Muscle Involvement in Sarcoidosis: A Retrospective and Followup Studies

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ABSTRACT. ***Objective.*** Muscle involvement is a frequent histopathologic feature of sarcoidosis, but its clinical presentation has been rarely reported and its longterm outcome is unclear. We describe the features and outcome of 5 patients with muscle sarcoidosis.

Methods. A retrospective study from hospital charts over the period 1985–2001 in 2 academic rheumatology centers.

Results. Muscle involvement was identified in 5 patients (3 women, 2 men) aged 37 to 61 years, out of a cohort of 45 patients with sarcoidosis. No symptomatic muscle involvement was observed in the 20 patients with Lofgren syndrome of our series. Muscle involvement was the initial feature of the disease in 2 patients. Three patients had nodular type and the 2 others the myositic type. Chronic myopathy was not observed. Followup of patients with muscle disease ranged from 30 to 144 months (mean 72.6). Prednisone was used at a starting dose from about 0.33 to 1 mg/kg/day in all patients, then progressively tapered, and was associated with use of hydroxychloroquine (HCQ) in 2 cases. One poorly compliant patient was persistently prescribed 40 mg/day prednisone and HCQ by his general practitioner and was still complaining of diffuse myalgia at the last 30-month followup visit. One patient also receiving HCQ experienced cardiac and renal relapse, leading to transient increase in steroid dosage, but remained symptom-free 3 years after steroid discontinuation. Muscle relapses occurred in the 3 other patients. Muscle symptoms disappeared after readministration of corticosteroids or increase of prednisone dosage. The first patient was symptom-free without any treatment at the last followup visit. The second was still taking 10 mg/day 144 months after disease onset because of steroid-dependent myalgia. The last patient was asymptomatic with 10 mg/day prednisone at the last evaluation.

Conclusion. Symptomatic muscle involvement may be an initial feature of chronic, and usually the systemic form of, sarcoidosis. It responds to corticosteroid therapy, but relapse seems to be frequent. (J Rheumatol 2006;33:98–103

Key Indexing Terms:
SARCOIDOSIS MUSCLE TREATMENT OUTCOME

Sarcoidosis is a granulomatous disorder of unknown etiology characterized by the accumulation of T lymphocytes and monocytes in the involved organs, noncaseating granulomas, and disorganized architecture of normal tissue[1]. Every organ can be affected by the granulomatous process, most frequently the lungs, lymph nodes, skin, eyes, and musculoskeletal system. The diagnosis is based on the association of clinical symptoms, thoracic radiograph findings, histological evidence of disease, and exclusion of other causes of granulomatous diseases, especially tuberculosis[1].

Asymptomatic muscle involvement occurs in 50% to 80% of patients with sarcoidosis, and it is characterized by an inflammatory process including granulomatous formation appearing in muscle biopsy[2]. Muscle involvement resulting in clinical symptoms is rare and has been described in less than 0.5% to 2.3% of patients with sarcoidosis[3].

We describe an additional series of sarcoidosis patients with symptomatic muscle involvement and presenting with unusual features.

MATERIALS AND METHODS

We retrospectively reviewed the medical records of consecutive patients with sarcoidosis seen in the rheumatology departments of 2 university hospitals between 1985 and 2001. We identified 45 patients in whom sarcoidosis was diagnosed based on suggestive clinical and radiological findings (hilar adenopathy, polyarthralgia, erythema nodosum, uveitis, or other) combined with either biopsy evidence of a noncaseating epithelioid granuloma with giant cells (n = 25) or Lofgren syndrome (n = 20), and identified patients with clinical muscle involvement defined as myalgia, weakness, and/or muscle nodules. Muscle involvement was confirmed by histology,

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imaging (magnetic resonance imaging, gallium scintigraphy), electromyographic study, or elevated muscle serum enzyme concentrations.

In these patients, the following data were obtained from hospital records: (1) demographics: age at diagnosis, sex, ethnic origin; (2) time from muscle symptom onset to diagnosis; (3) clinical information relevant to sarcoidosis, including clinical history data on principal complaints and physical examination, including proximal and distal muscle strength in upper and lower limbs; (4) laboratory values of erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and levels of serum muscle enzymes, e.g., creatine kinase (CK), lactate dehydrogenase (LDH), aldolase (ALD), aspartate aminotransferase (AST), and alanine aminotransferase (ALT); (5) chest radiograph; (6) findings from histopathological studies of tissue specimens; (7) followup duration; (8) number of muscle symptom flares; and (9) treatments used (dosage, duration) with their therapeutic and side effects. When available, findings from skeletal muscle exploration including ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), gallium-67 scintigraphy, or electromyography (EMG) were also recorded.

