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## Corticosteroid-Induced Myopathy

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Updated: Dec 4, 2008

### Introduction

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#### Background

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 endogenous corticosteroid production can arise from adrenal tumors. An excess of exogenous corticosteroid can result from steroid treatment for asthma, chronic obstructive pulmonary disease, and inflammatory processes, such as polymyositis, connective tissue disorders, and rheumatoid arthritis.<sup>1, 2, 3</sup>

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#### Pathophysiology

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.<sup>4, 5</sup> 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 sarcolemmal excitability. Sedentary lifestyle may increase the risk of muscle weakness in a patient taking corticosteroids, since corticosteroids seem to affect less active muscles preferentially. Two distinct types of steroid myopathy exist, acute and chronic. The chronic (or classic) form occurs after prolonged use of corticosteroids and has a more insidious course. The acute form is less common, is associated with rhabdomyolysis, and occurs abruptly while the patient is receiving high-dose corticosteroids.

#### Frequency

##### United States

The exact incidence of steroid myopathy is unknown; sensitivity to particular medications varies among patients.

#### Mortality/Morbidity

The weakness seen with steroid myopathy typically resolves after the corticosteroid dose is reduced or discontinued, although recovery can take weeks or months. Case studies have reported a lack of full recovery, as well as difficulty weaning patients off of mechanical ventilation. Osteoporosis, which can occur as a comorbidity with steroid myopathy, can result from the corticosteroid or from decreased mobility and respiratory impairment.<sup>6</sup> Other comorbidities include joint contractures, pressure ulcers, and deep vein thrombi, although these can occur in any condition causing weakness and immobility. Mortality has not been described. Some case studies have reported patient mortalities, but they provided no indication that steroid myopathy was the cause.

#### Sex

For a given dose of steroid, women appear to be twice as likely as men to develop muscle weakness, although the reason is unclear.

## Clinical

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### History

- Chronic (classic) steroid myopathy
  - This form is the classic presentation of steroid myopathy.
  - This condition can develop after prolonged administration of prednisone at a dose of 40-60 mg/d.<sup>4,5</sup> Although there is no clear length of time, onset of weakness has been found to occur within weeks to years following initiation of corticosteroid administration.
  - Several studies have suggested that the risk for steroid-induced myopathy is greater in severely asthmatic patients who use oral steroids.<sup>7</sup> One study, however, found no significant difference in the prevalence of myopathy in oral steroid users and inhaled steroid users.
  - Fluorinated steroids seem to produce weakness and myopathy more frequently than do nonfluorinated ones.
  - The insidious onset of proximal muscle weakness of the upper and lower limbs is a prominent clinical feature.
  - Progressive proximal muscle weakness of the upper and lower limbs is reported.
  - Patients typically complain of a progressive inability to rise from chairs, climb stairs, and perform overhead activities.
  - Patients initially note little difficulty with hand strength.
  - The facial and sphincter muscles usually are spared.
  - Myalgias can become a prominent feature with time.
  - Contrary to previous beliefs, several studies have shown involvement of the respiratory muscles (eg, the diaphragm); thus, pulmonary symptoms may be present.<sup>6</sup>
- Acute steroid myopathy
  - This form is encountered less frequently than is the chronic type.
  - Acute, generalized weakness, including weakness of the respiratory muscles, typically occurs 5-7 days after the onset of treatment with high-dose corticosteroids.<sup>6</sup> Some case reports describe the development of muscle weakness after the administration of a single dose of corticosteroid.
  - One study indicates a possible correlation between the occurrence of acute steroid myopathy and the total dose of steroid administered; acute atrophy was encountered with total doses of greater than 5.4 g of hydrocortisone in 6 days, whereas no signs of myopathy were noted with total doses of less than 4 g.
  - Previous systemic corticosteroid use does not appear to contribute to the development of myopathy.

### Physical

- Chronic (classic) steroid myopathy
  - Proximal muscle weakness is more pronounced than is distal muscle weakness; however, severe relative weakness of the anterior tibialis muscle can be found.
  - Pelvic girdle muscles usually are affected more severely and earlier than are pectoral girdle muscles.
  - Muscle bulk typically is normal, but muscle atrophy can occur.
  - Muscle stretch reflexes typically are normal.
  - Sensory examination should be normal.
- Acute steroid myopathy

- Generalized muscle weakness, not limited to a more proximal distribution, is noted.
- Muscle stretch reflexes typically are normal.
- Sensory examination should be normal.

