WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.453
Volume 15, Issue 2, 157-169. Review Article ISSN 2277–7105

DEXAMETHASONE TABLET: USE IN COVID-19, PHARMACOLOGICAL PROFILE, PHARMACOLOGICAL ACTIVITY, ROLE OF NANOMEDICINE

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Article Received on 15 Dec. 2025,
Article Revised on 05 Jan. 2026,
Article Published on 16 Jan. 2026,

https://doi.org/10.5281/zenodo.18255642

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How to cite this Article: Mr. S. Dinesh*[1], Mr. V. Kathir[1], Mr. B. Gowrisankar[1], Mr. A. Thirumurugan1, Mr. Praveenkumar[2], Dr. C. Jothimanivannan[3] (2026). Dexamethasone Tablet: Use In Covid-19, Pharmacological Profile, Pharmacological activity, Role Of Nanomedicine. World Journal of Pharmaceutical Research, 15(2), 157–169.

ABSTRACT

Dexamethasone is a long-acting synthetic glucocorticosteroid with vast anti- inflammatory and immunosuppressive activities. Dexamethasone became a significant drug during the COVID-19 pandemic based on evidence showing a decrease in hospital-related fatality rates for patients requiring supplementary oxygen therapy or mechanical ventilation. Dexamethasone pharmacologically inhibits inflammation by interacting with intracellular glucocorticoid receptors, which in turn inhibits pro-inflammatory cytokines, suppresses the activation of immune cells, and stabilizes cellular membrane function. In COVID-19, dexamethasone mainly exerts a modulating effect in terms of mitigating the exaggerated immune response in addition to controlling the resultant cytokine storms, thus improving pulmonary function and clinical outcomes in severe COVID-19. Dexamethasone has a long biological half- life, an oral route of administration, a low incidence of mineralocorticoid effects, and thus it can be used systemically. Adverse effects that can be encountered with dexamethasone administration include immuno-suppression, hyperglycemia, hypertension, irritation of the gastrointestinal mucosa, emotional dysregulation, and susceptibility to secondary infections, particularly if there is inappropriate

administration for a long period of time.

KEYWORDS: *Glucocorticosteroid, Dexamethasone, COVID-19, mineralocorticoid, anti-inflammation, Cushing's syndrome, nanomedicines, MERS, SARS and SARS-CoV-2.*

INTORODUCTION

Coronavirus disease 2019 (COVID-19), caused by a new severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has recently been a serious global health concern because of its contagious nature and the levels of morbidity and mortality. Although a large number of individuals who contract the virus may have only mild to moderate symptoms, a larger number may progress to severe complications like pneumonia, hypoxemia, acute respiratory distress syndrome (ARDS), systemic inflammatory response syndrome, and multi- organ failure. Several recent researches and studies have proved that the severity of COVID-19 is not only caused by the viral infection but is primarily a consequence of the excessive "cytokine storm" response by the immune system of the individual.

With the onset of this hyperinflammatory response, corticosteroids received renewed consideration as therapeutic options because of their established anti-inflammatory and immunosuppressive properties. Of these corticosteroids, dexamethasone, which is a synthetic long-acting glucocorticoid, has been identified as one of the most successful therapeutic options offered for severe or critical cases of COVID-19 infections. It has been administered using several dosage forms, namely oral tablets, among others, due to its.

considerable utility in clinical settings for managing several conditions that trigger inflammation, allergies, autoimmune disorders, as well as malignancies.

The pharmacological properties of dexamethasone include high glucocorticoid activity and low mineralocorticoid effects, good oral bioavailability, and a long half-life. The drug works by acting on intracellular glucocorticoid receptors to affect the expression of genes involved in the inhibition of inflammatory cytokine production, prevention of immune cell activation, and regulation of vascular permeability. In patients infected with COVID-19 who are on supplemental oxygen or mechanical ventilation support, dexamethasone reduced mortality by attenuating the toxic effects of excessive inflammation on the respiratory and systemic systems.

