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ODONTOGENIC INFECTIONS

Dental Science

KEYWORDS: Odontogenic Infections, Head and Neck Infections, Infections, Microbial flora in Head And Neck Infections, Dental Abscess, Dental Infections, Antibiotic Sensitivity

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ABSTRACT

PURPOSE : To review the characteristics of head and neck infections, accumulate prospective data of the microbial flora in head and neck infections and their antibiotic sensitivity.

MATERIALS AND METHODS: A clinical trial was conducted on 90 patients with head and neck infections of odontogenic origin. Pus sample was collected from the site of infection under controlled aseptic conditions.

RESULTS: Male patients dominated over female patients, mandibular molar were commonly involved affecting submandibular, sublingual and pterygomandibular space. A total of 141 strains were isolated. Although culture and sensitivity showed resistance to penicillin group of drugs but clinically not relevant. Carbapenams had no resistance from all strains of microorganisms.

CONCLUSION: The gold standard to date in the management of odontogenic infections is removal of etiology, drainage of pus and penicillins with β- lactamase inhibitors. Fulminant infections usually require aggressive therapy.

INTRODUCTION

Invasion and multiplication of pathogenic microorganisms into body tissue produces injury and progress to manifest as infection through a variety of cellular and toxic mechanisms.[1]

Maxillofacial infections have affected the human race since a very long time. Egyptians had described signs of dental abscesses and osteomyelitis.[2] These infections may be odontogenic (pulp necrosis) or non odontogenic (tonsils, sinuses).Infection may manifest itself as periapical or periodontal abscess, cellulitis or osteomyelitis.[3,4]

The odontogenic infections are mainly caused by endogenous bacteria in the oral cavity. The most common organisms are *Streptococcus, Eubacterium, Bacteroides* and *Fusobacterium*.[5] The anaerobic bacteria are not pathogenic themselves, they are dependent on invasive bacteria like *Streptococcus, Stphylococcus* to establish an infection. *Bacteroides melaninogenicus* is the most commonly isolated anaerobic bacterium alone or in combination with *Fusobacterium, Peptococcus or Peptostreptococcus.*

The principle of management of infection was first described by Hippocrates "TO REMOVE ETIOLOGY AND DRAIN THE PUS". Discovery of penicillin significantly changed the course of treatment, but after 4 years of mass production of Penicillin around 1943 antibiotic resistant microorganism began to develop.1 To combat Penicillin resistance synthetic antibiotics have subsequently been synthesized however, resistance to even these newer drugs is fast becoming evident.[2]

This study had been designed to assess the most common microorganism causing odontogenic infection and their antimicrobial susceptibility in western Uttar Pradesh to provide guidelines for effective treatment. In this study comparison was made with other studies in context of source of infection, age, gender, site involved, micro-organisms involved and their antibiotic sensitivity.

MATERIAL AND METHODS

This is a prospective clinical trial carried out in Subharti Dental College and Hospital, Meerut, U.P., India

Inclusion criteria:

1. All patients with head and neck infections of odontogenic origin
2. Above 12 years of age
3. Infections not responding to routine antibiotics

Exclusion criteria: None

All the patients were subjected to the same clinical protocol:

1. Complete clinical and drug history of the patients were recorded
2. Sample was collected under sterile conditions

a. Pus sample was collected using a 5ml syringe and 18 gauze needle. (Fig. 1)

b. Infected tissue samples (e.g. sinus tracts) were collected using a sterile swab. (Fig. 2)

c. Infected tissue (e.g. tooth) stored and transported in a sterile normal saline bottle. (Fig. 3)

d. The samples for anaerobic culture were transported in Robertson Cooked media (RCM). (Fig. 4)

The steps involved in the processing of samples have been described in the flow chart. (Fig. 5)

Patients characteristics recorded were age, gender, facial spaces involved, micro-organisms isolated and their antibiotic susceptibility.

RESULTS

A total of 90 samples were collected, out of theses 27 were excluded as no growth was present in them and remaining 63 were included in the study. The relevant criteria/ features of the case history are summarized in Table 1.

