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Review Article

Odontological Drugs and Their Action on Some Biological Organisms

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Abstract

Odontology is the study of teeth, of theirs diseases and treatment of these. Many odontological drugs are commonly used in dental practice. Antibiotics are indicated for the treatment of odontogenic infections, oral non-odontogenic infections, as prophylaxis against focal infection, and as prophylaxis against local infection and spread to neighboring tissues and organs. In addition to antibiotic, antifungals (drugs for classes azoles, imidazoles and polyenes), antiviral such as antimicrobial mouthwashes and nucleases inhibitors are also indicated for the treatment. These drugs prescription is almost invariably associated with the prescription of Nonsteroidal Antiinflammatory Drugs (NSAIDs), topical corticoids, local anaesthesic for odontological pain and/or Sodium Fluoride for dental caries. Odontalogical drugs act on several levels of metabolism either of microorganisms' constitutive material (e.g. wall, membrane, cytoplasm and nuclear materials for antibiotics, antivirals, antifungals and oxidizing substances) to destroys them or of humans system cells (receptors, enzymes, hormones for painful, inflammation, local anaesthesic and dental building drugs) to inhibit or stimulate them for the best functioning.

Keywords: Odontological drugs; Nonsteroidal Antiinflammatory Drugs; Microorganisms

Introduction

Odontology is the study of teeth, of theirs diseases and treatment of these. Dental practitioners should only prescribe within their competence and must make an appropriate assessment of the patient's condition, taking into account their medical history and any current medication, when prescribing [1]. Dental prescriptions provide short-term treatment or treatment specifically for surgical procedures; nevertheless, dentists require knowledge about drugs and must follow the international rules for prescribing [2]. There are no clinical indications for drugs which have controlled drug prescription requirements to be prescribed in primary dental care [1]. The most frequent reason for prescription is infection, when, in fact, pain is the main reason that patients go to the dentist. Often, pain is the result of infection; it should always be treated. It is very important to use appropriate diagnostic methods to differentiate the type and the origin of the pain so that proper treatment can be provided [2]. The most prescribed drugs in dentistry are the local anesthetics (Lidocaine) used during dental procedures, antibiotics (Amoxicillin, Ampicillin, Penicillin V, Clindamycin, doxycycline, Erythromycin, Clarithromycin, Metronidazole), NSAIDs (Ibuprofen, Paracetamol, Ketorolac, Naproxen, Diclofenac, Aspirin), antifungals (Fluconazole, Miconazole) and antiviral (Hydrogen peroxide, Aciclovir, Penciclovir). Ibuprofen, Paracetamol, penicillin and amoxicillin are the most used [2]. It has been estimated that 10% of all antibiotic prescriptions are related with dental infections [3]. In addition to these drugs, topical corticoids (Hydrocortisone, Betamethasone), analgesic (Diazepam) are also used for the treatment of dental pain and Sodium fluorine for dental caries. These drugs act either on microorganisms (bacterial, fungi or viral) or human system (receptors of cell, hormons, enzymes or odontostomatological tissues). Interactions of some of those drugs prescribed in dental practice are almost harmful.

Drugs for Bacterial Infections

While there are some classes of completely synthetic antibacterial agents, most drugs in use today are antibiotics, that is, natural compounds of microbial origin or derivatives thereof [4]. Despite the high incidence of odontogenic infections, there are no uniform criteria regarding the use of antibiotics to treat them [3]. Antibiotics are isolated from both prokaryotic and eukaryotic soil microorganisms, which use them as chemical weapons in their struggle against one another for the same ecological niche. There are literally thousands of known antibiotics. Most antibiotics are toxic for both bacterial and eukaryotic cells, including human ones [4]. Specific damage to bacteria is particularly feasible when a substance interferes with a metabolic process that occurs in bacterial but not in host cells [5]. Antibiotics are appropriate for oral infections where there is evidence of spreading infection (cellulitis, lymph node involvement and swelling) or systemic involvement (fever, malaise) [1]. According to Poveda-Roda et al. [3], antibiotics are typically prescribed in dental practice for some of the following purposes: (a) as treatment for acute odontogenic infections; (b) as treatment for non-odontogenic infections; (c) as prophylaxis against focal infection in patients at risk (endocarditis and joint prostheses); and (d) as prophylaxis against local infection and systemic spread in oral surgery. The most classes of bacterial drugs used for odonto-stomatological treatment are: penicillins, nitro-imidazoles, macrolides, lincosamides and tetracyclines.

