these cases may be difficult. Suppression of neutrophil functions has also been stated to contribute to the tendency for infection in diabetes. In diabetics, impairment of other bactericidal functions may exist, such as those of chemotaxis, phagocytosis, and reactive oxygen generation. Investigations have indicated that suppression of neutrophil function in diabetes allows microbial invasion and multiplication.[7]

Investigation of the pathogenic potential of individual species of micro-organisms is fraught with difficulties[8] as the micro-organisms are indigenous, their profile changes during an individual's life span and varies depending on anatomic location, environmental factors, and host defenses. These dynamics may influence our ability to isolate organisms.[9]

MATERIALS AND METHODS

A prospective study design was employed for comparison of the cohorts. All patients presenting with signs and symptoms suggestive of maxillofacial infection were thoroughly screened with a complete case history and detailed local and systemic examination. Relevant radiographs and hematological investigations were performed. For the cases diagnosed as having maxillofacial infection (odontogenic/non-odontogenic), initial segregation into two groups, that is, diabetics in Group I and non-diabetics in Group II was carried out taking into account a known history of DM. Those patients who did not give a history of DM but had a Random Blood Glucose value >130 mg/dl were also assigned to the diabetic group (Group I). Patients presenting with wound sepsis, history of anti-cancer chemotherapy or any other additional comorbidity such as chronic renal failure, severe anemia, etc., were excluded from the study.

Collection of Pus Samples

Sample collection was done preferentially by closed aspiration using an 18-gauge needle and 10 ml disposable syringe. Site of aspiration was chosen after careful examination and site was cleansed with isopropyl alcohol. Intra-oral site was prepared using 0.2% chlorhexidine mouth rinse. Subsequently sterile dry gauze was used to wipe the area clean. Maximum sample was aspirated in a single attempt to avoid contamination of the aspirate. In cases, where significant aspirate was not available, sterile culture swabs were introduced into the wound after incision and drainage. A few drops of sterile water were used to keep the swabs hydrated during transport.

Immediately on aspiration residual air was evacuated from the syringe and the needle was capped with a serial rubber cork. The sample was transported to laboratory avoiding any delay.

Monitoring of Clinical Progress

Patients were monitored through a process of daily clinical evaluation and periodic change of dressings as required by the quantum of soakage. Patients were examined for reduction in magnitude of swelling and cessation of pus discharge. The period of recovery was recorded by determining the number of days required for complete cessation of discharge and that required for at least 70% reduction in swelling. The latter was assessed subjectively by two independent observers blinded to the cohorts.

Antibiotic administration was continued for 72 h after cessation of discharge in full therapeutic dose.

Assessment of Microbial Profile

Pus samples were processed and smear studies by Gram staining were done and reported. All samples were subjected to aerobic, anaerobic, and fungal cultures. All the culture media used in the study were prepared by reconstituting the commercially available dehydrated media from HiMedia, India. For aerobic culture, samples were inoculated on blood agar, MacConkey's agar, and in Peptone water/Nutrient broth. For anaerobic culture, sample was transferred to Robertson's Cooked Meat medium and after a delay of 24 h, inoculation was done on Vancomycin/Kanamycin blood agar. The third part of the sample was inoculated on Sabouraud Dextrose Agar for fungal growth.

Incubation was done at 37°C for 18–24 h. If there was no growth, new plates were streaked and were re-incubated along with old plates at 37°C for 18–24 h. Plates for anaerobic cultures were incubated in anaerobic jar at 37°C in an anaerobic workstation (Don Whitley, India) using Gaspak.

If any of the plates showed, growth then smear was prepared after describing colony characteristics. Smear was stained by Gram's Method for assessment of morphologic characteristics. Thereafter, for Gram-positive cocci, the Catalase Backtracking sensitivity, Op tchin sensitivity, Coagulase test, and growth in 6.5% sodium chloride were used, whereas, for gram negative bacilli; oxidase test, catalase test, indole test, urease test, citrate test, and

triple sugar iron test were used. In addition, growth on any media was reconfirmed by taking a pure culture. To identify fungal growth, one plate was incubated at 25°C in a cooling incubator (Remi, India) and the other at 37°C. After positive growth, direct microscopy was done to identify the gross characteristics of the fungi.

Before concluding negative growth, anaerobic plates and fungal plates were incubated for to 14 days and 1 month, respectively. For plates incubated under aerobic conditions, negative cultures were concluded if no growth was observable 72 h post incubation.

