



**INTERNATIONAL JOURNAL OF
PHARMACEUTICAL SCIENCES**
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: <https://www.ijpsjournal.com>



Review Paper

Assessing Corticosteroid Related Metabolic Disturbances in Oad Patients: A Review

Jeffnisha J., Angitha Binu, Reshma Babu*, Mathan S., Shaiju Dharan

Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram.

ARTICLE INFO

Published: 26 May 2026

Keywords:

Obstructive airway disease,
asthma, COPD,
Corticosteroids,
Hypertension,
Hyperglycaemia

DOI:

10.5281/zenodo.20390947

ABSTRACT

Obstructive Airway Disease (OAD) encompasses a group of conditions characterized by airflow obstruction within the lungs, makes harder to breath. OAD covers conditions like asthma, Chronic Obstructive Pulmonary Disease (COPD), bronchitis and bronchiectasis. The prevalence of these disease differs across regions. In India, for instance, asthma affects around 3% of the population. In 2021 COPD was linked to roughly 3.5 million deaths worldwide, placing it among the most common causes of death globally. Persistent cough especially at night, wheezing, shortness of breath, chest tightness are the most commonly occurring symptoms of asthma. These can vary from person to person. Treatment aims to ease symptoms, improve breathing, prevent flare-ups and slow disease progression. Corticosteroids work by turning off the genes that trigger inflammation in the airway lining. They also reduce blood vessel widening and leakiness, which helps prevent immune cells from rushing to the site and causing further swelling. Despite their benefit they also have various side effects. This includes Hyperglycaemia, Hypertension, Weight gain, Osteoporosis, GI side effects, Dermatological adverse effects, Cardiovascular side effects and so on. Steroids acts on the liver and enhances the hepatic glucose production, thus it makes alteration in blood glucose level. They impairs the mineralization of bone matrix and also impairs the absorption of calcium from gut thus leads to bone disorder. GI side effects are associated with the increase in the dose and duration of treatment. Steroid uptake also causes immune suppression and leads to further infections

INTRODUCTION

Obstructive airway disease interfere with the normal flow of the air in and out of the lungs,

leading to breathing difficulties.^[1] These problems can arise in both upper and lower airways, and narrowing the bronchial tubes.^[2] Obstructive

*Corresponding Author: Reshma Babu

Address: Assistant Professor, Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram.

Email ✉: reshmanandu123@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



airway disease includes Asthma, COPD, Bronchitis and Bronchiectasis.^[2,3]

Asthma is a long- term condition that can affect people of all ages.^[4,5] It inflames and tightens the airways, making breathing difficulty.^[4,6] It causes coughing, wheezing and shortness of breath and which may be exacerbated by triggers.^[6] Genetic predisposition, low birth weight, prematurity, exposure to tobacco smoke and pollutions, some infections caused in airways by viruses can increase the risk of asthma.^[4]

COPD is a serious and long term condition which is characterised by the limitation in air flow which is not fully reversible.^[7] It is the fourth leading cause of death worldwide. There are two forms Chronic bronchitis and emphysema.^[8] Chronic bronchitis is a long- term inflammatory condition which causes persistent productive cough.^[9] Emphysema causes damage to the lung alveoli and decreases the elasticity of the air sacs in the lungs.^[10] Smoking is the major risk factor of COPD.^[8]

Corticosteroids play a key role in the management of OAD due to its anti- inflammatory and immunosuppressive property. They are used in both inhaled and systemic ways, based on the severity of the condition. Although corticosteroids are highly effective, they can lead to several side effects. These potential risks, which often vary based on the dose and length of treatment, can sometimes restrict their long- term use. The most commonly occurring adverse effects include Osteoporotic fractures, suppression of the hypothalamic- pituitary- adrenal axis, Cushingoid features, Diabetes and Hyperglycemia, Myopathy, Glaucoma and Cataracts, Psychiatric disturbances, Immunosuppression, Cardiovascular disease, Gastrointestinal and Dermatologic adverse effects.^[11]