RESULTS

Patient characteristics and organ involvement. We identified 5 cases of sarcoidosis with symptomatic muscle involvement, 3 women and 2 men, mean age 49 years (range 37–61 yrs) at disease onset (Table 1). Two patients (Patients 1 and 2) had initial muscle features when referred to our departments for diagnosis. Patient 1 presented with isolated muscle symptoms, whereas Patient 2 also had dyspnea, dermo-hypodermic lesions over the ankles, and polyarthralgia of large joints at initial presentation. In 3 other patients (Patients 3, 4, 5), muscle symptoms developed 13 to 19 months after the diagnosis of pulmonary sarcoidosis. Patient 3 had pulmonary and liver involvement at initial presentation. Muscle involvement occurred 19 months later as part of a flare that included cutaneous and articular features. The course of the disease was then complicated by myocardial and renal (renal failure and proteinuria) involvement.

Patient 4 had had acute large joint arthralgia, and cervical and low back pain. Erythematous plaques were noted on the palms of his hands and over his right knee and right ankle. He had repeated episodes of dry cough, low grade fever, headache, and conjunctivitis. Chest radiograph showed left and right lower lobe infiltrates. Bronchoalveolar lavage showed an increased number of lymphocytes and an elevated angiotensin-converting enzyme (ACE) concentration, and transbronchial lung biopsy revealed epithelioid and giant cell granuloma. Gallium scintigraphy showed a mildly increased uptake in mediastinal lymph nodes. An ophthalmological examination found dry eye syndrome. Cerebrospinal fluid analysis revealed an elevated total protein level and 11 lymphocytes per ml. Systemic sarcoidosis was diagnosed with pulmonary, joint, cutaneous, ocular, and meningeal involvement. The patient reported full recovery after treatment of oral prednisone starting at 50 mg/day. Steroid therapy was tapered after 13 months. Nodular sarcoidosis of left sural muscle developed after stopping corticosteroid therapy and was diagnosed 42 months later. Serum aldolase level was found repeatedly to be elevated (2.5 to 3.5 times normal), but EMG of the lower limbs and gallium scintigraphy scans were normal. The patient refused muscle biopsy. Rapid improvement was observed after treatment with prednisone 20 mg/day tapered over one year. The case of Patient 5 has been described in detail[4]. Epididymitis and polyadenopathy were the initial presentation of sarcoidosis in this patient; muscle symptoms appeared 13 months later and multisystemic sarcoidosis was diagnosed 18 months after the first symptoms.

Other organ involvement was observed in the course of the disease in the 5 patients. Pulmonary involvement was present in 4 patients, 3 with stage II (lymphadenopathy and

Table 1. Patient characteristics and specific organ involvement.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age, years, sex	61 F	42 M	56 F	49 F	37 M
Ethnic origin	French West Indies	Caucasian	Caucasian	Caucasian	French West Indies
Pulmonary involvement	—	Stage I	Stage II	Stage II	Stage II
Joint involvement	—	Arthralgia	Arthralgia	Arthralgia	Arthritis
Cutaneous features	—	+	+	—	+
Neurological involvement	—	Peripheral neuropathy	CNS and peripheral neuropathy	Meningitis	—
Salivary gland involvement	Biopsy +	+	—	—	+
Lacrimal gland involvement	—	+	—	Dry eye	+
Myocardial involvement	—	—	Echo-cardiography +	—	—
Liver involvement	—	—	Biology and biopsy +	—	—
Other	—	—	Renal involvement and vasculitis	—	Peripheral adenopathy and epididymal involvement

CNS: central nervous system.

parenchymal disease) and one with stage I disease (hilar and mediastinal lymph node enlargement). Arthralgia and/or arthritis, predominantly of large joints of the lower limbs, were observed in 4 patients. Cutaneous features and neurological involvement were noted in 3 patients each. Patient 2 had peripheral neuropathy (predominantly sensation) of the limbs confirmed by EMG study; Patient 3 developed paresthesia of the lower limbs, and cerebral MRI showed subcortical lesions, while EMG detected lower limb peripheral neuropathy. Patient 4 had meningeal involvement confirmed by cerebrospinal fluid analysis. Salivary glands were involved in 3 patients, but this was almost always asymptomatic. Myocardial, liver, epididymal, renal, and lymph node involvement resulting in severe disease were noted once each (Table 1).

