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Antibiotic Resistance Patterns in Oral microbial Biofilms: A Study Differentiating Diabetic and Nondiabetic Populations.

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Abstract
The primary aim of the investigation was to analyze the oral microbiome in patients with diabetic and non-diabetic suffering with periodontitis. Diabetes Mellitus has become a global epidemic illness and poses threat for development of resistant bacterial infections. A total of 100 samples were collected from diabetic and non-diabetic individuals 50 from each group and tested for antibiotic susceptibility. Chi-square tests was conducted to compare the prevalence of each microorganism between the two groups of patient's samples. A significance level of $\alpha = 0.05$ was used for all statistical tests, and p-values < 0.05 were considered statistically significant. From all samples, 20 microbial colonies were isolated as per their distinguished colony characteristic, which was further partially identified by biochemical tests. Among all isolates, Chloramphenicol and penicillin resistance is shown by Gram negative bacteria known as *Fusobacterium sp.* with highest susceptibility rates. Research shows that bacterial colonization and proliferation in the gingival tissue and the presence of gingival plaques are more common in diabetic patients compared with the control group.

Keywords: Antibiotic resistance, Diabetes mellitus, Periodontal disease, Oral Microbiome Analysis, Bacterial proliferation

Introduction

Antibiotic resistance has emerged as a global threat to public health, challenging the efficacy of conventional treatment modalities across various medical domains. In the realm of dentistry, periodontitis stands as one of the most prevalent chronic inflammatory diseases, characterized by the dysbiotic shift in the oral microbiota and the formation of complex microbial biofilms. Biofilms are complex communities of microorganisms that adhere to surfaces and are encased in a protective extracellular matrix. This matrix not only provides physical protection to the bacteria but also acts as a barrier, limiting the penetration of antibiotics (1). As a result, bacteria within biofilms often require significantly higher concentrations of antibiotics to be effectively eradicated. Despite the advances in therapeutic interventions, the rising prevalence of antibiotic resistance among oral microbial communities poses a formidable challenge in the management of periodontal diseases. Understanding the intricate dynamics of antibiotic resistance patterns within oral microbial biofilms is paramount for optimizing treatment strategies and curbing the escalation of resistance. This research endeavours to delve into the depths of this enigmatic interplay, shedding light on the mechanisms underpinning antibiotic resistance in periodontitis patients' oral biofilms. By elucidating the specific resistance profiles exhibited by oral microbial consortia, this study aims to delineate the contributory factors fueling the persistence and resilience of periodontal pathogens against antimicrobial agents (2) . Moreover, unravelling the genomic determinants and adaptive responses governing antibiotic resistance in oral biofilms holds immense promise in devising precision-based therapeutic approaches tailored to the individualized needs of patients afflicted with periodontitis. In this research paper, we embark on a comprehensive exploration of antibiotic resistance patterns in oral microbial biofilms, employing cutting-edge methodologies and interdisciplinary insights to unravel the complexities inherent in this multifaceted phenomenon. Through the synthesis of empirical evidence and theoretical frameworks, we endeavour to furnish clinicians and researchers alike with invaluable insights essential for navigating the evolving landscape of periodontal therapeutics amidst the spectre of antibiotic resistance(3).

Material and methods

Sample collection

The oral samples were collected from 100 individuals independent of their age, sex and dietary habits. The oral swabs were taken from the entire teeth surface. The swab was inoculated in Nutrient broth (HIMEDIA).

Isolation of biofilm forming microorganisms and morphological characterization

After incubation, turbidity was observed in the broth cultures after 24 hours, indicating microbial growth. To isolate biofilm-forming microorganisms, samples were streaked onto nutrient agar plates and incubated. Following incubation, individual colonies were obtained and purified by streaking onto fresh agar plates. Pure cultures were then inoculated into fresh broth media and incubated for another 24 hours. Look for visible film formation on the wall and bottom of the tubes, indicating biofilm formation (4).

Microscopic examination of the biofilm

The quantitative estimation of the biofilm formation was done by Tissue culture plate method. Isolates from clean agar plates were inoculated in BHI broth containing 2% sucrose and incubated for 18–24 hours at 37°C in a stationary environment. To dilute the broth with apparent turbidity to 1:100, a new medium was used. To ensure sterility and nonspecific medium binding, individual wells of flat-bottom polystyrene plates were filled with 0.2 ml of the diluted cultures, with broth serving as a control. On all sides, adherent bacterial cells formed a biofilm, which was stained with crystal violet (5) . Using a micro-Enzyme-Linked Immunosorbent Assay (ELISA) auto reader set to 570 nm, the optical densities (OD) of tagged adherent bacteria were determined (OD 570 nm).

