Don't blame the bones!

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An 83-year-old woman who was under treatment for a community-acquired pneumonia presented with dehydration, generalized myalgia, proximal lower limb weakness and associated hypercalcaemia. A ^{99m}Tc HDP (hydroxymethylene diphosphonate) 600-MBq whole-body bone scan performed to exclude bony metastases demonstrated no bone lesions, but showed abnormal intense soft-tissue activity in the shoulder and pelvic girdle musculature, findings suggestive of a proximal myopathy. She had been treated with the antibiotics co-amoxiclav and clarithromycin for 14 days, and in addition was on a regular dose of statin (simvastatin) for hyperlipidaemia.

Clarithromycin is a macrolide antibiotic metabolized by the liver where it is also a potent inhibitor of cytochrome P450 (CYP)3A, the major enzyme responsible for the metabolism of hydroxymethylglutaryl coenzyme A reductase inhibitors such as simvastatin [1]. The concomitant use of clarithromycin and simvastatin has been reported to cause rhabdomyolysis and myopathy [2, 3]. This interaction may be worsened by dehydration and renal insufficiency. Her blood biochemistry confirmed this with creatine kinase levels (CK) raised to 25,544 U/l indicative of significant muscle injury. Skeletal muscle damage leads to tissue oedema and inflammation followed by an influx of calcium into the muscles [4]. Calcium levels in the tissues are strongly correlated with ^{99m}Tc HDP uptake which acts as a ligand adsorbing onto

tissue calcium [4]. This striking myopathic pattern of soft-tissue tracer uptake on the bone scan is explained on this basis.

Conflicts of interest None.

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