Review Article

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Role of Steroids In Oral And Maxillofacial Surgery

Sathiyashree Sekar[1*] Rohini Thirunavukarasu[2] Saravanan Balasubramaniam[3] S. Gidean Arularasan[4]

[1]CRRI, Tamilnadu Government Dental College and Hospital, Chennai, Tamilnadu, India.
[2]Assistant Professor, Department of Oral and Maxillofacial Surgery, Tamilnadu Government Dental College and Hospital, Chennai, Tamilnadu, India.
[3]Assistant Professor, Department of Oral and Maxillofacial Surgery, Tamilnadu Government Dental College and Hospital, Chennai, India.
[4]Assistant Surgeon, Christian Medical College, Vellore, India.

ABSTRACT

Background: The beneficial effects of steroids were first reported by Hench in 1949[1]. They have a wide array of use in multiple medical specialities such as dermatology, rheumatology ,ophthalmology, immunology, oncology and oral and maxillofacial surgery. Glucocorticoids and their synthetic derivatives are hormones which at pharmacologic doses act as inflammatory and immunosuppressants. Corticosteroid is a versatile drug which has a wide area of application in the field of oral and maxillofacial surgery. It is used mainly for its anti-inflammatory, anti-allergic and analgesic action. Review of literature has shown that corticosteroids definitely have a profound effect on reducing post -surgical morbidities such as pain, swelling and oedema. The purpose of this paper is to review the various use of this versatile, multifaceted drug in the field of Oral and Maxillofacial Surgery.

Keywords: Corticosteroids, pain, swelling.

INTRODUCTION

The beneficial effects of steroids were first reported by Hench in 1949 [1]. They have a wide array of use in multiple medical specialities such as dermatology, rheumatology, ophthalmology, immunology, oncology and oral and maxillofacial surgery. Glucocorticoids and their synthetic derivatives are hormones which at pharmacologic doses act as inflammatory and immunosuppressants [2].

Cortical or hydrocortisone – the major glucocorticoid in humans is secreted by the adrenal cortex. The secretion is regulated by the adrenocorticotropic hormone (ACTH) from anterior pituitary gland. They have a range of physiologic actions such as influencing carbohydrate, protein, and fat metabolism, regulation of blood pressure and cardiovascular function and modulation of the immune system [3]. Corticosteroid drugs are synthetic analogues of cortical hormone. They act by binding to specific intracellular receptors on entering the target tissues and mimic the effects of the naturally occurring hormone.

The purpose of this paper is to review the various use of this versatile, multifaceted drug in the field of Oral and Maxillofacial Surgery.

1.Ulcerative and Vesiculobullous lesion:

Glucocorticoids because of their anti-inflammatory and immunosuppressive effects are mainly used as palliative in acute phase of the disease or as long term suppressors of the general host defence.

1.1 Recurrent apthous stomatitis:

The role of steroids in the management of recurrent apthous stomatitis is based on the assumption that apthous is a non-infectious inflammatory process. Steroids by directly acting on T-lymphocytes alter the response of effector cells to precipitants of

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*Correspondence Dr. Sathiyashree Sekar.
Tamilnadu Government Dental College and Hospital, Chennai, Tamilnadu, India.
Email: ssathiyashree@gmail.com

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immune pathogens. Topical steroids effective are fluocinonide, triamcinolone and clobetasol [4].

1.2 Behcet's Disease:

Immunosuppressive therapy is the key in treating Behcet's disease. The role of steroids as an anti-inflammatory is by their action on the neutrophils. It is used in the acute phase of the disease at a dose of 40-60mg /day [5].

1.3 Oral sub mucous fibrosis:

Steroids act as an immunosuppressant by opposing the action of soluble factors released by sensitized lymphocytes following activation by specific agents and as anti-inflammatory by preventing fibrosis by decreasing fibroblast proliferation and deposition of collagen [6].

1.4 Oral lichen planus:

Steroids function as an immunosuppressant by suppressing T cell function and decreasing IgG synthesis and also reduce tissue destruction by decreasing antigen release. They have no effect on hyperkeratosis lesions while atrophic and erosive type respond to local and systemic steroids [7].

1.5 Melkerson Rosenthal syndrome:

Steroids act by reducing the swelling and preventing persistent tissue oedema [8].

1.6 Post herpetic neuralgia:

The pain in the acute phase is due to abnormal pain discharge in the dorsal gray matter of the cord secondary to inflammation of segmental dorsal root ganglia. The persistence of pain may be associated with post inflammatory fibrosis in the root ganglia or sensory roots and steroids by inhibiting the inflammation in the acute phase are used in post herpetic neuralgia [9].

