

Prednisolone Induced Psychosis in an Adolescent Female with Nephrotic Syndrome- A Case Report

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I. Introduction

Over the last 50 years, exogenous corticosteroids have been used in the treatment of various medical illnesses. A number of adverse reactions have been established, including disturbance of mental state.

The average incidence of diagnosable psychiatric disorders due to steroid therapy is reported to be about 6%, but more patients suffer from mild symptoms which do not fulfill any diagnosis. Affective reactions such as depression, mania and hypomania are the most common side effects, along with psychosis, anxiety and delirium.

There is no clear mechanism model for corticosteroid-induced psychiatric disorder, but it appears to be dose-dependent. The incidence seems to be even higher at megadoses of methylprednisone.

Although it is a well-known complication in adulthood, literature about steroid-induced psychotic disorder in children and adolescents is insufficient.

II. Case Report

A previously healthy 12-year-old girl was referred to our pediatric nephrology department with a two-month history of nephrotic syndrome. She had received oral prednisone in several doses ranging from 16 mg/day to 48 mg/day during the past two months. The histopathological changes were considered as focal segmental glomerulosclerosis. At this point, high-dose methylprednisone was started on an outpatient basis. After one month she developed some behavioral changes such as fears, anxiety and sleep disturbances at home. Her parents took her to the pediatric emergency department on the second day of her complaints.

On admission, she had visual hallucinations and persecutory delusions. She claimed that her parents are not her real parents; they have been replaced by someone else. She had fears of being kidnapped and murdered. She had exhibited bizarre behavior such as praying differently and talking to herself. For the last two days, she experienced insomnia. She was oriented, aware of the time, place and the people around her. Her mother reported that she had never exhibited psychological problems in the past, and there was no family history of mental disorders.

At the time of hospitalization, her vital signs and physical and neurological examination findings were completely normal. Her weight was 38.5 kg (3-10%), height 137 cm (<3%) and blood pressure 120/70 mmHg. She was taking methylprednisone at more than 1 mg/kg per day at this presentation. All laboratory tests including complete blood count, coagulation tests, blood urea nitrogen (BUN), serum creatinine, electrolytes, liver enzymes, blood gases, and C-reactive protein/liver enzymes, blood gases, and C-reactive protein (CRP) levels were in normal limits except for hypoalbuminemia (1.6 g/dl) with proteinuria (1.8 g/m²/day). Her cranial magnetic resonance imaging (MRI) and electroencephalography (EEG) were also normal.

We considered the possibilities of delirium, drug intoxication and steroid-induced psychotic disorder in the differential diagnosis. Patients with delirium have impaired consciousness, fluctuations in their symptoms, and their EEG shows a generalized slowing activity. In drug intoxication patients have perceptual disturbances and intact reality testing. Our patient had impaired reality testing, normal cognitive functions, normal EEG, and her symptoms had begun after the initiation of mega doses of steroid. We thus diagnosed her as having steroid-induced psychotic disorder according to DSM-5 criteria. A regime of olanzapine 5 mg was initiated seven days after starting the treatment, her symptoms dramatically improved and MP were continued, to be given every other day for two weeks. Three weeks later, she had no psychiatric signs or symptoms. The only side effect was mild sedation at bedtime. Because her recent psychotic reaction had become a risk factor for further steroid therapy, we decided to continue with olanzapine 5mg as prophylaxis. At the last visit, six months after beginning olanzapine treatment, she remained free of psychiatric symptoms, despite the use of prednisone, and she was tolerating the medication well.

III. Discussion

The use of corticosteroids in various forms and doses has been known as the main therapeutic approach in childhood nephritic syndrome. Children routinely receive high-dose corticosteroid therapy; as many as 45% will have a frequently relapsing course and some will be steroid-dependent. Children with nephrotic syndrome often experience serious problems with depression, anxiety and increased aggression during high-dose prednisone therapy.

Female sex, past psychiatric history, prednisone dose of more than 40 mg/day and long-term administration are considered to be the major risk factors for steroid psychosis. In our case, three of these risk factors - female gender, over 1 g prednisone and steroid treatment for more than two years - were present.

Steroid psychosis often occurs from a few days to two weeks after administration of this agent. Although our patient had been receiving prednisone for more than two years, she had not developed any psychiatric manifestations. When she experienced psychiatric symptoms, her albumin level was 1.6 g/dl and she had significant proteinuria. The explanation for this may be that synthetic steroids bind to serum albumin, at which point they are inactive.

Therefore, higher levels of free and active fraction of steroids along with low plasma albumin levels will expose the patient to more adverse effects¹⁰. Interestingly, the incidence of psychosis in nephrologic patients is higher than other groups of patients treated with steroids.

Thus, those patients with disease causing low levels of serum proteins would be predisposed to experience more adverse effects with steroids. Our case had normal serum albumin level (3 g/dl) with normal renal function in the period without psychiatric signs/symptoms

IV. Conclusion

Referring to the described case above, we suggest that psychotic reaction should be taken into account as a possibility in all children who develop behavioral changes during corticosteroid treatment; atypical antipsychotics such as olanzapine may be considered not only as a part of the treatment but also as a prophylactic agent.

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