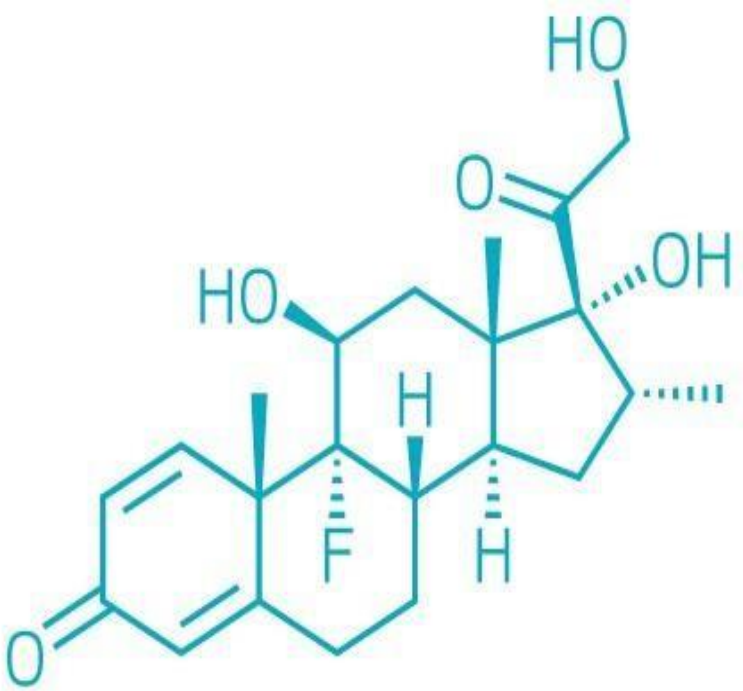
INTRODUCTION

The molecular weight for dexamethasone is 392.47. It is designated chemically as 9-fluoro-11β,17,21-trihydroxy-16α-methylpregna-1,4diene-3,20-dione.

The empirical formula is C22H29FO5 and the structural formula is;



Dexamethasone

Dexamethasone, a synthetic adrenocortical steroid, is a white to practically white, odourless, crystalline powder. It is stable in air. It is practically insoluble in water.

INDICATIONS AND USAGE

- Allergic states: Control of severe or incapacitating allergic conditions intractable to adequate trials of conventional treatment in asthma, atopic dermatitis, contact dermatitis, drug hypersensitivity reactions, perennial or seasonal allergic rhinitis, and serum sickness.

- Dermatologic diseases: Bullous dermatitis herpetiformis, exfoliative erythroderma, mycosis fungoides, pemphigus, and severe erythema multiforme (Stevens-Johnson syndrome).

- Endocrine disorders: Primary or secondary adrenocortical insufficiency , congenital adrenal hyperplasia, hypercalcemia associated with cancer, and nonsuppurative thyroiditis.

- Gastrointestinal diseases: To tide the patient over a critical period of the disease in regional enteritis and ulcerative colitis.

- Hematologic disorders: Acquired (autoimmune) haemolytic anaemia, congenital (erythroid) hypoplastic anaemia (DiamondBlackfan anaemia), idiopathic thrombocytopenic purpura in adults, pure red cell aplasia, and selected cases of secondary thrombocytopenia.

- Miscellaneous: Diagnostic testing of adrenocortical hyperfunction, trichinosis with neurologic or myocardial involvement, tuberculous meningitis with subarachnoid block or impending block when used with appropriate antituberculous chemotherapy.

- Neoplastic diseases: For the palliative management of leukemias and lymphomas.

- Nervous system: Acute exacerbations of multiple sclerosis, cerebral edema associated with primary or metastatic brain tumor, craniotomy, or head injury.

- Ophthalmic diseases: Sympathetic ophthalmia, temporal arteritis, uveitis, and ocular inflammatory conditions unresponsive to topical corticosteroids.

- Renal diseases: To induce a diuresis or remission of proteinuria in idiopathic nephrotic syndrome or that due to lupus erythematosus.

- Respiratory diseases: Berylliosis, fulminating or disseminated pulmonary tuberculosis when used concurrently with appropriate antituberculous chemotherapy, idiopathic eosinophilic pneumonias, symptomatic sarcoidosis.