1.7 Pemphigus Vulgaris and Vegetans:

Steroids do not prevent the binding of pemphigus antibodies in the target tissue but interfere with lymphocyte subpopulation thereby they lower autoantibody production leading to reduced vascular permeability and decreased leakage of antibodies ,complement and inflammatory cells to the focus and inhibit the release of proteolytic enzymes thereby stabilizes the epithelial membrane. Lever and Schaumberg[10] recommended a two tier regimen of steroids for Pemphigus.

Mild disease- 40mg prednisolone and immunosuppressant (azathioprine) for one year every other day.

Severe disease: 200-400mg/day for 5 to 10 weeks and then 40mg /day for 1 week after that 30 mg/day for 1 week and then 2mg /day after which the patient is treated in the mild disease drug regimen [10].

1.8 Benign Mucous Membrane Pemphigoid:

Steroids as anti-inflammatory lower enzyme release , reduce cell migration and decrease

1.9 Pemphigus Vulgaris and Vegetans:

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Leakage of humeral factors [11].

1.10 Bullous pemphigoid:

In case of bullous pemphigoid for localized lesions topical steroids are used and for severe disease systemic steroids are used [12].

1.11 Lupus erythematosus:

Steroids reconstitute T suppressor cell function , cause disappearance of n-DNA antibodies from serum and in ANA titre. Subepidermal deposits of Igs partly resolve . There is decreased phagocytic activity , T cell function and reduced inflammatory action of immune complexes [13].

1.12 Psoriasis:

Topical and intralesional steroids act by epidermal proliferation [14].

1.13 Mucocele/ Infectious mononucleosis:

Steroids are used in a dosage of prednisolone 60mg/day [15].

2. Temporomandibular Disorders:

Temporomandibular disorders include a wide array of clinical disorders involving temporomandibular joints, masticatory muscles or both. They are one of the most common musculoskeletal disorders causing orofacial pain. The most common signs and symptoms are pain, tenderness, trismus , and mandibular movement .Oral corticosteroids used in temporomandibular disorders are hydrocortisone 20-240mg/day, prednisolone 5-60mg/day, dexamethasone 0.75-9mg/day or betamethasone 0.6-7.2mg/day [16]. They have been used mainly for acute TMJ discomfort, TMJ arthritis. Their role in TMJ disorders are mainly anti inflammatory which are multiple blockade of phospholipase A2 which decreases the production of pro inflammatory and leukotrienes and decreases the number and activity of proinflammatory cells. A recent study [17] concluded that in patients with TMJ closed lock ,medical management with a 6 day regimen or oral methyl prednisolone followed by 3-6 weeks of NSAID therapy worked equally as well over a 5 year period as arthroscopy or open joint therapy in reducing jaw pain and dysfunction as measured by Craniomandibular index.

In uncontrolled case studies [18]of arthritic TMJs intra articular steroids 0.7 ml of methyl

Prednisolone acetate 40mg/ml combined with local anaesthetic in children or 1ml of triamcinolone acetonide / 1ml in adult seem to significantly reduce pain and improve function. It has also been reported that aggressive use of intra articular steroids has lead to articular cartilage destruction , infection and disease progression due to anti anabolic effect [19]. Hence this mode of treatment should be reserved only for severe case and repeated injections must be avoided. Similarly, the use of oral corticosteroids should be limited to more than 2 weeks. Patients with severe disc interference disorders, inflammatory conditions such as capsulitis, synovitis, TMJ osteoarthritis / rheumatoid arthritis are those who benefit the most from this treatment regime. Intra articular injection should not be used more frequently than every 3-4 months.

3. Keloid and Hypertrophic scars:

Hypertrophic scars and Keloids are abnormal wound responses representing a connective tissue response to trauma, inflammation, surgery or burns. Hypertrophic scars are typically raised, red or pink and sometimes pruritic but they do not exceed the margins of the original wound whereas keloids infiltrate into surrounding normal tissue. Hypertrophic scars continue to evolve over time without a quiescent or regressive phase.

Intralesional corticosteroid injections have become a mainstay in the management of hypertrophic scars and keloids either alone or in conjunction with other therapeutic procedures. They soften and flatten keloids but cannot narrow hypertrophic scars or eliminate keloids. Intralesional steroids decrease fibroblast proliferation, collagen synthesis, glycosaminoglycan synthesis and pro inflammatory mediators. The most commonly used drug for steroid injection is triamcinolone acetonide(TA) -5-10mg/ml injected into the upper dermis of a developing hypertrophic scar every 3-6 weeks [20]. The injections are discontinued when the scar becomes stable or there are signs of tissue atrophy , hypopigmentation or telangiectasia . In case of pre-existing steroids three monthly intralesional injection of TA-40mg/ml mixed with equal parts of 2 % lidocaine [21] are used . Some authors recommend the addition of hyaluronidase to disperse the injection. The use of topical steroids is indicated only in superficial lesion such as those occurring from dermabrasion.

