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MRI patterns of thigh muscle involvement in immune-mediated necrotizing myopathy and dermatomyositis

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Abstract

Background Immune-mediated necrotizing myopathy (IMNM) and dermatomyositis (DM) are characterized by weakness, hyperCKemia, associated autoantibodies, and varying extramuscular manifestations. Muscle MRI, currently subordinate to histopathology and serology in idiopathic inflammatory myopathy (IIM) classification, has an evolving role. Our study aims to define thigh muscle MRI involvement in IMNM and DM by direct comparison.

Methods This single-center, retrospective, cross-sectional study included 25 participants, who met IIM classification criteria (14 IMNM, 11 DM) and had available thigh MRI. Clinical and paraclinical data were available and reviewed. 11 muscles were graded for edema on MRI using a semi-quantitative scale (0: normal, 1: <30% of muscle involvement, 2: 31–75%, 3: > 75%). For 3 participants with no significant muscle edema, muscle fatty infiltration was scored according to the same scale. Using linear mixed-effects models, muscle scores were compared between the two groups and a secondary analysis was performed of only edema scores, excluding the 3 participants with fatty infiltration scores.

Results The most affected muscles in IMNM were the semimembranosus (3.0 [2.7-3.0] {median [IQR]}), biceps femoris-long head (BF-LH) (2.7 [2.0–3.0]), and adductors (2.5 [2.0–3.0]). In DM, the most affected muscles were the vastus lateralis (2.7 [2.3-3.0]), vastus intermedius (2.9 [2.2-3.0]), vastus medialis (2.3 [1.7–2.7]), semitendinosus (2.2 [1.0-2.7]), rectus femoris (RF) (2.0 [1.0-2.8]), biceps femoris-short head (BF-SH) (1.9 [1.0-2.7]), gracilis, and sartorius. Intergroup statistical difference of scores was significant ($p < 0.01$) for 10/11 thigh muscles excluding the RF ($p = 0.19$), supporting an inverse relationship of muscle involvement for DM and IMNM. The secondary comparative analysis of only muscle edema scores was significant ($p < 0.05$) for the same 10/11 muscles with a consistent direction for all comparisons.

Conclusion DM and IMNM affect disparate thigh muscles on MRI. DM preferentially affects the anterior thigh, semitendinosus and BF-SH in the posterior thigh, and gracilis in the medial thigh, whereas IMNM preferentially affects the posterior thigh (semimembranosus and BF-LH) and adductors in the medial thigh.

Keywords Immune-mediated necrotizing myopathy, Dermatomyositis, Inflammatory myopathy, MRI, Classification

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Background

Idiopathic inflammatory myopathies (IIMs) are a heterogeneous group of immune-mediated disorders with evolving subtypes, comprising inclusion body myositis (IBM), dermatomyositis (DM), antisynthetase syndrome, immune-medicated necrotizing myopathy (IMNM), and polymyositis. IMNM is characterized by markedly elevated creatine kinase (CK) levels and often autoantibodies against HMG-CoA reductase (HMGCR) or signal recognition particle (SRP). Myofiber necrosis with absent or minimal lymphocytic infiltration, focal major histocompatibility complex (MHC) class I upregulation of myofibers, and patchy sarcolemmal and endomysial microvessel membrane attack complex deposition are characteristic on histopathology [1, 2]. DM manifests clinically with weakness, rash, and other potential extramuscular manifestations (e.g., polyarthritis, calcinosis, and interstitial lung disease). Muscle biopsies show perifascicular atrophy in addition to perivascular and perimysial inflammation. Myofiber MHC class I upregulation and myxovirus resistance protein A (MxA) staining are also characteristic, often with perifascicular accentuation [3]. Myofiber necrosis (particularly in Mi-2 seropositive cases) may be present [4]. Indeed, differentiation of IIM subytpes is not always possible on clinical grounds (e.g., NXP2 seropositive dermatomyositis sine dermatitis [5] and anti-HMGCR myopathy with dermatomyositis-like rash [6]), whereas histopathology separates the various forms of IIM and is integral to their classification [7].

