# Magnetic Resonance Imaging Criteria for Distinguishing Between Inclusion Body Myositis and Polymyositis

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***ABSTRACT.*** ***Objective.*** To develop diagnostic imaging criteria for polymyositis (PM) and sporadic inclusion body myositis (sIBM).

***Methods.*** We investigated 220 patients with suspected inflammatory myopathies by magnetic resonance imaging (MRI). Findings were compared with the results of clinical and biological examinations and muscle biopsy. PM and IBM were diagnosed in 25 patients each. Quantitative and qualitative MRI analysis of the 3 muscle groups of the 2 thighs included fatty infiltration, atrophy, inflammation, and the type and distribution of the lesions.

***Results.*** MRI was abnormal in all patients. Fatty infiltration and atrophy were more frequent in patients with sIBM ($p < 0.05$). Inflammation as the sole abnormality was preferentially encountered in PM ($p = 0.05$). Widespread abnormalities were more frequent in sIBM ($p < 0.01$). Abnormalities in PM tended to be distributed along the fascia. Involvement of the anterior group, an asymmetrical distribution, and a distal predominance were all more frequent in sIBM ($p < 0.001$).

***Conclusion.*** Despite some overlap in MRI findings between the 2 diseases, MRI was useful for distinguishing PM from sIBM. (J Rheumatol 2002;29:1897–906)

*Key Indexing Terms:*
MAGNETIC RESONANCE IMAGING DIAGNOSTIC CRITERIA
POLYMYOSITIS INCLUSION BODY MYOSITIS

Polymyositis (PM), dermatomyositis (DM), and sporadic inclusion body myositis (sIBM) are idiopathic inflammatory myopathies (IIM). These disorders are characterized histologically by endomysial inflammation, but have distinct immunologically mediated pathogenic mechanisms[1,2]. IIM is classically diagnosed on the basis of the history, physical findings, serum creatine kinase (CK) concentrations, electromyography, and muscle biopsy. IBM can be mistaken for PM because of various clinical, biochemical, electromyographic, and immunopathologic similarities[1,3,4]. Histopathologic findings are also similar. The 2 conditions can be distinguished by the presence of muscle fiber vacuoles rimmed by granular material and by electron microscopic observation of intranuclear and cytoplasmic inclusion bodies in sIBM[5]. Unlike PM, sIBM is notoriously refractory to corticosteroids, standard immunosuppressive therapies, intravenous immunoglobulin, and plasmapheresis[3,6]. The potential adverse effects of these drugs mean that it is essential to distinguish between the 2 forms as early as possible.

Magnetic resonance imaging (MRI) can detect inflammation and fatty infiltration in IIM[7-13]. Fat saturated spin echo T2 weighted sequences and short tau inversion recovery (STIR) sequences are best suited to visualizing inflammatory modifications[9,10,14-16], but high signal intensity modifications on such sequences are not specific for IIM[17,18]. MRI signs of inflammation, atrophy, and fatty infiltration have different distributions among the different muscles, predominating in the proximal or distal part of the thighs[10,16,19-21]. The few reports of MRI characteristics of sIBM[10,12,22] have described certain distinctive findings[12], but no comparative MRI studies of sIBM and PM have been performed.

We prospectively compared muscle MRI findings in patients with PM and s-IBM. Inflammatory changes, fatty infiltration, and the type and distribution of abnormalities on T2-weighted and contrast sequences were compared with clinical data.

## MATERIALS AND METHODS

*Patients.* Between 1995 and 2000, 220 consecutive patients with various myopathies underwent MRI examination at our institution. This study was

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approved by the local ethical committee. Sixty-four consecutive adults with PM or sIBM diagnosed between April 1995 and January 2000 were eligible for analysis. Fourteen patients with PM or sIBM were excluded because their MRI data were incomplete. Of the remaining 50 patients, 25 had PM and 25 had sIBM. There were 26 men and 24 women, with a mean age of 54.9 years (range 19–77). The diagnosis of PM was established according to Bohan and Peter[23]. SIBM was confirmed by light and electron microscopy.

Muscle strength was tested using the modified British Medical Research Council grading system[24], with a maximal score for each muscle of 11 points. The degree of muscle weakness was determined from the total score for 8 proximal muscles. The theoretical maximum strength score was 88. All patients were tested independently by specialists in neuromuscular diseases (PC, TP, and PG). Serum levels of CK were determined in the same laboratory with the same technique (normal < 110 U/l).

Diagnosis was based on histological and immunohistological studies performed on surgical muscle biopsies in the same pathology department, blinded to MRI data.

*MRI*. MR imaging was performed with a 1.5 T GE Medical System device equipped with a body coil. All the patients underwent spin-echo (SE) T1 weighted 440/11 [repetition time (TR) in ms/echo time (TE) in ms]. Patients underwent 2 types of T2 weighted sequences because of system improvements during the study. All patients had at least one of the T2 weighted sequences (STIR or fast T2 fat saturation). Forty-three patients underwent fast SE T2 weighted sequences (3400/102) with fat saturation; 24 patients underwent STIR sequences with TR 5500 ms, TE 34 ms, and inversion recovery time 150 ms (5500/34/150). Postcontrast fat saturation SE T1 weighted sequences were obtained within 5 min after intravenous injection of 0.1 mmol/kg gadolinium-DTPA in 35 patients. Axial 7 mm sections with a 15 mm gap were obtained from the iliac crest to the knee joint with a 512 × 256 matrix and a 42 × 42 cm field of view.

Quantitative signal intensity measurements using the built-in software system were performed on the 4 sequences. An operator defined region of interest (ROI) was chosen in homogeneous pathological areas identified by visual inspection in both the proximal and distal parts of the quadricepital, posterior, and adductor muscle groups. Six signal-intensity measurements (with standard deviations) were obtained per sequence, as well as noise measurements. A total of 24 values were obtained per patient.