Not with standing the proven advantage, there are some side effects associated with the use of

dexamethasone. The immunosuppression property of the drug is known to increase susceptibility to infections, prolong viral clearance, and also make patients prone to attenuation of underlying diseases, such as diabetics mellitus and hypertension. Other side effects of the drug may include irritation of the gastrointestinal tract, mood stabilization, imbalance of electrolytes, and adrenal insufficiency, among others.

The purpose of this introduction is to offer general information on the significance of dexamethasone tablet efficacy during the treatment of COVID-19. This background information details the pharmacological and therapeutic effects of dexamethasone on COVID-19 and its side effects. Awareness about all of these factors is required for using dexamethasone appropriately.

CHEMICAL AND PHYSICAL PROPERTIES OF DEXAMETHASONE

Dexamethasone is a white, odourless crystalline powder. It is stable when exposed to air. It is practically insoluble in water (≤ 0.1 mg/mL).[27] The molecular formula is 9α-fluoro-16α-methyl hydrocortisone, and the structural formula is shown in Fig. 1:

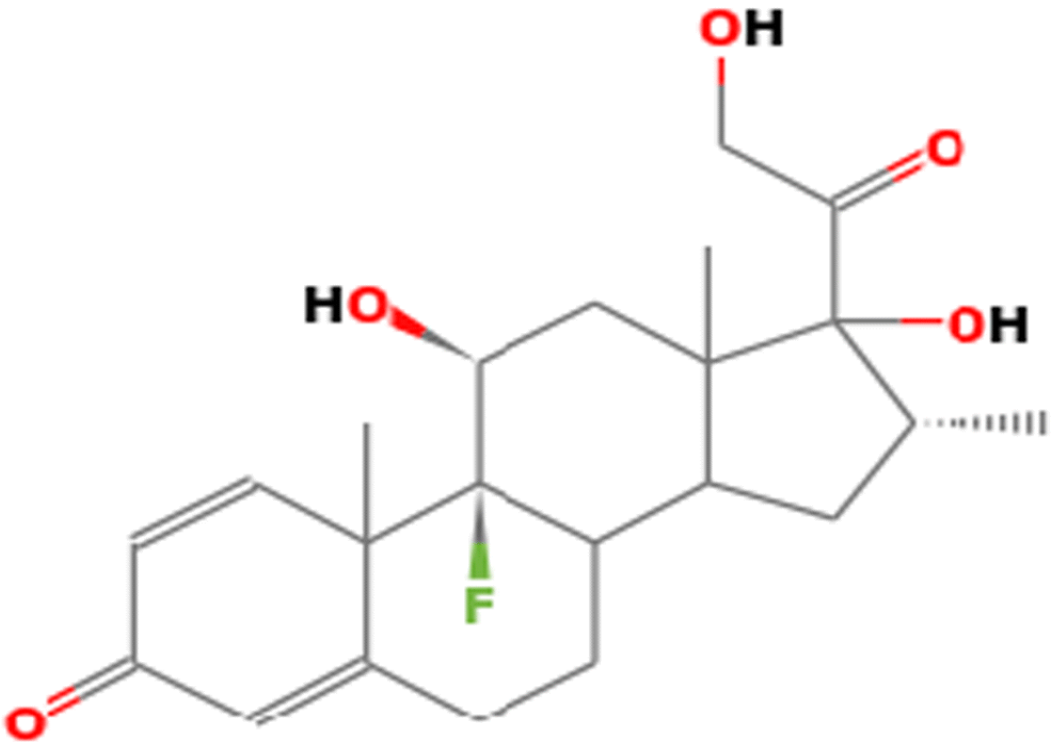

Fig.1: The chemical structure of dexamethasone.

