



Antibiogram of Extended-Spectrum Beta-Lactamase-Producing Gram-Negative Bacteria Isolated from Patients with Periodontal Diseases Attending Dental Clinics in Enugu

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Summary

INTRODUCTION

This study was designed to determine the antibiogram and prevalence of Gram-negative bacteria (GNB) in patients with gingivitis and chronic periodontitis.

MATERIALS AND METHODS

Exactly 180 gingival sulcus samples of patients' jaws collected using paper points were placed in double strength nutrient broth and analysed using standard microbiological techniques. Identified bacteria were phenotypically screened for ESBL (Extended-Spectrum Beta-Lactamase)-production using Double Disk Synergy Test (DDST). Antimicrobial susceptibility of isolated ESBL-producing bacteria was determined using the Kirby-Bauer disk diffusion method.

RESULTS

Results showed that age group > 36 years and above had the highest incidence of chronic periodontitis (46.5 %) and gingivitis (25 %). Pocket probing Depth \geq 4 mm was common among chronic periodontitis patients (51.1 %) while probing depth \leq 3 mm was prevalent amongst gingivitis patients (11.8 %). Bleeding on probing site \geq 50 % was more predominant among chronic periodontitis patients with frequency of 41.5 %; followed by 28.2 % for patients with gingivitis. *K. pneumoniae* 65(19.2 %), *E. coli* 44(12.9 %), *Salmonella* spp 35(10.3 %), *K. oxytoca* 34(10 %), and *P. aeruginosa* 13(3.8 %) were the GNB isolated.



CONCLUSION

Out of the bacteria isolated, 106 (31.2 %) were phenotypically identified to be ESBL-producers. Isolates were generally susceptible to gentamicin (50 %) and colistin sulphate (100 %), but completely resistant (100 %) to chloramphenicol, cefotaxime, ceftazidime, trimethoprim-sulfamethoxazole, and tetracycline respectively.

Multiple Antibiotic Resistance Index (MARI) values of the bacterial isolates ranged from 0.2-0.9. Periodontal and gingival sites of patients in this study harboured multidrug-resistant ESBL-producing GNB which may limit treatment options for periodontitis and gingivitis.

Keywords: Gram-Negative Bacteria, Antibiotic Resistance, Periodontitis, Gingivitis, ESBL.

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Introduction

Chronic periodontitis is a multifactorial infectious disease of the supporting tissues of the teeth initiated by disturbances in the subgingival biofilm and homeostasis, with high prevalence worldwide [1].

The leading role of Gram-negative subgingival microflora has been defined, with a group of fully identified bacteria including *Porphyromonas gingivalis*, *Treponema denticola*, *Tannerella forsythia*, *A. actinomycetemcomitans*, *Prevotella intermedia*, *Camphylobacter rectus*, *Fusobacterium nucleatum*, *Capnocytophaga* sp. and *Parvimonas micra* [2]. It is a complex infectious disease resulting from the interplay of bacterial infection and host response to bacterial challenge. Factors inherent to the host such as heredity, smoking and environmental factors are important determinants in the progression and severity of the disease. The presence of enterobacteria in the oral cavity is basically due to oral-faecal transmission, deficient oral hygiene, or contamination from grooming accessories, food or drink [3]. Another outstanding feature of Enterobacteriaceae is their implication as key pathogens in some cases of refractory

periodontitis. Similarly, these opportunistic microorganisms may be major players as a result of the inadequate use of antibiotics, which may suppress normal oral microbials, leading to persistent colonization by opportunist pathogens [4].

Antibiotics are often prescribed to treat periodontal infections; however, studies have shown that many oral anaerobic bacteria have developed resistance to various antibiotics by production of beta lactamases [5]. However, microbial resistance to these antibiotics and the dissemination of resistance genes among oral microorganisms needs further investigation, as the oral cavity may harbour some multidrug-resistant microorganisms, particularly enteric rods and cocci.

Extended spectrum beta-lactamases (ESBLs) are defined as enzymes produced by certain bacteria that are able to hydrolyze extended spectrum cephalosporin. They are therefore effective against beta-lactam antibiotics like ceftazidime, ceftriaxone, cefotaxime and oxyimino-monobactam [6].

Carbapenems and cephamycin are effective against ESBL producing strains. Generally, ESBLs are inhibited by clavulanic acid and tazobactam. ESBLs are found in Gram-negative bacteria, especially in



Enterobacteriaceae and *Pseudomonas aeruginosa* [7]. Data regarding the prevalence of Enterobacteriaceae in Nigeria population are lacking.