Penicillins

Various penicillins are obtained through chemical acylation on amine function of 6 - aminopenicillanic acid, which is obtained

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by fermentation [4]. 6-aminopénicillanic acid is the basic nucleus of penicillins. Penicillins, β-lactam antibiotics, are obtained from cultures of *Penicillium notatum* (mold fungi). They disrupt cell wall synthesis by inhibiting transpeptidase, it is an inhibitors of cell wall synthesis. When bacteria are in their growth and replication phase, penicillins are bactericidal; as a result of cell wall defects, the bacteria swell and burst. It is obvious that penicillins exert their bactericidal action on growing or multiplying [5]. Penicillins are generally well tolerated [5]. As for antibiotics, penicillin, mainly amoxicillin which is appropriate since it is the first choice in dentistry [3].

Amoxicillin, one derivative of penicillin G, is an acidoresistant aminopenicillin with large spectrum. It is usually effective at treating of dental abscesses usually caused by viridans *Streptococcus* spp. or Gram-negative organisms, and is as effective as phenoxymethylpenicillin or penicillin V but is better absorbed [1]. Amoxicillin can be protected from destruction by penicillinase by combination with inhibitors of penicillinase (*clavulanic acid*, *sulbactam*, *tazobactam*) [5].

Nitro-imidazoles

Imidazole is weakly basic. The nitrogen atom N3 can bind proton to form imidazolium cation. The N1 nitrogen atom is not basic because its electron pair participates in conjugation with the double bonds [6]. 5-nitroimidazoles are semi-synthetic derivatives of the azomycin produced by Streptomyces which have an antifungal or antiparasitic activity. The addition of a nitro substituent in position 5 lead to antibacterial activity of these molecules.

Nitro-imidazole derivatives, such as metronidazole, damage DNA by complex formation or strand breakage. This occurs in obligate anaerobic bacteria. Under these conditions, conversion to reactive metabolites that attack DNA takes place (e. g., the hydroxylamine shown) [5]. Inside anaerobic cells, nitroimidazoles are reduced by ferredoxin to nitro radical anions: $R\text{-}NO_2$ + ferredoxin-Fe^{2+} $R\text{-}NO^{2-}$ + ferredoxin-Fe^{3+}.These radicals can react with DNA and cause strand breaks [4]. The effect is bactericidal [5]. Aerobic human or bacterial cells are not affected because they don't contain sufficiently strong reducing agents. In addition, any radicals that may be formed will be reoxidized by oxygen: $R\text{-}NO^{2-} + O_2$ $R\text{-}NO_2 + O^{2-}$ The superoxide formed in the second reaction will be scavenged by peroxidase [4]. Metronidazole is well absorbed via the enteral route; it is also given intravenous or topically (vaginal insert). Because metronidazole is considered potentially mutagenic, carcinogenic, and teratogenic in humans, it should not be used for longer than 10 days, if possible, and should be avoided during pregnancy and lactation [5].

Macrolides

Macrolides suppresses advancement of the ribosome. Their action is predominantly bacteriostatic and is directed mainly against Gram-positive organisms. Intracellular germs such as chlamydias and mycoplasms are also affected. Macrolides are effective orally [5]. Among other uses, it is suitable as a substitute in allergy or resistance to penicillin. Gastrointestinal disturbances may occur. Because of inhibition of CYP isozymes (CYP3A4) the risk of drug interactions is present [5].The prototype of this group is *erythromycin*.

Clarithromycin like azithromycin are new semi-synthetic derivatives of erythromycin with similar activity; however, their elimination is slower [5,6]. Their macrocycle with a size made up of 14 atoms carries a lactone function, onto which are grafted two or more sugars, one of which is amino. These are basic molecules [6].

