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Essential drugs used in prosthetic dentistry

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Keywords

Analgesic, Antibiotics, Implant, Pharmacotherapeutics, Prosthetic dentistry, Salivary substitute, Xerostomia

Abstract

Pharmacology is the branch of medicine and biology concerned with the study of drugs. The two primary areas of concern are the effects of drugs on biological system and how biological system affects the effectiveness and metabolism of drugs. As a dental specialty, Prosthodontics deals with patients of all ages, especially geriatric population, emphasizing the need for understanding the local and systemic effects as well as the adverse effects of drugs. The aim of this article was to provide necessary information about the common drugs used in prosthodontics, which is crucial while prescribing medications in a more knowledgeable way. Hence in this article a brief review of various drugs that are used in complete, partial, fixed, and implant prosthetic dentistry are mentioned.

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Introduction

Medications are a major factor in enhancing patient's response during the pre and post treatment stages. A clinical procedure's success or failure depends mostly on how effectively the pharmacologic principles of locally acting drugs are implemented.[1] A solid foundation in pharmacology and up to date knowledge of most recent developments in pharmaceuticals are essential. According to WHO medication is any material or product that is used or meant to be utilized to alter or examine physiological systems or pathological conditions to benefit the patient.[2] Prosthodontics is the speciality of dentistry which deals with patients of all ages, hence making it more important to know about local and systemic effects of drugs and their

side effects.[3] Various drugs are administered during pretreatment, treatment phase and post treatment phase. Drugs can be broadly classified into therapeutic drugs with localized effect for clinical use in the oral cavity and drugs used for general and systemic pharmacological effect.[4]

This article provide a brief overview of medications used in removable prostheses, fixed prostheses and implant prostheses. **Table 1.**

Describe the various commonly employed drugs in prosthodontics with note on generic, commercial drug name, dosage, indications, contraindications, interactions and side-effects of each drug.

1. Drugs Implicated In Removalble Prostheses

The use of removable prosthesis in the mouth alters the oral environment significantly and can have consequential effect on mouth, resulting in mucosal reaction, burning mouth syndrome, residual ridge resorption, abutment caries, periodontal disease .[5]

1.1 Denture stomatitis

Also known as denture sore mouth or chronic atrophic candidiasis. Palatal mucosa in contact with denture is affected and is chronically erythematous and oedematous6.

Table 1. Description of commonly used drugs in prosthodontic clinical practice

Condition	Class	Drug Generic name	Drug Brand name & combination	Drug interaction	Dosage	Indication	Contraindication	Side effect
Denture stomatitis	Antifungal (Polyene)	Nystatin	Mycostatin, Nilstat, Nyamyc, Nystat Rx	No serious, moderate or mild interactions with other drugs	Oral tablet- 500,000 units	Denture stomatitis, Oral candidiasis	Hypersensitivity	Nausea, bitter taste
Denture stomatitis	Antifungal (Azole)	Clotrimazole	Canesten, Lotrimin, Mycelex, Candid	May inhibit CYP3A4, interaction with drugs metabolized by this pathway (e.g. tacrolimus, cyclosporine) Rifampicin may reduce antifungal effect	Topical solution- twice daily for 2-4 weeks Oral troches- 10mg dissolved slowly in mouth 5 times daily for 14 days	Mild oral candidiasis, Prophylaxis of oral candidiasis in immunocompromised patients	hypersensitivity	Local irritation, itching, burning sensation, erythema, edema, pruritic, urticaria
Denture stomatitis	Antifungal (Azole)	Miconazole	Daktarin, Micatin, Monistat	Inhibits CYP3A4 and CYP2C9Can increase effect of warfarin,phenytoin, sulfonylureas	Topical cream- 2-3 times daily Oral gel- 5-10ml 4 times daily	Oral candidiasis including under dentures	Hypersensitivity	Local irritation, itching, burning
Denture stomatitis	Antifungal (Azole)	Fluconazole	Diflucan, Trican, Zolcan, Flucozole	Enzyme CYP 3A4, CYP2C9 inhibitor. Increase the plasma level of warfarin, phenytoin,cyclosporin, oral hypoglycemics, statins. Rifampicin decreases fluconazole level	50-400mg oncedaily	Moderate to severe or refractory oral candidiasis	Pregnancy, Hepatic disease, Renal impairment	Nausea, vomiting, diarrhoea, abdominal discomfort, headache, alopecia, rashes, hepatic necrosis
Xerostomia	Cholinergic aganist	Pilocarpine	Salagen	Beta blockers may enhance bradycardis, hypotension. Cholinesterase inhibitors increase cholinergic side effects and anticholinergic drugs antagonize effects of pilocarpine	5mg orally, 3-4 times daily	Xerostomia from head and neck radiotherapy, Sjogren's syndrome related dry mouth	uncontrolled asthma or chronic pulmonary disease and in β-adrenergic blocker users and should be used with caution in patients with active gastric ulcers or uncontrolled hyperten sion narrow-angle glaucoma and iritis,	excessive sweating, cutaneous vasodilatation, emesis, nausea, diarrhoea, persistent hiccup, bronchoconstriction, hypotension, bradycardia, increased urinary frequency, and vision problems
Sialorrhea	Cholinergic antagonist	propantheline	Banthine, Pro Banthine	May reduce absorption of digoxin, levodopa. Enhance anticholinergic effects with antihistamine, tricyclic antidepressants, phenothiazine	15mg, 30-60 minutes before procedure	Antisialogogue to reduce salivary secretion before dental treatment	glaucoma, prostatic hypertrophy, severe gastrointestinal disorders (ulcerative colitis, obstructive disease, intestinal atony), and myasthenia gravis	flushing, rapid pulse, pupillary dilation, blurred vision, hyperthermia, dry skin, urinary retention, and constipation
Traumatic ulcer	Quarternary ammonium compound	Benzalkonium hexachloride	Herpetrol	No serious, moderate or mild interactions with other drugs	0.13% benzalkonium hexachloride with 1% menthol		Hypersensitivity/allergy	Local irritation, burning, itching, redness, allergic contact dermatitis
Traumatic ulcer	corticosteroid	Triamcinolone acetonide	Kenacort Orabase	Minimal interaction with topical/oral gel	2-4 times daily up to 14 days	Recurrent apthous ulcer, oral lichen planus, traumatic ulcer	Hypersensitivity, Avoid in untreated oral herpes simplex	Burning sensation, dryness and irritation of oral mucosa, oral thrush with prolonged use
Pain	Local anaesthetic	Lidocaine	Xylocaine	Beta blocker and cimetidine reduce hepatic metabolism	2% lidocaine solution, maximum	Infiltration anaesthesia, nerve block anaesthesia,	Hypersensitivity, severe hepatic impairment, cardiovascular disease,	Dizziness, tinnitus, tremor, seizure, hypotension, bradycardia, rash,