PHARMACOLOGICAL PROFILE OF DEXAMETHASONE

Dexamethasone is a synthetic, fluorinated long-acting glucocorticoid. It is more potent than

hydrocortisone, and its equivalent dose is 0.75 mg of dexamethasone to 20 mg of hydrocortisone and has a half-life of 36 to 72 hours. Dexamethasone is highly absorbed from the gastrointestinal tract and is distributed to all tissues of the body and crosses the placenta and blood-brain barriers. Dexamethasone is about 70 to 80% bound to plasma proteins, and its metabolism occurs in the liver by CYP3A4 enzyme and is primarily cleared through urinary secretion as inactive compounds. Its pharmacological actions include binding to intracellular glucocorticoid receptors and translocation of the compound-receptor complex to the cell nucleus to alter gene transcription. These actions include inhibiting phospholipase A2, reducing synthesis of prostaglandins and leukotrienes, inhibiting secretion of inflammatory cytokines like IL-1, IL- 6, and TNF-alpha, inhibiting migration of leucocytes and lysosomal membrane stabilization. In pharmacology, dexamethasone is highly effective as an anti-inflammatory, immunosuppressive, anti-allergic, and anti-edemic and has metabolism including increased gluconeogenesis, proteolysis, and redistribution of body fat. Due to its properties, it is commonly used to treat many conditions including inflammatory and autoimmune diseases, allergies, cerebral edema, shock, malignancies, and severe COVID-19 needing oxygen therapy. Adverse reactions to its uses are those that lead to hyperglycemia, gastric irritation, hypertension, susceptibility to infections, adrenal failure, and osteoporosis especially through its chronic usage.

PHARMACOLOGICAL ACTIVITY OF DEXAMETHASONE

The pharmacological properties of dexamethasone are mainly attributed to its high glucocorticoid activity, which manifests a pronounced anti-inflammatory, immunosuppressant, anti-allergic, and anti-edematous effect. Dexamethasone, upon administration, crosses easily through the cell membrane, reaching intracellular glucocorticoid receptors primarily located in the cytoplasm. The compound forms a receptor-drug complex, which migrates to enter the nucleus. It then responds to particular DNA sequences identified as glucocorticoid response elements. This leads to modification in gene expression. This genomic effect means enhanced protein synthesis of anti-inflammatory compounds like lipocortin, which also results in reduced genes coding for inflammation-produced mediator compounds. This leads to reduced phospholipase A2 inhibition, which directly reduces arachidonic acid liberation followed by reduced prostaglandins, leukotrienes, and thromboxane production. This makes dexamethasone a useful anti-inflammatory drug due to its mediator inhibition effect.

Further, dexamethasone is known to reduce the synthesis and secretion of pro-inflammatory cytokines like interleukin-1, interleukin-6, and tumor necrosis factor-alpha, and inhibit the activation, migration, and phagocytosis of leucocytes and macrophages. This corticosteroid drug inhibits lysosomal membrane stabilization and capillary per meability, causing reduction in swelling and redness of affected areas of inflammation. Its immunosuppressive properties occur through T-cell inhibition, inhibition of antibody secretion, and suppression of cell-mediated immunity, thereby being used in autoimmune and hypersensitivity diseases. Dexamethasone is known to possess strong anti-allergic properties through inhibition of histamine release and diminishment of hypersensitivity reactions.

Additionally, dexamethasone exerts significant metabolic effects such as stimulation of gluconeogenesis and elevation of blood glucose levels, protein catabolism, negative nitrogen balance, and redistribution of body fats with minimal salt and water retention effects due to its low mineralocorticoid activity. The above-mentioned pharmacological interactions of dexamethasone render it highly efficient in treating severe cases of inflammation, allergy, and immunological reactions as well as potentially deadly conditions such as cerebral edema and inflammation related to COVID-19.

PHARMACOLOGICAL ACTION OF DEXAMETHASONE

Dexamethasone Tablet is a synthesized, long-acting glucocorticoid corticosteroid. It has widespread anti-inflammatory and immunosuppressive properties. In COVID-19, dexamethasone corticosteroid use has been recommended in cases of moderate to severe COVID-19 infection, especially in individuals requiring supplemental oxygen therapy and/or mechanical ventilation. Severe infection of COVID-19 has been characterized by an exaggerated immune response, referred to as a cytokine storm. This condition leads to severe inflammation of the lungs, pulmonary edema, and acute respiratory distress syndrome (ARDS). Dexamethasone corticosteroid has been found to reduce mortality in severe cases of COVID-19 infection; however, it does not reduce mortality in mild cases of COVID-19 that do not require oxygen.

