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## Review Article

### **Management Of Dental Patients On Corticosteroid Therapy**

<sup>1</sup>Muskan Khurana, <sup>2</sup>Suman Sharma, <sup>3</sup>Kajal

#### ABSTRACT:

Many dental patients are receiving long-term systemic corticosteroid therapy. The corticosteroids act as double edged sword that is it helps in the dental treatment as well as also has a profound systemic effect on patients taking this medication for a long term. This article enlightens about the management of the patient receiving such medication and requiring dental therapy, the guidelines and handling the adverse effects. It also briefs about the use of Corticosteroids in certain dental procedures.

Keywords: Corticosteroids, adrenal atrophy, management, dental procedures, anxiety control, steroid therapy.

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Corresponding author: Muskan Khurana, Dental Practitioner, Jalandhar, Punjab, India

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#### INTRODUCTION

Corticosteroids and their synthetic analogues are commonly used for their potent anti-inflammatory and immunosuppressive properties in the treatment of a variety of medical conditions characterized by chronic inflammatory and immune phenomena. [1][2]Prolonged therapy with corticosteroids may produce many diverse side effects as well as depress normal function of the adrenal cortex. Every practicing dentist must have a basic understanding of adrenal physiology and the body's response to exogenous corticosteroids in order to properly manage dental patients who are receiving systemic corticosteroid medications. The purpose of this article is to review these topics, with focus on the management of dental patients receiving corticosteroid therapy.

#### PHYSIOLOGY OF CORTISOL

Adrenal cortex secretes three types of hormones: glucocorticoids, androgens, and mineralocorticoids. These are related to the body's response to stress and the physiological response of the vascular system to

tissue damage [3][4]. A total of 24–30 mg/day of cortisol is secreted by the adrenal gland in normal adults<sup>[5]</sup>. However, during periods of stress, this secretion level can spike up to300 mg/day<sup>[6]</sup>. Diurnal variations; the response of individuals to stress; and the effectiveness of control mechanisms involving the hypothalamus, pituitary gland and adrenal glands play a central role in regulating the daily secretion level of cortisol.

When high doses of corticosteroids equivalent to >30 mg of cortisol are administered for two weeks, these regulatory mechanisms may be altered and may return to normal only over a period of up to one year. Meanwhile, the body's capacity to react to stress can recover in just 15–20 days<sup>[5][7]</sup>. A total of 10–20 mg/day of cortisol is secreted, with almost 50% of this being secreted in the early hours of the day<sup>[6]</sup>.

#### FEEDBACK MECHANISM

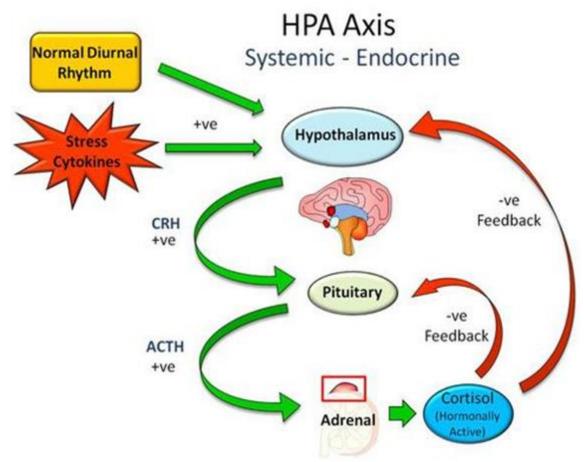
The secretion by adrenal cortex is controlled by Feedback system of HYPOTHALAMUS-PITUITARY-ADREANAL AXIS.

<sup>&</sup>lt;sup>1</sup>Dental Practitioner, Jalandhar, Punjab, India;

<sup>&</sup>lt;sup>2</sup>Post graduate student, Department of Paediatric and Preventive Dentistry, Genesis Institute of Dental Sciences and Research Ferozepur, Punjab, India;

<sup>&</sup>lt;sup>3</sup>Tutor, Genesis Institute of Dental Sciences and Research Ferozepur, Punjab, India

THE CORTISOL PATHWAY<sup>[8]</sup>



In response to corticotropin-releasing factor (CRF) from the hypothalamus, the anterior pituitary gland secretes adrenocorticotropic hormone (ACTH) which, in turn, causes the synthesis and release of glucocorticoids from the adrenal cortex. Sufficient amounts of glucocorticoids in circulation serve as a negative feedback mechanism for CRF synthesis, resulting in cessation of ACTH release. The hypothalamus may also release CRF in response to stimulation of the central nervous system by trauma or anxiety. During periods of stress, the negative feedback mechanism may be avoided for the increased need for glucocorticoids.

