

A Study on Assessment of Corticosteroid Drug Induced Psychosis and Treatment Based Outcomes

R. Gautham Chakra^{*1}, Badvel Manvitha², Kanamanayanapalli Nethra², Yalagalapalle Bhavana³, Shaik Tasleem², Panabaka Gangothi², Shaik Mohammad Khaja Mohiddin², Matli Yamuna², P. Rathish², Dudekula Hasan²

¹Assistant Professor, Department of Pharmacy Practice, Saastra College of Pharmaceutical Education and Research, Totapalli Gudur (M), Varigonda(V), Nellore-524311, Andhra Pradesh, India

²B.Pharm student, Saastra College of Pharmaceutical Education & Research, Totapalli Gudur (M), Varigonda (V), Nellore-524311, Andhra Pradesh, India

*Corresponding E-mail: gauthamrowdhra05@gmail.com

KEYWORDS

Corticosteroids, Psychosis, Mood disturbances, Psychological Interventions, Drug treatment.

Received 27 Jan 2026
Revised 11 Feb 2026
Accepted 25 Feb 2026
Published 09 Mar 2026

ABSTRACT

Introduction: Corticosteroids are integral to treatment across various medical domains. They are synthetic analogs of natural steroid hormones produced by the adrenal cortex like glucocorticoids (GCs), mineralocorticoids and sexual hormones. **Aim:** The study aimed to assess corticosteroid drug induced psychosis and treatment based outcomes. **Methodology:** The prospective observational study was carried out for a period of 6 months. The study was conducted in General Medicine department in a tertiary care hospital. A written and informed consent was obtained from the recruited patients. A Total of 85 patients were enrolled in the study. **Results & Discussion:** In our study 46-55 years age patients were more 33(28.82%) compared to other ages. In our study Male patients were more 51 (60 %), compared to Female patients were 34(40 %). The oral route drug administration patients were more 56 (65.88%), compared to parenteral route drug administration patients were 29(34.11%). The Management of steroid drug induced psychosis includes Psychological support patients were more 39(45.88%) compare to Psychological therapy patients were 22(25.88%), Psychological interventions patients were 24(28.23%). The Treatment for steroid drug induced psychosis includes Lithium prescribed patients were more 25 (29.41%) compare to other prescribed drugs. **Conclusion:** The effective diagnosis primarily involves excluding other potential causes, while prevention primarily focuses on minimizing dosages and avoiding the unnecessary prolongation of drug treatment. Although steroid-induced psychosis can typically be managed by tapering and discontinuing the offending steroid, patients may still experience persistent mood disturbances and psychosis.

INTRODUCTION

Corticosteroids are integral to treatment across various medical domains. They are synthetic analogs of natural steroid hormones produced by the adrenal cortex like glucocorticoids (GCs), mineralocorticoids and sexual hormones (testosterone and androstenedione). The mechanism of action of corticosteroids involves the activation of intracellular glucocorticoid receptors, which act as ligand-activated transcription factors, thereby modifying gene transcription in various ways¹⁻³.

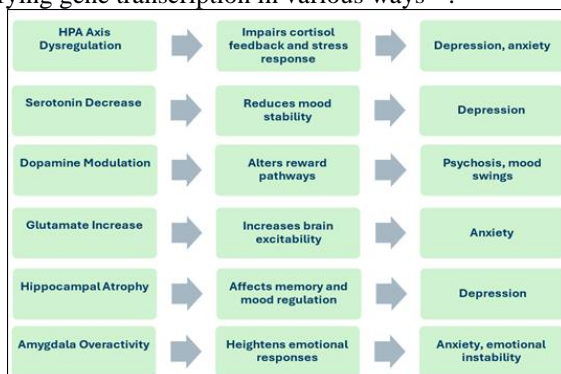


Fig 1: Mechanisms in steroid drug induced psychosis

Epidemiology

CIPDs have been reported in all age groups. The incidence of corticosteroid-induced psychiatric symptoms is very wide, ranging from 2% to 60%, and it is estimated to be seen in 5–18% of the patients treated with corticosteroids. A recent systematic review and meta-analysis described the pooled proportion of CIPDs: 22% for depression, 11% for mania, 8% for anxiety, 16% for delirium, and 52% for behavioral changes⁴.

Risk Factors: Age and Gender

However, some evidence suggests that women are more susceptible to developing depression, in contrast to men, who are more susceptible to developing mania, confusion, or disorientation. Regarding age, the risk of depression, mania, delirium, confusion, and disorientation increases with age, but suicidal behavior and panic disorder are more common in younger people⁵⁻⁸.