*Qualitative analysis*. After training sessions, all images were read independently by 4 experienced radiologists (ED, EA, SP, and JCF) who were blinded to the clinical and histopathological data. The final evaluation was achieved by consensus between 2 of the readers. The 3 muscle groups were graded separately. The anterior muscle group consisted of the rectus femoris, vastus intermedius, vastus lateralis, and vastus medialis; the posterior muscle group consisted of the semitendinous muscle, semimembranous muscle, biceps femoris, and biceps longus; the adductor muscle group consisted of the gracilis, adductor, and sartorius muscles. Interrater agreement was calculated in a previous study using Cohen's kappa coefficient on 46 patients with PM, DM, or IBM[25]. The kappa coefficients of each sequence were as follows: SE T1 weighted sequences with fatty infiltration k = 0.93, fast T2 fat saturation k = 0.90, STIR k = 0.89, SE T1 postcontrast fat saturation k = 0.90.

Five groups of MRI criteria were studied:

1. Fatty infiltration was defined as areas of signal intensity equivalent to subcutaneous fat on T1 weighted images. The abnormalities were graded using a 5 point scale, as follows: grade 0 = no abnormalities, grade 1 = abnormalities affecting less than 25% of the muscle, grade 2 = 25% to 50%, grade 3 = 50% to 75%, grade 4 = 75% to 100%. Ten muscles were studied, and the maximum total extension score was thus 40 per patient.
2. Changes in muscle cross sectional area were defined subjectively by comparison with the opposite side and with other muscle groups, by drawing an oblique line through the posterior aspect of the anterior group and the posterior aspect of the femur. Tested in 10 healthy subjects, this line separated the thigh into 2 roughly equal parts.
3. Inflammatory involvement was graded separately on T2 weighted sequences, STIR sequences, and postcontrast sequences. Areas of high signal intensity on T2 weighted fat saturation sequences were considered to represent inflammation. On postcontrast images, inflammatory areas were defined by a gradient of more than 40 points in signal intensity between pre- and postcontrast images. The extent of inflammation was graded using the same 5 point scale as for fatty infiltration.
4. Pattern of involvement: 0 = normal muscle, 1 = doubtful involvement of the muscle, 2 = nodular aspect, 3 = signal intensity changes along the fascia, 4 = large area of signal intensity changes (> 1/3 of the muscle circumference).
5. Location of the abnormalities: (a) Frequency of the abnormalities (by muscle and muscle group). (b) The symmetrical nature of abnormalities on the 2 thighs was graded separately for inflammation and fatty infiltration on 3 consecutive slices (0 = symmetrical, 1 = mildly or moderately asymmetrical, 2 = very asymmetrical) and for each muscle group (quadricepital, adductor, and posterior). Asymmetry was defined by changes on signal intensity > 25% between the 2 thighs on 3 consecutive slices. (c) The distal or proximal predominance of the abnormalities was evaluated for each muscle group and for the 4 sequences (0 = no predominance, 1 = proximal, 2 = distal).

Correlations were sought between fatty infiltration in contrast to disease duration, age, steroid treatment, and muscle strength scores; and also between inflammation compared to CPK levels and muscle strength scores.

*Statistical analysis*. Quantitative values were compared between the 2 diagnostic groups by Student t test or the Mann-Whitney test. Categorical measures were compared with the Pearson chi-square test, with Yates' correction if necessary, or Fisher's exact probability test for small samples. Correlations were calculated using the Spearman correlation coefficient. Results are expressed as means ± SD. Differences were considered significant at $p < 0.05$.

## RESULTS

### Clinical findings (Table 1)

The sIBM group comprised 17 men and 8 women. All fulfilled the usual diagnostic criteria for sIBM, with characteristic muscle histology[6]. None had a history of malignancy or intercurrent connective tissue disorders. The mean age was 60.8 ± 13.0 years. The average interval between the first symptoms of sIBM and diagnosis was 43.1 ± 37.8 months. The mean CK level at the time of MRI studies was 584 ± 182 IU/l. The mean muscle strength score was 63.0 ± 8.3.

The PM group comprised 16 women and 9 men. All fulfilled the usual diagnostic criteria for PM[23,26]. None had a history of malignancy or intercurrent connective tissue disorders. Mean age was 49.1 ± 12.5 years ($p < 0.05$ vs the

*Table 1*. Characteristics of the patients.

| | PM, N = 25 | IBM, N = 25 |
|---|---|---|
| Age, yrs | 49.1 ± 12.5 | 60.8 ± 13.0 |
| Male/female | 9/16 | 17/8 |
| Mean CPK, IU/l | 3329.2 | 584.9 |
| Mean strength score | 65.04 | 63.02 |
| Time to diagnosis, mo | 9.9 | 43.12 |
| Patients treated with | | |
| Corticosteroids, 5–60 mg/day | 24 | 20 |
| Methotrexate | 9 | 3 |
| Intravenous immunoglobulin | 8 | 15 |
| Azathioprine | 3 | 2 |

sIBM group). Eight patients had esophageal disorders (proximal dysphagia or uncoordinated peristalsis due to striated muscle dysfunction). Raynaud's disease was present in 3 patients. Visceral involvement included 5 cases of pulmonary fibrosis (one anti-Jo1 syndrome) and 3 cases of cardiomyopathy (one of dilated cardiomyopathy and 2 of conduction abnormalities). The average interval between the first symptoms of PM and diagnosis was 9.9 ± 15.0 months. The mean CK level was 3329 ± 1238 IU/l. The mean muscle strength score was 65.0 ± 11.6.

MRI findings (Table 2)

All the patients had MRI abnormalities consisting of fatty infiltration, atrophy, and/or inflammation.

*Fatty infiltration*. Signal intensity measurements on T1 weighted images showed a significant difference between the PM and sIBM groups ($p < 0.001$) in both the proximal (210 ± 143 vs 394 ± 215, respectively) and distal muscle groups (279 ± 177 vs 452 ± 160). In the distal part of the adductor group, signal intensity in all sequences showed a wide range of values owing to the absence or small cross sectional area of the adductor muscle at that level. These values were therefore excluded from the statistical analysis.