Of these 63 patients 45 were male and 18 female (2.5:1). The age range recorded was 12 years to 75 years with majority belonging to 3rd and 5th decade of their life. 50 patients gave a positive history of antibiotic consumption ranging from 1 to 5 days before reporting for treatment at this centre. (Table 1)

Most common cause of infection was caries (71%) most commonly involving the mandibular molar teeth (67%). (Graph 1, 2)

36% of these cases presented as localized abscess and 64% had progressed to space infection, with submandibular space (25%) being most commonly involved. (Graph 3)

Samples were collected from all the patients and sent for culture and sensitivity. A total of 190 strains were isolated with a range of 1 to 3 strains per patient. (Graph 4)

According to the gram staining reports Gram Positive Cocci (58%) were most commonly identified followed by Gram Negative Bacilli (38%) and Gram Negative Cocci (4%). No Gram positive Bacilli were seen.

Most commonly isolated aerobic organism was *Coagulase Negative Staphylococcus* (12.1%) followed by *Streptococcus viridans* (9.2%), *Streptococcus species* (7.8%), *Staphylococcus species, Escherichia coli* (E. coli) {Extended Spectrum β lactamase (ESBL) positive} (7.1%), *Pseudomonas aeruginosa and Klebsiella species* (6.4%), Methicillin resistant *Staphylococcus aureus (MRSA)* (5.7%), *Enterococcus faecalis* (4.9 %), Methicillin sensitive *Staphylococcus aureus (MSSA), Citobacter ferundii* (4.3%) and α- *Hemolytic Streptococci, Citobacter species, Acinobacter species, Enterococcus species, Klebsiella oxytoca* (ESBL Positive), *Klebsiella pneumonae, Morganella morgani* (3.5%). (Graph 4)

The commonly isolated anaerobic organisms are Peptoniphilus assachrolyticus (5%), Peptostreptococcus Anaerobius (20%), Parvimonas micra (5%), Vellionella species (5%), Provetella species (70%), Porphyromonas species (50%), Bacteroids species 6%), Fusobacterium species (12%). (Graph 5)

In the culture sensitivity reports obtained it was observed that *Streptococcus viridans* showed 100% sensitivity to Aminopenicillin with β- lactamase inhibitors, fourth generation Cephalosporins, third generation Cephalosporins with β- lactamase inhibitors, Aminoglycosides, Glycopeptides and Oxazolidinone. 69.2% sensitivity to third generation Cephalosporins. 100% resistance to Cotrimoxazole.

Streptococcus species [illegible]- lactamase inhibitors, fourth generation Cephalosporins, Glycopeptides and Oxazolidinone. 72.7% sensitivity to Aminoglycosides and Nitrobenzene derivatives. 72.7% resistance to Aminopenicillin and third generation Cephalosporins. 100% resistance to narrow spectrum Penicillin and Cotrimoxazole.

Staphylococcus species showed 70% sensitivity to Nitrobenzene derivatives. 50% sensitivity to Aminopenicillin, first generation Fluoroquinolones.100% resistance to Aminopenicillin with β-lactamase inhibitors, Cotrimoxazole and Ureidopenicillin.

MSSA showed 100 % sensitivity to Aminopenicillin with β- lactamase inhibitors, third and fourth generation Cephalosporins, Carbapenam and Oxazolidinone. 50% sensitivity to Aminopenicillin, Macrolides, Glycopeptides and Clindamycin.100% resistance to narrow spectrum Penicillin.

MRSA showed 100% sensitivity to Glycopeptides, Carbapenam and Oxazolidinone. 50% sensitivity to Aminopenicillin with β- lactamase inhibitors, fourth generation Cephalosporins and Macrolides.62.50% resistance to Clindamycin and Aminoglycosides.100% resistance to narrow spectrum Penicillin and second generation Cephalosporins.

Coagulase Negative Staphylococcus showed 100% sensitivity to Carbapenam, first generation Fluoroquinolones, Aminoglycosides, Glycopeptides and Oxazolidinone. 76.5% sensitivity to Clindamycin. 70.6% to Macrolides. 100% resistance to narrow spectrum Penicillin and fourth generation Cephalosporins.

E Coli showed 100% sensitivity to Carbapenam and Nitrofurantoin. 50% sensitivity to Ureidopenicillin.100% resistance to Aminopenicillin with β- lactamase inhibitors, Cephalosporins, Macrolides and Clindamycin.

Pseudomonas aeruginosa showed 100% sensitivity to Ureidopenicillin, Aminoglycosides, Fluoroquinolones and Carbapenam. 55.6% resistance to third generation Cephalosporins with Sulfbactum. 100% resistance to Aminopenicillin with β-lactamase inhibitors and second generation Cephalosporins.

Most of the bacteria showed sensitivity to Carbapenam followed by Aminoglycosides, Oxazolidonone and Glycopeptides and resistance to Aminopenicillin with β lactamase, Cephalosporins and Fluoroquinolones.