Lincosamides

The lincosamides, nowadays represented by lincomycin and its derivative 7-chloro-7-deoxy-, clindamycin, are constituted a hygric acid alkylated in position 4 and substituted via an amide function by a group 6-amino thiooctopyrannoside. *Clindamycin* has antibacterial activity similar to that of erythromycin. It exerts a bacteriostatic effect mainly on Gram-positive aerobic, as well as on anaerobic pathogens. Clindamycin is a semisynthetic chloro analogue of lincomycin, which derives from a *Streptomyces* species. Taken orally, clindamycin is better absorbed than lincomycin, has greater antibacterial efficacy and is thus preferred. Both penetrate well into bone tissue [5].

Tetracyclines

Tetracycline family is considered the classical broad-spectrum antibiotic, which means that it is clinically effective against both Gram-positive and Gram-negative bacteria, both aerobic and anaerobic ones [4]. Tetracyclines inhibit the binding of tRNA-AA complexes and their action is bacteriostatic. They are absorbed from the gastrointestinal tract to differing degrees, depending on the substance. For example, absorption being for doxycycline and minocycline is nearly complete [5]. Concurrent ingestion of antacids or milk would, however, be inappropriate because tetracyclines form insoluble complexes with plurivalent cations (e.g., Ca^{2+}, Mg^{2+}, Al^{3+}, $Fe^{2+/3+}$) resulting in their inactivation. Tetracyclines accumulate in growing teeth and bones to cause an irreversible yellowbrown discoloration of teeth and a reversible inhibition of bone growth [5]. It also increases photosensitivity of the skin and causes *hepatic damage*, mainly after i.v. administration. Because of these adverse effects, tetracycline should not be given after the second month of pregnancy and not prescribed to children aged 8 years and under.

Tetracyclines owe their name to their common tetracyclic structure (naphthacene carboxamide nucleus), on which are substituted substituents in positions 5, 6, 7. Based on their half-life, doxycycline is a second generation tetracycline.

Drugs for Fungal Infections

The most commonly used antifungal drugs are polyene antibiotics - in particular, amphotericin B - and synthetic triazoles such as fluconazole. The selective toxicity of both polyenes and triazoles is based on the occurrence of ergosterol in the fungal cell membrane, where it takes the place filled by cholesterol in mammalian cells [4]. Infections due to fungi are usually confined to the skin or mucous membranes: local or superficial mycosis [5]. Mycoses are most commonly due to dermatophytes, which affect the skin, hair, and nails following external infection, and to *Candida albicans*, a yeast organism normally found on body surfaces, which may cause infections of mucous membranes, less frequently of the skin or internal organs when natural defenses are impaired [5]. Polyene antibiotics bind directly to ergosterol to induce membrane damage. Inhibition of ergosterol biosynthesis is the therapeutic principle of triazoles such as fluconazole and ketoconazole. The target enzyme is 14-α sterol demethylase, which is a cytochrome P450 enzyme [4].

Imidazoles & triazoles

Imidazole and triazole derivatives inhibit synthesis of ergosterol,

an integral constituent of cytoplasmic membranes of fungal cells. Fungi stop growing (fungistatic effect) or die (fungicidal effect). The spectrum of affected fungi is very broad [5]. Angular cheilitis in denture-wearing patients is usually caused by infection with *Candida* spp and there is an associated denture stomatitis that should be treated concurrently. Unresponsive cases can be treated with miconazole cream or ointment, except those patients taking warfarin or statins [1]. Completely synthetic, they have undergone rapid evolution. They are classified into imidazoles or triazoles depending on 2 or 3 nitrogen respectively within the azole cycle. All the azoles in development nowadays are constituted by triazoles.

Because they are poorly absorbed and poorly tolerated systemically, most imidazoles are suitable only for topical use (clotrimazole, econazole, oxiconazole and other azoles). Fluconazole and itroconazole are newer orally effective triazole derivatives. Owing to its hydroxyl group, fluconazole is sufficiently water-soluble to allow formulation as an injectable solution. Both substances are slowly eliminated (plasma t½ ~ 30 hours) [5]. Fluconazole and other triazoles, which act as inhibiting ergosterol biosynthesis, have the widest spectrum of activity and are most widely used in practice [4].