Extent of fatty infiltration: Fatty infiltration was found in at least one muscle in 41 patients (17 PM, 24 sIBM). Nine patients had no fatty infiltration (8 PM, one sIBM). The degree of fatty infiltration in the anterior group was higher in sIBM than in PM: 19 sIBM and 5 PM patients were graded 3 or 4 ($p < 0.001$), and 9 PM and 5 sIBM patients were graded 1 or 2. The mean total score for the extent of fatty infiltration was 15 ± 6 in PM patients and 22 ± 7 in sIBM patients ($p < 0.001$). Fatty infiltration was found exclusively in the quadriceps muscle in 10 sIBM and one PM patient ($p < 0.001$), while involvement of all the muscles was similarly frequent in the 2 groups (11 PM, 11 sIBM).

Distribution of fatty infiltration: Fat infiltration was widespread in 22 sIBM (Figure 1) and 7 PM patients ($p < 0.0001$), and was predominant in the fascia in 9 patients (7 PM, 2 sIBM; $p = 0.05$). Fatty infiltration was asymmetrical between the 2 thighs in 12 cases (11 sIBM, one PM; $p < 0.001$). Asymmetrical involvement of the anterior group was significantly more frequent in patients with sIBM (11 sIBM vs one PM; $p < 0.001$). Asymmetrical involvement was seen in the posterior muscle group of 6 sIBM and 2 PM patients (NS), and in the adductor group of 5 sIBM and 3 PM patients. Fat infiltration was predominantly distal in 16 sIBM patients and 3 PM patients ($p < 0.001$). The grade of fat infiltration differed within a given muscle group in 30 patients (17 sIBM and 13 PM). No specific muscle was significantly more frequently involved in one of the 2 conditions.

*Atrophy*. Thirty-nine patients (23 sIBM and 16 PM) had atrophy of at least one muscle. Atrophy was seen in the ante-

*Table 2*. Comparative MRI evaluation of PM and s IBM. All data are percentages.

| | Patient Group | | |
|---|---|---|---|
| | PM (n = 25) | IBM (n = 25) | p |
| Fatty infiltration | | | |
| Presence of fatty infiltration | 68 | 96 | < 0.03 |
| Anterior group exclusively | 4 | 40 | 0.0021 |
| Anterior group. Grade 3 or 4 | 20 | 76 | 0.001 |
| All 3 groups involved | 44 | 44 | NS |
| Fascial pattern | 28 | 8 | NS |
| Widespread pattern | 28 | 88 | 0.0001 |
| Asymmetrical | 4 | 44 | 0.0009 |
| Distal predominance | 12 | 64 | 0.0002 |
| Atrophy | | | |
| Presence of atrophy | 64 | 92 | 0.02 |
| Anterior group atrophy | 16 | 60 | 0.0014 |
| Undulating fascia | 32 | 64 | 0.02 |
| Inflammation | | | |
| Presence of inflammation | 72 | 72 | NS |
| Inflammation in posterior group | 32 | 8 | 0.03 |
| Fascial pattern | 40 | 20 | NS |
| Widespread pattern | 16 | 44 | 0.03 |
| Asymmetrical | 8 | 10 | NS |
| Postcontrast images | PM (n = 19) | IBM (n = 16) | |
| Contrast uptake in anterior group | 74 | 88 | NS |
| Contrast uptake in posterior group | 53 | 31 | NS |
| Global analysis | PM (n = 25) | IBM (n = 25) | |
| Isolated inflammation | 20 | 0 | 0.05 |

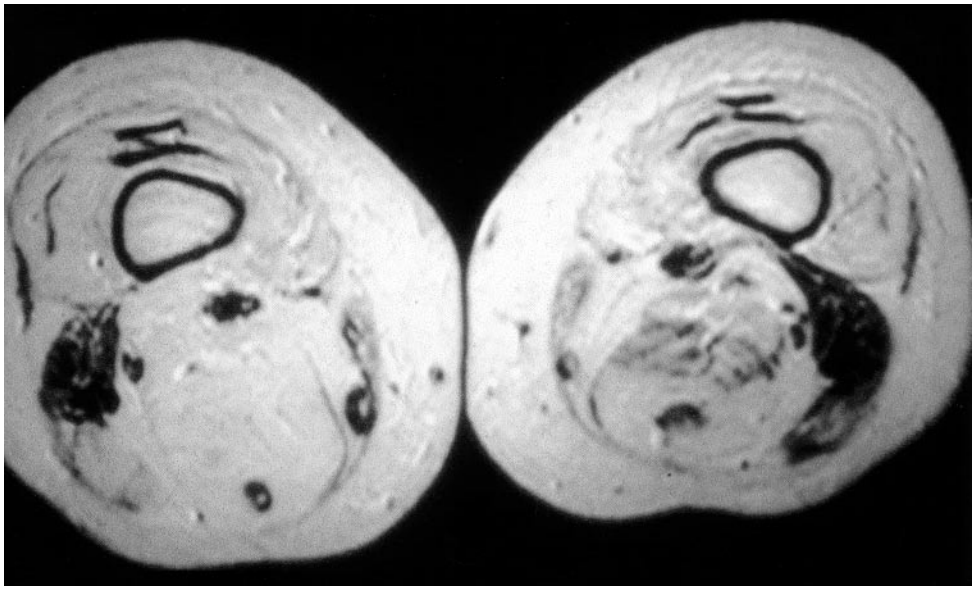

*Figure 1*. Axial SE T1 weighted image through the midsection of the 2 thighs in a patient with a 70 month history of sIBM. Note the diffuse fatty infiltration of the quadricepital and adductor groups, and the partial involvement of the posterior group (the biceps is preserved). This pattern of involvement is referred to in the text as widespread. The cross sectional area of the muscles is unchanged.

rior group in 15 patients with sIBM and 4 with PM ($p < 0.001$). There was no significant difference between the 2 diagnostic groups regarding overall frequency of atrophy, involvement of the posterior or adductor muscle group, or heterogenous involvement of muscles within a given muscle group.