DISCUSSION

In this study the mean age of 38.7 + 16.7 years was recorded with maximum number of patients belonging to the third and fifth decade. Kohli at al[3] reported an age range of 10 – 70 years with maximum number of patients belonging to the third and fourth decade,3 Yuvaraj et al [6] reported that maximum number of patients belonged to the second and third decade of their life ranging from 4 – 73 years and a mean of 33 +14 years.[6]

Caries (71.1%) was the most common cause of infection which correlates with various other studies (65%).[7,8] Sanchez et al[8] (33.8%) observed dental caries as the most frequent cause of infection followed by post extraction infectious process and pericoronitis.[8]

Mandibular third molar (27.8%) was the most commonly involved tooth which was consistent with the findings of Storoe et al,[9] Flynn et al[10] (68%). Some studies reported mandibular 1st molar (33.2%) to be the most common offending tooth.[11,12] Some reported the highest incidence involving lower second and third molar.[13] Common involvement of the mandibular molar teeth may be due to difficulty in maintenance of oral hygiene due to poor accessibility and difficulty in restorative treatment.

Submandibular space was the most commonly involved space (35.4%), this finding correlates with various other studies also.[11-15] Flynn et al[10] however observed that the most frequently involved space was Pterygomandibular followed by Submandibular, Lateral Pharyngeal and Buccal.[10] Gupta et al[16] observed that the most commonly infected space was Vestibular followed by Submandibular and Buccal space.[16]

Single space involvement was more commonly seen than multiple space involvement in our study. Multiple space involvement may be related to ignorance, fear of dental treatment or lack of adequate dental facilities.

The pathogenic microbiota of the oral cavity is complex and fluctuates with age, disease and conditions. Studies indicate majority of infections consist of mixed aerobic and anaerobic flora (65-70%) or are exclusively anaerobic whereas only 5% are exclusively aerobic.[3] The microbial flora of these infections is typically polymicrobial with an average of 2.2 to 6.1 strains per specimen.[17] In our study a total number of 141 strains were isolated with a mean of 2.3 + 0.3 strains per patient similar to 2.6 isolates per specimen,2 2.2 strains per swab[18,19] but was less when compared to 4 strains per abscess,[20] 5.5 strains per swab.[21]

The most commonly isolated organism in our study Coagulase Negative *Staphylococcus* (12.1%) followed by *Streptococcus viridans* (9.2%), *Streptococcus sps* (7.8%), *Staphylococcus sps, Escherchia coli* (E. Coli) (7.1%) in contrast to the other studies carried out in 2012 where *Streptococcus sanguis, Strptococcus mitis, Enterococcus faecalis* and β-hemolytic Streptococci were frequently isolated strains. [22] α-

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hemolytic Streptococci was most commonly isolated in a few studies.[9,13,14] Streptococcus viridans was most commonly isolated in some studies.[2,23]

Maxillofacial infections are polymicrobial in nature. Both anaerobic and aerobic bacteria are commonly isolated. It is very rare that only aerobic bacteria are isolated. But, anaerobic bacteria are not the pathogenic bacteria. They are dependent on aerobic bacteria for inoculation and propogation of infection. *Staphylococci* produces enzyme coagulase which results in abscess formation. *Streptococci* produces enzymes streptokinase, streptodornase, hyaluronidase, these enzymes break down fibrin and connective tissue and lyse cellular debris resulting in cellulitis.[24]

Staphylococcus Spesies have showed 100% resistance to Aminopenicillin with β-lactamase inhibitors, Cotrimoxazole and Ureidopenicillins. Rega et al[2] also observed poor susceptibility to Penicillin (27.3%) and Ampicillin (41.2%).[2]

MRSA was found resistant to narrow spectrum penicillin and Second generation Cephalosporins and Amoxicillin. Kohli et al also recorded resistance to Amoxicillin and Ampicillin.[3]

Staphylococcus aureus is a virulent organism and rapidly developing resistance to various antibiotics. Vancomycin is recorded as the antibiotic of last resort for this bacteria but Vancomycin resistant S. aureus has also been reported.[9]

E. Coli has become resistant to front line antibiotics including third generation Cephalosporins, Aminoglycosides and even Quinolones.25 In our study they were found resistant to Aminopenicillin with β-lactamase inhibitors, Cephalosporins, Macrolides and Clindamycin.

