myalgias, muscle enlargement, slow relaxation of the reflexes, and marked (up to 100-fold) increase of the serum CK level.

Excess corticosteroids can result from endogenous Cushing's syndrome or can be caused by exogenous glucocorticoid administration. Iatrogenic corticosteroid myopathy (or atrophy) is the most common endocrine-related myopathy. However, muscle weakness is rarely the presenting manifestation of Cushing's syndrome and, in virtually all instances of corticosteroid myopathy, other factors contributing to weakness are also present. Therapy consists of reducing the corticosteroid dose to the lowest possible level. Exercise and adequate nutrition prevent and may improve weakness.

Toxic Myopathies

Many drugs have been associated with muscle damage, proximal weakness, elevated CK levels, myopathic EMG readings, and abnormalities on muscle biopsy. Symptoms generally improve after stopping the medication. Some drugs can produce acute, rapidly progressive muscle destruction and myoglobinuria, particularly the hypocholesterolemic drugs, including the statins and fibric acid derivatives. In some patients statin use has been associated with a subsequent autoimmune necrotizing myopathy associated with antibodies to HMG-CoA.

Critical illness myopathy (CIM), also termed, acute quadriplegic myopathy, develops in a patient in the intensive care setting and is often discovered when a patient is unable to be weaned off a ventilator. The cause of the diffuse weakness is the prolonged daily use of either high-dose intravenous glucocorticoids or nondepolarizing neuromuscular blocking agents, often both. Patients often have had sepsis and multi-organ failure. The diagnosis of critical illness myopathy can be confirmed by muscle biopsy, which shows the loss of myosin-thick filaments on electron microscopic examination. Treatment is supportive after discontinuation of the offending agents.

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