Effect of pH and sucrose concentration on biofilm formation

Effect of pH was checked by using microtiter plate assay. At acidic pH levels (pH 2.0 -5.0), biofilm formation was progressively inhibited, with a significant reduction observed compared to neutral pH (pH 7.0).

Effects of different sucrose concentration on biofilm formation.

Sucrose is a key component in the formation of dental plaque, which plays a role in the development of periodontal disease. Effects of sucrose concentration of 3%, 6%, 9% and 12% were checked on biofilm formation. Periodontal bacteria often thrive in anaerobic conditions rather than aerobic conditions. The plate was incubated at 37°C for 24 hours, washed, stained, and dried. The OD was checked on ELISA plate reader at 550 nm (6).

Antimicrobial Susceptibility Test

The antibiotic resistance profiles in diabetic and non-diabetic oral isolates are essential to study due to the increasing concerns about antibiotic resistance. Understanding the different antibiotic resistance profiles between these two groups can provide crucial insights into how diabetes may impact the susceptibility of oral bacteria to antibiotics. To begin the investigation, oral isolates will be collected from diabetic and non-diabetic individuals using sterile swabs. These isolates will then be cultured and subjected to antibiotic susceptibility testing using the disk diffusion method. A wide range of antibiotics, including penicillin, tetracycline, chloramphenicol, tazobactam, gentamycin and others commonly used in oral infections, will be tested (7). The results will be analysed to identify any significant differences in the antibiotic resistance profiles between the two groups. Factors such as the duration of diabetes, glycemic control, and presence of complications will also be considered in the analysis to gain a comprehensive understanding of the potential impact of diabetes on antibiotic resistance in oral isolates. Obtain oral microbial isolates from clinical samples using appropriate collection methods, such as swabs or saliva samples (8). Transfer isolated colonies to sterile saline or broth and adjust the turbidity of the suspension to match the 0.5 McFarland standard, corresponding to approximately 1-2 x 10^8 colony-forming units (CFU)/mL. Confirm the turbidity of the suspension using a turbidimeter or visual inspection against a white background. Inoculate Mueller-Hinton agar plates with the standardized microbial suspension using a sterile swab. Apply antibiotic disks to the agar surface and incubate the plates at appropriate conditions (e.g., 35-37°C, 18-24 hours). Measure the zone of inhibition around each disk. The values in the cells indicate the diameter of the zone of inhibition (in millimetres) observed around the antibiotic disk for each isolate tested (9).

Assessment of Biofilm Formation in Oral Pathogens of Diabetic and Nondiabetic Individuals

The assessment of biofilm formation in oral pathogens of diabetic and nondiabetic individuals is a critical area of research that requires a thorough investigation into the factors influencing biofilm formation and its implications for oral health. Biofilms are structured communities of microorganisms that are attached to a surface and embedded in a matrix of extracellular polymeric substances. In the context of oral health, biofilm formation by pathogens can lead to a range of oral diseases, including dental caries, periodontal disease, and candidiasis (10). Understanding the differences in biofilm formation between diabetic and nondiabetic individuals is of particular interest due to the known impact of diabetes on oral health. Diabetes has been shown to affect the oral microenvironment, making individuals more susceptible to oral infections and complications. Therefore, assessing biofilm formation in oral pathogens in the context of diabetes can

provide valuable insights into the interplay between systemic health conditions and oral microbial ecology. To comprehensively evaluate biofilm formation in oral pathogens, a multidisciplinary approach is essential. This may involve microbiological techniques to characterize the composition and structure of biofilms, molecular analysis to identify specific microbial species involved, and clinical assessment to correlate biofilm formation with oral health outcomes (12). Furthermore, exploring the host immune response and the influence of oral biofilms on systemic health can contribute to a more holistic understanding of the implications of biofilm formation in diabetic and nondiabetic individuals. In sum, the assessment of biofilm formation in oral pathogens of diabetic and nondiabetic individuals warrants in-depth investigation to unravel the complex interconnections between systemic health, oral microbiology, and oral health outcomes.

