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Antibiotic resistance in oral and maxillofacial surgery - A comprehensive review

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Abstract

Antibiotic resistance has emerged as a major challenge in oral and maxillofacial surgery (OMFS), affecting the management of odontogenic infections, postoperative complications and reconstructive procedures. The increasing prevalence of multidrug-resistant pathogens, combined with the overuse and misuse of empirical broad-spectrum antibiotics, has significantly reduced the effectiveness of conventional antimicrobial protocols. Odontogenic infections are predominantly polymicrobial, involving viridans group streptococci, Prevotella, Porphyromonas, and Fusobacterium species, whereas non-odontogenic infections increasingly demonstrate virulent nosocomial pathogens such as Klebsiella pneumoniae, Acinetobacter baumannii, methicillin-resistant Staphylococcus aureus, and Pseudomonas aeruginosa. Resistance mechanisms including β-lactamase production, horizontal gene transfer, ribosomal target modification, and biofilm-mediated phenotypic tolerance have further complicated treatment outcomes.

This review discusses the evolving microbial spectrum, current resistance patterns of commonly used antimicrobials and the molecular mechanisms responsible for treatment failure in OMFS. In addition, major areas of antimicrobial misuse including prolonged perioperative prophylaxis, unnecessary prescriptions for low-risk outpatient procedures and inaccurate penicillin allergy labeling, are critically analyzed. The review also highlights evidence-based antimicrobial stewardship strategies such as single-dose perioperative prophylaxis, penicillin de-labeling protocols, culture-guided therapy, and continuous prescription audits. Appropriate integration of surgical intervention with rational antibiotic use remains essential to reduce resistance, improve clinical outcomes, and preserve the long-term efficacy of antimicrobial therapy in modern oral and maxillofacial surgery.

Keywords: Antibiotic resistance, oral and maxillofacial surgery, antimicrobial stewardship, odontogenic infections, biofilm

Introduction

The oral cavity is one of the most anatomically complex and microbially dense ecosystems in the human body, containing diverse bacterial, viral and fungal communities organized within structured biofilms [1]. Under normal conditions, this commensal microbiota maintains a balanced relationship with the host immune system. However, pathological triggers such as advanced dental caries, pulpal necrosis, periodontal destruction, trauma, and surgical interventions can disrupt this equilibrium and transform resident microorganisms into opportunistic pathogens. In Oral and Maxillofacial surgery, this microbial reservoir plays a central role in postoperative infections and deep fascial space infections of the head and neck [2].

The management of maxillofacial infections has traditionally depended on two complementary strategies: prompt surgical intervention and adjunctive empirical antimicrobial therapy. Surgical procedures such as incision, drainage, decompression of fascial spaces and debridement of infected hard or soft tissues remain the gold standard for reducing bacterial load and improving the local tissue environment [3]. Systemic antibiotics provide important supportive protection by limiting regional spread to critical anatomical spaces, preventing systemic sepsis, and reducing infectious failure of bone grafts, dental implants, and microvascular free flaps [4].

However, the widespread and prolonged use of antibiotics has created a major clinical concern. Defensive prescribing, extended perioperative prophylaxis, and unnecessary broad-spectrum antibiotic use for routine outpatient procedures have placed continuous selective pressure on the oral microbiome [5]. This has accelerated the emergence and expansion of antibiotic-resistant traits among common oral pathogens. As a result, OMFS clinicians increasingly encounter multidrug-resistant organisms and virulent nosocomial pathogens that reduce the effectiveness of conventional empirical protocols [6]. This resistance burden increases the risk of treatment failure, prolonged hospitalization, critical-care complications, salvage surgical procedures, and greater healthcare costs [6].

This review evaluates the evolving microbial spectrum in OMFS infections, discusses the molecular and clinical mechanisms underlying antimicrobial resistance, and outlines evidence-based antimicrobial stewardship strategies required to preserve antibiotic efficacy in contemporary oral and maxillofacial surgery.

Evolving Pathogenic Spectrum in OMFS Infections

Maxillofacial infections are broadly classified into odontogenic and non-odontogenic infections. Recent longitudinal studies demonstrate distinct microbial profiles and evolving resistance patterns within both categories [7].

