MR imaging of pelvic and thigh muscles in congenital muscular dystrophy

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To define and compare the magnetic resonance (MR) imaging findings of pelvic and thigh muscles in merosin-deficient and merosin-positive congenital muscular dystrophy, 10 patients with merosin-positive and six patients with merosin-deficient muscular dystrophy were examined in a 0.5 T MR imaging unit. Intensity and atrophy scores were given to individual muscles by two radiologists and were calculated for three muscle groups (pelvic, anterior thigh and posterior thigh muscles). Rectus femoris was affected less than the vastus muscles in 40 percent of cases in merosin-positive patients, whereas there was no selective sparing in merosin-deficient patients. Sartorius and gracilis were relatively spared in both groups. The most consistently affected muscles were gluteus maximus, adductor magnus and brevis in merosin-positive patients. Atrophy was more prominent in the adductor muscles in the merosin-deficient group. Intensity scores of anterior thigh muscles of the merosin-positive group were significantly higher than those of the merosin-deficient group ($U = 8$, $p = 0.016$). When stepwise logistic regression model was applied, intensity score of the anterior thigh muscles was found to be the best differentiating variable. The regression analysis model formed was able to differentiate the two forms with a sensitivity of 80 percent and specificity of 83 percent.

Key words: muscle, muscular dystrophy, magnetic resonance imanging.

Congenital muscular dystrophy (CMD) is a heterogeneous group of diseases characterized by muscle weakness at birth or within the first few months of life, dystrophic changes in muscle, early onset hypotonia, joint contractures, delayed motor milestones and a relatively slow progression[1]. At least three different types can be identified within CMD nosology: Type I (classical form) with a normal intelligence and no apparent central nervous system (CNS) involvement, Type II (Fukuyama CMD) presenting with severe CNS manifestations, and uncommon forms such as muscle-eye-brain disease and Walker-Walburg syndrome[2]. The discovery of merosin deficiency in some CMD patients has led to subclassification of the classical form to the merosin-positive CMD and merosin-deficient CMD forms[3]. Merosin-deficient CMD is a clinically homogeneous disorder caused by mutations of the LAMA 2 gene[4]. Three genes responsible for merosin-positive CMD have been mapped so far: Fukuyama CMD on 9q31, CMD with early rigidity of the spine (CMD-RSS) on 1p35-36, and muscle-eye-brain disease on 1p32-34[5-7]. When compared to the merosin-positive form, merosin-deficient CMD is more uniform in its clinical and pathological features and tends to be more severe, with marked motor disability and associated severe respiratory problems[8]. Merosin-deficient CMD and merosin-positive CMD have been further distinguished by the demonstration of CNS anomalies and white matter changes in magnetic resonance (MR) imaging studies of merosin-deficient CMD patients[9,10]. However, there is no study in the literature comparing the muscle involvement patterns of the two

groups. In this study our purpose was to define and compare the MR imaging findings of pelvic and thigh muscles in merosin-deficient and merosin-positive CMD patients.

Material and Methods

Ten merosin-positive CMD patients (6 males, 4 females, mean age: 7 years) and six merosin-deficient CMD patients (4 males, 2 females, mean age: 3.8 years) were included in our study (Table I). MR imaging was performed in a 0.5 Tesla system (Gyroscan T5, Philips, The Netherlands). T1-weighted (TR = 475, TE = 25), proton density weighted (TR = 2500, TE = 25), and T2-weighted (TR = 2500, TE = 80) axial images were obtained with a surface coil, and additional T1-weighted coronal images were taken using the body coil. Slice thickness was 5 mm with a gap of 2 mm (FOV: 220-280 mm, NSA: 2, matrix: 154 x 256). Ten patients (5 males, 5 females, mean age: 7 years) without any neuromuscular problems or malnutrition were also scanned using the same protocol, forming the control group. Normal and patient volunteers all gave informed consent according to the guidelines and procedures approved by the local human studies committee.