Antibiotic Susceptibility Testing

Samples from plates exhibiting positive growth were re-inoculated on Muller-Hinton Agar for rapidly growing non-fastidious organisms. For fastidious organisms, the agar was supplemented with 5% sterile, defibrinated blood. Antibiotic sensitivity studies for the microbial isolates were done by the standard Kirby-Bauer Disk Diffusion technique.

Interpretations were carried out based on the diameter of the zone of inhibition as, Sensitive, Moderately Sensitive or Resistant, using the manual provided by HiMedia Pvt Ltd, India. Antibiotic used were Amoxicillin-Clavulanic acid, Ampicillin, Ampicillin-Sulbactam, Cefadroxil, Clarithromycin, Clindamycin, Linezolid, Norfloxacin, Azithromycin, Vancomycin, Amikacin, Cefoperazone-Sulbactam, Cefotaxime, Ceftazidime, Ceftriaxone, Cefuroxime, Ciprofloxacin, Erythromycin, Gentamycin, Imipenem, Levofloxacin, Penicillin G, Metronidazole, Ornidazole, and Ofloxacin.

Statistical Analysis

The clinical and laboratory data so obtained were analyzed statistically to meet the objectives of the study using the SPSS V.19 (IBM, Chicago, USA). Chi-squire (χ^2) test was applied for the comparison between the groups. The statistical significance was kept at $P \leq 0.05$.

RESULTS

A total of 56 patients with maxillofacial infection fulfilling the inclusion and exclusion criteria were enrolled in the study, 26 patients in Group I (diabetic group), and 30 patients in Group II (non-diabetic group).

Age and Gender

The age of the patients ranged from 15 to 75 years with a mean age of 51.84 years in Group I and 31.16 years in Group II. A majority of the patients (38.5%) within Group I were within the age group of 56–65 years whereas 43% of the Group II patients were in the age range of 15–25 years. Those in Group I were thus significantly older, and this difference was found to be statistically significant [Table 1 and Graph 1].

Table 2 shows the gender distribution of the subjects, and it was observed that a statistically significant difference existed with respect to this parameter between the two cohorts. A reversal of sex ratio was observed when the gender characteristics of the two cohorts was analyzed [Graph 2], with males dominating in Group I and females in Group II.

Table 1: Age distribution of the patient

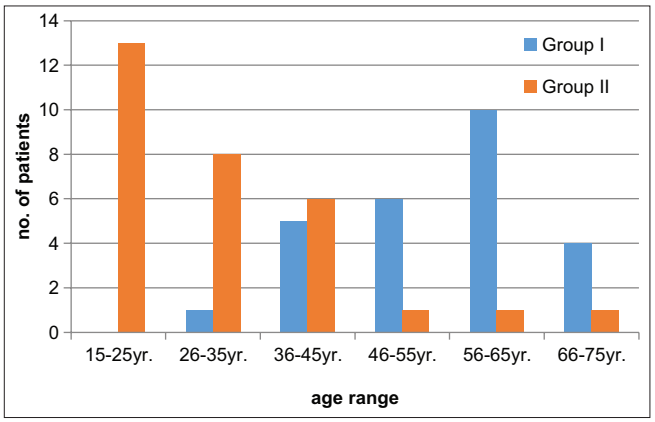
Age	Group I (%)	Group II (%)	*P*-value
15–25 years	0 (0)	13 (43)	0.000 (S)
26–35 years	1 (3.8)	8 (26.7)	
36–45 years	5 (19.2)	6 (20)	
46–55 years	6 (23.1)	1 (3.3)	
56–65 years	10 (38.5)	1 (3.3)	
66–75 years	4 (15.4)	1 (3.3)	

S: Significant, NS: Non significant

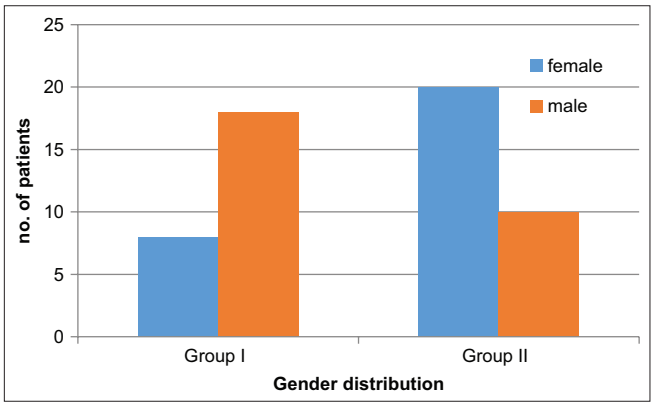
Table 2: Gender distribution of patient

Gender	Group I (%)	Group II (%)	*P*-value
Female	8 (30.8)	20 (66.7)	0.007 (S)
Male	18 (69.2)	10 (33.3)	