From the pharmacologic profile point of view, dexamethasone is a highly potent, fluorinated glucocorticoid compound. It is 25-30 times more potent than hydrocortisone and has very weak mineralocorticoid effects. It is readily absorbed when taken orally, distributes evenly in all tissues of the body, passes the blood-brain and placental barriers easily, gets metabolized in the liver, and is excreted in the urine. Its biological half-life is quite long and ranges

between 36-72 hours. This enables the drug to be administered once every The pharmacological properties, mechanism of action, and activities of dexamethasone include binding to intracellular glucocorticoid receptors in the cytoplasm. The drug-receptor complex then translocates to the nucleus, binds to glucocorticoid response elements on the DNA, and alters the transcription of genes. There is an increase in the production of anti-inflammatory. proteins like lipocortin, reduction in the activities of phospholipase A_2, thereby decreasing the release of arachidonic acid, and subsequently reducing the formation of prostanoid eicosanoids like prostaglandins, leukotrienes, and other mediators of inflammation.

Additionally, dexamethasone decreases the production of pro-inflammatory cytokines like IL-1, IL-6, and TNF-α, prevents leukocyte migration, and decreases capillary permeability, thereby stabilizing lysosomal membranes. All these mechanisms demonstrate drug properties that are potent in being anti-inflammatory, immunosuppressive, anti-allergic, and in displaying intense anti-edema activities. These are major in controlling lung infections in cases of COVID-19.

Dexamethasone pharmacokinetics depend on patient's renal and/or hepatic function because this corticosteroid is primarily metabolized by the liver and then cleared by the kidneys. Adverse effects of dexamethasone depend on dosage and duration of treatment because for shorter courses, a patient can experience hyperglycemia, gastric irritation, mood changes, hypertension, and infection susceptibility, but for longer courses, adrenal gland suppression, Cushing's syndrome, osteoporosis, muscular wasting, delayed healing of wounds, peptic ulcer, and growth failure in pediatric patients can occur.

DEXAMETHASONE NANOMEDICINE FOR COVID-19

It is the first medication proven to be life-saving in individuals infected by the COVID-19 virus. Based on the world’s largest randomized controlled trials (RCTs) of medicines for the treatment of COVID-19 infections, the so-called RECOVERY trials1, six medications are currently being tested. Besides the highly potent anti-inflammatory steroid dexamethasone, there are the anti-malaria medication hydroxychloroquine, the antibacterial azithromycin, the anti-HIV combination lopinavir-ritonavir, the anti-inflammatory monoclonal antibody tocilizumab, and convalescent plasma from recovered patients. The first and third press release of the chief investigators of the RECOVERY trials reported on the first findings, of which none were clinically significant to the anti-COVID-19 efficacy of hydroxychloroquine

and lopinavir- ritonavir given to hospitalized patients.The second announcement, placed on August 21, reported that dexamethasone (6 mg per day; given orally or intravenously for 10 days) leads to a 35% lower-than-expected number of deaths by individuals hospitalized, and on the intensive care unit (ICU) of hospitals, who receive mechanical ventilation1,2, and by individuals on oxygen therapy not receiving mechanical ventilation, it leads to a 20% lower-than-expected hospital mortality rates1. Besides, it shortens hospital stays by one day (in total, 12 days in patients given dexamethasone, compared to 13 days in patients receiving standard of cares), and it significantly raises the probability of hospital discharge within the 28 days of the trials (65% versus 61%, $p < 0.001$)1,2. It is the first indication of improved outcomes of acute respiratory distress syndrome by dexamethasone, which were reported just a short time ago3, and it bears global significance. It bears not only because it proved to be the first, and, until now, sole medication significantly improving the chances of hospitalized patients of surviving the COVID-19 pandemic, but, in addition, because it had already gained world-wide popularity as an almost perfect antidote against inflammatory states, and it is known to be extremely cheap4.