- Rheumatic disorders: As adjunctive therapy for short-term administration in acute gouty arthritis, acute rheumatic carditis, ankylosing spondylitis, psoriatic acute arthritis, rheumatoid arthritis, including juvenile rheumatoid arthritis

  For the treatment of dermatomyositis, polymyocitis, and systemic lupus erythematosus.

Indication and Usage

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Dermatologic diseases: Bullous dermatitis herpetiformis, exfoliative erythroderma, mycosis fungoides,

pemphigus, and severe erythema multiforme (Stevens-Johnson syndrome).

Endocrine disorders: Primary or secondary adrenocortical insufficiency (hydrocortisone or cortisone is the drug of choice; may be used in conjunction with synthetic mineralocorticoid analogs where applicable; in infancy mineralocorticoid supplementation is of particular importance), congenital adrenal hyperplasia, hypercalcemia associated with cancer, and nonsuppurative thyroiditis.

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maintenance therapy). For the treatment of dermatomyositis, polymyositis, and systemic lupus erythematosus.

Contraindicatios

Systemic fungal infections (see WARNINGS, Fungal infections).

Dexamethasone tablets are contraindicated in patients who are hypersensitive to any components of this product.

General:

Rare instances of anaphylactoid reactions have occurred in patients receiving corticosteroid therapy (see ADVERSE REACTIONS).

Increased dosage of rapidly acting corticosteroids is indicated in patients on corticosteroid therapy subjected

to any unusual stress before, during, and after the stressful situation.

Cardio-renal:

Average and large doses of corticosteroids can cause elevation of blood pressure, sodium and water

retention, and increased excretion of potassium. These effects are less likely to occur with the synthetic

derivatives except when used in large doses. Dietary salt restriction and potassium supplementation may be necessary. All corticosteroids increase calcium excretion.

Literature reports suggest an apparent association between use of corticosteroids and left ventricular free

wall rupture after a recent myocardial infarction; therefore, therapy with corticosteroids should be used with great caution in these patients.

Endocrine:

Corticosteroids can produce reversible hypothalamic-pituitary adrenal (HPA) axis suppression with the

potential for corticosteroid insufficiency after withdrawal of treatment. Adrenocortical insufficiency may result from too rapid withdrawal of corticosteroids and may be minimized by gradual reduction of dosage. This type of relative insufficiency may persist for months after

discontinuation of therapy; therefore, in any situation of stress

occurring during that period, hormone therapy should be

reinstituted. If the patient is receiving steroids already, dosage

may have to be increased.

Metabolic clearance of corticosteroids is decreased in hypothyroid patients and increased in hyperthyroid patients. Changes in thyroid status of the patient may necessitate adjustment in dosage.

Infections:

General:

Patients who are on corticosteroids are more susceptible to infections than are healthy individuals. There

may be decreased resistance and inability to localize infection when corticosteroids are used. Infection with any pathogen (viral, bacterial, fungal, protozoan or helminthic) in any location of the body may be associated with the use of corticosteroids alone or in combination with other immunosuppressive agents. These infections may be mild to severe. With increasing doses of corticosteroids, the rate of occurrence of infectious complications

increases. Corticosteroids may also mask some signs of current infection.

Fungal Infections:

Corticosteroids may exacerbate systemic fungal infections and therefore should not be used in the presence of such infections unless they are needed to control lifethreatening drug reactions. There have been cases reported in which concomitant use of amphotericin B and hydrocortisone was

followed by cardiac enlargement and congestive heart failure (see PRECAUTIONS, Drug Interactions, Amphotericin B injection and potassium- depleting agents).

Special pathogens:

Latent disease may be activated or there may be an exacerbation of intercurrent infections due to pathogens, including those caused by Amoeba, Candida, Cryptococcus, Mycobacterium, Nocardia, Pneumocystis, Toxoplasma.

It is recommended that latent amebiasis or active amebiasis be ruled out before initiating corticosteroid

therapy in any patient who has spent time in the tropics or any patient with unexplained diarrhea.

