Addison’s Disease presenting with Muscle Spasm

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Abstract
Primary hypoadrenalism has various causes and protean manifestation. We report a young female patient who presented with severe muscle spasm as her primary complaint. On evaluation she was found to be a case of Addison’s disease secondary to adrenal tuberculosis. Her muscle spasm disappeared rapidly with replacement dose of glucocorticoid.

Introduction
Severe muscle spasm solely due to adrenal insufficiency is a very rare entity. We discuss a case of a young Indian female who presented with severe muscle spasm which was corrected on correcting the adrenal insufficiency.

Case Report
A 35 yr old female patient presented to us in our out patient department with inability to extend her hip joint for the past one and a half months. This deformity in the hip was progressive and any attempt to extend the hip was painful. As a result she was bed ridden for the past couple of weeks. The patient also complained of nausea, generalised fatigue and recent loss of weight. She denied any history of fever, cough, and back pain. There was no history of any contact with tuberculosis. She does not suffer from diabetes mellitus, hypertension and didn’t have any drug for a prolonged period. She had undergone myomectomy for uterine fibroids 6 months back without any per operative or post operative complications.

On examination the patient was afebrile. Her blood pressure was 90/60mm of Hg and pulse rate was 84/min. She had hyperpigmentation of face, hands, elbows and feet and also of the oral mucous membranes (Figure 1). It was not possible to extend the hip joints to beyond 90 degrees (Figure 2). The examination of all systems and that of the spine was normal.

Complete haemogram revealed Hb of 11g%, WBC count of 9,100/cumm with a differential of N 68, L 22, E 10 and a platelet count of 1.5 lac/cu mm. Her fasting glucose value was 89 g/dl and urea, creatinine values were within normal limits (38 mg/dl and 0.9 mg/dl respectively). Her serum sodium was 131 meq/L and serum potassium was 5.1 meq/L. HIV ELISA was negative.

Her serum cortisol level at 8.00am was < 1 mcg/dl and failed to rise significantly after administration of Synacthen 250 mcg. X Ray of the hip joints and spine were unremarkable. Ultrasonography of the abdomen revealed a hypoechoic space occupying lesion in the right adrenal gland measuring 2.6 cm X 5 cm. There was no abnormality in the psoas muscle. MRI revealed an irregular mass in right suprarenal region (Figure 3). It showed no abnormality in the hip joints or lumbo-sacral spine. FNAC was taken from the lesion and smears showed acid fast bacilli on Ziehl Nielsen staining. Her serum prolactin, FSH and LH were within normal limits. Her TSH levels were 4.29 mIU/ml and T4 and T3 were 1.5 ng/ml and 3.53 pg/ml respectively. Her serum CPK was 50 IU/L and SGOT and LDH levels were 24 U/L and 60 u/l respectively. EMG was normal.

The patient was started on replacement dose of prednisolone i.e. 7.5 mg/day immediately after the serum cortisol results. Significant improvement of the spasm was seen within 24 hours and within 48 hours patient started walking around with near complete improvement in the spasm. We did not add fludocortisone because of poor affordability and subsequent normalisation of blood pressure and potassium levels. A few days later the FNAC results arrived and she was started on anti-tubercular drugs and the dose of steroids was stepped up keeping in mind excess metabolism by rifampicin. Other symptoms like nausea and generalised fatigue also improved gradually. The patient was discharged in a stable condition and is in our follow up now. She continues to take steroids and anti-tubercular drugs and has had no further complaints.

Discussion
Primary adrenocortical insufficiency occurs as a result of destruction of more than 90% of adrenal cortex leading to a deficiency of both cortisol and aldosterone. Autoimmune disease is the most important cause of primary adrenocortical insufficiency worldwide (in up to 80% of the cases†). But in India Tuberculosis remains to be the most important cause of Addison’s disease. One case series from south India (in the group with tuberculosis compared with the normal population. But both studies show a decreased response ACTH stimulation in the patients of tuberculosis and a significant improvement in response to ACTH was seen after treatment with antitubercular drugs by the latter. The usual symptoms are weakness, nausea, vomiting, generalised pigmentation and gastrointestinal disturbances. There may also be severe hypotension, unconsciousness, and severe hypoglycaemia in times of stress, a condition known as addisonian crisis. Reversible flexion contractures of the thigh consequent to adrenal or pituitary damage have been rarely
Flexion contractures have preceded the diagnosis of adrenal insufficiency, with time intervals ranging from 3 months to 5 years. A few patients have come to medical attention due to bizarre painful muscle spasms, more commonly involving trunk and lower extremities, which are triggered by touching or manipulating the limb. Glucocorticoid therapy relieves these symptoms but mineralocorticoid therapy is found to be ineffective.

The rapid relief of the muscle spasm in the discussed patient following the use of replacement dose of steroid and in the absence of any anti inflammatory or muscle relaxant drugs strongly suggests the adrenal insufficiency as the cause of muscle spasm in our patient. Psoas abscess, another important cause of muscle spasm, was excluded by imaging studies.

References