BIOSCIENCES BIOTECHNOLOGY RESEARCH ASIA, June 2013. Vol. **10**(1), 339-343

Efficacy of Corticosteroids after Orthognathic Surgery

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(Received: 06 January 2013; accepted: 26 February 2013)

Steroids are used in post-operative pain, swelling, trismus, nausea, vomiting and reduction of inflammatory reaction after orthognathic surgery. The use of short term systemic corticosteroid therapy has become common after oral and maxillofacial surgery, especially orthognathic surgery. The most commonly administered types of corticosteroids are betamethasone, dexamethasone, and methylprednisolone, administered intravenously, orally or by injection into the masseter muscle. This article gives a review of the efficacy of corticosteroids after orthognathic surgery.

Key words: Steroids, Surgery, Pain, swelling, Orthognathic surgery, Postoperative.

The term "corticosteroid" or "corticoid" includes natural gluco and mineralo corticoids and their synthetic analogues (Tripathi, 2006). In the late 1960s and early 1970s, corticosteroid administration was advocated in conjunction with various oral surgical interventions to minimize edema and possibly decrease pain and promote neuro-regeneration (Guernsey and DeChamplain., 1971). Corticosteroids are drugs with one of the broadest spectrum of clinical utility. Corticosteroids are a class of chemicals that includes steroid hormones naturally produced in the adrenal cortex of vertebrates and analogues of these hormones that are synthesized in laboratories. Corticosteroids are involved in a wide range of physiological processes, including stress response, immune response, and regulation of inflammation, carbohydrate metabolism, protein catabolism, blood electrolyte levels, and behaviour(Tripathi., 2006). Uses of corticosteroid

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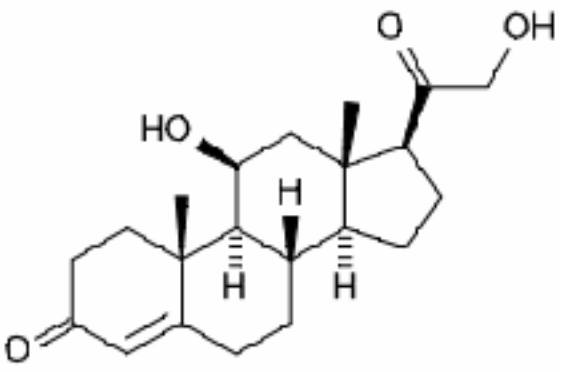
- To reduce inflammation (asthma, arthritis) and swelling (cerebral oedema)
- To suppress the immune response (systemic lupus erythematosis)
- To reduce nausea and vomiting (as in cancer chemotherapy)
- To reduce terminal pain (associated with cancer) as replacement therapy (in Addison's disease)
- Also used to treat brain edema, shock conditions, certain types of blood cancer (B- and T-cell lymphoma) (Jehn and Osborne., 1997) as well as conditions involving adrenal cortex insufficiency

As early as in the 1930s, the hormone cortisone was isolated from the adrenal glands and its efficacy for treatment of rheumatoid arthritis was empirically demonstrated in patients suffering from this debilitating disease. Due to their immunosuppressive effects, corticosteroids or glucocorticoid hormones are used to reduce organ rejection after transplantation and to treat auto-immune diseases, including multiple sclerosis, rheumatoid arthritis and inflammatory bowel



Scheme 1. Structure of Corticosteroid

disease. The synthesis and secretion of cortisol, the naturally occurring glucocorticoid in humans, is subject to a negative feedback loop and under tight control by a careful balance between adrenocorticotropin hormone, secreted from the pituitary gland in the brain and corticotrophin hormone secreted from the hypothalamus in a pulsatile and circadian fashion (Balsalobre *et al*., 2000). Corticosteroids inhibit leukocyte migration to sites of inflammation and thus reduce the general symptoms of inflammation (Cato and Wade., 1996). First known use of corticosteroid was in 1944 (Webster, 2012).

The corticosteroids are synthesized from cholesterol within the adrenal cortex. Most steroidogenic reactions are catalysed by enzymes of the cytochrome P450 family. They are located within the mitochondria and require adrenodoxin as a cofactor (except 21-hydroxylase and 17α-hydroxylase). Aldosterone and corticosterone share the first part of their biosynthetic pathway. The last part is mediated either by the aldosterone synthase (for aldosterone) or by the 11β-hydroxylase (for corticosterone). These enzymes are nearly identical (they share 11β-hydroxylation and 18-hydroxylation functions), but aldosterone synthase is also able to perform an 18-oxidation. Moreover, aldosterone synthase is found within the zona glomerulosa at the outer edge of the adrenal cortex; 11β-hydroxylase is found in the zona fasciculata and zona glomerulosa (Corticosteroids-Wikipedia).

