Clinical and Experimental Rheumatology 2016; 34: 1098-1100.

Do dermatomyositis and polymyositis affect similar thigh muscles? A comparative MRI-based study

N. Pipitone[1], A. Notarnicola[2], G. Levrini[3], L. Spaggiari[3], A. Scardapane[4], F. Iannone[2], G. Lapadula[2], P. Pattacini[3], G. Zuccoli[5], C. Salvarani[1]

[1]Department of Rheumatology, Department of Internal Medicine, Azienda Ospedaliera ASMN, IRCSS, Reggio Emilia, Italy;
[2]Department of Rheumatology, University of Bari, Italy;
[3]Department of Radiology, Department of Internal Medicine, Azienda Ospedaliera ASMN, IRCSS, Reggio Emilia, Italy;
[4]Department of Radiology, University of Bari, Italy;
[5]Department of Rheumatology, Children's Hospital of Pittsburgh, USA.

Nicolò Pipitone, MD, PhD
Antonella Notarnicola, MD
Gabriele Levrini, MD
Lucia Spaggiari, MD
Arnaldo Scardapane, MD
Florenzo Iannone, MD, PhD
Giovanni Lapadula, MD
Pierpaolo Pattacini,
Giulio Zuccoli, MD*
Carlo Salvarani, MD*

*These authors share senior authorship.

Please address correspondence to:
Dr Nicolo Pipitone,
Reumatologia,
Arcispedale S.M. Nuova,
Viale Risorgimento 80,
42100 Reggio Emilia, Italy.
E-mail: pipitone.nicolo@asmn.re.it

Received on September 14, 2015; accepted in revised form on January 18, 2016.

Key words: MRI, myositis

Competing interests: none declared.

ABSTRACT

Objective. *Dermatomyositis (DM) and polymyositis (PM) commonly cause weakness of the thigh muscles. However, it is debated whether DM and PM affect similar thigh muscles. Muscle oedema on fat-suppressed MRI sequences is thought to represent active inflammation. In this study, we aimed to assess which thigh muscle groups are preferentially inflamed in DM and PM, respectively, using short-tau inversion-recovery MRI sequences.*

Methods. *We analysed 71 patients from 2 Rheumatology centres, 31 with DM and 40 with PM diagnosed according to the Bohan and Peter criteria. MRI oedema (1=present, 0=absent) was assessed bilaterally on fat-suppressed sequences in 17 pelvic floor and thigh muscles. An MRI oedema score (range 0-17) was calculated by adding the separate scores bilaterally and dividing them by two. Inter-rater variability was assessed by intraclass correlation coefficient. Fisher's exact test was used to compare binomial data.*

Results. *Age and gender ratio were similar in patients with DM and PM. Disease duration (months, mean±SD) was shorter (20±31) in DM than in PM (53±69) (p=0.02). The intraclass correlation coefficient between the radiologists involved was 0.78. Muscle oedema was more common in DM than in PM except in the posterior thigh muscles. In particular, 68% of patients with DM had involvement of at least one anterior thigh muscle versus 38% of patients with PM (p=0.02).*

Conclusion. *Compared with PM, DM affects more thigh muscles, except those of the posterior compartment, which are equally involved in both disorders. These findings may be useful to target physiotherapy at the more frequently affected muscles.*

Introduction

The idiopathic inflammatory myopathies (IIM) include dermatomyositis (DM) and polymyositis (PM) (1, 2). DM differs from PM in terms of pathogenesis, histological features and the presence of a typical skin rash (3), while both disorders are characterised by muscle weakness, myopathic changes on EMG, and elevated serum muscle enzymes (1). However, while clinically overt muscle weakness has a proximal distribution in both conditions, subtle differences have been noted in the pattern of the muscles involved using whole-body (4) and thigh (5-7) MRI. The aim of this study was to define the pattern of inflamed thigh muscles in patients with DM and PM using short tau inversion recovery (STIR) MRI sequences.

