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Corticosteroids in Oral Mucosal Lesions: Myths Versus Reality

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ABSTRACT

Since its discovery in the 1040s, glucocorticoids have offered a cure for a variety of illnesses. Because of their anti-inflammatory and immunosuppressive qualities, corticosteroids are a valuable therapeutic tool for the treatment of oral mucosal ulcers. The purpose of this article is to educate the physician on the various possibilities for steroid therapy, allowing them to make decisions depending on the drug's qualities and the underlying condition.

INTRODUCTION

Since their introduction more than 50 years ago, glucocorticoids have completely changed the way many diseases are managed. Corticosteroids comprise both the synthetic steroid hormones made for pharmacotherapeutics and the endogenous steroid hormones generated from the adrenal cortex.[1-3] Although there are numerous therapeutic benefits to steroids, it is important to consider their side effects and long-term implications when treating a patient. Oral submucous fibrosis, pemphigus, lichen planus, and other mucosal illnesses can all be treated with topical and systemic steroids. On the other hand, a dental practitioner may occasionally encounter a patient who is receiving long-term steroid treatment for a systemic illness like lupus or arthritis. Such individuals' dental conditions should be managed with consideration for the impact of long-term steroid therapy and how it affects dental care.

Pharmacology of Steroids

Glucocorticoids, mineralocorticoids (sometimes called corticosteroids) and sex hormones are secreted by the adrenal cortex. Cholesterol is the starting material for the first two.

Hydrocortisone, prednisone, triamcinolone, dexamethasone, clobetasol, and mometasone are the several classifications for corticosteroids.

Glucocorticoids affect how proteins, lipids, carbs, calcium, and electrolytes are metabolized. Corticosteroids have a wide range of physiological and metabolic effects, although their primary pharmaceutical benefits are related to their anti-inflammatory and immunosuppressive properties. All forms of inflammation and allergic responses are suppressed by glucocorticoids. By inhibiting the function of white blood cells, stabilizing the lysozyme membrane, preventing plasminogen activation, and lowering the production of inflammatory mediators such prostaglandins and leukotrienes, glucocorticoids produce these effects.

In addition to topical use, systemic (oral and parenteral) administration of glucocorticoids is possible. They undergo hepatic metabolism before being conjugated and eliminated by the urine. Synthetic steroids have a slower metabolism, which prolongs their effects. Both topical and systemic steroids are used to treat a variety of immune-mediated and inflammatory mucosal diseases. Their function in a few of the mucosal disorders that are frequently seen has been covered.

Recurrent Aphthous Stomatitis

One of the most frequent and unpleasant mucosal disorders that people encounter is probably recurrent aphthous stomatitis (RAS). A day or two before to ulceration, RAS is typically accompanied by a localized burning sensation in the affected area. The ulcers themselves are circular to oval, painful, superficial, well-defined, and covered in a pseudomembrane with an erythematous halo surrounding them. Three clinical manifestations of RAS are possible: Seventy-five to eighty-five percent of instances of aphthae are minor cases. Usually measuring less than a centimeter in diameter, they recover in a week or two without any problems. On the mucosa covering the minor salivary glands, major aphthae typically develop. They often develop during puberty, typically affect the lips, soft palate, and throat, and leave scars that take weeks to months to heal.

Merely 5%–10% of all RAS cases are herpetiformaphthae instances. They appear as tiny (3–5 mm) ulcer crops that clump together and resemble herpes simplex infection. RAS's etiopathogenesis is not fully understood. By far, the diagnosis of RAS is clinical, and it is important to rule out any underlying conditions. RAS is managed symptomatically. It is preferable to apply topical triamcinolone acetonide (1% in an adhesive base) four times a day.[4] High intensity topical medicines such betamethasone, fluocinonide, or clobetasol held directly over the lesion in a gauze may be necessary in severe cases. In addition, if there are no periods of remission and the ulcers are constant, intralesional injections of betamethasone, dexamethasone, or trimetholone may be warranted.[3]