Similarly, corticosteroids should be used with great care in patients with known or suspected Strongyloides (threadworm) infestation. In such patients, corticosteroidinduced immunosuppression may lead to

Strongyloides hyperinfection and dissemination with widespread larval migration, often accompanied by severe enterocolitis and potentially fatal gram-negative septicemia.

Corticosteroids should not be used in cerebral malaria.

Tuberculosis:

The use of corticosteroids in active tuberculosis should be restricted to those cases of fulminating or

disseminated tuberculosis in which the corticosteroid is used for the management of the disease in conjunction with an appropriate antituberculous regimen.

If corticosteroids are indicated in patients with latent tuberculosis or tuberculin reactivity, close observation is necessary as reactivation of the disease may occur. During prolonged corticosteroid therapy, these patients should receive chemoprophylaxis.

Vaccination:

Administration of live or live, attenuated vaccines is contraindicated in patients receiving

immunosuppressive doses of corticosteroids. Killed or inactivated vaccines may be administered.

However, the response to such vaccines cannot be predicted. Immunization procedures may be

undertaken in patients who are receiving corticosteroids as replacement therapy, e.g., for Addison's disease.

Viral infections:

Chickenpox and measles can have a more serious or even fatal course in pediatric and adult patients on

corticosteroids. In pediatric and adult patients who have not had these diseases, particular care should be taken to avoid exposure. The contribution of the underlying disease and/or prior corticosteroid treatment to the risk is also not known.

If exposed to chickenpox, prophylaxis with varicella zoster immune globulin (VZIG) may be indicated. If exposed to measles, prophylaxis with immune globulin (IG) may be indicated. (See the respective package inserts for VZIG and IG for complete prescribing information.) If chickenpox develops, treatment with antiviral agents should be considered.

Ophthalmic:

Use of corticosteroids may produce posterior subcapsular cataracts, glaucoma with possible damage to the optic nerves, and may enhance the establishment of secondary ocular infections due to bacteria, fungi, or

viruses. The use of oral corticosteroids is not recommended in the treatment of optic neuritis and may lead to an increase in the risk of new episodes. Corticosteroids should not be used in active ocular herpes simplex.

Precautions

General:

The lowest possible dose of corticosteroids should be used to control the condition under treatment. When reduction in dosage is possible, the reduction should be gradual.

Since complications of treatment with corticosteroids are dependent on the size of the dose and the duration of treatment, a risk/benefit decision must be made in each individual case as to dose and duration of treatment and as to whether daily or intermittent therapy should be used.

Kaposi's sarcoma has been reported to occur in patients receiving corticosteroid therapy, most often for chronic conditions. Discontinuation of corticosteroids may result in clinical improvement.

Cardio-renal:

As sodium retention with resultant edema and potassium loss may occur in patients receiving corticosteroids, these agents should be used with caution in patients with congestive heart failure, hypertension, or renal insufficiency.

Endocrine:

Drug-induced secondary adrenocortical insufficiency may be minimized by gradual reduction of dosage. This type of relative insufficiency may persist for months after discontinuation of therapy; therefore, in any situation of stress occurring during that period, hormone therapy should be reinstituted. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently.

Gastrointestinal:

Steroids should be used with caution in active or latent peptic ulcers, diverticulitis, fresh intestinal

anastomoses, and nonspecific ulcerative colitis, since they may increase the risk of a perforation.

Signs of peritoneal irritation following gastrointestinal perforation in patients receiving corticosteroids may be minimal or absent.

There is an enhanced effect due to decreased metabolism of corticosteroids in patients with cirrhosis.

Musculoskeletal:

Corticosteroids decrease bone formation and increase bone resorption both through their effect on calcium

regulation (i.e., decreasing absorption and increasing excretion) and inhibition of osteoblast function. This,

together with a decrease in the protein matrix of the bone secondary to an increase in protein catabolism, and reduced sex hormone production, may lead to inhibition of bone growth in pediatric patients and the

development of osteoporosis at any age. Special consideration should be given to patients at increased risk of osteoporosis (e.g., postmenopausal women) before initiating corticosteroid therapy.