S: Significant, NS: Non significant



Graph 1: Age distribution of the patients



Graph 2: Gender distribution of patients

Presentation

The extensiveness of infection in the two groups was evaluated through assessment of the number of facial compartments involved (characterized dichotomously as single or multiple), fever and total white blood cell (WBC) counts.

Number of Spaces Involved

Majority of patients in the diabetic cohort (57.7%) had multiple space involvement as opposed to the non-diabetic group where 60% of the patient had a single space involvement. A statistically significant difference was thus observed with respect to this parameter [Table 3 and Graph 3].

Fever

Significantly higher proportion of patients in the non-diabetic group is presented in an afebrile state as opposed to the diabetic group. The majority of patient in diabetic group were noted to have mild fever on presentation [Table 4 and Graph 4].

Total WBC Count

The leukocyte response was considered under four heads; normal, mild, moderate, and high. The reference range for the same is indicated in Table 5. Analysis of this parameter showed that the counts were significantly raised in the diabetic cohort as compared to non-diabetic patients as in the latter group significantly higher no. About 73.3% of patients had normal counts. These differences were noted to be statistically significant [Table 5 and Graph 5].

Table 3: Number of space involved

No. of space	Group I (%)	Group II (%)	*P*-value
No space	4 (15.4)	7 (23.3)	0.005 (S)
Single	7 (26.9)	18 (60.0)	
Multiple	15 (57.7)	5 (16.7)	

S: Significant, NS: Non significant

Table 4: Fever

Fever	Group I (%)	Group II (%)	*P*-value
No fever	7 (26.9)	19 (63.3)	0.013 (S)
Mild 99–100°F	14 (53.8)	10 (33.3)	
Moderate 101–102°F	5 (19.2)	1 (3.3)	
High >102°	0	0	

S: Significant, NS: Non significant

Table 5: Total leukocyte counts

TLC (cells/mm³)	Group I (%)	Group II (%)	*P*-value
Normal (4500–10000)	5 (19.2)	22 (73.3)	0.000 (S)
Mild (>10000–15000)	16 (61.5)	6 (20.0)	
Moderate(>15000–20000)	5 (19.2)	2 (6.7)	
High (>20000)	0	0	

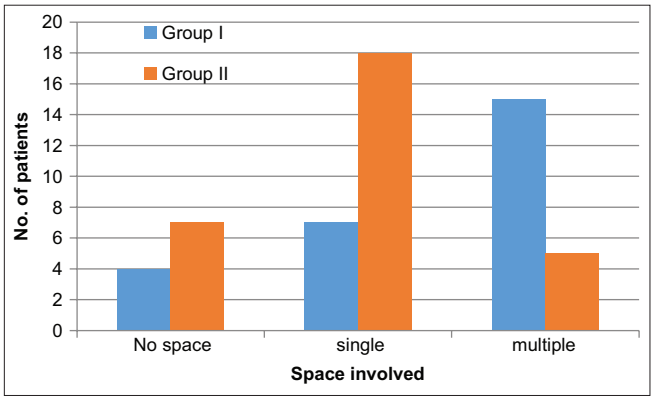
S: Significant, NS: Non significant, TLC: Total leukocyte counts

Comparison of Isolates

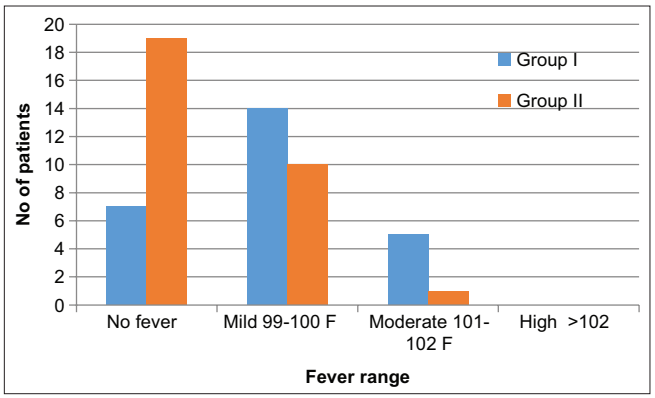
Microbial profile of the infections was determined through Gram staining characteristics, and isolation in culture.

