Clinical and dermoscopic features of livedoid vasculopathy

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To the Editor: Livedoid vasculopathy (LV) is a thrombo-occlusive disorder involving superficial and mid-dermis vessels, characterized by painful erythematous or purpuric macules or papules, small crusted ulcers, and porcelain-white atrophic scars that remain after several months.[1,2] Dermoscopy is a non-invasive and effective image analysis technology[3] that can aid dermatologists in visualization of the pigmentation pattern and vascular structure, as well as other morphologic features of the epidermis and dermis. This study was performed to identify the clinical and dermoscopic features of LV in patients with LV who were treated in our hospital from October 2017 to August 2018. The study was approved by the Ethical Committee of the First Affiliated Hospital of Zhengzhou University (No. 2018-YB-05). The study was performed in accordance with the established guidelines and regulations. We have obtained all appropriate patient consent forms. In the form, the patients have given their consents for their images and other clinical information to be reported in the journal.

Dermoscopic examination was performed using a Canon D60 digital camera (Canon, Japan) with Dermlite Foto dermoscopy (3 GEN, San Juan Capistrano, CA, USA). The diagnosis of LV was confirmed by histopathology in all patients. Clinical data were recorded including patient sex and age, disease course, and lesion characteristics. All data were analyzed using SPSS Statistics, version 22.0 (IBM Corp, Amonk, NY, USA). Mean ± standard deviation and median values were used to describe quantitative data. Correlations of qualitative data were assessed using the Chi-squared test. P values <0.05 were considered statistically significant.

Twenty patients with LV (15 women and 5 men; mean age: 20.5 ± 12.6 years [range: 9–56 years]) were included in this study. The duration of illness ranged from 2 to 60 months (median, 12 months). The lesions were typically on bilateral lower limbs, primarily the ankles, and dorsum of the feet. Ten (50%) patients exhibited LV on the calf, ankle, and dorsum of the foot; eight (40%) patients exhibited LV on the ankle and dorsum of the foot; one (5%) patient exhibited LV on the calf; and one (5%) patient exhibited LV on the dorsum of the foot. There were no significant sex-related differences in the incidence site (P > 0.05).

The most common clinical features were painful erythematous or purpuric macules or papules, varying in size, and shape (n = 20, 100%) [Figure 1A]. Some patients exhibited satellite infarction and painful ulcers (n = 17, 85%). Central ivory or porcelain-white stellate atrophic scars and punctate telangiectasia (n = 17, 85%) and peripheral brownish pigmentation (n = 8, 40%) were visible in some patients.

The pathogenesis of LV is unclear; it is currently presumed to be mainly related to blood hyper-coagulability and autoimmunity. Among our patients, one was rheumatoid factor positive, one exhibited low C3 level, and two were anti-nuclear antibody positive with low titer. Furthermore, protein S activity was elevated in two patients, protein S was reduced in three patients, protein C activity was reduced in two patients, folate level was reduced in one patient, and homocysteine levels were elevated in three patients.

Histopathology analyses of patients with LV revealed segmental hyalinization or fibrinoid degeneration of dermal vessels, proliferation of the endothelium, and intraluminal thrombosis (n = 20, 100%) [Figure 1B]. Some patients exhibited mild perivascular lymphocyte infiltration (n = 10, 50%), red blood cell extravasation (n = 10, 50%) and basal cell layer hyper-pigmentation (n = 8, 40%).

In our patients, we found multiple dermoscopic features: (1) pink or white background (n = 20, 100%); (2) vascular structures (eg, irregular linear vessels and glomerular vessels) around the lesion (n = 20, 100%) [Figure 1C]; (3) central shallow crusted ulcers or ivory-white structureless areas with peripheral pigment networks (n = 20, 100%) [Figure 1D]. These findings were consistent with the results of prior studies.[4,5] Some patients (n = 8, 40%) exhibited blurred dark-red globules, similar to glomerular vessels (ie,
punctiform vessels that resemble renal glomeruli and exhibit histopathology findings of curled and dilated capillaries in dermal papilla) but with distinct color; these globules were not previously reported. The most common dermoscopic features of LV were pink or white background with irregular linear and glomerular vessels; the specific features of LV were crusted ulcers or ivory-white structureless areas with peripheral pigment network.

Previous studies of dermoscopic features of LV reported the presence of central shallow crusted ulcers or ivory-white atrophic scar-like areas, as well as a pigment network and greater numbers of telangiectatic linear and glomerular vessels; they also demonstrated the relationships of these dermoscopic features with pathology findings. Pathologic dilated or thickened blood vessels in the dermis led to the dermoscopic linear and glomerular vessels; hyper-pigmentation of the basal cell layer or melanin within melanophages led to the presence of a pigment network; and dermal fibrosis led to the manifestation of a central ivory-white area.[4] Similar to previous reports regarding the dermoscopic features of LV, our study revealed central small crusted ulcer areas or porcelain-white areas, as well as peripheral reticular pigmentation and greater numbers of vessel structures. Blurred dark-red globules were also observed in some patients. We presume that the dark-red unstructured areas are associated with vascular embolism and may become painful ulcers and infarcts during disease progression.

If dermoscopy reveals central white structureless areas with a peripheral pigment network and greater numbers of vascular structures, as well as occasional dark-red structureless areas, a diagnosis of LV should be considered. Pathology analysis of the lesion is necessary to confirm the diagnosis.

Conflicts of interest

None.

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