

## The Coexistence of Amyopathic Dermatomyositis and Fibromyalgia

Sir,

Amyopathic dermatomyositis (ADM) is an uncommon variety of dermatomyositis (DM) and affects patients without muscle weakness or laboratory evidence of muscle involvement (1–3). In primary idiopathic DM up to one-third of patients present initially with only skin changes which may last for several years before muscle weakness develops (1–7). A few cases have been followed for as much as 10–12 years without signs of muscle involvement. It has been suggested that nearly 10% of all patients with DM describe their disease as ADM (2–4).

Criteria for the diagnosis of ADM include pathognomonic Gottron's papules, together with the characteristic heliotrope, periorbital, violaceous erythema associated with oedema. Other characteristic skin lesions include periungual telangiectasia and macular, violaceous erythema on the hands, arms, chest, neck or shoulders. One or two pathognomonic signs associated with one or more characteristic signs and a compatible skin biopsy are required for the diagnosis of ADM. Several other skin manifestations have been described, such as pruritus, photosensitivity, poikiloderma, scalp involvement with alopecia and a seborrhoeic pattern of cutaneous involvement (1, 6, 8, 9).

The prevalence of fibromyalgia in another connective-tissue disease, systemic lupus erythematosus, is high (10), but has not been described in connection with DM. Such a case is reported here.

### CASE REPORT

A 52-year-old woman had initially presented about 5 years previously with an erythematous, slightly scaly, eruption on her face involving the front, the cheeks and particularly the facial fold. She also had an erythematous rash on her breast. The diagnosis was seborrhoeic dermatitis or perhaps psoriasis and she was prescribed a mild steroid cream with an antifungal component. Four months later she returned because of worsening symptoms; on this occasion her skin manifestations consisted of periocular, heliotrope, violaceous erythema associated with oedema with extension to the front. On the trunk, including the upper part of the abdomen, the breast region, the neck and shoulders, there was a reddish-blue erythema with telangiectasia. Erythema and papules were noted on the bony prominences of the elbows, hips and knees. Her fingers were erythematous and felt swollen. She ascribed her symptoms to sun exposure.

The diagnosis was changed to DM and a biopsy was obtained from the trunk and processed for routine histologic staining and direct immunofluorescence (DIF) studies. Using light microscopy a perivascular lymphocytic infiltration was observed in the dermis while DIF examination showed a bandlike deposition of IgM, complement and fibrin. Serum levels of creatine phosphokinase, aldolase and lactic acid dehydrogenase were normal. Antinuclear antibodies were not detected. The patient was about 20 kg overweight. She was periodically depressed, had difficulty sleeping, did not feel refreshed in the morning and complained of general fatigue and widespread pain above and below the waist (arms, shoulders, back, thighs and knees) which had lasted for several years. On examination she experienced extreme pain upon pressure (4 kg) at 12 fibromyalgia tender points and her muscles felt hypertonic. Muscle strength was apparently normal. Because of progressing skin symptoms, which caused her much distress, she was treated with moderate doses of

prednisolone, 20 mg daily, which were tapered off after 6 weeks. The patient underwent extensive investigation for associated malignancy but this was not found. She was later referred to the National Hospital, University of Oslo, and was examined at the Department of Dermatology and the Center for Rheumatic Diseases. Her skin symptoms were at that time in regression but the diagnosis of fibromyalgia was sustained. Muscle biopsy and electromyography (EMG) were normal.

The patient has been followed for 5 years without laboratory signs of muscle involvement. Because of her fibromyalgia symptoms she has now received a disability pension. Recurrence of her skin symptoms necessitates treatment with prednisolone. Her laboratory data, including muscle enzymes and antinuclear antibodies (ANA), are normal.

### DISCUSSION

By definition the term ADM should be reserved for those patients who have characteristic and pathognomonic skin lesions of DM without any evidence of muscle disease at any time during the course of the disease. As some cases may develop signs of muscle involvement after 4 years, one could use the term premyopathic DM (8). EMG and muscle enzymes are not always correlated and muscle histology may depend on the biopsy site; however, it is difficult to convince a patient to undergo another muscle biopsy. MRI may be warranted in patients with ADM (1, 7) and would certainly have been of use in the present case.

