Case of Acquired Epidermodysplasia Verruciforms
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Abstract
Acquired type epidermodysplasia verruciformis (EDV) has historically been linked to individuals infected with human immunodeficiency virus (HIV) (1) and those on chronic immunosuppressant therapy for solid organ transplant (2). Recently, with the expansion of immunosuppressant drugs being used to treat a variety of immune mediated diseases, there has been an increase in EDV like syndrome presentations in case reports and literature. The trend is evident in an increasing number of adult and pediatric patients treated with biologic agents and immunosuppressant drugs for a variety of conditions such as atopic dermatitis (3), graft-versus-host disease (GVHD) (4), leukemia (5), and in this case systemic lupus erythematosus (SLE). (6). There is no standardized treatment of EDV and/or acquired EDV, which is primarily case based and often delivers variable unsatisfactory results (1).

Case Report
A 36 y.o. African American female with a 12-year history of SLE presented with a 3-year history of persistent flat topped verrucous white macules on her face, neck and back (Fig. 1). Her current regimen for treatment of SLE with renal involvement includes prednisone at 10mg daily plus azathioprine 150mg daily, which was added approximately 3 years ago after failing a trial of cyclosporine. The patient noticed multiple white patches developing on her forehead and cheeks and attributed the new findings to a childhood history of eczema. Biopsy was obtained revealing negative PAS, hyperkeratosis, hypergranulosis, and acanthosis with scattered large keratinocytes with vacuolated sea-blue cytoplasm, consistent with findings seen in epidermodysplasia verruciformis (Fig. 2). The patient denied any family history of similar skin findings, skin cancer, recurrent recalcitrant warts. She denies any prior personal history of warts or similar skin findings during childhood. She was previously negative for HIV testing by her rheumatologist, therefore the diagnosis of acquired epidermodysplasia verruciformis was established secondary to immunosuppression from prednisone and azathioprine.

Treatment included switching azathioprine for plaquenil 200mg BID and initiation of tazarotene 0.05% cream nightly, glycolic acid lotion 15% BID, cimetidine 400mg nightly, and topical imiquimod 3.75%. cream.

Discussion
EDV is a rare autosomal recessive disease associated with mutations of EVER1 and EVER2 genes on chromosome 17(7). These mutations downregulate cell-mediated immunity via zinc transport proteins and predispose those affected to all subtypes of the human papilloma virus (HPV). Patients with EDV are particularly susceptible to the beta subgroups HPV 5, 8, which are not pathogenic in immunocompetent hosts (8). Inherited forms present with recalcitrant flat warts in childhood that later develop flat topped pityriasis versicolor like patches/plaques which progress to non-melanoma skin cancers in sun exposed areas starting at the third and fourth decade (9). The disease is difficult to manage and often disfiguring and disabling, advising those affected to strict sun protection and domiciles in cloudy climates as UV radiation plays a pivotal role in malignant transformation of lesions (10).

References