Polyene

Polyenes are macrocyclic structures which show a hydrophobic side by the presence of 4 to 7 conjugated double bonds, and a side made more hydrophilic by the presence of substituents hydroxyls and an internal ester cycle. The polyene antibiotics amphotericin B and nystatin are of bacterial origin (e.g: *Streptomyces nodosus*) [5]. Polyene antibiotics class of antibiotics that contain an extended polyene moiety and bind to ergosterol or other sterols in cell membranes [4]. They insert themselves into fungal cell membranes and bind directly to ergosterol to induce membrane damage (hydrophilic channels formation); it is an antibiotic ergosterol-dependent membrane permeabilization [4,5].

Amphotericin B is active against most organisms responsible for systemic mycoses. Because polyene antimycotics are nonabsorbable, it must be given by infusion, which is, however, poorly tolerated (chills, fever, CNS disturbances, impaired renal function, and phlebitis at the infusion site) [5]. Applied topically to skin or mucous membranes, amphotericin B is useful in the treatment of candidal mycosis. Because of the low rate of enteral absorption, oral administration in intestinal candidiasis can be considered a topical treatment. Likewise, *nystatin* is only used topically (e.g., oral cavity, gastrointestinal tract) against candidiasis [5].

Drugs for Viral Infections

They lack a metabolic system and depend on the infected cell for their growth and replication. Targeted therapeutic suppression of viral replication requires selective inhibition of those metabolic processes that specifically serve viral replication in infected cells [5].

Antimicrobial mouthwashes

Hydrogen peroxide (H_2O_2) is an unstable compound that decomposes easily to give water and oxygen [6]. If oxygen reacts with two electrons hydrogen peroxide is formed. When oxygen reacts with four electrons water is formed [4]. Hydrogen peroxide is an oxidant, free radical unstable, highly reactive which tends to make chain reactions [7] and toxic to cells [8]. Hydrogen peroxide acts as an oxidizing agent or an antimicrobial mouthwashes controls plaque accumulation if tooth brushing is painful and also helps to control secondary infection in general [1,6]. It has a weak bactericidal effect, and due to the release of oxygen can cleanse abrasions from unwanted particles [6]. Hydrogen peroxide also acts as mucosal ulceration and inflammation [1].

Chlorhexidine is used as an antimicrobial mouthwashes controls plaque accumulation if tooth brushing is painful and also helps to control secondary infection in general [1].

Nuclease inhibitors

Nuclease inhibitors are those compounds or drugs that inhibit the conversion of nucleic acids to nucleotides. They concern deoxyguanosine analoguous as aciclovir, penciclovir and ganciclovir which bear acyclic strand instead of sugar. The function of aciclovir guanine residue has pka about 9. The molecule is then also acid as phenol. Penciclovir is a less toxic analogue of ganciclovir, where the oxygen in the straight chain has been replaced by an isosteric methylene group.

Among virustatic antimetabolites, acyclovir has both specificity of the highest degree and optimal tolerability because it undergoes bioactivation only in infected cells, where it preferentially inhibits viral DNA synthesis. The high therapeutic value of aciclovir is evident in severe infections with herpes simplex viruses and varicella-zoster viruses. In these cases, it can be given by i.v. infusion. Aciclovir may also be given orally despite its incomplete (15-30%) enteral absorption. In addition, it has topical uses. Because host DNA synthesis remains unaffected, adverse effects do not include bone marrow depression [5].

Odontogenic Pain

Pain is a designation for a spectrum of sensations of highly divergent character and intensity ranging from unpleasant to intolerable. Pain stimuli are detected by physiological receptors (sensors, nociceptors) least differentiated morphologically, viz., free nerve endings [5]. Most dental pain is due to inflammation which those of the periodontal tissues occurs in response to the presence of dental plaque microorganisms and results in bleeding on probing [9]. Pain sensation can be influenced or modified as follows: Elimination of the cause of pain, Lowering of the sensitivity of nociceptors (antipyretic analgesics, local anesthetics), Interrupting nociceptive conduction in sensory nerves (local anesthetics), Inhibition of pain perception (opioids, general anesthetics) and Altering emotional responses to pain, i. e., pain behavior (antidepressants as co-analgesics) [5].

