**STATIN INTOLERANCE - NOVEL BIOMARKERS, MECHANISMS, DEFINITION, AND EFFECTIVE MANAGEMENT. A WAY TO IMPROVE THERAPY ADHERENCE AND AVOID STATIN DISCONTINUATION?**

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Over the past 3 decades, statins have become the cornerstone of prevention and treatment of atherosclerotic cardiovascular and metabolic diseases. Albeit generally well tolerated, they can elicit a variety of muscle-associated symptoms that represent the most important reason for treatment discontinuation, switching or non-adherence. The statin-associated myopathy has been systematically underestimated by randomized controlled trials as compared with the incidence observed in clinical practice and obtained from patients’ registries. This discrepancy has several reasons among which the lack of reliable diagnostic test(s) and validated questionnaire to assess muscle symptoms are recognized as unmet needs. Therefore it is a large need to look for the cellular and molecular biomarkers to signal/diagnose muscle-related complaints.

When initiating statin therapy, attention to risk factors for statin intolerance is strongly recommended. The nocebo effect coupled with the challenges of diagnosing statin myopathy undermines drug adherence that is critical for achieving the benefits of lipid-lowering and cardiovascular risk reduction. A temporal relationship should be made between the initiation of therapy and development of symptoms to aid in diagnosis. To limit errors in the diagnosis of statin intolerance, improvements in clinician-patient communication about the side effects and benefits of statins should be attempted. The mainstay of treatment is statin cessation or statin dose reduction and evaluation of alternative causes for muscle related symptoms. Most symptoms usually resolve within 2 weeks of discontinuing therapy. The patient can be re-challenged with the same stain at a lower dose or an alternative statin. Non-statin lipid lowering therapies offer an alternative to patients who cannot tolerate statins.