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Prevalence of oral pathogens in oral cavities, dental implants, fixed bridges among the people in South India

Bibin.G.Anand and R.Mala

Abstract
The present study was aimed to isolate most prevalent pathogens present in dental implants and to assess their antimicrobial susceptibility profile against antibiotics. The investigation consisted of 75 dental samples. 10 were implants, 17 were natural teeth as abutments for fixed bridges and 48 were with cavities. All microorganisms were identified by biochemical tests. Predominant genera prevalent in rejected implant were *Pseudomonas aeruginosa, Enterobacteria and Flavobacterium. Bacillus subtilis, Streptococcus* sp *and P. aeruginosa* were found around the cavities teeth. The susceptibility profile of the isolates to antibiotics was assessed by well diffusion method. *B. subtilis* was resistant to metrogel, cephalosporin and penicillin. All *P. aeruginosa* species were multidrug resistant. Thus the present study reveals the current status of antibiotic resistance of dental pathogens.

Keywords: Dental implants, dental pathogens and Multi Drug Resistance.

1. Introduction
Mouth represents a dynamic ecological niche. The composition of normal microbiota varies with age. Microbial flora of oral cavity is highly complex and various surfaces of normal mouth are inhabited by abundant microbial community. Oral cavity of new born baby is sterile from eight hours to first few days of life. The environment is changed by eruption of deciduous teeth. Dental surface and gingiva are colonized by *Streptococcus mutans* and *Streptococcus sanguinis* with the formation of first teeth. *Streptococci* adhere to the gums and cheek but not to the teeth (Ko¨no¨nen, 2005).Oral cavity are harboured by anaerobes as the number of teeth increases. It is also influenced by diet. During childhood, the formation of the gingival sulcus will provide a favourable habitat for anaerobic species. Besides these species, *Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans*) and *Campylobacter rectus* are found as common members of the oral microbiota of healthy children, whereas *Porphyromonas gingivalis* and *Prevotella intermedia* appear to be transient organisms. (Ooshima *et al*,2003) Formation of permanent teeth are also associated with many aerobic and anaerobic organisms. Aerobes include *Staphylococcus epidermidis*, *Neisseria* and *Diptheroids.* Anaerobe includes *Lactobacilli, Bacteroids, Actinomyces, Veillonella, and Spirochetes.* Almost 20-80% of adult has Candia species in oral cavity. The complexity of the oral microbiota increase with time (Ko¨no¨nen *et al*, 2007).With loss of teeth microbial population decreases due to an unsuitable microenvironment (Ko¨no¨nen *et al*, 1991). Bacteria attach to the surface by biofilm formation. Biofilm formation on the surface of teeth is called Plaque. A dynamic equilibrium exists between dental plaque bacteria and the innate host defense system. The number and types of microbial population depends on oxidation reduction potential, pH, diet, diurnal variation, oral hygiene and the intake of antibiotics. An array of host defence mechanisms including the flow of saliva, desquamation of oral mucosa, lysozyme, lactoferrin, secretory immunoglobulins, phagocytic cells and calcium phosphate helps to clean the oral cavity.
Dental disease is caused by the accumulation of bacterial metabolites on to the teeth and gingival tissues. Uncared plaque can turn into tartar and lead to gingivitis or periodontal disease. Proper periodontal infection control before instalment of dental implants may prevent bacterial complications (Van Winkelhoff *et al*, 2000 and, Fu¨rst, *et al*, 2007).