Under normal conditions, 15 to 30 mgof endogenous cortisol are produced daily. During periods of maximal stress, the output of cortisol may increase up to 60mg.

## MECHANISM OF ACTION OF GLUCOCORTICOIDS

During and after any surgical procedure classical signs of inflammation including redness, elevated temperature, increased pain level, tissue enlargement due to fluid extravasation, and loss of function can be seen<sup>[9]</sup>. An appropriate inflammatory response is critical for an effective healing process in response to tissue damage. Cortisol plays a central role in reducing the signs and symptoms of inflammation by inhibiting the enzyme phospholipase A2, which converts phospholipids into arachidonic acid. This in

turn blocks the formation of other products, such as prostaglandins, leukotrienes, and thromboxane A2 which are mainly responsible for inflammatory response in body<sup>[5]</sup>. Cortisol also makes the lysosomal membranes more stable, thereby reducing the release of lysozymes, which propagate an inflammatory response at the site of injury. Furthermore, cortisol reduces capillary permeability, which in turn reduces inflammatory fluid extravasation. These alterations can suppress host resistance to bacterial or fungal pathogens and increase frequency of infections<sup>[10-13]</sup>. The severity of infections for persons on steroid medication also appears to be greater<sup>[12-14]</sup>. This increased susceptibility to infection can be critical for the patient subjected to bacteremia produced by dental procedures.

#### RELATION TO DENTAL PROCEDURES

Extractions: Surgical extraction of mandibular third molars is considered the most common oral surgical procedure<sup>[15]</sup>. Patients experience a range of adverse signs and symptoms following extraction, including pain, facial oedema, and trismus due to masticatory muscles inflammation <sup>[16]</sup>. In such patients, corticosteroids can help in anti-inflammatory effect by reducing fluid transudation, decreasing cell exudates, inhibiting vascular dilatation, and reducing fibrin deposition around the inflamed area. Given its analgesic and anti-inflammatory effects, a

combination of diclofenac and prednisolone has been found to be highly effective in reducing postoperative pain and swelling<sup>[17]</sup>.

**Temporomandibular Joint Disorders**: TMDs are the third most prevalent disorders causing pain worldwide<sup>[18]</sup>. Difficulty in opening the mouth, pain, tenderness in the muscles of mastication, and deviation in jaw movements are frequent manifestations of TMDs. Cortisol has been used in the management of patients with signs and symptoms of TMDs or TMJ arthritis.

**Oral Submucous Fibrosis:** Cortisol and its synthetic analogues have been topically used in patients with oral submucous fibrosis, particularly in patients with ulcers and mucosal pain. Symptomatic relief is achieved because of the anti-inflammatory properties of cortisol<sup>[19-20]</sup>. The use of a topical triamcinolone ointment thrice a day is preferred<sup>[21]</sup>.

**Bell's Palsy:** In patients with Bell's palsy, 60 mg/day of prednisolone is administered during the first five days of treatment, following which the dosage is tapered over the next six days with a reduction of 10 mg/day ending with an 11-day regimen <sup>[22]</sup>. It has to be borne in mind that the treatment should be initiated during the first 72 hours of symptom onset.

For persons on continuous, long-term glucocorticoid therapy, elective dental surgery procedures should be postponed until steroid therapy is discontinued or modifications are explored with the prescribing physician. If the dental procedure, such as emergency tooth extraction, cannot be delayed, it should be limited to localized regions in order to minimizethe size of the wound. All possible safeguards should be employed to prevent contamination of the site, and prophylactic antibiotics should be administered.