MRI is a useful imaging technique for evaluation of soft tissues and can readily demonstrate muscle edema, atrophy, and fatty infiltration as well as fascial edema. It is operator independent and can visualize a large field including superficial and deep structures [8]. MRI has a well-established role in guiding muscle biopsy site selection in myositis and may represent a cost-effective approach [9]. As it stands, there is inconsistent inclusion of imaging in classification criteria for IIM. IBM represents an exception: pathognomonic ultrasonographic and MRI features (reflective of clinically evident deep finger flexor and quadriceps involvement) have been reported [10, 11], which have resulted in formal incorporation of supportive ultrasonographic and MRI criteria for IBM classification [12].

Previous studies have investigated the MRI features of DM and IMNM. In DM, MRI shows symmetric proximal inflammation of the shoulder and hip girdle [8]. A study involving whole body MRI noted that DM was associated with patchy inflammation on MRI, posited to reflect a patchy distribution of ischemic damage [13]. Fascial involvement is often widespread in DM [14]. A recent study demonstrated prevalent involvement of the lumbar paraspinal muscles, gluteus medius and minimus, adductor magnus, and hamstrings in IMNM [15]. A large systematic MRI study demonstrated that fatty infiltration tends to be mild in DM and PM and a prominent, early feature of IMNM [14]. This study found that fatty infiltration was more prominent in anti-SRP(+) than anti-HMGCR(+) IMNM.

To date, there have been few studies that have systematically evaluated and compared the forms of IIM directly on thigh muscle MRI. The aim of our study is to characterize the MRI features of IMNM and DM by direct comparison, which may contribute to a more robust classification for IIM, involving noninvasive parameters.

Methods

25 participants, who were seen as patients in the Oregon Health & Science University (OHSU) myositis clinic from 2016-present, were included. Serology was performed on all patients and muscle biopsy in the majority (omitted in a minority of seropositive cases). 14 participants met European Neuromuscular Centre (ENMC) classification criteria [16] for IMNM and 11 met European Alliance of Associations for Rheumatology and American College of Rheumatology (EULAR/ACR) classification criteria [17] for DM. Thigh MRI had been performed for clinical purposes. In all cases except participants 1 and 3 (Table 1), imaging was performed prior to initiation of immunomodulatory therapy. 18 patients had bilateral thigh MRIs, and 7 patients had unilateral MRIs. A total of 43 unilateral MRIs of the thigh were analyzed: 23 in the IMNM group and 20 in the DM group. We did not include antisynthetase syndrome patients, since MRI was only available for 3 patients. IBM and polymyositis patients were excluded.

Anti-HMGCR antibody testing was performed by enzyme-linked immunosorbent assay (RDL Reference Laboratory, Los Angeles, CA). Reference values are defined by the performing lab as follows: negative, <20 U/mL; weak positive, 20–39 U/mL; moderate positive, 40–59 U/mL, and positive, >59 U/mL. Comprehensive Myositis Autoantibody Profile, which includes Jo-1, PL-7, PL-12, EJ, OJ, Mi-2, SRP, PM/ScL, Ku, U1RNP, U2RNP, Ro60, TIF1-γ, NXP2, MDA5 and SAE autoantibodies, was performed by S35-immunoprecipiation, RNA-immunoprecipitation, and immunoblotting (Oklahoma Medical Research Foundation, Oklahoma City, OK).

