Case Report

Managing Steroid-Induced Mania after Optic Neuritis: A Case Report

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ABSTRACT

Corticosteroids are critical in the treatment of systemic disorders such as optic neuritis, emphasizing early intervention for visual preservation. While their therapeutic effectiveness is well established, the possibility of systemic adverse effects, particularly in the neuropsychiatric domain, requires consideration. We present a case of a 42-year-old man who developed steroid-induced mania after receiving therapy for optic neuritis. This case emphasizes the need for early recognizing and addressing psychological problems in such situations. We used Risperidone to treat the psychiatric symptoms. This case report highlights the need for more study into optimizing care in similar scenarios.

Keywords: VA: Visual Acuity, RE: Right Eye

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INTRODUCTION

Optic neuritis is an inflammatory disease of the optic nerve caused by infections, trauma, autoimmunity, vascular insufficiency, metastases, toxins, or nutritional deficiencies with demyelination being the most common cause.[1] Early recognition of the cause of optic neuritis is important to start appropriate treatment and preserve vision. The disease commonly presents with pain on eye movement, followed by subsequent blurring of vision. Typically, optic neuritis tends to resolve spontaneously. Approximately 10% of patients may experience the development of chronic optic neuritis characterized by a gradual and progressive loss of vision.[2] Interferon beta-1a proves to be an effective treatment choice when optic neuritis is linked to multiple sclerosis.[3] The main aim of the treatment is to reduce the inflammation, give symptomatic relief to the patient and visual recovery. Corticosteroids have emerged as a cornerstone of treatment for optic neuritis due to their potent anti-inflammatory properties, which expedite recovery, although they do not have any effect on the final visual outcome.[4]

While the therapeutic benefits of corticosteroids are well-documented, there are a spectrum of well-recognized side effects including the medical and psychiatric issues.[5] One-fifth of the individuals who get high doses of corticosteroids experience psychiatric symptoms. These symptoms have been reported to be dose-dependent and to occur more often during the first few weeks of medication. Steroid-induced psychological alterations range from moderate symptoms like anxiety, sleeplessness, or irritability to severe symptoms like mania, psychosis, and delirium, amongst these Mania and hypomania are more commonly reported.[5] Although the specific cause of steroid induced psychiatric symptoms is uncertain, the inferred mechanism is stress generated by exogenous steroids on the hypothalamic-pituitary-adrenal (HPA) axis.[6] Management that combines steroid dose reduction or removal with an antipsychotic drug are successful in treating this syndrome.

We present a case of a 42-year-old male who presented to psychiatry with mania induced by steroids following the treatment of optic neuritis. This case highlights the importance of recognizing and addressing psychiatric side effects associated with corticosteroid, emphasizing the need for further research and refined management strategies in such situations.

Case Presentation

A 42-year-old male normotensive, normoglycemic presented to ophthalmology with sudden loss of vision in his right eye. His visual acuity in the right eye was 6/60 with impaired color vision (5/24 plates on 24 Ishihara Plate Test) and decreased contrast sensitivity. A right relative afferent pupillary defect was present. His fundus examination showed circumferential optic disc swelling with healthy macula. Visual Field Testing revealed a right central scotoma. However visual acuity of the left eye was 6/6 with normal optic disc and macula.

Detailed ophthalmological and neurological evaluation was done along with relevant panel of laboratory investigations. The Blood complete picture was within normal limits with Erythrocyte sedimentation rate (ESR) of 8mm/hr and C reactive protein (CRP) of 0.2mg/dl. Liver and renal function tests, as well as serum electrolyte levels, were all within normal ranges. Lumber Puncture Protein Electrophoresis showed no oligoclonal bands. Magnetic Resonance Imaging (FLAIR) of brain was unremarkable with no periventricular plaques or gliosis. Given that all investigations yielded normal results, the patient was treated according to the guidelines for Acute Idiopathic Optic Neuritis based on his medical history and clinical features. The patient was prescribed Methylprednisolone 1gIntravenous (IV) for 3 days followed by oral prednisolone (1mg/kg/day) for 11 days, subsequently tapered over 3 days. Daily monitoring of his visual function tests (visual acuity, color vision, contrast sensitivity, pupillary reflex, fundus examination) was performed (Figure 1). The patient started showing improvement in his visual acuity on the 3rd day of starting steroids with complete recovery of visual acuity to 6/6 and color vision by the end of the treatment.

A week following the initiation of treatment with steroids he began to manifest psychiatric symptoms of insomnia and talkativeness. The patient was referred for psychiatric evaluation. Initially there was insomnia and later he had talkativeness, expansive ideas, overspending, heightened libido, and increased appetite. Patient's family denied any past psychiatric symptoms, or any history of psychiatric illness in the family. His premorbid personality traits included extroversion, ability to make and maintain long standing relationships and a good stress coping ability.