Although bacteria can colonize different surfaces within the oral cavity (Salvi *et al*,2008) only a few studies, as reviewed by (Sachdeo *et al*, and Pye *et al*,2009) considered the presence of periodontal pathogens in edentulous individuals. Bloodstream is sterile under normal conditions. Transient bacteremia occurs when bacteria enter the bloodstream. Bacteremia in dentistry frequently occurs following invasive procedures such as extractions and periodontal surgery (Heimdahl *et al*, 1990 and Rajasuo *et al*, 2004), non-invasive procedures such as periodontal probing (Kinane *et al*, 2005, and Daly CG *et al*, 2001), root canal treatment (Savarrio *et al*, 2005), orthodontic treatment (Erverdi *et al* 2000) and oral hygiene procedures (Bhanji *et al*, 2002 and Crasta *et al*, 2009). In a healthy person, bacteremia in the bloodstream is countered by normal defence mechanisms (Daly *et al*, 2001). Bacteraemia may cause Infective Endocarditis (IE) in patients with cardiac anomalies or in patients with a compromised immune system (Janket *et al*, 2003. Prolonged infection in the oral cavities leads to the loss of teeth and then this leads to the implantation .The dental implants are most widely used in case of teeth loss, there are various classes of dental implants the success of the implants is by the osseointegration .The time span for the osseointegration ranges from 90-180 days and this success will be based when it is under a good clinical strategy. The implant failure was due to poor oral hygiene, mechanical stress given to the implant, and smoking (Evasic R,1990 and Branemark *et al*, 1969).The microbes play a major role in implant infection the most prevalent microbes like *Streptococcus,Actinomycetes,Fusobacterium,Staphylococci ,Pseudomonas and Enterococci,* are reported by(Passariello *et al*, 1993: Rams *et al*, 1990, : Alcoforado *et al*, 1991,and Rams *et al*, 1991) Therefore, antibiotic prophylaxis is recommended for the prevention of bacteraemia in susceptible patients. Antibiotic prophylaxis aims to reduce the amount of bacteria in blood and bacteria adherence in sterile vegetations (Boyle N *et al*, 2006). Different scientific organizations have recommended various prophylactic antibiotic regimens. The guidelines suggested by the American Heart Association (AHA) and British Society of Antimicrobial Chemotherapy (BSAC) are often used. AHA and BSAC suggest prophylaxis in all procedures involving dento-gingival manipulation or endodontics (Gould *et al*, 2006; Wilson *et al* 2008 and Piñeiro *et al*, 2010). Unfortunately, there is insufficient scientific data concerning the incidence of bacteremia and types of bacteria species following the installation of dental implants (Lee *et al*, 2009). So the present study is aimed to isolate and identify dental pathogens present in rejected implants, natural teeth with cavities and abutments for fixed bridges and their sensitivity to antibiotics.

2. Methodology

2.1 Isolation of Dental Pathogens.

Dental samples were obtained from Dr. G. RathnaKumar, Rajam clinic Madurai, India. The samples consisting of infected tooth, fixed bridges and the rejected implants were collected in phosphate buffered saline. Dental pathogens were isolated by serial dilution in blood agar medium by spread plate technique.

2.2 Identification of Dental Pathogens

2.2.1. Carbohydrate Fermentation Activity

The isolates were inoculated into the tubes of glucose, sucrose and lactose broth and incubated at 37 ˚C for 24 hrs. All carbohydrate broth cultures were assessed for evidence of acid and gas production. Acid production can be observed by colour change in the medium and gas production can be observed as bubble collected in Durham tube. Negative controls were also maintained.

2.2.2. Triple Sugar Iron Test

Triple Sugar Iron (TSI) agar slant was prepared. Culture was inoculated by stabbing into the agar butt (bottom of the tube) with an inoculating wire and then streaking the slant in a wavy pattern. It was incubated for 18 to 24 hours at 35 °C and observed for changes in the butt and on the slant.

2.2.3. IMViC Tests

2.2.3.1. Indole test

Using sterile technique, each organism was inoculated in **SIM(Sulphide Indole Motility medium)** broth deep into its appropriately labelled deep tube by means of a stab inoculation and incubated at 37 °C for 24 to 48 hours. Few drop of Kovac's reagent was added and observed for change in colour.

2.2.3.2 Methyl Red Test

Using sterile technique, the organisms were inoculated into MR-VP broth tubes and incubated for 24 to 48 hours at 37°C. Few drops of methyl red indicator was added and observed for change in colour.

2.2.3.3 Voges-Proskauer Test

Using sterile technique, the organisms were inoculated into MR-VP broth tubes and incubated for 24 to 48 hours at 37 °C. Few drops of Barritt's reagent was added and observed for colour change.

