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STEROIDS IN IMPACTED MANDIBULAR THIRD MOLAR SURGERIES: A NARRATIVE REVIEW

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ABSTRACT

Background: Corticosteroids have been widely used by oral surgeons for reducing swelling caused by wisdom teeth surgery. However, they have not been proven to decrease pain. This study was aimed at reviewing previous studies pertaining to corticosteroids and its relief effect following surgical extraction of impacted mandibular third molar. **Objective:** This review examined the effect of corticosteroid on the postoperative sequelae following surgical extraction of impacted mandibular third molars. **Material and methods:** An electronic search was undertaken in PUBMED from 2010-2026 for appropriate English papers on effect of corticosteroids following surgical extraction of impacted mandibular third molar based on a series of keywords in different combinations. Prospective, as well as retrospective studies (randomized/nonrandomized clinical trials, meta-analysis, cohort studies, case-control studies, and case reports) were considered. **Results:** Forty-five acceptable, relevant articles were selected for review. The review identified the various types and most commonly used steroids, routes of administration, indications, contraindications, adverse effect and complications in the management of post-operative sequelae following impacted mandibular third molars extractions **Conclusion:** The study revealed that administration of corticosteroids pre- or post-operatively led to significant improvement on post-operative sequelae following third molar surgical extraction.

KEYWORDS : Corticosteroids, mandible, impacted third molar, surgical extraction.

INTRODUCTION

Surgical extraction of impacted mandibular third molars is one of the most common procedures in oral and maxillofacial surgery and is associated with significant tissue trauma and pronounced acute postoperative sequelae include pain, facial swelling, and trismus. These contribute to patient morbidity and adversely affect quality of lifel. The severity of these sequelae can be ameliorated effectively by pharmacological management2.

Since their discovery, corticosteroids have been used in almost all areas of medicine and by nearly every route 3. Corticosteroids are synthetic analogues of the natural steroid hormones produced by the adrenal cortex and include glucocorticoids and mineralocorticoids. The synthetic hormones have varying degrees of glucocorticoid and mineralocorticoid properties. Glucocorticoids are predominantly involved in metabolism and have immunosuppressive, anti-inflammatory, and vasoconstrictive effects. While mineralocorticoids regulate electrolytes and water balance by affecting ion transport in the epithelial cells of the renal tubules4.

The term corticosteroids in practice, however, are generally used to refer to the glucocorticoid effect. Glucocorticoids are primary stress hormones that regulate a variety of physiologic processes and are essential for life 5. Corticosteroids are among the most widely prescribed drug classes worldwide, with an estimated market of more than 10 billion USD per year 6. An estimated one percent of the total adult population in the United Kingdom receives oral glucocorticoids at any one time

Corticosteroids like dexamethasone, prednisolone, methylprednisolone and hydrocortisone have been widely used to minimize postoperative inflammation and improve recovery following third molar surgery and other oral surgical procedures7. Their anti-inflammatory effects are primarily achieved through inhibition of inflammatory mediators and suppression of the inflammatory cascade8. All these agents are potent synthetic glucocorticoid characterized by high anti-inflammatory efficacy, prolonged duration of action, and a favorable safety profile when administered for short periods9. They exerts their effects by inducing the anti-inflammatory protein annexin-1, reducing the production of inflammatory mediators such as prostaglandins, serotonin, bradykinin, and lymphokines, and suppressing the activity of inflammatory cells including lymphocytes, monocytes, and eosinophils10. Consequently, they are most frequently prescribed in dental practice for the prevention and management of postoperative inflammatory sequelae.11.

Therefore, this review aims to critically evaluate the current evidence regarding the benefits, indications, contraindications, methods of administration, and potential complications of corticosteroid therapy in the management of postoperative sequelae following impacted mandibular third molar surgery.

MATERIALS AND METHODS

An electronic search was undertaken in PUBMED without time restriction for appropriate English papers on steroids based on a series of keywords in different combinations: various routes of steroid application, its uses, side effects and complications. Prospective, as well as retrospective studies (randomized/nonrandomized clinical trials, meta-analysis, cohort studies, case–control studies, and case reports) were considered. The reference lists of original and review articles were also sought. Letters to the Editor, historical reviews, and unpublished articles were not considered. The structure of the current literature review was tailored to principally summarize the pertinent information.

