malignant cells correlate with their metastatic potency, our findings provide evidence that natalizumab might have a protective effect on melanoma development and give reassuring data for its use in treatment of MS.

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OBSERVATION

Cholesterol Embolization Syndrome With an Atypical Proximal Presentation Simulating Calciphylaxis

Cholesterol embolization syndrome (CES) is associated with endovascular procedures or anticoagulation, which disrupt athromomatous plaques within large arteries releasing cholesterol crystals. These crystals lodge in the vasculature of various organs, most commonly the skin. Cutaneous involvement classically presents in the distal lower extremities as livedo reticularis (49%), gangrene (35%), cyanosis (28%), ulceration (17%), nodules (10%), purpura (9%), and splinter hemorrhages. We report a case of CES with proximal cutaneous lesions mimicking calciphylaxis.

Report of a Case | A woman in her 50s with an approximately 20 pack-year smoking history experienced a myocardial infarction, prompting anticoagulation therapy with heparin, cardiac catheterization, and stent placement at an outside hospital. Postoperatively, she initiated aspirin and clopidogrel treatment. Seven days after initiation, her lower back, buttocks, and thighs developed painful erythematous plaques, which progressed to nodules and ulcerations over days to weeks. A biopsy performed at the outside hospital suggested vasculitis. Prednisone treatment was initiated, and clopidogrel therapy was discontinued. As her ulcers continued to enlarge and her proximal arms developed mottling, she was referred to our clinic for evaluation.

At presentation, she had normal blood pressure and no gastrointestinal or neurologic complaints. On the buttocks, thighs, and right calf were firm, tender, erythematous nodules and ulcerations with overlying eschar (Figure 1). Livid pleomorphic plaques were present on the lower back, buttocks, thighs, and proximal arms bilaterally. The distal extremities appeared normal. We considered CES, given the livedoid changes and history of endovascular intervention, while calciphylaxis was considered based on the appearance and location of the lesions. We also considered medium-vessel vasculitis.

A 6-mm punch biopsy specimen from an ulcer on the buttock demonstrated cholesterol clefts in the lumen of deep dermal arteries without evidence of calciphylaxis or vasculitis (Figure 2). Aside from leukocytosis (white blood cell count, 18,000/μL), findings of other blood tests were normal, including vasculitis assays such as antinuclear antibody and anti-neutrophil cytoplasmic antibody tests. Results of retinal evaluation by our ophthalmology colleagues were unremarkable.
The histologic findings were diagnostic for a proximal cutaneous presentation of CES without systemic involvement, likely caused by anticoagulation therapy (heparin) and endovascular intervention (cardiac catheterization). It is unclear if aspirin or clopidogrel contributed to the manifestations. Supportive measures were initiated, including wound care, smoking cessation, and weight loss. Her cardiologist initiated pain management measures as well as treatment with statins and antihypertensive agents. The skin lesions healed over 1 year.

Discussion | In CES, disruption of atheromatous plaques causes microemboli to occlude downstream vessels. Cholesterol embolization syndrome due to endovascular procedures usually occurs hours to weeks after the procedure, whereas anticoagulation-associated CES presents weeks to months after initiation of anticoagulation.

Cutaneous involvement of the distal lower extremities manifesting as livedo reticularis, pain, and/or blue toes with intact pulses is the most frequent finding, seen in 35% to 90% of patients. Common systemic manifestations include hypertension (66%) and acute renal failure (33%). Other findings include gastrointestinal manifestations (19%-33%); Hollenhorst plaques in the retinal arterioles (22%); and, rarely, central nervous system involvement.

When found on histopathologic examination, cholesterol clefts within vascular lumen are pathognomonic for CES. As vessel involvement may be focal and segmental, step sections into the tissue block may be required to identify the involved vessel(s).

Standard treatment is supportive and includes local wound care. Resection of ischemic tissue can provide symptomatic improvement. Additionally, referral to a cardiologist should be considered. Statin therapy may stabilize atherosclerotic plaques, reducing recurrent embolization. Anticoagulation and endovascular intervention directed at the emboli source should be reserved for life-threatening cases, as additional dislodgement of cholesterol crystals may occur following such treatment.

Given the cutaneous features of CES, dermatologists often play a role in diagnosing this condition. Proximal cutaneous lesions are atypical but do occur and may cause diagnostic confusion with calciphylaxis. A high index of suspicion in the appropriate clinical context and a low threshold for skin biopsy are required to diagnose CES.
Acinetobacter baumannii Emerging as a Multidrug-Resistant Skin and Soft-Tissue Pathogen: Parallels to Methicillin-Resistant Staphylococcus aureus

Over the last 30 years, the gram-negative coccobacillus Acinetobacter baumannii has risen to microbiologic notoriety for its ability to successfully evade nearly all available antibiotics. It is now a well-established source of nosocomial bacteremia and pneumonia, known to cause hospital outbreaks, particularly in the intensive care unit. Less often it represents a source of nosocomial skin and soft-tissue infection (SSTI) in the setting of war wounds, surgical sites, and burns.1

Most reports of A baumannii infection, both nosocomial and community-acquired (CA) SSTI, involve cellulitis of peau d’orange appearance with overlying vesicles that, when untreated, progress to necrotizing fasciitis with coalescent bullae.2 The condition is uncommon and has been reported solely in compromised hosts.3 To our knowledge, A baumannii has never been reported as a cause of CA-SSTI in a healthy patient.

However, unpublished and anecdotal cases of increasingly drug-resistant A baumannii presenting as SSTI in healthy patients are known to exist. Molecular typing experiments reveal community and nosocomial A baumannii isolates to be genetically distinct, existing as separate reservoirs with unique virulence and resistance patterns. Until recently, multidrug resistance was considered a hospital-based phenomenon.4 We report herein the case of a healthy woman with CA multidrug-resistant A baumannii.

Report of a Case | A woman in her 50s with no comorbidities or history of hospitalization presented with tender, violaceous, indurated dermal plaques with central ulceration and peripheral erythema and edema (Figure, A) that had progressively enlarged over the previous 7 to 8 months. Tissue culture results were specific for A baumannii with sensitivity to polymyxin B and tigecycline. The patient was admitted for treatment with intravenous tigecycline and discharged to home with an inpatient-administered peripherally inserted central catheter to facilitate a subsequent 2 weeks of treatment. Following treatment, the patient underwent extensive wound care for 6 months prior to reepithelialization with resulting scarring, dyschromia, and neuropathic pain (Figure, B).

Discussion | The evolution of A baumannii infection bears a striking similarity to the now established methicillin-resistant Staphylococcus aureus (MRSA) epidemic; MRSA was initially regarded as a nosocomial pathogen transmitted between health care facilities and limited to a small number of strains with distinct virulence patterns. While the first CA cases occurred in those with predisposing risk factors, it eventually began ap-