Gram's Stain

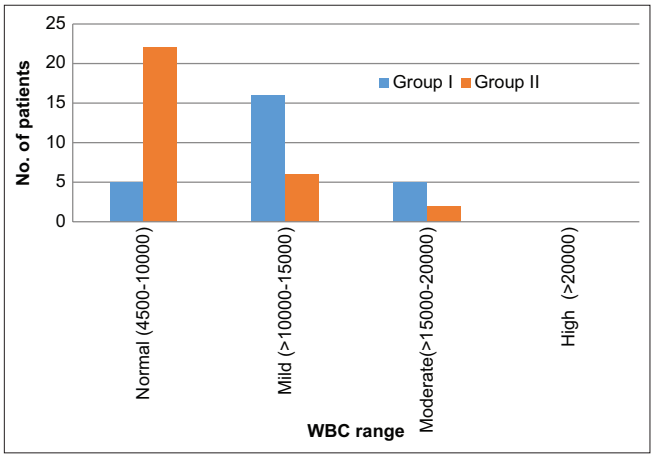
The organisms identified on the basis of staining characteristics were categorized as purely gram positive, mixed population of Gram positive and Gram negative and purely Gram negative. No organisms were identifiable in one patient belonging to diabetic cohort. Comparison of



Graph 3: Number of space involved



Graph 4: Fever



Graph 5: White blood cell count at presentation

the two groups on the basis of Gram staining characteristics showed no statistically significant differences. Majority of infections in both groups (69.2% and 83.3% in Groups I and II, respectively) were caused by purely Gram-positive organisms [Table 6 and Graph 6].

Bacterial Isolates

35 isolates in each group accounting for 11 different organisms were identified from 51 patients (24 in Group I and 27 in Group II). 2/26 and 3/30 patients in Groups I and II, respectively, had sterile cultures. Overall, *Staphylococcus aureus* was the most frequently isolated organism collectively as well as independently in both cohorts. Comparison of microbial isolates between the two groups did not how ever reveal any significant statistical differences [Table 7 and Graph 7].

Antibiotic Susceptibility Profile

In vitro susceptibility to different antibiotics was assessed and compared between the groups for the purpose of evaluation; moderate sensitivity was also regarded as susceptibility. Susceptibility pattern to 26 antibiotics tested for each isolate was compared between the two groups. Analysis for *in vitro* susceptibility showed statistically significant differences between the two groups for ceftazidime, erythromycin, and levofloxacin. The resistance to levofloxacin was significantly higher in diabetic cohort whereas resistance to erythromycin and ceftazidime was found to be significantly higher in the non-diabetic cohort. Overall, excellent responses were evident for clindamycin, linezolid, and metronidazole in both the groups [Table 8 and Graph 8].

Table 6: Gram stain

Gram stain	Group I (%)	Group II (%)	*P*-value
No stain	1 (3.8)	0 (0)	0.333 (NS)
Gram-positive	18 (69.2)	25 (83.3)	
Mixed	7 (26.9)	5 (16.7)	

S: Significant, NS: Non significant

Table 7: Bacterial isolates

Isolates	Group I (%)	Group II (%)	*P*-value
Staphylococcus aureus	13 (50)	10 (43.5)	0.161 (NS)
Streptococcus mutans	3 (11.5)	8 (26.7)	0.139 (NS)
Pseudomonas aeruginosa	3 (11.5)	2 (6.7)	0.431 (NS)
Klebsiella pneumoniae	5 (19.2)	1 (3.3)	0.068 (NS)
Streptococcus viridans	2 (7.7)	4 (13.3)	0.407 (NS)
Actinomyces	0 (0)	1 (3.3)	0.536 (NS)
Streptococcus salivarius	1 (3.8)	2 (6.7)	0.554 (NS)
Streptococcus sanguinis	4 (15.4)	3 (10)	0.418 (NS)
Staphylococcus epidermidis	2 (8.0)	2 (6.7)	0.622 (NS)
Streptococcus milleri	1 (3.8)	2 (6.7)	0.554 (NS)
β hemolytic streptococcus	1 (3.85)	0 (0)	0.464 (NS)

