

Table Summary of Cases

Patient	Sex	Age, y	Presenting Symptom	Lesion Site	History of Atopy	Results
1	F	5	Dyshidrotic eczema	Side and dorsum of toes	Yes	Potassium dichromate +++
2	F	11	Acute eczema	Dorsum of feet	No	Potassium dichromate +++
3	F	15	Dyshidrotic eczema	Dorsum of toes	Yes	Colophony ++
4	F	7	Dyshidrotic eczema	Plantar aspect of toes	No	Potassium dichromate ++
5	F	4	Pustulosis	Soles and dorsum of feet	Yes	Potassium dichromate +++
6	F	7	Dyshidrotic eczema	Dorsum of feet	Yes	Potassium dichromate +++ Cobalt chloride ++
7	F	3	Acute eczema	Dorsum of feet and heels	Yes	Potassium dichromate + Mercapto mix ++

reactions, 5 had a history of atopic dermatitis. While some authors sustain that allergic contact dermatitis is less common in patients with such a history, others claim that it is equally common, and still others claim that it is more common.⁷ Whatever the case, we believe that patch testing could be a useful diagnostic aid in children with foot dermatitis, of allergic or other origin, in whom lesions do not improve with appropriate treatment.

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L. Gámez,* I. Reig, N. Martí, Á. Revert, E. Jordá

Servicio de Dermatología, Hospital Clínico Universitario de Valencia, Spain

*Corresponding author.

E-mail address: lucieta_gp@yahoo.es (L. Gámez).

Dermatomyositis and Livedoid Vasculopathy as the Initial Manifestation of a Tumor

Dermatomiositis y vasculopatía livedoide: primera manifestación de una neoplasia

To the Editor:

Dermatomyositis is an idiopathic inflammatory myopathy that is associated with very characteristic skin manifestations.¹ In many cases, the disorder is accompanied by occult neoplasms,^{2,3} and patients with dermatomyositis and associated skin ulcers have been observed to have a poorer prognosis than those without.^{4,7} Many of the ulcers described in the literature are clinically compatible with atrophie blanche-like lesions.

We describe the case of a 72-year-old man with a 3-month history of poikiloderma-like plaques on the chest, the back, and the top of the thighs; he also experienced

occasional episodes of flushing and swelling around the eyes. Physical examination revealed diffuse erythema with a poikilodermal appearance in the lumbar area, together with erythematous papules on the metatarsophalangeal and interphalangeal joints and periungual telangiectasia.

Results of a complete blood count, coagulation studies, and biochemistry analyses performed to corroborate a suspected diagnosis of dermatomyositis were all normal, as were chest radiograph and abdominal ultrasound findings. Autoantibodies and tumor markers were negative. The histopathologic study of a biopsy specimen taken from the erythematous plaque on the abdomen showed interface dermatitis with interstitial mucin deposits (Figure 1). These findings confirmed the diagnosis of dermatomyositis and treatment was initiated with oral corticosteroids, 0.5 mg/kg body weight.

The patient's general health progressively deteriorated during follow-up and he also developed dysphagia for solids, prompting him to visit the emergency department. Examination revealed considerable worsening of skin lesions; the erythema had spread to the upper and lower limbs and the upper back,

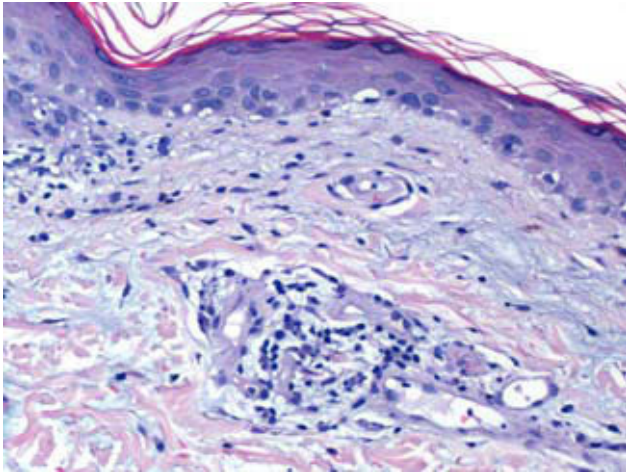


Figure 1 Foci of compact orthokeratotic hyperkeratosis with slight epidermal thinning and a moderate superficial inflammatory infiltrate formed by lymphocytes, together with interface dermatitis with vacuolar degeneration and Civatte bodies (hematoxylin-eosin staining, original magnification $\times 40$).