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Condition	Class	Drug Generic name	Drug Brand name & combination	Drug interaction	Dosage	Indication	Contraindication	Side effect
management			Lignox Lignospan	of lidocaine, epinephrine containing formulation interact with beta blockers and increase risk of arrythmia	dose of 4.5mg/kg	soft tissue anaesthesia	seizure disorder	urticaria
Pain management	Local anaesthetics	Bupivacaine	Marcaine, Sensorcaine	May prolong effects of beta blockers, may increase cardiotoxicity risk of antiarrythmic drugs	Infiltration: 0.25-0.5% solution, upto 1.3mg/kgsingle dose	Local anaesthesia (infiltration, nerve block,spinal, epidural)	Hypersensitivity, severe heart block, bradyarrythmia, severe hypotension	Dizziness, tremor, seizure, hypotension, bradycardia, allergic reaction
	Local anaesthesia	mepivacaine	Carbocaine, polocaine	May prolong effects of beta blockers, may increase cardiotoxicity risk of antiarrythmic drugs	Infiltration: 1-2% solution, maximum 6.6mg/kg	Local anaesthesia (infiltration, nerve block)	Hypersensitivity, severe heart block, bradyarrythmia, severe hypotension	Dizziness, tremor, seizure, hypotension, bradycardia, allergic reaction
Gingival retraction	Sympathomimetic	Racemic epinephrine	Racord, Sil-Trax EPI retraction cord	Systemic absorption may interact with beta blockers and reduce effect	0.01-0.05% racemic epinephrine solution	Gingival retraction for crown and bridge impression procedure, subgingival margin exposure	Hypertension, arrythmia, angina, hyperthyroidism	Transient gingival blanching, mild tissue irritation
Infection control and prophylaxis	Antibiotics	Amoxicillin	Amoxil, Moxatag, Trimox, Moxilin, Novamox	May increase the effect of warfarin and increase risk of bleeding, increase toxicity of methotrexate due to reduced clearance	1gm orally, 1 hour before surgery	Prevention of early implant infection and peri implantitis, reduction of post surgical bacterial contamination at the implant site,	Hypersensitivity to amoxicillin, penicillin, history of severe allergic reactions, used with caution in renal impairment, hepatic dysfunction	Nausea, vomiting, diarrhoea, rash, rarely anaphylaxis, angioedema, urticaria, pseudomembranous colitis, hepatotoxicity.
		Cephalexin	Keflex, Cepol, Sporidex, Ceporex, Biocef, Cefolac	Probenecid reduces renal excretion and increase cephalexin level, cephalexin reduces clearance of metformin and cause lactic acidosis, increase effect of warfarin and increase bleeding risk	Prophylaxis: 1gm orally 1 hour before surgery Post operative infection management: 250-500mg orally every 6 hours for -7 days	Early implant infection, secondary infections at the surgical site like cellulitis, abcess, peri implantitis	History of hypersensitivity to cephalosporin, penicillin, severe renal impairment, history of clostridium difficile associated diarrhoea	Nausea, vomiting, diarrhoea, hypersensitivity, anaphylaxis, pseudomembraneous colitis
		Clindamycin	Dalacin C, cleocin, Clindac A, Clinsol, Clindacin	Antagonistic effect on erythromycin, azithromycin, clarithromycin. Kaolin reduce absorption of clindamycin	Preoperative prophylaxis: 600mg orally 1 hour before implant surgery Post operative regimen: 300mg orally every 6-8 hours for 5-7 days	Alternative antibiotic for penicillin allergic patients undergoing implant surgery, bone grafting or sinuslift procedure	History of hypersensitivity, pseudomembraneous colitis, severe liver dysfunction	Nausea, vomiting, abdominal pain, rash, urticaria, pesudomembraneous colitis
Pain reliever	Non opioid analgesic	Acetaminophen	Crocin Dolo-650 Tylenol	Hepatotoxic drugs (e.g., isoniazid, rifampicin, carbamazepine, phenytoin): increased risk of liver damage. Alcohol: potentiates	500–1000 mg orally every 4–6 hours. Maximum daily dose: Up to 4 g/day in healthy adults.	Relief of mild-to-moderate pain after: Tooth extraction prior to prosthesis fabrication. Dental implant placement. Minor pre-prosthetic	Known hypersensitivity to acetaminophen. Severe hepatic impairment or active liver disease. Chronic alcohol consumption.	Common: nausea, rash, headache. Hepatic toxicity: dose-dependent; Renal toxicity: with chronic excessive use.

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			Panadol Calpol	hepatotoxicity. Warfarin: chronic high-dose acetaminophen may enhance anticoagulant effect → ↑ bleeding risk. Enzyme inducers (barbiturates, anticonvulsants): reduce therapeutic effect and ↑ toxicity risk.		surgeries. Soft tissue soreness or traumatic ulceration from ill-fitting dentures. As an alternative to NSAIDs in patients with gastric irritation, renal issues, or when anti-inflammatory effect is not essential.		
Pain reliever	Analgesic (NSAID)	Ibuprofen			200-400mg every 6-8 hr	Inflammatory pain, TMD flare up, post operative swelling	Active peptic ulcer, GI bleed, chronic kidney disease, uncontrolled hypertension, heart failure, 3rd trimester pregnancy	
Pain reliever	Analgesic (NSAID)	Naproxen	Naprosyn Anaprox Aleve	Increase Bleeding risk: with anticoagulants (warfarin, heparin), antiplatelets (aspirin, clopidogrel). Cause GI toxicity: with corticosteroids or alcohol. Decrease Renal clearance: with ACE inhibitors, diuretics, cyclosporine. Increase Lithium and methotrexate levels: due to reduced clearance.	250–500 mg orally every 12 hours	Relief of post-operative pain and inflammation after: Dental implant placement Tooth extractions (prior to denture fabrication) Pre-prosthetic surgical procedures Control of TMJ inflammation/pain in patients undergoing occlusal rehabilitation. Management of musculoskeletal pain associated with prosthesis adaptation.	Hypersensitivity to naproxen or other NSAIDs. History of NSAID-induced asthma, urticaria, or allergic reaction. Active peptic ulcer or GI bleeding. Severe renal, hepatic, or heart failure. Patients on anticoagulant therapy (relative contraindication in dental surgeries). Late pregnancy	GI effects: nausea, gastritis, peptic ulcers, bleeding. Renal impairment (esp. in long-term use). Cardiovascular risk: hypertension, edema, rare risk of thrombotic events. CNS: dizziness, headache. Hypersensitivity: skin rashes, bronchospasm
	Analgesic (NSAID)	Diclofenac	Voveran Voltaren Cataflam Diclomol	↑ Bleeding risk: with anticoagulants (warfarin, heparin), antiplatelet agents (aspirin, clopidogrel). ↑ GI toxicity: with corticosteroids or alcohol. ↓ Antihypertensive effect: ACE inhibitors, diuretics, β-blockers. ↑ Toxicity: with methotrexate, cyclosporine, lithium.	50 mg tablet, 2–3 times daily	Relief of post-operative pain, swelling, and inflammation after: Dental implant placement Surgical tooth extractions (before denture construction) Pre-prosthetic surgeries (ridge reduction, tori removal) Symptomatic relief in TMJ disorders (pain, inflammation). Myofascial pain management in patients adjusting to new prostheses.	Hypersensitivity to diclofenac or other NSAIDs. Peptic ulcer, GI bleeding, or perforation. Severe hepatic, renal, or cardiac disease. History of asthma, urticaria, or allergic reactions to NSAIDs.	GI: nausea, gastritis, ulcers, GI bleeding. Hepatic: elevated liver enzymes, rare hepatotoxicity. Renal: nephrotoxicity, fluid retention. Cardiovascular: increased risk of thrombotic events, hypertension, edema. CNS: headache, dizziness. Hypersensitivity: rashes, pruritus, bronchospasm.
	Analgesic (COX 2 Selective)	Etoricoxib	Arcoxia Nucoxia Etoshine	↑ Risk of bleeding: with anticoagulants (warfarin), antiplatelet drugs (clopidogrel, aspirin). ↑ Renal toxicity: with ACE	60–90 mg orally once daily	Post-surgical pain and inflammation after: Implant placement Pre-prosthetic surgical procedures (ridge reduction,	Hypersensitivity to etoricoxib or other NSAIDs. Active peptic ulcer, GI bleeding. Severe hepatic or renal impairment. Established ischemic heart disease,	GI (lower risk than traditional NSAIDs): dyspepsia, abdominal pain, diarrhea. Cardiovascular: hypertension, edema, ↑ risk of thrombotic events