PREVALENCE OF OAD:

In 2019 it was estimated that about 262 million people were affected by asthma and 455,000 death cases.^[4] The prevalence varies widely among countries and the geographical areas. Asthma affects an estimated 3% of the population in India and ranks as the second leading cause of death among chronic respiratory illness.^[12]

In contrast, COPD is a major global health concern, responsible for about 3.5 million deaths in 2021, making it the fourth leading cause of death worldwide.^[7] As of 2020, the global prevalence of COPD was around 10.6%.^[13] According to the World Health Organization, approximately 65 million people suffer from moderate to severe COPD, contributing to nearly 5% of all deaths worldwide.^[14]

SYMPTOMS:

Symptoms can differ widely between individuals and also vary depending on the specific condition. Persistent cough especially at night, wheezing, shortness of breath, chest tightness are the symptoms of asthma and these can be triggered by factors such as dust, smoke, chemical fumes, pollens, pet dander, feathers and even strong scents like perfumes.^[4]

The symptoms of COPD is same as asthma but have persistent, daily cough with phlegm, wheezing, progressive shortness of breath and fatigue.^[8]

DIAGNOSIS:

Lung function tests are commonly used to assess how well the airways are working. One such test, Spirometry which measures how much air a person can exhale and how quickly they can do it. Peak Expiratory Flow (PEF) test measures the speed of the air blown out using maximum effort. An arterial blood gas test helps doctors see how well your lungs are working by checking the levels of oxygen and carbon dioxide in your blood.

In addition, imaging techniques like chest X- ray, CT scan are often used to get a clearer view of the lungs and detect any abnormalities.^[15]

TREATMENT:

The main aim of the treatment is to relieve symptoms, improve the lung function, reduce exacerbation and prevent disease progression. A comprehensive approach that includes pharmacological, non- pharmacological and preventive treatments is required for effective therapy.

For the treatment of acute symptoms bronchodilators are used. These medicines help open up your airways by calming the muscles around them, so you can breathe more easily. Salbutamol is a short- acting beta2- agonist which provide quick symptomatic relief. They provides actions within 15- 20 minutes and the action lasts for 4-6 hours. Long- acting beta 2 agonist are used for maintenance therapy, particularly in moderate to severe diseases.^[16]

Anticholinergics such as Ipratropium bromide and Tiotropium bromide are available, this blocks the neurotransmitter called as Acetylcholine.^[16]

If the condition is severe then bronchodilators can be combined with inhaled steroids. Steroids helps to reduce the swelling and inflammation of the airways, an makes easier for breathing.^[17]

Corticosteroids reduces the inflammatory mediator including T lymphocytes, eosinophils, mast cells and dendritic cells.

They suppress the activated inflammatory genes in airway epithelial cells including the gene that codes for cytokines, chemokines, adhesion molecules, inflammatory enzymes and receptors.^[18] It decreases the vasodilation and permeability of capillaries, and also decreases the leukocyte migration to the site of inflammation.^[19]

Inhaled corticosteroid are primarily used to manage chronic, persistent asthma, while short-term oral corticosteroids are typically prescribed

during asthma exacerbation to provide quick relief.^[20] Lower dose of steroid produces anti-inflammatory effect whereas higher dose provides immune suppressive action.^[19]

Antibiotics can also be used to treat flare- ups caused by infections.^[17] Antibiotics prophylaxis are given to patients with severe obstruction and frequent exacerbation.^[21] They are agents which works by killing the bacteria that causes symptoms. The mainly used drugs in the treatment of COPD exacerbation includes Amoxicillin with clavulanic acid, Macrolides or Tetracyclines.^[22]

METABOLIC COMPLICATIONS:

Corticosteroid are accepted as the first line agent for the treatment of asthma, despite their benefits in various conditions they also have numerous side effects.^[23] They affect all the metabolic pathways in human body.^[24]

