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Beyond Myopathy: Statin-Associated Peroneal Tendinitis After High-Intensity Therapy in Secondary Prevention

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Introduction: Statins are among the most widely prescribed drugs in cardiovascular prevention. Although statin-associated myopathy is well recognized, tendinopathy and tendon rupture are rarer and less frequently identified.[1,2]

Case report: 60-year-old woman was referred to a cardiologist following routine annual follow-up with an immunologist. Six months prior she had begun to experience exertional angina (CCS class II), accompanied by dyspnea on exertion. Her medical history included untreated dyslipidemia, a family history of premature coronary artery disease, hypothyroidism treated with levothyroxine, and Sjögren's. Laboratory testing demonstrated hypercholesterolemia (LDL-C 4.9 mmol/L) and markedly elevated lipoprotein(a) at 254 nmol/L. Transthoracic echocardiography revealed no abnormalities. CT coronary angiography demonstrated a suspected obstructive lesion in the proximal LAD. Following initial assessment, the patient was diagnosed with chronic coronary syndrome. Coronary angiography revealed an intermediate ostial LAD stenosis and a significant mid-LAD stenosis. Intravascular ultrasound confirmed a significant calcified ostial LAD lesion with a minimal lumen area of 3.9 mm². Percutaneous coronary intervention was performed with implantation of a drug-eluting stent extending from the left main to the LAD using a provisional technique, together with an additional stent in the mid-LAD. The patient was discharged on the first post-procedural day on dual antiplatelet therapy (aspirin and ticagrelor) and atorvastatin 80 mg combined with ezetimibe 10 mg due to failure to achieve LDL-cholesterol targets. At the 8-week review she remained free of angina and had a normal exercise stress test. LDL-cholesterol had decreased to 1.7 mmol/L. However, she reported intermittent swelling and pain around the ankle, occasionally associated with erythema, without preceding trauma. Symptoms were sometimes triggered by prolonged standing or walking but also occurred at rest. Episodes of swelling and marked tenderness lasted several days and resolved spontaneously. Given suspicion of statin-associated tendinitis, musculoskeletal ultrasound of the lower leg and ankle was performed, demonstrating peritendinous effusion and Power Doppler signal around the peroneal tendons consistent with active tendinitis. The statin dose was reduced, and therapy was switched to rosuvastatin 10 mg combined with ezetimibe 10 mg. Owing to intolerance of high-intensity statin therapy the patient was considered a candidate for PCSK9 inhibitor therapy.

Conclusion: The precise molecular mechanism of statin-related tendon injury remains unclear, although in vitro studies suggest that imbalance in matrix metalloproteinase activity may contribute. Dose reduction or switching to an alternative statin may alleviate symptoms in some patients. In those with severe symptoms or tendon rupture, alternative lipid-lowering therapy such as PCSK9 inhibition should be considered.

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