RESULTS

Forty-five acceptable, relevant articles were selected for review. The review identified the various types and most commonly used steroids, routes of administration, indications, contraindications, adverse effect and complications in the management of post-operative sequelae following impacted mandibular third molars extractions.

DISCUSSIONS

Types and Most Commonly Used Steroids in Third Molar Surgeries

Steroids are a family of lipid-soluble molecules characterized by a core structure of four fused carbon rings. They are primarily classified by their function in the body and fall into three main categories: Corticosteroids, Sex Hormones, and Anabolic Steroids—12.

However, the scope of this review will cover only the corticosteroids which were divided into glucocorticoids and mineralocorticoids. The glucocorticoids includes: Dexamethasone , Prednisone, Cortisone, Hydrocortisone while the mineralocorticoids include the Aldosterone—12.

Prescription of drugs pertaining to pain control has been widespread after dental surgeries. However, since the outcome of dental surgeries is unpredictable, analgesic drugs like narcotics can be inadequate for pain relief, therefore, alternative method suggested is the use of glucocorticoids, which is one of the most effective medications to control postoperative pain and inflammation 13. Glucocorticoids are well known to suppress inflammation and are utilized in oral surgeries to relieve pain and reduce trismus and swelling. One of the most potent steroidal anti-inflammatory drugs is dexame-thasone14, synthetic glucocorticoids that has no mineralocor-ticoid effect. This drug has a minimal unfavorable impact on leukocyte chemotaxis, which indicates the movement of cells outside the circulatory system toward the site of injury15.

Dexamethasone

Glucocorticoids are well known to suppress inflammation and are utilized in oral surgeries to relieve pain and reduce trismus and swelling. One of the most potent steroidal anti-inflammatory drugs is dexamethasone14, a synthetic glucocorticoid that has no mineralocorticoid effect. This drug has a minimal unfavorable impact on leukocyte chemotaxis, which indicates the movement of cells outside the circulatory system toward the site of injury 15. Dexamethasone is at least 25 times more potent than hydrocortisone and at anti-inflammatory doses, dexamethasone does not have the sodium-maintaining properties of hydrocortisone which encourages fluid retention, hence the preference for dexamethasone use in oral surgery 16. Glucocorticoids such as dexamethasone also control the rate of synthesis of anti-inflammatory genes in molecular mechanisms and are similar to hormones produced by the adrenal glands17.

Indications

Dexamethasone has a wide variety of uses in the medical field. As a treatment, dexamethasone has been useful in treating acute exacerbations of multiple sclerosis, allergies, cerebral edema, inflammation, and shock. Patients with conditions such as asthma, atopic and contact dermatitis, and drug hypersensitivity reactions have benefited from dexamethasone18. Clinicians use it as a diagnostic agent for Cushing disease19. Dexamethasone is useful in the treatment of chemotherapy-induced nausea and vomiting. It is also used in the prevention and treatment of altitude sickness. It has also been used in the treatment of spinal cord compression due to metastases in oncological cases20. Concisely,in oral and maxillofacial surgery, it is used to treat TMJ disorders by reducing pain and inflammation via intracapsular injections, airway compromise resulting from massive swellings following facial space infections, intra-oral lesions like aphthous ulcers15.

Mechanism of Action

Dexamethasone is a potent glucocorticoid with very little or no mineralocorticoid activity21. Dexamethasone's effect on the body occurs in a variety of ways. It works by suppressing the migration of neutrophils and decreasing lymphocyte colony proliferation. The capillary membrane becomes less permeable, as well. Lysosomal membranes have increased stability. There are higher concentrations of vitamin A compounds in the serum, prostaglandin, and some cytokines (interleukin-1, interleukin-12, interleukin-18, tumor necrosis factor, interferon-gamma, and granulocyte-macrophage colony-stimulating factor) become inhibited21

Pharmacokinetics

Dexamethasone median time to peak concentrations (Tmax) is 1 hour (range: 0.5 to 4 hours). A high-fat, high-calorie diet decreased C max by 23% of a single 20 mg dose of dexamethasone, it is about 77% bound to human plasma proteins in vitro.The mean terminal half-life of dexamethasone is 4 hours (18%), and oral clearance is 15.7 L/hr following a single dose of dexamethasone. Dexamethasone is metabolized by CYP3A4, renal excretion of dexamethasone is less than 10% of total body clearance. Less than 10% of dexamethasone is excreted in the urine.