Oral Lichen Planus

It's a long-lasting inflammatory disease with an unknown T-cell-related etiology. Clinically, comparable medication-induced lesions are referred to as oral lichenoid drug responses. The appearance of oral lichen planus (OLP) can be bullous, reticular, papillary, atrophic, erosive, or plaque-like. The atrophic and erosive types are indicative.[5] Steroids are essential for managing lichen planus symptoms, even though a cure is still unattainable. The primary treatment is topical steroids; however, in cases of acute aggravation or numerous, large lesions, systemic

corticosteroids must be administered.[6,7] 0.025 percent Effective treatments for the symptoms of OLP include fluocinonide, topical betamethasone, fluticasone propionate spray, topical mometasonefuroatemicroemulsion, clobetasol propionate in orabase, and mouthwash, topical, intralesional triamcinolone acetonide..It has been demonstrated that 0.1% fluocinoloneacetonide is superior than triamcinolone in the treatment of OLP.[8] When treating resistant cases of erosive lichen planus, systemic corticosteroids should be investigated. The lowest therapeutic dose of prednisolone is 40–80 mg for 6-7 days.[9] Withdrawal must be tapered by lowering 5 mg per week if systemic steroid use is required for longer than two weeks in order to avoid triggering an adrenal crisis.[10]

Oral Submucous Fibrosis

A potentially malignant disorder of the oral cavity, oral submucous fibrosis (OSF) has a prevalence rate of 2.01% and a reported malignant transformation rate of 2.3%–7.6%. It causes a pronounced rigidity, growing loss of tissue mobility, and eventually the inability to open the mouth.[11,12,13,14,15,16] Steroids aid in OSF by lowering inflammation, which in turn prevents fibrosis by fibroblast proliferation and collagen deposition. For 20 weeks, biweekly intralesional injections of hyaluronidase and dexamethasone (4 mg/ml) are administered. For four weeks, triamcinolone (10 mg/ml) and hyaluronidase were administered intralessionally every two weeks. These facilitate better tongue opening.[16] Furthermore, systemic hydrocortisone (100 mg/day) or prednisone (30–40 mg/day for 14–28 days and subsequently tapered) may be useful in reducing burning feeling.

Erythema Multiforme

A dermatological hypersensitivity reaction to infectious agents like herpes and certain drugs is called erythema multiforme. In almost half of the instances, the herpes simplex virus is thought to be the cause. Treatment of the suspected infection or stopping the problematic medication are the first steps in management. Steroids function as adjuvants. It is possible to use mouthwash, adhesive paste, or topical clobetasol propionate. To prevent the long-term usage of systemic steroids, prednisone or dexamethasone pulse treatment may be recommended systemically.[17]

Pemphigus

Pemphigus is a persistent mucocutaneous illness that frequently starts in the mouth and causes excruciating blisters.[18] Since it is an autoimmune condition, managing the symptoms and stopping the disease process are frequently the main goals of care. 0.05% fluocinoloneacetonide or 0.05% clobetasol propionate is recommended for mild Pemphigus lesions. Prednisone at a high dosage of 100–200 mg is used to treat severe instances until symptomatic regression takes place. The dosage is lowered progressively until it is kept at about 50 mg per day.[19]

Mucous Membrane Pemphigoid

The vesiculobullous autoimmune illness known as mucous membrane pemphigoid (MMP), commonly referred to as cicatricialpemphigoid, affects the mucosa and skin. Because the condition is aggressive and incurable, high dosages of steroids are needed to control it.[20] Topical steroids can be used to manage gingival desquamation but have a

limited effect in MMP. Prednisolone (1-2 mg/kg/day) is the first line of treatment; immunosuppressants are then combined and the dosage is gradually tapered down. In cases where a daily dose over 100 mg is necessary, pulsed intravenous corticosteroids are advised.[18, 21]

Bullous Pemphigoid

It is a subepidermal blistering vesiculobullous autoimmune illness. It has been discovered that 0.05% clobetasol propionate works well topically. Systemic steroids are necessary for severe illness. 20 mg of prednisone per day is advised. In situations that don't yield, triamcinolone acetonide (3–10 mg/ml) may be administered intralesionally.[17]

Lupus Erythematosus

It could be chronic (discoid) or acute (systemic). Lupus oral ulcerations occur during flare-ups and are temporary. It is recommended to use topical betamethasone or clobetasol.[17] Prednisone (10–20 mg/day) may be used systemically or on different days. In some circumstances, intralesional triamcinolone may also be helpful.[22, 23]

CONCLUSION

Steroids offer the classic "Magic Bullet," a one-stop solution that cures practically every illness. It is still a two-edged sword, though. It does cure many diseases quickly and effectively. The doctor must, however, be aware of the indications, contraindications, and extra cautions that need to be taken when experimenting with steroids. The doctor must also be aware of the dose adjustments and modifications that need to be tailored to each patient based on the patient's condition, response, and disease process. Steroids can still serve to safely relieve human suffering if they are used wisely.

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