Table I. Diagnosis and Clinical Features of Our Patients

Case	Age (year)	Sex	Diagnosis	Functional status
1	12	M	MP-CMD	sits w/o support
2	4	M	MP-CMD	sits w/o support
3	7	M	MP-CMD	sits w/o support
4	9	F	MP-CMD	sits w/o support
5	10	F	MP-CMD	walks alone
6	5	M	MP-CMD	sits w/o support
7	7	M	MP-cMD	stands w/o support
8	5	F	MP-CMD	walks alone
9	2	M	MP-CMD	sits w support
10	9	F	MP-CMD	sits w/o support
11	1	M	MD-CMD	no head control
12	2	F	MD-CMD	no head control
13	11	M	MD-CMD	sits w support
14	4	M	MD-CMD	sits w/o support
15	4	M	MD-CMD	sits w/o support
16	1	F	MD-CMD	sits w support

M: male; F: female; MN-CMD: merosin-deficient congenital muscular dystrophy; MP-CMD: merosin-positive congenital muscular dystrophy; w: with; w/o: without.

All studies were retrospectively reviewed by two radiologists. Iliopsoas, gluteus maximus, gluteus medius, gluteus minimus, obturator internus, piriformis, and pectineus muscles (in the pelvis); rectus femoris, vastus lateralis, vastus medialis, vastus intermedius, sartorius, and gracilis muscles (in the anterior thigh); and biceps femoris, semimembranosus, semitendinosus, adductor magnus, adductor brevis and adductor longus muscles (in the posterior thigh) were evaluated for atrophy and intensity changes. Atrophy in muscles was evaluated by a four-grade scale by comparing the muscle size with the control group:

0: no atrophy.
1: mild atrophy.
2: moderate atrophy.
3: severe atrophy.

Intensity changes observed in muscles were also graded on a four-level scale[11]:

1: normal muscle signal intensity (homogeneous, hypointense signal contrasting sharply with subcutaneous and intermuscular fat).

2: slightly hyperintense, patchy intermuscular signal changes.

3: markedly hyperintense, patchy but widespread intramuscular signal changes.

4: total, homogeneous hyperintense signal change throughout the entire muscle, egual to the signal intensity of adjacent subcutaneous or paramuscular fat.

Atrophy and intensity scores of pelvic (gluteus muscles, iliopsoas, pectineus, piriformis), anterior thigh (sartorius, gracilis, quadriceps femoris) and posterior thigh (semimembranosus, semitendinosus, biceps femoris, adductor muscles) muscle groups were calculated for each patient. The scores of merosin-deficient and merosin-positive CMD patients for each of these muscle groups were compared by Mann-Whitney U test. Atrophy and intensity scores of merosin-deficient and merosin-positive CMD patients were also investigated by logistic regression analysis and stepwise logistic regression analysis.

Results

Atrophy and intensity scores of patients are summarized in Tables II and III. In merosin-positive CMD patients, intensity changes in the vastus muscles were prominent, and intensity scores were either 3 or 4 in nine cases (n = 9.90%). There was no clear sparing of the rectus femoris muscle within the quadriceps muscle group. However, the intensity score of

Table II. Atrophy Scores of Pelvic and Thigh Muscles

Case	Iliopsoas	Gluteus maximus	Gluteus medius	Gluteus minimus	Obturator internus	Piriformis	Pectineus	Sartorius	Gracilis	Rectus femoris	Vastus medialis	Vastus intermedius	Vastus lateralis	Semimembranosus	Semitendinosus	Biceps femoris	Adductor longus	Adductor magnus	Adductor brevis
1	1	1	1	1	1	1	1	0	0	1	1	1	1	1	1	1	0	1	1
2	3	3	3	3	3	3	1	2	1	3	3	3	3	3	3	3	3	3	3
3	0	0	0	0	1	2	0	0	0	0	0	0	0	0	0	0	1	0	0
4	1	0	0	0	1	3	2	0	0	0	0	0	0	0	0	0	2	2	2
5	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
6	2	1	1	1	1	0	2	0	0	0	2	2	2	1	1	1	0	2	2
7	3	2	2	2	2	2	2	2	1	3	2	2	2	2	2	2	2	2	2
8	1	2	2	2	0	0	0	0	0	0	1	1	1	0	0	0	1	3	3
9	3	3	3	2	2	2	2	3	3	1	3	3	3	3	2	3	2	1	1
10	3	3	3	2	3	2	2	2	0	2	3	3	3	1	0	2	1	3	3
11	1	1	1	1	1	2	1	1	1	2	2	2	2	1	1	2	3	3	3
12	1	2	1	1	2	1	0	1	1	1	1	1	1	1	1	1	3	3	3
13	1	0	0	0	1	1	1	0	0	1	1	1	1	0	0	0	0	0	0
14	1	3	3	3	2	3	1	1	1	2	2	2	2	2	2	2	2	2	2
15	1	0	0	0	1	1	0	0	0	1	1	1	1	1	1	1	3	3	3
16	0	2	2	2	2	2	0	1	1	2	2	2	2	2	3	3	3	3	3