S: Significant, NS: Non significant

DISCUSSION

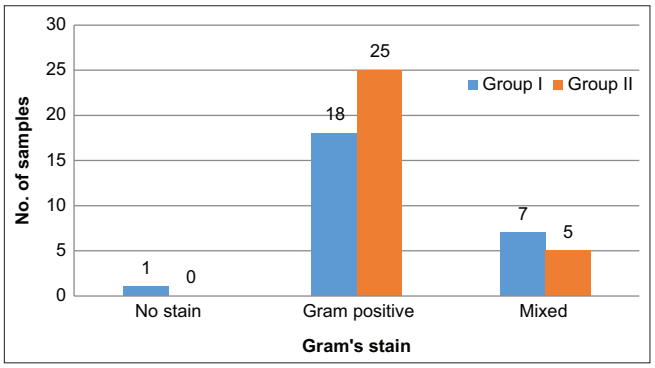
Treatment of maxillofacial infections is a routine practice in oral and maxillofacial surgery. Most of the times the origin is odontogenic.[5] Primary etiology may be carious tooth or a periodontal pocket and sometimes may be a partially erupted tooth.

Infections usually spread into a potential anatomical space present in maxillofacial region following the path of least resistance. Host defense plays the most important role, it may be compromised due to multiple factors.[5] Diabetes has been considered as one of the major factors adversely affecting the host defense.[10] Diabetes represents a major public health problem worldwide. India leads the world in so far as diabetic population is concerned and the same is expected to touch 69.9 million by 2025 (Mohan and Sandeep 2007).[11]

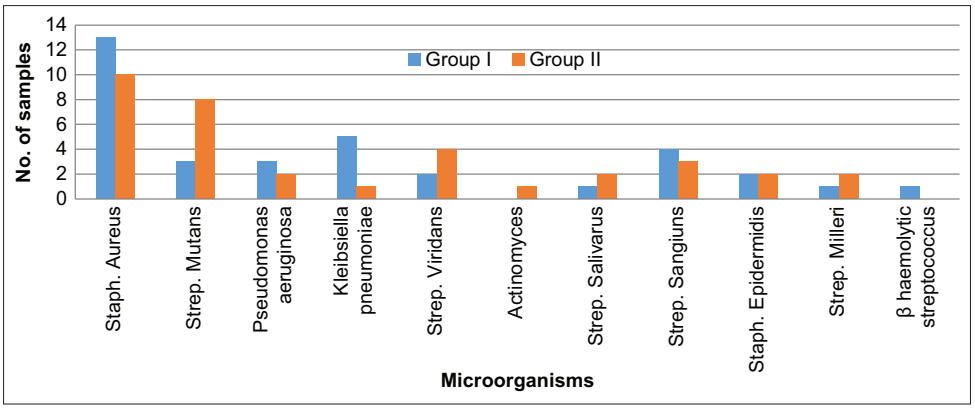
The mechanisms by which diabetes predisposes the infections are stated to be hyperglycemia, disturbed neutrophil function, depressed cellular immunity, abnormalities in compliment activation along with the vascular abnormality, all of which predisposes the patients to a higher risk of infections.[13] In a retrospective cohort study Shah and Hux (2003)[10] concluded that infection should be considered as a complication of diabetes.

It is known today that more than 500 different strains of microorganisms inhabit the oral cavity in a normal healthy adult.[12] Various studies have reported that usually harmless commensals of oral cavity turn pathogenic in certain conditions. Sharma *et al.* (2011)[13] also reported that the increased occurrence of oral infection in diabetic patients can be attributed to the changed environment of oral cavity which enhances the growth of bacteria.

Earlier there was a debate over the microbial aspect of odontogenic infections. In 1970, it was thought that odontogenic infections had a predominance of aerobic and microaerophilic organisms, mainly staphylococci