This study was designed to determine the antibiogram of ESBL-producing Enterobacteriaceae isolated from oral cavities of patients diagnosed with gingivitis and periodontitis visiting Federal College of Dental Technology and Therapy (FCDT & T) Clinics, Enugu, Nigeria.

Material and Methods

Study Population

A total of 180 patients (106 males and 74 females) attending Federal College of Dental Technology and Therapy (FCDT & T) Clinic, Trans Ekulu, Enugu, Nigeria for the treatment of periodontal and gingival diseases were the study population. In this descriptive study, complete periodontal evaluation was performed (full mouth) on all patients, using a Williams probe (Williams color-coded probe PQW, Hu-Friedy, Chicago-Illinois, USA). Periodontal evaluation; including gingival margin, bleeding on probing, probing depth, and clinical attachment level were done and classified according to the recommendations of the 1999 International Consensus of the American Academy of Periodontology [8].

Exclusion and Inclusion

Inclusion criteria were patients diagnosed with chronic periodontitis or gingivitis, with at least 10 teeth, without systemic compromise, over 20 years old, and who had not received prior periodontal treatment within the last 6 months. Exclusion criteria were patients undergoing antibiotic or corticosteroid therapy within the last three months, pregnant or lactating women, smokers and diabetic patients. A questionnaire was designed to collect socio-demographic data (age, gender, marital status, educational statuses) and other relevant data about

gingivitis and chronic periodontitis using periodontal disease index (PDI).

Ethical Clearance

The ethical clearance for this study (Reference no: FCDTT/REC/VOL 2/2018/104) was obtained from the research and ethical committee of Federal College of Dental Technology and Therapy Enugu, Nigeria.

Collection of Specimens and Sampling Procedure

A total of 180 samples from gingival sulcus were collected from patients visiting FCDT&T clinic, Enugu. Calibration was performed after which periodontal probing was done. Six (6) surfaces of all teeth were measured in order to select which surface(s) to include in the sampling (mesial vestibular, mesial lingual/palatal and vestibular/palatal interproximal surfaces).

To take the sample from the gingival sulcus, 5 sites with pocket probing depth ≥ 4 mm and clinical attachment level ≥ 2 mm were selected for the group with chronic periodontitis, while pocket probing depth ≤ 3 mm and absence of gingival inflammation were selected for the group with gingivitis. Supragingival film was removed with sterile gauze. The zone was then isolated with sterile cotton wool and paper points was placed depending on the depth of the gingival sulcus [9]. Paper points were removed after 1 minute in the gingival sulcus and placed in Eppendorf tubes containing 900 μ l thioglycollate broth supplemented with hemin and menadione. Test tubes containing the samples were then transported to the Department of Applied Microbiology, Ebonyi State University, Abakaliki, Nigeria for bacteriological analysis.



Bacteriological Analysis of Samples

Each sample was pre-enriched in prepared sterile peptone broth and incubated at 37 °C for 24 hrs. After incubation, the turbid broth was sub-cultured on nutrient agar, cetrimide agar, MacConkey agar, brain heart infusion agar, blood agar, cysteine lactose electrolyte deficient (CLED) agar and incubated at 37 °C for 24 hrs. After microbial growth, the macroscopic characteristics of colonies were observed and a Gram stain was performed following biochemical tests. Bacterial isolates were further identified using the API 20E (Biomerieux S.A., Marcy-l'Etoile/France) identification system.

Antibiotic Susceptibility Testing

Antibiotic susceptibility test of bacterial isolates was determined using the Kirby-Bauer disc diffusion method according to the recommendations of the Clinical and Laboratory Standard Institute CLSI [10]. Isolates were sub-cultured on nutrient agar, incubated at 37 °C for 18-24 hours. Then the colonies of each of the isolates were adjusted to 0.5 McFarland turbidity standard (equivalent to 1.5×10^8 cfu/ml) in sterile nutrient broth.