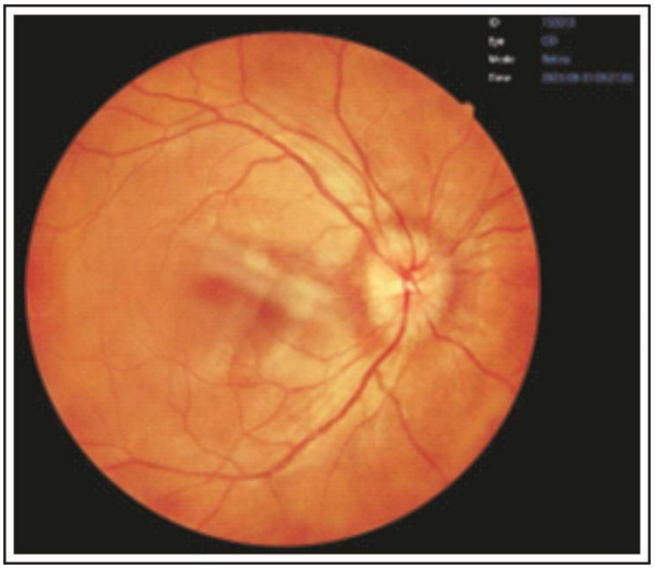

Figure 1: Inflamed disc of the right eye of the patient.

During examination, he was over talkative, overfamiliar, overinclusive and difficult to interrupt. He had a pressured speech. His mood was elated. Formal thought disorder contained circumstantiality with grandiose delusions in thought content along with ideas of reference. Notably, his orientation and cognitive functions remained intact, ruling out the possibility of delirium. However, he displayed a lack of insight regarding his psychiatric symptoms. At the time of psychiatric review, the patient's physical examination was done including a detailed neurological examination which was unremarkable. His Young Mania Rating Scale (YMRS) Score came out to be 29 (moderate mania).[7] A diagnosis of steroid induced mania was made using the diagnostic and statistical manual, DSM 5-TR.[8] Subsequently, he was initiated on a treatment regimen consisting of Risperidone (2mg twice daily) and Procyclidine (5mg once daily). Within one week of commencing this treatment, a significant improvement was observed. His mood symptoms started returning to baseline, with the resolution of formal thought disorder although

grandiose ideas persisted. This therapeutic regimen was maintained for an additional week before a gradual reduction in medications over the subsequent two weeks. The patient remained on antipsychotics for approximately four weeks before the complete discontinuation of treatment. A follow-up assessment of his YMRS score yielded a score of <7, indicative of minimal manic symptoms. He is currently undergoing fortnightly follow-up appointments to monitor his progress.

DISCUSSION

Steroids have been commonly used for the treatment of optic neuritis since long and IV administration of corticosteroids are considered standard of care in the treatment of acute optic neuritis as they speed the rate of recovery.[9]However, when used in high dose or for extended period of time it can lead to serious side effects. Apart from weight gain, mood changes, increased blood pressure and an increased risk of infection, steroids can induce psychosis. Although rare, steroid-induced mania poses a substantial therapeutic problem. The onset of manic symptoms during the therapy of optic neuritis demanded a rapid transfer from ophthalmological management to psychiatric diagnosis and intervention.

Neuropsychiatric complications associated with steroid therapy range from anxiety and sleep disruptions to severe mood symptoms such as depression, hypomania, mania, and psychotic illnesses. These symptoms usually appear within a few days after starting steroids, but they can occur at any time during or after therapy. The specific pathophysiology is unknown; however, it is most likely caused by exogenous steroid-related stress on the HPA axis.[6] This imbalance can cause neurocognitive and emotional abnormalities such as delirium, mania, and mood changes, which may be connected to elevated dopamine levels.

An essential facet of this case was the judicious initiation of antipsychotic therapy, with Risperidone. The selection of Risperidone was well-founded, considering its established efficacy in managing manic symptoms and its potential to address the neurochemical imbalances associated with corticosteroid-induced mania. There is evidence from a small number of patients that antipsychotics such as haloperidol and risperidone are beneficial in adults who have delusions or hallucinations after receiving corticosteroids.[10] In addition, the concurrent use of Procyclidine aimed to mitigate potential extrapyramidal side effects associated with antipsychotic therapy.

The subsequent clinical response to treatment was marked by the resolution of symptoms. This report highlights the importance of continued psychiatric follow-up, a structured tapering of medications, and vigilant monitoring for symptom recurrence. This patient's maintenance phase on antipsychotics for approximately four weeks allowed for a gradual reduction in medications, ultimately yielding a Young Mania Rating Scale (YMRS) score indicative of minimal manic symptoms.

CONCLUSION

This case study emphasized the importance of close monitoring during corticosteroid treatment and the importance of a multidisciplinary approach for handling psychiatric problems in the context of optic neuritis. It elucidated the critical role of Risperidone as a valuable intervention for clinicians dealing with such scenarios. Nonetheless, more research in this area is needed.

Patient's Consent: Researchers followed the guidelines set forth in the Declaration of Helsinki.

Conflict of Interest: Authors declared no conflict of interest.

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