We observed a previously unreported feature, in which the fascia draws an undulating line between the atrophic vastus intermedius and vastus intermedialis (Figure 2). This sign was present in the quadricepital muscle group of 24 patients (16 sIBM and 8 PM; $p < 0.05$) (Figure 3). Its presence correlated with the grade of atrophy: it was present in 12/14 patients graded $\geq$ 3 for atrophy, and absent in all 14 patients graded 0 ($p < 0.05$).

*Inflammation (Figure 4).* Distribution: Inflammation was found in 36 patients (18 PM and 18 sIBM). A slight difference was seen between the 2 diagnostic groups regarding involvement of the posterior group on fast T2 fat saturation sequences (8 PM vs 2 sIBM; $p < 0.05$).

Pattern: Fast T2 weighted images showed fascial inflammation in 10 PM and 5 sIBM patients (NS) and widespread inflammation in 4 PM and 11 sIBM patients ($p < 0.05$). A similar distribution was seen on postcontrast images. Asymmetry was slightly more frequent in the anterior group than in the other muscle groups in sIBM patients, on both fast T2 and postcontrast sequences (2 PM vs 5 sIBM; NS).

STIR, fat saturation fast T2, and postcontrast sequences (Figures 5 and 6): On postcontrast images, signal intensity in both the distal and proximal portions of the anterior group was higher in sIBM than in PM. Five patients were excluded from signal intensity analyses because fat saturation was inadequate. The proportion of patients with signs of inflammation was similar in the PM and sIBM groups. Contrast uptake was seen in the anterior group of 14 PM and 14 sIBM patients, and in the posterior group of 10 PM and 5 sIBM patients (NS).

Areas of contrast enhancement were superimposed on areas of signal hyperintensity on STIR and fast T2 fat saturation images.

*Combined data*. Isolated inflammation, without fatty infiltration or atrophy, was noted in 5 PM and no sIBM patients ($p = 0.05$). Fatty infiltration, atrophy, and inflammation were found simultaneously in 16 sIBM and 6 PM patients ($p < 0.05$).

Correlations between MRI and other findings

Among the 24 patients with grade 3 or 4 fatty infiltration, the mean disease duration was significantly shorter in the 5 patients with PM than in the 19 patients with sIBM, 57 vs 91 months ($p < 0.001$). Nine PM and 5 IBM patients were graded 1 or 2 for fatty infiltration, with a mean disease duration of 38 and 60 months, respectively. The differences disappeared when we compared mean scores in patients with a disease duration of less than 4 years (7 IBM and 18 PM patients); both groups had a mean score for fat infiltration of 4.

Correlations between the extent of inflammation on MRI and muscle biopsy were not tested, because the interval between biopsy and MRI exceeded one month in most cases. CK levels did not correlate with the presence of