## Differential Diagnoses

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Myasthenia Gravis

### Other Problems to Be Considered

#### Myopathies

Inflammatory myopathies (eg, polymyositis/dermatomyositis)

Muscular dystrophies

Drug/toxin-induced myopathies

#### Neuropathies

Diabetic amyotrophy

Motor neuron disease

Critical illness neuropathy

#### Neuromuscular junction disease

Eaton-Lambert syndrome

## Workup

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### Laboratory Studies

- Chronic (classic) steroid myopathy
  - Serum levels of creatine kinase typically are within the reference range.
  - Creatinine excretion in the urine increases dramatically and can precede the clinical appearance of myopathy by several days.<sup>1</sup>
  - Myoglobinuria and rhabdomyolysis are absent.
- Acute steroid myopathy - In most cases, high levels of serum creatine kinase are found, as well as associated myoglobinuria.

### Other Tests

- Muscle biopsy in chronic (classic) steroid myopathy<sup>8</sup>
  - Muscle biopsy shows preferential atrophy of type II fibers, particularly the fast-twitch glycolytic fibers (type IIB).<sup>6, 9</sup>
  - Some atrophy of other type II fibers and, to a small degree, type I muscle fibers can occur.
  - Increased variation in the diameter of muscle fibers occurs.
  - A lack of evidence of muscle fiber inflammation is reported.
  - There is a distinct lack of necrosis or regeneration of muscle.
  - Less active muscles appear to be affected preferentially.
- Muscle biopsy in acute steroid myopathy - Muscle biopsy shows focal and diffuse necrosis of all fiber types,

without predilection for type II fibers.

- Electromyography (EMG) and nerve conduction studies (NCSs) in chronic (classic) steroid myopathy<sup>10</sup>
  - Motor and sensory NCS results typically are normal.
  - Repetitive stimulation studies do not reveal significant decrement or increment.
  - EMG studies reveal normal insertional activity with little abnormal spontaneous activity (positive sharp waves and fibrillation potentials).
  - EMG may reveal a mild decrease in motor unit action-potential amplitude during maximal recruitment.
  - In moderate to severe cases, studies may show an early recruitment pattern.
- EMG and NCS in acute steroid myopathy<sup>11</sup> - Some case reports have indicated abnormal EMG findings, including abnormal spontaneous activity (positive sharp waves and fibrillation potentials), early recruitment, and small, polyphasic motor units. There have also been findings suggestive of the development of associated neuropathy following high-dose corticosteroid treatment.

## Histologic Findings

Muscle biopsy typically shows a preferential atrophy of type II fibers, particularly the fast-twitch glycolytic fibers (type IIB), with some atrophy of other fiber types.<sup>9, 6</sup> There is a distinct lack of necrosis or regeneration of muscle. Some studies, however, have reported focal and diffuse necrosis of all fiber types, without predilection for type II fibers.

## Treatment

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### Rehabilitation Program

#### Physical Therapy

Some literature suggests that aerobic exercises and resistance training may help to prevent weakness or reduce its severity. Although there are no definitive recommendations regarding therapy for steroid myopathy, it would seem reasonable to direct therapy to address the weakness and resulting impaired mobility. Range-of-motion exercises (either passive, active-assisted, or active, depending on the degree of weakness) and stretching exercises should be performed to prevent joint contractures. As a general rule, resistance exercises should be limited to muscles with greater than antigravity strength. Bed mobility, balance activities, transfer training, and gait training should be included to address decreased mobility. However, high intensity exercise should be avoided, because, according to some preliminary animal research models, it may be harmful.<sup>12</sup>

#### Occupational Therapy

Occupational therapy may focus on maximizing the patient's ability to independently perform activities of daily living. Training may include the use of assistive devices to enhance the patient's performance of self-care tasks, such as a balanced forearm orthosis to allow positioning of the upper arm in a manner that permits more independent feeding. Other adaptive equipment may include a raised toilet seat and similar devices that allow the patient to rise from a sitting position, and/or a motorized lift for ascending stairs.

### Consultations

Any adjustment of a patient's corticosteroid medications should be coordinated with the physician who has been prescribing those agents. Given reports of respiratory muscle weakness causing respiratory impairments,<sup>6, 13</sup> consider consultation with a pulmonologist. Consultation with a neurologist can be considered for assistance with diagnosis and for the exclusion of other potential causes of weakness. A physiatrist can also be consulted for assistance with diagnosis and with the management of a therapy program.

