

Steroid Induced Myopathy- Men In Barrell Presentation: A Case Report

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ABSTRACT

Development of proximal muscle weakness and atrophy in association with cushingoid features is a complication of chronic corticosteroid administration. Cases of acute steroid myopathy have been described in literature but were associated with use of high dose intravenous steroid especially in ICU setting. Here we are presenting a case of 15 year old male who developed acute onset proximal weakness of both upper limb (men in barrel presentation) following short course of oral prednisolone.

Keywords: Steroid Induced Myopathy, Men In Barrell, muscle weakness.

INTRODUCTION

Corticosteroids were introduced for therapeutic use in 1948, and in 1958, Dubois reported the first patient with myopathy resulting from iatrogenic corticosteroids.¹ Steroid myopathy is usually an insidiously progressing disease that causes weakness mainly to the proximal muscles of the upper and lower limbs and also to the neck flexors. Cushing originally described this in 1932, and Muller and Kugelberg first studied it systemically in the year 1959.^{2,3} Since corticosteroid therapy's introduction into clinical practice, both acute and chronic steroid myopathies have been well recognized. Earlier case reports of acute steroid myopathy (ASM) usually involved patients with asthma who were receiving high-dose IV corticosteroids for status asthmaticus.⁴ The acute form, also known as critical illness myopathy, presents as acute quadriplegia and usually occurs in ICU patients who receive high-dose IV corticosteroids and/or non-depolarising neuromuscular blocking agents to facilitate mechanical ventilation. However, Geeta A Khwaja in 2009 reported a case of acute myopathy following short course low-dose oral steroid therapy in adult patient.⁵ Here we are describing a case of acute steroid induced myopathy, in which the patient developed bilateral upper limb proximal muscle weakness following short course oral-steroids.

CASE PRESENTATION

A 15 year old male patient presented with 20 days history of proximal upper limb weakness. He noticed difficulty in combing hair, dressing and undressing himself especially for upper wear. There was no weakness in lower limb nor any history of bulbar involvement. There was no diurnal fluctuation of weakness.

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Patient was giving history of fever with rash 1 month back. He was prescribed antibiotics and prednisolone 20 mg from a local practitioner since past one month.

On examination at presentation, significant findings were puffy looking face with an otherwise undernourished body. On motor examination, power was reduced in bilateral upper limbs proximally (3/5) without any weakness of lower limbs. Higher mental functions and cranial nerves were intact. Rest of the systemic examination was normal.

Routine investigation including complete blood count, kidney function tests and serum electrolytes were normal. Liver function tests revealed mildly elevated hepatic enzymes (SGOT = 230mg/dL, SGPT = 113mg/dL). Serum CPK was 24 U/L ((reference range, 22-198 U/L). The viral markers for HBsAg, HCV and HIV were also negative. Chest X-ray was also normal. EMG done was showing mixed small and normal amplitude MUAP's with early complete interference pattern. Oral steroids were stopped and patient was advised for high protein diet, vit E and vit D supplementation along with physiotherapy. Gradually over the period of month weakness completely recovered.

DISCUSSION

Myopathy has been recognised as a dreadful side-effect of glucocorticoid administration since its introduction as a therapeutic agent. Initially, Cushing described it in 1932. And it was then Muller and Kugelberg who first studied it systemically in 1959.⁶ It occurs more commonly with chronic usage of fluorinated steroids (such as dexamethasone, betamethasone, and triamcinolone) as

compared to non-fluorinated ones (such as prednisolone or hydrocortisone).⁷

In literature, two distinct clinical patterns of steroid induced myopathy have been described— chronic and acute.⁸ The chronic form of steroid myopathy is more common, also called the classical form. It is insidious in onset and usually occurs after prolonged administration of oral corticosteroids.⁸ In addition to this, administration of prednisolone in doses > 10 mg/day (or its equivalent) has also been reported to predispose patients to muscle injury.⁹⁻¹¹

The acute form of steroid myopathy is usually uncommon. It is seen to occur in ICU patients receiving high dose IV corticosteroids with or without non-depolarising neuromuscular blocking agents, to facilitate mechanical ventilation. It can, however, also occur with high-dose glucocorticoid use alone.¹²⁻¹⁶ Also being referred to as acute quadriplegic myopathy, acute illness myopathy, critical illness myopathy, and myopathy associated with thick filaments. It may also develop in critically ill patients with sepsis, multi-organ failure, or transplant recipients receiving high-doses of IV corticosteroids during the perioperative phase.¹⁹⁻²¹ Clinical presentation is that of a limb girdle syndrome. Neck flexor and proximal muscle weakness is more pronounced than distal muscle weakness. Proximal muscle weakness of the lower and upper extremities is significantly related to the cumulative dose of steroid. Muscle bulk typically is normal, but muscle atrophy can occur.

It is interesting to note, myopathy following a single oral dose of prednisolone (40 mg) and acute myopathy following administration of a single low-dose of parenteral triamcinolone, though uncommon, has also been reported.^{6,17} The exact mechanism of the muscle weakness is unclear but may be related to decreased protein synthesis, increased protein degradation, hypokalaemia, and/or decreased sarcolemmal excitability.¹⁸

CONCLUSION

From our specific case, we shed light on the uncommon presentation of short-term oral steroids causing acute myopathy and presenting clinically as a limb-girdle syndrome rather than acute quadriplegic myopathy i.e. men in barrel phenotype. It is therefore imperative that a possibility of steroid myopathy be kept in mind for all patients who develop acute muscle weakness after starting oral or parenteral steroids. Early recognition of the problem and timely drug withdrawal carries a good prognosis for recovery.

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