2.2.3.4 Citrate Utilization Test

Using sterile technique, organisms were inoculated into Simmon's citrate agar slants and incubated for 24 to 48 hours at 37 °C. Following incubation the tubes were observed for change in colour

2.2.4. Catalase Test

The isolate was heavily inoculated into Nutrient agar slant by means of a streak inoculation and incubated at 35°C for 18 to 24 hours. Slants were set in an inclined position and several drops of a 3% solution of H_2O_2 was added over the growth on the slant and observed for the appearance of gas bubbles.

2.2.5. Oxidase Test

Single streak-line inoculation of the isolates was made on agar surface. The plates were incubated in an inverted position for 24 to 47 hours at 35 °C. 2 to 3 drops of oxidase reagent was added to the surface colonies and observed for colour change.

Isolation and identification of pathogens from Dental Samples

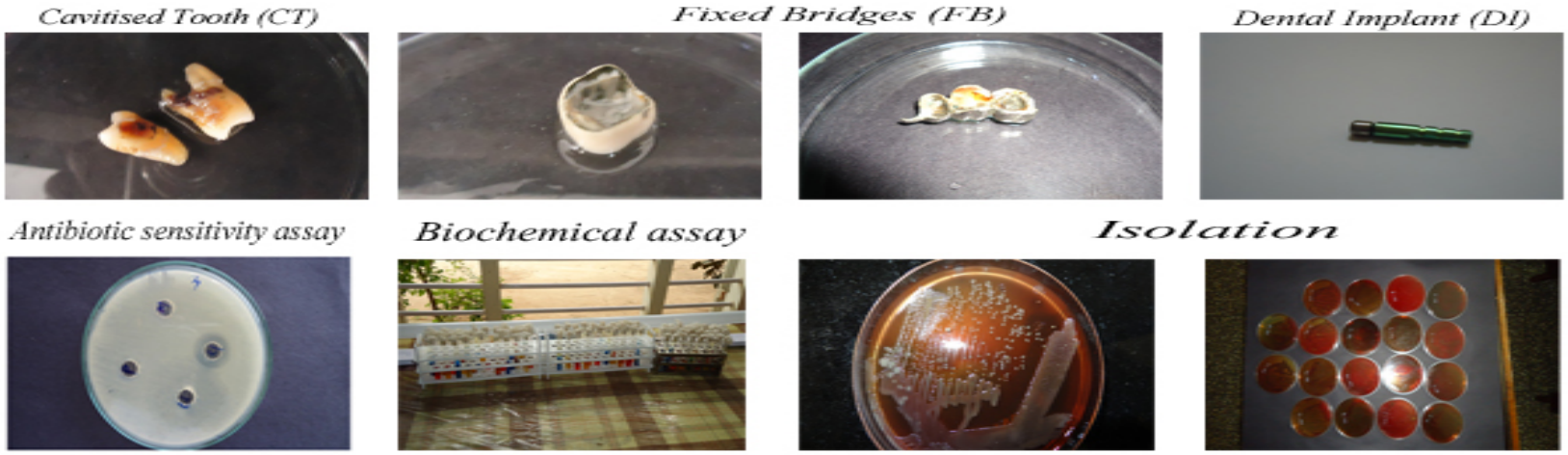

Table 1: Bio Che Test for Identification of Dental Pathogens

	Gram staining	Manitol	Motility	Glucose	Lactose	Sucrose	Hydrogen Sulphide	Gas Production	Peptone	Citrate	Oxidase	Catalase	Indole	Organism
1	G-ve Bacilli	+	+	-	+	+	-	-	-	+	+	+	+	*Pseudomonas aeruginosa sp1*
2	G-ve Bacilli	+	+	+	+	+	-	-	-	-	+	-	+	*P. aeruginosa. sp 2*
3	G+ve Bacilli	-	+	+	+	+	-	-	-	-	+	-	+	*Bacillus subtilis*
4	G-ve Bacilli	+	+	+	+	+	-	-	-	-	+	-	+	*P. aeruginosa. sp 3*
5	G-ve Bacilli	+	+	-	+	+	-	+	_	+	+	+	+	*Pseudomonas sp 4*
6	G-ve Bacilli	+	+	+	+	+	-	-	-	-	+	-	+	*Escherichia coli sp1*
7	G-ve Bacilli	+	+	+	+	+	-	-	-	-	+	-	+	*P. aeruginosa. sp 5*
8	G-ve Bacilli	+	+	+	+	+	-	-	-	-	+	-	+	*Escherichia coli sp2*
9	G+ve Cocci	+	-	+	+	+	-	-	-	+	+	+	-	*Staphylococcus albus*
10	G-ve Bacilli	+	+	+	+	+	-	-	-	-	+	-	+	*Escherichia coli sp3*
11	G-ve Bacilli	+	+	+	+	+	-	-	-	-	+	-	+	*Escherichia coli sp4*
12	G-ve Bacilli	-	-	-	-	-	-	-	-	+	+	+	-	*Flavobacterium*
13	G-ve Bacilli	+	+	-	+	+	-	+	-	+	+	+	+	*P. aeruginosa sp6*
14	G-ve Bacilli	+	+	+	+	+	+	+	+	+	+	+	-	*Enterobacter*

Table 2: Isolated and identified Micro organisms (Pathogens) from the dental samples

Sl. No	Sample Id	Organism
1	DI_1	*Pseudomonas aeruginosa sp 3, sp 4, sp 5 Enterobacteria, Flavobacterium., Escherichia coli sp1,*
2	DI_2	*P. aeruginosa. sp 4 Enterobacteria and Flavobacterium.*
3	DI_3	*Enterobacteria, Flavobacterium.*
4	DI_4	*P. aeruginosa. sp 4, sp 5, Enterobacteria and, Flavobacterium. , E. coli sp1.*
5	DI_5	*E. coli sp1 P. aeruginosa sp 1 sp6*
6	DI_6	*P. aeruginosa sp 5 sp6*
7	DI_7	*P. aeruginosa. sp 4 sp6, E. coli sp3 sp4, Enterobacteria*
8	DI_8	*P. aeruginosa. sp 1 sp2 sp6 , E. coli sp1, Enterobacteria P. aeruginosa sp 5*
9	DI_9	*P. aeruginosa .sp 2 ,Enterobacteria and Flavobacterium.*
10	DI_{10}	*P. aeruginosa. sp 1 sp2 sp3 sp5 , E. coli sp4,*
11	FB_1	*Flavobacterium. Bacillus subtilis, P. aeruginosa. sp 2 E. coli sp3*
12	FB_2	*Flavobacterium., P. aeruginosa. sp 1, sp5, sp6*
13	FB_3	*Bacillus subtilis, Streptococcus sp P. aeruginosa. sp 4 sp5, sp6*
14	FB_4	*Flavobacterium. Bacillus subtilis, Streptococcus sp P. aeruginosa. sp 6*
