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Lower Extremity Ulcers and the Satisfied Search

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A 62-year-old man with hypertension, diabetes mellitus, and coronary artery disease (CAD), on peritoneal dialysis, presented with a nonhealing left lower extremity ulcer (Figure 1). Treatment with empiric antibiotics showed no improvement and cultures remained persistently negative. A surgical specimen revealed pathological changes consistent with calciphylaxis (Figures 2 and 3).

With a mortality between 30% and 80% and a 5-year survival of 40%,[1-3] calciphylaxis, or calcific uremic arteriolopathy, is devastating. Dialysis and a calcium-phosphate product above 60 mg^2/dL^2 increased the index of suspicion (our patient = 70).[4] As visual findings may resemble vasculitis or atherosclerotic vascular lesions, biopsy remains the mainstay of diagnosis. Findings include intimal fibrosis, medial calcification, panniculitis, and fat necrosis.[5]

Management involves aggressive phosphate binding, preventing superinfection, and surgical debridement.[6] The evidence for newer therapies (sodium thiosulfate, cinacalcet) appears promising,[7-10] while the benefit of parathyroidectomy is equivocal.[11] Despite therapy, our patient developed new lesions (right lower extremity, penis) and opted for hospice services.

FIGURE 1. A 3-cm × 5-cm lesion on the lateral portion of the distal left lower extremity with surrounding erythema and eschar.

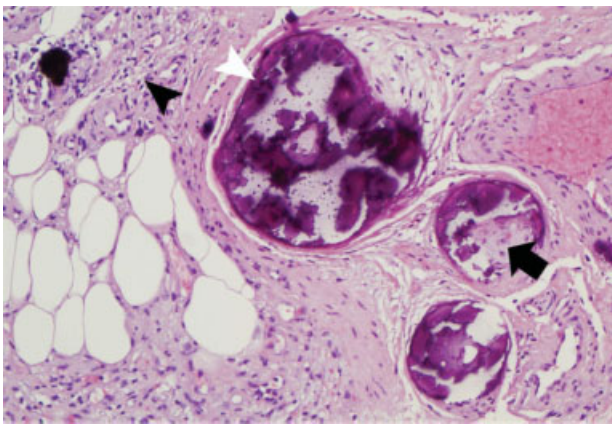

FIGURE 2. Histopathological specimen showing epidermal ulceration (white arrowhead), dermal fibrosis (black arrowhead), arterial mural calcification (white arrow), and arterial thrombosis (black arrow).

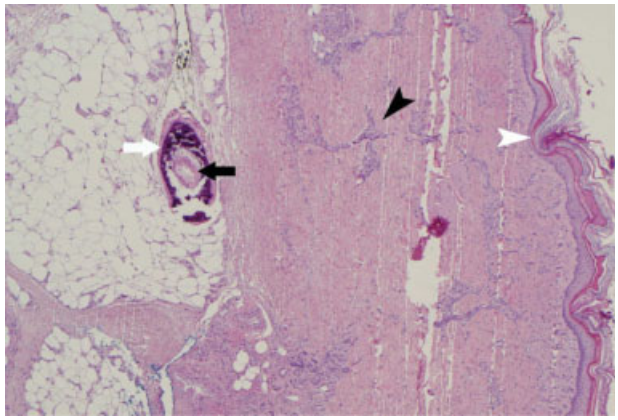

FIGURE 3. Calcification (white arrowhead) and thrombosis (black arrow) of small-sized to medium-sized hypodermic arterioles in a background of fat necrosis and septal panniculitis (black arrowhead), consistent with calciphylaxis.

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