#### ADRENAL ATROPHY

The most important side effect that occurs during prolonged administration of glucocorticoids is adrenal atrophy and loss of normal function in the adrenal cortex. Exogenous glucocorticoids in circulation provide negative feedback which inhibits the production of CRF and subsequent release of ACTH. With reduced or no hormonal stimulation, the adrenal cortex has decreased output and loses its ability to respond to demand. The degree of adrenal atrophy is dependent upon the dose of glucocorticoid, the route of administration, the duration of therapy, and the frequency of administration<sup>[23]</sup>. Relatively small doses (less than 30 mg) given daily for periods of less than 1 week has shown to cause little alteration in pituitaryadrenal function<sup>[24]</sup>. It has been shown that administration of glucocorticoids on an alternate-day schedule helps to prevent significant pituitary-adrenal suppression but is not successful in controlling the systemic disease. However, large, prolonged applications of cortisone to the skin of adults have not produced any major systemic effects. During periods of stress the patient's requirement for glucocorticoids is increased and may exceed the fixed dose that the patient is taking. Since the patient's pituitary-adrenal system is unable to respond to this need, the patient is left in a state of acute adrenal insufficiency or "adrenal crisis." Following discontinuation of daily therapeutic doses of exogenous glucocorticoids, patients will eventually regain normal pituitary-adrenal function. While the exact time for complete recovery varies widely, and it has been concluded that 1 year is required for most persons to regain normal response<sup>[26-27]</sup>.

## MANAGEMENT OF DENTAL PATIENTS TAKING CORTICOSTEROIDS

In the history of present illness or past medical history if the patient presents with the Corticosteroid therapy or have taken in the last year thenadditional information regarding dosage, route of administration, frequency of doses, and duration of therapy is necessary. If a patient is not presently taking glucocorticoids but has in the past, the length of time since the medication was last received is equally essential. Patients receiving glucocorticoids in doses greater than the equivalent of 30 mgof cortisone daily for a continuous period of one week or longer have a possibility of developing adrenal crisis. Persons who have discontinued this type of therapy within the past year are also at risk. The higher the daily dosage, the longer the period of administration, or the shorter the period since discontinuation, the greater is the potential risk of adrenal crisis. Because of the inherent management problems with the high-risk patient, elective dental procedures should be delayed if there is a possibility of an eventual change in the patient's adrenal status. If it is decided that dental procedures must be performed, consultation with the prescribing physician is mandatory.

Since adrenal crisis is due to the patient's inability to produce sufficient amounts of endogenous cortisol in response to stress, all efforts should be made to minimize the degree of anxiety experienced. In addition to anxiety control, blood glucocorticoid levels must be increased prior to a stressful situation. This can be accomplished through administration of a "steroid prep." A steroid prep is a controlled increase of glucocorticoids given prior to the patient's dental appointment which is slowly reduced to a normal dosage level over a 2- to 3day periodfollowing dentalprocedure. Failure to use a schedule of gradual reduction can result in adrenal insufficiency, which can cause collapse and death. [28-30]

Several regimens of management for the risk patient during dental therapy have been suggested<sup>[31]</sup>. Dentists must carefully evaluate the extent of dental procedures and estimate the apprehension of patients prior to discussing their recommendations with physicians. The following guidelines may be used to categorize management:

- I. A. Routine non-invasive dental therapy (examination, impressions, etc.)
- B. A non-apprehensive patient and

- C. Slight risk of pituitary-adrenal suppression.
- -No steroid prep is required.
- -Light sedation is optional.
- II. A. Routine invasive dental therapy (restorations, routine endodontics, scaling and root planing, biopsy) or
- B. A moderately apprehensive patient or
- C. Relatively greater risk of pituitary-adrenal suppression.
- -A steroid prep consisting of doubling' the daily dosage of glucocorticoid prior to the procedure followed by gradually reducing the dose each day for 3 days back to the normal dosage.
- -Light to moderate sedation.
- III. A. Combination of two or three factors from Category II or
- B. Extensive invasive dental therapy (long restorative or endodontic procedures, multiple extractions, periodontal or endodontic surgery) or

C. an extremely apprehensive patient or

- D. severe risk of pituitary-adrenal suppression.
- -A steroid prep consisting of quadrupling'9 the daily dosage of glucocorticoid prior to the procedure followed by gradual reduction over a 2- or 3-day period to the normal dosage.
- -Moderate to heavy sedation.