We obtained an OHSU Institutional Review Board (IRB) approval waiver. Our study met criteria for a waiver of informed consent as outlined by the Common Rule (45 CFR 46.116(f)). The following de-identified data were collected by chart review: duration of symptoms prior to MRI acquisition, age at MRI acquisition, history of immunomodulatory prior to MRI, CK level, muscle biopsy site, histopathological findings, autoantibody results, and muscle involvement (Table 1). Thigh MRIs had been performed with a standardized protocol of coronal and

Table 1 Demographics, clinical features, and diagnostic results of participants

n	Group	Dz dur. ā MRI acqn (m)	Age (y), MRI acqn	Tx, ā/ p̄ MRI acqn	Sex	CK (U/L)	Muscle Bx site	Histopathologic features	Ab	MRI seq.
1	IMNM	206	56	ā (17 y)	M	19,699	R VL	moderate MN, no I	SRP	T1W
2	IMNM	1	66	p̄	F	19,734	L quads	severe MN, mild perim. I	SRP	STIR
3	IMNM	74	46	ā (6 y)	F	4694	ND	--	SRP	T1W
4	IMNM	2	70	p̄	F	12,218	L quads	moderate MN, no I	HMGCR	STIR
5	IMNM	27	68	p̄	F	3403	ND	--	HMGCR	T2FS
6	IMNM	2	74	p̄	M	3188	ND	--	SRP	STIR
7	IMNM	3	67	p̄	F	15,530	R quads	mild MN, no I	HMGCR	STIR
8	IMNM	7	52	p̄	F	10,160	L RF	moderate MN, no I	SRP	STIR
9	IMNM	2	65	p̄	F	18,244	ND	--	HMGCR	STIR
10	IMNM	33	70	p̄	M	12,073	L deltoid	severe MN, no I	HMGCR	T1W
11	IMNM	4	72	p̄	F	6017	L quads	moderate MN, no I	HMGCR	T2FS
12	IMNM	2	78	p̄	M	11,025	L biceps	severe MN, no I	HMGCR	T2FS
13	IMNM	3	60	p̄	M	19,856	L quads	mild MN, no I	HMGCR	T2FS
14	IMNM	25	52	p̄	F	964	L deltoid	mild MN, no I	HMGCR	T2FS
15	DM	2	58	p̄	F	1535	L biceps	PA, MxA, MHC-1, moderate perim. I	TIF1-γ	T2FS
16	DM	10	49	p̄	F	178	L deltoid	PA, MxA, MHC-1, no I	SAE	T2FS
17	DM	2	73	p̄	F	4679	R thigh	PA, MHC-1, mod MN, mild perim. I	Mi-2	STIR
18	DM	0.5	43	p̄	F	6985	R thigh	PA, MxA, MHC-I, moderate MN, no I	NXP2	STIR
19	DM	1	65	p̄	F	15,242	L deltoid	PA, MxA, MHC-1, muscle infarct, perim. I	MSA/ MAA(-)	T2FS
20	DM	1	80	p̄	M	10,922	R VL	MxA, MHC-1, moderate MN, no I	NXP2	STIR
21	DM	3	68	p̄	M	7800	R thigh	PA, MxA, MHC-I, severe, MN, no I	Mi-2	T2FS
22	DM	2	73	p̄	F	710	ND	--	TIF1-γ	STIR
23	DM	6	9	p̄	F	273	ND	--	TIF1-γ	T2FS
24	DM	3	42	p̄	F	853	L quads	PA, MxA, MHC-1, mild perim. I	SAE	T2FS
25	DM	3	38	p̄	F	83	L quads	PA, MxA, MHC-1, mild perim. I	TIF1-γ	STIR

ā, before; Ab, antibody; acqn, acquisition; Bx, biopsy; CK, creatine kinase; DM, dermatomyositis; dur., duration; Dz, disease; HMGCR, HMG-CoA reductase; I, inflammation; IMNM, immune-mediated necrotizing myopathy; L, left; m, months; MHC-1, major histocompatibility complex class 1 myofiber upregulation; MAA, myositis-associated autoantibodies; MN, myofiber necrosis; MSA, myositis-specific autoantibodies; MxA, myxovirus resistance protein A staining; ND, not done; p̄, after; PA, perifascicular atrophy; perim., perimysial; quads, quadriceps; R, right; seq., sequence; SRP, signal recognition particle; STIR, short tau inversion recovery; T1W, T1 weighted; T2FS, T2 fat saturated; Tx, treatment; VL, vastus lateralis; y, years

