PAIN MEDICINE CASE REPORTS

ISSN 2768-5152

Volume 10, Number 5, pp. 401-404

STATIN-ASSOCIATED MYOPATHY MIMICKING LUMBAR RADICULOPATHY: A CASE SERIES

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Background: Chronic low back pain (cLBP) is often managed conservatively, but it is vital to rule out certain causes before pursuing targeted treatment. A thorough evaluation, including medication reconciliation, ensures appropriate care and may prevent costly or invasive treatments for many ubiquitous conditions, including patients taking statin medications.

Case Report: We present a series of 3 cases in which patients on statin medication presented to a pain management clinic with symptoms initially resembling mechanical and/or radicular chronic LBP (cLBP). In all 3 cases, medication reconciliation revealed high-dose statin medication, and supervised dose reduction or discontinuation was temporally linked with the alleviation of symptoms.

Conclusion: This is the first known case series reporting suspected statin-associated muscle symptoms that mimic lumbar radiculopathy. These cases underscore the importance of comprehensive clinical evaluation, including medication reconciliation. Clinicians should consider medication-related side effects in the differential diagnosis of new or atypical musculoskeletal pain.

Key words: Case series, statin myopathy, statin-associated muscle symptoms, lower back pain, spinal stenosis, lumbar radiculopathy

BACKGROUND

Low back pain (LBP) is a leading cause of disability and physician visits, affecting one in 4 adults in the United States. A significant subset of LBP patients present with radicular symptoms such as pain, numbness, or weakness in the lower extremities. While many cases are self-limiting, refractory symptoms often prompt advanced imaging to identify structural etiologies and subsequently guide pain management strategies (1).

However, lumbar radiculopathy is one of the many conditions that present with radiating low back pain. A comprehensive history and physical examination, along with imaging, are therefore critical in identifying the true pain generator. Statin-associated muscle symptoms (SAMS), for example, remains a well-documented adverse effect of statin medications, one of the most ubiquitous medication classes worldwide (2). While generally well-tolerated, SAMS can present with nonspecific myalgias and weakness, most often in the proximal muscles (3). The PRIMO study found that 10.5% of patients with high-potency statins reported muscle-related symptoms with pain in the lower extremities being most common (4). This clinical presentation of lower extremity pain can create a diagnostic challenge, as it may closely mirror symptoms of lumbar radiculopathy.

Both chronic low back pain (cLBP) and statin myopathy are prevalent conditions, which may lead to diagnos-

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Disclaimer: There was no external funding in the preparation of this manuscript.
Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.
Patient consent for publication: Consent obtained directly from patient(s).
This case report adheres to CARE Guidelines and the CARE Checklist has been provided to the journal editor.
Accepted: 2026-02-04, Published: 2026-07-31

tic confusion. Many providers may overlook medication reconciliation, which can result in potentially unnecessary, wasteful, and costly workups or invasive procedures that may miss the underlying cause (5,6).

Here, we present a series of 3 cases in which statin medication was temporally associated with symptoms that mimicked lumbar radiculopathy. These cases highlight the critical importance of medication reconciliation in the differential diagnosis of new or worsening musculoskeletal pain. To the authors' knowledge, this is the first reported case series of SAMS presenting in a pattern mimicking lumbar radiculopathy.

CASE REPORT

Patient A: 79-Year-Old Man

A 79-year-old man with cLBP and a medical history of hyperlipidemia, which he managed with statin therapy, developed new-onset leg pain mirroring radicular symptoms while on a daily regimen of 80 mg of simvastatin. His baseline cLBP, which affected the bilateral lower back and right buttock, had been stable, with numeric rating scale (NRS) score of 0-2/10, managed conservatively. He developed radicular pain localized to the left knee that radiated to the shin and worsened when lying down, rated 3-4 out of 10 on the NRS. MRI revealed moderate to severe spinal canal stenosis at L2-3, moderate left neuroforaminal stenosis at L4-5 and L5-S1, and moderate tricompartmental knee osteoarthritis. These imaging findings, which could explain the new symptoms, contributed to diagnostic uncertainty. In the month after the onset of symptoms, the patient's cardiologist switched his prescription from simvastatin to a daily regimen 40 mg of atorvastatin . At the one-month follow-up, the patient reported that this new lower extremity pain had "almost entirely resolved," with NRS improving to 0 out of 10.