- IV. Combination of two or more of the situations in Category III or a long surgical procedure.
- -Hospitalization of the patient where administration of extensive doses of glucocorticoids, heavy sedation, and appropriate monitoring can be provided.

# ADRENAL CRISIS IS CHARACTERIZED BY ONE OR MORE OF THE FOLLOWING PHYSIOLOGIC SYMPTOMS

- 1. Hypotension
- 2. Severe weakness
- 3. Hyperthermia
- 4. Hypoglycemia
- 5. Hyperkalemia and
- 6. Deterioration of the cardiovascular system.

#### CLINICALLY, PATIENTS WILL EXHIBIT

- (1) progressive severe mental confusion
- (2) nausea and vomiting and
- (3) intense pain in abdomen, lower back and legs.

If unchecked, these symptoms may lead to loss of consciousness, coma, and death  $^{[32\text{-}33]}$ 

IN CASE OF EMERGENCY SCENAROIS EVEN AFTER ALL PRECAUTIONS, the following REGIMEN should be followed. All administration of steroid preps should be by mutual agreement with the physician in charge of glucocorticoid therapy.

#### TREATMENT OF ADRENAL CRISIS (NEW ENGLAND JOURNAL OF MEDICINE)

Treatment	Dose and Procedure
Treatment	Dose and Procedure
Adults	
Hydrocortisone*	Provide prompt administration at a dose of 100 mg intravenously (or intramuscularly if intravenous access is not feasible), followed by 200 mg every 24 hr, given as a continuous infusion as intravenous (or intramuscular) boluses (50 mg) every 6 hr; if initial treatment is successful (usually after 24 hr), oral hydrocortisone at 2 to 3 times the usual dose can be given, with tapering down to the usual dose over the next 2 to 3 days?
Fluids	Provide intravenous administration of 1000 ml of normal saline (0.9% isotonic sodium chloride in the first hour, with intravenous dextrose to 5% concentration in normal saline added if the patient has hypoglycemia; subsequently, administer crystalloid fluids according to standard resuscitation guidelines;
Children	
Hydrocortisone	Provide prompt administration at a dose of 50 mg per square meter of body-surface area intrave nously (or intramuscularly if intravenous access is not feasible), followed by 50–100 mg per square meter every 24 hr, given as a continuous infusion or as intravenous (or intramuscula boluses (12.5–25 mg per square meter) every 6 hr; if initial treatment is successful (usually after 24 hr), oral hydrocortisone at 2 to 3 times the usual dose can be given, with tapering down to the usual dose over the next 2 to 3 days †
Fluids	Give a bolus of normal saline at a dose of 20 ml per kilogram of body weight, with repeated doses up to a maximum of 60 ml per kilogram in the first hour, along with intravenous dextrose, 0.5–1 g per kilogram, if the patient has hypoglycemia; provide subsequent administration of crystalloid fluids according to standard resuscitation guidelines:
Adults and children	
Possible additional measures	Antibiotic therapy, admission to intensive care or high-dependency unit, administration of low-dose heparin  Prompt investigation of other causes when hypotension persists despite adequate initial treatment  Consideration of precipitating events (e.g., sepsis, gastroenteritis)

- \* If hydrocortisone is unavailable, another parenteral glucocorticoid, such as dexamethasone (4 mg every 24 hours), methylprednisolone (40 mg every 24 hours), or prednisolone (25 mg bolus followed by two 25-mg doses, for a total of 75 mg in the first 24 hours; thereafter, 50 mg every 24 hours), may be used.
- † Fludrocortisone replacement is not required if hydrocortisone doses exceed 50 mg every 24 hours but is typically administered in adults and children with primary adrenal insufficiency when oral hydrocortisone is started.
- ‡ Circulatory status, body weight, and relevant coexisting conditions should be taken into account.

#### CONCLUSION

When treating patients with a history of glucocorticoid therapy, the precise information from the medical history should be taken to determine the relative risk of pituitary-adrenal suppression. If the patient is at risk, the dentist must determine the need for the following prophylactic measures:

- (1) systemic antibiotic therapy
- (2) anxiety control
- (3) increased administration of glucocorticoids (steroid prep).

If a patient experiences adrenal crisis, the dentist must be capable of interpreting the signs and administering appropriate therapy.

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