The cutaneous manifestations of DM are polymorphic and may be misleading in the absence of overt muscle involvement, as was the case at the initial examination of our patient. Although the skin symptoms responded to oral corticosteroid therapy, regression was not complete. This patient had typical fibromyalgia with subjective and objective manifestations but surprisingly there were no laboratory findings of muscle disease. There are several overlapping symptoms in DM and fibromyalgia, e.g. lethargy, fatigue, arthralgia and, of course, muscle weakness (6). This can lead to misinterpretation and also to a delay in diagnosis and therapy. Our patient fulfilled the ACR-90 classification criteria of fibromyalgia (11), with chronic widespread pain for >3 months and  $\geq 11$  tender points. According to these criteria a distinction between primary and secondary fibromyalgia is no longer made and the presence of associated symptoms is not required. Furthermore, a second clinical disorder does not exclude the diagnosis of fibromyalgia.

We do not know the ultimate outcome of ADM with regard to the development of malignancy and muscle involvement. ADM has been regarded as one end of a clinical spectrum, with the combination of skin and muscle symptoms in the middle and polymyositis at the other end (3, 6). It is not yet known if all patients with ADM will eventually develop muscle disease. It has been advocated that an aggressive approach to treating the skin disease may prevent the development of muscle disease in patients who initially have only skin involvement (3). Only prospective studies can prove this proposal. Corticosteroids may induce myopathy but there seems to be no such relationship in the present case as the therapy was begun years after the symptoms of fibromyalgia first appeared. The coexistence of

ADM and fibromyalgia in this patient is probably a coincidence, but certainly an interesting one.

## REFERENCES

1. Kovacs SO, Kovacs SC. Dermatomyositis. *J Am Acad Dermatol* 2000; 39: 899–920.
2. Cosnes A, Amaudric F, Gherardi R, Verroust J, Wechsler J, Revuz J, et al. Dermatomyositis without muscle weakness. Long-term follow-up of 12 patients without corticosteroids. *Arch Dermatol* 1995; 131: 1381–1385.
3. Euwer RL, Sontheimer RD. Amyopathic dermatomyositis: a review. *J Invest Dermatol Suppl* 1993; 100: 124–127.
4. Rockerbie NR, Woo TY, Callen JP, Giustina T. Cutaneous changes of dermatomyositis precede muscle weakness. *J Am Acad Dermatol* 1989; 20: 629–632.
5. Caproni M, Salvatore E, Bernocchi E, Fabri P. Amyopathic dermatomyositis: report of three cases. *Br J Dermatol* 1998; 139: 1116–1118.
6. Euwer RL, Sontheimer RD. Amyopathic dermatomyositis (dermatomyositis sine myositis). Presentation of six new cases and review of the literature. *J Am Acad Dermatol* 1991; 24: 959–966.
7. Stonecipher MR, Jorizzo JL, White WL, Walker FO, Prichard E. Cutaneous changes of dermatomyositis in patients with normal muscle enzymes: dermatomyositis sine myositis? *J Am Acad Dermatol* 1993; 28: 951–956.
8. Trautmann C, Abdel-Naser MB, Soehnchen R, Detmar M, Orfanos CE. Pramyopathische versus amyopathische Dermatomyositis. *Hautarzt* 1995; 46: 47–52.
9. Katayama I, Sawada Y, Nishioka K. The seborrhoeic pattern of dermatomyositis. *Br J Dermatol* 1999; 140: 978–979.
10. Grafe A, Wollina U, Tebbe B, Sprott H, Uhlemann C, Hein G. Fibromyalgia in lupus erythematosus. *Acta Derm Venereol* 1999; 79: 62–64.
11. Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. *Arthritis Rheum* 1990; 33: 160–179.

*Accepted September 8, 2000.*

Per Thune  
Dermatology Section, OMNIA Romeriksklinikken, NO-2010 Strømmen, Norway.