Case Report

Dexamethasone-induced withdrawal seizure

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ABSTRACT

Dexamethasone-induced withdrawal seizure, which is a very rare and uncommon event, occurred after discontinuation of steroid therapy that was taken to increase weight by the patient. The pathogenesis is not well understood. In this submission, we have highlighted the fact that withdrawal of steroid has a propensity to cause seizure as a rare withdrawal phenomenon.

Key words: Dexamethasone, seizure, withdrawal phenomenon

INTRODUCTION

Dexamethasone is a very potent, long-acting highly selective glucocorticoid exhibiting both anti-inflammatory and immunosuppressant properties. Glucocorticoids are important mediators of the stress response, regulating both glucose homeostasis and the immune system and have wide clinical use as anti-inflammatory agents because of their profound effects on immune and inflammatory processes.[1] It binds to specific receptor proteins in the target tissues to regulate the expression of corticosteroid responsive genes, changing the levels and arrays of proteins synthesized by the various target tissues.[2] Dexamethasone is a commonly used drug used in the treatment of allergic states, dermatological diseases, endocrine diseases, hematological disorders, neoplastic diseases, nervous disorders, ophthalmic, respiratory, renal disease and rheumatic disorders. It is relatively well tolerated but has some adverse effects, particularly in high doses. Two categories of adverse effects result from the therapeutic use of corticosteroids: Those resulting from withdrawal of steroid therapy and those due to continued use at supraphysiological doses. The adverse effects from both categories are potentially life threatening. However, discontinuation of steroid therapy can present a significant clinical challenge. Treatment with supraphysiological doses of corticosteroid at levels commonly used for the treatment of inflammatory and autoimmune disorders will suppress the hypothalamic–pituitary–adrenal (HPA) axis. The most severe complication of steroid cessation is acute adrenal insufficiency characterized by lethargy, malaise, anorexia, myalgia, headache and fever.[3] It has been frequently associated with hyperglycemia, peptic ulceration, delayed healing, osteoporosis, posterior sub-capsular cataract, psychiatric disturbances and suppression of the HPA axis after prolonged use. No case of steroid-induced withdrawal generalized tonic clonic seizure (GTCS) has been reported earlier, which is very unusual and rare. We hereby report a case of dexamethasone–induced GTCS in a young boy after prolonged use.

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CASE REPORT

A 19-year-old male, educated till class 8, currently working as a tailor in a factory presented to the psychiatry outpatient department. The patient gave a history that 3 months back he had joined the gym where a friend told him that if he takes

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dexamethasone he would have better muscles and hence better looks. Therefore, he took 0.5 mg of dexamethasone tablet twice a day to increase his weight, although we do not have any written proof that he had taken the medications that he claims. He started taking these tablets as he thought he would look good on gaining weight. Although there was no preoccupation with weight or body image, he was not even spending excessive time thinking about his looks. There was no interference with his social life or his work. He took dexamethasone tablets for 2 months and had a weight gain of around 3 kg and also suffered mild gastritis, although not documented. Subsequently, he could not take the tablets for 2 days and on the second day he experienced GTCS characterized by tonic, clonic movements with up-rolling of eyes and tongue bite with loss of consciousness. There was also postictal drowsiness for 30 min. After the first seizure, he had gone to the emergency department of a nearby hospital, then dilantinzed exact details of which are not available, and he did not follow-up there and he did not even inform them about his steroid intake. Thereafter, assuming that the episode was due to missing medications, he again started taking the dexamethasone tablets, but at a dose of 0.5 mg once a day. After taking these tablets for 1 month, he again missed doses for 2 days and on the second day had a GTCS. Subsequently, he reported to the hospital outpatient department. There was no family history of seizures. There was no history of smoking, alcohol consumption or any other illicit substances abuse; there was no history of head injury or high-grade fever. The 19-year-old male patient was investigated regarding the seizure and to evaluate any adverse effect on any organ due to chronic steroid intake. Hemogram, electrolytes, blood sugar, liver function test and lipid profile were within normal limits. Contrast-enhanced computed tomography of the head and electroencephalography were normal. The patient was advised to stop the drug and was given lorazepam for 2 weeks and gradually tapered over 2 weeks; he did not develop any further seizure.

DISCUSSION

Corticosteroids exert a number of indirect effects on the central nervous system through maintenance of blood pressure, plasma glucose concentrations and electrolyte concentration. Steroid receptors are expressed in different areas of the brain, and their role is related to the regulation of various neurotransmission, including serotonin and dopamine.[3] Increasingly direct effect of corticosteroids has been recognized, including effects on mood, behavior and brain excitability. The mechanisms whereby corticosteroids affect neuronal activity are unknown, but it has been proposed that steroids produced locally in the brain (neurosteroids) may regulate neuronal excitability.[4] The discontinuation of steroid therapy can present a significant challenge. A further literature search revealed that glucocorticoid courses of less than 3 weeks duration will not lead to HPA axis suppression, no matter what the steroid dose.[5] Therefore, in patients who have received more than physiological doses of systemic corticosteroid (approximately 1 mg dexamethasone) for greater than 3 weeks, withdrawal should not be abrupt. Steroid withdrawal syndrome is understood poorly.

The case presented here is an extremely rare glucocorticoid-induced withdrawal phenomenon and has not been reported in any literature till date. We tried to rule out other causes of seizure in a 19-year-old male, e.g. substance abuse, infections, tumor, head injury, electrolyte imbalance or any other medication use. In this case, the patient had seizures on both occasions within 2 days of stopping dexamethasone, which clearly indicated that this is due to the drug withdrawal. Mostly, withdrawal symptoms are due to acute adrenal insufficiency but here the mechanism of seizure after withdrawal is unclear and still needs to be elucidated. The incidence and the onset of such symptoms are quite variable depending on several factors; in any case, all healthcare professionals should be aware of such a possibility. Furthermore, such an event should be recognized early and treated accordingly. This case report should alert physicians prescribing steroids about the potentially life-threatening withdrawal phenomenon.

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