Clinical muscle symptoms and results of muscle investigations. Table 2 displays clinical muscle symptoms and the results of other investigations. Four patients complained of myalgia. Palpable nodules were found in 3 patients, although muscle weakness was not observed. Serum muscle enzymes were normal, except for aldolase in Patient 4. Serum aldolase level was 19 IU/l at first and checked at 24 IU/l (normal < 7.5 IU/l). Muscle ultrasonography was performed in 2 patients (quadriceps in Patient 1, triceps sural in Patient 4) and showed a high echogenic formation in the left vastus medialis of Patient 1. MRI of skeletal muscles (thighs in 2 cases, triceps surae and deltoid muscles in one case each) showed no abnormalities in 2 cases (Patients 2 and 4), and revealed 2 nodules in the left quadriceps of Patient 1 and multiple bilateral nodules in thigh muscles of Patient 5. The latter patient had diffuse increased uptake on gallium scintigraphy. Bilateral inflammatory involvement of lungs, mediastinal, hilar and perihilar regions, skeletal muscles, and parotid and lacrimal glands was noted in gallium-67 citrate uptake results along with appearance of the "leopard-man" sign in this patient. Nevertheless, gallium scintigraphy showed no abnormal muscle uptake in 2 other patients, Patients 2 and 4, but revealed increased uptake in bilateral pulmonary hila, and occasionally in salivary and lacrimal glands. EMG displayed a myopathic pattern in 2 patients (Patients 2 and 3) and was normal in one patient (Patient 4). Histological analysis of muscle biopsies performed in 2 patients showed typical noncaseating epithelioid granulomas with giant cells.

Laboratory test results and histological findings of other organ biopsies. ESR was normal (< 15 mm/h) in 2 patients and elevated in 3 patients, with a mean value of 38 mm/h (range 26–48). CRP level was > 5 mg/l in 3 patients (range 22–86). The ACE serum concentration was elevated in 3 patients. Blood calcium was normal in all patients, but urine calcium excretion was increased in 2 cases (Table 3).

Histological examinations of transbronchial biopsies in 2 patients were normal in the first (Patient 1) and showed noncaseating epithelioid granulomas with giant cells in the second (Patient 4). Noncaseating epithelioid granulomas were also observed on salivary gland specimens in Patient 1, on

Table 2. Clinical muscle symptoms, and laboratory, imaging, electromyography, and histology findings.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Duration of muscle symptoms before diagnosis, mo	6	1	1	42	5
Muscle symptoms	At presentation	At presentation	Appeared 19 mo after diagnosis of sarcoidosis	Appeared 13 mo after diagnosis of sarcoidosis	Appeared 13 mo after initial symptoms of sarcoidosis (epididymitis)
Muscle weakness	—	—	—	—	—
Myalgia	+	+	+	+	—
Palpable nodules	+	—	—	+	+
CK/LDH	Normal	Normal	Normal	Normal	Normal
Aldolase	3.6	Normal	5.3	19 (N < 7.5)	4.8
Ultrasonography	High echogenicity formation in left vastus medialis	ND	ND	Normal (triceps sural)	ND
MRI	Two nodules in left quadriceps (vastus medialis 2 x 6.5 cm and crural 1 x 1.5 cm)	Normal (deltoid)	ND	Normal (triceps sural)	Multiple nodules in bilateral thigh muscles
Gallium scintigraphy	ND	No abnormal muscle uptake	ND	No abnormal muscle uptake	Diffuse increased uptake in skeletal muscles
EMG	ND	Myopathy pattern on upper limbs; neuropathy pattern	Myopathy and neuropathy pattern	Normal (lower limb)	ND
Muscle biopsy	Noncaseating granulomas	Noncaseating granulomas	ND	ND	ND

CK: creatine kinase, LDH: lactate dehydrogenase, MRI: magnetic resonance imaging, EMG: electromyography, ND: not done.