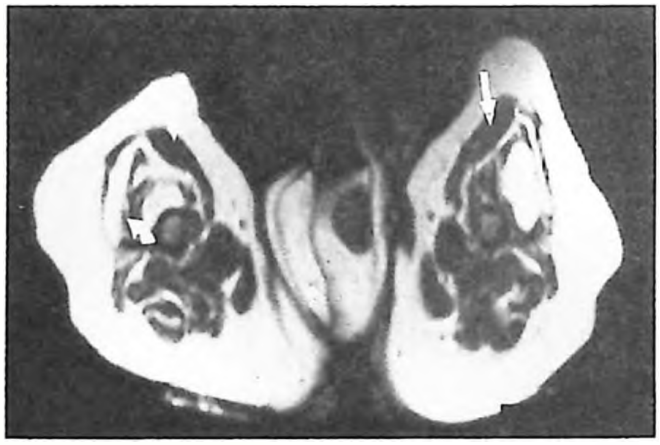
0: no atrophy.
1: mild atrophy.
2: moderate atrophy.
3: severe atrophy.

Table III. Intensity Scores of Pelvic and Thigh Muscles

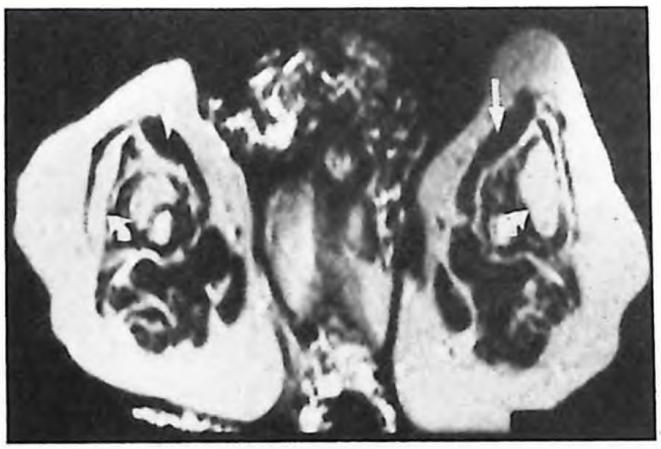
Case	Iliopsoas	Gluteus maximus	Gluteus medius	Gluteus minimus	Obturator internus	Piriformis	Pectineus	Sartorius	Gracilis	Rectus femoris	Vastus medialis	Vastus intermedius	Vastus lateralis	Semimembranosus	Semitendinosus	Biceps femoris	Adductor longus	Adductor magnus	Adductor brevis
1	3	3	3	3	3	2	3	2	1	4	4	4	4	3	3	3	2	3	3
2	1	1	1	1	1	1	1	1	1	1	4	4	4	1	1	1	1	1	1
3	2	4	4	4	2	3	4	2	2	4	4	4	4	4	4	4	2	4	4
4	4	4	4	4	3	3	4	2	2	4	4	4	4	4	4	4	2	4	4
5	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	4	3	2	3
6	2	4	2	2	1	1	1	2	1	2	4	4	4	3	2	2	1	4	4
7	2	3	2	2	1	1	2	3	2	3	3	3	3	2	2	3	2	2	2
8	2	4	2	2	1	1	2	1	1	2	2	2	2	1	1	1	2	4	4
9	3	4	1	1	2	2	1	4	4	1	4	4	4	1	4	4	1	2	2
10	3	4	2	2	4	2	2	4	1	2	4	4	4	4	1	4	2	4	4
11	1	2	2	3	3	2	1	1	1	3	3	3	3	2	2	3	3	3	3
12	2	3	3	3	2	2	1	1	1	2	2	4	2	1	2	2	4	4	4
13	3	4	4	4	4	4	4	2	2	3	3	3	3	3	3	3	3	3	3
14	1	3	3	3	3	3	2	2	2	2	3	3	3	3	3	3	3	1	3
15	2	4	4	4	1	2	4	1	1	4	3	3	3	1	2	1	1	3	3
16	1	2	2	2	1	2	1	1	1	2	3	3	3	2	2	2	1	3	3