4. Central Giant Cell granuloma:

Central giant cell granuloma is by WHO definition " an intraosseous lesion consisting of cellular fibrous tissue that contains multiple foci of haemorrhage, aggregations of multinucleated giant cells and occasionally trabeculae of woven bone". It is an uncommon non neoplastic lesion accounting for 7 % of all benign lesions of the jaws in tooth bearing areas. It commonly affects children and young adults with a female predilection. They are

classified as aggressive or non aggressive based on the clinical and radiographic features. The treatment for CGGG depends on many factors such as location, size and radiographic appearance. CGGG can be managed surgically by enucleation or En bloc resection. The non surgical treatment modalities include radiation, systemic injection of calcitonin, alpha interferon. In 1988 Jacoway et al [22]first reported the use of intralesional corticosteroid indicating microscopic similarities between sarcoidosis and CGGGs suggesting that similar therapeutic regimen would be of value in treating both conditions. Carlos and Sedano [23] suggested that steroids may cause cessation of bone resorption by two possible mechanisms

By inhibition of extracellular production of lysosomal proteases.

By apoptotic action on osteoclast like cells.

The protocol suggested by Terry and Jacoway derived by Francis Howell [22] suggested intralesional injection of a mixture consisting of equal parts of triamcinolone acetonide(10mg/ml) and local anaesthesia . The recommended dose is 2ml/2cm of radiolucency. The injections are given in multiple locations throughout the lesion in a weekly regimen for atleast six week.

5. Steroids and third molar surgery:

Corticosteroids are found effective in reducing post operative pain, swelling and trismus in third molar surgeries. Normal daily production of cortisol is 15-30 mg however upto 300 mg can be produced in times of crisis. Steroids can be given by intravenous, intra muscular, subcutaneous routes. Studies have shown that intramuscular injections are effective irrespective of the site. Masseteric muscle [24] makes a good alternative site. Submucosal injections was found to be as effective as intramuscular route. Submucosal administered corticosteroids due to its proximity to the surgical site will act on eicosanoids subsequently and prevent subsequent inflammatory process . Another approach to introduce corticosteroids directly to the surgical wound was endo alveolar approach [25]. They are found to have direct inhibitory effect on signal transmission in nociceptive C fibres and ectopic neuroma discharge in injured nerve.

Oral steroids is the most common route. Current studies [26] suggest that preoperative intake 1hr before surgery was the preferred dose because corticosteroids are more effective before the onset of inflammatory process. Post operative administration may only prevent further propagation of inflammation but is unable to reverse inflammation that has occurred.

Results from various studies [27] have reported that corticosteroids consistently provided a favourable effect in reducing oedema. Oral prednisolone of more than 20mg were found to be effective in reducing trismus. Reduction of post operative pain is usual after resolution of oedema, in addition the analgesic effect could be attributed to the inhibitory effect of corticosteroids on prostaglandins. Alcantara et al [28] showed that 8 mg dexamethasone was superior to 40mg methyl prednisolone in reducing post operative pain. The analgesic effect was suspected to the by anti inflammatory and immune suppressive effect . The anti inflammatory action results in decreased production of anti inflammatory mediators which play a major role in amplifying and maintaining pain perception. Swelling in addition causes tissue tension which leads to pain. The reduction of swelling by corticosteroids have to found to amplify analgesic action by lessening pain that arises from tissue tension. Corticosteroids acts on prostaglandin differently than NSAIDS, hence better analgesia can be achieved with co therapy [29] .

Jarrah et al [30] reported a study which confirmed the synergistic effect of corticosteroid with ibuprofen in controlling post operative pain and trismus as opposed to corticosteroids alone.

6. Corticosteroids and orthognathic surgery:

Systemic corticosteroid administration in patients undergoing orthognathic surgery has been controversial. Perioperative corticosteroids are used in orthognathic surgery to prevent post operative complication. In a randomised prospective double blind trial [31] twenty three patients who required Bilateral Sagittal Split Osteotomy(BSSO)were split into three groups and were given either placebo, preoperative dexamethasone 16mg iv , 3 pre postoperative doses 8mg iv energy 6hr. In both dexamethasone groups there was a significant reduction in facial swelling

on postoperative day. However Munro et al [32]compared a preoperative dose of dexamethasone 0.5 mg/kg followed by a two day with placebo in children having mandibular on maxillary osteotomy and found no significant difference between control and experimental groups . Glucocorticoids have been found to reduce Post operative Nausea and Vomiting(PONV) by depleting 5-HT3 in neural tissue and prevent its release in the gut and they have a synergistic action with 5HT3 antagonists. One trial [33]evaluated the incidence of early postoperative nausea and vomiting following orthognathic surgery. Corticosteroids seemed to have a statistically significant effect on this outcome when compared with metoclopramide. Three trials [34,35,36] evaluated inflammatory markers following orthognathic surgery. C-reactive protein, fibrinogen, granulocytes statistically mere decreased with corticosteroid. One trial [37] showed a statistical improvement in mechanical-touch threshold with postoperative corticosteroid following orthognathic surgery.