Route of Administration of Dexamethasone

Several routes to administering glucocorticoids had been attempted and studied in oral and maxillofacial surgeries

following surgical extraction of the impacted mandibular third molar. These include oral, submucosal, intramuscular, intra-alveolar, or intravenous routes8 and they are available in both oral and injectable forms. There remains no definite consensus about the best route for administration of glucocorticoid following IMTM surgical extraction22.

According to Al-Shamiri et al. 23, in a comparative study of 8 mg oral dexamethasone either preoperatively or postoperatively significantly lessens the postoperative sequelae (pain ,trismus and swelling) of third molar surgeries, with their findings leaning toward preoperative administration. Sabhlok et al. 8 used 4 mg of oral dexamethasone postoperatively every day for five days, demonstrating that it is useful for treating postoperative sequelae. According to Grossi et al. 24, submucosally administered dexamethasone achieved significant reduction in postoperative edema compared to other administration routes.

However, Al-khateeb et al., (2009) in a clinical evaluation of efficacy of submucosal infiltration of dexamethasone versus methylprednisolone following third molar surgery, found that post-operative pain and trismus were significantly reduced in patients who received methylprednisolone than in those who received dexamethasone.

Bamgbose et al.25 conducted a study using intravenous dexamethasone with a maximum of 16 mg within 24 hours. Their findings complemented the amplified effects of dexamethasone when used with diclofenac sodium after third molar surgery 25. Intramuscular injections were found to exhibit similar effects to the intravenous route. Klongnoi et al.22 mentioned enhanced postoperative pain relief and reduced swelling in impacted lower third molar surgeries with preoperative 8 mg intramuscular dexamethasone injection in the deltoid muscle.

A study by Majid and Mahmood 26, regarding the topical (intra-alveolar) application of dexamethasone, showed a significant reduction in postoperative sequalae.

The administration of dexamethasone through the pterygomandibular space was studied by Latt et al. 27 in 2016 while the sublingual route of dexamethasone was recommended by Gozali et al. 28 for patient comfort in 2017. It was claimed to have a faster onset and, at the 8 mg dose, was believed to be advantageous compared to the intramuscular method to alleviate effectively pain symptoms 28. The intra-masseteric approach was investigated by Nandini 29 using 8 mg dexamethasone, and they claimed that it was another way to reduce postoperative sequelae compared to the systemic approach.

Another new method was reported in 2020, where the intraosseous route was utilized and compared to the submucosal route. Kaewkumnert et al. 30 found that the latter was more efficacious than the former due to the possibility of heightened tension with discomfort created by intraosseous injection in the alveolar bone.

Adverse Effect

Although dexamethasone is generally well tolerated, it does have its drawbacks as a medication. The most frequently reported adverse effect by patients is the presence of insomnia after use. Other frequent adverse effects include acne, indigestion, fluid retention, electrolyte imbalances, weight gain, increased appetite, anorexia, nausea, vomiting, acne, agitation, and depression. There have been reports of adrenal suppression, arrhythmias, spermatogenic changes, glaucoma, hypokalemia, pulmonary edema, pseudotumor cerebri, and increased intracranial pressure. Steroid-induced osteonecrosis of the femoral head (long-term treatment). Hepatotoxicity: Likelihood score: A (well-established cause of liver injury when given in high doses, due to reactivation of hepatitis-B or a hepatocellular injury after high dose treatment31.

Contraindication

Dexamethasone use is contraindicated if patients have systemic fungal infections, hypersensitivity to dexamethasone, or cerebral malaria. Another contraindication is to administer live or live-attenuated vaccines during dexamethasone use. The immune system will be suppressed, placing the patient at risk of infection. It is still permissible to administer killed or inactivated vaccines. However, it bears mentioning that corticosteroids may attenuate immune response, and it is unpredictable if immunity develops32.

