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Dermoscopy of early non-ulcerated livedoid vasculopathy

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Abstract

Livedoid vasculopathy is a rare disease related to a hypercoagulable state. It can lead to painful and chronic relapsing ulcerations. We report dermoscopic findings of early, non-ulcerated livedoid vasculopathy in five patients. A mixture of ivory-white atrophic areas with multiple erythematous papules in the center of the lesions is characteristic. With dermoscopy, these papules correspond to glomerular vessels. In the periphery of the lesions some telangiectatic linear vessels complete the picture, although they could be absent in some patients.

Keywords: livedoid vasculopathy, leg ulcers, pain, dermoscopy, glomerular vessels

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Introduction

Livedoid vasculopathy is an uncommon disease and is a purely cutaneous form of ischemia. The disease is particularly painful due to vascular occlusion, which commonly progresses to ulceration and subsequently heals slowly over weeks or months. It does not show a complete clearing of the lesion, but gives rise to pearly atrophic scars (white atrophy), punctate telangiectasia, and brownish pigmentation accompanied by a racemous livedo (1).

The disease can be accompanied by atrophie blanche (AB), a porcelain-white scar that may be seen at the base of a healed ulcer. Livedoid vasculopathy is responsible for about 1% of leg ulcers (2). The lower legs, in particular the dorsum of the feet and ankles, are most often affected.

The disease is more common in patients over 45. Venous stasis can also be associated with this disease. Other comorbidities are arterial hypertension and treatment with anticoagulants. A recent study in Germany estimated the percentage of leg ulcers in those patients to be as high as 14.3% (3).

Patients and methods

We report the use of dermoscopy to further characterize such lesions in patients with early, non-ulcerated livedoid vasculopathy without clotting disorders. The duration of these complaints was between 6 and 12 months.

Dermoscopic examination was performed using a DermLite camera with polarized light (10× magnification; 3Gen, Dana Point, CA). The DermLite cam contains polarization filters, and therefore immersion liquids do not need to be applied during skin examination.

Five consecutive patients (four females and one male between 43 and 50 years old) with painful livedoid vasculopathy of the ankles without ulcerations and without venous insufficiency were evaluated.

Results

Although our patients had no ulceration, all five showed variable amounts of ivory-white areas surrounded by erythematous lesions. The erythematous lesions had glomerular-like vessels in all five patients (Figs. 1 and 2). In three patients, some telangiectatic linear vessels were noted in the periphery of the lesions (Fig. 2). Pigmentary changes were completely absent.

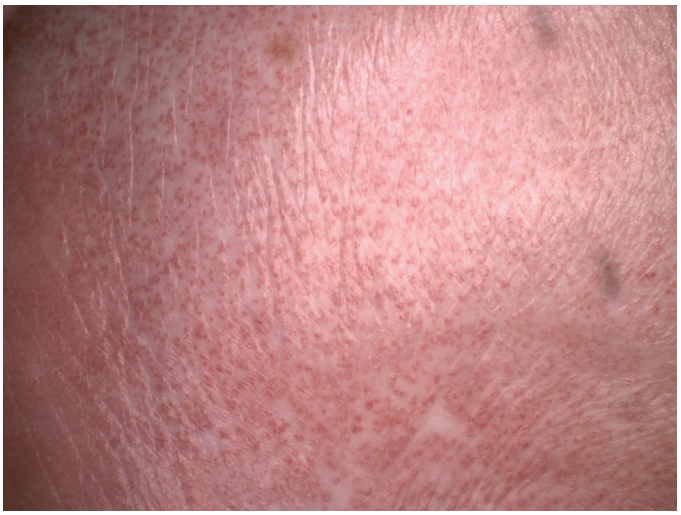

Figure 1 | Early lesion of livedoid vasculopathy on the ankle with ivory-white areas and multiple small red papules.