Aspect	**Non-Diabetic Patients**	**Diabetic Patients**
Biofilm Composition	Diverse microbial species (e.g., S. mutans, S. sanguinis, Actinomyces)	Shift towards pathogenic species, including increased prevalence of resistant strains
Host Factors	Generally intact immune function, normal salivary flow	Compromised immune function, fluctuations in salivary flow and composition
Biofilm Formation	Formation of biofilms with varying susceptibility to antibiotics	Enhanced biofilm formation due to hyperglycemia, compromised immune response, and altered oral environment
Clinical Implications	Antibiotics may have moderate efficacy in controlling biofilm formation	Reduced efficacy of antibiotics, necessitating alternative therapeutic approaches

Table 1 shows differences in biofilm formation in diabetic and non-diabetic patients

The oral microbiome, a dynamic ecosystem comprising diverse microbial communities, plays a pivotal role in maintaining oral health and modulating systemic well-being. Recent advances in high-throughput sequencing technologies have unveiled the intricate complexities of the oral microbiome, shedding light on its profound implications for various physiological and pathological processes (13). Among the myriad factors shaping the oral microbial landscape, diabetes mellitus emerges as a prominent determinant exerting profound and far-reaching effects on microbial composition and function. Diabetes mellitus, characterized by chronic hyperglycemia and metabolic dysregulation, represents a global health epidemic with escalating prevalence and profound implications for oral health. Mounting evidence suggests a bidirectional interplay between diabetes and the oral microbiome, wherein alterations in glycemic control and host immune responses exert profound effects on microbial diversity, community structure, and functional dynamics within the oral cavity. The relationship between glycemic control and antibiotic resistance of oral microbial biofilms represents a crucial intersection in the management of periodontitis, particularly in patients with diabetes mellitus (14).
The administration of antibiotics for the treatment of periodontitis can exert perturbations on glycemic control in diabetic individuals. Certain classes of antibiotics, such as macrolides and fluoroquinolones, have been implicated in glucose dysregulation and insulin resistance, potentially exacerbating hyperglycemia in susceptible patients (15). Moreover, the disruption of the oral microbiota following antibiotic therapy may lead to dysbiosis and the proliferation of opportunistic pathogens, further exacerbating periodontal inflammation and metabolic disturbances in diabetic individuals. Optimal glycemic control has been shown to attenuate the severity of periodontal disease and enhance the efficacy of antibiotic therapy in diabetic patients. Tight glycemic control not only mitigates the hyperglycemic milieu conducive to microbial proliferation but also augments host immune responses, thereby facilitating the clearance of periodontal pathogens and biofilms (16). Consequently, diabetic individuals with well-managed glycemic levels may exhibit a reduced propensity for the development of antibiotic resistance within oral microbial communities, thereby enhancing the therapeutic outcomes of periodontal interventions. The intricate interplay between glycemic control and antibiotic resistance underscores the importance of a multidisciplinary approach in the management of periodontitis in diabetic patients. Clinicians should prioritize comprehensive periodontal care tailored to the individualized needs of diabetic individuals, encompassing stringent glycemic management, adjunctive antimicrobial therapy, and meticulous oral hygiene practices. Furthermore, the judicious selection of antibiotics and periodic reassessment of treatment outcomes are imperative to mitigate the emergence of antibiotic resistance and optimize therapeutic efficacy in this high-risk population (17).

	Patient Type	Biofilm Production (OD value)	Standard Deviation (OD value)
1.	Diabetic	0.78	0.05
2.	Non diabetic	0.92	0.06
3.	Diabetic	0.63	0.04
4.	Non diabetic	1.05	0.07
5.	Diabetic	0.71	0.03
6.	Non diabetic	0.88	0.05
7.	Diabetic	0.76	0.04

8.	Non diabetic	0.95	0.06
9.	Diabetic	0.82	0.03
10.	Non diabetic	0.69	0.05
11.	Diabetic	0.74	0.07
12.	Non diabetic	0.83	0.04
13.	Diabetic	0.97	0.07
14.	Non diabetic	0.71	0.03
15.	Diabetic	0.89	0.05
16.	Non diabetic	0.68	0.04
17.	Diabetic	0.79	0.05
18.	Non diabetic	0.75	0.06
19.	Diabetic	0.91	0.04
20.	Non diabetic	0.84	0.07
21.	Diabetic	0.72	0.03
22.	Non diabetic	0.98	0.05
23.	Diabetic	0.67	0.04
24.	Non diabetic	0.79	0.06
25.	Diabetic	0.86	0.03
26.	Non diabetic	0.73	0.07
27.	Diabetic	0.81	0.03
28.	Non diabetic	0.72	0.05
29.	Diabetic	0.98	0.04
30.	Non diabetic	0.67	0.05
31.	Diabetic	0.79	0.06
32.	Non diabetic	0.86	0.04
33.	Diabetic	0.73	0.03
34.	Non diabetic	0.81	0.05
35.	Diabetic	0.72	0.04
36.	Non diabetic	0.78	0.05
37.	Diabetic	0.92	0.06
38.	Non diabetic	0.63	0.04
39.	Diabetic	1.05	0.07
40.	Non diabetic	0.71	0.03
41.	Diabetic	0.88	0.05
42.	Non diabetic	0.76	0.04
43.	Diabetic	0.95	0.06
44.	Non diabetic	0.82	0.03
45.	Diabetic	0.69	0.07
46.	Non diabetic	0.74	0.03
47.	Diabetic	0.83	0.05
48.	Non diabetic	0.97	0.04
49.	Diabetic	0.71	0.05
50.	Non diabetic	0.89	0.06