In patients with cirrhosis, diverticulitis, myasthenia gravis, renal insufficiency, or ulcerative diseases such as peptic ulcer disease or ulcerative colitis, it is important to use caution when prescribing dexamethasone. Recommendations include using dexamethasone cautiously during pregnancy as there is an increased risk of oral cleft formation.

Clinical experience has shown that large doses can increase blood pressure. In patients with recent myocardial infarction, it is advised to caution as an increase in free wall rupture of the left ventricle has been reported using dexamethasone.

Suppression of the hypothalamus-pituitary-adrenal axis (HPA axis) occurs with use, and therefore the rapid withdrawal of dexamethasone is not recommended. It is important to gradually increase and decrease any corticosteroid due to its effect on the HPA axis.

Latent diseases such as fungal (candida, cryptococcus, pneumocystis), parasitic (toxoplasmosis, amebiasis, Strongyloides), and bacterial (mycobacterium, Nocardia) infections may become active due to suppression of the immune system33.

Steroid use may inhibit bone formation and may lead to the formation of osteoporosis. Therefore, caution is necessary when prescribing dexamethasone to populations at higher risk for osteoporosis34.

Prednisolone

Another most commonly prescribed glucocorticoids is prednisolone. Prednisolone is a synthetic analogue of cortisol which has a half-life of 2.1- 3.5 hours. It is about 4 times as potent as hydrocortisone, and has duration of action of 18 – 36 hours and low mineralocorticoid activity35.

Indications

When used in low doses, prednisolone serves as an anti-inflammatory agent. At higher doses, they are considered as immunosuppressant. Corticosteroids inhibit the inflammatory response to a variety of inciting agents and, it is presumed, delay or slow healing They inhibit edema, fibrin deposition, capillary dilation, leukocyte migration, capillary proliferation, fibroblast proliferation, deposition of collagen, and scar formation associated with inflammation.

Prednisolone being a corticosteroid drug with predominant glucocorticoid and low mineralocorticoid activity, this makes it useful for the treatment of a wide range of inflammatory and autoimmune conditions such as asthma, uveitis, pyoderma gangrenosum, rheumatoid arthritis, urticaria, angioedema, ulcerative colitis, pericarditis, temporal arteritis, Crohn's disease, Bell's palsy, multiple sclerosis, cluster headaches, vasculitis, acute lymphoblastic leukemia, autoimmune hepatitis, lupus, Kawasaki disease,[22] dermatomyositis, post-myocardial infarction syndrome and sarcoidosis. Prednisolone can also be used for allergic reactions ranging from seasonal allergies to drug allergic reactions, as an immunosuppressant

for organ transplants, prednisolone in lower doses can be used in cases of adrenal insufficiency due to Addison's disease. However, in oral and maxillofacial surgery, apart from its use in managing trismus ,pain and swellings emanating from third molar surgeries, it is specifically used in the treatment of Juvenile idiopathic arthritis, recurrent aphthous stomatitis (RAS), Behcet's syndrome, pemphigus vulgaris, bullous pemphigoid, mucous membrane pemphigoid, erythema multiforme and Stevens-Johnson syndrome,.

Route of Administration of Prednisolone

Various routes of administration have been described for prednisolone, which include oral, intramuscular, intravenous and submucosal routes Prednisolone is available in both oral and injectable forms. It also has a long record of efficacy and safety. Prednisolone has been used extensively in oral and maxillofacial surgery for their active anti-inflammatory effects. Most previous studies reported administration of prednisolone through oral, intramuscular or intravenous routes.

Adverse Effect

These include: Increased appetite, weight gain, nausea, malaise, Increased risk of infection. Dermatological effects including reddening of the face, bruising/skin discoloration, impaired wound healing, skin atrophy, skin rash, edema, and abnormal hair growth.

Hyperglycemia; patients with diabetes may need increased insulin or diabetic therapies. Lower response to hormones, especially during stressful instances such as surgery or illness.

Change in electrolytes: rise in blood pressure, increased sodium and low potassium, leading to alkalosis. Gastrointestinal system effects: swelling of the stomach lining, reversible increase in liver enzymes, and risk of stomach ulcers. Muscular and skeletal abnormalities, such as muscle weakness/muscle loss, osteoporosis (see steroid-induced osteoporosis), long bone fractures and tendon rupture.