Odontogenic Infections

Odontogenic space infections commonly present as mixed anaerobic–aerobic polymicrobial infections. The

predominant microorganisms include viridans group streptococci (VGS), particularly Streptococcus salivarius and Streptococcus mitis groups along with Gram-negative obligate anaerobes such as Prevotella intermedia, Porphyromonas species and Fusobacterium species [8].
Within these infections, VGS initially consume local oxygen, thereby reducing the oxidation–reduction potential of the tissue environment. This favors the proliferation of obligate anaerobes such as Prevotella and Fusobacterium, facilitating rapid spread through deep fascial spaces and promoting tissue necrosis [9].

Non-Odontogenic Infections
Non-odontogenic infections differ significantly from the normal oral microbial profile and are increasingly associated with virulent aerobic pathogens. Recent surveillance studies have demonstrated a post-pandemic increase in nosocomial pathogens among hospitalized patients, particularly Klebsiella pneumoniae, Acinetobacter baumannii, Staphylococcus aureus (including MRSA strains) and Pseudomonas aeruginosa [10, 11].
These pathogens are frequently observed in patients requiring prolonged intensive care, undergoing complex microvascular free-flap reconstruction, or presenting with compound maxillofacial fractures exposed to the external environment.

Current Resistance Patterns of Commonly Used Antimicrobials in OMFS

Antibiotic Class / Agent	Historical Role in OMFS	Observed Resistance Status and Clinical Reality
Penicillin G/V, Aminopenicillins (e.g., Amoxicillin)	Standard empirical first-line therapy for odontogenic infections	Resistance rates frequently exceed 30–40%, primarily because of β-lactamase production by anaerobic bacilli (Schmid, 2026). Resistance as high as 100% has been reported in peri-implantitis-associated biofilms.
Aminopenicillins + β-lactamase Inhibitors (e.g., Co-amoxiclav)	Escalated empirical therapy for severe odontogenic and fascial space infections	Remains highly effective, with most surveillance studies reporting resistance rates below 10%.
Lincosamides (e.g., Clindamycin)	Traditional alternative for patients with reported penicillin allergy	Significant reduction in efficacy has been observed. Clindamycin resistance ranges from approximately 30% to 82.3% in post-pandemic odontogenic cohorts.
Nitroimidazoles (e.g., Metronidazole)	Anaerobic coverage, commonly combined with β-lactams	Demonstrates good activity against deep fascial anaerobic infections; however, emerging resistance has been identified in implant-associated biofilms.
Cephalosporins (e.g., Cefazolin, Ceftriaxone)	Perioperative prophylaxis and broader Gram-negative coverage	Generally maintain favorable susceptibility profiles, although increasing levofloxacin resistance and carbapenem-resistant Acinetobacter baumannii (CR-AB) strains have been reported globally.

Molecular Mechanisms of Resistance
The increasing loss of efficacy of commonly used antimicrobials in oral and maxillofacial surgery is mediated by several molecular mechanisms operating at genetic, cellular, and biofilm levels [12]. Understanding these mechanisms is essential for improving antimicrobial selection and developing targeted therapeutic strategies.

1. Beta-Lactamase Production and Horizontal Gene Transfer
Historically, Gram-negative obligate anaerobes such as Prevotella species (P. intermedia, P. melaninogenica) and Porphyromonas species were highly susceptible to aminopenicillins such as amoxicillin. However, prolonged antibiotic exposure has promoted the emergence of β-lactamase-producing strains [13].

Amoxicillin + Beta-Lactamase → Hydrolyzed Penicilloic Acid (Inactive)

Resistance is primarily mediated by the cfxA gene family (cfxA2, cfxA4, and cfxA5), which encode Class A β-lactamases. These enzymes hydrolyze the β-lactam ring of penicillin molecules, converting active amoxicillin into inactive penicilloic acid and preventing binding to penicillin-binding proteins (PBPs) [14]. The cfxA genes are commonly located on mobile genetic elements such as plasmids and conjugative transposons, facilitating rapid horizontal gene transfer. Within polymicrobial infections, β-lactamase-producing Prevotella species may create a localized "protective umbrella," thereby protecting susceptible organisms such as viridans group streptococci [15].

2. Ribosomal Target Alteration and the MLSB Phenotype
Clindamycin has traditionally served as an alternative antibiotic in penicillin-allergic patients because of its excellent bone penetration and anaerobic coverage. However, increasing resistance has significantly reduced its clinical utility [16].
Resistance is mainly associated with erythromycin ribosome methylation (erm) genes, including erm (A), erm(B), and erm(C), which encode methyltransferase enzymes. These enzymes methylate adenine residue A2058 within the 23S rRNA of the 50S ribosomal subunit, altering the antibiotic-binding site and preventing stable clindamycin attachment [17]. Because macrolides, lincosamides and streptogramin B antibiotics share overlapping ribosomal binding sites, this mechanism produces the Macrolide-Lincosamide-Streptogramin B (MLSB) resistance phenotype, resulting in broad cross-resistance and limited oral treatment options [17].

