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CASE AND RESEARCH LETTERS

Deep Dissecting Hematoma or Stage IV Dermatoporosis

Hematoma profundo disecante o estadio IV de dermatoporosis

To the Editor:

The term dermatoporosis has recently been proposed to refer to the structural changes that appear with age.¹

The signs and symptoms start to develop after 60 years of age and are complete between 70 and 90 years. The morphological signs of dermatoporosis include senile purpura, skin atrophy, and scarring. The loss of protective function leads to delayed healing, lacerations after minimal trauma, and the formation of deep dissecting hematomas that, if not drained, give rise to large areas of necrosis.²

We present the case of a 93-year-old woman with a history of type 2 diabetes mellitus, systemic hypertension, hypertensive heart disease, chronic respiratory insufficiency, ductal breast carcinoma, and trigeminal neuralgia.

She was on the following treatment: clopidogrel (75 mg/d), acarbose (100 mg/8 h), metformin chlorhydrate (850 mg/d), lactulose (2 sachets [20 g]/d), acetaminophen (650 mg/8 h), carbamazepine (100 mg/d), furosemide (40 mg/d), diltiazem (120 mg/12 h), venlafaxine (75 mg/d), anastrozole (1 mg/d), enalapril (20 mg/d), and inhalers (salbutamol and ipratropium bromide). She was admitted for a hematoma that had appeared 5 days earlier on the left lower limb. The patient stated she had suffered a mild trauma, developing erythema and mild intermittent pain in the leg left 10 days earlier (Figure 1).

There was a hematoma measuring 20 cm in its largest diameter on the anterolateral aspect of the left leg. A year earlier she had developed a hematoma of 25 by 15 cm on the right leg that required surgical drainage and use of vacuum-assisted closure treatment (Figure 2). Ultrasound revealed a large hematoma on the anterolateral aspect of the leg left, extending from the popliteal region to the ankle, and located mainly in the subcutaneous tissue. It had a thickness of approximately 5 cm (Figure 3). Surgical debridement was performed with subsequent topical cleansing and dressing, achieving progressive reepithelialization.

The complete blood count with formula and platelet count was normal, except for a hemoglobin of 8.9 g/dL,

2.58 × 10⁶/μl red blood cells, and a hematocrit of 24.7%. The partial thromboplastin time was normal. Cryoglobulins and anticardiolipin antibodies were negative. The liver and renal function tests were normal and antineutrophil cytoplasmic antibodies (ANCA) were negative.

Dermatoporosis is the term used to refer to the clinical manifestations and complications of cutaneous insufficiency. Four stages have been described. Stage 1 is characterized by the presence of senile purpura, skin atrophy, and scars. Stage 2 also includes some localized lacerations. In stage 3 there are multiple lacerations and delayed healing. Stage 4 is associated with deep dissecting hematomas that give rise to large areas of necrosis.²

Deep dissecting hematomas appear on the legs after minimal trauma. The vessels bleed easily due to age-related fragility and their position beneath an atrophic skin. The blood from the vessels collects between the subcutaneous tissue and the muscle fascia and presents clinically as red, edematous areas with locally increased temperature; this can lead to clinical confusion with cellulitis and treatment with oral antibiotics. If the subcutaneous hematoma is not drained at that stage, the skin becomes ischemic and large areas of necrosis develop, requiring extensive surgical debridement.

The differential diagnosis of conditions that progress to large areas of necrosis must include vascular occlusive syndromes that affect the skin, mainly those of noninflammatory cause, although some of inflammatory origin can produce clinical lesions with minimal inflammation and with skin necrosis. These syndromes present clinically with retiform purpuric lesions that can progress to areas of necrosis.³

In a series of 34 patients with dermatoporosis, the deep dissecting hematoma was situated in the legs.⁴ The mean age of the patients was 81.7 years and 85.3% were women.

The previously prescribed treatments of those patients included inhaled corticosteroids (12%) and anticoagulant medication in 29% (acetylsalicylic acid, clopidogrel, and acenocumarol). The patients stated they had suffered an injury to the leg and reported pain, erythema, and edema, though without fever or systemic symptoms. Half of the patients presented disorders associated with slow healing, such as diabetes mellitus, chronic venous insufficiency, arterial insufficiency, or polyneuropathies.

In patients with signs of dermatoporosis and with a history of diabetes, venous or arterial insufficiency,



Figure 1 Large hematoma in the right leg with a superficial necrotic ulcer.



Figure 2 Ulcer on the other leg after drainage and subsequent vacuum-assisted closure treatment.

polyneuropathy, or on prolonged corticosteroid treatment or anticoagulants, caution is required when evaluating painful, erythematous nodular lesions in the legs.⁴

We present a case of deep dissecting hematoma in a 93-year-old patient on treatment with clopidogrel and inhaled corticosteroids.



Figure 3 Ultrasound of the leg right with a large hematoma located mainly in the subcutaneous tissue, with a thickness of about 5 cm.

References

1. Saurat J.H. Dermatoporosis. The functional side of skin aging. *Dermatology*. 2007; 215:271-2.
2. Kaya G., Saurat J.H. Dermatoporosis: A chronic cutaneous insufficiency/fragility syndrome. Clinicopathological features, mechanisms, prevention and potential treatments. *Dermatology*. 2007; 215:284-94.
3. Jones A., Walling H. Retiform purpura in plaques: A morphological approach to diagnosis. *Clin Exp Dermatol*. 2007; 32:596-602.
4. Kaya G., Jacobs F., Prins C., Viero D., Kaya A., Saurat J.H. Deep dissecting hematoma: An emerging severe complication of dermatoporosis. *Arch Dermatol*. 2008; 144:1303-8.

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