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Statins And Tendinopathy Involving Multiple Tendons of The Foot: A Case Report

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1. Summary

A 60-year-old man with hypertension developed progressive left foot pain approximately two weeks after starting atorvastatin 20 mg for newly identified hypercholesterolaemia. The pain became disabling, limiting weight-bearing and preventing attendance at work. Plain radiographs showed no acute abnormality or fracture. An initial clinical diagnosis of peroneal tendinopathy was subsequently extended to include the tibialis anterior and foot extensor tendons. Symptoms persisted despite changes in footwear, analgesia and advice regarding rest and exercises. Following clinician-advised withdrawal of atorvastatin, pain improved markedly within 24–48 hours and had almost resolved after one week. The temporal relationship and positive dechallenge support suspected atorvastatin-associated tendinopathy, although they do not establish causation.

2. Case Presentation

A 60-year-old man with a background of hypertension underwent routine blood testing, which demonstrated hypercholesterolaemia. Atorvastatin was commenced at a dose of 20 mg. He initially tolerated treatment without reported adverse effects.

Approximately two weeks after treatment initiation, he developed mild pain in the left foot. Over the following days, the pain intensified and became predominantly localised to the medial and lateral aspects of the forefoot. He changed to footwear with a softer sole, but this did not alleviate his symptoms. Analgesics also provided no relief. The pain progressed to the extent that he had difficulty bearing weight and mobilising and was unable to attend work.

3. Investigations and Clinical Assessment

Plain radiographs of the left foot and ankle showed no acute abnormality and no evidence of fracture. On subsequent review, a clinical diagnosis of peroneal tendinopathy was made. As symptoms persisted, involvement of the tibialis anterior tendon and the extensor tendons of the foot was also identified clinically.

4. Management and Outcome

Initial management comprised advice on rest and analgesia, followed by exercises. This approach did not provide symptomatic relief. A medication review identified the recent introduction of atorvastatin, prompting consideration of a drug-associated tendinopathy. The patient was advised by his clinician to discontinue atorvastatin as a trial of withdrawal. Within 24–48 hours of discontinuation, he reported a marked reduction in pain, accompanied by improved ability to bear weight and mobilise. After one week without atorvastatin, his pain had almost completely resolved and he was mobilising again.

5. Discussion

This case describes suspected atorvastatin-associated tendinopathy involving the peroneal, tibialis anterior and extensor tendons of one foot, identified clinically. Symptoms began approximately two weeks after atorvastatin initiation, progressed despite conservative measures and improved markedly within 24–48 hours of clinician-advised withdrawal, with near-complete resolution after one week. The temporal relationship and positive dechallenge support a possible drug association but do not establish causation. The rapid symptomatic improvement should not be interpreted as evidence of structural tendon recovery.

Published case reports have described statin-associated tendon disorders, including tibialis anterior tenosynovitis. The involvement of several tendons in a single foot is a distinctive feature of the present case, rather than evidence of a previously unreported anatomical association.

6. Clinical Context and Potential Mechanisms

Tendon symptoms are often multifactorial. Metabolic conditions, including diabetes and hypercholesterolaemia, may contribute to tendon pathology, while age, obesity and rheumatoid arthritis can also influence susceptibility. Mechanical loading and local factors should be considered alongside medication exposure rather than treated as evidence of a drug-related cause. Other medicine classes associated with tendinopathy include fluoroquinolones, long-term glucocorticoids and aromatase inhibitors.

Statin-associated tendon disorders have been reported at several sites, including the Achilles tendon, shoulder, elbow and hand or

wrist. The Achilles tendon is prominent in published reports, but estimates of its frequency depend on the reporting population and should not be generalised to all statin users. The present case draws attention to the possibility of clinically apparent involvement of less commonly reported foot tendons.

Experimental findings provide a plausible, but unproven, mechanism. In a three-dimensional human tenocyte model, simvastatin exposure increased the release of matrix metalloproteinases, including MMP-1 and MMP-13, and produced a weaker, disrupted extracellular matrix. These findings suggest that altered collagen turnover and matrix integrity could contribute to tendon symptoms.

7. Clinical Evidence, Frequency and Timing

The epidemiological evidence remains mixed, and tendinopathy should be distinguished from tendon rupture. A 2023 nationwide cohort [1] study found a higher hazard of the combined tendinopathy outcomes among statin users than matched non-users (hazard ratio 1.435; 95% confidence interval 1.411–1.460). A 2019 study [2] of two Swedish population cohorts similarly reported associations with trigger finger and with shoulder tendinopathy, the latter reaching statistical significance in men. These observational findings support an association but remain susceptible to confounding, including the contribution of hypercholesterolaemia itself.

By contrast, a UK propensity score-matched study [3] of new statin users aged 45 years or older found no clear increase in Achilles or biceps tendon rupture (hazard ratio 0.95; 95% confidence interval 0.84–1.08). A large 2024 US claims analysis [4] likewise found no association between statin use and increased risk of the combined tendon rupture and tendinopathy outcome among patients with atherosclerotic cardiovascular disease. A 2025 two-sample Mendelian randomisation study [5] found no statistically significant causal relationship between genetically proxied statin use and Achilles tendon injury or Achilles tendinitis, although some analyses were limited by the number of available genetic instruments. A 2025 systematic review [6] of 12 clinical studies reported heterogeneous findings, including positive tendinopathy associations, null rupture findings and conflicting outcomes after tendon repair. Collectively, these studies do not demonstrate a consistent increase in tendon rupture risk, nor do they exclude an uncommon medication-associated tendon disorder in an individual patient.

Spontaneous reports cannot reliably establish the frequency of statin-associated tendinopathy. The 96-patient pharmacovigilance series was a collection of reported cases, not a cohort of statin-treated patients from which a population prevalence of 2.09% could be calculated. Although useful for detecting potential safety signals, these reports lack an exposure denominator and are subject to reporting bias. Time to onset varies. In the same series, 59% of tendon manifestations occurred within the first year of treatment, while early case reports described onset after one to two months. The 2023 cohort found stronger associations at lower cumulative defined daily doses, which weakened as cumulative exposure increased. These dose categories do not correspond directly to fixed three- or six-month treatment periods. Symptoms began comparatively early in this case, at approximately two weeks, but timing alone does not establish a drug-related cause.

8. Clinical Implications

The principal lesson is the importance of reviewing recently introduced medicines when tendon pain is persistent, disabling or involves several tendons. In this case, the temporal relationship and marked improvement after withdrawal support suspected atorvastatin-associated tendinopathy, while the clinical diagnosis and limited follow-up warrant caution in attributing causality. Any decision to withdraw, replace or reconsider lipid-lowering therapy should remain clinician-led and balance symptom severity against the patient's cardiovascular risk and the established benefits of treatment.

Learning points

- Consider a medication-associated tendon disorder when new, persistent or disabling tendon pain follows the introduction of treatment.
- A temporal relationship and positive dechallenge support a possible drug association but do not establish causation or structural tendon recovery.
- Distinguish tendinopathy from tendon rupture when interpreting the evidence and keep lipid-lowering treatment decisions clinician-led and individualised to cardiovascular risk.

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