Genetics

In fact, single-nucleotide polymorphisms (SNP) related to the glucocorticoid receptor (GR) have been described in patients with CIPDs, and a positive association has been found between some polymorphisms of the BCL-1 gene and the occurrence of affective symptoms during corticosteroid treatment.

Understanding these genetic influences is crucial for personalized medicine and tailoring treatments to minimize risks.

Risk Factors Related to Drug

Route of Administration

But some evidence suggests that there may be a higher risk of neuropsychiatric effects when the medication is administered intravenously (IV) compared to orally. IV methylprednisolone had a higher risk of developing neuropsychiatric effects⁹⁻¹¹.

Type of Corticosteroid

Studies have documented that patients taking prednisolone have a higher risk of developing CIPDs. However, it is important to note that this type of steroid is the most prescribed. Therefore, this is not sufficient to conclude that the use of prednisolone alone is a risk factor. prednisone, prednisolone, and methylprednisolone were most often associated with causing mania. Some evidence suggests that patients receiving intravenous methylprednisolone (IVMP) have a higher incidence rate of CIPDs, which is significantly higher than patients receiving any other corticosteroid treatment.

Clinical Evaluation

A detailed patient history and physical examination are critical for diagnosing steroid-induced psychosis.

Psychiatric History:

- Previous mental health disorders (bipolar disorder, schizophrenia, anxiety, depression).
- Family history of psychiatric conditions.
- Prior experiences with corticosteroids and psychiatric symptoms.

Mental Status Examination (MSE):

- Appearance and behavior: Agitation, restlessness, or confusion.
- Mood and affect: Signs of mania, depression, or emotional instability.
- Thought processes: Disorganized thinking, paranoia, delusions.
- Perception: Presence of hallucinations (visual or auditory).

Laboratory Tests & Metabolic Screening:

- Electrolyte imbalances (low sodium, high calcium) may contribute to psychiatric symptoms.
- C-reactive protein (CRP) and inflammatory markers can assess ongoing systemic inflammation.
- Thyroid function tests to rule out hyperthyroidism-induced psychiatric effects.
- Neuroimaging and Other Diagnostic Tools
- In some cases, additional assessments may be required to exclude other causes of psychotic symptoms¹².
- Computed Tomography (CT) or Magnetic Resonance Imaging (MRI): Used to help rule out brain lesions, tumors, or strokes that may mimic psychosis¹³⁻¹⁶.
- Positron Emission Tomography (PET) Scan: Assess brain metabolism in severe or persistent cases.
- Electroencephalogram (EEG): Identifies seizure-related causes of psychiatric symptoms.

Pathophysiology:

- Steroids alter brain chemistry, potentially increasing the risk of mood instability, hallucinations, and

cognitive impairment. Corticosteroids affect brain function through several mechanisms:

- Hypothalamo-Pituitary-Adrenal (HPA) Axis Dysfunction: Disrupts stress response and increases cortisol levels.
- Neurotransmitter Imbalance: Alters dopamine and serotonin levels, which regulate mood and perception.
- Glutamate Excitotoxicity: Excessive glutamate activity can cause cognitive and emotional instability.

Clinical Manifestations

Steroid-induced psychosis can present in various ways, from mild mood swings to severe delusions and hallucinations. Recognizing early warning signs can help prevent progression to full-blown psychosis.

Psychotic Features

In some cases, steroid-induced psychosis may involve hallucinations, delusions, or cognitive impairment, though symptom severity varies among individuals.

Hallucinations: Seeing, hearing, or feeling things that aren't real.

Delusions: Fixed, false beliefs (e.g., paranoia, grandiosity).

Cognitive Impairment: Confusion, disorganized thinking, and memory loss.

Timeline of Symptom Onset

Symptoms can appear rapidly, especially in high-dose steroid users.

Mood disturbances: Typically onset within days.

Anxiety or panic attacks: 1-2 weeks

Hallucinations and delusions: 1-4 weeks (high-dose users)

Treatment

There is no specific or clear solution for managing patients with CIPDs; therapeutic measures should be individualized according to the severity of the CIPDs and the indication for corticosteroid therapy.

Below are some useful strategies to relieve symptoms:

- Divide the dose into multiple doses per day.
- Decrease the dose.
- Switch from the IV to the oral route.

Treatment Approaches

Lowering the steroid dose is often the first approach to managing symptoms, but some individuals may require additional psychiatric support or medication.