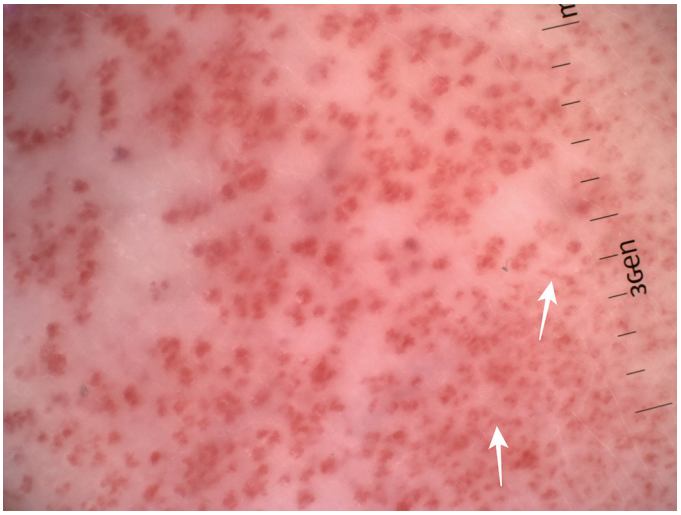

Figure 2 | Dermoscopy of early livedoid vasculopathy with polarized light. Multiple glomerular vessels are seen in the center of the lesions interrupted by ivory-white atrophic areas. Some telangiectatic linear vessels can be seen (arrows) in the periphery (right side).

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Discussion

Our study has two major limitations: a) the small number of patients and b) the lack of histology. Nevertheless, it is of interest because there is not much information available on this subject. Here we present dermoscopic features of early livedoid vasculopathy without ulcerations. There is only one other study by Hu et al. reporting on the use of dermoscopy in livedoid vasculopathy. They investigated nine female patients with ulcerated livedoid vasculopathy (4). The ivory-white atrophic scar-like areas they noted were also seen in our patients. Because they had no history of ulceration, ulceration does not seem to be a *conditio sine qua non*. We suggest that these ivory-white lesions are a result of tissue fibrosis. Hu et al. observed some hyperpigmentation in a reticular pattern in the periphery of the lesions (4). This seems to be a secondary change either due to ulceration with pronounced inflammation or due to concomitant venous insufficiency. In our study, hyperpigmentation was not visible. The major finding in our study and in the study by Hu et al. was the formation of glomerular vessels (4). We also observed some telangiectatic linear vessels in the periphery of the lesions as described elsewhere (4). We suggest that these vascular formations might be later symptoms.

Glomerular vessels are not specific to livedoid vasculopathy. They have been described in chronic sun-damaged skin with precancerous lesions (5–7), in the periphery of hyperkeratotic Bowen's disease (8) and basal cell carcinoma (9), in poromas (10), in psoriasis (11), and in cutaneous leishmaniasis (12, 13). In contrast to other tumorous or non-tumorous cutaneous lesions, the glomerular vessels in early livedoid vasculopathy show a more regular and even distribution over the entire lesion, which might be a more characteristic feature.

Livedoid vasculopathy is caused by a hypercoagulable state, leading to obstruction of small dermal blood vessels by intraluminal thrombi. In addition, the platelets may be characterized by increased aggregation ability (Table 1). The histopathology of livedoid vasculopathy is characterized by intraluminal thrombosis, proliferation of the endothelium, and segmental hyalinization of dermal vessels. Treatment of choice is with oral rivaroxaban (14).

Table 1 | **Important procoagulant factors in livedoid vasculopathy.**

Factor	Description
Protein C deficiency	Protein C is a vitamin K–dependent anticoagulant protein that inactivates coagulation factors Va and VIIIa, and its deficiency (< 55%) results in a thrombophilic state.
Protein S deficiency	Protein S is a cofactor of protein C and inactivates the coagulation factors Va and VIIIa; type 1 and type 3 are known as quantitative defects, whereas type 2 is a qualitative defect that has been infrequently observed.
Factor V Leiden (FVL) mutation	FVL thrombophilia is caused by a mutation in the gene for factor V (F5G1691A mutation resulting in the amino acid substitution FVR506Q).
Antithrombin (AT) III deficiency	Hereditary AT deficiency is rare; the autosomal dominant trait typically reduces functional AT levels to 40 to 60% of normal.
Prothrombin gene mutation	Prothrombin G20210A mutation.
Hyperhomocysteinemia	May occur isolated, but is also a feature of inherited metabolic disorders, including homocystinuria, due to mutation in the CBS gene, and N(5,10)-methylenetetrahydrofolate reductase deficiency, caused by mutation in the MTHFR gene (607093); homocysteinemia/homocystinuria and megaloblastic anemia can result from defects in vitamin B12 (cobalamin; cbl) metabolism.

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