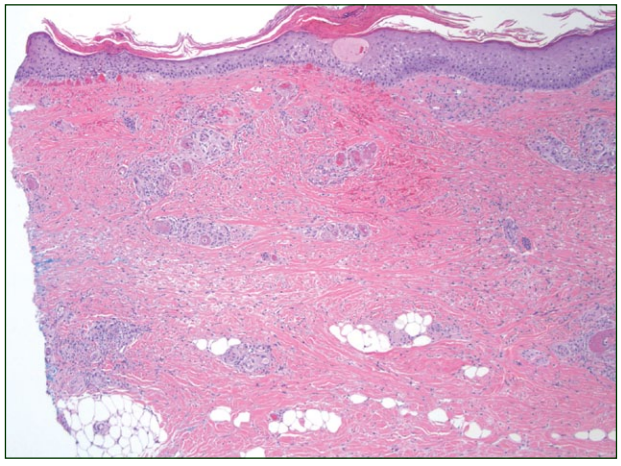
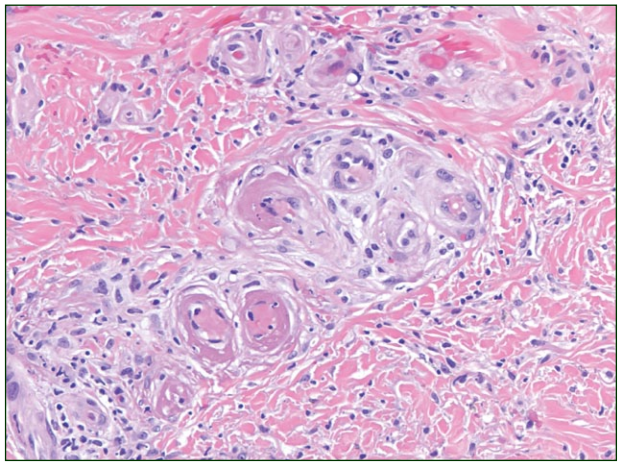


H&E, original magnification ×4.



H&E, original magnification ×20.

A biopsy was taken from the left medial malleolus of a 49-year-old woman with a history of bilateral, painful, and recurrent venous ulcers. The best diagnosis is:

a. cholesterol thromboembolism
b. leukocytoclastic vasculitis
c. lipodermatosclerosis
d. livedoid vasculopathy
e. polyarteritis nodosa

PLEASE TURN TO PAGE 181 FOR DERMATOPATHOLOGY DIAGNOSIS DISCUSSION

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The authors report no conflict of interest.
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Livedoid Vasculopathy

Livedoid vasculopathy (LV), also known as atrophie blanche, is associated with chronic venous insufficiency.[1] The clinical presentation of LV consists of multiple, recurrent, painful, punched out ulcers on the lower legs that heal as white, atrophic, stellate scars. The epidermis may be atrophic, ulcerated, or necrotic, with associated dermal sclerosis (Figure 1).[2] Upper and mid dermal capillaries are dilated and occluded with fibrinous material, and vessel walls are hyalinized and surrounded by a scant perivascular lymphocytic infiltrate (Figure 2).[3] Leukocytoclastic vasculitis consists of angiocentric neutrophilic inflammation with vessel wall alterations (eg, fibrinoid necrosis, inflammatory cells within vessel walls), and karyorrhectic debris is not present (Figure 3).[1] Lipodermatosclerosis (LDS) also clinically presents on the lower legs and is associated with chronic venous insufficiency. Vascular changes may be similar to LV with pericapillary fibrin deposition in the upper dermis and dermal sclerosis; however,

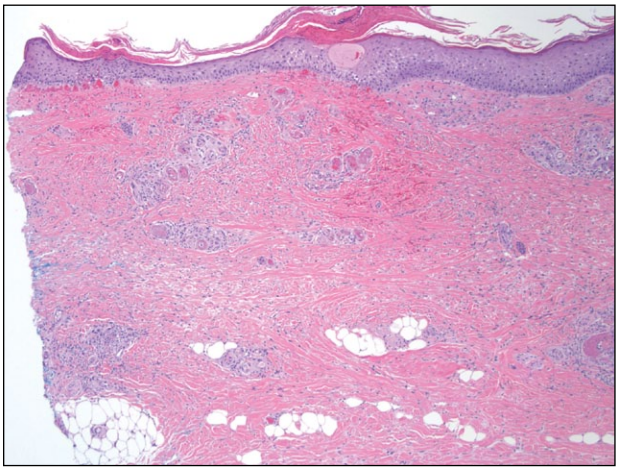

Figure 1. In livedoid vasculopathy, the epidermis is associated with dermal sclerosis that often is atrophic, ulcerated, or necrotic (H&E, original magnification ×4).

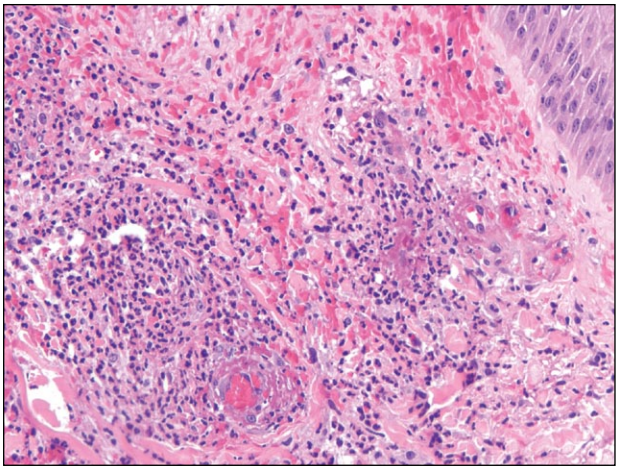

Figure 3. Angiocentric neutrophilic inflammation and vessel wall alterations characteristic of leukocytoclastic vasculitis (H&E, original magnification ×20).