The standardized broth culture was incubated for 10 minutes and using sterile swab stick, the standardized broth culture of the isolates was inoculated onto Mueller-Hinton agar plates. The surface of the medium was streaked in four directions while the plates were rotated approximately 60° to ensure even distribution. The inoculated Mueller-Hinton agar plates were allowed to dry for about 10 minutes at room temperature with the lid closed. After the agar surface has dried for few minutes, antibiotic impregnated discs (Oxoid, UK) of known concentrations; tetracycline (30

µg), ampicillin (10 µg), amoxicillin (30 µg), penicillin (10 µg), cefotaxime (30 µg), ceftriaxone (30 µg), cefotaxime (30 µg), amoxicillin/clavulanic acid (30 µg), ceftazidime (30 µg), cefepime (30 µg), cefotetan (30 µg), cefuroxime (30 µg), gentamicin (30 µg), kanamycin (30 µg), tobramycin (30 µg), trimethoprim-sulfamethoxazole (30 µg) and ciprofloxacin (5 µg) were carefully applied on the inoculated Mueller-Hinton agar plates using sterile forceps.

The plates were then incubated at 37 °C for 24 hours. After 30 minutes, the plates were inverted and incubated for 24 hours. A ruler was used to measure the diameter of each zone of inhibition in mm on the underside of the plate. The inhibitory zone diameter was interpreted as susceptible, intermediate or resistant according to the criteria of CLSI.

Screening for ESBL

Production

The presence of ESBL was presumptively detected in isolates using double disk synergy test (DDST), in line with CLSI criteria [10] by inoculating a standardized (0.5 McFarland turbidity standards) suspension of the test organism on Mueller-Hinton (MH) agar (Oxoid, UK). Antibiotic disc of amoxicillin/clavulanic acid (30 µg) was placed at the center of the MH agar plate and antibiotic discs containing cefotaxime (30 µg) and ceftazidime (30 µg) each was placed at a distance of 15 mm (center to center) from the central disc (amoxicillin/clavulanic acid), and the plate was incubated at 37 °C for 18-24 hours.

ESBL production was suspected phenotypically when the zones of inhibition of the cephalosporins (cefotaxime 30 µg and ceftazidime 30 µg) increased in the presence of amoxicillin/clavulanic acid disk (30 µg). A ≥ 5 mm increase in the inhibition zone diameter for either of the cephalosporins (cefotaxime



and ceftazidime) tested in combination with amoxicillin-clavulanic acid versus its zone when tested alone confirmed ESBL production phenotypically [11].

Multiple Antibiotic

Resistance Index (Mari)

The Multiple Antibiotic Resistance Index was calculated as the ratio of the number of antibiotics to which the bacterial isolates were resistant /the total number of antibiotics against which the isolates were tested [11, 12].

Statistical Analysis

Statistical analysis was performed using SPSS 17.0 version statistical software package. Comparison between categorical variables was calculated using the T-test and ANOVA. Results were considered statistically significant if the p value is less than 0.05 ($p < 0.05$).

Results

The socio-demographic data of patients revealed that out of the 180 patients diagnosed with chronic periodontitis, 106 (31.1 %) were males while 74 (21.8 %) were females (Table 1). There was no statistically significant difference in the prevalence of chronic periodontitis and gingivitis between males and females. Age group >36 years & above recorded higher incidence of chronic periodontitis (46.5 %) and gingivitis (25 %) than that among age $> 20-35$ years with incidence frequencies of 13.5 % and 15 % for chronic periodontitis and gingivitis respectively .

Pocket probing depth ≥ 4 mm (51.1 %) was common among chronic periodontitis patients while probing depth ≤ 3 mm (11.8 %) was prevalent amongst patients with gingivitis. Bleeding on probing site ≥ 50 % was more predominant among chronic periodontitis patients (41.5 %), followed by patients with gingivitis (28.2 %) as presented in Table 2.

A total of 191(56.2 %) Gram-negative bacteria were recovered from patients with periodontitis and gingivitis. *Klebsiella pneumoniae* was the most predominant enterobacteria species isolated with a frequency of 65(19.2 %), followed by *E. coli* with frequency of 44(12.9 %). *Pseudomonas aeruginosa* was the least predominant with frequency of 13(3.8 %). Bacterial species with high frequencies of 63 (18.5 %) and 57 (16.8 %) were recorded for periodontal and gingival sites (upper right jaw and lower left jaw) respectively (Table 2). There was no statistically significant difference in the frequency of bacteria between the upper and lower jaw.