Neurological effects, including involuntary movements (convulsions),headaches, and vertigo.

Psychosocial behavioral and emotional disturbances with aggression being one of the most common cognitive symptoms, especially with oral use. Nasal septum perforation and bowel perforation (in some pathologic conditions).

Discontinuing prednisolone after long-term or high-dose use can lead to adrenal insufficiency.

Pharmacokinetics

Prednisolone has a large therapeutic window, considering the dosage required to produce a therapeutic effect is a few times higher than what the body naturally produces. Prednisolone is 70–90% plasma protein bound, it binds to proteins such as albumin.

Both prednisolone phosphate and prednisolone acetate go through ester hydrolysis in the body to form prednisolone. It subsequently undergoes the usual metabolism of prednisolone. Concomitant use of prednisolone and strong CYP3A4 inhibitors such as ketoconazole is shown to cause a rise in plasma prednisolone concentrations by about 50% owing to a diminished clearance. Prednisolone predominantly undergoes kidney elimination and is excreted in the urine as sulphate and metabolites of glucuronide conjugate.

Contraindication: Systemic fungal infections and hypersensitivity to prednisolone

Methylprednisolone

Methylprednisolone is an FDA-approved medication used for managing and treating various conditions. Methylprednisolone is a potent prescription corticosteroid that suppresses the immune system and rapidly reduces severe inflammation. It is widely used to treat acute asthma attacks, severe allergies, rheumatoid arthritis, and flare-ups of multiple sclerosis or inflammatory bowel disease.

Understanding the intricate pharmacology of this synthetic corticosteroid helps healthcare professionals tailor treatment plans to meet individual patient needs. This initiative enhances clinician proficiency regarding methylprednisolone therapy to ensure patient-centered care. A systematic review of methylprednisolone shows that it has a potency of 5 and are examples of intermediate-acting steroidal drugs with an action time between 12 and 36 hours. Meanwhile, the same review shows that methylprednisolone and dexamethasone are the most often corticosteroids used after impacted third molar surgery to decrease the initial inflammatory response. These can be administered by injection into the surgical area or systemically. This review has also shown that methylprednisolone, at standard doses, was considered more effective than dexamethasone and prednisolone for treatments and five times more potent than hydrocortisone. It suggests that methylation of prednisolone makes it more potent by aiding interaction with a cellular target rather than increasing its stability.

Indications of Methylprednisolone

The following indications for methylprednisolone administration approved by the U.S. Food and Drug Administration are categorized below by organ system.

In dental practice, apart from uses in managing post-operative sequelae (pain, trismus and swelling) following third molar surgeries by maxillofacial surgeons, oral medicine specialists uses methylprednisolone to treat Bell's palsy which is an idiopathic inflammation of the facial nerve (the seventh of twelve paired cranial nerves) which occurs almost always on one side of the face only. It is characterized by facial muscle weakness, hyperacusis, decreased tearing, and loss of taste on the anterior two thirds of the tongue. Because Bell's palsy results from inflammation and edema of the facial nerve, corticosteroids constitute the standard medicine in the treatment of this condition. For adults, prednisolone or methylprednisolone at doses of 1 mg/kg/day for 7 to 10 days, taken in divided doses in the morning and evening, is suggested.

Moreso, in the management of acute trigeminal nerve injuries, traumatic facial nerve paralysis, chronic facial pain, and allergic diseases involving maxillofacial area, methylprednisolone has been found useful.

In dermatology, it is used in treating atopic dermatitis, contact dermatitis, pemphigus vulgaris, pemphigus foliaceous, bullous pemphigus, erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis, while in endocrinology, it is used for congenital adrenal hyperplasia associated with cancer, hypercalcemia associated with cancer, Primary or secondary adrenocortical insufficiency (as a second-line treatment in conjunction with mineralocorticoids).

Methylprednisolone is used in gastroenterology to treat inflammatory bowel disease (acute exacerbations) ,while in hematology it can be used for autoimmune hemolytic anemia, Congenital (erythroid) aplastic anemia, Immune thrombocytopenia.

Neurologist used methylprednisolone to treat acute exacerbation of multiple sclerosis, whereas in ophthalmology, uveitis, scleritis, chorioretinitis, iritis and iridocyclitis, keratitis, optic neuritis, retinal vasculitis, allergic conjunctivitis has been treated using methylprednisolone.