Tapering Strategies:

- Gradual dose reduction instead of abrupt discontinuation to avoid adrenal insufficiency.
- If tapering is not an option, switching to a lower-dose or alternative anti-inflammatory medication may help.

Steroid Alternatives:

- Non-steroidal immunosuppressants (e.g., methotrexate, azathioprine, hydroxychloroquine) in autoimmune diseases.
- Biologic therapies (TNF inhibitors, monoclonal antibodies) for inflammatory conditions.
- Psychotherapy and Supportive Care
- In addition to medication, counseling and social support play a key role in helping patients recover.
- **Cognitive Behavioral Therapy (CBT):** Helps patients manage anxiety, paranoia, and mood instability.

- **Family Education and Support Groups:** Caregivers should know potential symptoms and treatment strategies.

Prevention and Monitoring

Not all patients on steroids will develop psychiatric symptoms; proactive screening and monitoring help identify high-risk individuals early.

- Risk Assessment Before Starting Steroid Therapy
- Before prescribing steroids, healthcare providers should:
- Screen for psychiatric vulnerabilities: Evaluate patients for a history of depression, anxiety, or bipolar disorder.
- Discuss potential side effects: Patients should be educated about mood and behavior changes.
- Evaluate alternative treatments: Use the lowest effective dose of steroids for the shortest duration possible.

Non-Pharmacological Approaches:¹⁷⁻¹⁹

Psychoeducation: Educating the patient and family about SIP, its causes (steroid effects on the brain), and management strategies reduces fear and improves coping.

Cognitive Behavioral Therapy (CBT): Helps individuals monitor and challenge irrational thoughts, manage distress from hallucinations, and develop coping skills.

Mindfulness & Meditation: Practices like mindfulness and prayer can help individuals ground themselves and reduce the intensity of distressing thoughts or voices.

Distraction Techniques: Engaging in activities like listening to soothing music, creative arts (drawing, writing), or physical tasks can redirect focus from psychotic symptoms.

Social & Occupational Therapy: Training in social skills and engaging in meaningful activities (like volunteering) can improve functioning and reduce isolation.

Ecotherapy & Nature Exposure: Spending time in nature can have calming effects and improve mental well-being.

Family Therapy: Involving family members to create a supportive home environment and improve communication.

Lifestyle Modifications: Regular physical activity and a balanced diet support overall brain health.

Environmental Support: Providing a calm, structured environment with soothing sensory input (like quiet music).

METHODOLOGY

The prospective observational study was carried out for a period of 6 months. The study was conducted in General Medicine department in a tertiary care hospital. A written and informed consent was obtained from the recruited patients. A Total of 85 patients were enrolled in the study.

Study Design: It was Prospective observational study.

Study Period: The Present study was conducted for a period of six months.

Study site: The Present study was conducted in oncology department in a tertiary care hospital.

Sample size: It was 85 Patients.

Inclusion criteria

- Patients with age of more than 18 years.
- Patients who are willing to give consent.
- Patients receiving treatment for steroid drug induced psychosis.

Exclusion criteria

- Patients below 18 years.
- Patients who were not willing to join in the study.
- Special population including pregnant women and lactating women.
- Psychiatric abnormalities.

Institutional ethics committee (IEC) consideration:

The research protocol was submitted to ethical committee and ethical Committee was permitted to perform the research work in the selected department of a tertiary care hospital.

Patient data collection and management:

The data collection form contains information regarding age, sex, diagnosis, past medical history, medication history, laboratory data, and diagnosis, dose and frequency of administration and duration of therapy was collected from the patients treatment chart.

Statistical analysis:

The data was represented as percentages. The P<0.05 was considered to indicate a statistically significant difference.

RESULTS AND DISCUSSION

Table 1: Age In our study 25-35 years age patients were 12 (14.11%), 36-45 years age patients were 19(22.35%), 46-55 years age patients were 33(28.82%), 56-65 years age patients were 21(24.70%).

S.No	Age	Total (N=85)	Percentage (%)
1.	25-35	12	14.11
2.	36-45	19	22.35
3.	46-55	33	28.82
4.	56-65	21	24.70
	Total	85	

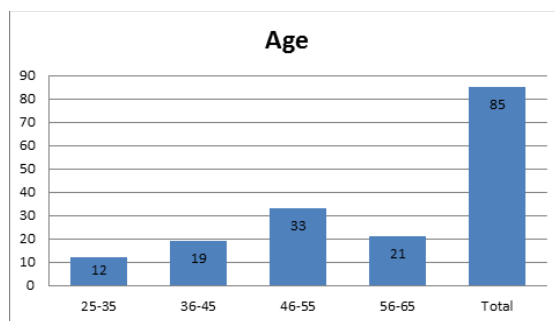


Figure 1: Age

Table 2: Gender: In our study Male patients were 51 (60%), Female patients were 34(40%).