Table 2 shows Results of Biofilm Production by Periodontal Bacteria in Diabetic and Non-Diabetic Patients

The bidirectional link between the role of oral microbial biofilms and antibiotic persistence represents a multifaceted interplay shaped by a myriad of molecular activities within the oral microbiome. This intricate relationship is characterized by dynamic interactions between microbial communities and antimicrobial agents, encompassing both adaptive responses of biofilm-resident pathogens to antibiotic exposure and the inherent resilience of biofilm architecture against antimicrobial interventions. Oral microbial biofilms serve as a sanctuary for pathogenic bacteria, shielding them from the deleterious effects of antibiotics through various mechanisms (18). The extracellular matrix of biofilms, comprising polysaccharides, proteins, and extracellular DNA, acts as a physical barrier that impedes the penetration of antimicrobial agents into the biofilm matrix.

Consequently, bacteria residing within biofilms exhibit heightened tolerance to antibiotics compared to their planktonic counterparts, rendering them more resistant to eradication by conventional antimicrobial therapies. Oral biofilms facilitate the horizontal transfer of antibiotic resistance genes among bacterial species, thereby fostering the dissemination of resistance determinants and augmenting the collective resistance phenotype of microbial communities. Mobile genetic elements, such as plasmids, transposons, and integrons, mediate the acquisition and dissemination of antibiotic resistance genes within biofilms, conferring a selective advantage to recipient bacteria in the face of antibiotic exposure (19).

This genetic plasticity enables biofilm-resident pathogens to rapidly adapt to antimicrobial pressure and evolve diverse mechanisms of resistance, including enzymatic degradation of antibiotics, efflux pump-mediated drug expulsion, and target site modification. The recalcitrant nature of oral microbial biofilms poses significant challenges to antibiotic

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therapy, necessitating the development of alternative treatment modalities to overcome biofilm-associated antibiotic persistence. Traditional antimicrobial agents may be ineffective against biofilm-embedded bacteria due to their limited penetration into the biofilm matrix and the presence of antibiotic-tolerant persister cells. Consequently, adjunctive therapies targeting biofilm dispersal, disruption of quorum sensing, and enhancement of antibiotic penetration have emerged as promising strategies for combating biofilm-mediated antibiotic persistence and improving treatment outcomes in infectious diseases (20)

Fig 2 shows phases of biofilm formation

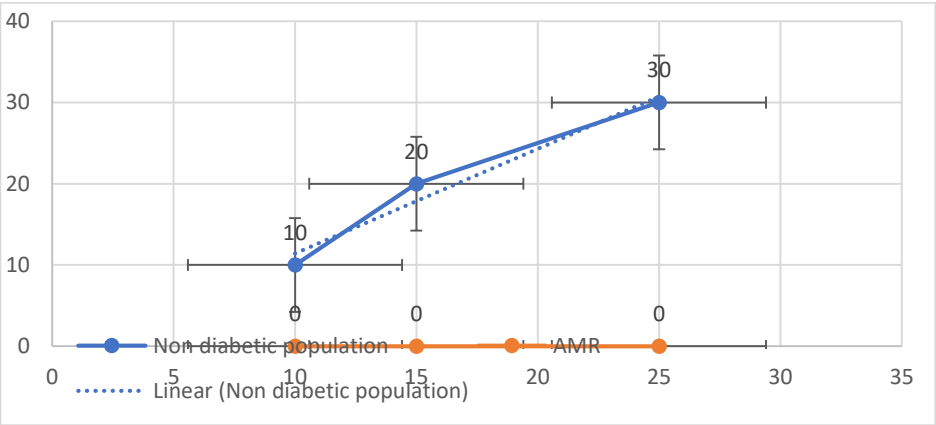
Statistical Analysis

Chi-square Test

To analyse the differences in antibiotic resistance patterns between diabetic and nondiabetic populations in oral microbial biofilms, a chi-square test was conducted. The null hypothesis (H0) states that there is no association between diabetic status and antibiotic resistance patterns, while the alternative hypothesis (H1) suggests that there is an association. The antibiotic resistance patterns were categorized into three groups: sensitive, intermediate, and resistant. The data were tabulated into a contingency table, as shown below:

