Letter to the Editor

Are we under-estimating basic first line drug regimes of beta-lactam antibiotics clindamycin and metronidazole in dental oral and maxillofacial infections?

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Dear Sir,

Dental, oral and maxillofacial clinicians are involved in the treatment of infections on day to day basis. In spite of drastic advances in technology for creating the complex antibiotic molecules, we still continue to face the challenge to fight the microbes involved in the infections of the oral and maxillofacial region. Laskin and Strauss, correctly emphasize "the impressive ability of bacteria and viruses to adapt, change, and mutate in response to our pharmacologic bombardment is a testimony to the complex, surreptitious, and unpredictable nature of these small yet hardy microbes".[1] As we change, alter, extract or create newer antibiotic molecules, the microbes change or alter their genetic makeup to combat and survive these drugs. We as researches, bearing in mind to win, keep our battle fields as agar plates and side line the clinical outcome of these maxillofacial infections with conventional or older drugs. A single microbe showing resistance to an antibiotic on a culture media plate, instigates to discover a drug that would kill and destroy. With these *in vitro* thoughts, we most often tend to forget that maxillofacial and odontogenic infections have a mixed microflora, and there exists a concept of co-aggregation,[2] pathogenic synergy,[3] and interdependency among these microorganisms.[4] In an interesting study on subcutaneous abscesses in mice demonstrating mortality based on synergistic effect of bacteroides, Clostridium, Fusobacterium, anaerobic cocci, and aerobic bacteria, it was concluded that synergistic potential between various bacterial strains increases mortality, than when compared to individual bacterial strain.[5] With this clinical evidence in hand, culturing a single isolate on an agar plate in an aerobic environment and testing the antibiotic sensitivity for this individual bacterial stain, should not be considered a valid data to use higher or newer antibiotics in maxillofacial infections. There have been reports based on *in vitro* studies on increasing antibiotic susceptibility to drugs in quinolone group and other synthetic broad spectrum antibiotics,[6] which remain unnecessary in primary empirical therapy for maxillofacial infections. Chunduri, *et al.*, have demonstrated higher antibiotic sensitivity to amoxicillin, amoxicillin/clavulanate and clindamycin, thus providing an evidence based guide to continue using these first line drugs for empirical therapy.[7] Apart from beta-lactam, clindamycin, a drug promoted for prophylaxis for endocarditis in cases of penicillin allergy, considering the spectrum and susceptibility of the bacteria species involved in dental infections, would also be an effective drug in maxillofacial infections currently. In addition to its anti-infective properties, clindamycin has high oral absorption, significant tissue penetration, including penetration into bone, and stimulatory effects on the host immune system. Short term treatment regimes as required for oral and maxillofacial infections are usually spared with complications associated with clindamycin.[8] Löfmark, *et al.*, reported metronidazole as a standard for anaerobic infections due to its cost effectiveness, generally good activity and lower resistance against pathogenic anaerobic bacteria, favourable pharmacokinetic and pharmacodynamic properties, and minor adverse effects.[9] In a recent study on microfloral senstivity and clinical outcomes in maxillofacial infections, sensitivity to penicillin was 81.3%. The resistance shown by Staphylococcus to Penicillin group did not show any descent in the patients' clinical course. The following reasons were attributed by the authors for a better clinical outcome in spite resistance: 1. Most maxillofacial infections are mixed in nature, 2. In interdependent, synergistic mixed infections, one bacterial species sensitive to penicillin may render the entire pathogenic complex non-pathogenic, 3. *In vitro* resistance does not necessarily infer *in vivo* resistance, particularly in mixed infections, 4. The source of infection had usually been removed and/or surgical incision and drainage accomplished, which, even without antibiotics, can effect a cure.[4]

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