1 : normal muscle signal intensity (homogeneous, hypointense signal contrasting sharply with subcutaneous and intermuscular fat).
2 : slightly hyperintense, patchy intermuscular signal changes.
3 : markedly hyperintense, patchy but widespread intramuscular signal changes.
4 : total, homogeneous hyperintense signal change in whole muscle, equalling the signal intensity of adjacent subcutaneous or paramuscular fat.

the rectus femoris was less than that of the vastus muscles in four cases (40%). Gracilis seemed to be spared with no atrophy in seven cases (70%), and with intensity scores of 1 or 2 in eight patients (80%) (Fig. 1). Although not as prominent as with the gracilis, the sartorius also seemed to be relatively spared compared to other thigh muscles. Its atrophy score was 0 in six cases (60%), 2 in three cases (30%), and 3 in two cases (20%), and its intensity score was 1 or 2 in six cases (60%).



(a)



(b)

Fig. 1. Four-year-old patient with merosin-positive congenital muscular dystrophy (CMD) (Case 2). T1-weighted (a) and T2-weighted (b) axial images of thigh muscles. Intensity score of rectus femoris muscle (arrows) is 1 whereas it is 4 for the vastus muscles (curved arrows). Atrophy scores of these muscles are all 4.

Gluteus maximus was the most consistently affected muscle in merosin-positive CMD patients, with an intensity score of 3 or 4 in nine cases (90%) (Fig. 2). Adductor magnus and brevis also demonstrated significant and consistent intensity changes, with intensity scores of 3 or 4 in eight cases (80%). In contrast, the intensity score of the adductor longus was either 1 or 2 in all 10 cases (100%).

In patients 3, 4 and 5, atrophy score was either 0 or 1, despite high intensity scores in all muscles (Fig. 3). However, although these patients demonstrated a common muscle involvement pattern on MR imaging, their clinical presentation was heterogeneous: patients 3 and 4 could only sit without support and patient 5 could walk alone.

Atrophy scores of the vastus muscles were less than 3 in all patients (100%) with merosin-deficient CMD (Fig. 4). Selective sparing of the

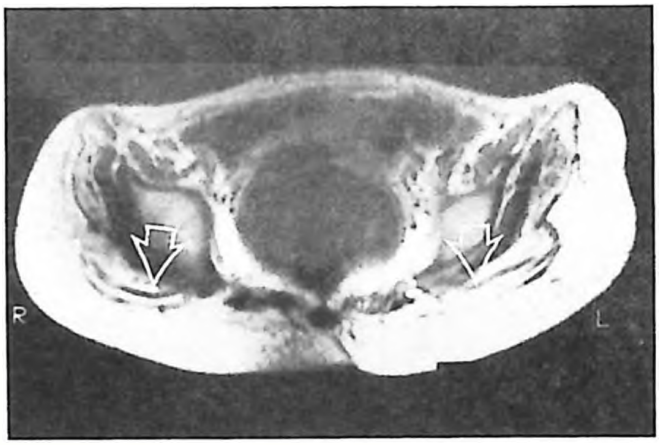


Fig. 2. Nine-year-old patient with merosin-positive congenital muscular dystrophy CMD (Case 10). T1-weighted axial images of pelvic muscles demonstrate severe atrophy and intensity changes in the gluteus maximus muscles (open arrows).