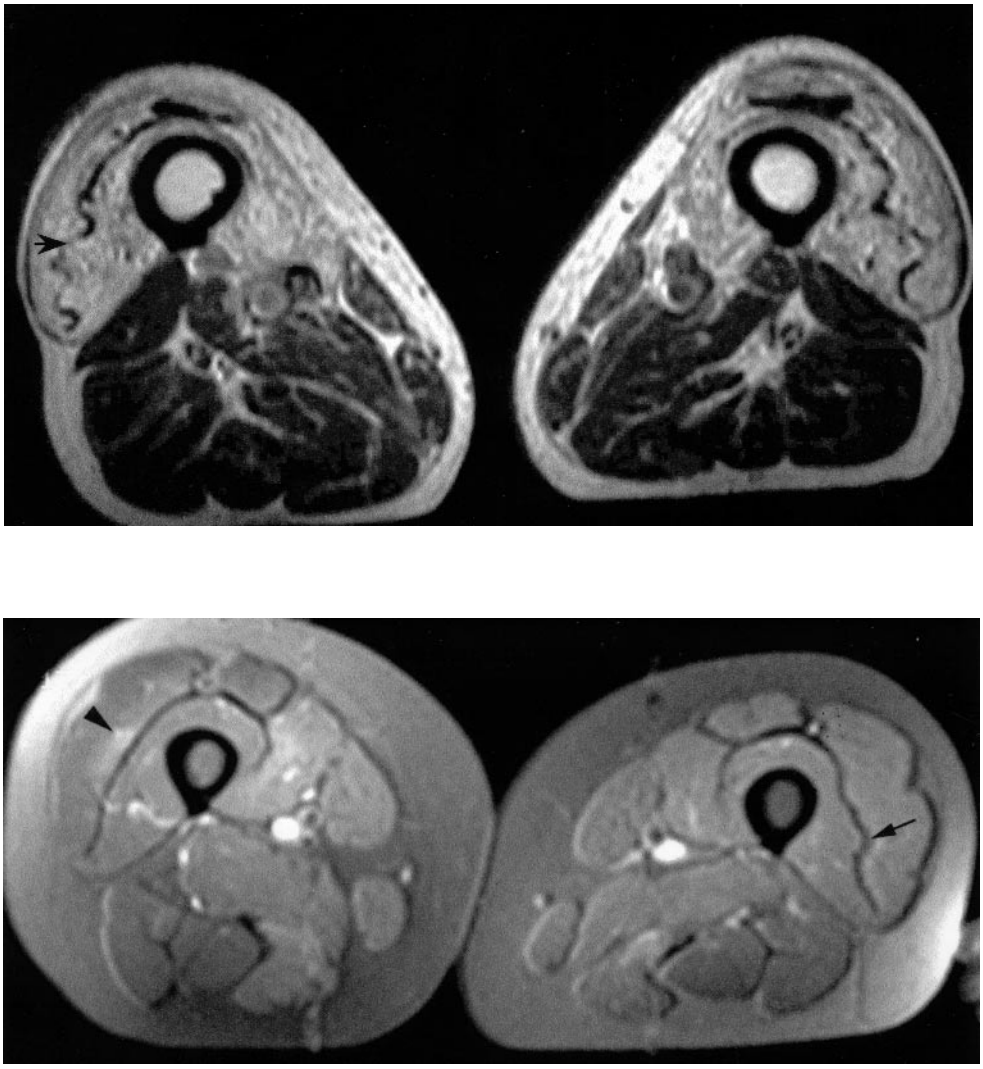

*Figure 2*. A. Axial SE T1 weighted image through midsection of the 2 thighs in a patient with sIBM. Note diffuse fatty infiltration throughout the quadricepital muscles. The fascia between the vastus intermedius and the vastus lateralis appears as an undulating line (arrow). B. Axial SE T1 weighted image after contrast injection with fat saturation (PM onset 11 mo previously). Left: contrast uptake is visible in the quadricepital group (arrowhead); muscle volume is preserved and the fascia describes a curved line. Right: slight muscle volume loss is visible, and the curve described by the fascia is somewhat irregular (arrow).

atrophy or muscle inflammation on MRI. CK levels showed a negative correlation with the extent of fatty infiltration in both diagnostic groups (PM, r = 0.535 and IBM, r = 0.469; $p < 0.001$). CK levels correlated negatively with muscle strength in patients with PM (r = –0.226), but not in sIBM ($p < 0.001$). No correlation was found between muscle strength and the presence of fatty infiltration, inflammation, or atrophy on MRI. The cumulative dose of steroid therapy was not different in the PM and sIBM groups. No difference in the cumulative dose was found between patients with grade 3 or 4 fatty infiltration and patients with no fatty infiltration, or between patients with and without atrophy.

## DISCUSSION

MRI has proven valuable for detecting muscle abnormalities in various forms of idiopathic inflammatory myopathy[4,9,17,22,26-31]. In particular, it was more sensitive than muscle biopsy for detecting disease activity (89% vs 66%). Although not as specific for IIM as muscle biopsy (88% vs 100%), MRI has a positive predictive value equivalent to that of biopsy (97% vs 100%) and a better negative predictive value (64% vs 38%)[12].

We prospectively analyzed and compared muscle MRI findings in patients with PM and sIBM. We precisely assessed fatty infiltration and inflammation/edema in each

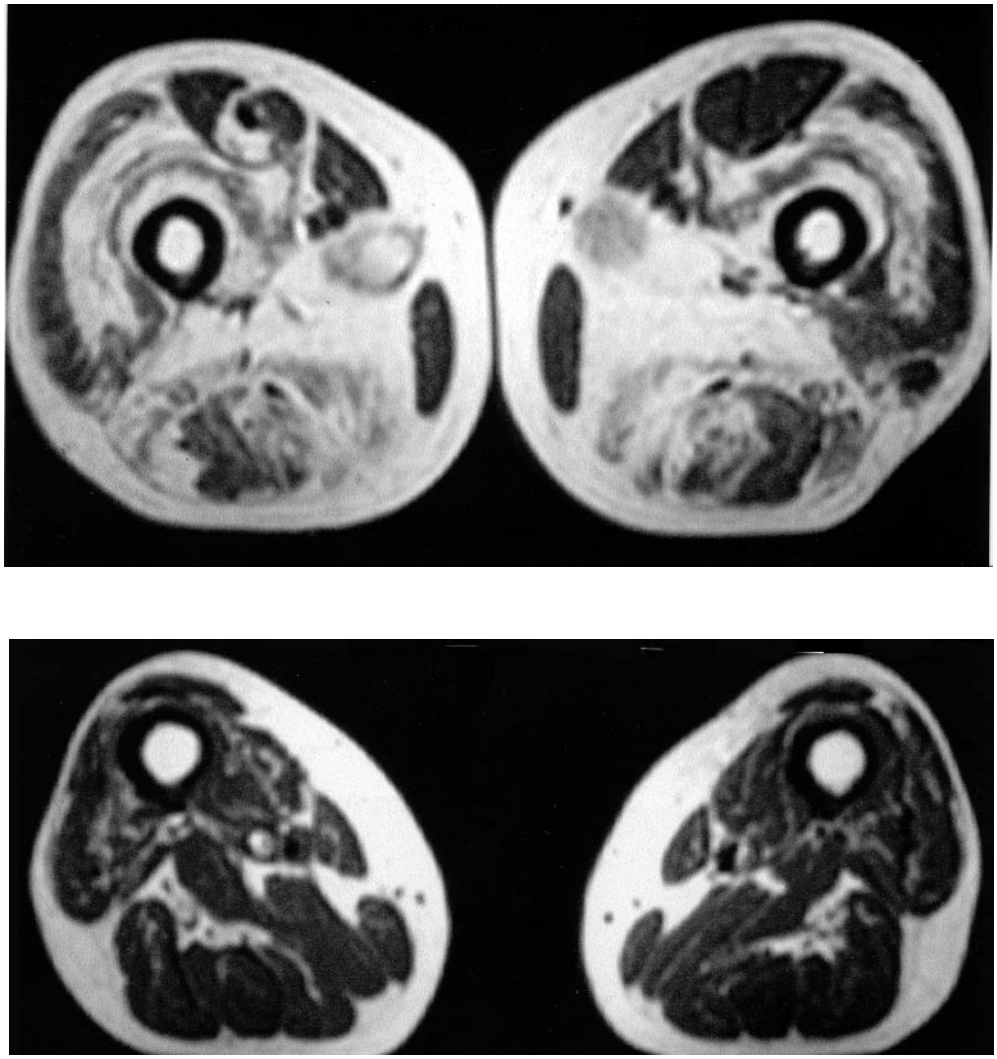


*Figure 3*. Atrophy and fatty infiltration have different MRI aspects. Axial SE T1 weighted images of the thighs in a patient with PM. A. Fatty infiltration is present in all the muscle groups with predominance along the fascia. B. SE T1 weighted image in a patient with IBM. Atrophy of the quadricepital group is visible, with very slight fatty infiltration.

form of myositis and in each thigh muscle. We also measured signal intensities, graded atrophy, and described the pattern of abnormalities, including their symmetric/asymmetric and distal/proximal distribution.

