Steroid induced psychosis: a case report

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Abstract

Since the early 1950s, corticosteroids have been a commonly used class of medication to suppress the immune system and decrease inflammation. They have been used as an effective treatment for a wide variety of medical conditions including allergies, rheumatic diseases, gastrointestinal disorders, ophthalmic conditions, dermatological conditions, asthma, back pain, chronic obstructive pulmonary disease, systemic lupus erythematosus, and cancer. Adverse psychological side effects including psychotic symptoms have been associated with different types of corticosteroids and with various forms of use, either oral, inhaled or intravenous. These can occur at any stage of treatment including rapid discontinuation (withdrawal). The incidence of steroid-induced severe psychiatric symptoms in adults is about 5-6%, and mild to moderate reactions occurred in about 28%.(1) Other studies showed that it can be up to 60%, and recent studies show an increased rate of psychopathologies in this population. (2)

In this paper, we discuss the case of a 21 years old female patient, without any previous psychiatric history. She was admitted with psychosis a week after the administration of 60 mg/day of prednisolone for ulcerative colitis. The sudden onset of the symptoms, their quick response to the discontinuation of prednisolone and antipsychotic therapy and the complete recovery of the patient support the diagnosis of corticosteroid-induced psychosis. The patient’s medical history and the lab findings excluded other etiological factors. She was treated with olanzapine, an atypical antipsychotic, because of her distinct psychotic symptoms, and she showed significant improvement.

Key words: Steroids, psychosis, ulcerative colitis, side effect
Ms. M, is a single 21-year-old university student with no previous psychiatric history or family history. She was recently diagnosed with ulcerative colitis and started on prednisolone at a dose of 60 mg daily. Three days later, she developed poor sleep and rapid pressured speech. She was extremely anxious and fearful about her future. She was suspicious and irritable. She believed that Satan was controlling her mood and her behavior. There were repeated suicidal attempts that were prevented by her family. Psychiatry consultation was requested after one week for assistance in evaluating and managing her acute behavioral changes. She was assessed and transferred to our tertiary psychiatric Hospital in the capital. There was no history of drug or alcohol abuse. She had no known drug allergy. There was no known family history of mood or psychotic disorder. On examination, she was detached, sometimes agitated, easily distracted and talkative. Her mood was irritable without moments of euphoria. She was showing passivity-(made action) - “believed Satan was controlling her behavior”. She was partially cooperative for the physical exam which revealed an anorexic lady with BMI of 16.

Laboratory examinations including complete blood cell count with differentials, comprehensive metabolic profile and thyroid function, lipid profile, liver function, bone profile and kidney function tests were within normal limits. Her fasting blood sugar was 8mmol/l, but HBA1C was 5.7%. Serology tests for HIV, hepatitis, and syphilis, were negative. Her CT-brain and EEG were normal.

During her admission at the psychiatric hospital, prednisolone was tapered off as advised by the gastroenterologist at the regional hospital. She was asymptomatic regarding her ulcerative colitis. The patient was extremely disturbed and was given intramuscular injections of haloperidol 5mg and promethazine 25mg to calm her down. She was started on Clonazepam 0.5mg bid, Procyclidine 5mg bid and Risperidone 2mg, increased to 4mg at bed. Risperidone was then switched to Olanzapine as she developed extrapyramidal side effects (rigidity and tremor).

She was stabilized and discharged on prednisolone 10mg/day, olanzapine 10 mg/bedtime and clonazepam 0.5 mg bid when needed. Two weeks later she remained well. She denied psychotic, elated or depressed mood and suicidality. Clonazepam was discontinued, and Olanzapine was gradually decreased. In the following two months, her mood remained stable, and she resumed social activities. There were no signs of relapse while gradually discontinuing olanzapine. Six months after stopping Olanzapine there was no evidence of relapse, and she remained asymptomatic.

Since the introduction of corticosteroids in the early 1950s; they have been associated with neurobehavioral effects that are common, complex, unpredictable and often severe and sometimes potentially life-threatening. (3,4) About 10% of inpatients receive steroids, and up to 3% of the general population is on long-term glucocorticoid treatment (5). Lewis and Smith reviewed 13 studies comprising 2,555 patients who were treated with corticosteroids. They found the prevalence of severe psychiatric syndromes ranged from 1.6% to 50% (6). In another study, it is estimated that about 20% of patients who receive corticosteroids of greater than 40 mg of prednisone or its equivalent will develop a severe psychiatric disorder that will require intervention (7).