Hyperglycemia, Obesity, Hypertension, Osteopenia are associated with both short and long term use of steroids, whereas Osteoporosis, Adrenal suppression and skin changes usually occurs with long term use of steroids.^[25] Inhaled corticosteroid causes dose related systemic adverse effects, but comparing to oral corticosteroids therapy the adverse effects are low.^[23]

HYPERGLYCEMIA:

Corticosteroid induced hyperglycemia is observed in one- third of patients who undergone steroid therapy.^[26,27,28] The risk factors includes age, BMI and family history of diabetes.^[30]

Steroids acts on the liver and enhances the hepatic glucose production.^[26] They increase insulin resistance and decreases the production and secretion of insulin.^[29] Acting on liver cells it directly increases the endogenous glucose production via activating a number of genes which involved in hepatic metabolism of carbohydrates leading to enhanced gluconeogenesis. High blood



sugar increases the risk of infections.^[26] Metformin has been used widely as a blood sugar lowering agent. ^[26,28] In the intestine, metformin reduces glucose absorption in the proximal region, a process thought to be linked to enhanced glucose utilisation by the enterocytes.^[26]

OSTEOPOROTIC FRACTURES & OSTEONECROSIS:

Steroid induced osteoporosis is the most common form of secondary osteoporosis.^[31] Corticosteroid impairs the mineralization of bone matrix and also impairs the absorption of calcium from gut. They also decrease the bone formation by reducing the activity and lifespan of osteoblasts, promoting their apoptosis as well as the apoptosis of osteocytes. Research shows that even 5mg/day of prednisone leads to bone loss, associated with reduction in bone mineral density.^[11] High concentration of glucocorticoid decrease the rate of bone formation, osteoblast number and osteocytes number.^[31]

In women hormone replacement therapy can be used to prevent osteoporosis. Use of Bisphosphonates can reduce the bone breakdown and increase the bone density in spina and hip. Raloxifene is a selective estrogen receptor modulator commonly prescribed to treat osteoporosis in post menopausal women. Calcitonin is a hormone produced by the thyroid gland which may slow bone loss and prevent spine fractures. Studies shows that use of statins, which are cholesterol lowering agent by women for at least a year can reduce the risk of bone fractures.^[32]

CARDIOVASCULAR ADVERSE EFFECTS:

The use of corticosteroid is associated with Hypertension, Hyperglycemia, Obesity and also Hyperlipidaemia.^[11] Patients with chronic use of steroid are at higher risk of CVA such as coronary artery disease, heart failure and stroke.^[34] The use

of steroid alters the mineralocorticoid activity which leads to the retention of free water in the body, and retention of sodium in kidney and this finally results in hypertension.^[11,35] It is associated with high dose and duration of treatment. Diuretics like hydrochlorothiazide, furosemide and spironolactone can be given to treat steroid-induced hypertension.^[33]

The impact of steroid in lipid profile is higher and pay way for other cardiovascular diseases. The activity of acetyl- CoA and free fatty acid synthetase was increased by corticosteroid, this leads to the elevation of total cholesterol, VLDL and triglycerides.^[36]

GASTROINTESTINAL ADVERSE EFFECTS:

The use of steroid causes gastritis, peptic ulcer, dyspepsia and abdominal distention. The use of NSAID along with steroid increases the risk of peptic ulcer and GI bleeding.^[11] When patient take high dose of steroids they inhibit the prostaglandin biosynthesis, thereby inhibiting the gastric alkaline response and produce severe lesions.^[37] If 20mg/day of prednisolone is taken for 1 month it significantly increase the secretion of gastric acid.^[38] The risk increases with the increase in dose and duration of treatment. Proton pump inhibitors are frequently prescribed as a treatment option.^[39]

DERMATOLOGICAL ADVERSE EFFECTS:

