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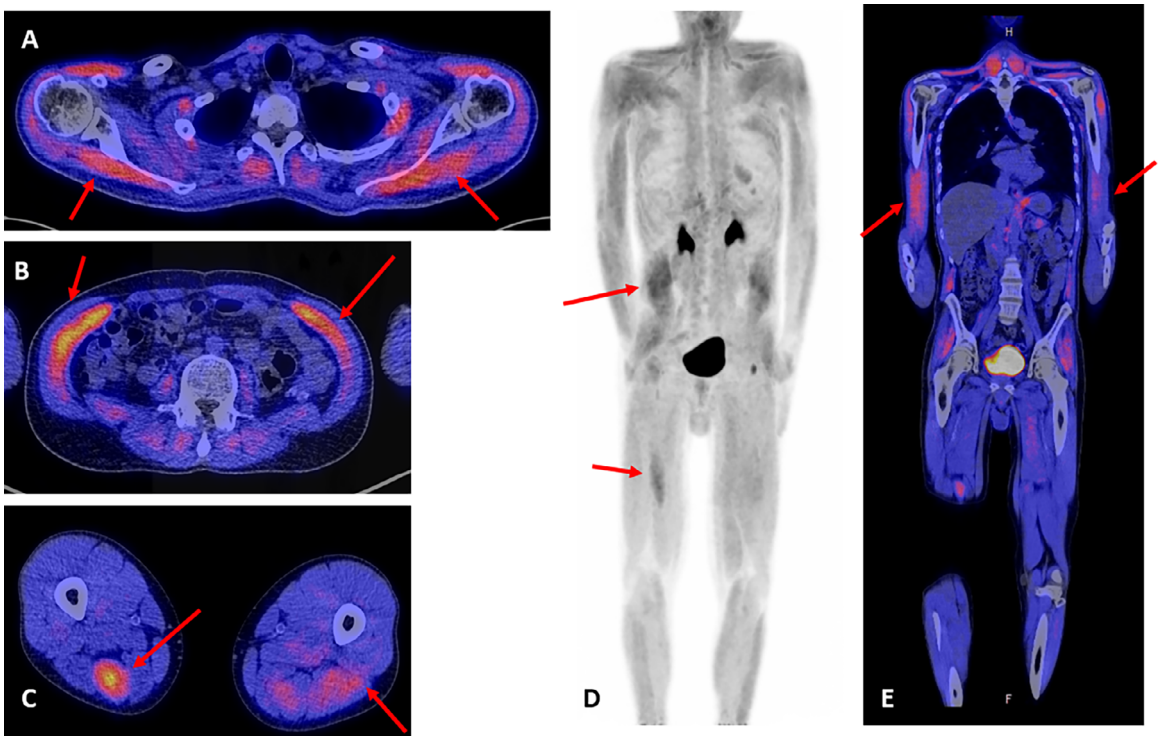
Clinical Images: Tissue remodeling in autoimmune statin-induced necrotizing autoimmune myopathy



The patient, a 54-year-old White male, developed progressive weakness and pain at his proximal muscles, preferentially involving the shoulders and thighs. He took atorvastatin for his hyperlipidemia in the context of metabolic syndrome for the last 10 months. A physical examination revealed reduced muscle strength in his legs and arms. His creatin kinase (CK) level was elevated at greater than 7000 U/L (normal range 21-232 U/L), as were his aldolase and myoglobin levels. Anti-hydroxymethylglutaryl CoA (Anti-HMG-CoA) reductase antibodies were positive, indicating statin-induced necrotizing autoimmune myopathy (NAM). Electromyography and biopsy findings were also consistent with NAM. Connective tissue disease, infection, and malignancy were ruled out. Despite atorvastatin discontinuation, the patient continued to have muscle weakness, persistent elevation in the CK level (>7000 U/L), and anti-HMG-CoA reductase antibodies. Since the onset of myositis 6 years ago, he received high-dose glucocorticoids, intravenous immunoglobulins, methotrexate, rituximab, cyclophosphamide, and azathioprine with limited or no success. A magnetic resonance imaging scan of the thigh showed active myositis with water rich lesions in T2 sequences. To test whether the ongoing myositis leads to tissue remodeling in the affected muscles, we performed a positron emission tomography (PET) scan using a ^{68}gallium-labeled fibroblast activation protein inhibitor (^{68}Ga-FAPI) as a tracer that binds to activated mesenchymal cells. The ^{68}Ga-FAPI PET computed tomography scan revealed prominent patchy tracer uptake in the muscles of the proximal and distal parts of the extremities as well as the abdomen and the hips (**A-E**, red arrows). Increased ^{68}Ga-FAPI uptake has been reported in tissue responses associated with tumors, immunoglobulin G4–related disease, and juvenile polymyositis (1–3). This case demonstrates that substantial tissue remodeling is associated with adult NAM.

Written informed consent was obtained from the patient for the publication of any potentially identifiable images or data included in this article (standard ethics approval #157_20B and the translational arthritis research database [TARDA]). Patients or the public was not involved in the design, conduct, reporting, or dissemination plans of our research. The data supporting the conclusions of this article will be made available by the authors, without undue reservation. The authors thank Louis Schuster for his technical assistance.

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