Graph 6: Gram stain



Graph 7: Bacterial isolates

Table 8: Antibiotic sensitivity pattern

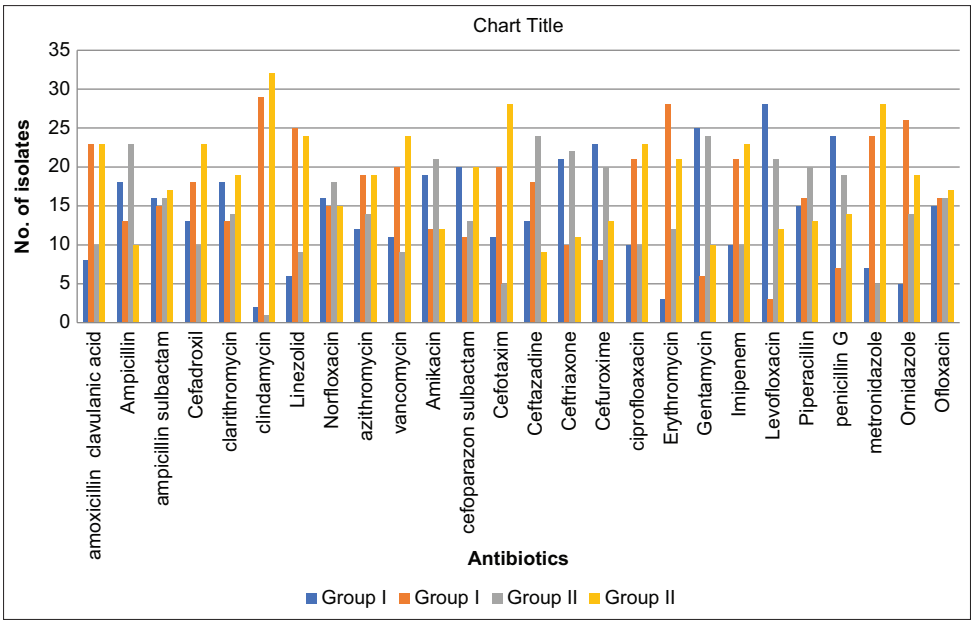
Antibiotic	Group I (%)		Group II (%)		*P*-value
	Resistant	Sensitive	Resistant	Sensitive	
Amoxicillin-clavulanic acid	8 (24.2)	23 (69.7)	10 (27.8)	23 (63.9)	0.864 (NS)
Ampicillin	18 (54.5)	13 (39.4)	23 (63.9)	10 (27.8)	0.585(NS)
ampicillin sulbactam	16 (48.5)	15 (45.5)	16 (44.4)	17 (47.2)	0.907 (NS)
Cefadroxil	13 (39.4)	18 (54.4)	10 (27.8)	23 (63.9)	0.585 (NS)
Clarithromycin	18 (54.5)	13 (39.4)	14 (38.9)	19 (52.8)	0.428 (NS)
Clindamycin	2 (6.1)	29 (87.9)	1 (2.8)	32 (88.9)	0.759 (NS)
Linezolid	6 (18.2)	25 (75.8)	9 (25)	24 (66.7)	0.708 (NS)
Norfloxacin	16 (48.5)	15 (45.5)	18 (50)	15 (41.7)	0.910 (NS)
Azithromycin	12 (36.4)	19 (57.6)	14 (38.9)	19 (52.8)	0.894 (NS)
Vancomycin	11 (33.3)	20 (60.6)	9 (25)	24 (66.7)	0.728 (NS)
Amikacin	19 (57.6)	12 (36.4)	21 (58.3)	12 (33.3)	0.919 (NS)
Cefoperazone sulbactam	20 (60.6)	11 (33.3)	13 (36.1)	20 (55.6)	0.124 (NS)
Cefotaxime	11 (33.3)	20 (60.6)	5 (13.9)	28 (77.8)	0.160 (NS)
Ceftazidime	13 (39.4)	18 (54.5)	24 (66.7)	9 (25)	0.042 (S)
Ceftriaxone	21 (63.6)	10 (30.3)	22 (61.1)	11 (30.6)	0.932 (NS)
Cefuroxime	23 (69.7)	8 (24.2)	20 (55.6)	13 (36.1)	0.479 (NS)
Ciprofloxacin	10 (30.3)	21 (63.6)	10 (27.8)	23 (63.9)	0.923 (NS)
Erythromycin	3 (9.1)	28 (84.8)	12 (33.3)	21 (58.3)	0.039 (S)
Gentamycin	25 (78.1)	6 (18.8)	24 (66.7)	10 (27.8)	0.517 (NS)
Imipenem	10 (30.3)	21 (63.6)	10 (27.8)	23 (63.9)	0.923 (NS)
Levofloxacin	28 (84.8)	3 (9.1)	21 (58.3)	12 (33.3)	0.039 (S)
Piperacillin	15 (45.5)	16 (48.5)	20 (55.6)	13 (36.1)	0.578 (NS)
penicillin G	24 (72.7)	7 (21.2)	19 (52.8)	14 (38.9)	0.224 (NS)
Metronidazole	7 (21.2)	24 (72.7)	5 (13.9)	28 (77.8)	0.700 (NS)
Ornidazole	5 (15.2)	26 (78.8)	14 (38.9)	19 (52.8)	0.066 (NS)
Ofloxacin	15 (45.5)	16 (48.5)	16 (44.4)	17 (47.2)	0.936 (NS)

S: Significant, NS: Non significant

and streptococci. With recent advances in the field of diagnostic microbiology and techniques, it is now known that odontogenic infections are poly microbial in origin.[14] Studies[5] comparing the microorganisms in diabetic and non-diabetic patients suffering from infection have also shown poly microbial origin with no significant differences between the groups.

