Detection of Human Papillomavirus Type 31 in a Case of Papillated Bowen’s Disease

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CASE REPORT
A 74-year-old male visited our clinic because of an asymptomatic skin lesion on the inner aspect of left thigh, which he had noticed for one year. He denied any history of arsenic ingestion, immunosuppressive therapy or radiotherapy. Past history was unremarkable. There are no lesions suspicious for human papillomavirus (HPV) infection during whole body skin examination. His wife was recently under gynecologic and dermatologic check-up and there is no evidence suggestive of HPV infections such as viral warts or genital dysplasia. Clinically, the lesion is characterized by a 12-mm well-circumscribed, black-brownish, exophytic, hyperkeratotic, verrucous nodule (Fig. 1). The skin tumor was completely surgically excised and sent for pathological examination. Histopathological findings revealed full-thickness cytologically atypical keratinocytes with loss of polarity in the epidermis (Fig. 2A). Numerous mitotic figures and scattered dyskeratotic keratinocytes were noted (Fig. 2B). In addition, prominent vacuolated keratinocytes in the upper third of the epidermis were also evident (Fig. 2C). Owing to the koilocytic cells found in the lesion suggestive of HPV infection, the formalin-fixed paraffin-embedded tissue specimens were used for HPV identification. HPV DNA was detected using consensus primer-mediated PCR assays. HPV genotyping was performed by using commercial EasyChip HPV blot (King Car Yuan Shan Institute, I-Lan, Taiwan). The specimen was positive for HPV type 31. The patient was uneventful without any recurrence after following up for one year.

DISCUSSION
Bowen’s disease, originally described in 1912 by John T, represents a squamous cell carcinoma in situ on the skin. The typical presentation is an irregular, asymptomatic, well-demarcated, scaly or crusted erythematous plaques from a few millimeters to several centimeters in diameter. They can occur anywhere on the skin, including sun-exposed and non-sun-exposed regions of the body. Etiologic factors include chronic arsenic ingestion, ionizing radiation, ultraviolet radiation exposure, immunosuppression and infection with human papillomavirus. Several clinical variants had been described such as periangual, subungual, intertriginous and pigmented Bowen’s disease. Histopathological findings were also extensively reviewed which included psoriasiform, atrophic, verrucous-hyperkeratotic, pigmented, clear cell, pagetoid, mucinous, and sebaceous variants.

HPV are well documented to involve in the pathogenesis of many anogenital dysplasia and cancer development. They are also found to be related to the development...
of genital Bowen’s disease. With the accumulation of many case reports and series, HPV have also been repeatedly detected in extragenital Bowen’s disease. Most of them are on the periungual or other areas of the hands and usually involves a mucosal type of HPV such as HPV-6, -11, -16, -31, -33, -34, -51, -54, -58, -61, -62, and -73. However, the lesion other than the hands such as the neck was also reported.

In 2006, Joannie D. Sun et al. analyzed 26 patients harboring the lesion of Bowen’s disease with an unusual clinicopathologic features. He coined the papillated Bowen’s disease—a distinct variant from other previously described variants. In his article, he concluded that this distinctive lesion was characterized by well-circumscribed, papillated, exophytic and endophytic, sometimes keratotic nodule. The histopathological examination usually revealed keratinocytes with prominent perinuclear halos similar to koilocytes found in HPV infections in addition to a conventional full-thickness epidermal dysplasia. However, the authors failed to demonstrate any HPV DNA in the specimen of all 26 patients. The striking negative results of all 26 cases may be due to lower sensitivity of in situ hybridization or low levels of expression of HPV DNA. The HPV of the specimen, if any, may be detectable by more sensitive screening methods such as polymerase chain reaction or southern blot hybridization.

HPV-31 is the oncogenic genotype of HPV that is notorious for the genesis of many skin diseases such as Bowenoid papulosis and cervical cancer. They had also been found in the genital as well as extragenital Bowen’s disease. According to Hama N et al., the elevated amount of HPV-31 DNA could have led to the emergence of squamous cell carcinoma from Bowenoid papulosis under the condition of decreased cellular immunity. This finding emphasized that HPV-31 are potentially capable of causing malignant transformation of the skin. In our case, it is
shown that mucosal type HPV-31 can also be found extragenitally and cause malignant transformation of the skin.

In conclusion, papillated variant is a distinct type of Bowen’s disease both clinically and pathologically. We had found that mucosal high-risk HPV such as type 31 in our case might have causal relationship to the development of this distinct variant. However, a large scale study should be conducted to define the true relationship between HPV infection and papillated Bowen’s disease.

REFERENCES