Steroid induces skin atrophy, causes thinning and fragility of the skin. They causes impairment in the cutaneous wound healing.^[11] They inhibit the migration of leukocytes and macrophages, reduces the collagen synthesis and wound maturation.^[11]

CUSHINGOID FEATURES:

It refers to weight gain and abnormal fat distribution caused by excess cortisol. They may even develop within first two months of treatment and even with low dose.^[11] Even low dose of

steroid ie; 5mg/day of Prednisone can results in Cushing syndrome.^[39] Patients with high BMI, youngest patients and those with higher caloric intake are at higher risk in developing Cushing syndrome.^[11] The adverse effects can be minimised by initiating the steroid therapy with low dose.^[40]

MYOPATHY:

Long-term use of corticosteroids can result in muscle weakness and shrinkage, particularly in the muscles closest to the body's core, such as those in the upper arms, thighs, and hips. These symptoms can develop gradually, typically appearing within a few weeks to months of starting treatment. The risk is directly proportional to the dose.^[11] Both catabolic and anti- anabolic processes are thought to be involved in corticosteroid induced myopathy. Steroid also induce myocyte apoptosis through receptor based and mitochondrial based signalling pathways involving cytochrome C and the caspase cascade.

Steroid inhibits the amino acid transport into the cell and down regulate differentiation of satellite cells into muscle fibers by blocking the transcription factor called myogenin, thus it leads to the inhibition of protein synthesis and myogenesis. They also lowers the serum potassium and phosphate level, which leads to muscle weakness.^[41] The symptoms will resolve within 2-3 weeks after the withdrawal of drug.^[11]

OPHTHALMOLOGIC EFFECTS:

The risk of Glaucoma and Cataract increases with the increase in the dose of the drug. The use of systemic steroid causes the increase in the intraocular pressure, which may cause optic nerve atrophy and visual field loss.^[11] After discontinuing the therapy the increased pressure will resolve within few weeks, however the damage caused to the optic nerve and vision loss are permanent.^[11]

Systemic use of steroid may leads to a rare adverse effect called as Central Serous Chorioretinopathy, a condition that cause fluid accumulation behind the retina leading to vision impairment. Cataract are associated with long term use of steroid especially in older adults. They also causes the retention of fluid in the eye leading to blurring of vision.^[42]

INFECTION:

The risk of infection increases with the increase in the dose. A meta- analysis found that the infection rate is higher for the patients using systemic steroid and when the dose is 10mg/day.^[11]

It decreases the production of inflammatory mediators and increases the risk of bacterial infections, viral infections such as atypical mycobacterial infection and cytomegalovirus infection and fungal infection including endemic mycoses, cryptococcosis, aspergillosis and candidiasis.^[43]

PSYCHIATRIC-DISTURBANCE:

The most common adverse effect of short term steroid therapy are euphoria and hypomania. They also cause disturbances of mood, cognition, sleep and behavioural changes.^[44] Psychosis is seen with patients who are taking steroids at high dose and for a prolonged time.^[11]

Chronic or high dose of steroid exposure enhances the activity of Dopaminergic neurotransmission leads to mania, hypomania or psychosis. They may also cause the reduction in Serotonin level leads to Depression.^[45]

ADRENAL SUPPRESSION:

Steroid- induced adrenal suppression results from the prolonged use of exogenous glucocorticoid.^[46] This suppress the hypothalamic- pituitary- adrenal axis and diminish endogenous cortisol production.

