research examining indoor tanning behavior among the estimated 0.8 million male indoor tanners 40 years or older is warranted given their increased frequency of indoor tanning and the lack of research or interventions focused on this demographic group.

The decrease in indoor tanning may be partly attributable to the increased awareness of its harms. Indoor tanning devices have been classified as carcinogenic to humans, their use has consistently been shown to increase skin cancer risk, and laws restricting access among minors may have changed public perceptions of their safety. In addition, a 10% excise tax on indoor tanning was implemented in 2010, which may have contributed to the decrease in indoor tanning.

This study is subject to certain limitations. Results from the National Health Interview Survey are generalizable only to the noninstitutionalized civilian adult population. In addition, the use of cross-sectional data does not permit a causal inference between behaviors and the frequency of indoor tanning.

The Surgeon General has highlighted the importance of reducing the harms from indoor tanning and of continued public health efforts to identify and implement effective strategies to reduce indoor tanning. Research regarding the motivations of indoor tanners could inform the development of new interventions. Physicians can also play a role through behavioral counseling, which is recommended for fair-skinned persons aged 10 to 24 years. Continued surveillance of indoor tanning will aid program planning and evaluation by measuring the effect of skin cancer prevention policies and monitoring progress.

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Observation

Acantholytic Dyskeratotic Epidermal Nevus

Acantholytic dyskeratotic epidermal nevus (ADEN) is histologically similar to Darier disease (DD), and distinguishing the 2 entities may be challenging. We describe the case of an 18-month-old boy with congenital epidermal nevi, histological acantholytic dyskeratosis, and the presence of sarcoendoplasmic reticulum calcium transport ATPase 2 (SERCA2) protein by immunohistochemical (IHC) staining (Abcam PLC). We concluded that the best diagnosis was ADEN.

Report of a Case | An 18-month-old otherwise healthy Hispanic boy was referred for evaluation of congenital linear verrucous lesions on the upper and lower extremities. The lesions followed the lines of Blaschko. His mother observed that the plantar lesions were painful with ambulation. He had been treated with topical retinoids and steroids since age 3 months without improvement. The patient’s birth and family history were unremarkable. Physical examination revealed hyperpigmented verrucous papules coalescing into plaques in a Blaschkooid distribution on the bilateral lower extremities, left groin, scrotum, and buttocks (Figure 1). Linear plaques were present on the scalp, left palm, left medial foot, and from the right heel to the right dorsal foot. Neither mucosal lesions nor nail abnormalities were present. A shave biopsy of lesional skin from the thigh showed papillomatous epidermal hyperplasia with zones of acantholytic dyskeratosis; IHC staining revealed SERCA2 protein in the lesional skin (Figure 2). In the context of the patient’s congenital presentation and lack of other clinical features or family history of DD, the patient was diagnosed with ADEN. He was subsequently observed for 7 years and continued to be without signs of Darier disease.

Discussion | Epidermal nevi are congenital malformations of the epidermis characterized clinically by verrucoid plaques on the skin often following lines of Blaschko and histologically by papillomatosis, acanthosis, and hyperkeratosis. Ten subtypes have been described. ADEN is a subtype distinguished by its “Darier-like” pattern of acantholysis and dyskeratosis.

Distinguishing between ADEN and DD relies on clinical-pathologic correlation. Both demonstrate histologic loss of adhesion between suprabasal keratinocytes (acantholysis), abnormal keratinization (dyskeratosis) in the form of corps ronds.
and grains, hyperkeratosis, and parakeratosis. Both present clinically with hyperkeratotic papules and warty plaques but differ in their age of onset and associated abnormalities. ADEN is often congenital, while DD typically manifests in the second or third decade of life. Typically, DD has associated nail abnormalities, mucous membrane involvement, palmoplantar tarpits, and punctate keratoses, while ADEN does not.2

Genetic testing is available, as is immunohistochemical staining of tissue for SERCA2 protein expression. The genetic difference between the 2 disorders remains controversial. Darier disease is caused by mutations in ATP2A2, which encodes the SERCA2 pump. Mutations of SERCA2 have been discovered in at least 2 patients with ADEN, leading some clinicians to classify ADEN as segmental DD resulting from postzygotic mosaicism.3 Others maintain that congenital or early childhood onset and linear distribution suggest ADEN and not segmental DD.4 There is 1 reported case of ADEN where lack of ATP2A2 mutation was confirmed by DNA analysis.5

In our patient, IHC staining identified SERCA2 protein in lesional skin. However, the IHC staining does not substantiate normal function of the protein. Genetic testing was not economically feasible for this family but may be appropriate when a patient desires genetic counseling, prenatal diagnosis, or identification of at-risk family members. The Genetic Testing Registry lists several laboratories that will perform sequence analysis of the ATP2A2 gene.6 With 96% specificity, the lack of ATP2A2 gene mutation nearly excludes a diagnosis of DD. Our case supports the premise that ADEN has clinical and prognostic features distinct from DD despite overlapping histopathologic findings. Although IHC staining of SERCA2 can be helpful, further study of genotype-phenotype correlations may allow better distinction of these 2 clinical entities.

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Severe Acne in Female-to-Male Transgender Patients
The number of patients diagnosed as having gender identity disorders (GIDs) has increased in the past decade. Sexual minorities receive little dermatologic interest, although they have specific skin disorders.1 Female-to-male transsexual patients (trans men) receive masculinizing doses of testosterone (T) to induce virilization.2 Our knowledge of the effects of T therapy on the skin of trans men is scarce,3 and very little has been published about it. Previous works conclude that T therapy used in trans men does