



# Acute Steroid Induced Myopathy after Single IM Dose of Steroid

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## Abstract

**Introduction:** AMS after a single IM dose of dexamethasone is very rare. And till now no one has reported similar case, on the other side the chronic myopathy induced by high IV corticosteroids is not unusual.

**Case presentation:** A 3 years old female had acute myopathy with rhabdomyolysis after a single dose of steroid with rapid improvement after discontinued the medication

**Conclusion:** In spite of that our case is rare; the physician should pay attention while using steroids, especially when clinical situation and laboratory tests are highly suspicious.

**Abbreviations:** ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; ASM: Acute Steroid Myopathy; AST: Aspartate Aminotransferase; CPK: Creatinine Phosphokinase; CRP: C-Reactive Protein; EMG: Electromyography; ESR: Erythrocyte Sedimentation Rate; IV: Intravenous

## Introduction

Steroid myopathy is usually an insidious disease process that causes weakness mainly to the proximal muscles of the upper and lower limbs and to the neck flexors. Cushing originally described it in 1932, and Muller and Kugelberg first studied it systemically in 1959. An excess of either endogenous or exogenous corticosteroids is believed to cause the condition. Excess of either endogenous or exogenous corticosteroids is believed to cause the condition [1,2].

Corticosteroids were introduced into clinical practice in 1948, and in 1958, Dubois [3] reported the first patient with myopathy resulting from iatrogenic corticosteroids. Since corticosteroid therapy's introduction into clinical practice, both acute and chronic steroid myopathies have been well recognized.

Chronic steroid myopathy is more common and develops after prolonged usage of steroids [3,4]. Acute steroid myopathy (ASM) is less common and develops early in the course of treatment, typically with high-dose intravenous (IV) steroids [4].

Earlier case reports of ASM usually involved patients with asthma receiving high-dose IV corticosteroids for status asthmaticus [5]. Geeta A Khwaja also reported on 2009 a case of Acute Myopathy Following Short-term Low-dose Oral Steroid Therapy in adult patient [6].

Acute myopathy developing from intramuscular corticosteroid has not been often reported. No case was found yet that described

a pediatric patient developing myopathy after a single dose of intramuscular corticosteroid therapy.

## Case presentation

A 3 years old, female known case of bronchial asthma step 1, was in usual healthy state till 3days back when she had upper tract infection which induced acute asthmatic exacerbation, this episode was treated by nebulized albuterol (ventolin) and one dose of IM dexamethasone (0.5 mg /kg /dose), respiratory symptoms improved but after 24 hours. The patient had generalized muscle weakness and myalgia with no skin rash or joint problem, after that her urine became dark.

Her mother sought medical advice in our ER, and she mentioned 2 similar attacks after steroid injections but without urine color changing.

- On examination, the patient looked ill with stable vital signs, no skin rash
- Deep tendon reflexes were normal, mild decreased muscle power and tenderness
- She was admitted for further investigation, where laboratory tests revealed
- ESR=11 mm/h, high liver and muscle enzymes CK =3200 LDH=404 AST=242 ALT=41 ALP=109

- Other serum biochemistry levels and renal function were normal
- Urine dipstick showed 4+ blood, urine analysis confirmed presence of myoglobin.
- Rhabdomyolysis was suspected and treated by adequate hydration and urine alkalinizing.
- The patient clinically improved and urine became clear
- Rheumatologist excluded connective tissue disease, including dermatomyositis.

Depending on acute course and rapid recovery neurologists diagnosed this case as rhabdomyolysis resulting from acute steroid myopathy.

After 4 days of admission, she was discharged with follow up as an outpatient.

EMG was done as outpatient and was normal; unfortunately the nerve conduction study was not done.

The following Table 1 is showing muscle and liver enzymes during admission.

**Table 1:** Table showing muscle and liver enzymes during admission.

	1 <sup>st</sup> Day of Admission	2 <sup>nd</sup> Day of Admission	3 <sup>rd</sup> Day of Admission
CK	3200	9636	355
LDH	404	1475	3394

After 2 weeks: The patient was doing well with normal physical examination and LDH =200 CK=250 ALT=44 AST= 53

**Discussion**

Steroid myopathy may be more frequent with the use of fluorinated steroids, such as dexamethasone or triamcinolone, than with nonfluorinated ones, such as prednisone or hydrocortisone. [5, 6] Although the exact mechanism of the muscle pathology is unclear, it may be related to decreased protein synthesis, increased protein degradation, alterations in carbohydrate metabolism, mitochondrial alterations, electrolyte disturbances, and/or decreased sarcolemma excitability [1].

Most of studies have shown that myoglobinuria secondary to drugs, when considered in the pediatrics population, occur more so in the second decade of life, our case alluded to the fact that it can occur in younger age groups as well [7].

Hypokalemia can induce rhabdomyolysis but in our patient, hypokalemia, as secondary to perhaps B2 agonist, or corticosteroids, could not have contributed to the development of myoglobinuria, as the patient’s biochemistry profile was normal [8].

The acute form of steroid myopathy is uncommon. It usually occurs in ICU patients who receive high dose IV corticosteroids and/or nondepolarising neuromuscular blocking agents to facilitate mechanical ventilation, but can occur with high-dose glucocorticoid use alone [6].

Acute, generalized weakness, including weakness of the respiratory muscles, typically occurs 5-7 days after the onset of treatment with high-dose corticosteroids.

Generalized muscle weakness, not limited to a more proximal distribution is noted.

Muscle stretch reflexes typically are normal and sensory examination should be normal [1]. Though most cases in the literature report a lengthy prolonged recovery phase sometimes taking 3-12 months for full recovery, our patient exhibited a rapid recovery..... one such speed recovery was reported.

In acute steroid myopathy, most patients have high levels of serum creatine kinase (CK). AST, ALT, as well as associated myoglobinuria.

Our patient, however fortunately did not develop ARF, as the serum urea nitrogen and serum creatinine remained within normal limits. It is recommended that a low threshold of clinical suspicion be employed; and serum CK as well as urine dipstick and microscopy to detect myoglobinuria should be obtained for patients in whom rhabdomyolysis may be possible [9]

Muscle biopsy was not done and as mentioned in other cases in adults is not diagnostically helpful EMG was normal in our case.

Interestingly, gender also seems to be a risk, as women are twice as likely as men to develop muscle weakness [10]... of note. Our patient was also a female, hence, there was a gender predilection for the likelihood of developing drug induced acute rhabdomyolysis.

Treatment of ASM is aiming to discontinue the rhabdomyolysis and prevent developing renal failure. No definitive treatment was found in literature except only to stop giving steroid, in addition to that weakness seen with steroid myopathy typically resolves after the corticosteroid dose is reduced or discontinued.

This case revealed clearly that even single IM dose of steroid can cause ASM.

**Conclusion**

We think that steroids still a cornerstone of treatment for most areas of Medicine, and it is unwise to abandon this drug, and despite the rarity of this case, our clinical threshold should be low to discover it, even after single dose.

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