Quantitative T2 Maps in Idiopathic Inflammatory Myopathy: Correction for Fatty Involution of Muscle

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INTRODUCTION: In clinical MR settings, active muscle disease in patients with idiopathic inflammatory myopathy (IIM) is typically determined on STIR imaging. However, assessment of disease on STIR images is difficult to quantify. Some investigators have advocated the use of quantitative T2 maps to more objectively measure active muscle disease in IIM. Muscle damage in advanced or chronic IIM can result in fatty replacement of muscle, a process which may potentially confound the interpretation of mono-exponential T2 relaxation values. We illustrate the use of fat fraction (FF) measurements, derived by a two point Dixon method with T2* correction, to correct bulk muscle T2 values for fatty muscle infiltration.

METHODS: A standardized 1.5 Tesla screening MR survey for IIM was developed, consisting of the following axial multi-slice 2D acquistions through the thighs: 1) T1 spin echo, 2) fast spin echo STIR, 3) double echo gradient echo (GRE) at consecutive in and out-of-phase echo times (TR 350/ TE 2.2, 4.4), 4) double echo gradient echo at consecutive in-phase echo times (TR 350/ TE 4.5, 9), and 5) multi-echo spin echo (TR 2400/ TE 25, 50, 75, 100). Fat fraction maps were calculated from the in and out-of-phase GRE sequence as:

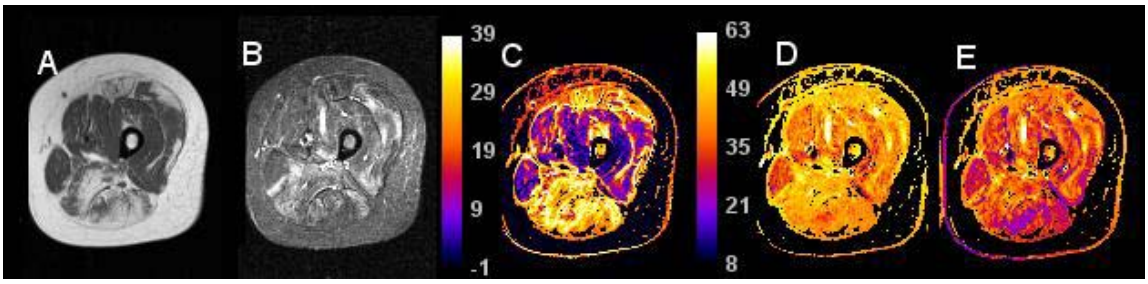
$$FF = [S(in) - S(out)] / 2*S(in)$$

where S(in) is corrected for T2* relaxation using the dual in-phase acquisition. For purposes of this study, muscle regions were determined by thresholding the GRE scans, rather than by a phase-based, fat-water separation procedure. Proton relaxation in muscle voxels was modeled as biexponential, with contributions from fat and muscle, where the signal for the nth echo in a multi-echo SE sequence is:

$$S(N) = S(0)* k* [r*FF *(\exp(-TEn/T2f)) + (1-FF)*(\exp (-TEn/T2m))]$$

T2f is the T2 relaxation of fat, and is assumed to be constant (52 msec). The proportional T1 and NH contributions to signal in the multi-echo SE sequence (r) for pure fat and pure muscle is based on a separate regional analysis of fat and non diseased muscle in 4 patients, and is also assumed to be constant (r =1.5). FF-corrected T2 (T2m) maps of muscle were generated by iterative, non-linear fitting of k and T2m to this model, where k is a scaling factor embodying hardware contributions to signal.

RESULTS: Sample images of the left thigh, from a patient with advanced polymyositis are shown below. T1 spin echo (A) shows muscle atrophy manifested by fatty involution of multiple muscles. STIR (B) demonstrates extensive inflammation involving atrophic and nonatrophic muscle. Fat fraction map (C) illustrates severe muscle damage. Mono-exponential T2 map (D) is quite different from FF-corrected T2 map (E), showing generally higher T2 values, and likely over-estimating disease in atrophic muscle regions, such as hamstrings in this case (color scales, in percent, are identical in D and E). In the atrophic but inflammed adductor magnus, standard and FF-corrected T2 values are 45.6 and 38.4 msec, while in non-atrophic and inflammed portion of vastus lateralis, standard and FF-corrected T2 = 42.6 and 38.9 msec, respectively.



DISCUSSION: Quantitative muscle T2 measurements may serve as a useful marker of active muscle disease in patients with IIM, particularly in the setting of clinical trials. Fat fraction can also be efficiently estimated in clinical MR surveys for myopathy, with the additon of two fast gradient echo scans. FF measurement may aid the quantification of muscle damage in IIM. More importantly, because the T2 of fat greatly exceeds that of healthy muscle, correction of muscle T2 values for FF can result in substantially different T2 results. The approach to muscle T2 measurement we have shown may improve the validity and utility of clnical T2 measurement as a objective, marker of active muscle disease in these patients.

REFERENCES:

Maillard SM, Jones R, Owens C, et al. Quantitative assessment of MRI T2 relaxation time of thigh muscles in juvenile dermatomyositis. Rheumatology 2004;43(5):603-8.