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				inhibitors, diuretics, cyclosporine. ↑ Methotrexate and lithium levels (reduced clearance). May reduce the effect of some antihypertensives.		tori removal) Extractions before complete/partial denture fabrication TMJ disorders: useful for pain and inflammation without significant GI irritation. Alternative to conventional NSAIDs in patients with gastritis or peptic ulcer history.	stroke, peripheral arterial disease, or uncontrolled hypertension	(MI, stroke). Renal: fluid retention, nephrotoxicity (esp. with long-term use). CNS: dizziness, headache. Hypersensitivity: skin rashes, pruritus.
	Analgesic (weak opioid)	Tramadol	Ultram Tramazac Dromadol Dolol	↑ Risk of serotonin syndrome: with SSRIs, SNRIs, MAO inhibitors, TCAs. CNS depression: additive effect with alcohol, benzodiazepines, antihistamines, opioids, anesthetics. Seizure risk ↑: with antidepressants, antipsychotics, bupropion. Reduced effect with CYP2D6 inhibitors (e.g., fluoxetine, paroxetine).	50–100 mg every 4–6 hours as needed Max: 400 mg/day	Moderate to severe post-operative pain after: Dental implant surgery Extensive pre-prosthetic surgery (ridge augmentation, tori removal) Difficult extractions (before complete denture construction) Alternative for patients unable to tolerate NSAIDs (e.g., GI ulcer patients, elderly with renal impairment).	Hypersensitivity to tramadol. Severe respiratory depression or acute intoxication with alcohol, hypnotics, or opioids. History of seizures or epilepsy (lowers seizure threshold). Concomitant use with MAO inhibitors or within 14 days of discontinuation. Severe hepatic or renal impairment.	CNS: dizziness, drowsiness, headache, seizures (dose-related). GI: nausea, vomiting, constipation. Respiratory depression (esp. at high doses or with other CNS depressants). Dependence and withdrawal risk
Neuralgia	Anticonvulsant (tricyclic compound)	Carbamazepine	Tegretol Zeptol Carbatrol Mazetol Epitol	Enzyme inducer (CYP450): reduces effectiveness of many drugs (warfarin, doxycycline, oral contraceptives). Erythromycin, clarithromycin, azole antifungals ↑ carbamazepine levels → toxicity risk. Phenytoin, phenobarbital: alter carbamazepine metabolism.	Adults (oral): with 100–200 mg once or twice daily Maintenance: 400–1200 mg/day in divided doses	Trigeminal neuralgia, Neuropathic orofacial pain syndromes, Helpful when pain complicates prosthodontic treatment planning or adaptation to dentures.	Hypersensitivity to carbamazepine, tricyclic compounds. History of bone marrow suppression. Hepatic porphyria. Use with MAO inhibitors.	CNS: dizziness, drowsiness, ataxia, diplopia, blurred vision. Hematologic: leukopenia, aplastic anemia (rare but serious). Dermatologic: skin rashes, Stevens–Johnson syndrom. GI: nausea, vomiting. Hepatic: ↑ liver enzymes, hepatotoxicity.
Neuralgia	Anticonvulsant (tricyclic compound)	Oxcarbazepine	Trileptal Oxetol Oxtellar XR Oxcarb	Enzyme inducer (weaker than carbamazepine): may reduce levels of oral contraceptives. Phenytoin: oxcarbazepine may increase phenytoin levels. CYP inducers (carbamazepine, phenobarbital, rifampicin): reduce oxcarbazepine levels.	Start with 300 mg twice daily Increase gradually by 300 mg/day every 3–7 days based on response. Usual maintenance: 600–1200 mg/day in divided doses	Trigeminal neuralgia (primary indication when carbamazepine is poorly tolerated). Atypical facial pain or neuropathic orofacial pain. Used when severe neuralgic pain interferes with prosthodontic treatment planning, denture adaptation, or occlusion-	Hypersensitivity to oxcarbazepine or eslicarbazepine. History of Stevens–Johnson syndrome with anticonvulsants. Severe hepatic impairment. Caution in renal impairment	CNS: dizziness, drowsiness, headache, fatigue, ataxia, diplopia. Hyponatremia (more common than with carbamazepine, especially in elderly). GI: nausea, vomiting, abdominal pain. Dermatologic: rash, pruritus, rare Stevens–Johnson syndrome. Psychiatric: irritability, mood

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				CYP inhibitors (verapamil, erythromycin, ketoconazole): may ↑ oxcarbazepine levels.		related procedures.		changes.
TMD	Skeletal muscle relaxant	Carisoprodol	Soma Carisoma Vanadom Sodol	Additive CNS depression: alcohol, benzodiazepines, opioids, antihistamines, antidepressants. Enzyme inducers/inhibitors (rifampin, phenytoin, carbamazepine, ketoconazole, erythromycin) alter metabolism. Potentiated sedative effect with other muscle relaxants or hypnotics. Avoid with CYP2C19 inhibitors (fluvoxamine, omeprazole)	250–350 mg three times daily and at bedtime Max duration: 2–3 weeks	Relief of acute musculoskeletal pain and spasm in TMD, especially: Myofascial pain dysfunction syndrome Muscle spasm due to stress-related bruxism or parafunctional habits Muscle tightness affecting mandibular movement during prosthodontic rehabilitation	Hypersensitivity to carisoprodol or meprobamate. History of drug abuse or dependence. Acute intermittent porphyria.	CNS: drowsiness, dizziness, sedation, impaired coordination. GI: nausea, vomiting. Allergic reactions: rash, pruritus (rare). Dependence, tolerance, and withdrawal symptoms with prolonged use.
TMD	Centrally acting muscle relaxant	cyclobenzaprine	Flexeril Amrix Fexmid	Additive CNS depression: alcohol, benzodiazepines, opioids, antihistamines. Avoid with MAO inhibitors (risk of hyperpyretic crisis, seizures, severe hypertension). Potentiates serotonergic drugs (SSRIs, SNRIs, TCAs, tramadol) → ↑ risk of serotonin syndrome.	5–10 mg, 3 times daily Max: 30 mg/day	Relief of acute muscle spasm and myofascial pain in TMD, especially when associated with: Bruxism or clenching Myofascial pain dysfunction syndrome Restricted mandibular movement due to muscle tightness	Hypersensitivity to cyclobenzaprine. Use within 14 days of MAO inhibitors. Hyperthyroidism. Recent myocardial infarction, arrhythmias, heart block, or congestive heart failure	CNS: drowsiness, dizziness, fatigue, confusion. Anticholinergic effects: dry mouth, blurred vision, constipation, urinary retention. Cardiac: tachycardia, arrhythmias (rare but possible). Serotonin syndrome risk if combined with serotonergic agents.

Nystatin, also known as Mycostatin, is the most commonly used antifungal in dentistry.[7]. It shows dose-dependent fungistatic or fungicidal activity against fungi, especially Candida albicans, which causes oral thrush and can colonize prosthetic devices.[8] Since it is not absorbed, tablets are held in the mouth until dissolved, and dentures can be disinfected by immersion in nystatin solution.[9] A polyene antibiotic, nystatin binds to ergosterol in fungal membranes, forming pores that increase permeability and cause cell death. Adverse effects include nausea and bitter taste.[10]

Another antifungal drug is clotrimazole, a triazole belonging to the azole group. Azoles act by inhibiting the 14α-demethylase enzyme, thereby impairing ergosterol synthesis.[11] Clotrimazole (Mycelex) is a fungistatic agent effective against Candida albicans infections of the oral cavity. It is administered as a 10 mg troche, dissolved in the mouth five times daily for 14 days. Since it is not absorbed systemically, adverse effects are minimal, limited to local irritation, itching, or burning.[12]

Triazoles are broad-spectrum antifungals that include fluconazole and itraconazole. Fluconazole is rapidly absorbed into body fluids, with 50–100 mg/day effective for oropharyngeal candidiasis and 100–200 mg/day for esophageal candidiasis. Side effects are uncommon but may include nausea, vomiting, and abdominal pain at doses above 200 mg.[13,14] Itraconazole is also effective in treating oropharyngeal and esophageal candidiasis.