Adrenal suppression occurs when the patient gets steroid therapy for more than 3 weeks. Adrenal crisis may result from abrupt withdrawal of drug without tapering.^[47] Adrenal suppression is associated with the sudden withdrawal of drug without tapering, thus gradual tapering is required as per the treatment protocols.^[11]

WEIGHT GAIN:

Steroids influence both fat metabolism and its distribution throughout the body. They increase appetite and increase the calorie intake, leading to weight gain.^[48] The drug stimulate hypothalamus, a part of the brain which controls hunger. It also increases the accumulation of fat in face, neck and abdomen.^[48,49] Decreases in the metabolism causes the body to burn fewer calories. Long term use affects the adrenal gland and leads to the release of high amount of cortisol, which is a stress hormone.^[50] This imbalance cause the retention of water and salt in the body, leading to blotting and weight gain.^[49]

CONCLUSION

Corticosteroids play a major role in the treatment of obstructive airway diseases like asthma and COPD due to their powerful ability to reduce airway inflammation and improve breathing. However, their benefits come with the potential for a wide range of side effects, especially when used in high doses or over a long period. These can affect blood sugar levels, bone strength, mental health, cardiovascular function, and immune response.

Because of this, it's essential for healthcare providers to take an individualized approach-prescribing the lowest effective dose for the shortest necessary time, and regularly monitoring patients for any complications. Educating patients about proper inhaler technique, lifestyle changes, and the importance of follow-up care can also

improve outcomes. With the right balance, corticosteroids can remain a safe and effective tool in managing chronic airway diseases while minimizing long-term harm.

REFERENCES

1. Alana Biggers. Obstructive lung disease: Symptoms, diagnosis, and treatment. Medical News Today. 2019. <https://www.medicalnewstoday.com/articles/324406#obstructive-vs-restrictive>
2. Broomfield A, Kenth J, Bruce IA, Tan HL, Wilkinson S. Respiratory complications of metabolic disease in the paediatric population: a review of presentation, diagnosis and therapeutic options. *Paediatric Respiratory Reviews*. 2019;32(1):55-65.
3. Category of respiratory disease characterised by airway obstruction. Wikimedia foundation, 2007. https://en.m.wikipedia.org/wiki/Obstructive_lung_disease
4. World Health Organization. Asthma [Internet]. World Health Organization. 2024. <https://www.who.int/news-room/fact-sheets/detail/asthma>
5. National Heart, Lung, and Blood Institute. What Is Asthma? National Heart, Lung, and Blood Institute; 2024. <https://www.nhlbi.nih.gov/health/asthma>
6. Hashmi MF, Cataletto ME. Asthma [Internet] PubMed. Treasure Island (FL): StatPearls Publishing; 2024 [Cited on 2024 May 3]. <https://www.ncbi.nlm.nih.gov/books/NBK430901/>
7. World Health Organization (WHO). Chronic obstructive pulmonary disease (COPD). 2024. <https://www.who.int/news-room/fact-sheets/detail/chronic-obstructive-pulmonary-disease>
8. Chronic obstructive pulmonary disease (COPD) [Internet]. MedlinePlus Medical Encyclopedia. 2023 [cited 2021 Jul 1].



