tissue culture, and from a papule for hematoxylin-eosin staining. Within an hour of obtaining the specimens, the pathologist reported frozen section findings of small organisms within the vasculature suggestive of either fungal spores, likely histoplasmosis, or staphylococcal bacteria. Permanent sections revealed a large neutrophilic pustule with inflammation extending throughout the dermis and prominent necrosis with basophilic structures filling necrotic vascular spaces (Figure 2A). Tissue Gram staining confirmed gram-positive cocci in the vessels (Figure 2B). Tissue culture, blood culture, bronchial washings, and fluid from bilateral empyemas all grew CA-MRSA.

Results from transthoracic echocardiogram and bone scans were negative. Magnetic resonance images of the brain showed multifocal lesions, likely septic emboli. Findings from workup for possible immunodeficiency were negative. The patient was sent to a rehabilitation facility to complete a 6-week course of intravenous vancomycin.

Discussion | The original classifications of CA-MRSA and hospital-acquired MRSA are no longer distinct: CA-MRSA now causes infections in health care settings, and hospital-acquired MRSA spreads in the community.1 In a study on disseminated Staphylococcus in children, MRSA was the causative agent in all cases. Children aged 5 to 12 years were most vulnerable, and trauma was found to be a common precipitating factor.2

Septic vasculitis caused by septicemia from Staphylococcus is an immune-complex negative, small-vessel neutrophilic vasculitis.3 Clinical lesions of septic vasculitis are characterized by retiform purpura, petechiae and ecchymoses, vesiculopustules, hemorrhagic bullae, and ulceration.4 In cases of septic emboli without evidence of vasculitis, some prefer the term septic vasculopathy. One study5 compared 32 patients with bacterial sepsis, cutaneous lesions, and biopsy-proven septic vasculopathy. Cutaneous lesions were an early finding of sepsis in most patients (91%; n = 29). Most cases (n = 18) involved the development of lesions simultaneously or within the first 24 hours of sepsis that were disseminated rather than localized. Most lesions presented as purpuric papules and plaques, followed by petechiae, vesicles and bullae, pustules, distal ischemia, and nodules.5 Septic vasculopathy had a mortality rate of 20% in this study,5 making early recognition and treatment essential.

In addition to cutaneous findings, staphylococcal sepsis can involve the lung, heart, kidneys, brain, bone, and joints,6 and it is important to evaluate for these potential sites of involvement. As this case report highlights, consulting dermatologists can play a role in identifying disseminated infections by recognizing the diverse skin manifestations and performing appropriate biopsies for analysis by frozen section, permanent section, and tissue culture.

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Safe Use of Therapeutic-Dose Oral Isotretinoin in Patients With a History of Pseudotumor Cerebri

Drugs common in the treatment of acne vulgaris, such as isotretinoin and isotretinoin, have been reported in association with pseudotumor cerebri (PTC), which can lead to severe, irreversible symptoms, including vision loss. There is a paucity of data on isotretinoin use in patients with prior PTC.

Report of Cases | Case 1. A female patient in her teens presented with a 2-year history of severe, nodular, cystic acne of her face (Figure 1A), chest, and back with significant scarring. Her hormonal workup was unrevealing. Two years earlier, she had received minocycline for acne and had developed an unusually severe headache; lumbar puncture confirmed PTC. Prompt discontinuation of minocycline treatment led to long-term PTC symptom resolution. However, her acne was recalcitrant to treatment for several months with oral contraceptives, spironolactone (200 mg/d), topical antibiotics, and topical retinoids; she also required frequent intraliesional triamcinolone for persistent painful cysts.

Isotretinoin therapy was initiated at 10 mg/d for 7 days, and the dose was increased slowly over 4 months to 40 mg/d. The patient noted skin dryness and facial erythema but developed no signs or symptoms concerning for PTC. After 9 months of isotretinoin therapy (cumulative dose, 120 mg/kg), her acne had markedly improved (Figure 1B).

Case 2. A woman in her late 20s presented with polycystic ovarian syndrome, severe inflammatory nodular acne with scarring (Figure 2A), and a history of minocycline-associated PTC (diagnosed at age 16 years via lumbar puncture). Her acne had not responded adequately to several topical retinoids at maximum concentrations, concomitant benzoyl peroxide, topical and oral antibiotics, spironolactone, and oral contraceptives. Multiple clinicians had avoided isotretinoin use given her history of PTC.
Owing to the recalcitrance of her acne to combination therapy with several agents, low-dose isotretinoin therapy was begun (initiated at 10 mg/d), and the dose gradually increased to therapeutic levels (cumulative dose, 140 mg/kg over 7 months of treatment). She experienced dramatic acne improvement with only mild residual scarring (Figure 2B). The only adverse effects were skin dryness and hypertriglyceridemia, both of which resolved after isotretinoin therapy was completed.