Staphylococci produce coagulase, an enzyme which can cause fibrin deposition, and so are frequently associated with abscess formation. Streptococci produces enzyme such as streptokinase, hyaluronidase and streptodornase, enzymes that break down fibrin and ground substance of connective tissue, and therefor are often associated with so frequently present with cellulitis.[9]

Multiple authors[9,13,15,17,18] have observed and reported a distinct predilection for occurrence of maxillofacial infections among males, the lone exceptions being Mahalle *et al.* (2014)[19] who reported a female predilection and Hunt *et al.* (1989)[20] who reported an equal gender distribution. In the present study, over all there was no distinct gender predilection, an observation akin to that of Hunt *et al.*[20] However, the interesting observation was that of



Graph 8: Antibiotics

a complete reversal of sex ratio between the diabetic and non-diabetic cohorts. This observation is entirely different from that reported in the literature generally.

Sanchez *et al.* (2011)[23] showed that infections are found in almost all ages, his study showing a range of 4–80 years. Patients in our study however had the age ranging from 15 to 75 years. Haug *et al.* (1991)[24] in his study found that most common age range for all odontogenic infection was 25–30 year. Our observations are like those of Haug *et al.* with maximal patients (13 out of 56) belonging to the age group of 15–25 years. In contrast with the general population the diabetic patients presenting with maxillofacial infections are generally much older, findings that have been mentioned by Huang *et al.* (2005),[25] The mean age in the diabetic population affected with maxillofacial infection has been reported to vary between 45 and 60 years. The mean age of diabetic cohorts in present study was 51.8 years and that of non-diabetic cohorts was 31.16 years. These observations concur with those reported in the literature.

Multiple space involvement at the time of presentation was significantly higher in the diabetic group in our study. This agrees with observations forwarded by Huang *et al.* (2005),[25] Rao *et al.* (2010),[5] and Juncar *et al.* (2014).[16] The most frequently involved spaces in the present study were submandibular and buccal spaces in both the cohorts. Other cohort studies[5,29] have also mentioned similar findings. Although Huang *et al.* (2005)[25] reported parapharyngeal space to be the most involved space in diabetics, these studies essentially evaluated deep neck space involvement.

Fever occurs as an acute inflammatory response to bacterial endotoxins and cell wall fragments. About 53.5% patients in the present study were found to be febrile at the time of initial presentation. This observation is in agreement with that of Bridgeman *et al.* (1995),[26] who reported fever in 50% of the patients with maxillofacial infections but differs from the findings described by Mathew *et al.* (2012) [27] and Lee *et al.* (2007)[18] who recorded fever in only 35% and 14.6% patients, respectively.

Rao *et al.* (2000)[5] while comparing the deep neck infections in diabetic and non-diabetic patients, reported pyrexia in 60% of diabetic patients compared to 61.3% of non-diabetic patients. Rao *et al.* (2010),[6] on the other hand, found pyrexia in a significantly higher number of diabetic patients with maxillofacial infections as compared to non-diabetics which are observations like those of the present study. It has been suggested[6] that these differences may be due to a higher penchant for bacteremia in the diabetic patients.

Bacterial infection initiates neutrophil release from the bone marrow and thus induces a neutrophil leukocytosis. Rao *et al.* (2010)[5] in their comparative studies in diabetic and non-diabetic patients found no statistically significant difference in leukocytosis. Chang *et al.* (2013)[28] reported higher WBC count in diabetic patients compared to non-diabetic patients. In our study too, significantly higher proportion of patients (80.7%) in the diabetic group had leukocytosis as compared to non-diabetic patients (26.7%). None of the patients however had leukocytosis exceeding 20,000 cells/mm^3, with the majority showing

mild leukocytosis. It may be supposed that higher tendency for leukocytosis in diabetic patients could be due to the problem of impaired chemotaxis which probably reduces WBC availability at the target site. Increased WBC released into the peripheral circulation may therefore be a compensatory response to improve leukocyte emigration.

