CASE REPORT

Antidepressant induced Mania and Steroid Psychosis in a Patient with Bell’s Palsy

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Abstract

Steroid is commonly used for various connective tissue diseases and immunological related disorders. Psychiatric side effects are common in patient with systematic treatment of steroid. The reported prevalence ranges from 6% to 28%. Antidepressant-induced mania occurs when the mood of a patient switches to manic or hypomanic from depression after the use of antidepressant. We reported a case of a 55 year old lady, who presented with agitation and grandiosity after the treatment with antidepressant. She was initially diagnosed as having Bell’s palsy with unilateral facial muscle weakness. Oral prednisolone was prescribed for seven days where she became depressed, having auditory hallucination and delusion of guilt. She was then started on antidepressant where she became irritable, agitated and developed grandiose delusion. The antidepressant was withheld and she was started on atypical antipsychotic. Her condition improved and discharged well after three days of stay in the ward.

Keywords: Steroid, psychosis, antidepressant-induced mania

Introduction

Bell’s palsy is an idiopathic palsy of the facial nerve (VII) resulting in a usually unilateral facial weakness or paralysis. The incidence rate of this disorder is about 23 per 100,000 annually, or about 1 in 60 or 70 persons in a lifetime. The pathogenesis of the paralysis is unclear and the diagnosis is made by excluding other causes which may cause facial palsy such as infection (Ramsay Hunt Syndrome, Lyme disease, HIV, Meningitis, Polio, TB), brainstem lesions (Brainstem tumour, Stroke, Multiple sclerosis), cerebello-pontine angle lesions (Acoustic neuroma) and systemic disease (Diabetes Mellitus, Sarcoid, Guillain-Barre). Within a few days of onset of Bell’s palsy, high doses of prednisolone (0.5mg/kg/12h PO for 5 days) should be given to reduce nerve edema, hence preventing the progression of weakness to total paralysis.

Corticosteroid is a class of steroid hormone that is produced in the adrenal cortex. It involves in a wide range of physiological processes such as stress response, immune response and regulation, carbohydrate
metabolism, protein catabolism and electrolyte homeostasis. There were two major groups of corticosteroid which is the glucocorticoids group that control carbohydrate, fat and protein metabolism and anti-inflammatory; and mineralcorticoid group that control electrolyte and water homeostasis in the body\(^1\).

Synthetic pharmaceutical drugs with corticosteroid-like effect are used in a variety of conditions, ranging from brain tumors to skin disease. Dexamethasone and its derivatives are almost pure glucocorticoids, while prednisolone and its derivatives have some mineralcorticoid action in addition to the glucocorticoid effect. Synthetic corticosteroid such as prednisolone is used in the treatment of arthritis, temporal arteritis, dermatitis, allergic reactions, asthma, hepatitis, systemic lupus erythematosus, inflammatory bowel disease, sarcoidosis and Bell’s palsy\(^1\).

Corticosteroids have been used in ophthalmology for almost 50 years. Hench, in 1949, was the first to report on the beneficial effects of ACTH and cortisone on rheumatoid arthritis\(^4\). Now, prednisolone is widely prescribed for various medical conditions. However, there are a number of well-known side effects with corticosteroid. They include increase blood glucose, Cushing’s syndrome, weight gain and osteoporosis\(^5\).

In addition to physical side effects, corticosteroids often induce psychiatric syndromes. Glaser divided the steroid induced psychiatric side effects in to two categories: (a) a primary affective disorder of either elation or depression, and (b) a more complex reaction with organic reaction and psychosis\(^6\). These symptoms are collectively known as “steroid psychosis”. The reported incidence range from 6% to 28% depends on severity\(^5\). Most often, the patient who received short term corticosteroids will have hypomania and euphoria. In contrast, a long term corticosteroids therapy is related to depressive symptoms. A serious paranoid state or depression with risk of suicide can be induced, particularly in patients with a history of mental disorder, however, is not a definite predictor for occurrence\(^5\). Female has an increase risk of develop the psychiatric disturbance with the unknown reason. Most of the neuropsychiatric disturbances occur during the early corticosteroid therapy. However, these disturbances can happen any time, even after stopping the corticosteroid therapy.

**Case report**

Mrs T is a 55 years old, Chinese lady, who works as a primary school teacher. She was admitted to University Malaya Medical Centre (UMMC) with a presenting complaint of irritability, talking irrelevantly, having auditory hallucination and grandiose delusion.

Mrs T was diagnosed to have Right Bell’s Palsy ten days prior to admission. MRI showed no significant abnormality except small old infux in the right temporal lobe. She was then treated with oral prednisolone 40mg daily for seven days. She complained of poor sleep and depressed mood associated with irritability after she was started on prednisolone. She also developed delusion of guilt whereby she felt excessive burden to her family for no reasons. Later, Mrs T started to have auditory hallucination where she heard “evil” talking to her and threatening to harm her. She was seen by a psychiatrist in a private hospital and diagnosed to have depression with psychotic features. She was treated with antidepressant (Fluoxetine 20mg daily).
After taken the medication, she became very irritable and agitated. She developed grandiose delusion where she claimed that she is the Jesus Christ and the ruler of the world. She commanded people to bow and pray to her in order to save the world.