Here, we would like to propose the nano-formulation of dexamethasone in order to enhance the treatment of COVID-19-related complications. On the preclinical front, a number of different diseases have already been effectively addressed using dexamethasone nanomedicines, such as: rheumatoid arthritis, inflammatory bowel disease, multiple sclerosis, liver fibrosis, wound healing, and cancer 10,11,12,7,8,9. In the context of cancer, indeed the efficacy of liposomal dexamethasone proved promising in syngeneic mouse models and mouse xenograft models10. Moreover, in multiple myeloma a promising result was observed, a disease in which dexamethasone is a mainstay drug in the context of both the induction and the maintenance phases. Notably, at the University Medical Center of the RWTH Aachen Hospital in Germany, the first in-man clinical study of the use of PEGylated liposomal dexamethasone began in the same year in patients presenting with progressive multiple myeloma11. The preliminary results are promising in terms of being safe in the dose range of 1-40 mg (dexamethasone equivalent), and signs of efficacy are observed.

The hypothesis that dexamethasone nanomedicines are useful for COVID-19 treatment has been made on the basis of the well-established notion that nanoparticles do accumulate significantly in macrophages upon intravenous administration, as well as upon inhalative administration (Fig. 2). In this respect, it should be noticed that the liposomal formulation of

amikacin, known by the brand name Arikayce, has been approved by the US FDA in 2019 for using Mycobacterium avium complex lung disease. As a nanomedicine formulation, it has been demonstrated that Arikayce effectively targets pulmonary macrophages wherein bacterial pathogen resides, thus contributing significantly to a more effective treatment of a bacterial infection rather than formulation of free amikacin. Thus, thinking along these lines, pulmonary administration of dexamethasone liposomes could be more efficient in terms of treatment of alveolar macrophages for intervening in (sub)acute phases of COVID-19.

On the other hand, intravenous administration opens a possibility of using liposomes and nanomedicines for effectively targeting dexamethasone to myeloid/lymphoid tissue, which are characterized by a high accumulation of phagocytic cells like spleen and bone marrow. Moreover, it also provides an efficient means of selective administration of a potent corticosteroid drug at a pathological location characterized by leaky vasculatures along which a large accumulation of phagocytic cells has been noticed, suppressing thus oedema induction induced through inhibition of proinflammagenesis of matrix metals proteinases/cytokines at this location of a bacterial infection. It is very important within this context to induce a characteristic of a "long-circulating" nanomedicine formulation in a manner utilizing PEGylation upon intravenous administration, especially due to an important effect of these characteristics in these formulations upon an increase in accumulation within an inflammatory macrophage population infiltrating at a pathological location. Also, importantly within this consideration, it avoids an immediate capture of these formulations within a liver- and spleen-localized macrophage population characterized by a capacity of effectively clearing these nanomedicines from a blood stream.