1.2 Mouthrinses containing anti-infective agents

Many mouth rinses contain ethanol as the primary local anti-infective agent, along with flavoring, sweeteners, colors, preservatives, and wetting agents.[15] Thymol and other phenolics have limited use due to poor taste. Hydrogen peroxide, though weak in antibacterial action, helps remove debris through oxygen release[16] The surface-active agent cetylpyridinium, a quaternary ammonium derivative, has mild bacteriostatic action but leaves an acrid aftertaste.[17.]

Povidine Iodine – is an iodophore. It combines a surface-active agent with halogen releasing molecule. It is the most likely antibacterial agent with most effectiveness. The iodophores taste unpleasant but they never sting or discolour like iodine solutions do. The council on dental therapeutics approves povidone-iodine to be utilized as a surgical scrub at concentrations between 7.5% and 10%.[15,18]

Chlorhexidine, a biguanide, is effective mainly against gram-positive bacteria, less so against gram-negative and fungi, and ineffective against viruses and spores.15 0.12% chlorhexidine digluconate is widely used for plaque control and gingivitis, with sustained release due to its binding to oral tissues and salivary proteins.19 It is also used as a 2–4% surgical scrub and as a mouthwash to aid healing after denture insertion or implant placement until suture removal.[20]

1.3 Sialagogues

Xerostomia may result from systemic diseases (Sjogren's syndrome, rheumatoid arthritis, diabetes insipidus, pernicious anaemia), radiation, drug side effects (anticoagulants, antidepressants, antihypertensives, antiretrovirals, hypoglycaemics, levothyroxine, multivitamins, NSAIDs, steroid inhalers), aging, or factors like depression, anxiety, stress, and malnutrition. Hyposalivation is diagnosed when stimulated salivary flow is ≤0.5–0.7 mL/min and unstimulated flow is ≤0.1 mL/min.[21] It can lead to dysphagia, dysgeusia, stomatitis, burning tongue, rampant caries, periodontal disease, poor denture tolerance, mucosal ulceration, and Candida albicans infection.[22, 23]

Management strategies aim to relieve symptoms and/or increase salivary flow. Simple measures include hydration, humidification at night, avoiding irritants, and using sugar-free gums/candies. Pharmacological options include mucosal lubricants, saliva substitutes, and stimulants.[21] Cholinergic agonists like pilocarpine (5 mg TID, ~3 h action) and cevimeline (30 mg TID, greater M3 selectivity) enhance salivary flow.[23,25] 1Side effects include sweating, flushing, nausea, diarrhoea, bronchoconstriction, bradycardia, hypotension, urinary frequency, and vision changes. Both are contraindicated in uncontrolled asthma, COPD, β-blocker use, and used cautiously in ulcers, hypertension, and CVD; pilocarpine is additionally contraindicated in narrow-angle glaucoma and iritis.[26]

Citric acid may relieve antidepressant-induced

xerostomia.[27] Anethole trithione and bromhexine, direct cholinergic agonists, show some benefit but require further evidence.[28]

Topical options include 1% malic acid spray (effective for drug-induced xerostomia but may erode enamel).[29] sugar-free chewing gums/candies to stimulate flow and reduce friction30 and saliva-substitute gels, rinses, or toothpastes.

1.4 Salivary substitutes

Saliva substitutes increase oral viscosity and mimic natural saliva without enhancing flow. They typically contain fluoride, calcium, phosphate, cellulose derivatives (carboxymethyl- or hydroxyethyl-cellulose), flavoring agents, and preservatives.[31] Only carboxymethylcellulose-based products are approved by the Council on Dental Therapeutics2. Their effect is short-lived, requiring frequent use, with variable patient relief and compliance issues. Current strategies focus on moistening and lubrication. Available forms include sprays, gels, solutions, or prosthesis reservoirs; common brands are Salix, Salivart, Orex, Moist-Stir, and Xerolube.[32]

The mucin-containing spray Saliva Orthana showed no significant benefit over placebo in a controlled trial.[33] Among oral lubricants, gel formulations are the most effective and well-accepted. Anhydrous crystalline maltose lozenges increased salivary flow and reduced xerostomia symptoms.[34] Topical application of the anticholinesterase physostigmine to oral mucosa also improved salivary secretion from minor glands, offering a potential alternative to systemic therapy.[35]

1.5 Antisialogogues

Cholinergic antagonists reduce salivary secretion by blocking receptors stimulated by sialagogues. Atropine and scopolamine, alkaloids from Atropa belladonna, are outdated but Council-approved.[36] Synthetic derivatives like methantheline and propantheline share similar actions, with propantheline being more potent.[2] For clinical use, these agents are administered before procedures: atropine (1–2 h), scopolamine (30–60 min), methantheline (30–60 min), and propantheline (30 min).[37]

Anticholinergic drugs cause typical parasympathetic blockade effects, including flushing, tachycardia, mydriasis, blurred vision, hyperthermia, dry skin, urinary retention, and constipation.[38] In dentistry, side effects are rare since effective salivary suppression occurs at low doses.[39] However, risks are higher in elderly patients with xerostomia. These drugs are contraindicated in glaucoma, prostatic hypertrophy, severe GI disorders (ulcerative colitis, obstruction, intestinal atony), and myasthenia gravis.[40]

1.6 Gagging

The gag reflex is a protective mechanism preventing aspiration.41 Gagging may be psychogenic (triggered by anxiety, fear, or sensory stimuli such as sight, smell, or imagination) or somatogenic (due to tactile or systemic stimuli).[42,43]

Trigger zones include the tonsillar pillars, tongue, palate, and posterior pharyngeal wall; the soft palate is particularly sensitive. Dental procedures like impressions, radiographs, and denture wearing often provoke gagging.[44,45]

When clinical and psychological approaches fail, pharmacological management may be required.[46] Options include local anaesthetics and centrally acting drugs such as antihistamines, sedatives, tranquilizers, parasympatholytic, and CNS depressants.[47] Since gagging is parasympathetically mediated, atropine derivatives are useful by blocking acetylcholine at vagal postganglionic sites, reducing salivation, GI contractions, and vomiting reflex.[48]

Topical anaesthetics (sprays, gels, lozenges) can desensitize oral mucosa but may themselves induce gagging, cause systemic toxicity, suppress the cough reflex, and risk aspiration pneumonia.[49] Greater palatine injections have been suggested but may trigger gagging and distort impressions.[44]

A safer approach is applying anaesthetic spray on gauze placed on the posterior palate until impression making is complete. Other reported pharmacological agents include trime thobenzamide and granisetron, an IV antiemetic.[51, 52]

1.7 Traumatic ulcer

The force of friction between the denture's tissue surface and the mucosa may be the reason for trauma in denture

users.53 The first line of treatment is to stop wearing dentures and apply topical benzalkonium hexachloride (20%) and mucopain or dolo gel, the latter additionally incorporates salicylates to feed antibacterial action. Triamcinolone acetonide (0.1%)-containing kenacort gel is recommended in the event of severe inflammation.[54]

1.8 Denture adhesive

With proper use denture adhesives are beneficial to the patient in increasing retention and stability, enhanced comfort, improved function, and in providing psychological satisfaction. They should not be used as an aid to compensate for denture deficiencies even though adhesives enhance denture performance.[55]

Denture adhesives are supplied as a paste, powder or cream.

The main ingredients of denture adhesives are classified into three groups.

Group 1 (Adhesive agents)

Tragacanth, gelatine, methylcellulose, acacia, hydroxyl-methyl cellulose, Karaya gum, sodium carboxyl-methyl cellulose, pectin, and synthetic polymers like acrylamides, acetic, polyvinyl and polyethylene oxide.[56]

Group 2 (Anti-microbial agents)

Sodium tetraborate, ethanol, hexachlorophene, and sodium borate.[56]

Group 3 (Other agents)

Plasticizing agents, flavoring agents like oil of peppermint, oil of wintergreen, and wetting agents, etc.[56]

Enhance retentive properties of denture by improving interfacial forces, increase cohesive and adhesive properties, increase viscosity of medium lying between denture am basal seat area, eliminate voids between basal seat an denture base.[57]

As the Adhesive powders absorb water, they swell to many times their original volume and the anions so formed, interact with cations in the proteins in the oral mucous membrane.[58]

The viscosity of the adhesive is increased by the thick saliva formed, thereby increasing the denture retention.

