



The pain of cholesterol-lowering therapy

Everyone seems to know statins cause muscle pain. The phenomenon of statin myopathy remains, at least in part, a conundrum to me—but apparently not to many patients with hypercholesterolemia who fear the muscle problems.

Given the perception that statin myopathy is common, the incidence of significant myalgias in clinical trials is surprisingly low and similar to that with placebo (generally less than 5%), and that of myositis or rhabdomyolysis is much rarer.

In this issue of the *Journal*, Dr. Genaro Fernandez and colleagues (page 393) discuss possible reasons for the discrepancy between the prevalence of statin-associated myalgias in clinical trials vs real practice. They suggest that patients more likely to develop myalgias are weeded out in the screening phase of clinical trials and that the trials may be too small and too short to capture this information. Yet in practice, many patients develop muscle pain shortly after starting statin therapy. I suggest another explanation—ie, that volunteers in clinical trials *want* to take the medication, while in the clinic my patients are reluctant to take one more medication and have trepidations about starting one that they “know” causes muscle pain.

But I don’t think *all* statin myopathy is due to the power of suggestion. Some patients clearly have drug-elicited elevations in creatine kinase (CK), and others (including me) experience significant myalgias with one statin but can tolerate another.

A challenge in my rheumatology clinic is distinguishing statin myopathy from other underlying problems in patients referred for evaluation of pain, weakness, or elevated CK. I first establish a temporal relationship between the drug initiation and the start of symptoms, and I look for other drugs or possible drug interactions that could be causing the problem, such as colchicine vacuolar myopathy in the setting of newly initiated statin therapy. I look for an alternative explanation for the pain syndrome, such as upper-arm pain and physical findings that suggest rotator cuff disease, or lateral hip-area pain due to bursitis. In some patients, statins may pose a metabolic challenge that unmasks (or brings to the physician’s attention) an underlying biochemical disorder of the muscle, such as myotonic dystrophy or even polymyositis.

Dr. Fernandez et al offer sound advice, as they suggest keeping an open diagnostic mind when evaluating patients with apparent statin myopathy. In particular, with these authors, I urge you to perform a careful personal and family history and a focused examination, ask about vigorous physical exercise, check the CK, and withhold and then rechallenge with the statin before ordering a slew of serologic and metabolic tests.

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