Methods

We retrospectively studied 71 patients from 2 Rheumatology centres, 31 with DM and 40 with PM diagnosed according to the Bohan and Peter criteria. PM was confirmed by consistent histological features in all cases. Histological features deemed consistent with PM included the presence of lymphocytes attacking non-necrotic muscle fibres or an endomysial lymphocytic infiltrate in the absence of red-rimmed vacuoles and a negative immunohistochemistry for muscle dystrophies, especially dyspherlinopathies.

Myositis was considered active if there was significant (grade 4 or less of the Medical Research Council scale) muscle weakness not explained by chronic damage, progressive worsening of muscle strength, a creatine kinase raised at least twice the upper limit of normal or a histology showing inflammatory features. Muscle weakness of 4 or less in at least four proximal muscle groups was considered in any case a prerequisite for defining active myositis.

Three magnets were utilised for this study (1 Tesla and 1.5 Tesla for centre A and 1.5 Tesla for centre B). Multiplanar multiecho MRI sequences including T1, T2, PD and STIR images were obtained. The technical factors regarding STIR sequences are as follows:

• 1 Tesla magnet for centre A (INTERA Philips 11.8 using 4 channel phase array body coil [Sense Body]) with the following parameters:

Axial STIR: Slice thickness 6 mm, FOV: 350-450 mm, Acquisition matrix 304 (Scan percentage 80%), Reconstruction matrix 512, TE: 55 ms, TR: 2500, Inversion Time: 150 ms, NSA:3, Sense factor no, Tse Factor 12. Coronal STIR: Slice thickness 6