axial T_1-weighted (T_1W) and T_2-weighted with/without fat suppression or short tau inversion recovery (STIR) images. MRI slices at the midthigh, a standardized level that captures the largest cross-sectional area of muscle [18, 19], were independently evaluated and graded by the director of the OHSU Advanced Imaging Research Center (WR), the director of the OHSU myositis clinic (NC), and a neuromuscular specialist with training in neuromuscular imaging (AW). Muscle edema, which correlates with inflammation [20] and myofiber necrosis [21] on muscle biopsy, was graded on T_2-weighted fat-saturated (T_2FS) or STIR images in all except 3 IMNM participants (1,3, and 10; Table 1), whose MRIs showed minimal to no abnormality on T_2-weighted images. Rather, on T_1W images in these 3 cases, there was significant muscle fatty infiltration, which was graded. MRI grades for fatty infiltration or edema were assigned using a modification of Fischer's scale [22] as follows: 0: normal, 1: <30% muscle involvement, 2: 31–75%, 3: > 75%.

Unilateral muscles were given discrete scores. Weighted Fleiss κ and percent agreement, acknowledging the strengths and limitations of each [23], were used to evaluate interrater reliability among the 3 independent raters. The average of the 3 raters' muscle scores was calculated to generate a final score for 11 midthigh muscles. 43 scores per muscle were generated from our study sample: 23 in the IMNM group and 20 in the DM group. Intragroup muscle score mean, median, and interquartile range (IQR) were calculated for each of the 11 muscles. To mitigate potential bias from multiple within-subject measures (i.e., bilateral MRI images), a linear mixed effects model was conducted using R (R Core team, 2024, version 4.4.1) including lmerTest package (Kuznetsova et al., 2017) to compare pooled muscle scores (11 individual muscle comparisons) in the IMNM group to those in the DM group. A second analysis was performed using only muscle edema scores (excluding the data from the 3 IMNM participants with muscle fatty infiltration on T_1W images) with otherwise constant parameters. The

Benjamini-Hochberg procedure was used to account for multiple comparisions (α = 0.05).

Results

In the IMNM group (average age at presentation: 61.8 +/- 12.2 y, 64.3% female), 5 participants had anti-SRP antibodies, and 9 participants had anti-HMGCR antibodies (Table 1). The average CK level was 11,200+/-6738 U/L. All biopsied samples confirmed myofiber necrosis ranging from mild to severe. Thigh MRI was performed at bimodal time points after symptom onset. For the majority of participants, it was performed within a year of symptom onset and for the remainder of participants, it was performed more than a year after symptom onset (for 2 cases more than 5 years). MRI showed that the most affected muscles (intragroup) were the semimembranosus (3.0 [2.7-3.0] {median [IQR]}), biceps femoris-long head (BF-LH) (2.7 [2.0–3.0]), and adductors (2.5 [2.0–3.0]) in both anti-SRP and anti-HMGCR seropositive patients (Fig. 1). There was relative sparing (intragroup) of the semitendinosus, sartorius, vastus lateralis (VL), vastus medialis (VM), biceps femoris-short head (BF-SH), and gracilis muscles in both antibody subgroups.

In the DM group (average age at presentation: 54.4 +/-20.7 y, 81.8% female), 2 participants were anti-Mi-2(+), 4 were anti-TIF1-γ(+), 2 were anti-SAE(+), 2 were anti-NXP2(+), and 1 was seronegative (Table 1). The average CK level was 4478+/-5159 U/L. All biopsied samples confirmed pathologic hallmarks of DM, including perifascicular atrophy, MxA staining, and MHC class I upregulation. Thigh MRI was performed within a year of symptom onset in all patients. The most affected muscles on MRI in this group were the VL (2.7 [2.3-3.0]; mean: 2.6), vastus intermedius (2.9 [2.2-3.0]; mean: 2.5), VM (2.3 [1.7–2.7]), semitendinosus (2.2 [1.0-2.7]), rectus