Non-steroidal anti-inflammatory drugs

Most odontogenic pain can be relieved effectively by Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), such as cyclooxygenase inhibitors ibuprofen and aspirin, which have anti-inflammatory activity [1]. Cyclooxygenases (Cox) localized to the endoplasmic reticulum are responsible for the formation from arachidonic acid of a group of local hormones comprising the prostaglandins, prostacyclin, and thromboxanes. NSAIDs (except ASA) are reversible inhibitors of Cox enzymes. These enzymes possess an elongated pore into which the substrate arachidonic acid is inserted and converted to an active

product. NSAIDs penetrate into this pore and thus prevent access for arachidonic acid, leading to reversible blockade of the enzyme. Traditional cyclooxygenase inhibitors inhibit both Cox-1 and Cox-2 and include the most common NSAIDs prescribed such as ibuprofen, aspirin, indomethacin, and diclofenac [4,5,7].

Paracetamol (acetaminophen), N-(4-hydroxyphenyl) acetamide, is a synthetic analgesic, antipyretic [6] and NSAID (less used). Called again pain killer or pain-killing, analgesic drugs exert effects at several stages of pain perception and transmission [5]. Paracetamol is a safe, well tolerated drug with few side effects when used as directed [10]. However, higher doses of paracetamol could be hepatotoxic [6]. In suicidal or accidental poisoning with acetaminophen (10g), the depleted store of thiol groups must be replaced by administration of acetylcysteine [5].

Ibuprofen, 2-(4-isobutylphenyl) propanoic acid, is the synthetic carbonic acid derivative of 2-phenylpropanoic acid. It is an analgesic (pain killer) and antipyretic with anti-inflammatory effects [4].

Diclofenac is carbonic acid which carboxyl group binds to serine 530, and replacement of this residue but not of arginine 120 or tyrosine 355 renders the enzyme insensitive to this inhibitor [4].

Aspirin with chemical name acetylsalicylic acid (2-acetoxybenzoic acid, acidum acetylsalicylicum) is an important and famous derivative of salicylic acid (2-hydroxybenzoic acid). In doses about 0.5-3 g/day it is a commonly used analgesic and antipyretic drug (e.g. Aspirin). In doses of approximately 100 mg/day it prevents aggregation of blood platelets [6]. Acetylsalicylic acid improves oral tolerability. Significantly reduces its ability to induce mucosal injury by acetylation of salicylic acid. After absorption of ASA into the portal circulation, the acetyl moiety is cleaved with a t½ of 15-20 minutes; salicylic acid causes a long-lasting blockade of COX-1-mediated thromboxane synthesis in platelets because of an irreversible acetylation of the enzyme [5].

Anxiolytic

An oral dose of a benzodiazepine may be used for premedication to aid anxiety management before dental treatment [1]. Benzodiazepines act on GABA a receptors in the central nervous system to hyperpolarize cells. They exhibit a broad spectrum of activity: they exert sedating, sleep-inducing, anxiolytic, myorelaxant, and anticonvulsant effects and can be used for induction of anesthesia. A typical metabolic pathway for benzodiazepines as exemplified by the drug diazepam. A long-acting substance such as diazepam is the drug of choice for controlled tapering of withdrawal. For longer lasting anxiolytic therapy, compounds should be selected that are eliminated slowly and ensure a constant blood level (e.g., diazepam). Binding of benzodiazepine agonists allosterically enhances binding of GABA and its action on the channel [5].

Topical corticosteroids

Some topical corticoids were prescribed by odontologist to treat mucosal ulceration and inflammation. The most important glucocorticoid is cortisol with three hydroxyl groups (11β, 17α, 21), in pharmacology known as hydrocortisone [6]. Hydrocortisone is a natural corticoid and others such as betamethasone and beclometasone are their synthesis analogous. Cortisol secretion is stimulated by hypophyseal ACTH [5] and its transformation from cortisone (a biologically inactive form that does not bind to glucocorticoid receptors) to cortisol also takes place in the liver [7]. This glucocorticoid steroid hormone affects metabolic regulation and inhibits inflammation [4]. Cortisol acts through nuclear steroid receptors and it has a significant effect on the immune reaction of the body, and has anti-allergic, anti-oedematous, anti-inflammatory and anti-exudative effects [7]. Angular cheilitis in denture-wearing patients is usually caused by infection with *Candida* spp and there is an associated denture stomatitis that should be treated concurrently. Unresponsive cases can be treated with hydrocortisone cream or ointment, except those patients taking warfarin or statins [1].