Neuro-psychiatric:

Although controlled clinical trials have shown corticosteroids to be effective in speeding the resolution of

acute exacerbations of multiple sclerosis, they do not show that they affect the ultimate outcome or natural

history of the disease. The studies do show that relatively high doses of corticosteroids are necessary to demonstrate a significant effect. An acute myopathy has been observed with the use of high Cardio-renal:

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demonstrate a significant effect. (See DOSAGE AND ADMINISTRATION.)

An acute myopathy has been observed with the use of high doses of corticosteroids, most often occurring in patients with disorders of neuromuscular transmission (e.g., myasthenia gravis), or in patients receiving

concomitant therapy with neuromuscular blocking drugs (e.g., pancuronium). This acute myopathy is

generalized, may involve ocular and respiratory muscles, and may result in quadriparesis. Elevation of

creatinine kinase may occur. Clinical improvement or recovery after stopping corticosteroids may require weeks to years.

Psychic derangements may appear when corticosteroids are used, ranging from euphoria, insomnia, mood

swings, personality changes, and severe depression, to frank psychotic manifestations. Also, existing emotional instability or psychotic tendencies may be aggravated by corticosteroids.

Ophthalmic:

Intraocular pressure may become elevated in some individuals. If steroid therapy is continued for more than

6 weeks, intraocular pressure should be monitored.

Information for patients:

Patients should be warned not to discontinue the use of corticosteroids abruptly or without medical

supervision. As prolonged use may cause adrenal insufficiency and make patients dependent on

corticosteroids, they should advise any medical attendants that they are taking corticosteroids and they should seek medical advice at once should they develop an acute illness including fever or other signs of infection.

Following prolonged therapy, withdrawal of corticosteroids may result in symptoms of the corticosteroid withdrawal syndrome including, myalgia, arthralgia, and malaise.

Persons who are on corticosteroids should be warned to avoid exposure to chickenpox or measles. Patients

should also be advised that if they are exposed, medical advice should be sought without delay. of corticosteroids, most often occurring in

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Drug Interactions

Amphotericin B injection and potassium-depleting agents: When corticosteroids are administered concomitantly with potassium-depleting agents (e.g., amphotericin B, diuretics), patients should be observed closely for development of hypokalemia. In addition, there have been cases reported in which concomitant use of amphotericin B and hydrocortisone was followed by cardiac enlargement and congestive heart failure. **Antibiotics**: Macrolide antibiotics have been reported to cause a significant decrease in corticosteroid clearance. **Anticholinesterases**: Concomitant use of anticholinesterase agents and corticosteroids may produce severe weakness in patients with myasthenia gravis. If possible, anticholinesterase agents should be withdrawn at least 24 hours before initiating corticosteroid therapy. **Anticoagulants**, oral: Co-administration of corticosteroids and warfarin usually results in inhibition of response to warfarin.Therefore, coagulation indices should be monitored frequently to maintain the desired anticoagulant effect.

Antidiabetics: Because corticosteroids may increase blood glucose concentrations, dosage adjustments of antidiabetic agents may be required.

Antitubercular drugs: Serum concentrations of isoniazid may be decreased.

Cyclosporine: Increased activity of both cyclosporine and corticosteroids may occur when the two are used concurrently. Convulsions have been reported with this concurrent use.

Dexamethasone suppression test (DST): False-negative results in the dexamethasone suppression test (DST) in patients being treated with indomethacin have been reported. Thus, results of the DST should be interpreted with caution in these patients.

Digitalis glycosides: Patients on digitalis glycosides may be at increased risk of arrhythmias due to hypokalemia.

Ephedrine: Ephedrine may enhance the metabolic clearance of corticosteroids, resulting in decreased blood levels and lessened physiologic activity, thus requiring an increase in corticosteroid dosage.