Newer adhesive materials provide stronger bio-adhesive and cohesive forces. Free carboxyl groups formed by the hydration of adhesive such as methyl cellulose, hydroxyl methyl cellulose, sodium carboxyl-methyl cellulose or poly methyl vinyl-ether maleic anhydride, etc. form electrovalent bonds that produce stickiness or bio adhesion.[59]

The increased viscosity of the adhesive creams results in their lateral spread excluding air and saliva thereby increasing the retention.

Mode of action-

Any residual adhesive should be removed from the tissue-bearing surface of the denture. Food debris on the tissue surfaces of the denture is wiped clean. Wet dentures before application of adhesive. Small amounts of adhesive are applied to the tissue-bearing surface of the denture.[60] In the maxillary denture - anterior alveolar ridge, the centre of the hard palate and posterior palatal seal region. In the mandibular denture – adhesive must be applied along the entire sulcus.[61]

Denture should be seated and held in place firmly by hand pressure for 5-10 s. Gauze is used to remove excess adhesive. Patient is advised to close into centric occlusion several times to spread the adhesive as a thin even layer.[60, 62]

1.9 Denture cleanser

Denture cleansers correspond to a variety of products designed to safely remove stains, deposits, and debris from the surfaces of dental prostheses, by means of various methods. An ideal denture cleanser should be simple to use, effectively remove organic and inorganic matter from denture surface, have bactericidal and fungicidal properties, and be compatible with all denture base materials.[63] Though mechanical cleansing using a soft denture brush and water is the most recommended as well as effective and safe method to clean dentures, for persistent accumulations or stains, overnight immersion in denture cleansers is a common practice.

Creams, pastes, gels and solutions or even tablets are available forms to clean dentures.[64] Soaking dentures in the cleaning solution varies from a few minutes to overnight

depending on the manufacturer's instructions whereas denture cleansing creams, pastes or gels are brushed on the denture after it is removed from the mouth and then rinsed off.

It includes- Oxidizing (bleaching) agents: Alkaline perborate, sodium perborate or postassium monopersulfate. These compounds remove staining and kill the bacteria harboured on a denture's surface, Reducing Solutions- Sodium hypochlorite, Effervescing agents - Perborate, carbonate or citric acid. Effervescing agents provide for the rapid disintegration of the product and create a mechanical cleansing action, Chelating agents - EDTA. This type of compound helps to remove the tartar that has accumulated on a denture's surface, Detergents- Sodium polyphosphate. These compounds assist in cleansing the denture. Additional compounds - Dye markers that provides a color change when the cleansing process has been completed, Flavorings and fragrances, Enzymes – Protease, amylase, Disinfectants- Potassium permanganate, glutaraldehyde.[64,65]

Most denture cleansers contain persulfates as bleaching/cleaning agents, which may trigger type IV (delayed) hypersensitivity after repeated exposure.[66]. This cell-mediated immune reaction (via cytotoxic T cells and cytokines) can cause irritation, tissue damage, rash, hives, gum tenderness, respiratory issues, and hypotension.

Commercially available denture cleanser is- Fittydent, Kleenex, Stain away, Polident, Triclean.[63]

Investigations and studies have pointed out that, the correct use of chemical cleansers is not associated to alterations in mechanical properties of denture base materials.[67, 68]

Till date allergy or harmful effects by the proper use of cleansers following manufacturer's direction have also not been reported.

2. Drugs Associated With Fixed Dental Prosthesis

2.1 Local anaesthetics

Local anaesthetics (LAs) produce reversible loss of sensation by blocking sodium channels in sensory nerves, preventing depolarization and impulse conduction.[69,74] They are classified as amides (lidocaine, mepivacaine, bupivacaine, prilocaine, ropivacaine) metabolized in the liver, and esters (procaine, benzocaine) hydrolysed by plasma pseudocholinesterase.[70,72] Amides rarely cause allergy, while esters may lead to hypersensitivity via PABA metabolites.[76,77] Their efficacy depends on lipid solubility, pKa, and tissue pH; inflamed/acidic tissues reduce action.75. Duration is influenced by vascularity and protein binding. Toxicity arises from overdose or intravascular injection, with CNS symptoms (tinnitus, tremors, convulsions, coma) and cardiovascular effects (tachycardia, hypertension, later depression). Bupivacaine and tetracaine are most cardiotoxic, lidocaine least.[78,79]

Commonly used agents include lidocaine (2% + 1:100,000 epinephrine; medium duration), mepivacaine (2% with levonordefrin or 3% plain for short procedures), and bupivacaine (0.5% + 1:200,000 epinephrine; long-acting for implants/ surgeries). Topical anaesthetics, such as benzocaine 20% (Mucopain, Dologel), are popular but more allergenic.[73] Vasoconstrictors like epinephrine prolong effect, reduce systemic absorption, and lower toxicity.[80,81] Safe maximum doses are 0.2 mg for healthy patients and 0.04 mg for those with cardiovascular disease.[82] Vasoconstrictor-containing LAs are contraindicated in pheochromocytoma, uncontrolled angina, arrhythmias, CHF, hyperthyroidism, severe hypertension, and recent MI or stroke.[82,83]

2.2 Agents used for gingival retraction

Gingival retraction is displacement of marginal gingiva away from the tooth to achieve proper margin during impression procedure.85,86 Preparations used for gingival retraction are usually cotton cords impregnated with either racemic epinephrine or aqueous solutions of metal salts.[87,88]

All the precautions for epinephrine as used in local anesthetic apply to its use in retraction cords because systemic absorption is nearly as rapid as intramuscular injection in areas of gingival abradement, and the cords contain a large dose of epinephrine. Shrinkage of gingival tissue presumably is a result of local epinephrine absorption, α-adrenergic receptor activation, vasoconstriction, and the resultant decrease in volume of this highly perfused tissue

2.3 Haemostatic agent

Thrombin can be applied as a liquid or powder on clotted blood free site. Thrombin must not be injected because of

extensive intravascular clotting that may result in death.[94]

Absorbable gelatine sponge can also be used which is available in the form of porous sheet and powder. Haemostatic property of gelatine can be improved b soaking in a solution of thrombin before application. It can be sutured into a desired area because it is absorbed within several weeks.[95]

Styptics may be considered as a concentrated form of an astringent. Ferric subsulfate solution (Monsel's solution) is a haemostatic but causes a black stain on teeth and is most effective when applied under pressure and left in place for several hours.[96]

Drugs Implicated In Implant Prosthesis

Dental implant has become a routine treatment option due to its high survival and success rates, offering a predictable solution for tooth replacement. A thorough understanding of the indications and protocol for the use of pharmacologic agents in implant dentistry is essential.