15	FB_5	*Flavobacterium. , Streptococcus sp P. aeruginosa. sp 3 sp5*
16	FB_6	*Flavobacterium. Bacillus subtilis, Streptococcus sp P. aeruginosa. sp 4*
17	FB_7	*Flavobacterium. Bacillus subtilis, E. coli sp1 sp3*
18	FB_8	*Bacillus subtilis, Streptococcus sp*
19	FB_9	*Flavobacterium. Bacillus subtilis, Streptococcus sp*
20	FB_{10}	*Flavobacterium. P. aeruginosa. sp 1 sp2 sp4 sp5*
21	FB_{11}	*Flavobacterium. Bacillus subtilis, Streptococcus sp*
22	FB_{12}	*Flavobacterium P. aeruginosa. sp 1*
23	FB_{13}	*Flavobacterium. Bacillus subtilis, Streptococcus sp E. coli sp1 sp3*
24	FB_{14}	*Bacillus subtilis, Streptococcus sp*
25	FB_{15}	*Flavobacterium. Bacillus subtilis, Streptococcus sp E. coli sp3*
26	FB_{16}	*Flavobacterium. Bacillus subtilis, Streptococcus sp*
27	FB_{17}	*Flavobacterium. Bacillus subtilis, Streptococcus sp P. aeruginosa. sp 4*
28	CT1	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3, sp4*
29	CT2	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
30	CT3	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3, sp4*
31	CT4	*Bacillus subtilis, Streptococcus sp and P. aeruginosa*
32	CT5	*Bacillus subtilis, Streptococcus sp and P. aeruginosa*
33	CT6	*Bacillus subtilis, Streptococcus sp and P. aeruginosa*
34	CT7	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
35	CT8	*Bacillus subtilis, Streptococcus sp and P. aeruginosa*
36	CT9	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp4*
37	CT10	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1 sp2*
38	CT11	*Bacillus subtilis, Streptococcus sp E. coli sp5 and P. aeruginosa*
39	CT12	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3, sp4*
40	CT13	*Bacillus subtilis, Streptococcus sp and P. aeruginosa*
41	CT_{14}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
42	CT_{15}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3*
43	CT_{16}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1, sp4*
44	CT_{17}	*Bacillus subtilis, Streptococcus sp P. aeruginosa E. coli sp3 and Flavobacterium.,*
45	CT_{18}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3*
46	CT_{19}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1 sp2*
47	CT_{20}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3*
48	CT_{21}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3*
49	CT_{22}	*Bacillus subtilis, Streptococcus sp E. coli sp1 and P. aeruginosa*
50	CT_{23}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3*
51	CT_{24}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
52	CT_{25}	*Bacillus subtilis, Streptococcus sp P. aeruginosa E. coli sp2 sp4 and Flavobacterium.,*
53	CT_{26}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
54	CT_{27}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2*
55	CT_{28}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3 ,sp4*
56	CT_{29}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
57	CT_{30}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp4*
58	CT_{31}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp4*