Estrogens, including oral contraceptives: Estrogens may decrease the hepatic metabolism of certain corticosteroids, thereby increasing their effect. **Hepatic Enzyme Inducers, Inhibitors and Substrates:** Drugs which induce cytochrome P450 3A4 (CYP 3A4) enzyme activity (e.g., barbiturates, phenytoin, carbamazepine, rifampin) may enhance the metabolism of corticosteroids and require that the dosage of the corticosteroid be increased. Drugs which inhibit CYP 3A4 (e.g., ketoconazole, macrolide antibiotics such as erythromycin) have the potential to result in increased plasma concentrations of corticosteroids. Dexamethasone is a moderate inducer of CYP 3A4. Co-adminstration with other drugs that are metabolized by CYP 3A4 (e.g., indinavir, erythromycin) may increase their clearance, resulting in decreased plasma concentration. **Ketoconazole**: Ketoconazole has been reported to decrease the metabolism of certain corticosteroids by up to 60%, leading to increased risk of

corticosteroid side effects. In addition, ketoconazole alone can inhibit adrenal corticosteroid synthesis and may cause adrenal insufficiency during corticosteroid withdrawal.

Nonsteroidal anti-inflammatory agents (NSAIDS): Concomitant use of aspirin (or other nonsteroidal anti-inflammatory agents) and corticosteroids increases the risk of gastrointestinal side effects. Aspirin should be used cautiously in conjunction with corticosteroids in hypoprothrombinemia. The clearance of salicylates may be increased with concurrent use of corticosteroids.

Skin tests: Corticosteroids may suppress reactions to skin tests.

Vaccines: Patients on corticosteroid therapy may exhibit a diminished response to toxoids and live or inactivated vaccines due to inhibition of antibody response. Corticosteroids may also potentiate the replication of some organisms contained in live attenuated vaccines. Routine administration of vaccines or toxoids should be deferred until corticosteroid therapy is stopped.

Pregnancy

Teratogenic effects: Pregnancy Category C. Corticosteroids should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Infants born to mothers who have received substantial doses of corticosteroids during pregnancy should be carefully observed.

ADVERSE REACTIONS

The following adverse reactions have been reported::

Allergic reactions: Anaphylactoid reaction, anaphylaxis.

Cardiovascular: Bradycardia, cardiac arrest, cardiac arrhythmias, cardiac enlargement, circulatory collapse,congestive heart failure, fat embolism, hypertension, hypertrophic cardiomyopathy in premature infants, myocardial rupture following recent myocardial infarction, edema, pulmonary edema, syncope, tachycardia, thromboembolism, thrombophlebitis, vasculitis.

Dermatologic: Acne, allergic dermatitis, dry scaly skin, ecchymoses and petechiae, erythema, impaired wound healing, increased sweating, rash, striae, suppression of reactions to skin tests, thin fragile skin, thinning scalp hair, urticaria.

Endocrine: Decreased carbohydrate and glucose tolerance, development of cushingoid state, hyperglycemia, glycosuria, hirsutism, hypertrichosis, increased requirements for insulin or oral hypoglycemic agents in diabetes, manifestations of latent diabetes mellitus, menstrual irregularities, secondary adrenocortical and pituitary

unresponsiveness,suppression of growth in pediatric patients.

Fluid and electrolyte disturbances: Congestive heart failure in susceptible patients, fluid retention, hypokalemic alkalosis, potassium loss, sodium retention.

Gastrointestinal: Abdominal distention, elevation in serum liver enzyme levels (usually reversible upon discontinuation), hepatomegaly, increased appetite, nausea, pancreatitis, peptic ulcer with possible perforation and hemorrhage, perforation of the small and large intestine (particularly in patients with inflammatory bowel disease), ulcerative esophagitis.

Metabolic: Negative nitrogen balance due to protein catabolism.

Musculoskeletal: Aseptic necrosis of femoral and humeral heads, loss of muscle mass, muscle weakness, osteoporosis, pathologic fracture of long bones, steroid myopathy, tendon rupture, vertebral compression fractures.

Neurological/Psychiatric: Convulsions, depression, emotional instability, euphoria, headache, increased intracranial pressure with papilledema (pseudotumor cerebri) usually following discontinuation of treatment, insomnia, mood swings, neuritis, neuropathy, paresthesia, personality changes, psychic disorders, vertigo. **Ophthalmic**:

Exophthalmos, glaucoma, increased intraocular pressure, posterior subcapsular cataracts. **Other**: Abnormal fat deposits, decreased resistance to infection, hiccups, increased or decreased motility and number of spermatozoa, malaise, moon face, weight gain.