- <https://medlineplus.gov/ency/article/000091.htm>
9. Widysanto A, Mathew G. Chronic bronchitis [Internet]. National Library of Medicine. StatPearls Publishing. 2025. <https://www.ncbi.nlm.nih.gov/books/NBK482437/>
 10. American Lung Association. Emphysema. American Lung Association [Internet]. 2023. <https://www.lung.org/lung-health-diseases/lung-disease-lookup/emphysema>
 11. Hodgens A, Sharman T. Corticosteroid. PubMed. Treasure Island. StatPearls Publishing. 2025 [Cited on 2023 May 1]. <http://www.ncbi.nlm.nih.gov/books/NBK554612/>
 12. Agrawal S, Pearce N, Ebrahim S. Prevalence and risk factors for self-reported asthma in an adult Indian population: a cross-sectional survey. *The International Journal of Tuberculosis and Lung Disease*. 2013;17(2):275–82.
 13. Boers E, Barrett M, Su JG, Benjafield AV, Sinha S, Kaye L, et al. Global Burden of Chronic Obstructive Pulmonary Disease through 2050. *JAMA Network Open*. 2023;6(12). doi:10.1001/jamanetworkopen.2023.46598.
 14. Verma A, Gudi N, Yadav UN, Roy MP, Mahmood A, Nagaraja R, et al. Prevalence of COPD among population above 30 years in India: A systematic review and meta-analysis. *Journal of Global Health*. 2021;11(2).
 15. National heart, lung and blood institute. COPD - diagnosis [Internet]. 2022. <https://www.nhlbi.nih.gov/health/copd/diagnosis>
 16. Cleveland Clinic. Bronchodilators: Asthma, Purpose, Types & Side Effects. Cleveland Clinic. 2022. <https://my.clevelandclinic.org/health/treatments/17575-bronchodilator>
 17. National Heart, Lung, and Blood Institute. COPD - Treatment. 2022. <https://www.nhlbi.nih.gov/health/copd/treatment>
 18. Barnes PJ. Corticosteroid Therapy For Asthma. *Pulmão RJ*. 2012;21(2):53–9. https://www.sopterj.com.br/wpcontent/themes/_sopterj_redesign_2017/_revista/2012/n_02/09.pdf
 19. Prednisolone [Internet]. DrugBank. 2005. <https://go.drugbank.com/drugs/DB00860>
 20. Ramadan AA, Gaffin JM, Israel E, Phipatanakul W. Asthma and Corticosteroid Responses in Childhood and Adult Asthma. *Clinics in chest medicine*. 2019;40(1):163–77. doi: 10.1016/j.ccm.2018.10.010.
 21. Al-Hasan MN, Al-Jaghbeer MJ. Use of Antibiotics in Chronic Obstructive Pulmonary Disease: What is Their Current Role in Older Patients? *Drugs & Aging*. 2020;37(9):627–33. doi: 10.1007/s40266-020-00786-7.
 22. Sherrell Z. Antibiotics for COPD exacerbation: Options, considerations, and more. [Internet]. MedicalNewsToday. 2023. <https://www.medicalnewstoday.com/articles/antibiotics-for-copd-exacerbation>
 23. Lipworth BJ. Systemic Adverse Effects of Inhaled Corticosteroid Therapy. *Archives of Internal Medicine*. 1999;159(9):941.
 24. Deshmukh C. Minimizing side effects of systemic corticosteroids in children. *Indian Journal of Dermatology, Venereology and Leprology*. 2007;73(4):218-21.
 25. Kulkarni S, Durham H, Glover L, Ather O, Phillips V, Nemes S, et al. Metabolic adverse events associated with systemic corticosteroid therapy—a systematic review and meta-analysis. *BMJ Open*. 2022;12(12):e061476–6.
 26. Sanpawithayakul K, Korbonits M. Metabolic complications of glucocorticoids- Prevention



