Steroid-Induced Myopathy in an Elderly

Uzma Tazeen[1], Primrose Wardhana[2]

[1] *Foundation Year, Hull and East Yorkshire Hospitals, United Kingdom*
[2]*Consultant Geriatric Medicine, Hull and East Yorkshire Hospitals, United Kingdom*

Conflict of Interest: None declared

This article has been peer reviewed.

Article Submitted on: 5th April 2012

Article Accepted on: 3rd July 2012

Funding sources: None declared

Correspondence to Dr Uzma Tazeen

Address: Hull and East Yorkshire Hospitals, United Kingdom

Email: dransaa@yahoo.com

Cite this article: Tazeen U, Wardhana P. Steroid-induced myopathy in an elderly. J Pak Med Stud.2012;2 (4): 144-145

Dear Editor,

Adverse drug reactions may represent up to 5% of all hospital admissions and account for around 5% of all in-patient mortalities [1]. Women are twice more likely to be affected than men [2]. Long term use of corticosteroids can have adverse consequences, which may include myopathy. This is a case report of an eighty-year-old man with known history of chronic obstructive pulmonary disease (COPD), managed with seretide and tiotropium with as required salbutamol inhaler with on and off oral steroids, presenting with proximal muscle weakness and difficulty weight bearing that progressively worsened in the last six months prior to presentation. He had a course of rehabilitation and showed marked improvement in his physical status on withdrawal of steroids.

An eighty-year-old man was admitted with gradually worsening mobility and recurrent falls for two years, he experienced difficulty in standing without assistance that progressively worsened over the last six weeks prior to presentation. His past medical history was significant for COPD for which he was on salbutamol, symbicort, tiotropium inhalers and intermittent prescription of prednisolone 30mg. On examination, he had an unsteady gait ranking 4 on the modified ranking scale with marked wasting of the quadriceps, and power in both his limbs were 4/5 with an unremarkable systemic examination. His creatinine kinase levels were high with a value of 2126 U/L (normal 24-195 U/L) with associated worsening of his renal function, urea 14 mmol/L (normal 2.1-7.6 mmol/L) creatinine 188 mmol/L (normal 56-127 mmol/L). Autoimmune antibody screen, thyroid function test, anti voltage gated channels, GM1, GQ1b, GAD antibodies and electromyography (EMG) performed were negative. Nerve conduction studies (NCS) demonstrated mild sensory degenerative polyneuropathy and possibly dorsal column lesion. Computed tomography (CT) scan of chest, abdomen was essentially normal. Magnetic resonance imaging (MRI) showed spondylolytic spondylolisthesis of L5 on S1 with impingement of bulging disc on L5 nerve root within its exit foramen. Muscle biopsy was resorted which demonstrated myelinated nerve fibers and mild myopathic changes, electron microscopy showed evidence of fiber atrophy with several tubuvesicular structure but no nuclear filamentous inclusions seen with no selective type 2 fiber atrophy. A diagnosis of steroid-induced myopathy was considered and steroids were tapered. Patient was given continuous input from physiotherapy and was discharged in around six weeks with improved mobility.

Steroids may cause myopathy due to decreased synthesis or increased protein degradation, modulting the carbohydrate metabolism, electrolyte disturbances, and decreased sarcolemmal excitability, although the exact mechanism remains unclear. Excessive steroids can also result secondary to tumors such as those of adrenal origin or iatrogenic [3-5]. Muscle biopsies are not required for the diagnosis of steroid-induced myopathy but in cases where they are performed show diffuse involvement and predilection for type II fibers in acute presentations [3]. Steroid-induced myopathy is frequently related with the use of fluorinated steroids, as compared to nonfluorinated steroids [2]. Corticosteroids may cause weakness of both skeletal and respiratory muscles usually in patients with COPD [7]. Glucocorticoid therapy may lead to increased apoptosis if it interferes with the signalling of insulin-like growth factor-I (IGF-1) [7]. Glucocorticoids influence muscle differentiation and degradation by enhancing the effect of ubiquitination on the MyoD which is an essential transcriptional switch for muscle development and regeneration [8].

Myopathy due to steroids can be acute or chronic [3] and in some instances may involve the respiratory muscles [9]. The chronic form occurs after prolonged use with a more insidious onset whereas the acute presentation, if present, may be associated with rhabdomyolysis and abrupt onset [9]. Myopathy secondary to steroid may predispose patients to osteoporosis and sedentary life style which exposes to risks of contractures, deep venous thrombosis (DVT) and pressure sores. Stopping the administration of steroids results in insidious response; however, complete recovery may take weeks to months [11]. The laboratory findings are generally non specific[11,

12]. Creatinine kinase (CK) and lactate dehydrogenase (LDH) levels may be within normal limits; however, before development of myopathy the urinary creatinine excretion may specifically rise [3]. Acute myopathy shows increased CK levels with myoglobinuria which is absent in cases of chronic use of steroids [9]. EMG and NCS remains normal in chronic phases but some case reports of acute onset have indicated abnormal spontaneous activity (positive sharp waves and fibrillation potentials) [9]. There is no definite treatment for reversing steroid-induced myopathy; however, stopping the administration of steroids or, in cases where it is essential to be given, administer low doses on alternate days or offering non-fluorinated forms may minimize the risk with improvement in weeks to months. Our patient sufficently showed improvement on withdrawal of steroids and had been ambulatory in the ward three to four weeks after appropriate dose reduction.

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