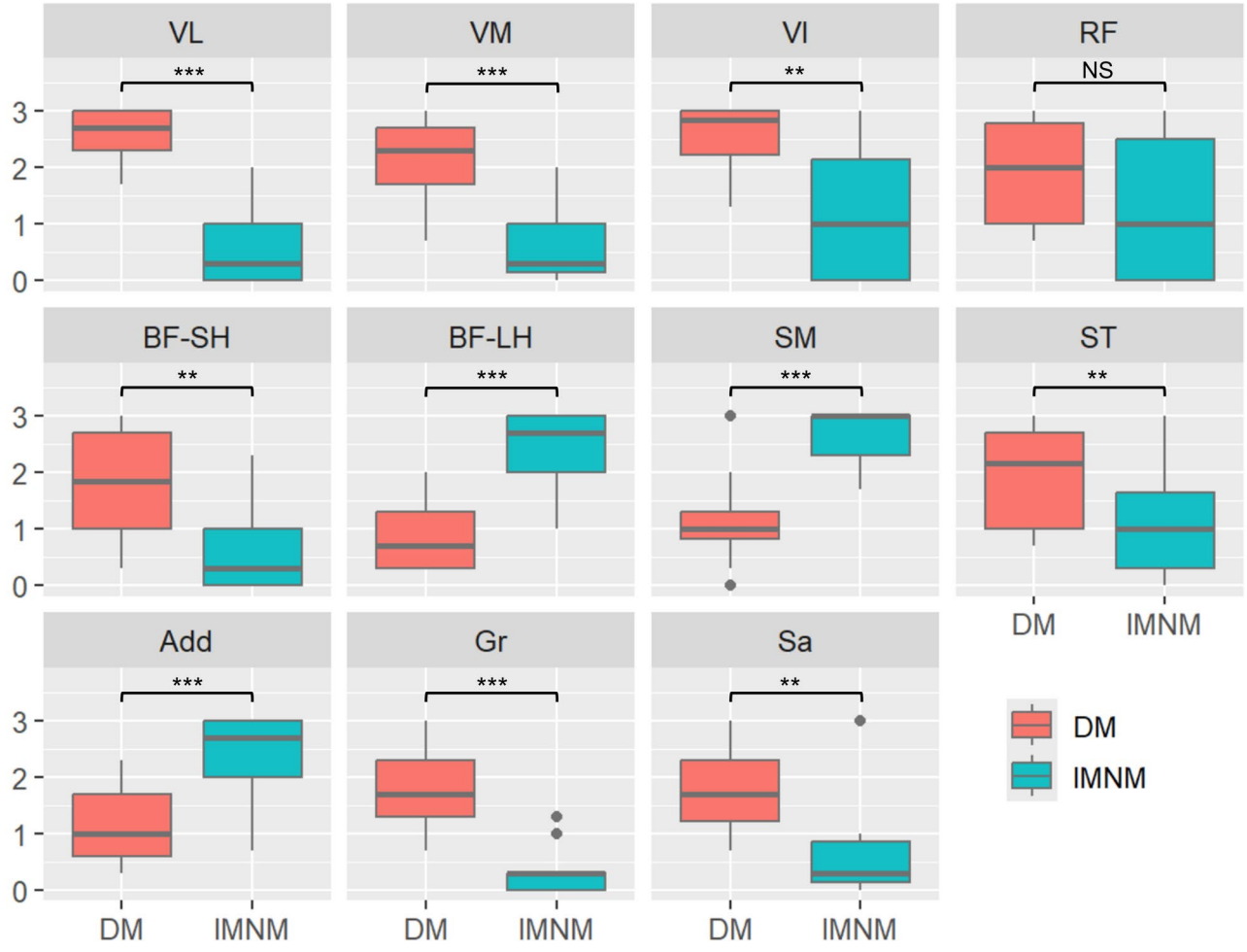

Fig. 1 Muscle grading of thigh MRIs in dermatomyositis (DM) and immune-mediated necrotizing myopathy (IMNM). Vastus lateralis (VL), vastus medialis (VM), vastus intermedius (VI), rectus femoris (RF), biceps femoris-short head (BF-SH), biceps femoris-long head (BF-LH), semimembranosus (SM), semitendinosus (ST), adductors (Add), gracilis (Gr), and sartorius (Sa) MRI scores (0–3, y-axis) demonstrated as boxplots (vermilion, DM; teal, IMNM), which include median, interquartile range, and outliers (dots). Linear mixed effects model comparison of pooled individual thigh muscles scores between DM and IMNM indicated by significance bars. ***, p-value < 0.001. **, p-value < 0.01. NS, not significant

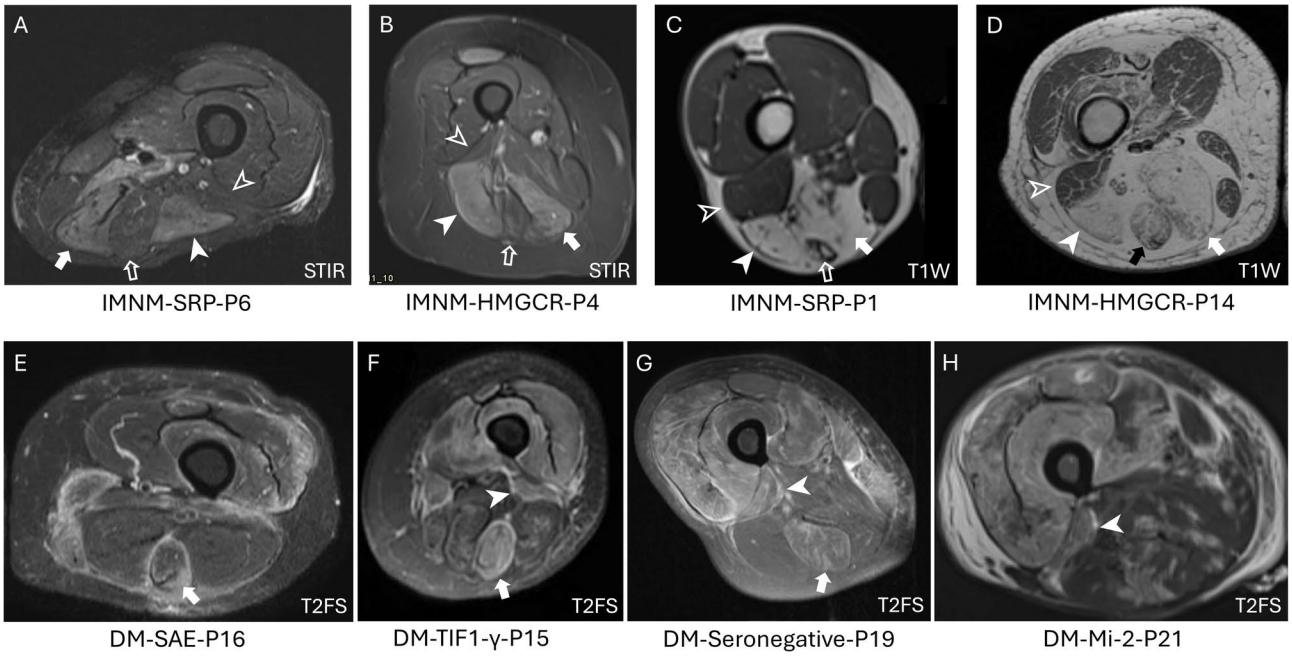

Fig. 2 Representative thigh MRIs of immune-mediated necrotizing myopathy (IMNM) and dermatomyositis (DM). (**A, B**) Muscle edema of selective posterior compartment muscles (semimembranosus [arrow] and biceps femoris-long head [BF-LH] [arrowhead], sparing the semitendinosus [open arrow] and biceps femoris-short head [BF-SH] [open arrowhead]) on short tau inversion recovery (STIR) images in IMNM cases with shorter disease duration. (**C, D**) On T1-weighted (T1W) images, complete fatty replacement of the semimembranosus (arrow) and BF-LH (arrowhead) with persistent sparing of the BF-SH (open arrowhead) in IMNM cases with prolonged disease duration. The semitendinosus is relatively spared (open arrow, C) or has undergone significant fatty replacement (black arrow, D). (**E-H**) Muscle and fascial edema of anterior compartment muscles, semitendinosus (arrow), and BF-SH (arrowhead) on T2-weighted fat-saturated (T2FS) images in DM