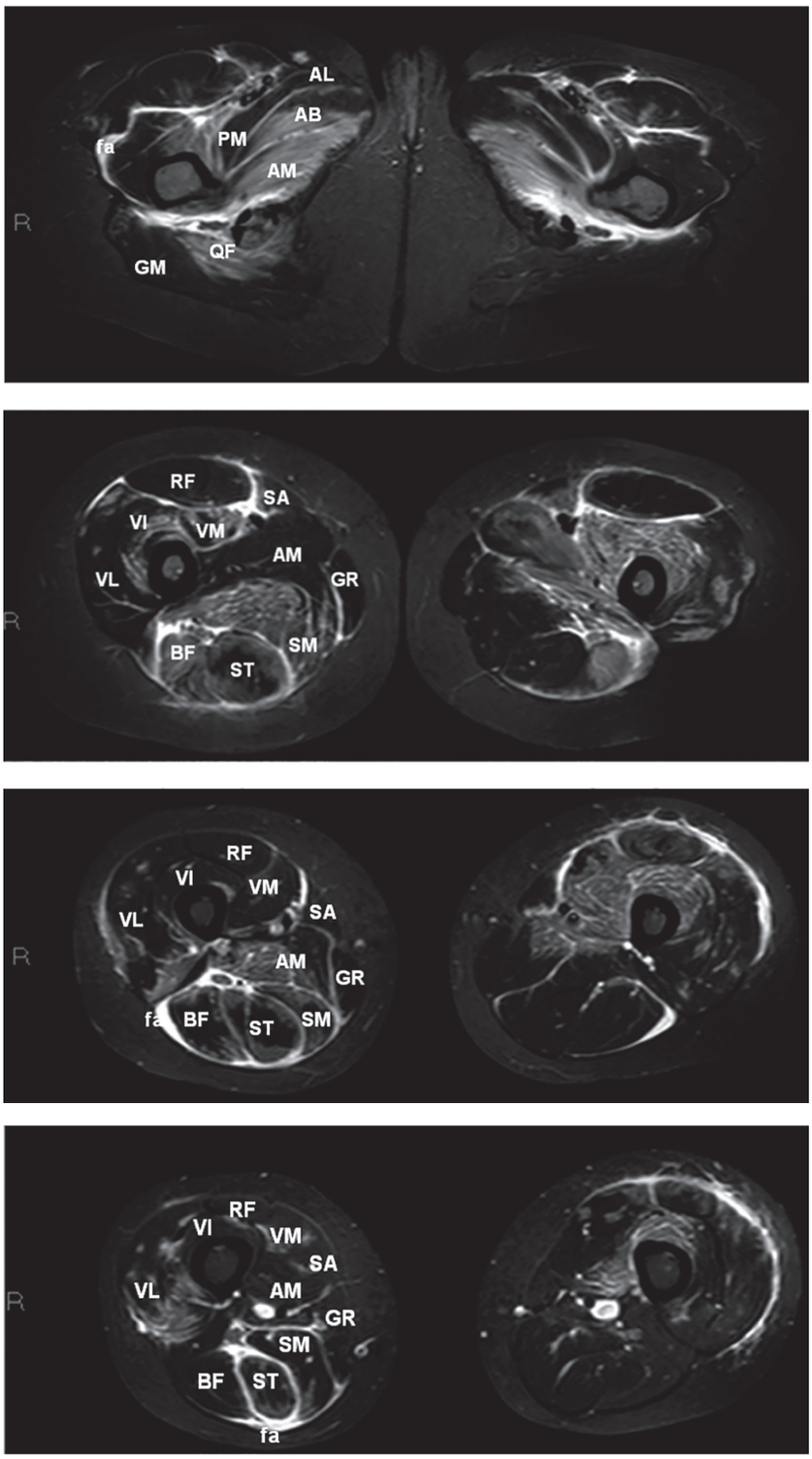

Fig. 1. Axial views (from top to bottom) of an MRI (short tau inversion recovery sequences) of the pelvic floor/thigh muscles showing bilateral diffuse oedema of nearly all muscle groups in a patient with DM. The MRI oedema score of this patient was 16.
AB: adductor brevis; AL: adductor longus; AM: adductor magnus; BF: biceps femoris; fa: fascia; GM: gluteus maximus; GR: gracilis; PM: pectineus muscle; QF: quadratus femori; RF: rectus femoris; SA: sartorius; SM: semimembranous; ST: semitendinous; VI: vastus intermedius; VL: vastus lateralis; VM: vastus medialis.

mm, FOV: 350–450 mm, Acquisition matrix 304 (Scan percentage 80%), Reconstruction matrix 512 TE: 55 ms, TR: 2500 ms, Inversion Time: 150 ms, NSA: 3, Sense factor no, Tse Factor 12;
• 1.5 Tesla magnet for centre A (Achieva Philips 2.6 using 16 channel phase array body coil [Sense Torso XL]).
Axial STIR: Slice thickness 6 mm, FOV: 350-450 mm, Acquisition matrix 336 x 299 (Scan percentage 80%), TE: 60 ms, TR: shortest, Inversion Time: 150 ms, NSA: 2, Sense factor 1.5
Coronal STIR: Slice thickness 4 mm, FOV: 350–450 mm, Acquisition matrix 336x265 (Scan percentage 80%), Reconstruction matrix 528 TE: 60 ms, TR: Shortest Inversion Time: 150 ms, NSA: 3, Sense factor 1.5.
• 1.5 Tesla magnet for centre B (Achieva Philips 1.8) using a 4-channel phase array body coil (Sense body).
Axial STIR: Slice thickness 7–10 mm, FOV: 350–450 mm, Acquisition matrix 320x400 (Scan percentage 80%), TE: 60 ms, TR: Shortest, Inversion Time: 150 ms, NSA: 2, Flip angle 90°, Sense factor 3.Coronal STIR: Slice thickness 4–6 mm, FOV: 350-450 mm, Acquisition matrix 320x400 (Scan percentage 80%), TE: 60 ms, TR: Shortest Inversion Time: 150 ms, NSA: 2, Flip angle 90°, Sense factor 3. MRI oedema (1 = present, 0 = absent) was assessed bilaterally on STIR sequences in 17 thigh/pelvic floor muscles.
An MRI composite oedema score (range 0–17) devised by one of the authors (GZ) was calculated by adding the separate scores bilaterally and dividing them by two as described elsewhere (8). A representative image of an MRI exam with the relative score is shown in Figure 1. Inter-rater variability was assessed by (single measures) intraclass correlation coefficient (ICC). Fisher's exact test was used for comparison of binomial data. Paired *t*-test was used to compare between-group continuous variables. Significance was set at $p<0.05$.
As this study was purely observational, ethics committee approval was not required according to local regulations.