A total of 106(31.2 %) Gram-negative bacteria isolated from patients with periodontitis and gingivitis were extended-spectrum beta-lactamase (ESBL)-producers. ESBL production was more prevalent amongst *K. pneumoniae* (14.1 %), followed by *Salmonella* spp (6.8 %), *K. oxytoca* (5.9 %), and *E. coli* (4.4 %) being the least, while *P. aeruginosa* was negative for ESBL production (Table 3).

Gram-negative bacteria recovered from patients with periodontitis and gingivitis were completely resistant (100 %) to amoxicillin-clavulanic acid, chloramphenicol, tetracycline, and trimethoprim-sulfamethoxazole. Isolates also exhibited resistance to ceftriaxone (93.3 %), aztreonam (88.9 %), ceftazidime (77.8 %), cefotaxime (66.7 %), gentamicin (66.7 %), and ciprofloxacin (55.6 %). Contrastingly, all isolates were highly susceptible to colistin sulphate (100 %) and imipenem (78.9 %) as shown in table 4.

Recovered Gram-negative bacterial isolates had multiple antibiotic resistance index (MARI) values ranging from 0.2 – 0.9



Table 1: Socio-Demography of Patients Visiting Federal College of Dental Technology and Therapy Enugu Dental Clinic for Treatment

Demographic and Dental relevant parameters	Chronic periodontitis (%)	Gingivitis (%)
Gender		
Male	106(31.1)	92(27.1)
Female	74(21.8)	68(20)
Age (year)		
>20-35	46(13.5)	51(15)
>36 & Above	158(46.5)	85(25)
Marital status		
Single	56(16.5)	71(20.9)
Married	97(28.5)	48(14.1)
Other	68(20)	0(0.0)
Educational status		
Formal	41(12.1)	27(7.9)
Informal	183(53.8)	89(26.2)
Destructive/Inflammatory Periodontal Region		
Lower Right Jaw	54(15.9)	31(9.1)
Lower Left Jaw	37(10.9)	48(14.1)
Upper right Jaw	58(17.1)	27(7.9)
Upper Left Jaw	61(17.9)	24(7.1)
Pocket probing depth		
≥ 4mm	174(51.1)	93(27.4)
≤ 3mm	33(9.7)	40(11.8)
Bleeding on probing site		
≤ 50% of sites	69(20.3)	34(10)
≥ 50% of sites	141(41.5)	96(28.2)
Clinical attachment level		
≥ 2mm	160(47.1)	121(35.6)
≤ 2mm	36(10.6)	23(6.7)

Table 2: Frequency Distribution of Gram-Negative Bacteria in Patients with Periodontitis and Gingivitis

Gram-negative bacteria	Periodontal/ Gingival Sites				Total Prevalence (%)
	Lower Right Jaw (%)	Lower Left Jaw (%)	Upper Right Jaw (%)	Upper Left Jaw (%)	
<i>P. aeruginosa</i>	0 (0.0)	2(0.6)	6(1.8)	5 (1.5)	13(3.8)
<i>E. coli</i>	7(2.1)	11(3.2)	15(4.4)	11(3.2)	44(12.9)
<i>Salmonella spp</i>	5(1.5)	17(5)	10(2.9)	3(0.9)	35(10.3)
<i>K. pneumoniae</i>	15(4.4)	20(5.9)	23(6.8)	7(2.1)	65(19.2)
<i>K. oxytoca</i>	11(3.2)	7(2.1)	9(2.6)	7(2.1)	34(10)
Total	38(11.2)	57 (16.8)	63 (18.5)	33 (9.7)	191(56.2)



Table 3: Phenotypic Detection of ESBLs in Gram-Negative Bacteria Isolated from Patients with Periodontitis and Gingivitis

Gram-negative bacteria	Periodontal/ Gingivae Sites				Total ESBL-positive (%)
	Lower Right Jaw (%)	Lower Left Jaw (%)	Upper Right Jaw (%)	Upper Left Jaw (%)	
<i>P. aeruginosa</i>	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
<i>E. coli</i>	6(1.8)	0 (0.0)	0 (0.0)	9(2.6)	15(4.4)
<i>Salmonella</i> spp	5(1.5)	15(4.4)	0 (0.0)	3(0.9)	23(6.8)
<i>K. Pneumoniae</i>	12(3.5)	17(5)	19(5.6)	0 (0.0)	48(14.1)
<i>K. oxytoca</i>	11(3.2)	0 (0.0)	9(2.6)	0 (0.0)	20(5.9)