S.No	Gender	Total (N=85)	Percentage (%)
1	Male	51	60
2	Female	34	40
	Total	85	

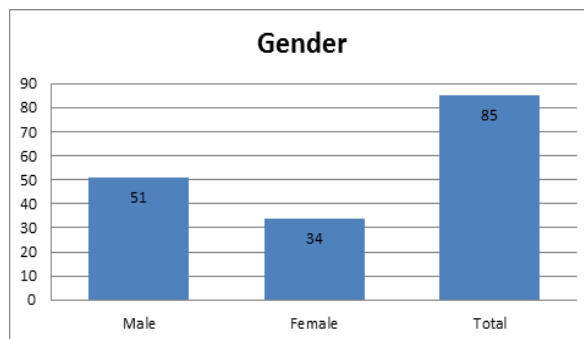


Figure 2: Gender

Table 3: Education status: In our study Literate patients were 54 (63.52%), illiterate patients were 31 (36.47%).

S.No	Education status	Total (N=85)	Percentage (%)
1	Literate	54	63.52
2	Illiterate	31	36.47
	Total	85	

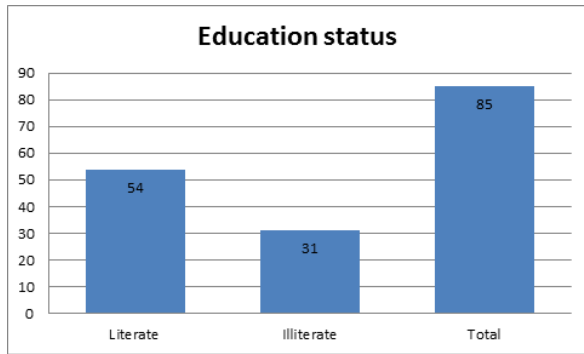


Fig 3: Education status

Table 4: Residential status: In our study rural area patients were 49 (57.64%), urban area patients were 36 (42.35%).

S.No	Residential status	Total (N=85)	Percentage (%)
1	Rural	49	57.64
2	Urban	36	42.35
	Total	85	

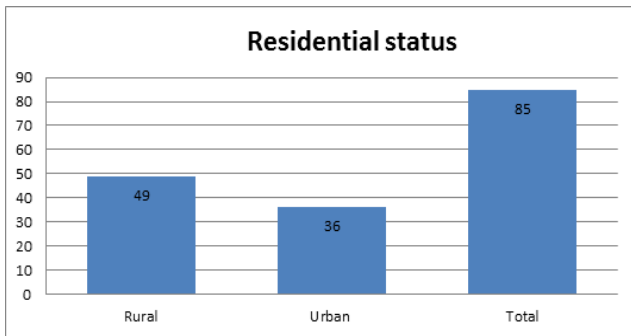


Figure 3: Residential status

Table 5: Economical status

In our study <15,000 INR monthly income patients were 14(16.47%),16,000-20,000 INR monthly income patients were 20(23.52%),21,000-25,000 INR monthly income patients were 41(48.23%),26,000-30,000 INR monthly income patients were 10(11.76%).

S.No	Economical status (Monthly)	Total (N=85)	Percentage (%)
1	<15,000 INR	14	16.47
2	16,000-20,000 INR	20	23.52
3	21,000-25,000 INR	41	48.23
4	26,000-30,000 INR	10	11.76
	Total	85	

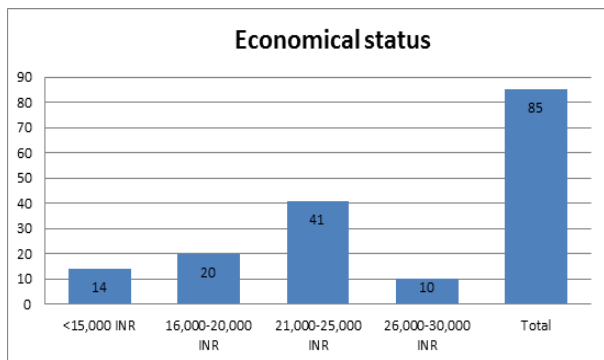


Fig 5: Economical status

Table 6: Etiological profile

S.No	Etiology	Total (N=85)	Percentage (%)
1	Prednisolone	22	26.88
2	Dexamethasone	13	15.29
3	Methylprednisolone	14	16.47
4	Past medical history of depression	15	17.64
5	Skin infections	12	14.11
6	Respiratory infections	9	10.58
	Total	85	