femoris (RF) (2.0 [1.0-2.8]), BF-SH (1.9 [1.0-2.7]), gracilis (1.7 [1.3–2.3]), and sartorius (1.7 [1.2–2.3]) (Fig. 1). There was relative sparing of BF-LH, semimembranosus, and adductors.

There was moderate interrater agreement (Weighted Fleiss $\kappa = 0.554$) and 83% percent agreement among independent scorers. The difference of graded scores between IMNM and DM participants was statistically significant for 10 of 11 muscles (Fig. 1), compatible with an inverse relationship of thigh muscle involvement on MRI. The RF was the sole muscle similarly affected in both groups ($p = 0.19$). Reanalysis including only muscle edema scores (excluding muscle fatty infiltration scores for 3 IMNM participants) was significant ($p < 0.05$) in the same muscles with a consistent direction for all comparisons (Additional file 1). The qualitative inverse pattern of muscle involvement in these two disorders is readily apparent (Fig. 2).

Discussion

The histopathological and serological characterization of IIM have contributed to classification criteria that clearly separate subtypes. On MRI, we have shown that IMNM affects the muscles in the posterior and medial thigh compartments with selective individual muscle involvement of the semimembranosus and BF-LH and adductors. DM affects the anterior compartment and gracilis in addition to the semitendinosus and BF-SH, with relative sparing of posterior thigh muscles prominently affected in IMNM. Notably, specific patterns of MRI involvement have been demonstrated in muscular dystrophies—a recent machine-learning approach to pattern recognition of muscle involvement on MRI proved to be highly accurate in the identification of the genetic basis of various muscular dystrophies based on the distribution of affected muscles [24]. Inferential analysis of the MRI pattern of selective muscle involvement in our study reached significance to discriminate IMNM from DM. A patchy pattern of MRI muscle involvement (i.e., quadriceps involvement in the case of DM and hamstring and adductor involvement in the case of INMN [13–15]) was corroborated by our study, but we have additionally discretely indexed individual muscle involvement (including selective involvement or sparing of muscle heads within the same muscle), which increases the diagnostic specificity of muscle MRI in evaluating IIM and separating subtypes.

Indeed, our study was designed specifically to evaluate the *distribution* of muscle involvement on MRI. Nonetheless, prominent fascial edema (Fig. 2E and F arrows) in DM and prominent fatty infiltration (the predominant MRI finding for several participants [Fig. 2C and D]) in

IMNM corroborates previously reported characteristic features of these disorders [13–15]. It is noteworthy that these pathological processes occurred in muscles similarly affected by muscle edema in our study sample. This is supported by similar results on reanalysis of only muscle edema scores (excluding data points for the 3 IMNM participants with significant fatty infiltration on T_1W MRI). Muscle edema and fatty infiltration, though not interchangeable, likely co-occur or occur in sequence in the same muscles in IMNM. We felt it important to include participants with predominantly muscle fatty infiltration in the primary analysis given fatty infiltration is an important feature of IMNM and a feature that may be misattributed to muscular dystrophy [25]. We acknowledge the potential contribution of prolonged disease duration and/or treatment effect to the prominent fatty infiltration and absence of muscle edema on thigh MRI in the abovementioned 3 IMNM participants, and thus, we similarly felt it important to perform a reanalysis excluding the data from these participants to remove such confounding variables.