They are glucocorticoids drugs secrete by human adrenal glands [11], which play a crucial role in adaptation of the organism to stress [6]. The Adrenal Cortex (AC) produces the glucocorticoid cortisol (hydrocortisone) in the zona fasciculata and the mineralocorticoid aldosterone in the zona glomerulosa which also vitally important in adaptation responses to stress situations, such as disease, trauma, or surgery [5]. As glucocorticoids suppress the synthesis of substances released during inflammation (eicosanoids), they are widely used as anti-inflammatory drugs. They have also a catabolic effect on protein metabolism and stimulate liver gluconeogenesis [6]. Betamethasone used as a mouthwash, is suitable for extensive inflammation or ulceration but should not be swallowed to minimize the risks of systemic effects [1].

Local anesthetics

Local anesthetics reversibly inhibit impulse generation and propagation in nerves. In sensory nerves, such an effect is desired when painful procedures must be performed, e.g., surgical or dental operations. they are capable of inhibiting this rapid influx of Na^+; initiation and propagation of excitation are therefore blocked Most local anesthetics exist in part in the cationic amphiphilic form. Since local anesthetics block Na^+ influx not only in sensory nerves but also in other excitable tissues, they are applied locally. Depression of excitatory processes in the heart, while undesired during local anesthesia, can be put to therapeutic use in cardiac arrhythmias [5].

Lidocaine, an antiarrhythmic drug, is synthetic local anesthetics which causes their anaesthesia on surface body such as mucosa [6]. The channel block caused by lidocaine can be observed with single channel molecules. The drug reduces the conductivity of the open state, which is referred to as a fast block. In addition, it also slows down the reactivation of the channel, which is observed as a slow block [4]. Clinically used local anesthetics are either esters or amides. Lidocaine is broken down primarily in the liver by oxidative N-dealkylation, with hydroxylation of the nitrogen (tertiary amines case). It is rapidly degraded in the body by cleavage and must be given intravenously [5].

Dental Caries

Fluorine deficiency causes dental caries. Due to lack of fluorine fluoroapatite of enamel is not formed and tooth substance is susceptible to action of oral acids produced from food residues by oral bacteria. Moreover fluorine probably prevents acid production by bacteria by inhibiting glycolysis. Thus the lack of fluorine produces cavities due to solubilization of enamel by acids produced in oral cavitiy [7].

Table 1: Summary table of classes with examples of some odontological drugs and theirs interactions [1,10].

Types	Classes of Drug	Examples of Drug	Common Interacting Drug(s)	Recommendation
Bacterial Infections	Penicillins	Phenoxymethylpenicillin, 250mg; Amoxicillin Capsules, 250mg and Co-amoxiclav 250/125 Tablets.		
	Nitro-imidazoles	Metronidazole Tablets, 200mg	Alcohol	Advise patients to avoid alcohol
			Warfarin	Do not prescribe metronidazole for patients taking warfarin
	Macrolides	Erythromycin Tablets, 250mg; Clarithromycin Tablets, 250mg		
	Lincosamides	Clindamycin Capsules, 150mg	Calcium channel blockers (e.g. nifedipine), carbamazepine, ciclosporin, domperidone, statins (e.g. simvastatin), theophylline, warfarin	Do not prescribe macrolide antibiotics for patients taking these drugs
	Tetracyclines	Doxycycline Capsules, 100mg		
Fungal infections	Imidazoles	Fluconazole Capsules, 50mg	Statins, warfarin, theophylline	Do not prescribe azole antifungals for patients taking these drugs
	Triazoles	Miconazole Oromucosal Gel, 24mg/ml		
	Polyene	Nystatine Oral Suspension, 100,000 units/ml		
Viral Infections	Nuclease inhibitors	Aciclovir Tablets, 200mg, 1%; Penciclovir Cream, 1%		
	Antimicrobial Mouthwashes	Chlorhexidine Mouthwash, 0.2%; Hydrogen Peroxide Mouthwash, 6%.		
Pains	Non-steroidal anti-inflammatory drugs	Paracetamol Tablets, 500mg; Ibuprofen Tablets, 400mg; Diclofenac Sodium Tablets, 50mg	Antihypertensive drugs esp. beta-blockers (e.g. altenolol), ACE inhibitors (e.g. lisinopril) and diuretics	Avoid prescribing NSAIDs or ensure course is for 5 days or less
			Anticoagulants (e.g. warfarin, dabigatran)	Do not prescribe NSAIDs for patients taking these drugs
			Aspirin	Do not prescribe NSAIDs for patients taking a daily low dose of aspirin
			Lithium	Do not prescribe NSAIDs
			Methotrexate	Avoid prescribing NSAIDs
			Selective serotonin reuptake inhibitors (SSRIs e.g. fluoxetine)	Avoid prescribing NSAIDs
			Systemic corticosteroids	Only prescribe NSAIDs in combination with a proton-pump inhibitor
		Aspirin Dispersible Tablets, 300mg	Alcohol	Advise patients to avoid alcohol for 12 hours after taking aspirin
			Clopidogrel	Avoid prescribing aspirin
			Non-steroidal anti-inflammatories (e.g. ibuprofen, diclofenac)	Do not prescribe aspirin for patients taking these drugs
			Selective serotonin reuptake inhibitors (SSRIs e.g. fluoxetine)	Avoid prescribing aspirin
			Systemic corticosteroids	Only prescribe aspirin in combination with a proton-pump inhibitor
			Warfarin	Do not prescribe aspirin for patients taking warfarin
	Anxiolytic	Diazepam Tablets, 2mg		
	Topical corticosteroids	Hydrocortisone Oromucosal Tablets, 2.5mg; Betamethasone Soluble Tablets, 500µg/Clenil Modulite®‡, 50µg/metered inhalation (beclometasone pressurized inhalation, CFC-free)		
	Local anesthetics	Lidocaine Ointment, 5%		