Table 4: Antibiotics Susceptibility Pattern of Clinical Isolates of ESBL-Producing Gram-Negative Bacteria Recovered from Patients with Periodontitis and Gingivitis

Antibiotics	Resistance (%)	Susceptibility (%)
Amoxicillin- clavulanic acid	100	0
Azetroneam	88.9	11.1
Cefotaxime	66.7	33.3
Ceftazidime	77.8	22.2
Ceftriaxone	93.3	6.7
Chloramphenicol	100	0
Colistin sulphate	0	100
Ciprofloxacin	55.6	44.4
Gentamicin	66.7	33.3
Imipenem	21.1	78.9
Tetracycline	100	0
Trimethoprim-sulfamethoxazole	100	0

Discussion

Periodontitis is of multi-bacterial origin and almost 12 putative periodontal pathogens have been identified [13]. Periodontal disease is the most frequent oral disease, second only to dental caries and a significant oral health issue with attendant socio-economic problems in Nigeria [14]. This study evaluated the antibiogram of ESBL-producing Gram-negative bacteria isolated from oral cavities of patients diagnosed with gingivitis and periodontitis visiting Federal College of Dental Technology and Therapy (FCDT & T) Clinic, Enugu, Nigeria. Demographic and dental relevant parameters

of the studied population in this work showed that the frequency of gingivitis was higher in males (27.1 %) than in females (20 %). This result is in agreement with other studies which reported higher gingivitis occurrence among males than females. Abdulaziz [15] reported 64 % and 34 % gingivitis prevalence in males and females respectively.

Gingivitis occurrence frequencies of 65.2 % and 38.9 % have also been reported in males and females respectively [16]. Similar finding was also reported by Majdy *et al.* [17] in Saudi population. Prevalence of study population with gingival bleeding on probing sites ≥ 50 % was 28.2 %, which is consistent



with the findings of Majdy *et al.* [17] and Dhakal *et al.* [16] who reported prevalence values of 28.8 % and 30 % respectively. The low prevalence of gingivitis (47.1 %) among the participants in this study is similar to the 31.8 % reported in Columbia [18].

In this study, chronic periodontitis was observed in age group >36 & above (46.5 %) than >20-35 (13.5 %). This result corroborates the work of Nazir *et al.* [19] who reported that the prevalence and severity of periodontal diseases is usually increased with age, significantly over 40 years old. It is still uncertain whether aging is a risk factor for the development of severe periodontal diseases, or if it is due to the prolonged exposure to real etiological factors in older patients. The observed difference in the prevalence of periodontal disease noted in this study between age group >36 & above and age group >20-35 may be explained by the negligible difference in tooth cleaning frequency and periodontal disease risk behaviour among older participants than younger participants [19].

The frequency of chronic periodontitis was higher in males (31.1 %) than in females (21.8 %). On the other hand, Raghianti *et al.* [20] reported that males are more prone to develop periodontal diseases because they usually display poorer oral hygiene than females. Although oral hygiene, as risk factor, was not considered in this study, but chronic periodontitis has been linked to male with smoking and kola-nut eating habits [21]. It's worth-noting that amongst the studied patients, 52.9 % had periodontitis while 47.1 % had gingivitis. The prevalence of chronic periodontitis (52.9 %) in this study is within the previous reported periodontitis prevalence (15-57 %) in Nigeria [9], and in Columbia where 25.6 % patients visiting dental clinics had gingivitis while 74.4 % had chronic periodontitis. In contrast, high gingivitis prevalence of 75.4 % among adult male

populations in Nigeria [21]; 67.3 % among American population [22], and 82 % among adult Nepalese [16] have been reported. The pattern of periodontal disease in this study pointed to the fact that prompt treatment intervention will lead to total restoration of periodontal health, thus halting progression to destructive periodontitis in the majority of the participants because gingivitis is known to be reversible with adequate care.

In this current study, the frequency of chronic periodontitis (46.5 %) and gingivitis (25 %) was extremely higher among the age group (>36 and above), reflecting periodontal disease as a disease of lifetime accumulation. The ebbing immunity with aging impairment of host response to disease may have increased the occurrence of periodontal disease among older participant in this study. In this study, patients with informal education had higher prevalence of periodontitis (53.8 %) and gingivitis (26.2 %) than patients with formal education.