2.4 Antimicrobials

An important complication to prevent after implant surgery is infection. Infection can lead to a multitude of problems, including pain, swelling, loss of bone, and possible failure of the implant.[97]. Because of the risk of morbidity from infections, antimicrobial therapy is an essential component of the surgical protocol. The most common antimicrobials used in implant dentistry consist of antibiotics (local and systemic) and antimicrobial rinses (0.12% chlorhexidine gluconate).[98,99]

2.4.1 Antibiotics

The use and understanding of the various antibiotic regimens available in implant dentistry are beneficial for the initial success and long-term maintenance of implant therapy.100. Antibiotic therapy in implant dentistry may be classified as either prophylactic (prevent infection) or therapeutic (treat infection).[98]

Prophylactic antibiotics-

Postoperative wound infections can have a significant effect on the success of dental implants. The main goal of the use of prophylactic antibiotics is to prevent infection during the initial healing period from the surgical wound site, thus decreasing the risk of infectious complications of the soft and hard tissues.[101,102] Although there is no conclusive evidence on the mechanism of preoperative antibiotics, most likely a greater aseptic local environment is achieved.[103] The antibiotic chosen for prophylaxis should encompass the bacteria most known to be responsible for the type of infection found with the surgical procedure. [104,105] Therefore, the following antibiotics are suggested against pathogens known to cause postoperative surgical wound infections in implant surgery are, Amoxicillin: the usual drug of choice, prophylactic dose is 1gram(g) 1hour(hr) before surgery.[106] If allergic, Cephalexin (nonanaphylactic allergy to penicillin), prophylactic dose is 1g 1hr before surgery or Clindamycin (anaphylactic allergy to penicillin), prophylactic dose is 600mg 1hr before surgery.106. For sinus involvement procedures (e.g., sinus grafts) the following antibiotics are suggested: 1. Augmentin 2. Ceftin (if history of recent use of antibiotics [within 4 weeks]) or doxycycline

Therapeutic use of antibiotics: postoperative infections

Acute postoperative infections have been shown to occur on the third to fourth day after surgery. Local signs of infection are pain, inflammation, bleeding, and exudate at the site of surgery. Systemic signs include fever, headache, nausea, muscle aches, vomiting, and weakness.[97] When surgical wound infections arise, a specific diagnosis is advantageous to treat the complication. When evaluating the various antibiotics possible that are effective against the bacteria in question, a broad-spectrum beta-lactam antibiotic is most often the first-line medication.[102]

The recommended treatment for intraoral infections associated with grafting or implant therapy include surgical drainage and systemic antibiotics Amoxicillin (500 mg): Two immediately, then one tablet three times daily for 1 week or if penicillin allergy exists then Clindamycin (300 mg): Two immediately, then one tablet three times daily for 1 week.[103,100] If no improvement is seen after 4 days, a culture and sensitivity test should be administered to select the antibiotic most effective against the responsible organisms.

2.4.2 Chlorhexidine

Another modality for antimicrobial prophylaxis for implant

surgery is the use of an oral rinse, 0.12% chlorhexidine digluconate.[107]

Chlorhexidine gluconate is a potent antibacterial that causes lysis by binding to bacterial cell membranes. It has high substantivity that allows it, at high concentrations, to exhibit bactericidal qualities by causing bacterial cytoplasm precipitation and cell death.[108] In the oral cavity, chlorhexidine has been shown to have a slow release from tissue surfaces over a 12-hour period. The use of chlorhexidine has been shown to be an effective adjuvant in reducing plaque accumulation, enhance mucosal health, improve soft tissue healing, treat periodontal disease, prevent alveolar osteitis.[109]

2.5 Analgesic

Effective pain control in oral implantology requires analgesic coverage before local anaesthesia wears off and a structured postoperative regimen. Pre-emptive analgesia, introduced before surgical stimuli, reduces postoperative pain intensity. Adequate analgesic blood levels are recommended prior to implant or grafting procedures, where factors such as tissue reflection, bone preparation, and surgical duration influence pain.[110]. Common pre-emptive medications include ibuprofen (400 mg), acetaminophen (1000 mg), and celecoxib (200 mg). Postoperatively, nonopioid options include acetaminophen, NSAIDs, COX-2 inhibitors, and tramadol. Acetaminophen provides analgesia and antipyresis with a ceiling dose of 4 g/day, but carries hepatotoxicity risk.[111] NSAIDs, both nonselective (ibuprofen) and selective (celecoxib), act by inhibiting COX enzymes and prostaglandin synthesis, reducing inflammation and hyperalgesia.[112, 113]

Nonselective NSAIDs may cause GI, renal, hepatic, and cardiac side effects.114 while COX-2 inhibitors reduce GI risk.[116] Combining acetaminophen (1 g q6h) with ibuprofen (400 mg q8h) provides superior analgesia.[117] Tramadol, with dual action on μ-opioid receptors and neurotransmitter reuptake, is effective alone or in combination with acetaminophen (Ultracet), especially in patients intolerant to NSAIDs or opioids.[118]

Opioids remain the mainstay for moderate-to-severe postoperative pain. They act as μ- and κ-receptor agonists, providing dose-dependent analgesia without a ceiling effect but with risks of euphoria, nausea, vomiting, constipation, sedation, respiratory depression, and dependence.[119] Codeine, though effective, is limited by side effects; hydrocodone and oxycodone (often combined with acetaminophen or aspirin) provide stronger analgesia, while sustained-release oxycodone carries high abuse potential. Meperidine, due to poor oral bioavailability and toxic metabolite accumulation, is not recommended orally. Combination analgesic therapy using nonopioids with opioids enhances analgesic efficacy through additive or synergistic effects while minimizing side effects. This multimodal approach is the preferred strategy for postoperative implant pain management, ensuring adequate comfort and reducing drug-related risks.[120]

2.6 Postoperative swelling and inflammation management

Management of postsurgical swelling is essential for pain control, reducing oedema, trismus, and infection risk. Surgical trauma triggers an inflammatory response mediated by COX enzymes and prostaglandins. NSAIDs like ibuprofen provide both analgesic and anti-inflammatory effects by inhibiting prostaglandin synthesis, making them effective for postoperative management.[110.] In addition, glucocorticosteroids are widely used due to their strong anti-inflammatory action. Dexamethasone is the preferred steroid for implant surgery, offering high anti-inflammatory potency with minimal mineralocorticoid effects. For optimal efficacy, it should be administered preoperatively (preferably in the morning to align with natural cortisol release), with use limited to ≤3 days post-surgery, not exceeding the cortisol-equivalent of 300 mg. A tapering regimen over 2–3 days minimizes side effects.[121, 122.]

Beyond controlling oedema and pain, dexamethasone also reduces neurosensory impairment by decreasing inflammation at nerve injury sites and supporting regeneration of the inferior alveolar nerve.[123]. Adjunctive cryotherapy further decreases postoperative inflammation by inducing vasoconstriction, thereby reducing blood and lymph flow at the surgical site. Ice packs or cold dressings should be applied externally for 20 minutes followed by a 20-minute rest period to prevent thermal injury. This

intermittent protocol prevents reactive vasodilation while effectively reducing swelling.[124]

2.7 Antianxiety agents

These are used in apprehensive patients due to stress during the dental procedure. Most popular and safest medications for these patients is Benzodiazepine.[125] The benzodiazepines are the most effective drugs available for dental-related anxiety. These drugs have depressant effects on the subcortical levels of the CNS. Under this Diazepam, Alprazolam works well as antianxiety medication during dental procedure. [126] Diazepam is usually not an effective agent for highly apprehensive patients unless administered intravenously. However, it is extremely effective if given orally the night before the procedure with a dose of 5 to 10 mg. Advantages of diazepam for dental procedures are that it reduces salivary flow and relaxes skeletal muscles. Midazolam is a fast-acting benzodiazepine that is twice as potent as diazepam. It is available as a syrup and also as a formulated injectable solution. Midazolam possesses anticonvulsant proper ties and also is an excellent muscle relaxant, sedative, and amnesic. The inhibitory effects in the CNS are intensified; therefore, midazolam should not be combined with other CNS depressant drugs. Triazolam is an orally administered benzodiazepine and short-term hypnotic drug. When given orally, this drug is fast acting and has been shown to be safe and effective for dental procedures. Studies have shown that triazolam given in doses of 0.25 to 0.5 mg does not produce adverse effects in respiration, heart rate, or arterial pressure. This drug is also ideal for patients with hypertension, because blood pressure has been shown to decrease by five points. [126]

3. Drugs Implicated In Dentin Hypersensitivity

Tooth sensitivity is a common complaint of patients in dental practices. Studies have demonstrated dentinal hypersensitivity to affect 10–30% of the population. There are various potential causes of tooth sensitivity and a variety of available non-invasive treatment options.