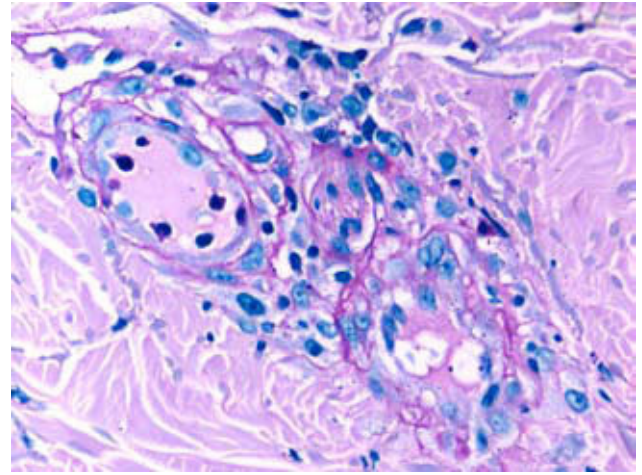


Figure 3 A homogeneous dermis and thrombosed vessels in the papillary dermis with occupation of the lumen by homogeneous eosinophilic, fibrillar material suggestive of fibrin-platelet thrombi (Periodic acid-Schiff staining, original magnification $\times 200$).

and there were also livedo reticularis-like ulcers, which, on healing, left porcelain-white atrophic scars (Figure 2). The patient also had considerable muscle weakness in the upper and lower limbs. It was decided to hospitalize the patient for stabilization and additional tests.

The analyses performed during admission showed a marked elevation of the muscle enzymes (creatinase kinase, 653 IU/L) and elevated tumor markers (carcinoembryonic antigen, 39.4 ng/mL and cytokeratin fragment 21-1, 5 ng/mL). The barium swallow study showed filiform stenosis of the pharyngoesophageal junction but the findings on upper gastrointestinal endoscopy were normal. The cranial computed tomography (CT) scan and muscle biopsy results were also normal although the electromyogram demonstrated a myopathic pattern.

Histopathologic study of the atrophic lesions showed thrombosis of the dermal vessels, which were surrounded

by fibrin deposits (Figure 3), leading to a diagnosis of livedoid vasculopathy.

The CT scan of the chest, abdomen, and pelvis revealed enlarged right subcarinal lymph nodes and a subcarinal mass with a histologically confirmed intermediate cell-type undifferentiated carcinoma. The diagnosis was refined to dermatomyositis with livedoid vasculopathy as the initial manifestation of lung cancer. Despite the initiation of palliative chemotherapy, the patient died of respiratory failure and hemodynamic instability.

Dermatomyositis, which consists of characteristic skin lesions and muscle weakness, was first described by Bohan and Peter¹ in 1975. The skin manifestations include a variety of alterations including Gottron sign, Gottron papules on the back of the interphalangeal joints of the hands, heliotrope rash, shawl sign, V-neck rash, mechanic's hands, cuticle dystrophy with telangiectasia, poikiloderma-like plaques, and subcutaneous calcinosis. Muscle involvement includes symmetric proximal muscle weakness, with or without respiratory muscle involvement or dysphagia, and alterations of muscle function tests (biopsy, electromyography, and/or serum muscle enzymes).