59	CT_{32}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp4*
60	CT_{33}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1 ,sp2*
61	CT_{34}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3*
62	CT_{35}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3*
63	CT_{36}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1, sp2*
64	CT_{37}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and Flavobacterium.,*
65	CT_{38}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3*
66	CT_{39}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
67	CT_{40}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3*
68	CT_{41}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
69	CT_{42}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3, sp4*
70	CT_{43}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1*
71	CT_{44}	*Bacillus subtilis, Streptococcus sp, P. aeruginosa E. coli sp2 and Flavobacterium.,*
72	CT_{45}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp3*
73	CT_{46}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2, sp3*
74	CT_{47}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp1, sp2*
75	CT_{48}	*Bacillus subtilis, Streptococcus sp P. aeruginosa and E. coli sp2*

DI-Dental Implants
FB-Fixed Bridges
CT-Cavitized Teeth

The isolated pathogens are then checked for its antibiotics resistance capacity the antibiotics used in our study are Metronidazole, Penicillin, Ciprofloxacin and cephalosporin.

Metronidazole is the widely used drugs for the treatment of anaerobic infections and the treatment of choice for most patients with mild to moderate Dental infection of bacterial origin, such as periapical abscess, periodontal abscess, acute pericoronitis of impacted or partially erupted teeth.

Penicillin is a group of antibiotics derived from Penicillium fungi the term "penicillin" is generically to refer penicillin G. All penicillins are β-lactam antibiotics and are used in the treatment of bacterial infections are caused by the susceptible, usually Gram-positive, organisms.

Ciprofloxacin a second-generation fluoroquinolone antibiotics, its spectrum activity includes most strains of bacterial pathogens including Gram (-) and Gram-(+) bacterial pathogens.

Cephalosporins are beta-lactam compounds in which the beta-lactam ring is fused to a 6-membered dihydrothiazine ring, thus forming the cephem nucleus. In general, first generation cephalosporins have better activity against gram-positive bacteria and less gram-negative activity, while third generation agents have few exceptions, have better gram-negative activity and it shows a less gram-positive activity. The fourth generation agent is active and exhibits both gram-positive and gram-negative activity.
ll the isolates were resistant to Metrogel and penicillin. Most of the isolates were resistant to cephalosporins. NCCLS standard is used to assess the sensitivity of organism is given in table 2. Antimicrobial susceptibility profile of isolates to antibiotics is given in table 3. Genes that confer resistance can be transferred between bacteria in a horizontal fashion. Antibiotic resistance evolves by natural selection. Continuous exposure to antibiotics selects for the antibiotic resistance as a process of natural selection. Many antibiotic resistance genes reside on plasmids, facilitating their transfer. So the isolates can be considered as multidrug resistant (MDR) or a superbug. Beta lactamase activity is present in *Staphylococcus* and *Pseudomonas.* Resistance to antibiotics are also developed by alternative efflux pump.
Pseudomonas spp and *Flavobacterium* were completely resistant to all the antibiotics revealing the severity of antibiotic resistance. So the present study explores the need for a new antimicrobial agent potent against dental pathogens. The susceptibility of *P. aeruginosa* to fluoroquinolones decreased after increasing use of fluoroquinolones (Pechère 2001).