Several studies have also documented a higher prevalence of periodontal disease in individuals with lower educational attainment [21] and may reflect lack of awareness on periodontal disease. Our study revealed that periodontal pockets were populated with 56.2 % Enteriobacteriaceae. This result is similar to documented data in China (57 %) [23], but differed from low frequencies reported in Chile (17.6 %) [24] and Brazil (31.2 %) [25]. These studies denote that prevalence is not uniform and varies with the population examined due to multitude of factors such as geographic area/economic status of the country, diet, sampling technique employed, and the condition of periodontal apparatus.

Klebsiella pneumoniae (19.2 %) was the most predominant enterobacteria in patients with chronic periodontitis and gingivitis in our study.



Gamboa *et al.* [18] also reported prevalence frequencies of 75 % and 22.8 % for *K. pneumonia* and *K. oxytoca* respectively. Other studies have highlighted the presence of other Gram-negative bacteria, such as *Escherichia coli* and *Pseudomonas aeruginosa* reported in this study in chronic periodontitis and gingivitis [26] except for *Salmonella* spp in which this study was the first to report prevalence in chronic periodontitis and gingivitis patients in Nigeria. *P. aeruginosa* which was isolated in this study has been reported by Persson *et al.* [27].

The frequencies of enterobacteria in this study may postulate a common source of contamination and transmission of this pathogen among periodontal patients basically due to oral-faecal transmission, deficient oral hygiene, or contamination from grooming accessories, food or drink. Exactly 31.2 % of isolates in this study were ESBL producers and our findings are consistent with the studies of Benachinmardi *et al.* [28] and Iwahara *et al.* [29] who reported prevalence of 26 % and 31% respectively in patients with periodontitis. Rams *et al.* [30] and Patel [31] reported high prevalence of ESBL producers among bacterial isolates with frequencies of 52.1 % and 69 % ESBL producers. ESBL were more predominant amongst *Klebsiella pneumoniae* (14.1 %).

In Nigeria, ESBL-producing *Klebsiella pneumoniae* of non-periodontal origin have been reported in Enugu [32], Ebonyi [32], and Abeokuta [33] with occurrence frequencies of 44.6 %, 6.7 %, and 5% respectively. The high frequency of ESBL-producers among patients will make the treatment of bacterial infections more difficult.

Isolates in this study exhibited high resistance to amoxicillin-clavulanic acid, chloramphenicol, tetracycline, and trimethoprim-sulfamethoxazole, ceftriaxone, aztreonam, ceftazidime, cefotaxime,

gentamicin, and ciprofloxacin. Similar antibiotic resistance patterns in this study have also been reported by Olowe *et al.* [33]. Contrastingly, all isolates in this study were highly susceptible to colistin sulphate (100 %) and imipenem (78.9 %). This is in agreement with previous studies [33] that reported that ESBL-producing Enterobacteriaceae were highly susceptible to colistin sulphate and imipenem. The effectiveness of colistin sulphate and in combination with other antibiotics against ESBL-Producing Enterobacteriaceae has been reported [34]. Multiple Antibiotic Resistant index (MARI) of isolates in this study ranged from 0.2 – 0.9 and this MARI value range shows that there is antibiotic abuse in our study area, thus depicting multidrug-resistant traits for bacterial isolates.

Expression of multi-drug resistance among the ESBL-producing Enterobacteriaceae demonstrates that systemically, healthy adult subjects may harbour multidrug-resistant clones in sites of chronic periodontal diseases.

Conclusion

The present study identified and implicated multidrug-resistant ESBL-producing Enterobacteriaceae in chronic periodontal diseases among patients visiting dental clinics in Enugu. This study also showed that isolates were generally highly resistant to amoxicillin-clavulanic acid, chloramphenicol, tetracycline, and trimethoprim-sulfamethoxazole, ceftriaxone, aztreonam, ceftazidime, cefotaxime, gentamicin, and ciprofloxacin which are commonly used to treat bacterial infections.

Interestingly, colistin sulphate and imipenem were the most effective antibiotics against the ESBL-producing bacterial isolates as they were highly susceptible to these antibiotics. Our study has shown that oral cavity may act as a reservoir and a source of



dissemination of ESBL-producing Enterobacteriaceae to other body sites. Thus, it is imperative to establish strong antibiotic usage policies to curb the increasing menace of antibiotic resistance. Further studies are also required to elucidate the pathogenicity and potential roles of Enterobacteriaceae in periodontitis.

Conflict of Interest

None to declare

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