Results

Age (years, mean±SD) was similar in patients with DM (53±16) and PM (57±15). The F:M ratio was similar in DM (23/8) and PM (31/9). Disease duration (months, mean±SD) was shorter (20±31) in DM than in PM (53±69) (p=0.02). The difference in disease duration was driven by 4 outliers in

the PM group with a duration greater than 190 weeks; when the analysis was performed without including these 4 subjects the difference was no longer significant (p>0.05). 26 (84%) patients with DM and 36 (90%) patients with PM were judged to have active disease (p=0.5). 23 (74%) patients with DM and 28 (70%) patients with PM were taking glucocorticoids (p=0.8), while 17 (55%) patients with DM and 25 (63%) patients with PM were taking immunosuppressants (p=0.6).

The ICC between the Radiologists involved was 0.78.

Muscle oedema was more common in DM than in PM except in the posterior thigh muscles (Table I). In particular, 68% of patients with DM had involvement of at least one anterior thigh muscle *versus* 38% of patients with PM (p=0.02).

Table I. Prevalence of involvement of thigh muscle groups in DM and PM (number and (%) of patients with muscle oedema). Axial muscles are those belonging to the pelvic floor, while the remaining muscles are those of the thighs.

Muscles	Compartment	DM (n=31)	PM (n=40)	p-value
Gluteus maximus	axial	17 (55%)	13 (33%)	0.09
Quadratus femoris	axial	9 (29%)	1 (3%)	0.002
Vastus lateralis	anterior	15 (48%)	11 (28%)	0.09
Ileopsoas	axial	8 (26%)	3 (8%)	0.049
Vastus medialis	anterior	14 (45%)	10 (25%)	0.08
Tensor fasciae latae	anterior	12 (39%)	4 (10%)	0.009
Rectus femoris	anterior	16 (52%)	10 (25%)	0.03
Sartorius	anterior	13 (42%)	11 (28%)	0.2
Gracilis	medial	15 (48%)	8 (20%)	0.02
Pectineus	medial	8 (26%)	2 (5%)	0.02
Adductor longus	medial	9 (29%)	6 (15%)	0.2
Adductor brevis	medial	12 (39%)	5 (13%)	0.01
Adductor magnus	medial	10 (32%)	10 (25%)	0.6
Short head biceps femoris	posterior	10 (32%)	6 (15%)	0.1
Long head biceps femoris	posterior	12 (39%)	12 (30%)	0.5
Semimembranous	posterior	10 (32%)	8 (20%)	0.3
Semitendineous	posterior	14 (45%)	10 (25%)	0.08

Discussion

Both DM and PM share the clinical hallmark of proximal muscle weakness. However, studies that have used fat-suppressed MRI sequences to look at inflamed muscles have noted subtle differences in their respective patterns of muscle involvement (4, 6, 7). Herein, using STIR MRI sequences we have provided evidence that DM affects more frequently than PM most thigh muscles, except those of the posterior compartment, which are equally involved in both disorders. To our knowledge, this is the largest study aimed at specifically investigating the pattern of thigh muscle involvement in adult patients with myositis using MRI.

Our findings are consistent with previous reports suggesting a preferential involvement of the anterior thigh muscles in DM compared with PM (7, 9, 10). Specifically, the study by Reimers *et al*., which looked in detail at the pattern of thigh muscle involvement in 58 patients with myositis, demonstrated a higher degree of signal intensity in DM *versus* PM in the rectus femoris, vastus lateralis, sartorius, gracilis, semimembranous, semitendinous and biceps femoris (9), in broad agreement with our results. However, Reimers *et al*. defined signal hyperintensity as brighter signal on T2 sequences, while STIR sequences are currently preferred to T2 sequences, because both can visualise inflammatory muscle oedema, but a brighter signal on T2 may also be due to fat replacement of the muscles (11).