Dentinal hypersensitivity has varying degrees of pain. The first steps in treating a symptom such as tooth hypersensitivity should be diagnosis and identification of aetiology followed by an attempt to reduce or eliminate the factors contributing to the symptom.

The most common aetiology of dentin exposure is gingival recession. When the gingival margin recedes past the cemento–enamel junction it reveals cementum. This thin cementum covering is easily lost, as a result of which dentinal tubules become exposed to the external environment. This exposure is thought to be influenced by aggressive toothbrushing, abrasive toothpastes, poor plaque control, facial piercings, periodontal disease, anatomical predisposition and orthodontic treatment. Avoiding and eliminating these known aetiological factors will help to prevent gingival recession and as a result will prevent dentinal hypersensitivity.

3.1 Dentifrice

In efforts to treat dentinal hypersensitivity, certain ingredients are added to dentifrices. The purpose of these ingredients is to relieve dentinal hypersensitivity by either eliminating nerve conduction or occluding the dentinal tubules. Such ingredients include potassium nitrate, strontium acetate, arginine and calcium carbonate, and calcium sodium phosphosilicate. Potassium nitrate depolarises the nerves within the dentinal tubules and inhibits their ability to transmit pain. strontium has the ability to occlude dentinal tubules. Strontium ions exchange with calcium ions, causing the formation of strontium crystals within dentinal tubules. The combination of arginine and calcium carbonate acts like strontium acetate in that it also occludes dentinal tubules and blocks the movement of fluid suspected to cause sensitivity. blend of calcium sodium phosphosilicate is designed to stimulate the remineralisation of enamel and simultaneously it occludes dentinal tubules. High fluoride concentration toothpastes can also serve as part of this phase of treatment of hypersensitivity. These are usually prescription-only fluoride toothpastes that can deliver 5000–12,500 ppm fluoride. High levels of topical fluoride will help in remineralisation and can relieve dentinal hypersensitivity.[127]

3.2 Dentin desensitiser

Dentin desensitisers are products used by dental professionals to treat dentinal hypersensitivity. Desensitisers have different ingredients, such as

fluoridehydroxyethyl methacrylate, glutaraldehyde, oxalate ingredients. With the exception of potassium nitrate, these desensitising agents occlude the dentinal tubules to relieve sensitivity. Fluoride varnish is a desensitiser, commonly used by dental professionals, which is applied by painting the solution onto the affected surfaces. The solution sets via interacting with saliva, thus allowing it to stay on the tooth surface and facilitating maximal uptake Cof fluoride. The combination of glutaraldehyde and hydroxyethyl methacrylate is currently a popular desensitiser and is commonly referred to by its brand name, Gluma, studies have shown Gluma to occlude tubules by penetrating up to a depth of 50–200 μm. Oxalate is another desensitiser used by dental professionals that works by combining with calcium ions present in saliva. The combination forms insoluble calcium oxalate crystals that precipitate within the tubules, eventually occluding them.[128]

3.3 Bonding Agent

Self-etch bonding systems typically contain acidic ingredients that condition the dentin, as well as monomers that combine on the dentin, forming a hybrid layer45. This layer provides a coating over the dentin and significantly reduces hypersensitivity over a 4-week period. When the previous options of non-invasive treatment of tooth hypersensitivity fail to improve the symptoms, bonding agents can be a recommended next step, which is essentially non-invasive and lacks significant adverse effects.[129]

4. Dental Hard Tissue Remineralization

The remineralization process is a natural repair mechanism to restore the minerals again, in ionic forms, to the hydroxyapatite (HAP) crystal lattice. It occurs under near-neutral physiological pH conditions whereby calcium and phosphate mineral ions are redeposited within the caries lesion from saliva and plaque fluid resulting in the formation of newer HAP crystals, which are larger and more resistant to acid dissolution. Numerous types of remineralizing agents and remineralizing techniques have been researched and many of them are being used clinically, with significantly predictable positive results. The recent research on remineralization are based on and potassium nitrate, as well as a combination of these biomimetic remineralization materials, having the capability to create apatite crystals within the completely demineralized collagen fibers.

Requirements of an ideal remineralization material are as follows:

- Diffuses into the subsurface or delivers calcium and phosphate into the subsurface
- Does not deliver an excess of calcium
- Does not favor calculus formation
- Works at an acidic pH
- Works in xerostomic patients
- Boosts the remineralizing properties of saliva

Fluoride inhibits demineralization as the fluorapatite crystals, formed by reaction with enamel apatite crystals, are more resistant to acid attack compared to HAP crystals. Second, fluoride enhances remineralization as it speeds up the growth of the new fluorapatite crystals by bringing calcium and phosphate ions together. Third, it inhibits the activity of acid producing carious bacteria, by interfering with the production of phosphoenol pyruvate (PEP) which is a key intermediate of the glycolytic pathway in bacteria. And also, the F− retains on dental hard tissue, the oral mucosa and in the dental plaque to decrease demineralization and enhance remineralization.[130]

Toothpastes can contain fluoride in various chemical forms mainly as sodium fluoride (NaF), sodium monofluorophosphate (Na2FPO3), amine fluoride (C27H60F2N2O3), stannous fluoride (SnF2), or combinations of these. Sodium fluoride directly provides free fluoride. Sodium monofluorophosphate is the fluoride of choice when calcium containing abrasives are used. The fluoride released is absorbed to the mineral surface, as a CaF2 or a CaF2-like deposit, in free or bound form. Stannous fluoride provides fluoride and stannous ions where the latter act as an antimicrobial agent.[131]

β-TCP (tricalcium phosphate)

Studies have shown that the combination of TCP with fluoride can provide greater enamel remineralization and build more acid-resistant mineral relative to fluoride alone. When it is used in toothpaste formulations, a protective

barrier is created around the calcium, allowing it to coexist with the fluoride ions. During toothbrushing, TCP comes into contact with saliva, causing the barrier to dissolve and releasing calcium, phosphate, and fluoride.

Functionalized TCP

Functionalized TCP is a low-dose calcium phosphate system that is incorporated into a single-phase aqueous or non-aqueous topical fluoride formulation. It provides a barrier that prevents premature TCP–fluoride interactions and also facilitates a targeted delivery of TCP when applied to the teeth.

Dicalcium Phosphate Dihydrate (DCPD)

DCPD is a precursor for apatite that readily turns into fluorapatite in the presence of fluoride. Researches have shown that inclusion of DCPD in a dentifrice increases the levels of free calcium ions in the plaque fluid, and these remain elevated for up to 12 hours after brushing, when compared to conventional silica dentifrices

ACP (Amorphous calcium phosphate)

ACP is the initial solid phase that precipitates from a highly supersaturated calcium phosphate solution and can convert readily to stable crystalline phases such as octacalcium phosphate or apatitic products. It plays as a precursor to bioapatite and as a transient phase in biomineralization

CPP–ACP is a two-phase system which when mixed together reacts to form the ACP material that precipitates onto the tooth structure and elevates calcium levels in the plaque fluid. GC Tooth Mousse Plus™ and MI Paste Plus™ are formulations of CPP–ACP with incorporated fluoride to a level of 900 ppm, where the fluorides give additive effects in reducing caries experience.[21] It is available as toothpastes, chewing gum, lozenges, and mouth rinses.