Livedoid vasculopathy, which was described by Milian in 1929, is characterized by the appearance of ulcers with a livedoid or reticular pattern that predominantly affect the lower limbs; these ulcers take weeks or months to heal and leave characteristic porcelain-white atrophic scars.^{6,7} The main cause of livedoid vasculopathy is hypercoagulability, which leads to thrombosis of the dermal vessels and subsequent ulceration.

Dermatomyositis with ulceration is more common in young patients. In our review of the literature,^{4,7} we saw that ulcers in elderly patients with dermatomyositis might be an indicator of poorer prognosis, and that in many cases, these ulcers are clinically compatible with ulcers due to livedoid vasculopathy.



Figure 2 Diffuse erythema with poikiloderma. Whitish scars with a livedoid appearance.

Careful consideration must be given to the possibility of occult neoplasms in dermatomyositis in middle-aged to elderly patients.^{3,8,9} The most common cancers found in association with dermatomyositis are ovarian, pancreatic, and lung cancers.

We have presented a case of dermatomyositis associated with cancer of the lung and presenting livedo reticulatus ulcers due to livedoid vasculopathy. We wish to stress the importance of screening for occult neoplasms in elderly patients with dermatomyositis, particularly when they have extensive skin manifestations.

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F. Vílchez,* E. Camacho, I. Alcaraz, N. López, E. Herrera

Servicio de Dermatología, Hospital Universitario Virgen de la Victoria, Málaga, Spain

*Corresponding author.

E-mail address: fvilchezm@hotmail.com (F. Vílchez).

Curettage for the Treatment of Molluscum Contagiosum: A Descriptive Study

Tratamiento mediante curetaje de moluscos contagiosos: estudio descriptivo

To the Editor:

Molluscum contagiosum (MC) is a viral skin infection and a frequent reason for consultation. The condition mainly affects children, sexually active individuals, and immunosuppressed patients.¹ In immunocompetent patients, MC is a self-limiting infection and usually resolves spontaneously within 6 months to 4 years, hence treatment is not always necessary.²⁻⁴

Multiple therapeutic options are available: *a)* surgical techniques (physical destruction of the lesions by cryotherapy or curettage), *b)* topical agents that produce a local inflammatory response by causing irritation (eg, 0.7%-0.9% cantharidin or 5%-20% salicylic acid), and *c)* topical immune-response modifiers (0.3%-3% imiquimod or cidofovir gel or cream). The choice of treatment depends on the patient (age, number of lesions, lesion sites, complications, history of atopic dermatitis or immunosuppression, fear, occupation, recreational activities, distance to medical center, etc), and on physician expertise (this can improve cure rates with the different therapeutic modalities).⁴⁻⁶

Few studies have investigated more commonly used or suggested treatments such as curettage and cryotherapy.⁷ The purpose of this study was to evaluate MC cases treated

by curettage in our department and the factors associated with a higher rate of therapeutic failure.

A descriptive study was conducted. Patients who consulted for MC between June 16, 2008 and March 15, 2009 were recruited and underwent treatment of all lesions by curettage in the Dermatology Department of Complejo Hospitalario Arquitecto Marcide-Novoa Santos, Ferrol, Spain.

In each case, the following variables were recorded in accordance with a pre-established protocol: sex, age, association with atopic dermatitis, number of household members affected, number of MC lesions at the first visit, anatomic site of the lesions (subdivided into head, neck, trunk, arms, legs, and pubic-anogenital region), number of body areas affected, and number of lesions 2 months after curettage.

The diagnosis of MC was based on the characteristic appearance of frequently umbilicated papules with a diameter of less than 1 cm. Patients were considered to be cured if they had no MC lesions. If necessary, a topical anesthetic cream of lidocaine plus prilocaine (EMLA cream) was applied under an occlusive bandage 1 hour before the procedure.

A descriptive study of the variables included was performed, with the quantitative variables expressed as the mean (SD) and qualitative variables as percentages. Categorical variables were analyzed using the χ^2 test and quantitative variables using the *t* test. The statistical analysis was performed using SPSS 15.0; *P* values less than .05 were considered significant.