Direct smear studies of gram staining were done to identify the gross phenotypic characteristics of organisms in pus sample with the intention that it can help in determining the initial antibiotic to be given before the culture sensitivity reports. None of the studies reviewed have reported direct smear evaluations and comparison. Direct smear examination showed a predominance of Gram-positive cocci in both the groups without any significant statistical difference. This also validates our choice of Clindamycin as the empiric antibiotic.

Lewis *et al.* (1986),[21] Storoe and Haug (2001),[14] Huang *et al.* (2004)[15] and Robertson and Smith (2009)[22] covering diverse populations and performed at different time periods show that maxillofacial infections are polymicrobial in nature with aerobic, anaerobic, and mixed micro flora. Some studies[9,32] show that these infections are predominantly caused by anaerobic organisms. Rega and Ziccardi (2006)[1] stated that maxillofacial infection was caused predominantly by aerobes and facultative anaerobes. The results of our study are in concurrence with those of Rega and Ziccardi (2006)[1] and Rao *et al.* (2010).[5] Many studies[14,21] have shown successful isolation of obligate anaerobes. We were unable to isolate obligate anaerobes in any of the samples, but this had no impact on the outcome of treatment. It is possible that the empiric uses of Clindamycin helped to target obligate anaerobes as well since this antibiotic has proven record of effectiveness against obligate anaerobes. Isolation of anaerobic bacteria remains a technique sensitive and challenging issue.

There was a low occurrence of no growth in our study, there were only 7.2% of the sample which showed no growth which agrees with Sklavounos *et al.* (1986)[29] and Konow *et al.* (1992)[30] who reported only 9.5% and 1.6% negative growths, respectively.

In the present study, most common organism isolated in both the groups was *S. aureus* (23/56 patients) which is similar to several other reports.[6,10,19] In contrast, Kuriyama *et al.* (2005)[22] found Streptococci viridans to be the predominant aerobe involved in such infections. Isolation of *S. aureus* has clinical significance as resistant strains are known to occur which do not respond to routine microbial therapy. Cohort studies comparing maxillofacial infection in diabetics and non-diabetics such as those by Rao *et al.* (2010),[5] Huang *et al.* (2005),[25] reported *Klebsiella* as the predominant organism in diabetic patients with maxillofacial infection. Second to *S. aureus*, *Klebsiella* was the next most dominant organism in the diabetic cohorts.

A total of 25 antibiotics were tested in the present study. Over all excellent responses were evident for clindamycin, linezolid, and metronidazole in both the groups. Chang *et al.* (2005)[31] also reported the high sensitivity for clindamycin, while Boyanova *et al.* (2006)[32] reported clindamycin and metronidazole to be highly efficient against gram negative rods.

There was no statistically significant difference in antibiotic susceptibility and resistance between the two cohorts except for ceftazidime, erythromycin, and levofloxacin. Other published studies in literature have not assessed the cohort differences in the antibiotic susceptibility of the causative organisms. The susceptibility characteristics have been assessed and described only with relevance to organisms isolated. Antimicrobial sensitivity is significantly influenced by the previous exposure to antibiotics and varies from individual to individual.

Lee *et al.* (2007),[18] Chang (2013)[28] reported higher no. of complications in diabetic patients, although we did not encounter complication in any of the two groups. This could be due to the fact that majority of the diabetics did not have uncontrolled diabetes at the time of presentation.

CONCLUSION

There are many authors who have published their studies assessing microbiology, clinical presentation and antibiotic susceptibility of the patient suffering with maxillofacial infections. we have performed a prospective study. After the evaluation of the results of the study we were able to state that; diabetic males are at higher risk of getting infections. Diabetic patients are at a higher risk of bacteraemia, the spread of infection is higher in diabetic patients. The response to empirical antibiotic therapy (clindamycin) in both the groups provide satisfactory results. There were no significant differences in organism isolated and antibiotic susceptibility between the groups. *Kleibsiella* (facultative anaerobe) isolated in higher numbers in diabetic population. Diabetic patients have a longer duration of illness, which can be due to the time taken to control the blood glucose levels.

Management of maxillofacial infections remains the same for both diabetic and non-diabetic population although diabetic patients have to be trated with little more caution and precaution as they are at the higher risk of developing complication.

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