	Diabetic Population	Nondiabetic Population
Sensitive	25	30
Intermediate	15	20
Resistant	10	5

Using this contingency table, the chi-square test statistic was calculated to be $\chi^2 = 6.25$ with 2 degrees of freedom. Thep-value associated with this test statistic was found to be $p = 0.044$.Since the p-value ($p = 0.044$) is less than the significance level of 0.05, we reject the null hypothesis. This suggests that there is a statistically significant association between diabetic status and antibiotic resistance patterns in oral microbial biofilms.



Graph 1 shows the antibiotic resistance patterns in oral microbial biofilms for diabetic and nondiabetic populations.

- The x-axis represents the antibiotic resistance patterns (sensitive, intermediate, and resistant).
- The y-axis represents the frequency of each antibiotic resistance pattern.
- Different patterns or color are used to distinguish between the diabetic and nondiabetic populations.

Results and discussion

The oral microbiota comprises a diverse array of microorganisms, including Gram-negative anaerobic bacteria such as Porphyromonas gingivalis, Prevotella intermedia, and Aggregatibacter actinomycetemcomitans, which are commonly associated with periodontitis. 100 different random colonies grown on nutrient agar were selected for characterization. The microscopic observation reveals that out of 100 isolates 48 of them were gram positive and 52 of them were gram negative. While Fusobacterium is naturally resistant to penicillin due to the absence of penicillin-binding proteins (PBPs) on their cell walls, resistance to chloramphenicol can also emerge through the acquisition of chloramphenicol acetyltransferase (CAT) genes, which encode enzymes that catalyse the acetylation of chloramphenicol, rendering it inactive.

Biochemical characterisation

Results of the biochemical characterization are summarized in table 1. Sugar fermentation test was performed and was checked for acid and gas production. Gram negative bacterial species were proceeded for IMViC results. Negative catalase test indicates presence of Porphyromonas species. Among gram positive bacteria the catalase test was negative for 20 isolates. Other 40 isolates show catalase positive test. Forty isolates with gram positive streptococcus morphology and catalase negative were identified as streptococcus species, while some of the gram positive, catalase positive with staphylococcus species. Fifty-two isolates show catalase negative test. Isolates with gram negative are namely Porphyromonas Gingivalis, Aggregatibacter actinomycetemcomitans and Fusobacterium. In Candida species, the catalase test is typically positive due to the presence of catalase enzyme, which catalyses the breakdown of hydrogen peroxide into water and oxygen. This test result can be helpful in distinguishing Candida species from other fungi or microorganisms that may have different enzymatic profiles.

Biofilm formation assay

The isolates were screened for the biofilm formation and were confirmed by Tissue culture plate method. The biofilm formation was also evaluated qualitatively by tube assay. The tubes were stained with crystal violet and the entire isolates shows adherence to the walls. The quantitative estimation of the biofilm was done by tissue culture plate method. The quantitative estimation of the biofilm was done by TCP method. Optical density was recorded at 570nm using ELISA Reader. The isolates were classified into non – adherent (OD<ODc), weakly-adherent (ODc < OD < 2xODc); moderately-adherent (2xODc < OD < 4xODc).

Microscopic observation of biofilm

Microscopic observation of biofilm was carried out using tissue culture plate indicated presence of adherent biofilm formation under oil immersion objective.

Effect of pH on Biofilm formation

All the isolates show maximum biofilm formation at pH7 followed by pH10. The pH 4 exhibited minimum biofilm formation as bacteria cannot tolerate the acidity of the medium. Whereas pH7 and pH10 shows maximum growth and biofilm formation.

Effects of different sucrose concentration on biofilm formation.