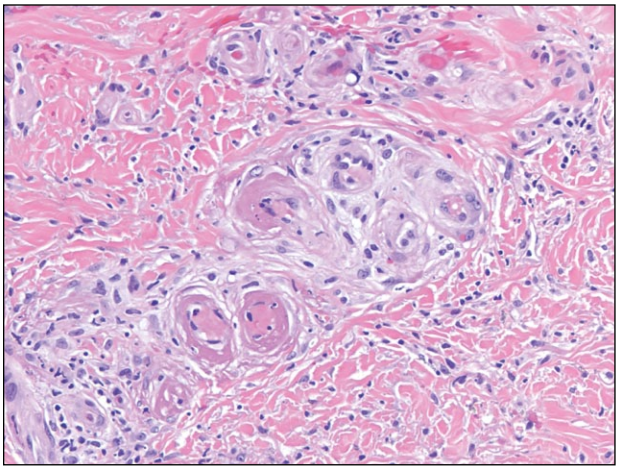

Figure 2. In livedoid vasculopathy, upper and mid dermal capillaries are dilated and occluded with fibrinous material, and vessel walls are hyalinized and surrounded by a scant perivascular lymphocytic infiltrate (H&E, original magnification ×20).

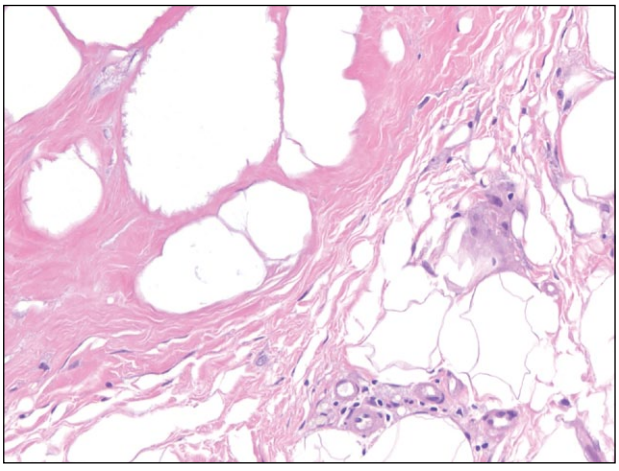

Figure 4. Lipodermatosclerosis is characterized by a predominantly mixed septal and lobular panniculitis with variable fat necrosis, septal hyalinization, and inflammation, often with lipomembranous changes (H&E, original magnification ×40).

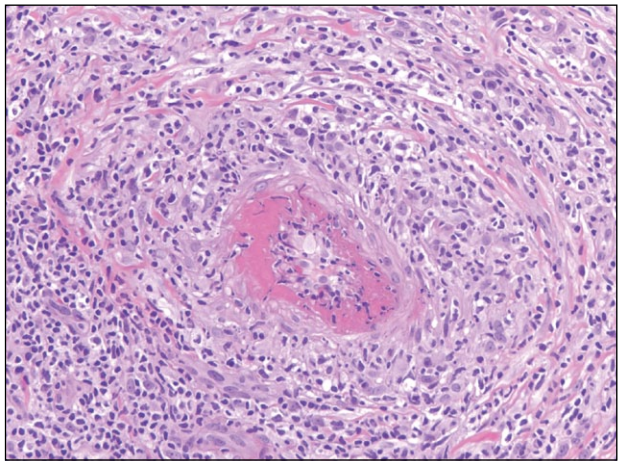

Figure 5. Polyarteritis nodosa is a form of leukocytoclastic vasculitis that predominantly involves vessels in the deep dermis and subcutis (H&E, original magnification ×20).

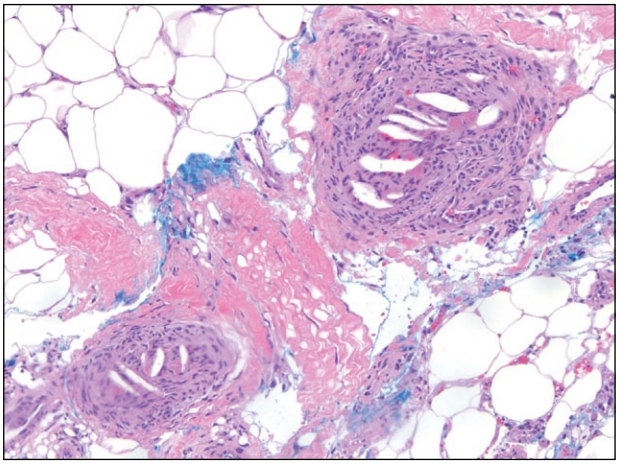

Figure 6. Cholesterol clefts within intravascular thrombi characteristic of cholesterol thromboembolism (H&E, original magnification ×20).

LDS usually is seen as a predominantly mixed septal and lobular panniculitis with a variable (dependent on the time of biopsy) amount of fat necrosis, septal hyalinization, and an inflammatory infiltrate composed of lymphocytes and foamy macrophages.[3] Lipomembranous changes often are present in LDS (Figure 4). Polyarteritis nodosa is a form of leukocytoclastic vasculitis of vessels in the deep dermis and subcutis (Figure 5), whereas LV involves the dermis and lacks leukocytoclasia. Cholesterol thromboembolism is characterized by cholesterol clefts within intravascular thrombi (Figure 6).

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