Sodium fluoride NaF (*natrii fluoridum*) is a pair of ions Na^+ and F^-. Sodium fluoride in tablets is administered in small doses to treat increased caries, mainly in children; multiple doses are used in the prophylaxis or treatment of osteoporosis. The fluoride ion (F^-) is the component of fluorapatite in bones and teeth (100-200mgkg^{-1}). Fluoride is important for the treatment of osteoporosis and it only known deficiency symptom is the increased incidence of tooth decay. It confers significant resistance to dental caries, with the topical action of fluoride on enamel and plaque considered more important in this effect than the systemic action [1]. Fluorides inhibit the growth of bacteria and plaques on the surface of teeth; therefore, they are often added to toothpastes, dental flosses, chewing gums or oral rinses [6]. Dental fluorosis is characterized by mottled teeth. Enamel becomes rough and loses characteristic lustre. Stained, chalky white patches are seen on surface of teeth. Pitting occurs due to loss of enamel and tooth surface appear corroded [7].

Odontological Drug Interaction in Biological System

Drug interaction is the side effects which may express only when

a drug is mixed with certain other things [12]. It can involve a variety of mechanisms, including those where the normal concentration of a drug in tissue fluid is either reduced or increased due to the effects of a second drug on its ADME (Absorption, Distribution, Metabolism, Excretion) properties or those where the pharmacological effects of the first drug are modified (reduced or enhanced) due to the pharmacological effects of the second drug. However, when two or more drugs are given at the same time, they may exert their effects independently or they may interact [10]. Some of these drugs can't help but trigger side effects because of their chemical structure [12]. With the increase in the number of older patients who have retained some or all of their teeth and who may be on one or more long-term medications, identifying potential drug interactions that may occur between drugs prescribed in dental practice and the patient's current medication is increasingly important [10]. Dentists are aware of potentially harmful interactions when prescribing some drugs. The most frequent interactions with side effects observed (Table 1) in dental practice are the incidence of myopathy after prescribing azoles and clarithromycin in those taking statins, the asthma symptoms exacerbated following the use of NSAIDs and the interactions of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), carbamazapine, azole antifungals and antibiotics with warfarin.

Conclusion

Among many odontological drugs, antibiotics, antifungal, antiviral, NSAIDs, topical corticoids, analgesic, local anaesthesic and Fluoride are commonly used in dental practice. Each of these drugs have or no a specificity action to destroys microorganisms or to inhibit or stimulate many parameters (receptors, enzymes, hormones, nerve impulse, dental cells synthesis) of body system for its good functioning.

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