Statin-Induced Myopathy: A Clinical Perspective

Abdulsalam A Al-Sulaiman, MD, PhD, FRCP(C)* Fahd A Al-Khamis, MD**

Statins are at the forefront of treatments for hyperlipidemia, coronary artery disease and stroke. Patients may not adhere to Statins therapy due to hepatic or neuromuscular side effects that include neuropathy and myopathy. The latter include myalgia, lassitude, fatigue, proximal muscle weakness with or without elevated creatine kinase (CK) or myoglobinuria. Studies suggest that these symptoms are under reported and may occur in as much as 5% or more. This article reviews the definition, incidence, possible mechanisms, risk factors, clinical presentation and suggested management of Statin-induced myopathy.

Bahrain Med Bull 2009; 31(2):

Statins, 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors, are essential in the prevention and treatment of patients with hyperlipidemia, ischemic heart disease and stroke. Recently statins are being investigated for use in patients with dementia. Since their introduction in 1987, statins have shown to be safe and effective but several serious adverse effects including hepatic and neuromuscular complications. The latter are mainly myopathy and to a lesser extent neuropathy. The incidence of neuromuscular complications is expected to rise with the widespread use of statins. It seems pertinent therefore to alert physicians about the potential risk factors, clinical features and management of Statin-induced myopathy.

Definitions

Myopathy in general terms refers to any muscle disease regardless of etiology. Pasternak and colleagues defines the spectrum of statins myopathy as follows: myalgia refers to muscle aches or weakness without creatinine kinase (CK) elevation and myositis refers to muscle symptoms with increased CK. Rhabdomyolysis is muscle symptoms with marked CK elevation more than 10 times the upper limits of normal. This definition has been challenged because there is no reference to specific histopathological terms particularly evidence of inflammatory changes.

*Department of Neurology
King Fahd Hospital of the University
Al-Khobar, KSA