Patient B: 89-Year-Old Man

An 89-year-old man with a history of lumbar stenosis and baseline sciatica experienced new-onset symptoms distinct from his baseline cLBP after initiating a high-intensity daily regimen of 80 mg of atorvastatin following a minor stroke. His pain had previously been managed conservatively and with epidural steroid injections. Within a year of starting atorvastatin, he began to experience new-onset unilateral myalgias and radiating pain distinct from his prior sciatica, as well as balance issues and ataxic gait. He contacted his cardiologist about the muscle spasms, joint pains, and low energy, suspecting these symptoms were related to the statin. After the consultation with the neurology and cardiology departments, the patient's atorvastatin dose was halved to 40 mg daily. Two months later, his neurologist documented complete resolution of the patient's myalgias, radiating lower-extremity pain, dizziness, ataxia, and impaired balance. This resolution was corroborated one month later by his cardiologist. The patient's serum creatine kinase (CK) level, obtained 3 months after the statin dose adjustment, was normal at 90 U/L. Subsequent follow-up visits over 6 months confirmed that the patient had tolerated the reduced dose, and relief was durable. His Modified Rankin Scale (mRS) score improved from 2 ("Slight disability") on the high-dose statin to one ("No significant disability") following dose reduction.

Patient C: 51-Year-Old Man

A 51-year-old man, an avid runner with no prior history of back or leg pain, developed debilitating bilateral leg pain, weakness, tingling, and numbness shortly after initiating a daily regimen of 5 mg of rosuvastatin for his hyperlipidemia. His symptoms worsened following an increase to 10 mg daily and then exacerbated exponentially after the addition of ezetimibe 10 mg daily. The patient's function declined from running regularly to being unable to walk more than one city block due to shooting leg pains and weakness. His primary care physician documented "sudden weakness of bilateral lower extremities while walking" and an uncoordinated gait during this period.

Despite an MRI that demonstrated severe central canal stenosis at L4-5 with degenerative anterolisthesis, the patient discontinued both rosuvastatin and ezetimibe. Within 2 months, his symptoms improved slightly, which allowed him to initiate physical therapy. His T-score in the Physical Function section of the Patient-Reported Outcomes Measurement Information System® (PROMIS®) improved from 34 to 44 by the third month after the cessation, reflecting a shift from moderate to mild dysfunction. By 7 months, he had successfully returned to high-level activities, including jogging and playing sports. His cardiologist noted the symptoms had been "clearly worse on statin." Lumbar extension range of motion improved from 15 degrees initially following discontinuation to 60 degrees by month 6 in physical therapy.

This case series is devoid of identifiable patient

information and is therefore exempt from written informed consent and IRB review in accordance with NYU Langone Health policy.

DISCUSSION

We present a series of 3 cases of patients with documented structural LBP who experienced newly emergent or acutely worsened lower extremity pain, weakness, and/or gait dysfunction after statin initiation, titration, or dose escalation. Notably, all 3 cases demonstrated clinical improvement after the discontinuation of their statin or the reduction of their doses. While the presence of structural spinal pathology may complicate the diagnosis, the temporal relationship between statin use and symptom fluctuation raises the possibility that SAMS may contribute to the clinical presentation.

SAMS encompasses a broad spectrum of clinical manifestations from simple myalgia to clinical rhabdomyolysis or necrosis, most commonly affecting proximal lower extremities symmetrically and often without CK elevation (7,8). The condition is usually self-limited, particularly in the more prevalent, less severe forms resulting in simple myalgia, and improves within 2-4 weeks of cessation. The Statin-Associated Muscle Symptom Clinical Index (SAMS-CI) is a validated diagnostic tool that correlates symptoms, temporal pattern, discontinuation, and rechallenge to assist clinicians in arriving at the diagnosis, particularly in cases of overlapping symptoms (9). Importantly, recurrence of similar symptoms within 4 weeks of reinitiating the statin (referred to as rechallenge) is considered indicative of a strong causal relationship (10). The prevalence of SAMS in real-world settings remains debated, but observational studies and registries suggest higher rates than are reported in randomized trials (8).