OVERDOSAGE

Treatment of overdosage is by supportive and symptomatic therapy. In the case of acute overdosage, according to the patient's condition, supportive therapy may include gastric lavage or emesis.

DOSAGE AND ADMINISTRATION

For oral administration.

It Should Be Emphasized That Dosage Requirements Are Variable And Must Be Individualized On The Basis Of The Disease Under Treatment And The Response Of The Patient.

Situations which may make dosage adjustments necessary are changes in clinical status secondary to remissions or exacerbations in the disease process, the patient's individual drug responsiveness, and the effect of patient exposure to stressful situations not directly related to the disease entity. If after long-term therapy the drug is to be

stopped, it is recommended that it be withdrawn gradually than abruptly.

For the purpose of comparison, the following is the equivalent milligram dosage of the various corticosteroids:

- Cortisone, 25
- Hydrocortisone, 20
- Dexamethasone 0.75

  These dose relationships apply only to oral or intravenous administration of these compounds. When these substances or their derivatives are injected intramuscularly or into joint spaces, their relative properties may be greatly altered.

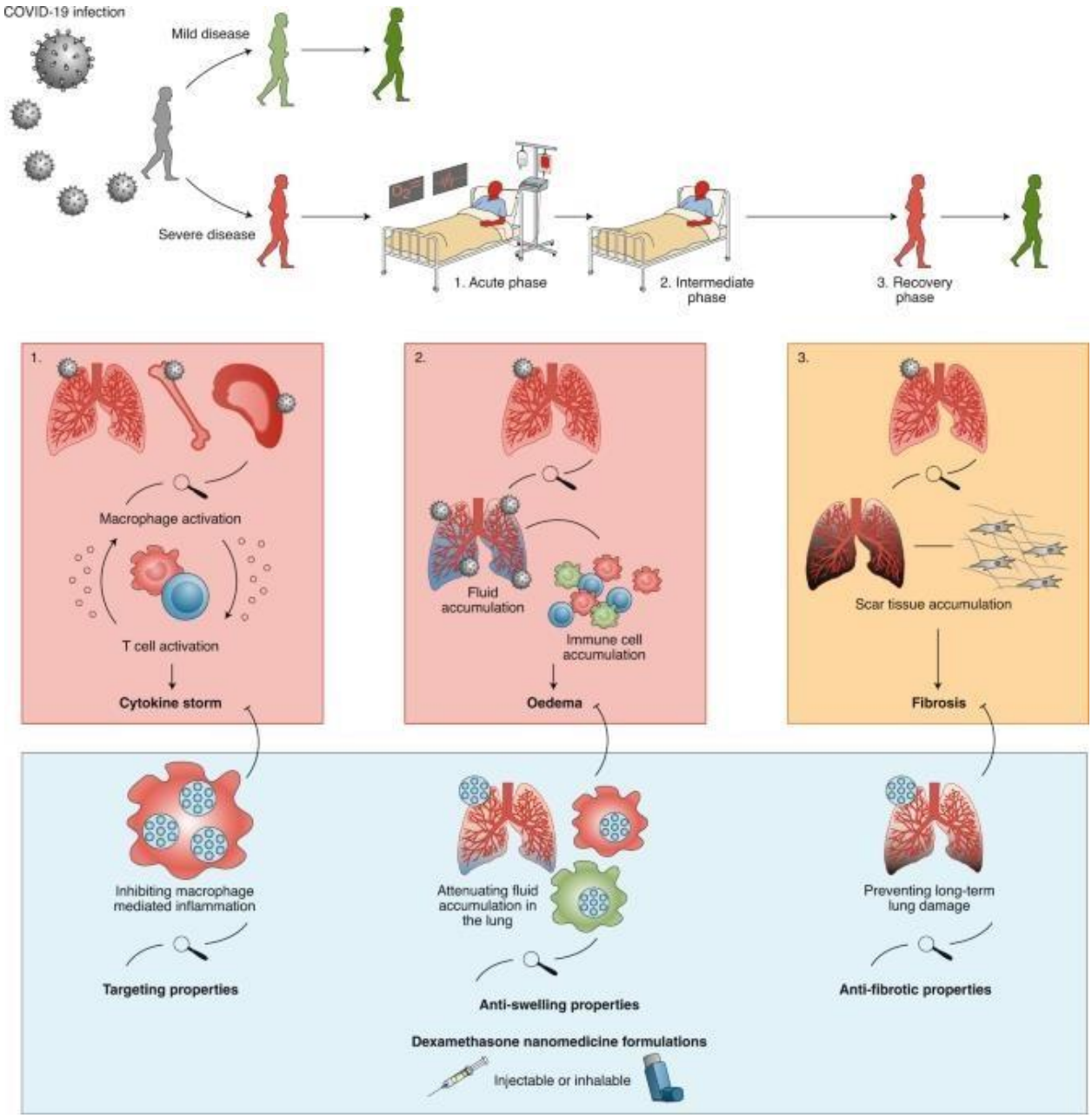
ROLE OF DEXAMETHASONE IN COVID 19 TREATMENT