The pathomechanism accounting for the specific pattern of muscle involvement in these two IIM subtypes is not known. We speculate that for DM, the muscles involved are reflective of the distribution of fascia (prominently affected in DM [14]) in the midthigh. Namely, the fascia lata is thickest along the lateral thigh as it overlies the VL, the most affected muscle in our DM sample. The lateral intermuscular septum, the thicker of the thigh's intermuscular septae, separates the VL from the BF-SH and partially contributes to the muscles' origin. Additionally, the gracilis, sartorius, and semitendinosus are contiguous with crural fascia as their tendons coalesce into the pes anserinus. Differential distribution of target antigenic expression may also play a role. When considering IMNM, the semimembranosus and BF-LH have been shown to provide the majority of force applied by the hamstrings during active knee flexion, which may be influenced by muscle architecture [26]. This characteristic may render these muscles more susceptible to muscle breakdown in IMNM.

We acknowledge that muscle MRI will not likely supplant muscle biopsy, serological testing, and above all, clinical reasoning in the evaluation of IIM. MRI is, however, noninvasive, widely availability, and provides at-the-ready results, in contradistinction to serological testing with variable processing time and specificity depending upon assay. The rationale for muscle MRI as a complementary diagnostic tool for IIM is predicated on its potential for speedy diagnostic confirmation of clinical suspicions in straightforward cases and its utility in challenging cases, represented in our study sample by participants with over two years of ongoing symptoms before correct diagnosis and appropriate treatment.

Our retrospective study has several limitations. The moderate sample size precluded direct comparison of thigh MRI in patients with anti-HMGCR IMNM to those with SRP seropositivity. Our study did not evaluate whole body MRI as others have [13], which may be a perceived limitation. This, however, was by design as our interest was in evaluating a pattern that may be identified on thigh MRI, which is pursued in clinical practice. Similarly, midthigh muscles (viz., as opposed to hip muscles captured on thigh MRI) were evaluated due to the large muscle mass and number of individual muscles at this level [18], which would be expected to have a higher power to detect a difference between groups. Due to our study's retrospective design, definitive statements on the temporal relationship of muscle MRI findings to symptom onset cannot be made. Furthermore, inferences cannot be made from our study about thigh MRI as a biomarker of treatment response or disease activity.

Conclusion

Our retrospective, cross-sectional study involving thigh MRI delineates a distinct pattern of thigh muscle involvement in IMNM compared to that in DM. Although our findings should be confirmed in additional studies, these patterns may further partition the subtypes of IIM, contribute to their classification, and aid diagnosis.

Abbreviations

ACR	American college of rheumatology
BF-LH	Biceps femoris-long head
BF-SH	Biceps femoris-short head
CK	Creatine kinase
DM	Dermatomyositis
ENMC	European neuromuscular centre
EULAR	European alliance of associations for rheumatology
HMGCR	HMG-CoA reductase
IBM	Inclusion body myositis
IIM	Idiopathic inflammatory myopathy
IMNM	Immune-mediated necrotizing myopathy
IRB	Institutional review board
MHC	Major histocompatibility complex
MxA	Myxovirus resistance protein A
OHSU	Oregon Health & Science University
RF	Rectus femoris
SRP	Signal recognition particle
STIR	Short tau inversion recovery
T_1W	T1-weighted
T_2W	T2-weighted
VL	Vastus lateralis
VM	Vastus medialis

Supplementary Information

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Supplementary Material 1

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Not applicable.

Author contributions
All authors contributed to the study conception and design. Material preparation was performed by KMV, NC, AWW, and DG. Data collection was performed by WDR, NC, and AWW. Analysis was performed by DC, AWW, and NC. The first draft of the manuscript was written by AWW and KMV, and all authors commented on previous versions of the manuscript. NC supervised the conduction of the study and review and editing of the manuscript. All authors read and approved the final manuscript.

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Data availability
The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the Declaration of Helsinki and met criteria for waiving informed consent as outlined by the Common Rule (45 CFR 46.116(f)). Ethical approval was waived by the Oregon Health & Science University Institutional Review Board in view of the retrospective nature of the study and given all the procedures being performed were part of the routine clinical care of the participants.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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