A 28-year-old man with an asymptomatic firm plaque on his right shin: what is your diagnosis?

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Received: 5 May 2019
Accepted: 15 May 2019

CLINICAL PRESENTATION

A 28-year-old man referred to the dermatology clinic with an asymptomatic firm, well demarcated violaceous plaque with bumpy surface on his right medial upper shin since two years ago. At first, the lesion was an erythematous patch and gradually became like a plaque. He had pain and sensation of heaviness in his leg (Figure 1). He had no other skin lesions and was otherwise healthy. There was no family history of the same skin lesion. There were no clinically significant abnormalities in laboratory evaluation. The result of serum screening for antinuclear antibodies (ANA) by ELISA was negative. The color Doppler sonography of the right leg demonstrated multiple varicose veins in the medial aspect of the right leg draining into the distal part of the right greater saphenous vein and incompetency of the right saphenofemoral junction. A punch biopsy was performed on the plaque.

Iran J Dermatol 2019; 22: 82-84

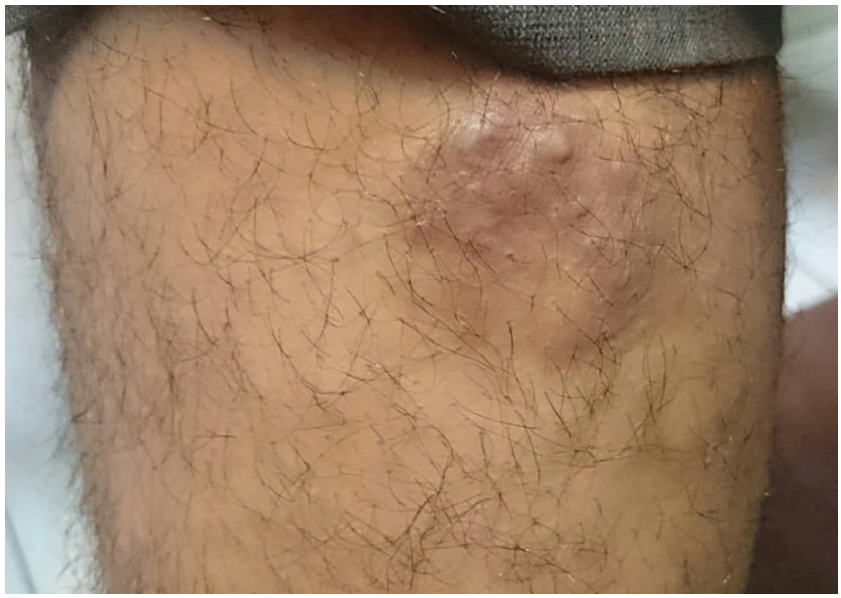

Figure 1. A 28-year-old man with an asymptomatic firm plaque on his right shin.

Diagnosis

Dermal focal mucinosis due to venous insufficiency

Microscopic Findings

Histolopathogical examination of the punch biopsy showed mild acanthosis with elongation of rete ridges and wide separation of collagen bundles in the upper and mid-dermis filled with bluish-gray thin strands and beads (interstitial mucin) and few eosinophils and plump fibroblasts in the upper and mid-dermis with minimal perivascular lymphocytic infiltrate (Figures 2 and 3). Alcian blue stain showed heavy mucin deposition in the upper and mid-dermis (Figure 4).

DISCUSSION

Dermal focal mucinosis is a condition characterized by localized areas of dermal deposition of mucin, an amorphous substance composed of hyaloronic acid and sulfated glycosaminoglycans [1,2].

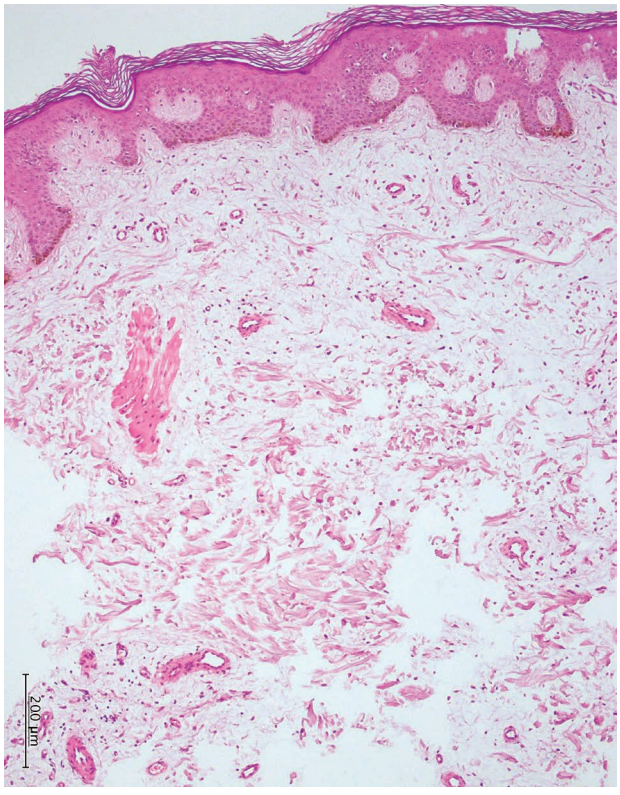

Figure 2. Mild acanthosis and focal basal hypermelanosis in the epidermis; separated collagen bundles and scattered fibroblasts mostly in the upper and mid dermis (H&E stain, 100×).