As SARS-CoV-2 is a recent virus and after several researches and clinical trials COVAXIN & COVISHIELD were introduced in India which have their role in decreasing the severity or prevent COVID-19 infection.

Most of the adverse outcomes of coronavirus disease are associated with severe inflammation, lung injury secondary to ARDS and thus diffuse alveolar damage . Therefore, to control the immune-mediated damage of lung tissue, corticosteroids drugs have been given in severe cases of coronavirus such as MERS, SARS and SARS-CoV-2

- **In this controlled, open-label trial comparing a range of possible treatments in patients who were hospitalized with Covid-19, we randomly assigned patients to receive oral or intravenous dexamethasone (at a dose of 6 mg once daily) for up to 10 days or to receive usual care alone. The primary outcome was 28-day mortality. Here, we report the final results of this assessment**

ROLE OF DEXAMETHASONE IN COVID-19



Does WHO recommend Dexamethasone for the treatment of covid -19 :-

On 2 September 2020, WHO issued an interim guideline on the use of dexamethasone and other corticosteroids for the treatment of COVID-19. The guidelines were developed by a panel of WHO and international experts and investigators and is based on evidence collected from seven clinical trials.

- The guidelines make two recommendations:

Recommendation 1:

WHO strongly recommends that corticosteroids (i.e. dexamethasone, hydrocortisone or prednisone) be given orally or intravenously for the treatment of patients with severe and critical COVID-19.

Recommendation 2:

WHO advises against the use of corticosteroids in the treatment of patients with non-severe COVID-19, unless the patient is already taking this medication for another condition.

Time and duration of medication should be once daily for 7-10 days.

Daily dose should be 6 mg of dexamethasone, equivalent to 160 mg of hydrocortisone (i.e. 50 mg every 8 hours or 100 mg every 12 hours), 40 mg of prednisone, 32 mg of methylprednisolone (8 mg every 6 hours).

The panel of experts made its recommendation on the basis of the moderate certainty evidence of a mortality reduction of 8.7% and 6.7% in patients with COVID-19 who are critically or severely ill.

Lethal Mucormycosis: How steroids act as trigger and what is the treatment?

- ✞ mucormycosis fungal spores are of low virulence and usually do not cause infection.

- ✞ There were very few cases before Covid-19, but a large number of cases are only being reported in recent times due to the coronavirus disease infection, which affects immunity.
- ✞ Since steroids like dexamethasone also repress the immune system, it is necessary to be extra careful regarding its use, now more so than ever.
- ✞ If COVID patients have uncontrolled blood sugars to begin with, and if you use steroid on top of that, their blood sugar is going to be very uncontrolled. High blood sugar will lead to acidic blood, and this particular fungus, it actually thrives in high blood sugar and high acidic environments.

Reference

- Centre For Drug Evaluation & Research
- Davidson's Principles & Practice of Medicine
- Harrison's Principles of Internal Medicine
- Ahmed Hassan Article
- KD.TRIPATHI
- Wikipedia