3. Biofilm Shielding and Phenotypic Tolerance (Persister Cells)
In chronic maxillofacial infections such as osteomyelitis, osteoradionecrosis, and peri-implantitis, microorganisms exist within highly organized biofilms rather than free-floating planktonic forms. Biofilm bacteria secrete an extracellular polymeric substance (EPS) matrix composed of exopolysaccharides, extracellular DNA, proteins, and lipids. This matrix acts as a physical and chemical barrier that limits antibiotic diffusion into deeper biofilm layers [18]. As biofilms mature, oxygen and nutrient depletion create metabolic gradients that induce bacterial dormancy.

This starvation response, mediated by signaling molecules such as guanosine tetraphosphate (ppGpp), promotes formation of dormant persister cells. Since most conventional antibiotics target active bacterial metabolism, these dormant cells demonstrate marked phenotypic tolerance and may survive antibiotic concentrations significantly higher than planktonic bacteria. Following cessation of therapy, persister cells may reactivate and contribute to chronic infection recurrence and implant or hardware failure in OMFS [19].

Critical Areas of Antimicrobial Misuse in Maxillofacial Surgery

The increasing burden of antibiotic resistance (ABR) in oral and maxillofacial surgery (OMFS) is strongly associated with long-standing clinical prescribing practices that continue despite limited supporting evidence. These practices contribute significantly to the emergence of multidrug-resistant organisms in both outpatient and inpatient settings [20].

1. Prolonged Perioperative Surgical Antibiotic Prophylaxis (SAP)

One of the major contributors to institutional resistance is the unnecessary continuation of surgical antibiotic prophylaxis into the postoperative period.

The "Single-Shot" Paradigm

The primary objective of surgical antibiotic prophylaxis is to maintain tissue drug concentrations above the minimum inhibitory concentration (MIC) of expected pathogens at the time of incision and throughout wound closure. A single preoperative dose administered within 60 minutes before surgery is generally sufficient for routine maxillofacial procedures [21].

The Fallacy of Postoperative Shielding

Extending antibiotic prophylaxis for several postoperative days in clean-contaminated procedures such as orthognathic surgery, cystectomy, or uncomplicated trauma fixation provides minimal additional protection against surgical site infections (SSIs) [22]. Instead, prolonged antibiotic exposure promotes ecological dysbiosis by eliminating normal commensal flora and creating favorable conditions for multidrug-resistant Gram-negative organisms, vancomycin-resistant enterococci (VRE) and Pseudomonas aeruginosa colonization [23]. Suppression of normal gut microbiota may also facilitate Clostridioides difficile overgrowth, resulting in pseudomembranous colitis, dehydration, and prolonged hospitalization.

2. Inappropriate Prophylaxis for Low-Risk Outpatient Procedures

Systemic antibiotics are frequently overprescribed in routine outpatient dentoalveolar procedures performed in healthy individuals.

Third Molar Extractions and Minor Biopsies

Randomized clinical trials have demonstrated that routine postoperative antibiotic administration following uncomplicated third molar surgery, alveoloplasty, or minor soft-tissue biopsy procedures does not significantly reduce the incidence of alveolar osteitis or postoperative infection in healthy patients. Instead, unnecessary antibiotic use increases the risk of hypersensitivity reactions, gastrointestinal adverse effects, and selection of resistant microbial strains [24, 25].

The Dental Implant Exception

Primary dental implant placement represents a unique evidence-based indication for antibiotic prophylaxis. Because titanium implants initially lack a vascular supply capable of delivering immune cells, they are particularly susceptible to early bacterial colonization during placement. Current evidence supports administration of a single preoperative 2 g oral dose of amoxicillin, or 600 mg of clindamycin/azithromycin in penicillin-allergic patients, approximately 1 hour before surgery. This protocol significantly reduces the risk of early implant failure [26].However, extending antibiotic therapy into the postoperative period does not provide additional benefits for implant integration and therefore remains unjustified.

3. The Penicillin Allergy Over-Labeling Crisis

A major challenge in OMFS is the high prevalence of unverified penicillin allergy labels among patients.

Reality of Allergy Over-Reporting

Although nearly 10% of patients report penicillin allergy, formal immunological evaluation demonstrates that more than 90% can safely tolerate β-lactam therapy. Many reported allergies are related to childhood viral exanthems or transient adverse effects rather than true IgE-mediated hypersensitivity [27].