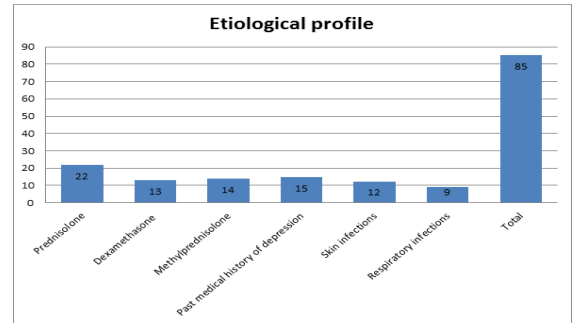


Figure 4: Etiological profile

Table 7: Drug prescriptions: In our study generic drug prescription patients were 63(74.11%), branded drug prescription patients were 22(25.88%).

S.No	Drug prescriptions	Total (N=85)	Percentage (%)
1	Generic	63	74.11
2	Branded	22	25.88
	Total	85	

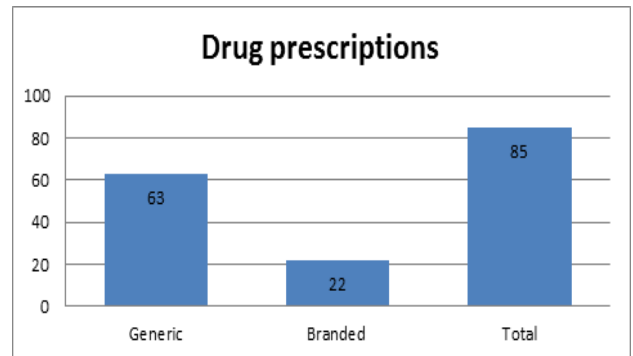


Fig 7: Drug prescriptions

Table 8: Route of drug administration

S.No	Route of drug administration	Total (N=85)	Percentage (%)
1	Oral route	56	65.88
2	Parenteral route	29	34.11
	Total	85	

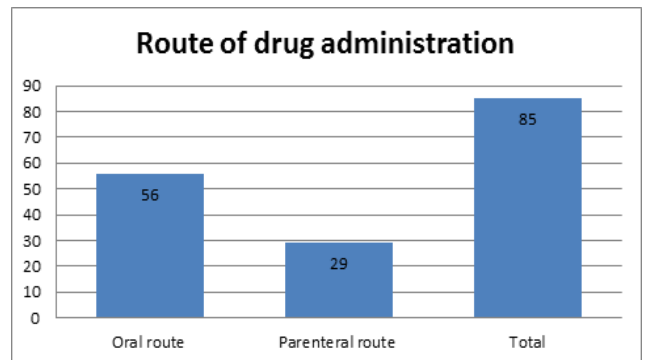


Fig 8: Route of drug administration

Table 9: Comorbidities

S.No	Comorbidities	Total (N=85)	Percentage (%)
1	Diabetes mellitus	15	17.64
2	Asthma	14	16.47
3	Hypertension	18	21.17
4	Epilepsy	21	24.70
5	Peptic ulcer	17	20
	Total	85	

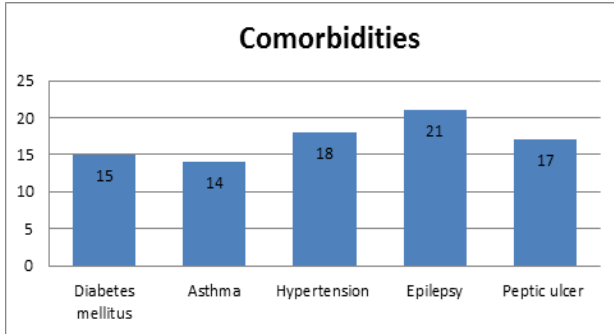


Fig 9: Comorbidities

Table 10: Clinical symptoms

S.No	Symptoms	Total (N=85)	Percentage (%)
1	Hallucination	32	37.64
2	Delusion	12	14.11
3	Confusion	13	15.29
4	Insomnia	17	20
5	Mania	11	12.94
	Total	85	