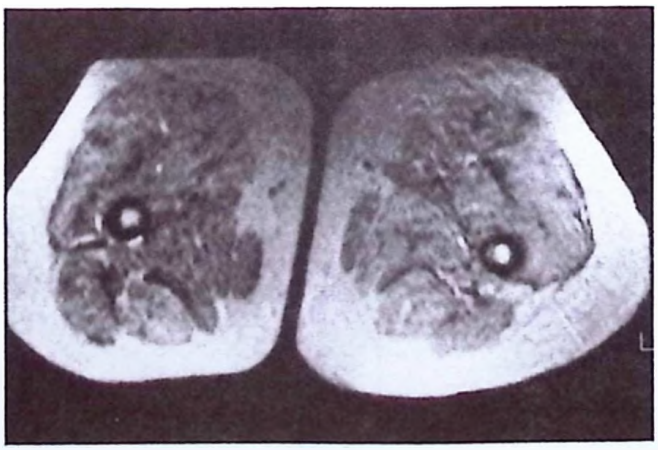


Fig. 3. 10-year-old patient with merosin-positive congenital muscular dystrophy CMD (Case 5). T2-weighted axial images of thigh muscles. Severe intensity changes (intensity score is 4 in semitendinosus, 2 in adductor longus and 3 in other thigh muscles) are noted in the thigh muscles without accompanying atrophy.

rectus femoris muscle was not observed. Intensity scores of the sartorius and gracilis were either 1 or 2, maximum atrophy score was 1 in all six cases (100%). Iliopsoas muscle seemed to be affected less than the other muscles. Atrophy was most prominent in the posterior thigh muscles, especially in the adductors (Fig. 5). Atrophy score of the adductors was either 3 or 4 in five patients (83%). In three patients, atrophy in the adductor muscles was not accompanied by intensity changes (50%).

The only statistically significant difference between the atrophy and intensity scores of merosin-deficient and merosin-positive CMD patients was the intensity score of the anterior thigh muscles. The intensity scores of the anterior thigh muscles of merosin-positive CMD patients were significantly higher than those in the merosin-deficient CMD patients ($U = 8$, $p = 0.016$). The differences between the other corresponding atrophy and intensity scores of merosin-deficient and merosin-positive CMD patients were not significant.

Logistic regression analysis was applied to determine the significance of atrophy and intensity scores in differentiation between the merosin-deficient and merosin-positive CMD patients. The success of the model is summarized in Table IVA and IVB. The model formed using the atrophy scores of pelvic and thigh muscles (IVA) was 100 percent sensitive and 67 percent specific in differentiating merosin-positive from merosin-deficient CMD patients. The model formed using the intensity scores of pelvic and thigh muscles (IVB) was 90 percent sensitive and 67 percent specific in differentiating merosin-positive from merosin

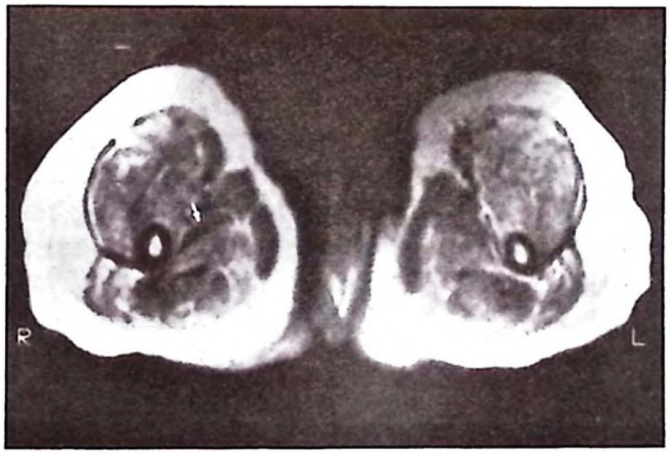


Fig. 4. Four-year-old patient with merosin-deficient congenital muscular dystrophy CMD (Case 15). T2-weighted axial images of thigh muscles show more prominent atrophy in adductor muscles compared to the other thigh muscles (small arrow).

