Detection of human papillomavirus in lesions of a patient with dermatosis papulosa nigra

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A case study of dermatosis papulosa nigra in a 46-year-old Caucasian. Small brown and red papules were expressed on the face. The clinical and histopathological findings were supported by detection of human papillomavirus type 23 in the skin lesions. Its role in keratinization disorders is discussed. The authors stress that this condition is rarely found in Caucasians.

Summary

**Key Words**
dermatosis papulosa nigra, human papillomavirus, Caucasian male

**Introduction**

Dermatosis papulosa nigra (DPN) is a nevoid defect of pilosebaceous follicles; it is common in the black population but very rare among Caucasians. Patients express small pigmented or reddish papules in the upper part of the face, and less frequent expression of the same in the lower part. The lesions resemble seborrheic keratoses (1,2,3,4).

**Case report**

A 46-year-old Caucasian male noticed verrucous and pigmented lesions on his forehead and malar region, which appeared a few weeks after exposure to the sun in summer. His family history was positive for malignant melanoma but negative for nonmelanomic skin cancer (NMSC).

Physical examination confirmed the expression of reddish papules with a fine verrucous surface, which resembled seborrheic keratoses (Fig. 1).

Analysis of blood samples showed that the man was immunologically competent.

The biopsy of a skin lesion revealed irregular acanthosis, hyperkeratosis and an increased concentration of melanin in the lower epidermis as well as a mild lymphocytic and histiocytic infiltrate around vessels and follicles. No cytopathic effects were observed in the keratinocytes.

For the identification of human papilloma viruses (HPV) two punch biopsies were taken from the affected and non-affected skin in the temporal region. After extraction the DNA was amplified with polymerase chain reaction (PCR) and hybridized. The method applied was described by Nuovo et al (5). HPV type 23 was detected in the lesional skin; results were negative in the non-affected skin.
The clinical and histopathological findings suggested a diagnosis of dermatosis papulosa nigra. Imiquimod (5% cream) was applied overnight three times a week.

At the follow-up evaluation after 12 weeks only a mild improvement was noted and the decision was made to remove the lesions by laser cauterization.

**Discussion**

HPV is a DNA epidermotropic virus inducing common warts, condylomata acuminata, carcinomas of the female genital tract, and also other conditions including epidermodysplasia verruciformis (EV), which is a rare inherited disorder. The above mentioned conditions express a wide spectrum of HPV types. Sometimes even the skin lesions on areas exposed to the sun may be transformed by HPV activity into carcinomas (5).

In recent literature cases of benign keratotic abnormalities were reported as being associated with HPVs (6, 7, 8), and many types of HPV have been recently identified even in normal skin (9).

HPV type 23 has also been detected in lesions of the benign form of EV (10).

Our patient was immunologically competent and the histopathology of the skin lesions did not produce evidence of a viral infection such as koilocytosis, or any other cytopathic effects. One hypothesis to explain the failure of imiquimod therapy is that the HPV is only occasionally present in the relevant skin sites.

The exact role of HPV in keratinization disorders is not known, but, in combination with prolonged exposure to ultraviolet light, it may be responsible for various histopathologic and molecular changes, including the development of NMSC (11, 12).

Further studies will provide useful information regarding the presence of HPV in normal skin and will contribute to a better understanding of the local and systemic involvement of the immunologic system (13).

**REFERENCES**


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