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Uncommon Onset of Steroid-Induced Psychosis at Replacement Dose in Secondary Adrenal Insufficiency: A Case Study of Partial Empty Sella Syndrome

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Abstract: ***Steroid-induced psychosis is typically linked to high-dose glucocorticoid therapy, but in rare cases, it can emerge even with standard replacement doses, particularly in patients with chronic adrenal insufficiency. This article presents a compelling case of a 48-year-old female who developed acute psychotic symptoms after being initiated on a low-dose prednisolone regimen for secondary adrenal insufficiency associated with partial empty sella syndrome. Interestingly, her symptoms surfaced despite the dose being well below the threshold commonly reported in similar cases. In my view, this highlights a noteworthy clinical nuance: patients with long-standing glucocorticoid deficiency may exhibit heightened sensitivity to even minimal hormonal shifts. It is evident that the underlying chronic hypo-cortisolemic state may predispose such individuals to abrupt neuropsychiatric reactions when hormone levels are corrected. This case underscores the need for vigilant monitoring and early psychiatric intervention during initial glucocorticoid replacement therapy. Moreover, it suggests a broader clinical implication patients must be properly informed about potential side effects, even when doses appear physiologically benign.***

Keywords: steroid-induced psychosis, adrenal insufficiency, prednisolone, empty sella syndrome, glucocorticoid replacement

1. Case Report

48 year old Female with no preexisting comorbidities presented with history of vomiting for 15 days associated with abdominal pain and poor appetite. She also had history of cold intolerance and constipation. History of amenorrhea for the past 3 years. There was no history of postural giddiness, hyperpigmentation, head injury, visual symptoms, headache, or past history of lactation failure. On examination blood pressure was 100/70 mm of Hg, otherwise there was no other clinical finding in general and systemic examination. Key investigatory findings

TEST NAME	RESULT	NORMAL VALUE
SERUM SODIUM	116mEq/L	135-155
SERUM POTASSIUM	4.0mEq/L	3.5-5.0
URINE SODIUM	139.10mEq/L	15.00-237.00
URINE POTASSIUM	34.60mEq/L	22.00-164.00
URINE OSMOLALITY	448 mOm/kgH2O	300.00-900.00
FREE T3	1.23pg/ml	2.60-4.80
FREE T4	0.25ng/dl	0.61-1.12
FREE TSH	9.891µIU/mL	0.34-5.60
SERUM CORTISOL	3.02µg/dL	<10
FSH	20.76mIU/mL	16.74-113.59
LH	5.85mIU/mL	10.87-58.64
INJ SYNTROPAC GIVEN CORTISAL IN 30MINS	9.5µg/dL	<10
SERUM CORTISOL IN 60 MINS	8.23µg/dL	<10

With this clinical history and investigatory findings, we concluded patient is having secondary adrenal insufficiency and secondary hypothyroidism. Magnetic Resonance Imaging of Sella revealed partial empty sella. Patient was started on oral prednisolone 5mg in the morning and 2.5 mg at evening. On the 3 rd day of starting prednisolone patient developed psychotic manifestations in the form of hallucinations and irrelevant talking. Psychiatry opinion was sought for the same and patient was started on olanzapine 5mg at bedtime and a possibility of steroid induced psychosis

was kept. After a week patient psychotic feature disappeared despite continuing steroid replacement and her antipsychotic medications were tapered and stopped. Replacement for hypothyroidism started 3 days after starting prednisolone to avoid adrenal crisis.

2. Observations

Glucocorticoids cause variety of neuropsychiatric manifestations like depression, emotional liability, mania, psychosis, sleep disturbances, memory deficits, pseudotumor cerebri and movement disorders. Steroid induced psychosis usually seen at an equivalent dose of 20 mg or more of prednisolone which is usually used for immunosuppressive indications for autoimmune disorders like systemic lupus erythematosus. Common risk factors include dose of glucocorticoids (more common with doses >40 mg of prednisolone), older age, hypoalbuminemia, and history of psychotic disorders. Though the mechanism of steroid psychosis is unclear people have postulated it could be due to increase in enzyme activity of tyrosine hydroxylase which leads to raise in levels of dopamine leading to psychotic features. But development of psychosis with replacement doses of glucocorticoids of 7.5 mg of prednisolone is uncommon. In our case patient developed psychotic symptoms with 7.5 mg/day of prednisolone which is much lower than the usual dose reported in literature. The postulated cause of this phenomenon could be due to underlying chronic low basal glucocorticoid status in these patients with underlying glucocorticoid deficiency and when exposed to replacement dose there is sudden relative increase in level of glucocorticoids in comparison to pre-existing state. Similar cases of steroid psychosis during the treatment of adrenal insufficiency have been reported in literature. This case report necessitates the clinicians to monitor for development of steroid induced psychosis during first time treatment of chronic adrenal insufficiency status.

3. Conclusion

When starting glucocorticoid replacement in a patient with long standing adrenal insufficiency its necessary for the clinicians to monitor for development of psychotic features and patient must be counselled regarding this so that it can be identified and managed early.

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