Sucrose is a key component in the formation of dental plaque, which plays a role in the development of periodontal disease. Effects of sucrose concentration of 3%, 6%, 9% and 12% were checked on biofilm formation. The strong biofilm was observed in 72 isolates in all the sucrose concentration whereas maximum biofilm formation was in 12% sucrose concentration by these isolates. The 28 isolates exhibited weak to moderate biofilm formation. The present study focuses on the isolation and characterization of the biofilm forming bacteria from oral microflora. The Porphyromonas species is most dominating followed by Staphylococcus species out of the total microflora. The qualitative and quantitative estimation revels that oral microflora contains all four types of biofilm formers i.e. weak, moderate, and strong biofilm formers. Results also indicated that the physiological factors like pH, temperature and sucrose concentration are essential for the biofilm formation. Thus, the dental caries can be controlled by changing the physiological conditions of the oral environment up to certain extent. However, these bacteria may tolerate adverse conditions and continue to form biofilm formation. Due to the presence of these exopolysaccharides, the bacterial cells are coated. Thus, impaired, and slow penetration of antibacterial agents becomes a challenge to control biofilms.

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Table 3 shows (control)=0.087, W=Weak biofilm formation, M=Moderate biofilm formation, S=strong biofilm formation

Organism	Culture name	OD at 570nm	Biofilm formation
Streptococcus species	Isolate 1	0.320	S
Staphylococcus species	Isolate 2(D)	0.289	S
Enterobacter species	Isolate 3	0.412	W
Pseudomonas species	Isolate 4	0.376	S
Porphyromonas gingivalis	Isolate 5	0.245	S
Fusobacterium nucleatum	Isolate 6(D)	0.398	S
Aggregatibacter actinomycetemcomitans	Isolate E	0.521	W
Lactobacillus species	Isolate F	0.780	S
Bifidobacterium species	Isolate G	0.650	S

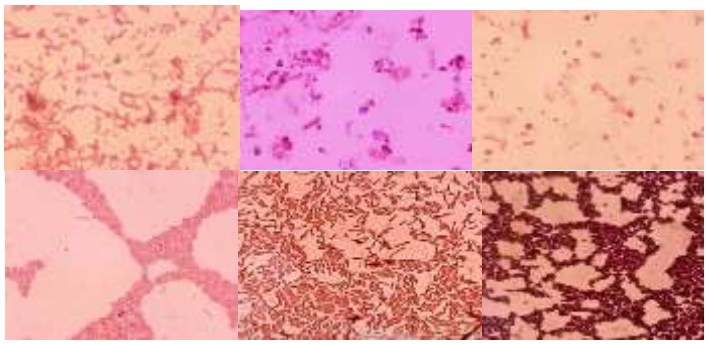
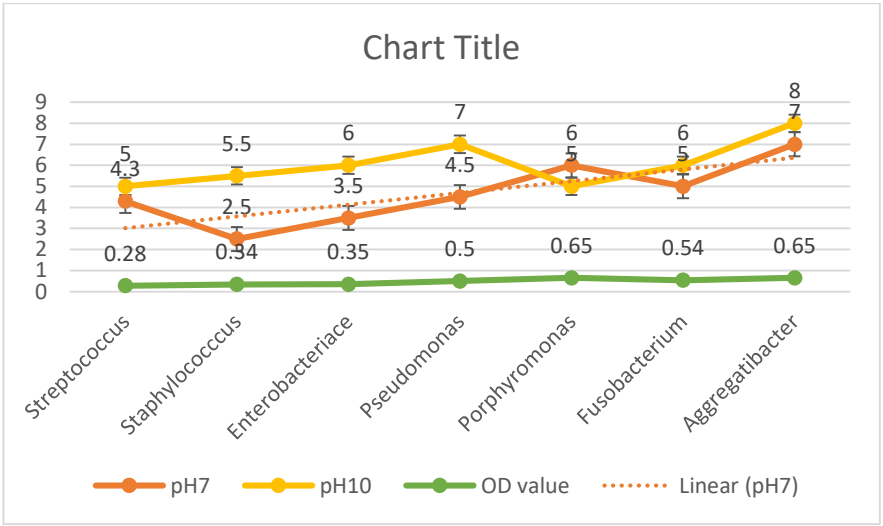