We found MRI abnormalities (fatty infiltration and/or atrophy and/or inflammation) in all the patients in this series. Sufficient differences were found between PM and sIBM (Table 2) to make MRI an element in the diagnosis in patients with IIM, even though definite diagnosis is based on histopathological findings of muscle biopsy. In particular, PM was characterized by isolated inflammation (frequently involving the posterior muscle group), a fascial pattern of involvement, and symmetrical lesions. In contrast, sIBM was characterized by more frequent fatty infiltration, the presence of all 3 lesion types (fat, atrophy, and inflammation), asymmetrical involvement between the 2 thighs, a distal predominance, anterior group involvement, and widespread lesions.

The fatty infiltration score was higher in patients with sIBM than in those with PM, but not when the disease duration was taken into account. The time course of fatty infiltration is thus apparently the same in the 2 diseases, but MRI investigations are generally carried out sooner in PM (mean 38 months after onset in this study) than in sIBM (85 months). Our results show that inflammation and fatty infiltration can coexist in the same patient, and sometimes in the same muscle, years after disease onset (Figures 6 and 7).

Inactivity and steroid treatment could have contributed to

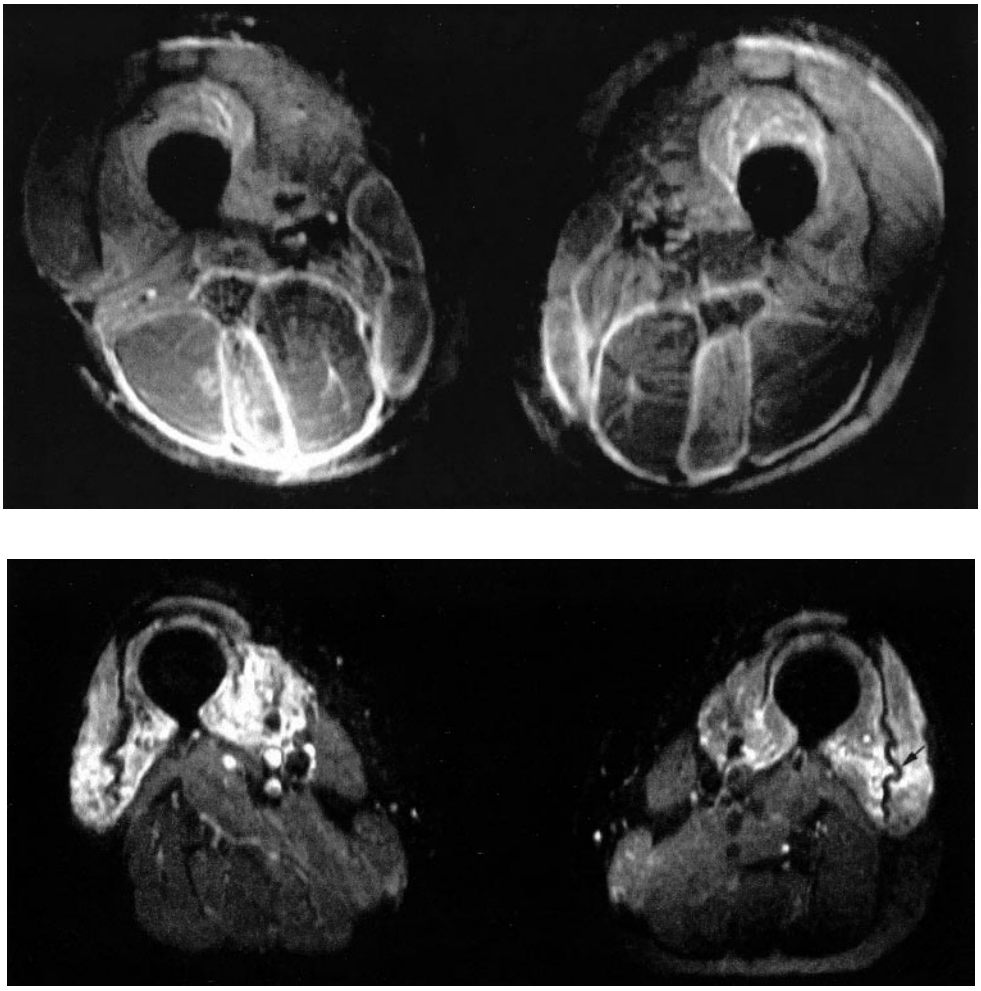


*Figure 4*. Axial STIR image through the midsection of the thighs in a patient with PM. A. Inflammatory infiltrates are visible as high signal intensity lines along the fascia, predominating in the posterior muscle group. B. Axial STIR image through midsection of thighs in a patient with a few months' history of IBM. Inflammation involves the quadricepital group exclusively. Slight atrophy is also visible, with an undulating fascia. Posterior and adductor groups are normal.

the progression of atrophy and/or fatty infiltration[8], but the treatments received by the 2 groups of patients prior to MRI were comparable.

We did not confirm the preferential involvement of particular thigh muscles reported by Reimers, *et al* in sIBM[10], although some patients had a single involved muscle with a particular group.

We used all 3 sequences (STIR, gadolinium contrast, and fat suppression) to grade the inflammatory involvement in 24 patients, and found no significant difference in their sensitivity. The value of postcontrast images is thus questionable in this setting.

One particularly interesting finding was the undulating nature of the fascia between the vastus intermedius and vastus lateralis in patients with significant muscle volume loss (Figures 2 and 3). This sign has not previously been described, and studies are under way to determine if it is characteristic of acquired muscle atrophy.