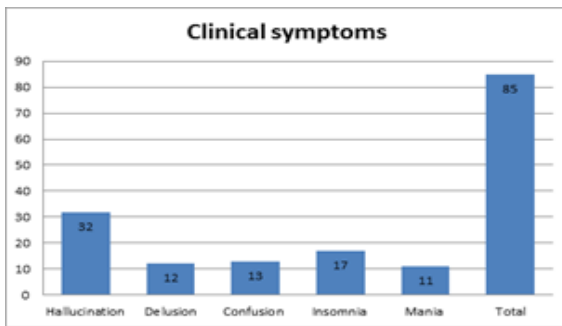


Figure 12: Clinical symptoms

Table 11: Laboratory test

S.No	Diagnosis	Total (N=85)	Percentage (%)
1	CT scan	14	16.47
2	MRI	22	25.88
3	EEG	27	31.76
4	ECG	10	11.76
5	Blood test	12	14.11
	Total	85	

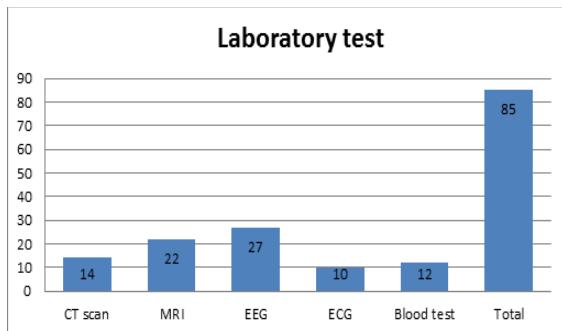


Figure 11: Laboratory test

Table 12: Management of steroid drug induced psychosis

S.No	Management	Total (N=85)	Percentage(%)
1	Psychological support	39	45.88
2	Psychological therapy	22	25.88
3	Psychological interventions	24	28.23
	Total	85	

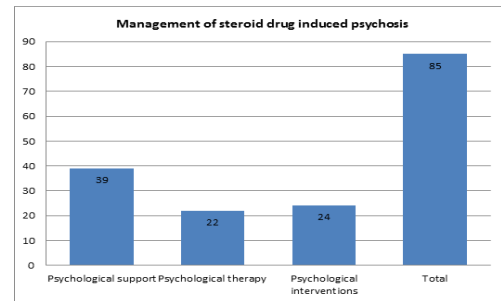


Figure 12: Management of steroid drug induced psychosis

Table 13: Treatment for steroid drug induced psychosis

S.No	Treatment	Total (N=85)	Percentage (%)
1	Lithium	25	29.41
2	Risperidone	20	23.52
3	Diazepam	13	15.29
4	Haloperidol	16	18.82
5	Olanzapine	11	12.94
	Total	85	

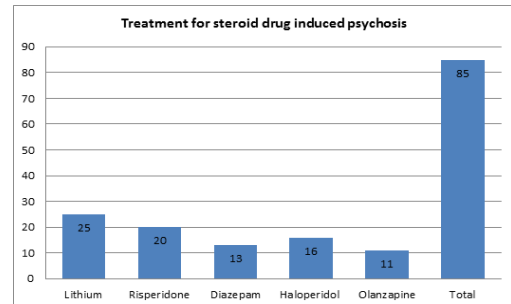


Figure 14: Treatment for steroid drug induced psychosis

DISCUSSION

- In our study 46-55 years age patients were more 33(28.82%) compared to other ages.
- In our study Male patients were more 51 (60 %), compared to Female patients were 34(40 %).
- In our study Literate patients were more 54 (63.52%), compared to illiterate patients were 31 (36.47%).
- In our study rural area patients were more 49 (57.64%), compared to urban area patients were 36 (42.35%).
- In our study 21,000-25,000 INR monthly income patients were more 41(48.23%) compared to other income categories.
- The etiological profile for steroid drug induced psychosis includes Prednisolone patients were more 22(26.88%) compared to other etiological profiles.
- In our study generic drug prescription patients were more 63(74.11%), compared to branded drug prescription patients were 22(25.88%).
- The oral route drug administration patients were more 56 (65.88%), compared to Parenteral route drug administration patients were 29(34.11%).
- The Epilepsy comorbidities patients were 21(24.70%) compare to other comorbidities.

- The Clinical symptoms for steroid drug induced psychosis include Hallucination patients were more 32(37.64%) compare to other clinical symptoms²⁰⁻²¹.
- The Laboratory test for steroid drug induced psychosis includes EEG patients were more 27(31.76%), compare to other laboratory examinations.
- The Management of steroid drug induced psychosis includes Psychological support patients were more 39(45.88%) compare to Psychological therapy patients were 22(25.88%), Psychological interventions patients were 24(28.23%)²².
- The Treatment for steroid drug induced psychosis includes Lithium prescribed patients were more 25 (29.41%) compare to other prescribed drugs²³.