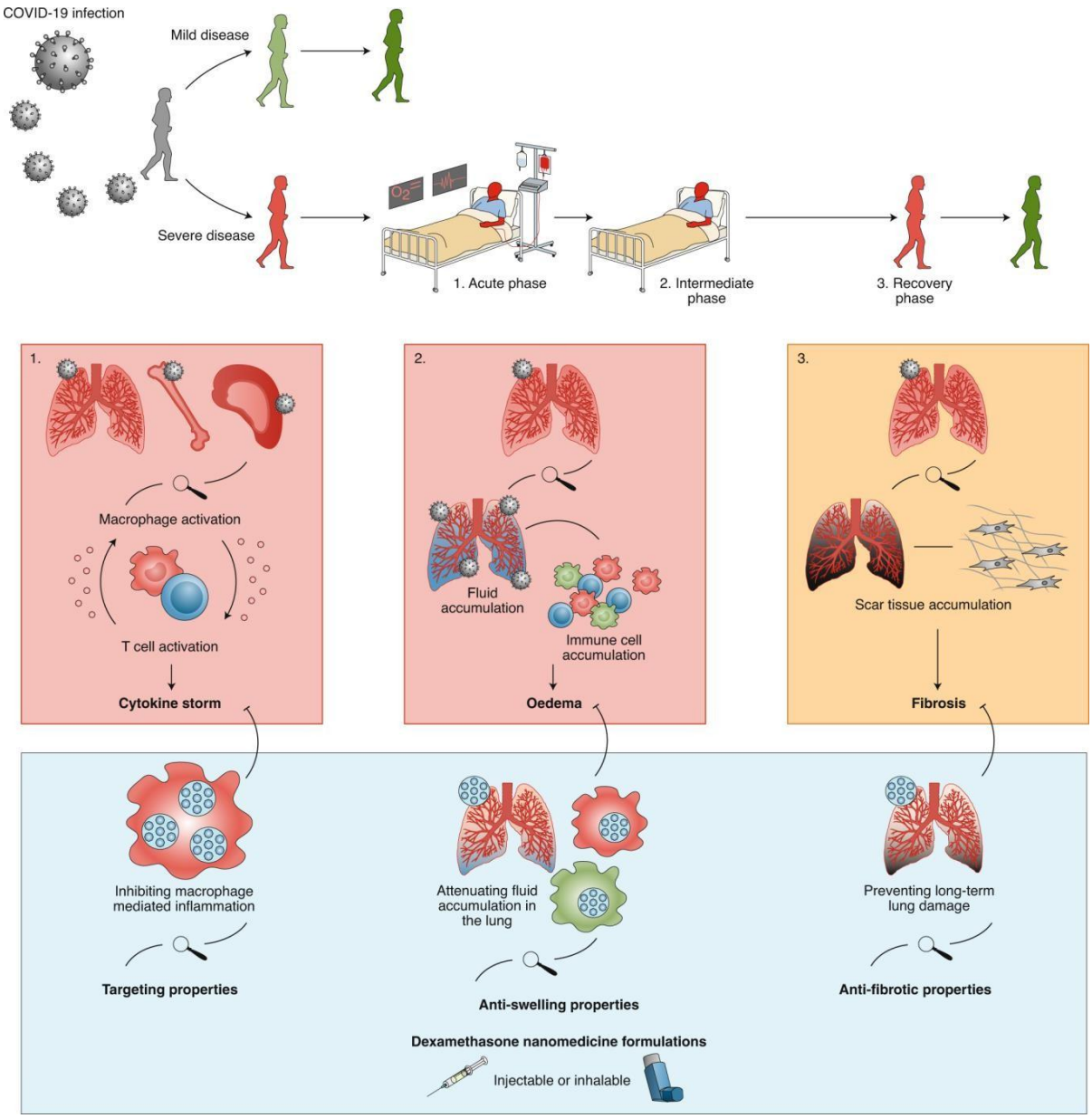

Fig. 2: Dexamethasone nanomedicines for COVID-19.

COVID-19 infection leads to several life-threatening manifestations that occur acutely and persist for long periods of time and include cytokine storm syndrome, development of edematous and fibrotic changes. The use of dexamethasone nanomedicines may prove helpful for better control of these manifestations of severe disease and for efficient recovery from life-threatening manifestations of COVID-19 infection during the acute and post-ICU phase. The dexamethasone nanomedicines are in no way close to the vaccine in terms of the control of the global cases of COVID-19. For several cases, the dexamethasone nanomedicines can assist in the daily management of the disease:

(1) Thus, as suggested above, nanomedicine formulations may aid in delivering the potent

corticosteroid effectively to inflammation-initiating and inflammation-propagating cells like phagocytic cells in the lungs, blood, and myeloid/lymphoid tissues. This would help manage MAS and cytokine storm, which have been known to contribute to COVID-19-related deaths5. Therefore, critically ill COVID-19 patients being ventilated or oxygenated would be expected to recover more effectively than before by the potent corticosteroid.