Nephrotic syndrome (idiopathic or secondary to lupus nephritis) is treated with methyl prednisolone by nephrologist and pulmonologist have been using it to treat aspiration pneumonitis,. asthma, Chronic beryllium disease, Disseminated pulmonary tuberculosis (as an adjunct to antituberculous chemotherapy), Eosinophilic pneumonia, Symptomatic sarcoidosis and rheumatologist uses it to treat acute rheumatic carditis, acute gout, ankylosing spondylitis, dermatomyositis and polymyositis, psoriatic arthritis, rheumatoid arthritis (including juvenile type), Systemic lupus erythematosus .

Mechanism of Action

Methylprednisolone and its derivatives, methylprednisolone acetate succinate and methylprednisolone sodium, are intermediate-acting synthetic glucocorticoids. They are typically prescribed as anti-inflammatory or immunosuppressive agents. As an anti-inflammatory, methylprednisolone is 5 times as potent as hydrocortisone (cortisol) while demonstrating minimal mineralocorticoid activity. Methylprednisolone diffuses passively across the cellular membrane and binds to the intracellular glucocorticoid receptor. This complex translocates into the nucleus to interact with specific DNA sequences and enhance or suppress the transcription of particular genes. The methylprednisolone-glucocorticoid receptor complex blocks the promoter sites of pro-inflammatory genes, promotes the expression of anti-inflammatory gene products, and inhibits the synthesis of inflammatory cytokines; these actions are accomplished primarily by blocking the function of transcription factors, such as nuclear factor-kappa-B (NF-kB).

Pharmacokinetics

Depending on the administration route and dose, Methylprednisolone exhibits rapid and linear absorption, with peak concentrations achieved 48 minutes after administration. The onset of action of intravenous methylprednisolone occurs within 1 hour. Methylprednisolone has an oral bioavailability of approximately 88%.

Methylprednisolone has a volume of distribution (Vd) of 24 ± 6 L and a steady-state volume of distribution (Vss) of 27 ± 8.2 L. This drug primarily binds to albumin in plasma. It undergoes hepatic metabolism to produce metabolites such as 20-carboxymethylprednisolone and 6β-hydroxy-20α-hydroxymethylprednisolone. Methylprednisolone is primarily excreted in urine, following a bi-exponential pattern. The half-life elimination of intravenous methylprednisolone is 0.25 hours, with an oral half-life of 2-5 hours.

Available Dosages: Methylprednisolone is available in tablet formulations of 2 mg, 4 mg, 8 mg, 16 mg, and 32 mg, injectable suspension formulations at concentrations of 20 mg/mL, 40 mg/mL, and 80 mg/mL, and as a powder for injection in doses of 40 mg, 125 mg, 500 mg, 1000 mg, and 2000 mg.

Hydrocortisone

Hydrocortisone, also known as 11β,17α,21-trihydroxypregn-4-ene-3,20-dione, is a naturally occurring pregnane steroid.63 It is the pharmaceutical form of cortisol, the body's primary stress hormone. Hydrocortisone plays an essential role in regulating metabolism, immune responses, inflammation, and the body's response to physical and emotional stress.

Mechanism of Action: Hydrocortisone works by binding to intracellular glucocorticoid receptors. The resulting complex enters the cell nucleus, where it regulates the expression of specific genes. This action suppresses the production of inflammatory mediators such as prostaglandins, leukotrienes, and cytokines while reducing the activity of immune cells. Consequently, hydrocortisone decreases inflammation, swelling, redness, and allergic reactions.

Pharmacokinetics: It is mainly administered intravenously with peak onset at 4-6 hours, well absorbed after intramuscular administration. Widely distributed and metabolized in the liver. Half-life: Plasma, 1.5-2 hr; biologic, 8-12 hr.

Medical Uses

In oral and maxillofacial practice, hydrocortisone is used in minor surgical procedures like tooth extraction, biopsy, periodontal surgery, genioplasty, etc: about 25 mg of hydrocortisone is administered after the surgery, while in surgical procedures such as panfacial fractures or bimaxillary surgeries, massive swelling with airway compromise as seen in Ludwig angina, about 50-75 mg of hydrocortisone is administered for 1 to 2 days.