Although not validated in external cohorts, the score we used has been shown to have high inter-rater reproducibility, to correlate with muscle strength (12), and to be sensitive to change (13). Therefore, our findings are in our opinion robust enough and have also clinical significance. Knowledge about preferential muscle involvement in DM and PM can aid in targeting rehabilitation at the more affected muscles, which is well known to impart major benefit to patients with myositis (14). In addition, awareness of the respective patterns of muscle involvement in DM and PM might assist in differentiating the rare, but not exceptional, forms of DM without skin changes from PM (15), although muscle biopsy is still warranted to secure the diagnosis.

References

1. DEVERE R, BRADLEY WG: Polymyositis: its presentation, morbidity and mortality. *Brain* 1975; 98: 637-66.
2. BARSOTTI S, TERENZI R, LA PAGLIA GM, BELLUCCI E, BALDINI C, NERI R: One year in review 2015: idiopathic inflammatory myopathies. *Clin Exp Rheumatol* 2015; 33: 593-601.
3. MEDSGER TA, JR., ODDIS CV: Classification and diagnostic criteria for polymyositis and dermatomyositis. *J Rheumatol* 1995; 22: 581-5.
4. CANTWELL C, RYAN M, O'CONNELL M *et al*.: A comparison of inflammatory myopathies at whole-body turbo STIR MRI. *Clin Radiol* 2005; 60: 261-7.
5. PORTER MJ: Temporal arteritis presenting as a submandibular swelling. *J Laryngol Otol* 1990; 104: 819-20.
6. CURIEL RV, JONES R, BRINDLE K: Magnetic resonance imaging of the idiopathic inflammatory myopathies: structural and clinical aspects. *Ann N Y Acad Sci* 2009; 1154: 101-14.
7. PARK JH, OLSEN NJ: Utility of magnetic resonance imaging in the evaluation of patients with inflammatory myopathies. *Curr Rheumatol Rep* 2001; 3: 334-45.
8. PIPITONE N, VERSARI A, ZUCCOLI G *et al*.: 18F-Fluorodeoxyglucose positron emission tomography for the assessment of myositis: a case series. *Clin Exp Rheumatol* 2012; 30: 570-3.
9. REIMERS CD, SCHEDEL H, FLECKENSTEIN JL *et al*.: Magnetic resonance imaging of skeletal muscles in idiopathic inflammatory myopathies of adults. *J Neurol* 1994; 241: 306-14.
10. PARK JH, VANSANT JP, KUMAR NG *et al*.: Dermatomyositis: correlative MR imaging and P-31 MR spectroscopy for quantitative characterization of inflammatory disease. *Radiology* 1990; 177: 473-9.
11. KUO GP, CARRINO JA: Skeletal muscle imaging and inflammatory myopathies. *Curr Opin Rheumatol* 2007; 19: 530-5.
12. PIPITONE N, NOTARNICOLA A, SCARDAPANE A *et al*.: Does MRI provide an added value over serum creatine kinase measurement in myositis? *Ann Rheum Dis* 2015; 74 (S2): 177.
13. PIPITONE N, NOTARNICOLA A, SPAGGIARI L *et al*.: Role of MRI in the follow-up of patients with myositis. *Ann Rheum Dis* 2015; 74 (S2): 831.
14. ALEXANDERSON H, LUNDBERG IE: Exercise as a therapeutic modality in patients with idiopathic inflammatory myopathies. *Curr Opin Rheumatol* 2012; 24: 201-7.
15. FIORENTINO D, CALLEN JP: Dermatomyositis. *In* WILLIAMS H, BIGBY M, DIEPGEN T, HERXHEIMER A, NALDI L, RZANY B (Eds.): *Evidence-based Dermatology*. John Wiley & Sons, 2009: 561.