The NLA Muscle Safety Task Force's comprehensive review of SAMS addresses the importance of diligent evaluation of patients with similar complaints to ours, considering a differential including orthopedic, central or peripheral neurologic, vascular, or rheumatologic causes (11). There are few individual case reports, including that of a 60-year-old woman who developed MRI-confirmed piriformis syndrome on long-term atorvastatin with antalgic gait (12). Importantly, her symptoms resolved with intermittent discontinuation and relapsed with rechallenge, showing that SAMS may manifest in atypical forms mimicking nerve root compression. In rare, severe instances, statins may cause a necrotizing autoimmune myopathy characterized by proximal muscle pain and weakness, profound CK elevation, and necrotizing changes on biopsy, that persist despite statin discontinuation (13).

In each case in this series, the pattern of symptom onset and resolution corresponded with statin exposure, including dose-dependent worsening in 2 patients. In Patient A, symptoms resolved fully and rapidly after switching statins, raising strong suspicion of a medication-related effect in the absence of other interventions. Particularly notable is that the original medication, 80 mg of simvastatin, is associated with a risk of myopathy and rhabdomyolysis over 10 times greater than that associated with other statins and doses, including 40 mg of atorvastatin, the medication to which he was switched (14,15). Patient B described the pain as distinctly different from his prior radiculopathy, and gait abnormalities emerged or worsened in all 3 cases. Patient C encountered a significant enough worsening of functional status, impairing his ability to participate in physical therapy, which was alleviated following statin cessation. That relief became durable with prolonged therapy.

Limitations of this series include its retrospective nature, small sample size, and lack of confirmatory testing such as muscle biopsy or electromyography. CK levels were drawn from only one of the 3 patients, and this was only done 3 months after cessation, following symptomatic improvement. Additionally, functional outcome measures were also available for only one of 3 cases. While lumbar spinal stenosis was present in all 3 patients and might have been a significant confounder, such improvement from statin cessation would be atypical from a purely structural etiology, particularly without concurrent intervention. Importantly, pre-existing musculoskeletal disease was identified as a significant risk factor that might predispose patients to myopathic side effects from statins in the PRIMO study (4).

CONCLUSION

SAMS represents a clinical spectrum manifesting as pain, weakness, cramping, gait impairment, or paresthesia that can mimic lumbar radiculopathy, stenosis, or other causes of structural LBP. Structural spinal pathology often coexists in the diagnosis, but it is imperative in clinical practice to take a thorough history of the patient, including medication reconciliation, to elucidate the true cause. A suspicion of SAMS is warranted when symptoms are atypical or linked temporally to statin therapy. A supervised diagnostic de-challenge and,

when appropriate, a confirmatory rechallenge, may clarify etiology, improve patient outcomes, and avoid unnecessary imaging and interventions. Future studies may seek to evaluate whether existing diagnostic tools can reliably distinguish SAMS from structural musculoskeletal pain and assess the safety and diagnostic value of supervised statin de-challenge in this population.

Author Contributions

Ruben Dovlatyan (RD): Conception of the case series, data de-identification, literature review, manuscript drafting with primary focus on discussion and conclusion, coordination of writing tasks, editing.

Blake Jones (BJ): Drafting of case descriptions, manuscript editing.

Bruno Alonso (BA): Drafting of introduction, literature review, manuscript editing.

Sidharth Sahni (SS): Drafting of discussion in collaboration with RD, literature review, manuscript editing.

David Jevotovsky (DJ): Drafting of abstract, literature review, manuscript editing.

Dylan Banks (DB): Assistance with drafting conclusion, manuscript editing.

Neal Rakesh (NR): Final manuscript review and editing.

Richard Lau (RL): Clinical evaluation of all patients, final manuscript review and editing.

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