Table 3. Laboratory test results and histological findings of other organ biopsies.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Inflammatory syndrome	—	+	+	—	+
ACE	169 (65–135)	Normal	88 (8–52)	Normal	124 (35–115)
Blood calcium	Normal	Normal	Normal	Normal	Normal
Urine calcium	Normal	Normal	Normal	Increased	Increased
Transbronchial lung biopsy	Normal	ND	ND	Noncaseating granuloma	ND
Salivary gland biopsy	Noncaseating granuloma	ND	ND	ND	ND
Liver biopsy	ND	ND	Noncaseating granuloma	ND	ND
Epididymal biopsy	NA	ND	NA	NA	Noncaseating granuloma
Lymph node biopsy	ND	ND	ND	ND	Epithelioid granuloma with necrosis

ACE: angiotensin-converting enzyme, ND: not done, NA: not applicable.

liver biopsy in Patient 3, and on epididymal biopsy in Patient 5 (Table 3).

Management and clinical course. Followup of muscle disease ranged from 30 to 144 months (mean 72.6). A tapering regimen of prednisone at a starting dose from about 0.33 to 1 mg/kg/day was used in all patients and associated with hydroxychloroquine (HCQ) in 2. Treatments and disease course are shown in Table 4. One poorly compliant patient (Patient 2) was persistently prescribed 40 mg/day of prednisone and HCQ by his general practitioner and was still complaining of diffuse myalgia at the last 30-month followup visit. One patient (Patient 3) also receiving HCQ experienced cardiac and renal relapse, leading to transient increase in steroid dosage (500 mg pulse of methylprednisolone for 2 days, followed by 60 mg/day prednisone), but remained symptom-free 3 years after steroid discontinuation. Relapses occurred in the 3 other patients (Patients 1, 4, and 5). The first developed myalgia, proximal muscle weakness of lower limbs, and increased creatine kinase (3 times normal) and ACE levels 6 years after withdrawal of steroid; he recovered after another 3 month course of prednisone at an initial dose of 20 mg/day. In the second patient, one year after steroid withdrawal, 2 nodules developed in the left forearm and disappeared after readministration of 30 mg/day prednisone; this patient was still taking 10 mg/day 144 months after disease onset because of steroid-dependent myalgia. In the last patient, complete remission was obtained at first, but muscle nodules relapsed with an increased muscle serum enzyme level when prednisone dosage was tapered under 5 mg/day after 3 years. An increase of prednisone dosage to 10 mg/day was found to be efficacious.

DISCUSSION

Muscular sarcoidosis was reported for the first time in 1908 and remains underdiagnosed. Asymptomatic muscle involvement occurs in 50% to 80% of patients, but symptomatic involvement is rare (0.5% to 2.5% of patients)[2,3,5,6]. Myopathy can be the initial manifestation of sarcoidosis, as in 2 of our patients, even though when diagnosis is confirmed, signs and symptoms of other organs are usually present[7-10].

Our short series of patients with muscular sarcoidosis reflects the diversity of muscle involvement and the variety of clinical presentation of the disease. Three types of symptomatic muscular sarcoidosis have been described in the medical literature: nodular involvement, acute myositis, and chronic myopathy.

Table 4. Treatments used and clinical course.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Initial treatment	CS	CS + HCQ	CS + HCQ	CS	CS
Followup duration, mo	99	30	48	144	42
Muscle relapse	Yes	No	No	Yes	Yes
Last clinical status and treatment	Asymptomatic without medication	Myalgia; plaquenil 400 mg/day and prednisone 40 mg/day	Asymptomatic with plaquenil 400 mg/day	Steroid-dependent myalgia; 10 mg prednisone	Improved; 10 mg/day prednisone

CS: corticosteroids, HCQ: hydroxychloroquine.

1. Nodular muscle involvement was diagnosed in 3 patients of our series. Diagnosis in 2 patients (Patients 1 and 5) was based on clinical examination and ultrasonography, MRI, or gallium scintigraphy. Nodular involvement was diagnosed in a third patient (Patient 4) in spite of normal radiologic and EMG findings because palpable muscle nodules were found in 2 different sites at the 2-year interval (relapse), with an increased serum aldolase level. The nodular type is the least common type of symptomatic muscle involvement in sarcoidosis[6,11]. It may involve any skeletal muscle; however, the lower limbs are more frequently affected. Nodules of various sizes are usually palpable, painless, and not accompanied by any limitation of movement or muscle weakness. However, contracture and myalgia may appear. Serum creatine kinase level and EMG studies are usually normal. MRI shows even small nodules owing to excellent tissue contrast, and MRI findings of the nodular type have been reported to be specific[12-14]. According to Otake, *et al*[12-14], nodules exhibit (1) a central "dark star" surrounded by a peripheral high signal area on T1 weighted proton-density, and T2 weighted axial images; and (2) 3 stripes on coronal and sagittal images: the inner stripe with decreased T2 signal intensity, and the outer stripes with increased signal intensity on coronal and sagittal images that correspond to the central dark star and the peripheral high signal areas on axial images, respectively. Pathological assessment has shown that the central area corresponds to fibrous tissue, whereas the peripheral area consists of zones infiltrated by inflammatory cells. However, some investigators consider that the characteristic MRI features of sarcoid nodules cannot be distinguished from benign or malignant lesions[15]. Muscle biopsy shows multiple granulomas with CD4 lymphocytes in the center and CD8 lymphocytes in the periphery and in the endomysium[16].