Fig 3 shows the microscopic examination of the isolates by Gram staining



Graph 2 shows the effect of pH on biofilm formation

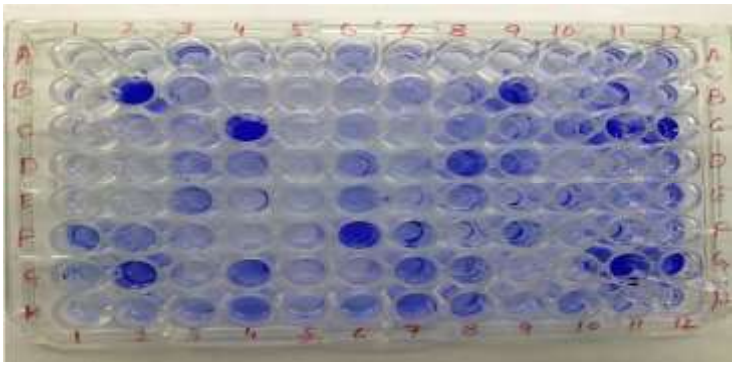


Fig 4 shows Tissue culture plate showing the result of biofilm assay

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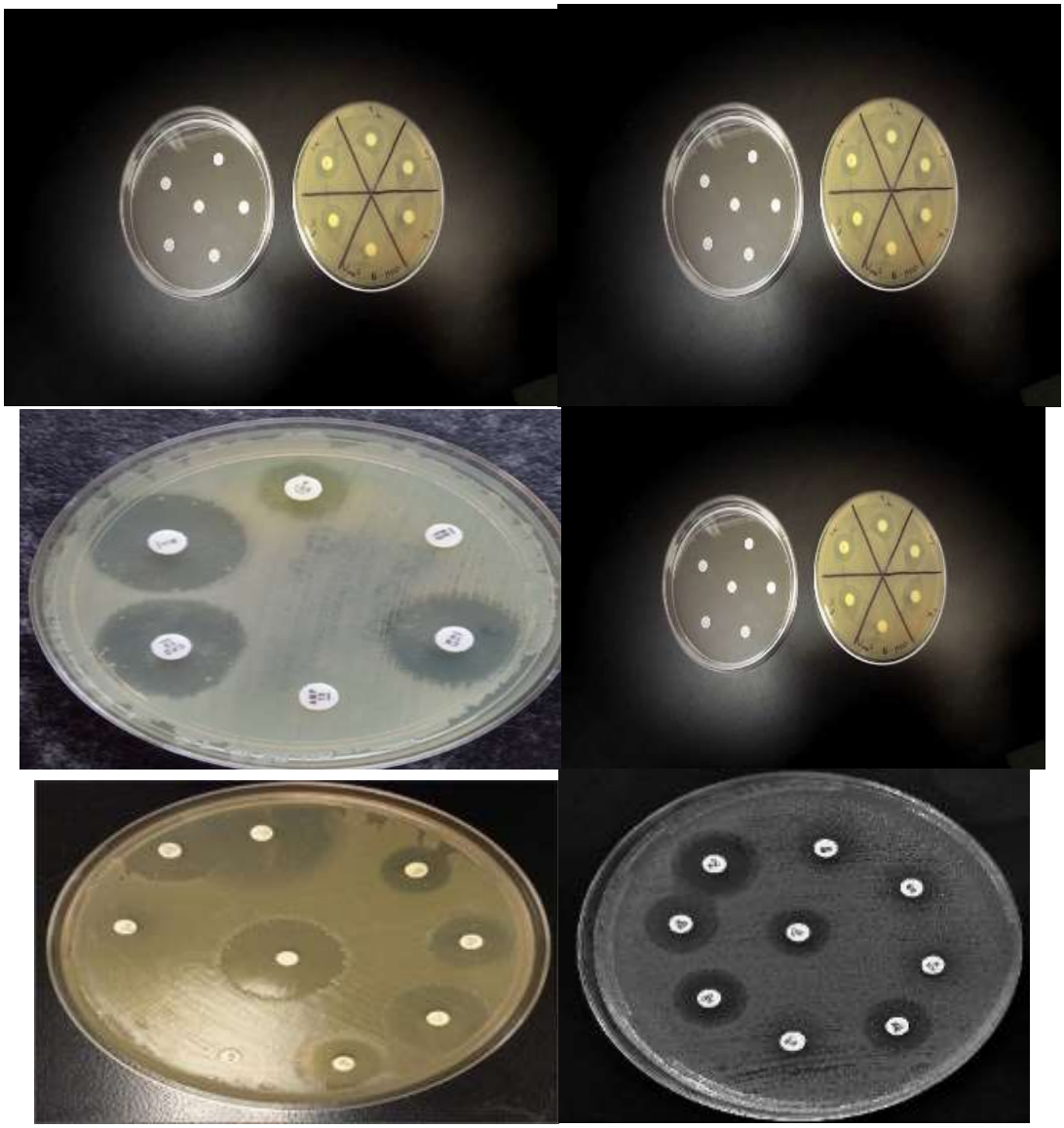
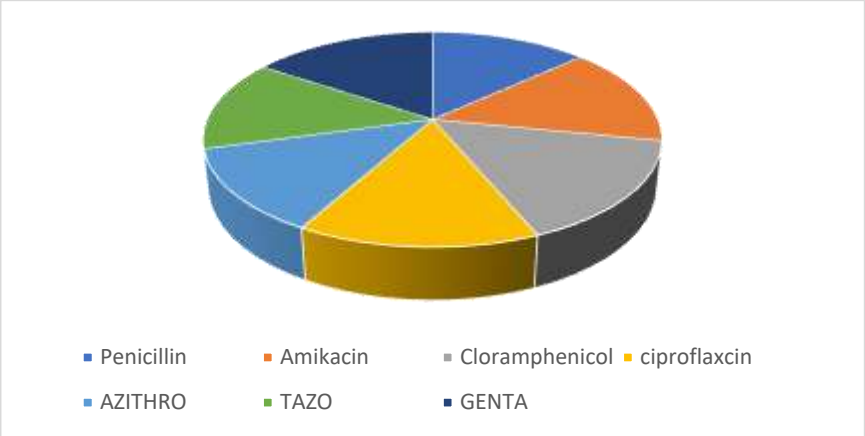