In sum, atrophy, fatty infiltration, and anterior group involvement are characteristic of inclusion body myositis, whereas isolated inflammation and global or posterior involvement are characteristic of polymyositis. This study shows that MRI helps distinguish between PM and sporadic inclusion body myositis, even in their later stages.

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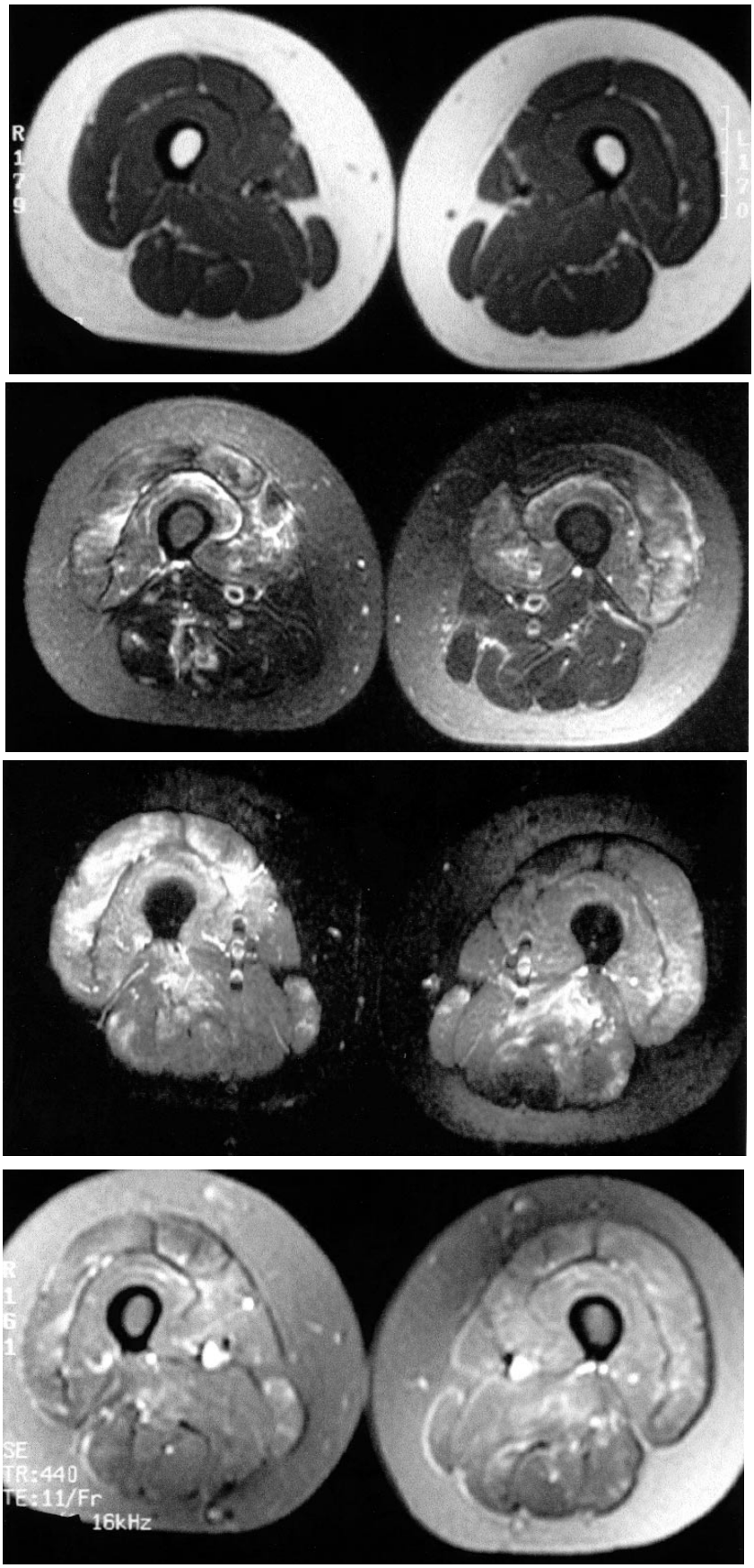


*Figure 5*. Axial images through midsection of the thighs in a patient with a 40 month history of PM. A. SE T1 weighted image, B. fast SE T2 weighted image with fat saturation, C. STIR image, D. fat saturation SE T1 postcontrast image. Muscle volume is preserved, and no fatty infiltration is visible on the SE T1 weighted image (A). Inflammation is mainly visible along the fascia, in the form of a hypersignal on the 3 sequences used to depict inflammation (B, C, D).