7. Bell's palsy:

Bell's palsy is acute idiopathic unilateral paralysis of the facial nerve. Vascular, inflammatory and viral causes have been suggested from paired serologic analysis and studies of the cerebral ganglia, suggesting an association between herpes and onset of facial paralysis. The most commonly reported regimen is 1mg/kg of oral prednisolone and upto 70mg/day split into twice daily dosing. The starting dose was continued for 6 days then tapered off over subsequent 4 days [38]. Early treatment with acyclovir in combination with prednisolone is recommended as possibly effective to improve facial function outcomes.

8. Retrobulbar haemorrhage:

Anderson et al (1982) recommend the use of very large doses of steroids, as soon as possible after injury, to reduce circulatory spasm, oedema and cell necrosis. Dexamethasone sodium phosphate is administered intravenously at a level of 3-4mg/kg body weight as an initial loading dose, followed by 1-3mg/kg 6 hourly for the following 24 hours, reducing to 1mg/kg over the next 2 days. If the response is encouraging ,the therapy is continued on a reducing basis for a further 5-7days. However, if there is no response after 48 hours the steroids are discontinued [39].

9.Management of patients on steroids:

The treatment protocol for patients on steroids depends on dosage, route of administration, frequency, duration of therapy of steroids and also the procedure to be undertaken. The possibility of adrenal crisis is more in patients receiving more than 30mg cortisone daily for a continuous period of one week or longer .Patients who have discontinued this type of therapy within the past one year are also at risk. The higher the daily dosage , the longer the period of administration or shorter the period since discontinuation , the greater the potential risk for adrenal crisis.

+Adrenal crisis occurs due to the inability of the patient to produce sufficient amounts of endogenous of in response to stress. To overcome these Kalkwarf et al [40] suggested a "Steroid Prep". A steroid prep is given prior to the surgical procedure which is slowly tapered to a normal dosage level over a 2-3 day period.

Adrenal crisis is characterized by hypotension ,severe weakness ,hyperthermia ,hypoglycaemia, hyperkalemia , deterioration of CVS- clinically patients will exhibit progressive severe mental confusion , nausea and vomiting ,intense pain in abdomen, lower neck and legs if unchecked leads to loss of consciousness , coma , and death.

Treatment of adrenal crisis [40]

1. Hydrocortisone (iv)

a)100 mg over 30 seconds

b)followed by continuous infusion of 100mg per 8 hour period until stable

2. 5% Glucose in physiologic saline (iv)

a)1l over 2 hour time period

b) if patient is diabetic

insulin dosage have to adjusted

3. Vasopressor

4. Antipyretics

For patients undergoing general anaesthesia for minor surgery 100mg hydrocortisone i.m should be administered and in case of major surgery usual glucocorticoid medication should be maintained and 100mg hydrocortisone should be delivered as a pre – operative dose followed by 50mg 8hourly for 48 hours .

10.Side effects of corticosteroids :

Apart from serving its role as an anti-inflammatory drug, it has many adverse effects. The side effects of corticosteroids depends upon the route of administration, drug dosage and duration of the treatment. It includes fluid retention, adrenal insufficiency, impaired growth, electrolyte abnormalities, weight gain, osteoporosis, fractures, osteonecrosis, cataract, insomnia , myopathy, moon face , peptic ulcer, diabetes, glaucoma , psychological problems and increased susceptibility to infection.

Adverse effects of glucocorticoids includes hypopigmentation, sub cutaneous fat wasting, skin atrophy , oral thrush , contact dermatitis, telangiectasia and cushingoid effects from systemic absorption.

CONCLUSION

Corticosteroid is a versatile drug which has a wide area of application in the field of oral and maxillofacial surgery. It is used mainly for its anti-inflammatory, anti-allergic and analgesic action. Review of literature has shown that corticosteroids definitely have an profound effect on reducing post - surgical morbidities such as pain, swelling and oedema. However, long term corticosteroids have potential side effects which could be life threatening if not treated at earliest. Therefore, the role versus benefit ratio should be assessed in prescribing steroids for the patients.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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