A 44-year-old man presented with an eruption of pruritic erythematous plaques on his lower extremities of 6 months’ duration that were unresponsive to antifungal cream or topical corticosteroid. His medical history was notable for chronic inflammatory demyelinating polyneuropathy (CIDP), which was diagnosed 1 year prior to presentation and was associated with lower extremity weakness and imbalance of 3 years’ duration. Punch biopsy of lesional skin showed a superficial and deep perivascular and interstitial lymphohistiocytic infiltrate with abundant interstitial neutrophils and rare eosinophils. He was diagnosed with interstitial granulomatous dermatitis (IGD), and the eruption improved with the initiation of oral dapsone 50 mg twice daily.

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**Case Report**

A 44-year-old man presented with an eruption of pruritic erythematous plaques on his lower extremities of 6 months’ duration; they were unresponsive to the antifungal agent terbinafine hydrochloride cream 1% and the topical steroid triamcinolone acetonide cream 0.1%. Onset was not associated with new medications, physical activity, or travel. The plaques were associated with a mild burning to itching sensation. No myalgia or arthralgia was present. His only medication was a daily multivitamin.

His medical history was notable for chronic inflammatory demyelinating polyneuropathy (CIDP), which was diagnosed 1 year prior to presentation. He had experienced gradual worsening of lower extremity weakness and imbalance over 3 years. Neurologic impairment limited to the bilateral lower extremities, including weakness; mild gait ataxia; decreased deep tendon reflexes at the bilateral quadriceps, gastrocnemius, and soleus muscles; and decreased vibratory sensation, had been appreciated. Cerebrospinal fluid examination showed a highly elevated protein level (194 mg/dL [reference range, 14–45 mg/dL]), with other parameters within reference range as well as negative cultures. Magnetic resonance imaging showed normal findings for the head but abnormal enhancement of lumbar nerve roots. Electromyography was interpreted as demyelinating neuropathy. Laboratory testing for complete blood count, erythrocyte sedimentation rate, and vitamin B₁₂ level, as well as serum protein electrophoresis test, antinuclear antibody assay, antineutrophil cytoplasmic antibody test, rheumatoid factor test, and human immunodeficiency virus serology, were negative or within reference range. The patient declined therapy for his CIDP.

On dermatologic examination, annular scaly plaques were present in a bilateral and fairly symmetric distribution limited to the lower extremities, sparing the soles (Figure 1). No linear cords within the skin were palpable. Potassium hydroxide preparation was negative for fungus. Punch biopsy showed a superficial and deep perivascular and interstitial lymphohistiocytic infiltrate with abundant interstitial neutrophils and rare eosinophils (Figure 2A). Leukocytoclasis, degenerated collagen, and amorphous basophilic debris were visible on higher magnification (Figure 2B). Gram, Ziehl-Neelsen, and Gomori methenamine-silver stains were negative for microorganisms. The patient was diagnosed with...
interstitial granulomatous dermatitis (IGD). The eruption did not respond to clobetasol propionate cream 0.05% and it recurred after a 3-week tapering course of prednisone. The eruption improved with the initiation of oral dapsone 50 mg twice daily; however, the medication was discontinued after 2 months due to moderate pancytopenia.

Comment

Interstitial granulomatous dermatitis is a rare dermatosis with diverse clinical features that may be idiopathic, medication induced, or related to autoimmune disease, particularly rheumatoid arthritis and systemic lupus erythematosus. It can present in association with arthritis and with indurated rope-like plaques, though a wide variety of morphologies are now recognized. Medications implicated in this eruption include calcium channel blockers, angiotensin-converting enzyme inhibitors, statins, and tumor necrosis factor α inhibitors. The condition has been successfully treated with oral dapsone, antimalarial agents, cyclosporine, and ironically tumor necrosis factor α inhibitors.

Figure 1. Eruption of erythematous scaly plaques on the dorsal feet and overlying metatarsophalangeal joints, with a striking degree of bilateral symmetry (A, right foot; B, left foot). Photographs courtesy of James B. Paulson, MD, Montezuma, Iowa.

Figure 2. Superficial and deep perivascular and interstitial lymphohistiocytic infiltrate with abundant interstitial neutrophils and rare eosinophils (A)(H&E, original magnification ×40). Leukocytoclasis, degenerated collagen, and amorphous basophilic debris were visible on higher magnification (B)(H&E, original magnification ×400). Photographs courtesy of James B. Paulson, MD, Montezuma, Iowa.
Chronic inflammatory demyelinating polyneuropathy is an acquired neuropathy characterized by progressive weakness and impaired sensorimotor function caused by autoimmune damage to the myelin sheath of peripheral nerves. The clinical presentation and course are highly variable, though the disease has been shown to respond to systemic corticosteroids, intravenous immunoglobulin, and plasmapheresis. Cutaneous findings generally are not described in CIDP, but some cases are associated with systemic lupus erythematosus. According to a PubMed search of articles indexed for MEDLINE using the terms "interstitial granulomatous dermatitis" or "chronic inflammatory demyelinating polyneuropathy" or "polyneuropathy," ours is the first reported case describing coexisting IGD and CIDP. The exact relationship between these disorders remains unclear, though the overlap in clinical presentation is intriguing.

REFERENCES