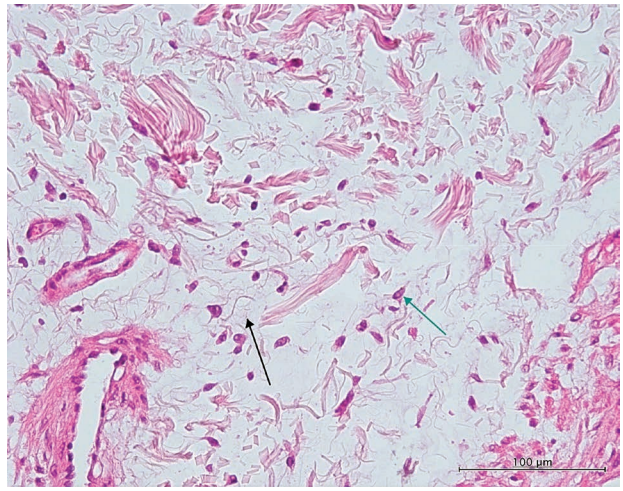

Figure 3. Separated collagen fibers and interstitial bluish stringy mucin (black arrow) and spindle-shaped fibroblasts (green arrow) (H&E stain, 400×).

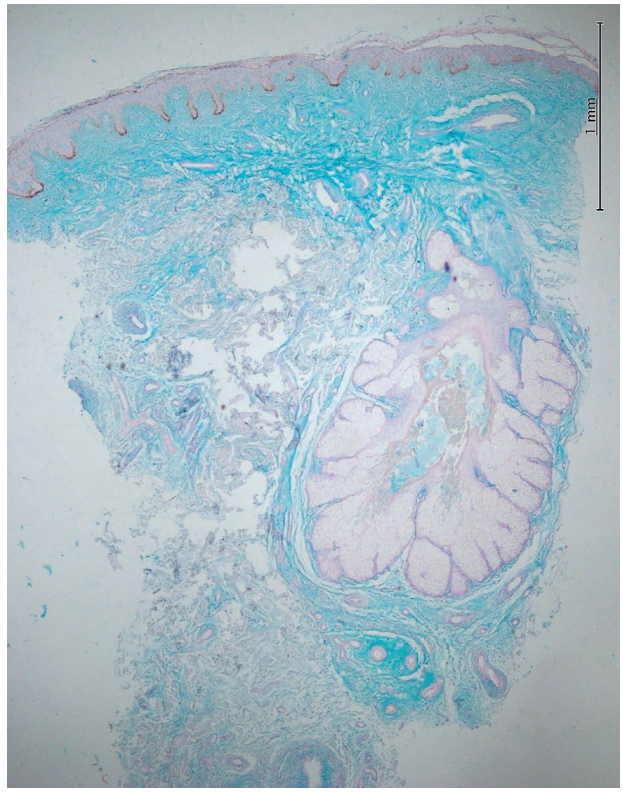

Figure 4. Dermal mucin deposition, predominantly in the superficial and mid dermis (Alcian Blue stain, 40×).

In the localized variants of dermal mucinosis, the patients develop small firm waxy papules or nodules and plaques. It usually appears on the upper and lower extremities, but it may occur at the sites of trauma or scar [3,4]. Minimal morbidity is associated with mucin deposition, but the pruritus can be intense [5]. This condition needs to be excluded

from scleromyxedema, which is usually associated with monoclonal gammopathy or thyroid disease, in dermal mucinosis; the skin is the only site of involvement and it has no association with sclerosis or systemic disease [2,4,6]. There are reports about its occurrence as the adverse effect of biological therapy [7,8] or its association with obesity [9,10], but in this case there is an association with venous insufficiency.

The pathogenic mechanism responsible for dermal mucin deposition in the setting of venous insufficiency is unclear, but hypoxia may play a central role. When venous insufficiency occurs, fibrinogen leaks into the surrounding tissues and the ensuing edema decreases the local oxygen delivery. Chondrocytes, in response to reduced oxygen tension, have been shown to increase the production of hyaloronic acid, the primary component of dermal mucin. Similarly, local tissue hypoxia, in response to venous insufficiency, could potentially increase the fibroblast biosynthetic activity and dermal hyaluronan production [1].

Dermal mucinosis in the setting of venous insufficiency is characterized by abundant dermal(upper and middle) mucin deposition, causing the separation of the collagen fibers, increase in the small blood vessel density, slightly thickened vessel wall, and no inflammatory infiltrate [7,9].

It does not require therapy and a wait and see approach is recommended. Topical application of corticosteroid or pimecrolimus or tacrolimus may be of some benefit. In the setting of venous insufficiency, if the ensuing hypoxia is decreased, the process may be halted; thus, compression stockings and laser treatment to ablate the new vessels are therapeutic options [1,4].

The patient was referred to a vascular surgeon to repair the varicose veins. At the 4-month follow up after vascular surgery, the induration and diameter of the lesion was less but the color change in the lesion did not returned to normal. The patient is currently under follow-up by dermatology department.

Conflict of Interest: None declared.

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