Clinical Consequences in OMFS

Unverified penicillin allergy labels frequently force clinicians to avoid first-line β-lactam therapies such as amoxicillin/clavulanic acid or cefazolin and instead prescribe clindamycin or macrolides. This practice contributes to increased treatment failures because clindamycin resistance associated with the MLSB phenotype and erm-mediated resistance has reached up to 82.3% in certain odontogenic and viridans streptococcal isolates [28]. In addition, clindamycin use is strongly associated with Clostridioides difficile overgrowth and gastrointestinal complications. Consequently, de-labeling low-risk patients before surgery has become an important component of antimicrobial stewardship in OMFS.

Critical Areas of Antimicrobial Misuse in OMFS

Several deeply embedded prescribing practices continue to accelerate antibiotic resistance within the specialty.

Prolonged Perioperative Surgical Prophylaxis (SAP)

Extension of surgical prophylaxis into the postoperative period remains a major driver of institutional resistance. Although a single pre-incisional dose effectively minimizes surgical site infections by preventing intraoperative contamination, prolonged postoperative administration offers no additional therapeutic advantage. Instead, it promotes colonization by multidrug-resistant Gram-negative organisms, Clostridioides difficile, and vancomycin-resistant enterococci (VRE) [29].

Inappropriate Prophylaxis for Low-Risk Interventions

Routine antibiotic administration for minor outpatient procedures continues despite international recommendations

against this practice. Clinical evidence demonstrates that healthy patients undergoing uncomplicated third molar extractions, minor dentoalveolar procedures, or soft-tissue biopsies derive minimal benefit from prophylactic antibiotic coverage. In contrast, primary dental implant placement remains a specific evidence-based indication for a single preoperative dose of amoxicillin [5]. Postoperative extension of therapy in uncomplicated cases remains unjustified.

Strategic Frameworks for Antimicrobial Stewardship (AMS)

To address rising antimicrobial resistance and align treatment practices with precision medicine, oral and maxillofacial surgery departments must implement structured antimicrobial stewardship (AMS) programs.

1. Re-evaluation of Prophylactic Protocols

Clinical protocols should transition from prolonged postoperative antibiotic administration to strict single-dose perioperative prophylaxis for routine major surgeries. Extended antibiotic coverage should be reserved only for selected high-risk procedures such as complex microvascular free-flap reconstruction or contaminated multifragment facial trauma cases [30].

2. Implementation of Penicillin De-labeling Models

Risk-stratification tools such as the PEN-FAST scoring system should be incorporated into preoperative assessment protocols.

PEN-FAST Score = Penicillin allergy history (<5 years ago) × 2 + Anaphylaxis/Angioedema × 2 + Severe Cutaneous Reaction × 2

Patients with low-risk scores (<3) may safely receive first- or second-generation cephalosporins, such as cefazolin, rather than being unnecessarily shifted to high-resistance alternatives such as clindamycin [31].

3. Active Audit, Feedback, and Culture Submission

Empirical broad-spectrum antibiotic therapy should not be solely relied upon in hospitalized fascial space infections. Tissue samples or purulent aspirates should be collected under aseptic conditions before initiating antimicrobial therapy. Continuous monitoring of institutional Defined Daily Doses (DDD), combined with regular audit and feedback programs, has been shown to improve prescribing practices and reduce unnecessary antibiotic consumption. [32]

Conclusion

Antibiotic resistance has become a major challenge in oral and maxillofacial surgery because pathogens increasingly demonstrate enzyme-mediated resistance, target-site modification, and biofilm-associated protection, thereby reducing the effectiveness of conventional antimicrobial therapy. Management of severe maxillofacial infections continues to depend primarily on prompt surgical intervention, including incision, drainage, decompression, and debridement, while antibiotics should serve only as adjunctive supportive therapy. Inappropriate prescribing practices such as prolonged postoperative prophylaxis and unnecessary antibiotic use for low-risk procedures contribute significantly to ecological dysbiosis, gastrointestinal complications, and the emergence of multidrug-resistant organisms. Preservation of antimicrobial efficacy therefore requires evidence-based stewardship strategies, including microbiological culture testing, prescription auditing, rational perioperative prophylaxis, and verification of reported penicillin allergies. Integration of precise surgical management with responsible antimicrobial use remains essential for improving patient outcomes and reducing the resistance burden in contemporary oral and maxillofacial surgery.

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