### Other Treatment

- In cases of myopathy caused by long-term corticosteroid use, decreasing the corticosteroid dose to below a 30 mg/d threshold may result in resolution of muscle weakness. In patients in whom myopathy has resulted from a short course of high-dose corticosteroid use, partial or complete recovery has been reported following the discontinuation of steroid administration.<sup>13</sup>
- Preliminary studies on rats suggest that creatine plays a part in the prophylaxis of steroid-induced myopathy. Further studies are needed to explore this possible treatment/prevention option.<sup>14</sup>

## Medication

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Various medications, including potassium supplements, phenytoin, vitamin E, and anabolic steroids, have been tried as potential treatments for steroid myopathy.<sup>4</sup> None have been clearly shown to prevent or reverse muscle weakness induced by steroid myopathy. The main treatment recommendations for steroid myopathy are a decrease in the dose of steroid to below a threshold level or the discontinuation of the corticosteroid's use. Alternate-day dosing could also be considered.<sup>15</sup> Another recommendation is that the currently administered steroid be exchanged for one that is not fluorinated.

## Follow-up

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### Deterrence/Prevention

- Consider the judicious use of steroids.

### Complications

- Although prior studies have reported full motor recovery, some patients may be left with varying degrees of residual weakness.

### Prognosis

- In chronic (classic) steroid myopathy, recovery from weakness may take weeks to months following discontinuation or dose reduction of the corticosteroid.
- In acute steroid myopathy, recovery may be prolonged (>6 mo).

### Patient Education

- Inform patients of the potential of development of myopathy when starting high-dose or long-term corticosteroid therapy.

## Miscellaneous

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### Medicolegal Pitfalls

- The main potential pitfall in diagnosing steroid myopathy relates to patients with polymyositis/dermatomyositis, which typically is treated with corticosteroids. The main symptom of polymyositis/dermatomyositis is proximal upper and lower extremity weakness. When these patients, while being treated with corticosteroids, develop increasing weakness, it can be difficult to determine whether the weakness is secondary to the polymyositis or to steroid myopathy.
- Laboratory studies can aid in differentiating between the 2 conditions. Creatine kinase typically is elevated

significantly in polymyositis/dermatomyositis. In steroid myopathy, it typically has been described that, although there is elevated urinary creatinine excretion, the serum creatine kinase is not significantly elevated.<sup>1, 11</sup> However, some studies have reported elevations of creatine kinase in some cases of the previously described acute form of steroid myopathy.

- On electrodiagnostic study, polymyositis typically demonstrates normal NCS results, as, commonly, does steroid myopathy. On EMG study, however, polymyositis demonstrates abnormal spontaneous activity and increased polyphasic waveforms with short durations. The classic form of steroid myopathy has been described as not demonstrating significantly abnormal EMG findings. Again, some studies have described an acute form of steroid myopathy that can demonstrate abnormal spontaneous activity, an early recruitment pattern, and small, polyphasic waveforms.<sup>10, 11</sup>
- The initial recommendation is to decrease or discontinue the use of the corticosteroid.<sup>13</sup> If the weakness improves, then steroid myopathy is the most likely diagnosis. If the weakness persists or worsens, then the most likely diagnosis is worsening of the polymyositis.

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## Keywords

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corticosteroid-induced myopathy, myopathy, steroid, steroids, corticosteroid, corticosteroids, polymyositis, dermatomyositis, adrenal tumor, adrenal tumors, hydrocortisone, prednisone, triamcinolone, dexamethasone, steroid myopathy, acute steroid myopathy, chronic steroid myopathy, critical illness myopathy, excess endogenous corticosteroid production, excess exogenous corticosteroid production, asthma, steroid treatment for asthma, steroid treatment for polymyositis, steroid treatment for connective tissue disorders, steroid treatment for rheumatoid arthritis, fluorinated steroids, nonfluorinated steroids, prolonged administration of prednisone

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Disclosure: Nothing to disclose

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Disclosure: Teva Neuroscience Honoraria for Speaking and teaching; Serono-Pfizer Honoraria for Speaking and teaching

### Acknowledgments

**Debra Ibrahim**, 4th year medical student, New York College of Osteopathic Medicine, class of 2008, assisted with the revision of this manuscript.

**Evish Kamrava**, 4th year medical student, St. George's University School of Medicine, class of 2009, assisted with the revision of this manuscript.

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