(2) Dexamethasone is a very active anti-oedema medication. Its strong activity against swelling is one of the factors of its action in various conditions, such as high-grade inflammatory cases and glioblastoma, and apparently also in COVID-19. Nano-formulation of dexamethasone may add to such activity through increased bioavailability and activity of said medication against hyper-activated immune cells within the swollen area of the lungs. The dexamethasone nanomedicine may also provide a means of maintaining activity of anti-inflammatory and anti- oedema medication in the weeks after hospital discharge. (3) Indeed, dexamethasone has been reported to be a highly potent anti-fibrotic drug. There have been several preclinical studies done to investigate the possibility that the anti-fibrotic properties of dexamethasone could be potentiated using a nanomedicine formulation7,8,9,10,11,12. Indeed, it has been reported that dexamethasone nanomedicines could be highly useful in the prevention of fibrotic conditions. Given that pulmonary fibrosis has recently appeared to be a major complication in the follow-up care management of COVID-19 patients, particularly those who have been intubated for a prolonged period of time17, dexamethasone nanomedicines could meet an urgent clinical need also at this level of COVID-19 follow-up management. But when thinking realistically about its potential in treating COVID-19, money and time are essential considerations for its use. The fact that dexamethasone is an established widely available and extremely low-cost medication that already has an established and proved role in saving lives in COVID-19 patients significantly sets the bar for a new nanomedicine product containing dexamethasone.

A nanomedicine product for dexamethasone clearly involves a higher degree of complexity for its composition and manufacture, and it first has to go through evaluation and registration for its use to be approved on the market, where it should fetch at least US$100 for its use to be economically feasible. It is our considered opinion that in this venture, therefore, the most important thing is to design appropriately its use in clinical trials so that one can clearly establish its actual value in a distinct manner. If dexamethasone given to COVID-19 patients using nanomedicine for targeted delivery is found to offer better patient outcomes, for

example, in terms of their shorter stay on mechanical ventilation or in costly ICU care19,20, then clearly this in itself is already an enormous accomplishment that easily balances out the greater degree of complexity and cost of this nanomedicine. And if in its use in these clinical trials, dexamethasone nanomedicines also proved to be able to perform better than its free form in improving patient survival rates in critically ill patients, then this is clearly an enormous accomplishment in its own right in our collective fight against this raging pandemic of COVID- 19 globally.

ADVERSE EFFECT / SIDE EFFECT

It has various adverse effects, especially when administered at higher doses or for longer periods of time due to its strong glucocorticoid effect. It brings about gastrointestinal side effects like irritation of the gastric mucosa, nausea, vomiting, heartburn, and peptic ulcers with gastrointestinal bleeding. It affects carbohydrates and brings about hyperglycemia, an aggravation of diabetes mellitus, and may cause steroid-induced diabetes. It brings about an imbalance of electrolytes with sodium and water retention with resultant hypertension and edema, with mild mineralocorticoid effects.

Dexamethasone is known to have immunosuppressive properties, which increase vulnerability to infections by bacteria, viruses, fungi, and opportunistic pathogens, while also masking signs of infection. Long-term outcomes include endocrine and metabolic disturbances like adrenal gland suppression, cushingoid appearance (moon face, buffalo hump, trunkal obesity), weight gain, skin manifestations of acne, and hirsutism. Other manifestations include skeletal disturbances like osteoporosis, skeletal muscle wasting, skeletal muscle weakness, and pathologic fracture, mainly occurring among older individuals. Neuropsychiatric manifestations such as insomnia, mood swings, irritability, anxiety, depression, euphoria, and steroid-induced psychosis may also occur. Skin manifestations include skin atrophy, bruising, striae, wound healing problems, and fragility of vessels. Ocular manifestations such as cataracts, glaucoma, and increased intraocular pressure may also occur. Moreover, acute insufficiency of the adrenal gland may occur because of the abrupt withdrawal of the medication, hence the need to taper dexamethasone carefully.

CONCLUSION

Dexamethasone Tablet has also proved to be a lifesaver in the treatment of moderate to severe COVID-19 infection, especially in patients on supplemental oxygen therapy or on mechanical ventilation. Due to its high glucocorticoid effectiveness, dexamethasone has

proved capable of mitigating the high intensity of inflammation and the subsequent cytokine storms that lead to pulmonary damage and multi-organ failures. Also, the pharmacokinetic properties possessed by dexamethasone that include high potency, high efficacy, prolonged action, anti-inflammatory, and immunosuppressant properties make the compound ideal for managing severe complications arising from COVID-19. Moreover, the combined role of nanomedicines in the delivery systems also has a vast potential in increasing the efficacy index in targeted lung delivery and minimizing systemic toxicity.