Bioactive Materials

A bioactive material is defined as a material that stimulates a beneficial response from the body, particularly bonding to host bone tissue and to the formation of a calcium phosphate layer on a material surface. Bioglass (BG) is a class of bioactive material which is composed of calcium, sodium, phosphate, and silicate. They are reactive when exposed to body fluids and deposit calcium phosphate on the surface of the particles.[131]

Nanomaterials

Nanoparticles have better ion release profiles than microparticles. Since it is difficult to directly use nanomaterials to remineralize teeth in the oral environment, these materials are often added to restorative materials as inorganic fillers, such as resin composites to release calcium, phosphate, and fluoride ions for remineralization of dental hard tissues.[132]

NanoHAP Particles

Nano-sized HAP (n-HAP) is similar to the apatite crystal of tooth enamel in morphology and crystal structure. So it can be substituted for the natural mineral constituent of enamel for repair biomimetically

ACP Nanoparticles

They are small spheroidal particles with a dimension in the nanoscale (40–100 nm). ACP nanoparticles, as a source of calcium and phosphate ions, have been added to composite resins, ionomer cements, and adhesives

5. Temporomandibular Disorder

Temporomandibular disorders (TMD) are a range of both musculoskeletal and degenerative conditions. The leading causes of TMD are an altered position of the intra-articular disc or abnormal muscle hyperactivity. The main symptoms include joint clicks, limitation of movement, and facial muscle tension, known as orofacial pain. Treatment options vary according to the joint's internal imbalance severity and osteodegenerative phenomena. There are non-invasive or minimally invasive treatments for patients in the early stages of internal disorders. While in advanced or complex cases, patients require joint replacements or invasive therapies.

NSAIDs treat patients with mild to acute TMJ inflammation, especially in disc dislocation cases, without reduction or acute trauma. These drugs must be taken for a minimum of 2 weeks. Several NSAIDs are used extensively in the dental field, such as ibuprofen, naproxen, diflunisal and ketorolac.[133]

Widely used drugs are opioids, and their action against moderate pain is very effective. In the treatment of TMD, their use is mild to severe pain. The most widely used opioid drugs are codeine, oxycodone, and hydromorphone for severe pain. If the oral route is not preferred, one can opt for the fentanyl patch.

Corticosteroid drugs have a chemical similarity to cortisol which is produced by the adrenal glands. These drugs are used to treat moderate to severe TMD. They block phospholipase A2, decreasing the production of prostaglandins and leukotrienes. Corticosteroids can be injected directly into the joint capsule or orally. Usually, intra-articular cortisone solutions are diluted with a local anaesthetic.

Other drugs used in the treatment of TMD are centrally-acting muscle relaxants. They are used and administered in patients with chronic pain. These drugs cause drowsiness and are therefore taken before going to bed. The most common muscle relaxants are carisoprodol, cyclobenzaprine, metaxalone and methocarbamol. Antidepressive drugs are widely used for the management of TMD pain. Tricyclic antidepressant drugs and selective serotonin reuptake inhibitors appear to be the most important. Several studies evaluate the efficacy of tricyclic antidepressants in the management and control of chronic pain. Furthermore, patients with chronic pain also suffer from depression and sleep disturbances. The exact mechanism of action is not fully known; however, it is probably given by inhibiting the serotonin reuptake and noradrenaline at the synaptic level of the central nervous system. The drugs used are amitriptyline, nortriptyline and desipramine.[133]

Benzodiazepines are drugs used to treat sleep disorders and muscle disorders. Several clinical trials have evaluated the efficacy of these drugs in treating TMD compared to a placebo. hese drugs have been discouraged due to adverse reactions such as sleepiness. Furthermore, these drugs show tolerance and dependence whereby the sudden discontinuation leads to symptoms including anxiety, agitation, restlessness, insomnia and convulsions.

6. Trigeminal Neuralgia

Trigeminal neuralgia (TN) is one of the most common causes of facial pain seen in dental and neurologic practices. The International Headache Society (IHS) defines TN as a “unilateral disorder characterized by brief electric shock-like pains, abrupt in onset and termination, and limited to the distribution of one or more divisions of the trigeminal nerve”. A variety of medical and surgical treatments are available for TN. Antiepileptic drugs (AEDs) are considered first line therapy for TN with different efficacy, and carbamazepine is the drug of choice. Surgical interventions are reserved for patients who do not respond to adequate medical therapy. Microvascular decompression (MVD), percutaneous trigeminal rhizotomies, and gamma knife radiosurgery (GKRS) are possibly effective in the treatment of TN.[134]

According to current evidence-based treatment guidelines published in 2008 from the AAN and EFNS, carbamazepine is established as effective (level A) and oxcarbazepine is probably effective (level B) for controlling pain in classic TN. These guidelines recommend carbamazepine (200-1200 mg/d) and oxcarbazepine (600-1800 mg/d) as a first-line therapy for classic TN.

Carbamazepine acts by inhibiting voltage-gated sodium channels, thereby reducing the excitability of neural membranes. In newly diagnosed cases of TN, the usual starting dose is 100 to 200 mg twice daily. The daily dose should be increased by 100 mg every other day until sufficient pain relief is attained or until intolerable side effects prevent further upward titration. The typical total maintenance dose is 300-800 mg/d, given in 2-3 divided doses. The maximum suggested total dose is 1200 mg/d. The dose may be tapered once pain is controlled, since remission may occur. Extended-release carbamazepine is useful as a night dose in patients with pain attacks during sleep, as drug levels do not fall. This not only keeps patients pain free during sleep but may reduce side effects. Common side effects include sedation, dizziness, nausea, vomiting, diplopia, memory problems, ataxia, elevation of hepatic enzymes, and hyponatremia, which may contraindicate it for elderly patients.[134]

Oxcarbazepine is a keto-analogue of carbamazepine that is rapidly converted into its pharmacologically active 10-monohydroxy metabolite. The keto derivative of

carbamazepine does not pass through the liver cytochrome system, resulting in an improved side effect profile and fewer drug interactions than with carbamazepine. Oxcarbazepine is an acceptable alternative to carbamazepine, which may have provided pain relief but has caused unacceptable adverse effects. Better tolerability can also be considered an advantage over carbamazepine. Oxcarbazepine can be started at 150 mg twice daily. The dose can be increased as tolerated in 300 mg increments every third day until pain relief occurs. Maintenance doses range between 300-600 mg twice daily. The maximum suggested total dose is 1800 mg/d.[134]

Second-line treatment is based on very little evidence. Three drugs are included in this class - lamotrigine, baclofen, and pimozide.[135]

The initial dose of lamotrigine is 25 mg twice daily and can be increased gradually to a maintenance dose of 200-400 mg/d in 2 divided doses. The dosage required for adequate pain relief varied widely from 100-400 mg/d. Common side effects are sleepiness, dizziness, headache, vertigo, and ataxia.

Baclofen, a skeletal muscle relaxant, is a GABA analogue that activates GABAB receptors and thus depresses excitatory neurotransmission. It is effective in controlling pain in TN at a dose of 60-80 mg/d. Baclofen can be used alone or in combination with carbamazepine. The initial dose is 10 mg/d for 3 days, which can be increased to 10-20mg /d every 3 days if needed. The maximum tolerated dose is 60-80 mg/d, administered 3-4 times per day. Typical side effects of baclofen include drowsiness, dizziness, weakness, fatigue, nausea, hypotension, and constipation. Abrupt discontinuation of baclofen can cause withdrawal symptoms

Third-line therapy

The newer AEDs tested within the past few years are gabapentin, pregabalin, topiramate, and levetiracetam[136]

Gabapentin, a GABA receptor agonist, acts primarily on presynaptic calcium channels of neurons to inhibit the release of excitatory neurotransmitters. Treatment can be started at a dose of 300 mg/d and may be gradually increased by 300 mg every 2-3 days as tolerated. For maximum efficacy, the dose can be increased to 1800 mg/d. Pregabalin is an analog of GABA, structurally related to gabapentin.

7. Conclusion

Geriatric prosthetic patients are mostly affected with different systemic diseases, so special care should be given during the prosthetic treatment phase. The prosthodontist might have to handle a medical emergency which arises while patients are in dental chair, which is another major factor.

So, clinician should practically apply the knowledge of basic pharmacology during operative procedure, prevent the incidence of drug interaction and thereby improve patient care. Therefore, a solid foundation in basic pharmacology, familiarity with pharmacotherapeutics, and an up-to-date understanding of the most recent developments in medicinal agents are essential for a competent and effective practitioner.

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