Pseudomonas aeruginosa has also been found resistant to Aminopenicillin with β-lactamase inhibitors and Second generation Cephalosporins. Ciprofloxacin and Imipenem are effective anti Pseudomonas drugs.[26]

Wraneke[23] in 2008 reported Moxifloxacin and Amoxicillin with Clavulinic acid to be the most potent antibiotic with Clindamycin and Doxycycline showing moderate effect and Penicillin showing the least activity in vitro.[23] Sobottka et al[27] reported susceptibility of bacteria to Amoxicillin with Clavulinic acid, Moxifloxacin and Levofloxacin.[27] In our study bacteria were found susceptible to Carbapenems, Teicoplanin, Linezolid, Gatifloxacin and 33% sensitivity to Amoxicillin with Clavulinic acid.

A note on changing microbial flora and antibiotic resistance:

Over the years due to advent of various antibiotics there has been significant improvement in the morbidity and mortality due to infectious diseases. But the bacteria also have adapted to the change. As mentioned by Neu[22] the development of resistant strains maybe due to

1. Chromosomal mutation
2. Inductive expression of a latent chromosomal gene
3. Exchange of genetic material through transformation, tranduction or conjugation.
4. Transpons (jumping genes)

The antibiotic agents are rendered inactive by:

1. Destruction or modification of the drug.
2. Prevention of access to the target.
3. Alteration of antibiotic target site.[22]

Head and neck infections are polymicrobial in nature thus the virulent organisms, organisms resistant to commonly employed antimicrobial agents and those present in greatest numbers need our attention [28]. It is important to note that due to interdependent, synergestic and mixed nature of infection, if even one bacterial species is sensitive to penicillin, it may render the entire pathogenic flora apathogenic.[6]

As mentioned by Kohli et al[3] following factors should be considered while selecting an antibiotic for head and neck infections:

1. Pateint's immune status and underlying systemic condition.
2. Severity of infection
3. Commonly encountered microbes
4. Pharmacokinetic properties of the drug
5. Drug allergies.[3]

The first principle of management of infection remains surgical drainage of abscess (Incision and Drainage or Debridement), removal of etiology. Decompression eases the pressure, improves vascularity of the area and obviates the use of antibiotics or increases the effectiveness of the antibiotics as vascular supply is restored.

CONCLUSION

The culture sensitivity test showed resistance to Amoxicillin and Amoxicillin with Clavulanic acid, clinically the patient still responded well to the drugs giving some hope that the orofacial infections do respond for common antibiotic therapy. In fulminant infections not responding to Penicillin, the drug of choice which may be considered is Carbapenem as we found no resistance to the drug.

Thus from this study it can be concluded that non-scientific prescription of antibiotics should be discouraged as a first step to prevent the fast emerging resistant strains. The gold standard to date for space infection treatment is incision and drainage and Penicillin with β- lactamase inhibitors. We suggest educative programmes for dental health providers regarding antibiotic use, conduct long term and large sample study along with refinement in culturing technique to arrive at bacterial database and their drug sensitivity. Adapt a healthy life style with appropriate balanced diet routine dental check-ups to avoid severe Oro- facial infections due to dental and periodontal pathology.

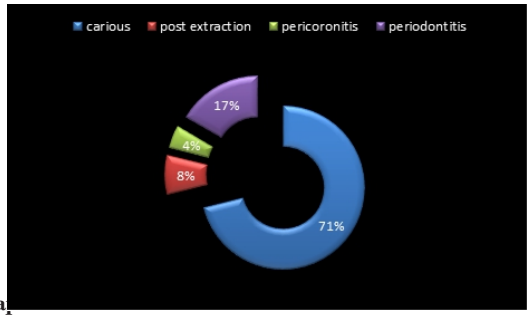
TABLES

Table 1: CASE HISTORY OF SIGNIFICANCE

PRESENTATION	Number of cases	PERCENTAGE OF CASES
PAIN	63	100%
SWELLING	45	71.4%
TRISMUS	19	30.2%
DYSPHAGIA OR DYSPNEA	13	20.6%
IMMUNOCOMPROMISED STATUS		
DIABETES	2	3.3 %
PREDNISOLONE THERAPY	1	1.6 %
PREVIOUS ANTIBIOTIC HISTORY		
CEPHALOSPORINS	10	15.9 %
AMINOPENICILLINS	20	31.7%
NITROMIDAZOLE	16	25.4 %
QUINOLONES	4	06.3%