Due to her condition, she was then referred to UMMC psychiatry department. Due to her unmanageable behavior, she was given parenteral diazepam 10mg and haloperidol 5mg in the emergency department and admitted to the psychiatric ward. She was started on atypical antipsychotic (Quetiapine XR 100mg ON). Her condition settled down on the second day of admission and she was discharged well after three days in the ward. For family history, Mrs T’s elder brother is a known case of depression and was on treatment.

**Discussion**

In the nineteenth century, Emil Kraepelin coined the term “manic-depressive insanity” which initially referred to all kinds of mood disorder, including episodic depression, mania, hypomania, cyclothymic disorders and many other mood variations. In 1957, Karl Leonhard, a German psychiatrist, further split the manic depressive insanity into major depressive disorder (unipolar depression) and bipolar disorder. This conceptualization was later adopted by the DSM first edition. Young and Klerman further sub-classified Bipolar disorder into 6 subtypes, namely Bipolar I: Mania and depression, Bipolar II: Hypomania and depression, Bipolar III: Cyclothymic disorder, Bipolar IV: Hypomania or mania precipitated by antidepressant drugs, Bipolar V: Depressed patients with a family history of bipolar illness, Bipolar VI: Unipolar Mania. In DSM-IV-TR, the occurrence of manic symptoms during treatment with antidepressants was termed as substance-induced mood disorder (with manic or mixed features).

The rate of antidepressant-associated mania in unipolar depression (1% - 6%) is much lower than in bipolar disorder (20% -40%). The mechanisms of ‘switching’ from depression to mania are not completely understood. However, there are evidence revealing the involvement of central catecholamine especially dopamine and serotonin. Studies have shown that the assumption of cocaine and sleep deprivation therapy can induce manic or hypomanic episodes through dopaminergic pathway. Serotonin can mediate Methylenedioxymethamphetamine (MDMA) which produce exciting and euphorizing effects.

Some authors argued that antidepressant-induced mania occurs solely among individuals with a pre-existing susceptibility to bipolar, hence, environmental factors (such as sleep deprivation in our case) will evoke the switch process from depression to mania or hypomania, this is termed bipolar diathesis model. In contrast, some authors viewed this type of mania more as adverse events of antidepressant because it typically resolves with antidepressant cessation although time-limited symptomatic management may still be needed. This concept is adopted by DSM-IV-TR, in which antidepressant-induced manias are classified as substance-induced mood disorders rather than as a subtype of bipolar illness. Back to our case, Mrs. T was fully recovered from the manic episode after the withdrawal of the antidepressant. Therefore, her manic episode could be an iatrogenic side effect of antidepressant.

Many family-pedigree studies suggest that bipolar and unipolar probands may represent the same underlying disorder, the only
differences are the severity in between them. Blacker et al found that depressed relatives of bipolar patients had a higher threshold of having bipolar features\(^1\). Mrs. T has a brother who also has depression and has been on antidepressant for many years.

Therefore, Mrs. T may already have some genetic loading which made her vulnerable to antidepressant-induced mania.

Although antidepressant-induced mania was first described in connection with the use of tricyclic antidepressants, the safety of the newer antidepressants such as selective serotonin reuptake inhibitor (SSRI) eg fluoxetine, paroxetine and SNRI eg venlafaxine, duloxetine still remains unknown. There are evidences that tricyclic antidepressant will exacerbate the acute symptoms of agitation and psychosis on patients who have steroid-induced psychiatric disturbances but little information is available for newer antidepressants\(^1\). Some even recommend if an antidepressant is needed for the treatment of steroid-induced psychiatric disturbance, SSRI such as fluoxetine, 20mg/day is the drug of choice\(^1\). However, Mrs. Teh developed manic symptoms while she was given fluoxetine for her steroid-induced psychiatric disturbances.

Minimal information was found on the treatment of steroid-induced psychiatric disturbances, most of the management strategies were based on case reports and anecdotal evidences. Despite that, the prognosis of steroid-induced psychiatric disturbances is good with complete recovery in more than 90% of the people. Treatment of steroid-induced psychiatric disturbances should begin with dose reduction or discontinuation of the steroid drugs\(^1\). Davis et al found that neuroleptics in low doses led to rapid symptom resolution in 83% of the patients who had steroid–induced psychiatric disturbances\(^1\). Now newer atypical agents such as olanzapine which are rarely associated with dystonic reactions or extrapyramidal side effects, are proven useful in treating psychiatric symptoms during steroid therapy and are recommended as first-line-treatment\(^1\). In this case, Mrs. T had completed her seven days’ course of prednisolone and her fluoxetine were withheld. She then recovered fully from the illness after she was treated with an antipsychotic medication.

In summary, systemic corticosteroids should be prescribed with care in those who are predisposed to psychiatric reactions, including those who have previously suffered from corticosteroid-induced psychosis, or who have a personal or family history of psychiatric disorders; Antidepressants should probably be avoided as first-line treatment in persons with mood symptoms likely secondary to steroid. Atypical antipsychotics generally are safe and effective in treating steroid-induced psychiatric disturbances.

References


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