Table 2: NCCLS Standards for the used antibiotics

NCCLS standards **R**-Resistant, **I**-Intermittent, **S**-Susceptible			
Ciprofloxacin 5µg	**Cephalexin**5µg	**Metrogel** 5µg	**Penicillin-** G 5µg
R-15mm	**R**-12mm	**R**-20mm	**R**-15mm
I-16-20 mm	**I**-13-15 mm	**I**-21-23 mm	**I**- 16-20 mm
S-21mm and above	**S**-16 mm	**S**-21 mm and above	**S**-28mm and above

Table 3: Antibiotic Sensitivity of the Dental Pathogens to the commonly used Antibiotics (diameter of inhibition zone in mm)

Organism	Inhibition Zone in (mm)			
	Metrogel 5µg	**Ciprofloxacin 5µg**	**Cephalexin 3µg**	**Penicillin-G 5µg**
P. aeruginosa. 1	0 (R)	41(S)	0(R)	0(R)
P. aeruginosa. 2	0(R)	31(S)	0(R)	0(R)
B. subtilis	0(R)	0(R)	0(R)	0(R)
P. aeruginosa. 3	0(R)	30(S)	13(I)	0(R)
P. aeruginosa. 4	0(R)	29(S)	13(I)	0(R)
E .coli 1	0(R)	33(S)	11(R)	0(R)
P. aeruginosa. 5	0(R)	41(S)	0(R)	0(R)
E.coli 2	0(R)	41(S)	0(R)	0(R)
S. albus	0(R)	17(I)	0(R)	0(R)
E .coli 3	0(R)	20(I)	18(S)	0(R)
E .coli 4	0(R)	31(S)	11(R)	0(R)
Flavobacterium	0(R)	31(S)	11(R)	0(R)
P. aeruginosa. 6	0(R)	19(I)	11(R)	0(R)
Enterobacter	0(R)	31(S)	22(S)	0(R)

The volume of antibiotic prescribed is the major factor in increasing rates of bacterial resistance (Costelloe *et al* 2010). A single dose of antibiotics leads to a greater risk of resistant organisms to that antibiotic in the person for up to a year. (Thomas *et al* 1998). Exposure of bacteria to suboptimal doses of antibiotics due to inappropriate dose or failure to take the prescribed dose for stipulated time contributes to the development of resistance among microbes. (Robicsek, *et al* 2006). Some types of efflux pumps can act to decrease intracellular quinolone concentration (Poole 2004).

Resistance to quinolones in Gram-negative bacteria is mediated by plasmid coded proteins which binds to DNA gyrase. Finally, mutations at key sites in DNA gyrase or topoisomerase IV can decrease their binding affinity to quinolones, decreasing the drug's effectiveness. Low antibiotic susceptibility is one of the most dangerous characteristics of *P. aeruginosa*. It is attributable to a concerted action of multidrug efflux pumps. They have, *mexAB-oprM*, *mexXY*, etc responsible for antibiotic resistance genes encoded in chromosomes. Hypermutation favours the selection of mutation-driven antibiotic resistance in *P. aeruginosa* strains, producing chronic infections, when the clustering of numerous different antibiotic resistance genes in integrons favours the concerted acquisition of antibiotic resistance determinants. (McCollister *et al* 2011).

Conclusion:

From our minor research study it can be concluded that the pathogens are the major cause for the failure of dental implants and this can be overcome by alternative antibiotics or enhancing the property of antibiotic and functionalising the dental implants. And there is a need for further investigation of alternatives

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