Consulted for an asymptomatic hypopigmented and scaly eruption. On examination, he was presented with widespread hypopigmented and mild scaly lesions simulating pityriasis versicolor on the trunk and multiple plane warts on hands.

A 12 year-old boy presented with a history of multiple asymptomatic hypopigmented and scaly eruption since he was 3 years old. There was no family history of skin cancer or similar lesions and no history of consanguinity. Physical examination revealed numerous flat-topped mild scaly papular lesions on face, neck, upper back, extensor surfaces of the forearms, and thighs (Fig. 1 A, B, C, D). Some of them appeared verrucous and other pityriasis versicolor-like. The mucous membranes, hair, and nails were unaffected. The lesions were refractory to ketoconazole cream.

Laboratory findings including complete blood count, urinalysis and serum glucose level were within normal limits. Hepatitis, tumor markers and anti-human immunodeficiency virus (anti-HIV) antibodies were negative.

The biopsy of one of these hypopigmented lesions showed an acanthotic epidermis with a prominent granular layer with dense keratohyaline granules and keratinocytes with clear cytoplasm, vesicular nuclei, and perinuclear halos.

**What’s your diagnosis?**
1. Verruca plana
2. Tinea versicolor
3. Epidermodysplasia verruciformis
4. Benign squamous papillomas

The diagnosis is epidermodysplasia verruciformis (EV).

**DISCUSSION**
Epidermodysplasia verruciformis is a rare lifelong cutaneous disorder associated with a high-risk of skin cancer. Cutaneous lesions are highly polymorphic and consist of pityriasis versicolor–like lesions; wart-like lesions; red macules; and seborrhic keratoses (SK)-like plaques, which usually
manifest on the face, trunk, and extremities.
EV results from an abnormal susceptibility to specific human papillomavirus (HPV) genotypes and to the oncogenic potential of some of them, particularly HPV5 and HPV8. Patients are unable to reject their lesions, and squamous cell carcinomas and Bowen’s disease can develop on sun-exposed areas.
EV is usually an autosomally recessive disorder. Approximately 50% of the patients carry mutations in the EVER 1, 2 genes on chromosome 17q25. There is a resulting defect in cell-mediated immunity, leading to an abnormal susceptibility to HPV infections and a propensity to developing skin cancer. Implicated HPV strains include serotypes 3, 5, 8-10, 12, 14, 15, 17, 19 to 25, and 47. Histopathology of the lesions exhibited typical enlarged vacuolated keratinocytes with pale cytoplasm and round keratohyaline granules in upper epidermal layers. A malignant transformation of skin lesions occurs in more than half of the patients in the fourth and fifth decades of life. The preferential location of premalignant lesions, the forehead and other sun exposed areas, as in our patient, is suggesting a role for ultraviolet light–induced carcinogenesis.
In reviewing the literature for treatment options, there was no evidence of a definitive treatment. Monotherapy with interferons, or in combination with retinoids or imiquinod, has been of value in the treatment of EV as well as surgical treatment.

REFERENCES