chronic kidney disease, hypothyroidism), and 3) prior severe statin myopathy (i.e., rhabdomyolysis, CK >5x ULN). Prior NLA guidance suggests withholding statin therapy for CK levels >3 times ULN[13] and the ACC/AHA guidelines recommends withholding statin therapy for CK levels >5 x ULN.[63] Post-treatment CK measurements may be useful in some patients with SAMS, but are particularly important in the evaluation of suspected myopathy or rhabdomyolysis.

Measurement of anti-_3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGCR) antibodies, electromyography, muscle strength testing, and muscle biopsy are neither pragmatic for clinical practice nor routinely recommended. These tests may be ordered by a neurologist or a clinical lipid specialist for evaluation of persistent weakness, chronic CK elevations, or muscle pain or tenderness that does not remit with statin withdrawal.[13] Measurement of levels of hepatic aminotransferase, alkaline phosphatase, and bilirubin are helpful to exclude severe hepatic impairment and measurements of blood urea nitrogen and creatinine are helpful to exclude renal dysfunction, both of which can impair statin metabolism and aggravate statin intolerance. Hypothyroidism is an important secondary cause of myalgia/myopathy that should be ruled out. Vitamin D levels can also be measured and supplemented if deficient. Refer to the vitamin D supplementation section for more details.

What management strategies are helpful to address SAMS?

A multifaceted approach is necessary after a patient experiences a perceived threat to well-being, quality of life, and/or functional status related to SAMS. Utilization of effective communication strategies is vital to establish a trusting patient-clinician relationship and optimize outcomes in patients with SAMS.[63] It is also important to validate and acknowledge patient-reported SAMS, while also emphasizing the risk of cardiovascular morbidity and mortality that may manifest without the use of statin treatment. This