DISCUSSION

The administration of corticosteroids is thought to inhibit mast cell production and secretion of cytokine, kinin and histamine. This should promote an inhibition of thromboxane and bradykinin, resulting in less blood vessel dilatation and less permeability (Huffman, 1977; Schaberg, 1984). Skelvred *et al*., (1982) have documented the beneficial uses of systemic corticosteroid therapy to suppress acute postoperative sequelae of surgical procedures. Schaberg *et al*., 1984 have described that methylprednisolone is effective for the control and management of postoperative facial edema following orthognathic surgery. Corticosteroids reduce edema by decreasing permeability of capillary endothelium and therefore reduce the amount of fluid, protein, macrophages, and other inflammatory cells entering areas of tissue injury (Brooks, 1986).

Patients treated with corticosteroids for long periods may develop a resistance towards a steroid-based therapy (Barnes, 1995). Neupert, 1990 has shown that systemic steroids are capable of decreasing trismus and global pain. Preoperative glucocorticoid therapy is effective in reducing postoperative swelling in orthognathic surgery (Dan *et al*., 2010). Patients with Systemic Lupus Erythematosus (SLE) are at higher risk with long-term steroid treatment (Sirois, 2003). Anne, (2010) have suggested that corticosteroid administered in combination with oral surgery produces significant decreases in edema and pain. An

Table 1. Complications of Short-Term Glucocorticoid Treatment

´ Allergic reaction – skin reaction/anaphylaxis
´ Skin changes – steroid acne/paper thin skin/ bruising
´ Increased serum glucose
´ Adrenal suppression (if high dose)
´ Disturbance of wound healing
´ Impaired immunity
´ Increased cardiovascular risk
´ Increased morbidity in pre-existing peptic ulcer disease
´ Glaucoma
´ Psychiatric disturbance – change in mood/ psychosis

elevated risk for the development of avascular necrosis, steroid-induced psychosis and adrenal suppression is present with the higher dosage required for orthognathic surgery. Hooley and Hohl, (1974) states that corticosteroids significantly reduces postoperative edema and decreases the average hospital stay. (Boc and Peterson, 1981; Gee, 1974) have recommended the use of steroids for orthognathic and traumatic oral surgical procedures when control of edema and prevention of vascular congestion in the nutrient flap is critical.

The biological effect of glucocorticoids on wound healing is thought to increase the risk of a number of adverse gastrointestinal events such as gastritis, formation of an ulcer and gastrointestinal bleeding (Salerno and Hermann, 2006). Precious *et al*., 1992 have reported steroid-induced acne due to corticosteroid treatment after orthognathic surgery. But none have reported severe complications.

Female gender and a history of psychotic behaviour were found to increase the risk of steroid induced psychosis (Lewis and Smith, 1983). A trial by Weber and Griffin, 1994 have been showed significantly decreased edema with 1 single preoperative dose of dexamethasone and doses administered before and after surgery and on the first postoperative day.

Alexander and Throndson, 2000 states that swelling and inflammation contribute towards pain and trismus but inflammation is an important part of wound healing. Extensive swelling can compromise the airway, the recovery of patient and surgical outcome.

A small number of trials have examined the effect of glucocorticoids on swelling in orthognathic surgery. Munro *et al*., 1986 have compared a preoperative dose of dexamethasone 0.5mg/kg followed by two day post-operative dose of 0.25mg/kg/day with placebo in children having mandibular or maxillary osteotomy. They found no significant reduction in facial swelling when photographs were assessed subjectively by an independent observer.

Schaberg *et al*., 1984 have reported that Computed Tomograms (CT) allow more objective evaluation of swelling. In a cohort study of 39 patients having Le-fort I or transoral vertical osteotomy, facial swelling as assessed on CT, was reduced at 24 and 72h, and was attributed to the timing of the dose of methylprednisolone .

Weber and Griffin,1994 states that in a randomised prospective double blind trial, 23 patient who required bilateral sagittal split osteotomy of the mandible were split into three groups and were given either placebo, preoperative dexamethasone 16mg intravenously or preoperative dexamethasone 16mg intravenously with three postoperative 8mg doses intravenously every 6h. In both dexamethasone group there was a significant ($p<0.5$) reduction in facial swelling on postoperative day one as assessed by computer scanning of clinical photograph, and was no stastical difference between two groups.

CONCLUSION

The use of short term systemic corticosteroids postoperatively after orthognathic surgery is safe. Preoperative intravenous dexamethasone significantly reduced postoperative inflammation and its associated edema after orthognathic surgery. The administration of corticosteroids in orthognathic surgery decreases edema and pain significantly, with no higher risk of infection and with minimum risk of other side effects. Though corticosteroids plays a beneficial role in post operative management of orthognathic surgery, risk of its administration must be seriously considered before using it.

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