CONCLUSION

In our study 46-55 years age patients were more 33(28.82%) compared to other ages. The etiological profile for steroid drug induced psychosis includes Prednisolone patients were more 22(26.88%) compared to other etiological profiles. The clinical symptoms for steroid drug induced psychosis include Hallucination patients were more 32(37.64%) compare to other clinical symptoms. The Management of steroid drug induced psychosis includes Psychological support patients were more 39(45.88%) compare to Psychological therapy patients were 22(25.88%), Psychological interventions patients were 24(28.23%). Corticosteroids, while indispensable in treating a variety of medical conditions, pose a significant risk for developing neuropsychiatric adverse effects²⁴. Steroid-induced psychosis is a severe adverse effect that can occur shortly after administering high doses of glucocorticoids. The clinicians should exercise caution while selection and prescribing corticosteroids, considering their potential neuropsychiatric side effects. Timely and appropriate evaluation is vital to minimize the future progression of risk of steroid drugs associated complications²⁵. The effective diagnosis primarily involves excluding other potential causes, while prevention primarily focuses on minimizing dosages and avoiding the unnecessary prolongation of drug treatment and lowering psychosis symptoms.

REFERENCES

[1] Busceti, C.L.; Ferese, R.; Bucci, D.; Ryskalin, L.; Gambardella, S.; Madonna, M.; Nicoletti, F.; Fornai, F. Corticosterone Upregulates Gene and Protein Expression of Catecholamine Markers in Organotypic Brainstem Cultures. *Int. J. Mol. Sci.* **2019**, *20*, 2901.

[2] Sterner, E.Y.; Kalynchuk, L.E. Behavioral and Neurobiological Consequences of Prolonged Glucocorticoid Exposure in Rats: Relevance to Depression. *Prog. Neuro- Psychopharmacol. Biol. Psychiatry* **2010**, *34*, 777–790.

[3] Sun, X.; Zu, Y.; Li, X.; Zhao, S.; Sun, X.; Li, L.; Zhang, X.; Wang, W.; Liang, Y.; Wang, W.; et al. Corticosterone-Induced Hippocampal 5-HT Responses Were Muted in Depressive-like State. *ACS Chem. Neurosci.* **2021**, *12*, 845–856.

[4] Pham, K.; Nacher, J.; Hof, P.R.; McEwen, B.S. Repeated Restraint Stress Suppresses Neurogenesis and

Induces Biphasic PSA-NCAM Expression in the Adult Rat Dentate Gyrus. *Eur. J. Neurosci.* **2003**, *17*, 879–886.

- [5] Pazini, F.L.; Cunha, M.P.; Azevedo, D.; Rosa, J.M.; Colla, A.; De Oliveira, J.; Ramos-Hryb, A.B.; Brocardo, P.S.; Gil-Mohapel, J.; Rodrigues, A.L.S. Creatine Prevents Corticosterone-Induced Reduction in Hippocampal Proliferation and Differentiation: Possible Implication for Its Antidepressant Effect. *Mol. Neurobiol.* **2017**, *54*, 6245–6260.
- [6] Brown, E.S.; Woolston, D.J.; Frol, A.B. Amygdala Volume in Patients Receiving Chronic Corticosteroid Therapy. *Biol. Psychiatry* **2008**, *63*, 705–709.
- [7] Zelleroth, S.; Stam, F.; Nylander, E.; Kjellgren, E.; Gising, J.; Larhed, M.; Grönbladh, A.; Hallberg, M. The Decanoate Esters of Nandrolone, Testosterone, and Trenbolone Induce Steroid Specific Memory Impairment and Somatic Effects in the Male Rat. *Horm. Behav.* **2024**, *161*, 105501.
- [8] Lorenzetti, V.; Allen, N.B.; Fornito, A.; Yücel, M. Structural Brain Abnormalities in Major Depressive Disorder: A Selective Review of Recent MRI Studies. *J. Affect. Disord.* **2009**, *117*, 1–17.
- [9] Yılmaz, T.; Gedikli, Ö.; Yildirim, M. Evaluation of Spatial Memory and Locomotor Activity during Hypercortisolism Induced by the Administration of Dexamethasone in Adult Male Rats. *Brain Res.* **2015**, *1595*, 43–50.
- [10] Olescowicz, G.; Sampaio, T.B.; De Paula Nascimento-Castro, C.; Brocardo, P.S.; Gil-Mohapel, J.; Rodrigues, A.L.S. Protective Effects of Agmatine Against Corticosterone-Induced Impairment on Hippocampal mTOR Signaling and Cell Death. *Neurotox. Res.* **2020**, *38*, 319–329.
- [11] Williams, D.M. Clinical Pharmacology of Corticosteroids. *Respir. Care* **2018**, *63*, 655–670.
- [12] Morita, M.; Gravel, S.-P.; Hulea, L.; Larsson, O.; Pollak, M.; St-Pierre, J.; Topisirovic, I. mTOR Coordinates Protein Synthesis, Mitochondrial Activity and Proliferation. *Cell Cycle* **2015**, *14*, 473–480.
- [13] Laplante, M.; Sabatini, D.M. mTOR Signaling in Growth Control and Disease. *Cell* **2012**, *149*, 274–293.
- [14] Kim, A.H.; Khursigara, G.; Sun, X.; Franke, T.F.; Chao, M.V. Akt Phosphorylates and Negatively Regulates Apoptosis Signal-Regulating Kinase 1. *Mol. Cell. Biol.* **2001**, *21*, 893–901.
- [15] Zhou, J.; Blundell, J.; Ogawa, S.; Kwon, C.-H.; Zhang, W.; Sinton, C.; Powell, C.M.; Parada, L.F. Pharmacological Inhibition of mTORC1 Suppresses Anatomical, Cellular, and Behavioral Abnormalities in Neural-Specific *Pten* Knock-Out Mice. *J. Neurosci* **2009**, *29*, 1773–1783.
- [16] David, D.J.; Samuels, B.A.; Rainer, Q.; Wang, J.-W.; Marsteller, D.; Mendez, I.; Drew, M.; Craig, D.A.; Guiard, B.P.; Guilloux, J.-P.; et al. Neurogenesis-Dependent and -Independent Effects of Fluoxetine in an Animal Model of Anxiety/ Depression. *Neuron* **2009**, *62*, 479–493.