Corticosteroid induced neuropsychiatric disorders vary from mild mood disturbances to severe psychotic symptoms (1,8). “Two large meta-analyses found that severe reactions occurred in nearly 6% of patients and mild to moderate reactions occurred in about 28%” (1). Steroid-induced psychosis has been described in 1.8% to 57% of steroid-treated patients (1). The association and mechanisms of corticosteroids induced neurobehavioral problems are not clear. A widely utilized classification index, first proposed in a 1,952 case series and review, consisted of four grades of symptom severity: The first grade consists of mild euphoria, lessened fatigue while the second consists of heightened euphoria and appearing hypomanic. Various responses including anxiety, phobia, obsession, or depression are symptoms of third grade, and grossly psychotic comes under the fourth grade.

Hypomania or mania is the most common psychiatric adverse effect of corticosteroid treatment. Some studies showed that hypomania or mania is associated with short term use of corticosteroids, however, prolonged and chronic use of corticosteroid can increase risk of depression. Patients who experience corticosteroid-induced depression during one treatment course may experience drug-induced mania in a subsequent course and vice versa (7,9). Psychiatric disturbances do not appear to be associated with an underlying disease either, with the exception of systemic lupus erythematosus which increases the risk by a factor of two or more (10,11).

Psychiatric disturbances can occur at any time during corticosteroid treatment; they may occur immediately after starting and even after treatment discontinuation. However, most of the mental disturbances occur early in the therapeutic course. The majority of cases occur during the first two weeks; Smith (1983) reported about 39% of cases with onset during the first week and 62% within 2 weeks. Hall et al. (1979) found that 86% of patients developed corticosteroid-induced psychiatric disorders within 1 week of starting treatment (12,13). Bhangle et al. (2013) found 60 to 85% of patients will develop symptoms within the first week of treatment and about 90% within six weeks of initiating treatment. Symptoms usually resolve within 3-11 months after discontinuation; however, in
some cases, psychosis may persist even after tapering has been completed. (8)

Even after discontinuation of treatment corticosteroids can cause neuropsychiatric disturbance usually termed “withdrawal syndrome”. It can result in anorexia, depression, fatigue, poor concentration, depersonalization, and psychosis with additional symptoms including lability, somatic concerns such as paresthesia, faintness, poor memory and focus, depersonalization and in some cases suicide. It usually lasts about 2 to 8 weeks after stopping treatment (1, 14, 15).

Corticosteroid dose is the strongest predictor of psychiatric disorder with a reported incidence of 1.3% at < 40 mg/day (prednisone equivalents), 4.6% at 41-80 mg/day and up to 18.4% at > 80 mg/day (16). However, corticosteroid dose does not predict the onset, severity or type of psychiatric symptoms seen.

Previous corticosteroid induced psychiatric disturbances or previous history of psychiatric illness does not predict the occurrence of corticosteroid induced psychiatric disturbances (1, 17, 18). It is more common in females, but no particular age group appears to be at increased risk; however, Fardet et al. (2012) found that the risk increased with age and those with previous psychiatric history are at greater risk for cognitive impairment (19).

Currently, there are no clinical guidelines on the most efficient treatment of corticosteroid induced neuropsychiatric disorders. There are no FDA-approved medications to manage steroid induced neuropsychiatric disorders. One of the important steps to reduce the side effect of corticosteroids’ treatment is to educate patients about potential adverse effects. The first step in managing any steroid-induced psychiatric symptom is to taper or discontinue if possible (tapering as necessary to reduce the risk of hypothalamic-pituitary-adrenal axis suppression). Also, evaluate for suicidal ideation or intractable agitation and consider hospitalization if either is present. Palliative pharmacotherapy is usually indicated in case the patient cannot tolerate corticosteroid cessation or dose reduction or develops psychosis, severe agitation, aggressive behavior, or other intolerable symptom complexes. Approximately 90% of patients treated with a taper only achieved recovery. (6)

Literature has shown that use of psychotropic drugs for corticosteroid-induced psychiatric disturbances vary significantly. Previous studies reported that typical antipsychotics like chlorpromazine and haloperidol are effective agents. (20). A recent review by Kusljic et al. 2015 showed that use of risperidone, aripiprazole, olanzapine, valproate and lamotrigine in managing corticosteroid-induced psychiatric disturbances are the most active agents in controlling corticosteroid-induced psychiatric disorder. (2).

In our case, we tapered the steroid therapy slowly for almost six weeks and administered antipsychotic medications because of her marked psychotic symptoms; risperidone was started then switched to olanzapine due to side effects. Also, benzodiazepines are used in steroid psychosis especially when managing corticosteroid-induced anxiety, panic, and insomnia. (21) In our patient clonazepam was used during admission to calm her down and reduce agitation.

In conclusion, our case is one of the several case reports that document steroid-induced psychosis. Corticosteroids are beneficial in autoimmune illness but have many serious side effects. Acute psychotic episodes are one of the serious complications. The patient and family must be educated about the risks of adverse psychiatric side effects of corticosteroids. They should be discontinued if possible and psychotropic treatment started to hasten recovery.

References