Case 3. A female patient in her teens presented with severe scarring, nodular acne and a history of PTC (confirmed by ophthalmologic and neurologic evaluation) that had developed after she had started isotretinoin therapy 3 years earlier. Her PTC resolved at that time with discontinuation of isotretinoin therapy. As with patient 2, her acne did not respond adequately to several topical retinoids at maximum concentrations, concomitant benzoyl peroxide, topical and oral antibiotics, spironolactone, and oral contraceptives. And again, as with patient 2, multiple clinicians had avoided isotretinoin use given her history of PTC.

Low-dose isotretinoin therapy was begun (initiated at 30 mg/d), and the dose increased gradually to therapeutic levels (cumulative dose, 120 mg/kg over 9 months of
Discussion | Pseudotumor cerebri is characterized by symptoms of increased intracranial pressure: headache, visual disturbances (blurry vision, diplopia), nausea and vomiting, and stiff neck. It is commonly idiopathic, primarily affecting women of childbearing age who are overweight. Less frequently, it can occur in association with other disease conditions (usually endocrinologic abnormalities such as polycystic ovarian syndrome). The pathophysiological mechanisms of drug-associated PTC are not fully understood. It has been postulated that tetracyclines may reduce cerebrospinal fluid outflow at the arachnoid villi, while oral retinoids (through excess retinols and retinol-binding protein) may be directly toxic to arachnoid villi function.

The patient was seen for follow-up every 6 months. The only adverse effect was skin dryness, which resolved after isotretinoin treatment was completed. Slow but progressive growth of the hyperpigmented patch on her left lateral ankle. The lesion was a 3.6 × 3 cm triangular hyperpigmented, asymptomatic patch consisting of confluent mottled black and brown macules and several satellite lesions consistent with an EB nevus.

The patient returned at age 9 years and the parents noted slow but progressive growth of the hyperpigmented patch on her left ankle. On examination, several brown macules coalescing into a 5.5 × 5-cm patch with surrounding satellite lesions was seen on her lateral ankle (Figure 1A). Biopsy was performed of a representative area, showing a compound nevus with large nested melanocytes and bridging at the dermal-epidermal junction consistent with an atypical compound nevus (Figure 1B).

The patient was seen for follow-up every 6 months. The atypical nevus remained unchanged until age 12 years, when 2 discrete areas of regression were noted (Figure 2A). On dermoscopy, discontiguous and irregular homogenous blue, brown, and gray areas with gray-brown structureless areas and a negative pigment network were observed, suggestive of malignant transformation. No induration was palpated except at the previous biopsy site.

Repeated biopsy of the areas of clinical regression showed atypical nevus with areas of dermal fibrosis (Figure 2B). Gene expression testing for 23 melanoma genes was performed yielding a melanoma gene expression test (Myriad myPath; Myriad Genetics) score of −7.1, where scores from −16.7 to −2.1 are consistent with a benign nevus.

Melanoma Gene Expression Markers for Surveillance of Epidermolysis Bullosa Nevi Malignant Transformation

The evaluation of malignant changes in epidermolysis bullosa (EB) nevi remains a challenge for clinicians; herein, we introduce the idea of employing melanoma gene expression markers to aid in thorough histopathologic examination of suspicious EB nevus lesions.

Report of a Case | A young girl with epidermolysis bullosa simplex (EBS) initially came under our care at age 4 years. Two years after her first presentation, she developed an irregular brown-black patch on her left lateral ankle. The lesion was a 3.6 × 3 cm triangular hyperpigmented, asymmetrical patch consisting of confluent mottled black and brown macules and several satellite lesions consistent with an EB nevus.

The patient returned at age 9 years and the parents noted slow but progressive growth of the hyperpigmented patch on her left ankle. On examination, several brown macules coalescing into a 5.5 × 5-cm patch with surrounding satellite lesions was seen on her lateral ankle (Figure 1A). Biopsy was performed of a representative area, showing a compound nevus with large nested melanocytes and bridging at the dermal-epidermal junction consistent with an atypical compound nevus (Figure 1B).

The patient was seen for follow-up every 6 months. The atypical nevus remained unchanged until age 12 years, when 2 discrete areas of regression were noted (Figure 2A). On dermoscopy, discontiguous and irregular homogenous blue, brown, and gray areas with gray-brown structureless areas and a negative pigment network were observed, suggestive of malignant transformation. No induration was palpated except at the previous biopsy site.

Repeated biopsy of the areas of clinical regression showed atypical nevus with areas of dermal fibrosis (Figure 2B). Gene expression testing for 23 melanoma genes was performed yielding a melanoma gene expression test (Myriad myPath; Myriad Genetics) score of −7.1, where scores from −16.7 to −2.1 are consistent with a benign nevus.

Letters