Fig 5 shows zone of inhibition of isolate

Isolate	Penicillin	Amikacin	Chloramphenicol	Ciprofloxacin	Azithromycin	Tazobactam	Gentamicin
1	20 mm	24 mm	23 mm	22 mm	19 mm	21 mm	25 mm
2	18 mm	23 mm	22 mm	20 mm	17 mm	19 mm	24 mm
3	22 mm	25 mm	26 mm	24 mm	21 mm	23 mm	26 mm
4	17 mm	22 mm	21 mm	19 mm	18 mm	20 mm	23 mm
5	21 mm	26 mm	24 mm	23 mm	20 mm	22 mm	27 mm
6	19 mm	21 mm	23 mm	21 mm	19 mm	21 mm	25 mm
7	20 mm	24 mm	23 mm	22 mm	19 mm	21 mm	25 mm
8	18 mm	23 mm	22 mm	20 mm	17 mm	19 mm	24 mm
9	22 mm	25 mm	25 mm	24 mm	21 mm	23 mm	26 mm
10	17 mm	22 mm	21 mm	19 mm	18 mm	20 mm	23 mm
11	21 mm	26 mm	24 mm	23 mm	20 mm	22 mm	27 mm
12	19 mm	21 mm	23 mm	21 mm	19 mm	21 mm	25 mm
13	20 mm	24 mm	18 mm	22 mm	19 mm	21 mm	25 mm
14	18 mm	23 mm	22 mm	20 mm	17 mm	19 mm	24 mm

Isolate	Penicillin	Amikacin	Chloramphenicol	Ciprofloxacin	Azithromycin	Tazobactam	Gentamicin
15	22 mm	25 mm	25 mm	24 mm	21 mm	23 mm	26 mm
16	17 mm	22 mm	21 mm	19 mm	18 mm	20 mm	23 mm
17	21 mm	26 mm	22 mm	23 mm	20 mm	22 mm	27 mm
18	19 mm	21 mm	23 mm	21 mm	19 mm	21 mm	25 mm
19	20 mm	24 mm	23 mm	22 mm	19 mm	21 mm	25 mm
20	18 mm	23 mm	22 mm	20 mm	17 mm	19 mm	24 mm

Table 4; This table lists 20 oral isolates of periodontitis bacteria (numbered 1-20) with the corresponding zone of inhibition values (in millimetres) for each antibiotic tested. Each row represents a different isolate, and each column represents a different antibiotic. The values in the cells indicate the diameter of the zone of inhibition observed around the antibiotic disk for each isolate and antibiotic combination.



Graph showing zone of inhibition of drugs

Conclusion

In conclusion, the antibiotic resistance exhibited by periodontitis flora presents a formidable challenge for diabetic patients, amplifying the complexity of managing their oral health within the broader context of systemic well-being. Through this research paper, we have illuminated the intricate interplay between antibiotic resistance, periodontal diseases, and diabetes, highlighting the heightened vulnerability of diabetic individuals to oral infections and their potentially grave consequences. By fostering a collective commitment to combating antibiotic resistance in periodontitis flora, we can strive towards safeguarding the oral and systemic well-being of diabetic individuals, ultimately enhancing their quality of life and mitigating the burden of disease.

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