- by metformin. *Annales d'endocrinologie*. 2023;84(4):483-97.
doi: 10.1016/j.ando.2023.05.002.
27. Cho JH, Suh S. Glucocorticoid-induced hyperglycemia: A neglected problem. *Endocrinology and metabolism*. 2024;39(2):222-38.
<http://dx.doi.org/10.3803/EnM.2024.1951>
28. Bonaventura A, Montecucco F. Steroid-induced hyperglycemia: An underdiagnosed problem or clinical inertia? A narrative review. *Diabetes Research and Clinical Practice*. 2018;139(1):203–20. doi: 10.1016/j.diabres.2018.03.006.
29. Vaishali Limbachia, Nunney I, Page DJ, Barton HA, Patel L, Thomason GN, et al. The effect of different types of oral or intravenous corticosteroids on capillary blood glucose levels in hospitalized in patients with and without diabetes. *Clinical Therapeutics*. 2023;46(2):e59-e63. doi: 10.1016/j.clinthera.2023.11.013.
30. Aberer F, Hochfellner DA, Sourij H, Mader JK. A Practical Guide for the Management of Steroid Induced Hyperglycaemia in the Hospital. *Journal of Clinical Medicine*. 2021;10(10): 2154. doi: 10.3390/jcm10102154.
31. Briot K, Roux C. Glucocorticoid-induced osteoporosis. *Rheumatic and Musculoskeletal Disorders Open*. 2015;1(1):4–14: e000014. doi: 10.1136/rmdopen-2014-000014.
32. Stanbury RM, Graham EM. Systemic corticosteroid therapy—side effects and their management. *British Journal of Ophthalmology*. 1998;82(6):704-8. doi: 10.1136/bjo.82.6.704.
33. Darrell Hulisz. Drug-Induced Hypertension. [Internet]. *US Pharm*. 2008;33(1):11-20. <https://www.uspharmacist.com/article/drug-induced-hypertension>
34. Alan IS, Alan B. Side Effects of Glucocorticoids. *IntechOpen*. 2017. <https://www.intechopen.com/chapters/58357>
35. Ferrari P. Cortisol and the renal handling of electrolytes: role in glucocorticoid-induced hypertension and bone disease. *Best Practice & Research Clinical Endocrinology & Metabolism*. 2003;17(4):575–89. <https://www.sciencedirect.com/science/article/abs/pii/S1521690X03000538>
36. Lau KK, Tancredi DJ, Perez RV, Butani L. Unusual Pattern of Dyslipidemia in Children Receiving Steroid Minimization Immunosuppression after Renal Transplantation. *Clinical Journal of the American Society of Nephrology*. 2010;5(8):1506–12.
37. Black HE. The Effects of Steroids Upon the Gastrointestinal Tract. *Toxicologic Pathology*. 1988;16(2):213–22.
38. Strickland RG, Fisher JM, Taylor KB. Effect of Prednisolone on Gastric Function and Structure in Man. *Gastroenterology*. 1969;56(4):675–86.
39. Caplan A, Fett N, Rosenbach M, Werth VP, Micheletti RG. Prevention and management of glucocorticoid-induced side effects: A comprehensive review. *Journal of the American Academy of Dermatology*. 2017;76(1):11–6.
40. Priyanka B, Prasanna D, Krupa S, B. Sreenivasulu. Corticosteroid Induced Cushing's Syndrome. *Indian Journal of Pharmacy Practice*. 2019;12(2):136-9.
41. Surmachevska N, Tiwari V. Corticosteroid Induced Myopathy. *Treasure Island: StatPearls Publishing*; 2023. [Cited on 2023 April 17]. <https://www.ncbi.nlm.nih.gov/books/NBK557731/>
42. Short-Term vs. Long-Term Steroid Use: Effects on Your Eyesight. *Sanjeevan*. 2025.