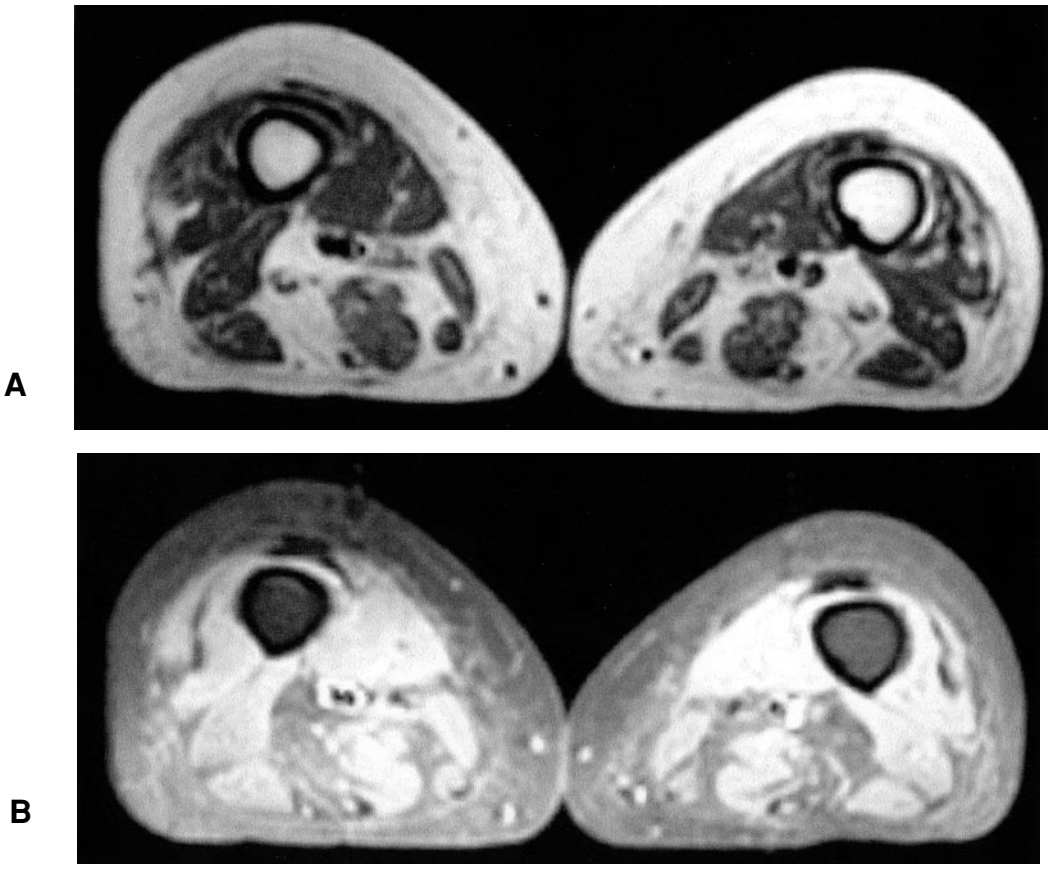


*Figure 6*. Axial SE T1 weighted images through midsection of the thighs in a patient with a 5 month history of severe PM. Precontrast (A) and 5 min postcontrast with fat saturation (B). Note diffuse enhancement of muscles of the 3 groups. Slight atrophy and fatty infiltration are already visible (A).

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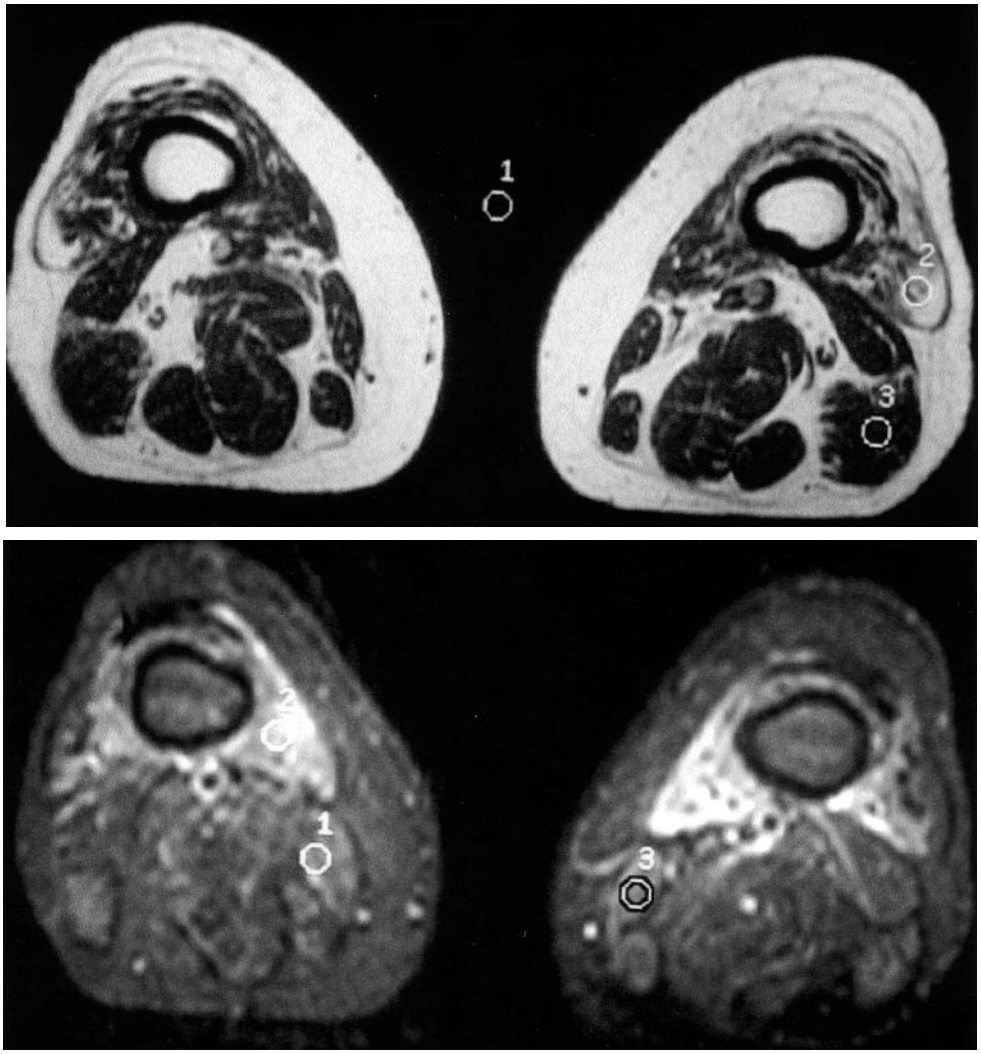


*Figure 7.* Axial images through distal part of the thighs in a patient with recently diagnosed sIBM, but with symptom onset 24 months previously. A. SE T1 weighted image. B. STIR image. Areas of inflammation are visible on the STIR sequence, mainly in the vastus lateralis and medialis (B). Fatty infiltration of the same areas is depicted on the SE T1 weighted image. Inflammation can thus be present in areas with fatty infiltration. Regions of interest numbered 1 to 3 were used to measure signal intensity.

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