- [17] Kristensen, A.S.; Andersen, J.; Jørgensen, T.N.; Sørensen, L.; Eriksen, J.; Loland, C.J.; Strømgaard, K.; Gether, U. SLC6 Neurotransmitter Transporters: Structure, Function, and Regulation. *Pharmacol. Rev.* **2011**, *63*, 585–640.
- [18] Lopes, I.S.; Oliveira, I.C.M.; Capibaribe, V.C.C.; Valentim, J.T.; Da Silva, D.M.A.; De Souza, A.G.; De Araújo, M.A.; Chaves, R.D.C.; Gutierrez, S.J.C.; Barbosa Filho, J.M.; et al. Riparin II Ameliorates Corticosterone-Induced Depressive-like Behavior in Mice: Role of Antioxidant and Neurotrophic Mechanisms. *Neurochem. Int.* **2018**, *120*, 33–42.
- [19] Rocha, M.A.N.D.; Silva, E.P.; Silva, R.N.M.; Sousa, G.R.D.; Barbosa-Filho, J.M.; Maia, M.D.S.; Lima, A.S.; De Souza-Ferrari, J.; Pereira, F.D.O. Riparin II-Type Benzamides as Novel Antibiofilm Agents against Dermatophytes: Chemical Synthesis, in Vitro, Ex Vivo and in Silico Evaluation. *J. Antimicrob. Chemother.* **2024**, *79*, 617–631.
- [20] Ge, C.; Wang, S.; Wu, X.; Lei, L. Quercetin Mitigates Depression-like Behavior via the Suppression of Neuroinflammation and Oxidative Damage in Corticosterone-Induced Mice. *J. Chem. Neuroanat.* **2023**, *132*, 102313.
- [21] Lin, L.; Herselman, M.F.; Zhou, X.-F.; Bobrovskaya, L. Effects of Corticosterone on BDNF Expression and Mood Behaviours in Mice. *Physiol. Behav.* **2022**, *247*, 113721.
- [22] Singh, M.; Agarwal, V.; Jindal, D.; Pancham, P.; Agarwal, S.; Mani, S.; Tiwari, R.K.; Das, K.; Alghamdi, B.S.; Abujamel, T.S.; et al. Recent Updates on Corticosteroid-Induced Neuropsychiatric Disorders and Theranostic Advancements through Gene Editing Tools. *Diagnostics* **2023**, *13*, 337.
- [23] Rome, H.P.; Braceland, F.J. The Psychological Response to ACTH, Cortisone, Hydrocortisone, and Related Steroid Substances. *Am. J. Psychiatry* **1952**, *108*, 641–651.

Source of Support: Nil. Conflicts of Interest: None declared.