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^{18}F-FDG Muscular Uptake in Statin-Associated Symptoms Without Myositis

How Long to Stop Treatment for Image Quality Improvement?

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Abstract: Statin-associated muscle symptoms are a frequent adverse effect of statin treatment and can lead to a statin-associated myopathy characterized by a significant serum creatine kinase increase. We report the case of an 80-year-old man who presented an increased muscular ^{18}F-FDG uptake in a statin-associated muscle symptom without creatine kinase abnormality or inflammation. Statin treatment was discontinued for 6 hours, 3 days, and 7 days on consecutive follow-up examinations. The 1-week window clearly enhanced image quality. This case illustrates the possibility of diffuse muscular ^{18}F-FDG uptake without myositis and the need for a minimal 1-week statin discontinuation to reduce muscular uptake.

Key Words: statin, PET/CT, ^{18}F-FDG, SAMS, myositis

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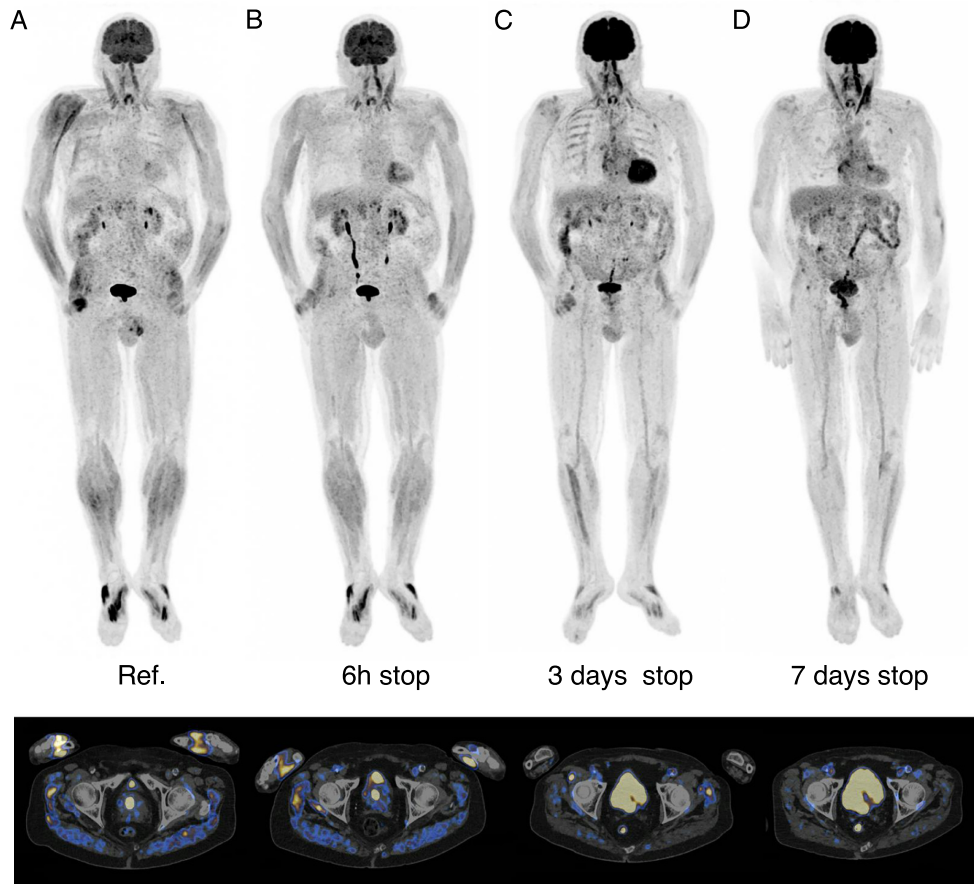

FIGURE 1. Statin-associated muscle symptoms (SAMSs) are a frequent adverse effect of statin treatment (7%–29%) and the main reason for nonadherence.[1] SAMS can occur with normal or slightly elevated serum creatine kinase (CK). The etiology is complex with multifactorial mechanisms and appears more frequently in women.[2] Myopathy is a rarer complication with muscular inflammation and increased CK. Diffuse ^{18}F-FDG muscular uptake on PET was reported in statin-related rhabdomyolysis[3,4] and other myopathies.[5–7] In men, but not in women, the risk of statin-related myopathy is dose-dependent.[2] In this case, a 80-year-old man was referred for ^{18}F-FDG PET in hemopathy follow-up. A long-term statin treatment was prescribed for cardiovascular prevention. On examination, the patient described mild chronic myalgia for more than 6 months. CK and C-reactive protein were normal. Clinically, the patient was classified statin-related myotoxicity (SRM) 1 on the 7-point SRM scale. Acquisitions were all obtained on PET/CT (Biograph mCT Flow; Siemens) 60 minutes after 3.5 MBq/kg ^{18}F-FDG injection. Table flow acquisition was 1.3 mm/s. The patient was fasting without any treatment for at least 6 hours, and glycemia was controlled and normal (5.4–7.0 mmol/L). Reference PET acquisition (**A**) showed an abnormal diffuse ^{18}F-FDG muscular uptake on MIP, more pronounced on shoulders, arms, hips, legs, and feet. This aspect remained similar on the second examination without treatment for 6 hours (**B**). On the follow-up with a statin discontinuation for 3 days (**C**) and 7 days (**D**), an image quality improvement was observed with less muscular uptake and a better ^{18}F-FDG bioavailability, especially on brain. The 1-week statin-free medication showed a better overall image quality except arm movements during the acquisition. Cervical hypermetabolisms were related to uptakes in contracting skeletal muscles. On pelvic transaxial fused PET/CT slices (below MIP images), there was an hypermetabolism of gluteal muscles, which was gradually reduced after 7 days of statin discontinuation. The 2 last acquisitions also revealed osseous and pulmonary focal uptakes related to hemopathy. Diffuse muscular uptake has been reported to alter the tumoral contrast and PET lesion detectability.[8] This case illustrates the possibility of diffuse muscular FDG uptake in SAMS without biological myositis and the need for a minimal 1-week statin discontinuation to improve image quality. This treatment interruption could be planned as the first biological modifications described in the literature were observed from a 5-day statin interruption[9] with only impact on long-term cardiovascular risks after a 3-month discontinuation.[10–12] Thus, this short statin discontinuation could be conceivable without impact on the patient cardiovascular risk. A study on a larger cohort would allow a better assessment of an ideal discontinuation time.