REFERENCE

1. World Health Organization. Corticosteroids for COVID-19: Living Guidance RECOVERY Collaborative Group. Dexamethasone in hospitalized patients with Covid-19. N Engl J Med. 2021; 384: 693–704.
2. WHO; 2020.
3. NIH COVID-19 Treatment Guidelines Panel. Coronavirus Disease 2019 (COVID-19) Treatment Guidelines.
4. Goodman & Gilman. The Pharmacological Basis of Therapeutics. 13th ed. McGraw-Hill; 2018.
5. Katzung BG. Basic & Clinical Pharmacology. 15th ed. McGraw-Hill; 2021.
6. Rang HP, et al. Rang and Dale's Pharmacology. 9th ed. Elsevier; 2020.
7. Barnes PJ. Glucocorticosteroids: current and future directions. Br J Pharmacol. 20 11; 163(1): 29–43.
8. Cain DW, Cidlowski JA. Immune regulation by glucocorticoids. Nat Rev Immunol. 2 017; 17: 233–247.
9. Buttgereit F, et al. Mechanisms of action of glucocorticoids. Ann Rheum Dis. 2004; 63: 145–152.
10. Meduri GU, et al. Corticosteroid treatment in ARDS. Chest., 2007; 131: 954–963.
11. Villar J, et al. Dexamethasone treatment for ARDS. Lancet Respir Med. 2020; 8: 267–276.
12. Fadel R, et al. Early short-course corticosteroids in COVID-19 pneumonia. Clin Infect Dis., 2020; 71: 2114–2120.
13. Russell CD, et al. Clinical evidence does not support corticosteroid treatment for CO VID-19. Lancet, 2020; 395: 473–475.
14. Rhen T, Cidlowski JA. Antiinflammatory action of glucocorticoids. N Engl J Med., 2 005; 353: 1711–1723.

15. Schäcke H, et al. Mechanisms involved in the side effects of glucocorticoids. Pharmacol Ther., 2002; 96: 23–43.
16. Liu J, et al. Cytokine storm in COVID-19. J Allergy Clin Immunol, 2020; 145: 347–349.
17. George PM, et al. Pulmonary fibrosis following COVID-19. Lancet Respir Med., 2020; 8: 807–815.
18. Ashwell JD, et al. Glucocorticoids in T-cell development and function. Annu Rev Immunol, 2000; 18: 309–345.
19. Barnes PJ. Anti-inflammatory actions of glucocorticoids. Clin Sci., 1998; 94: 557–572.
20. van der Goes MC, et al. Adverse effects of glucocorticoids. Ann Rheum Dis., 2 014; 73: 183–190.
21. Peters MC, et al. COVID-19 related cytokine storm. Nat Rev Immunol, 2020; 20: 363–374.
22. FDA. Dexamethasone drug label. U.S. Food and Drug Administration.
23. Chrousos GP. Glucocorticoid therapy and adrenal suppression. Endocrinol Metab Clin North Am., 2015; 44: 231–247.
24. Meijer OC, et al. Glucocorticoid receptor signaling. Endocr Rev., 2018; 39: 911–960.
25. Storm G, et al. Nanomedicine for inflammation. Nat Rev Drug Discov, 2017; 16: 69–87.
26. Metselaar JM, et al. Liposomal dexamethasone in inflammatory diseases. J Control Release, 2003; 90: 143–153.
27. Bancos I, et al. Glucocorticoid side effects. Lancet Diabetes Endocrinol, 2015; 3: 964– 975.
28. Coutinho AE, Chapman KE. Glucocorticoids and immune suppression. Trends Im munol, 2011; 32: 353–361.
29. Tleyjeh IM, et al. Systematic review of corticosteroids in COVID-19. Clin Microbiol Infect, 2021; 27: 269–279.
30. Zha L, et al. Corticosteroid treatment of COVID-19. J Infect, 2020; 81: e13–e20.