- <https://sanjeevan.in/short-term-vs-long-term-steroid-use-effects-on-your-eyesight/>
43. Mustafa SS. Steroid-induced secondary immune deficiency. *Annals of Allergy, Asthma & Immunology*. 2023;130(6):713-7. doi: 10.1016/j.anai.2023.01.010.
 44. Thomas P, Warrington MD, Michael Bostwick MD. Psychiatric Adverse Effects of Corticosteroids. *Mayo Clinic Proceedings*. 2006;81(10):1361–7. [https://www.mayoclinicproceedings.org/article/S0025-6196\(11\)61160-9/fulltext](https://www.mayoclinicproceedings.org/article/S0025-6196(11)61160-9/fulltext)
 45. Parasher A, Bez J. Steroid induced psychiatric adverse effects: an overview of risk factors, clinical features and management. *International Journal of Research in Medical Sciences*. 2020;8(6):2365-70. <https://www.msjonline.org/index.php/ijrms/article/view/8091>
 46. Noura Nachawi, Li D, M. Cecilia Lansang. Glucocorticoid-induced adrenal insufficiency and glucocorticoid withdrawal syndrome: Two sides of the same coin. *Cleveland Clinic journal of medicine*. 2024;91(4):245–55. doi: 10.3949/ccjm.91a.23039.
 47. Pazderska A, Pearce SH. Adrenal Insufficiency – Recognition and Management. *Clinical Medicine*. 2017;17(3):258–62. doi: 10.7861/clinmedicine.17-3-258 <https://pmc.ncbi.nlm.nih.gov/articles/PMC6297573/>
 48. Modi J. Do Steroids Make You Gain Weight? *Buzzrx.com*. 2022. <https://www.buzzrx.com/blog/do-steroids-make-you-gain-weight>
 49. Clinic TS. Understanding the Link Between Steroids and Weight Gain. *The Silhouette Clinic*. 2023. <https://thesilhouetteclinic.com/steroids-and-weight-gain/>
 50. Clinic C. Moon Face: Causes & Treatment. *Cleveland Clinic*. 2024. <https://my.clevelandclinic.org/health/symptoms/moon-face>.
 51. Peter K Wung, Troy Anderson, Kevin R Fontaine, Gary S Hoffman. Effects of glucocorticoids on weight change during the treatment of Wegener’s Granulomatosis. 2008;59(5):746-53.
 52. Stanley M.H Chan, Stavros Selemidis, Steven Bozinovski, Ross Vlahos, et al. Pathobiological mechanisms underlying metabolic syndrome in chronic obstructive pulmonary disease: Clinical significance and therapeutic strategies. 2019;198:160-88. doi: 10.1016/j.pharmthera.2019.02.013.
 53. Mesut Savas, Vincent L Wester, et al. Association between systemic and local corticosteroid use with metabolic syndrome and body mass index. 2017;102(10):3765-74.
 54. Imran Sulaiman, Breda Cushen, Garrett Green, et al. “Objective Assessment of Adherence to Inhalers by Patients with Chronic Obstructive Pulmonary Disease”. 2017; 195(10):1333-43.
 55. Marie T. Brown, Jennifer Bussell, Suparna Dutta, Katherine Davis, Shelvy Strong, Suja Mathew. Medication Adherence: Truth and Consequences. 2016;351(4):387-99. doi: 10.1016/j.amjms.2016.01.010.
 56. Ebtessam Islam, Chok Limsuwat, Gilbert Berdine, Raed Alalawi, Keneth Nugent. Corticosteroids cause hyperglycemia in patients with acute chronic obstructive pulmonary disease exacerbation. 2013;144(4):1-3.
 57. Herman Joseph Johannesmeyer, Kayvan Moussavi, Kerry Anne Rambaran, Kristica Kolyouthapong. Corticosteroid administration and glycemic outcomes during treatment of acute exacerbation of COPD.



2022;(8):1-8. doi:
10.1016/j.ajmo.2022.100027.

58. Spoorthy Kulkarni, Hannah Durham, Luke Glover.,2022. Metabolic adverse effects associated with systemic corticosteroid therapy – A systematic review and meta-analysis. *BMJ Open*. 2022;12(12):1-14.
59. Ruth E Costello, Belay B Yimer, et al. Glucocorticoid use is associated with an increased risk of hypertension. 2021;60(1):132-9.
60. Nur Atik, Rira Uji Hayati, et al. Correlation between steroid therapy and lipid profile in Systemic Lupus Erythematosus patients. 2020;12:41-46.
61. David B Price, Jaco Voorham, Robert Fogel. Inhaled corticosteroids in COPD & onset of type 2 DM & osteoporosis: matched cohort study. 2019;29(1):38.

HOW TO CITE: Jeffnisha J., Angitha Binu, Reshma Babu, Mathan S., Shaiju Dharan, Assessing Corticosteroid Related Metabolic Disturbances in Oad Patients: A Review, *Int. J. of Pharm. Sci.*, 2026, Vol 4, Issue 5, 6641-6650, <https://doi.org/10.5281/zenodo.20390947>