Graphs

Graph 1 Etiological factors



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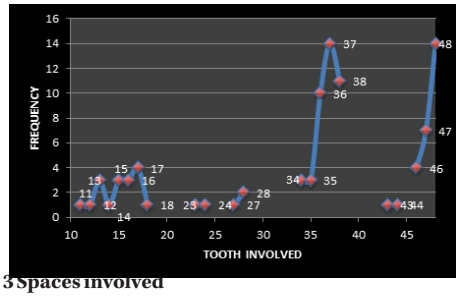
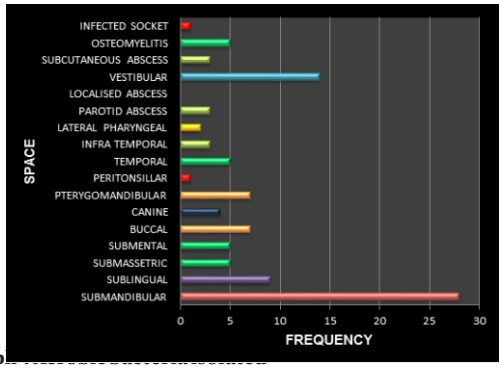


Graph 3 Spaces involved



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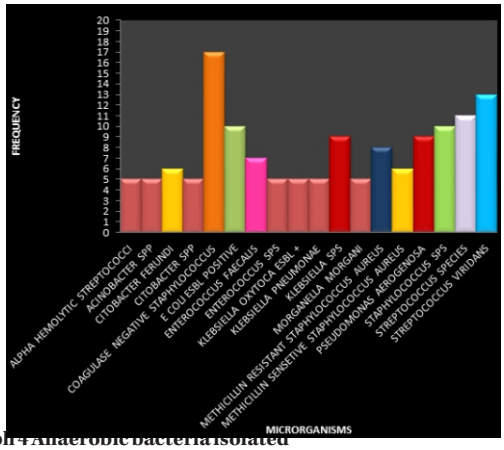

Graph 4 Anaerobic bacteria isolated

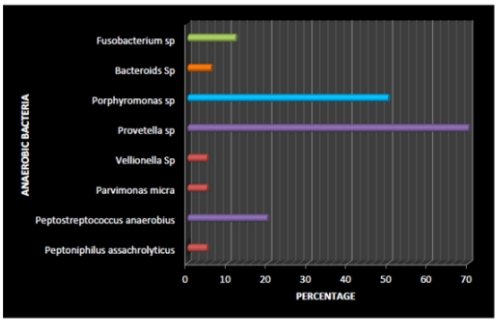

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Fig. 1 Sample collection

Fig. 1a Extraoral aspiration

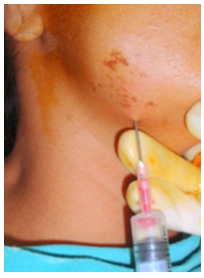


Fig. 1b Intraoral aspiration

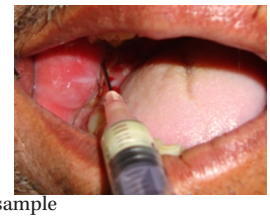


Fig. 1c Collected sample

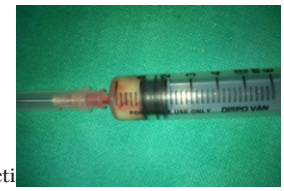


Fig. 2 Swab Collecti

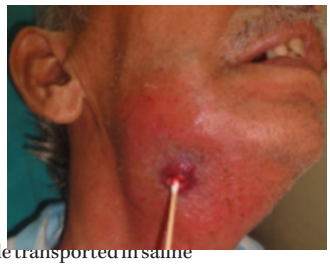
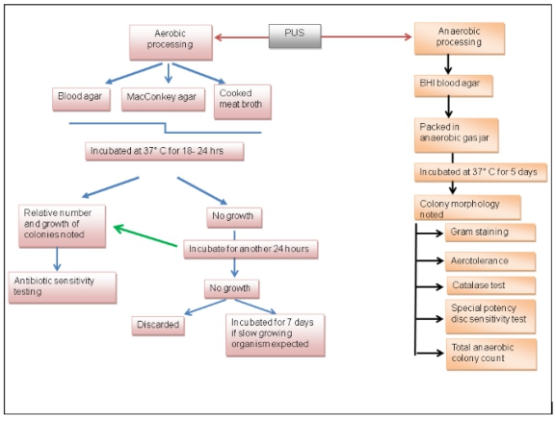


Fig. 3 Tissue sample transported in saline

Fig. 4 Sample transported in RCM

Fig. 5 Steps in the processing of the sample



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