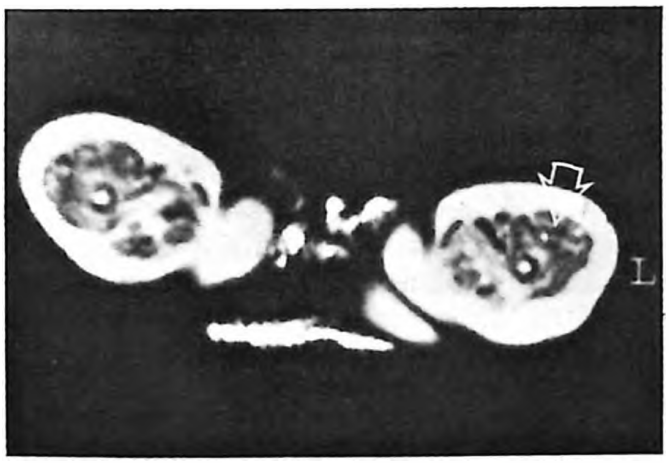


Fig. 5. Two-year-old patient with merosin-deficient congenital muscular dystrophy CMD (Case 12). T2-weighted axial images of thigh muscles. Relative sparing of anterior thigh muscles compared to the posterior high muscles is noted (open arrow).

Table IV-a. Logistic Regression Analysis Table Formed Using the Atrophy Scores of Pelvic Anterior and Posterior Thigh Muscles

		Predicted		
		Mer+CMD	Mer-CMD	Total
Observed	Mer+CMD	12	0	12
	Mer-CMD	2	4	6
	Total	14	4	18

Logistic regression coefficients.
exp (0.1042-0.6527VI+0.4156V2+0.2858V3).
V1: total atrophy scores of pelvic muscles;
V2: total atrophy scores of anterior thigh muscles.
V3: total atrophy scores of posterior thigh muscles.
Mer+CMD: merosin-positive congenital muscular dystrophy.
Mer-CMD: merosin-deficient congenital muscular dystrophy.

Table IV-b. Logistic Regression Analysis Table Formed Using the Insensity Scores of Pelvic Anterior and Posterior Thigh Muscles

		Predicted		
		Mer+CMD	Mer-CMD	Total
Observed	Mer+CMD	11	1	12
	Mer-CMD	2	4	6
	Total	13	5	18

Logistic regression coefficients.
exp (6.932+0.2385V4-0.6831V5-0.0543V6).
V1: total atrophy scores of pelvic muscles;
V2: total atrophy scores of anterior thigh muscles.
V3: total atrophy scores of posterior thigh muscles.
Mer+CMD: merosin-positive congenital muscular dystrophy.
Mer-CMD: merosin-deficient congenital muscular dystrophy.

deficient CMD patients. When stepwise logistic regression analysis was applied, the intensity score of the anterior thigh muscles was found to be the best differentiating variable between merosin-deficient and merosin-positive CMD patients. The success of the model is summarized in Table V. The model formed using the intensity score of anterior thigh muscles was 80 percent sensitive and 83 percent specific in differentiating merosin-positive from merosin-deficient CMD patients.

Table V. Logistic Regression Analysis Table Formed Using the Intensity Scores of Anterior Thigh Muscles

		Predicted		
		Mer+CMD	Mer-CMD	Total
Observed	Mer+CMD	11	1	12
	Mer-CMD	1	5	6
	Total	12	6	18

Logistic regression coefficients.
exp (7.1949-0.478V5).
V5: total intensity score of anterior thigh muscles.
Mer+CMD: merosin-positive congenital muscular dystrophy.
Mer-CMD: merosin-deficient congenital muscular dystrophy.