2. The 2 other patients (Patients 2 and 3) of our series had a myositic type of muscle involvement, with diffuse pain, characteristic EMG findings, and histologic analysis in one patient only. The acute myositic type occurs mainly in patients younger than 40 years and is characterized by diffuse muscle swelling and pain that progresses to muscle contracture, hardening, and hypertrophy[17,18]. Some patients can present with phlebitis-like acute, swollen, and painful calf or thigh. Fatigue, fever, joint symptoms, and erythema nodosum are generally associated[17]. Symmetrical proximal muscles are usually involved. Muscle weakness is rarely observed[9]. Muscle enzymes are frequently increased and EMG studies show patterns similar to polymyositis. Gallium scintigraphy reveals diffuse increased uptake[19-21]. MRI study may be normal because of the small size of the lesions[18]; however, T2 weighted images usually show diffusely increased signal intensity[21]. Muscle biopsy allows distinction from early onset polymyositis. The muscle fibers are dissociated by an interstitial cellular infiltrate, which contains epithelioid cells laid out in several concentric layers, lymphocytes, and giant cells. Perifascicular atrophy is observed in 50% of cases[17]. CD4/CD8 lymphocyte ratio by immunostaining is high, in contrast with the low ratio observed in polymyositis and inclusion myositis[22]. Nevertheless, few instances of coexistence of dermatomyositis and sarcoidosis have been described[23].

3. Although chronic myopathy has been reported as the most frequent type of symptomatic muscle sarcoidosis[11], we did not identify this form in our series. Chronic myopathy occurs mainly in women aged between 50 and 60 years and is characterized by a slow progressive symmetrical weakness and atrophy of the proximal muscle groups. An unusual case of abdominal wall muscle involvement has been reported[24]. This type must be distinguished from distal muscle involvement secondary to peripheral neuropathy, which has been well documented in sarcoidosis[6]. MRI and CT scans are normal, whereas gallium-67 citrate scintigraphy has been reported to exhibit a diffusely increased uptake pattern[14,18]. The extent of uptake depends on the degree of inflammation. Muscle enzymes are usually normal and a myopathic pattern can be observed in EMG studies. Remissions and exacerbations are noted during the course of the disease[22,25]. Muscle biopsy shows microscopic granulomas distributed among muscle fibers and fibrosis[18].

Corticosteroids are usually effective in patients with nodular lesions and acute myositis, with a starting dosage varying from 0.5 to 1 mg/kg/day. The earlier the treatment, the more effective it is. Although corticosteroid treatment was rapidly effective in 3 patients of our series, relapse was observed in all these patients several years later. The response is unpredictable in patients with chronic myopathy type. Excellent results have been achieved in some cases, whereas in others there has been no response to corticosteroids. Outcome might be complicated by corticosteroid-induced myopathy[26]. In steroid-dependent patients or in those who fail to respond to corticosteroids, chloroquine, azathioprine, and mainly methotrexate should be tried[11,18,27]. New agents, including thalidomide and infliximab, could be useful in selected cases. The effectiveness of these agents seems to lie in their ability to block tumor necrosis factor[28,29].

Symptomatic muscle involvement in sarcoidosis occurs in the chronic and usually systemic form of the disease, and is rarely its initial presentation. The clinical picture is variable and diagnosis often requires multiple investigations. Gallium scintigraphy is the most useful tool that can demonstrate muscle involvement in chronic myopathy. Serum muscle enzymes are usually increased, and EMG study shows a myopathic pattern in the acute myositic type. Muscle biopsy is needed to distinguish it from early polymyositis. Gallium scintigraphy and especially MRI are very useful for diagnosing nodular type. Improvement of muscle symptoms is achieved in the majority of cases with corticosteroid therapy, but the disease frequently relapses.

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