Meanwhile in major surgical procedures such as extensive head and neck tumor resection, deep neck dissection, mandibulectomy and maxillectomy surgeries with reconstruction, about 100-150 mg of hydrocortisone is administered for 2 to 3 days .

Hydrocortisone has numerous therapeutic applications, including: Treatment of adrenal insufficiency, such as Addison's disease, management of severe allergic reactions.

Treatment of inflammatory skin conditions, including eczema, dermatitis, and psoriasis, and management of autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus, treatment of inflammatory bowel diseases, including ulcerative colitis and Crohn's disease. Management of asthma and other inflammatory respiratory disorders and replacement therapy in patients with inadequate cortisol production .

Dosage Forms: Hydrocortisone is available in several formulations, including: Oral tablets, topical creams and ointments, injectable solutions, rectal creams, foams, and suppositories, eye and ear preparations. However, the choice of formulation depends on the condition being treated.

Side Effects

Pregnant women often use topical, inhaled, or systemic corticosteroid drugs for a variety of inflammatory and allergic conditions. Several investigations have reported that the use of corticosteroids during early pregnancy is associated with a 3- to 6-fold increased risk of orofacial clefts,. Although systemic corticosteroids are associated with a higher risk of orofacial clefts than topical corticosteroids, the latter is not without risk. It has been shown that application of hydrocortisone cream on eczematous skin is associated with significant increase in the level of plasma cortisol. In a study by Edwards et al., a significant association between topical corticosteroids and orofacial cleft was observed. Epidemiologic data have shown that low-to-moderate doses of inhaled corticosteroids taken during the first trimester of pregnancy are safe but raise concerns about high doses.

Although hydrocortisone is highly effective, prolonged or excessive use may cause adverse effects such as: Frequent: Insomnia, heartburn, nervousness, abdominal distention, diaphoresis, acne, mood swings, increased appetite, facial flushing, delayed wound healing, increased susceptibility to infection, diarrhea or constipation.

Occasionally, headache, edema, change in skin color, frequent urination. Rarely, tachycardia, allergic reaction (such as rash and hives), psychological changes, hallucinations, depression. Topical: Allergic contact dermatitis, purpura, itching, redness, irritation.

Adverse Reactions : Long-term therapy may cause hypocalcemia, hypokalemia, muscle wasting (especially in arms and legs), osteoporosis, spontaneous fractures, amenorrhea, cataracts, glaucoma, peptic ulcer disease, and CHF (Congestive heart failure)

Abruptly withdrawing the drug after long-term therapy may cause anorexia, nausea, fever, headache, sudden severe joint pain, rebound inflammation, fatigue, weakness, lethargy, dizziness, and orthostatic hypotension

Hydrocortisone offers several benefits: Rapid reduction of inflammation, Effective suppression of allergic reactions, Replacement of deficient cortisol in adrenal insufficiency, Availability in multiple dosage forms, Well-established safety profile when used appropriately.

Aldosterone

Aldosterone is a mineralocorticoid hormone (without anti-inflammatory or anti-allergic effect) produced in the zona glomerulosa of the adrenal cortex that influences water and salt regulation in the body. Aldosterone's primary function is to act on the late distal tubule and collecting duct of nephrons in the kidney, favoring sodium and water reabsorption and potassium excretion while also contributing to acid-base balance. To execute these tasks, it influences epithelial sodium channels, sodium-potassium exchange pumps, hyd-rogen ion ATPases, and bicarbonate-chloride antiporters . Because aldosterone is mainly for fluid retention management in the practice of medicine, it is not considered among the steroids used following third molar surgeries.

CONCLUSION

The use of steroids in the management of postoperative sequelae (pain, trismus and swelling) in dental practice following third molar surgeries cannot be over emphasized. It has been playing a central role in mitigating the postoperative effect of third molar surgeries on quality of life of patients. However, they are used either singly or in combination with other drugs, for the treatment of various diseases affecting oral and maxillofacial area.

However, corticosteroids carry the risk of potential side effects which are sometimes severe and life threatening. Therefore, benefits from corticosteroids should always be weighed against their potential risks in each patient.

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