Discussion

Imaging methods have been utilized more frequently in the evaluation of primary skeletal muscle diseases in recent years. These methods include conventional radiography, scintigraphy, ultrasonography (US), computed tomography (CT) and, more recently, MR imaging[12-16]. MR imaging can display both normal and pathological skeletal muscle[16]. It can characterize the muscle diseases in terms of the involved muscles, distribution of the involved muscles and pattern of fatty replacement. The thin intermuscular fat planes are easily imaged, allowing identification of individual muscles and muscle groups. Increased intramuscular signal intensity observed in most muscular diseases on both T1- and T2-weighted images is thought to represent fatty changes[17]. This increased intensity also facilitates the identification of the diseased muscle. The fatty nature of these intramuscular changes has been confirmed by chemical shift imaging[18]. Increased water content in the dystrophic muscles also adds to the increased signal intensity on T2-weighted images[19].

Different MR sequences have been used in the evaluation of primary muscle diseases. Murphy et al.[16] reported no advantage of IR (inversion recovery) or T2-weighted sequences over T1-weighted sequences. However, T2-weighted images seemed to be helpful in childhood inflammatory myopathies, with the increased contrast of diseased muscle[20]. Again, in inflammatory myopathies, increased signal on T2-weighted images has been reported to be present before any detectable abnormality on T1-weighted images[21,22]. In our study, intensity changes were best demonstrated on T1-weighted images. None of our cases displayed abnormal signal intensity on T2-weighted sequences in the absence of abnormality on T1-weighted sequences. However, T2-weighted sequences were helpful for the accurate grading of the muscles. We used mainly axial images in our evaluation. Coronal images provided additional information about the extent of the disease in a very few cases.

Although MR imaging findings of muscles in Duchenne muscular dystrophy have been well studied, with even an MR imaging grading system with functional correlation having been established, there is, to our knowledge, no report in the literature designed to study the muscle MR imaging findings in congenital muscular dystrophies[19,21,22]. Muscle involvement patterns in merosin-deficient and merosin-positive CMD patients have also not been investigated. Lamminen et al.[11] studied muscle MR imaging findings of muscular dystrophies, including nine patients with muscle-eye-brain disease, and concluded that the gracilis, sartorius and rectus femoris were relatively spared. Apart from this one stduy, all other muscle imaging studies in CMD have been performed with US[24-27]. Topaloğlu et al.[24] reported selective sparing of the rectus femoris muscle in eight out of 26 CMD patients. Heckmatt and Dubowitz[26] also reported one CMD case with selective sparing of the rectus femoris. However, in their series of 14 CMD cases, the same authors did not mention any selective involvement pattern identified by US[15]. Similarly, in another series of eight CMD patients studied with US, selective involvement or sparing of muscles was not reported[27]. In our study, the rectus femoris was affected less than the vastus muscles in 40 percent of cases in merosin-positive CMD patients, whereas there was no selective sparing in merosin-deficient CMD patients. The sartorius and gracilis were the two muscles which seemed to be relatively

spared in both merosin-deficient and merosin-positive CMD patients. We also noted a common involvement pattern in three cases in the merosin-positive group characterized by significant intensity changes in muscles without any atrophy. The most consistently affected muscles in merosin-positive CMD patients were the gluteus maximus, adductor magnus and adductor brevis according to the intensity scores, whereas the adductor longus was relatively spared among the adductors. Another striking feature was the more prominent atrophy of the adductors in the merosin-deficient CMD group. We think that these different patterns may help the clinician identify appropriate muscles for biopsy and plan courses of physical and rehabilitation therapy.

Ours is the first study to use atrophy and intensity scores for a regression analysis model to differentiate two separate muscle diseases. The regression analysis model formed by the intensity scores of the anterior thigh muscles could differentiate the merosin-deficient from merosin-positive CMD patients with a high level of sensitivity and specificity (80% and 82%, respectively). The intensity scores of the anterior thigh muscles in the merosin-positive CMD patients were significantly higher than in merosin-deficient CMD patients. These findings suggest that muscle MR imaging can also be used in the non invasive diagnosis of muscle diseases. However, further studies with larger series of patients are necessary in order to increase the confidence in the diagnosis.

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