

Human papillomavirus-induced periungual pigmented Bowen's disease

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Case report

A 50-year-old male presented with an ill-defined hyperpigmented macule on the left pointer finger (Figure 1). The lesion was reportedly first evident one year prior to presentation. Clinically, the lesion was asymmetrical. Notably, there was central hypopigmentation and obliteration of acral architec-

ture. Differential diagnosis included acral nevus, tinea nigra, subcorneal bleeding, exogenous pigmentation, melanoma and pigmented Bowen's disease.

Dermatoscopically, the lesion was chaotic with regard to the distribution of color (Figure 2). The lesion had different shades of brown and gray and its pattern was structureless. As noticed before by Cameron et al, a structureless pattern is



Figure 1. Clinical view. [Copyright: ©2012 Ramirez-Fort.]



Figure 2. Dermatoscopic view. [Copyright: ©2012 Ramirez-Fort.]

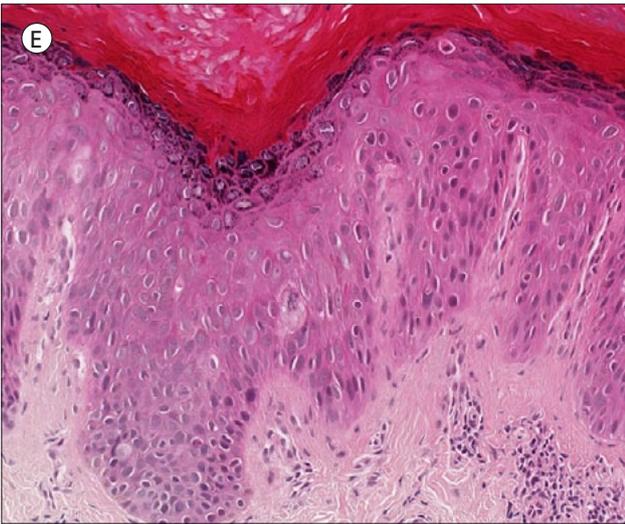
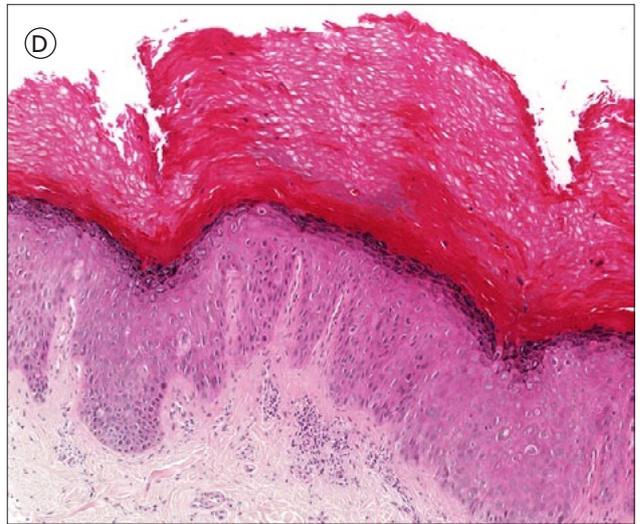
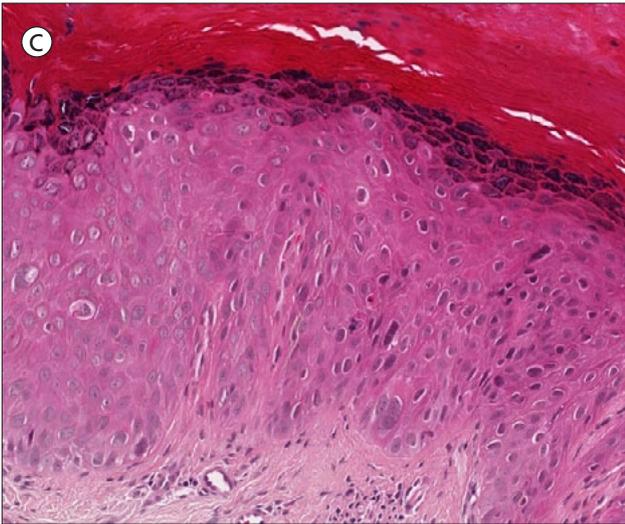
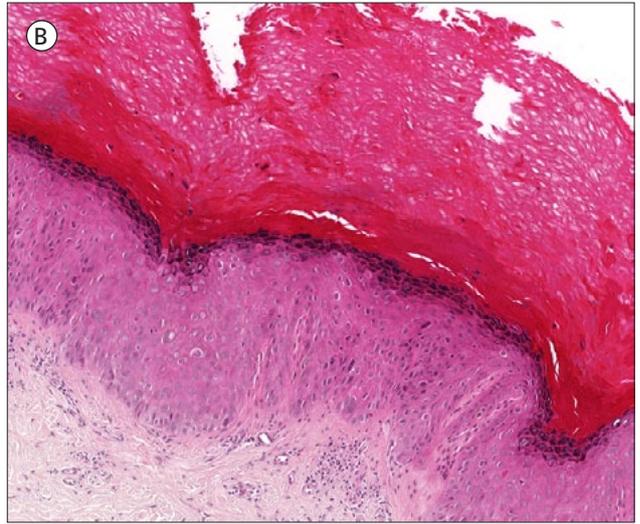


Figure 3A-E. Dermatopathologic view.
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in keeping with the diagnosis of pigmented Bowen's disease [1].

Histopathologic analysis of the lesion demonstrated a pigmented Bowen's disease (Figure 3). There was acanthosis with focal hypergranulosis and parakeratosis. The epidermis was composed of atypical keratinocytes and a few scattered dyskeratotic cells. The superficial dermis had a light perivascular lymphocytic infiltrate. Polymerase chain reaction sampling was positive for human papillomavirus (HPV), further confirming the diagnosis of an HPV-induced pigmented Bowen's disease. The lesion was excised completely and the surgical defect was closed primarily.

Discussion

Bowen's disease may be induced by chronic exposure to forms of electromagnetic radiation, such as ultraviolet light (UV) or x-ray. Bowen's disease may also be induced by infection with HPV, as in the case presented above. Bowen's disease induced by HPV usually occurs on genital skin; multiple regional lesions are termed "bowenoid papulosis."

Bowen's disease is further categorized into pigmented or nonpigmented. It has been speculated that the source of pigment in Bowen's disease may represent a collision between a solar lentigo and Bowen's disease. Although this explanation is reasonable for pigmented Bowen's disease on cutaneous sites chronically exposed to sunlight, it does not explain the pigmentation of "bowenoid papulosis" on genital skin or the case presented above. A more probable hypothesis considers neoplastic pigmentation as a direct reflection of the pigmentation present in the initial keratinocytes to proliferate. The hypothesis holds validity in the setting of a benign seborrheic keratosis or malignant lesion of Bowen's disease.

Some patients with bowenoid papulosis on genital skin develop subsequent periungual Bowen's disease. Reported cases have found the same HPV strains in genital and periungual lesions, suggesting anogenital-digital spread of HPV as a possible etiology for HPV-positive periungual Bowen's disease [2]. The literature reports only one other case of a pigmented, periungual Bowen's disease by Hu et al [3]. Although the HPV status of the lesion is unknown, the group clearly demonstrated the utility of dermatoscopy in evaluation and further monitoring of disease progression.

References

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