

patients. We report a case of a pair of father and daughter suffering from Bowen's disease. A genetic basis for Bowen's disease should be considered and investigated.

A 21-year-old Chinese woman presented to our department in 2007 with papules and plaques on trunk and limbs for more than 10 years. The patient first presented with an asymptomatic plaque about the size of a coin at the age of 10. The number of lesions gradually increased before generalizing rapidly during her pregnancy in 2004. The patient denied a history of exposure to radiation and immunosuppressive therapy. The patient lived in Jianshui County in Yunnan, China, where arsenic pollution is not significantly present. The physical examination revealed brownish, scaling patches and plaques [Figure 1a]. Cutaneous signs of arsenism,<sup>[1]</sup> including hyperkeratosis of the palms and soles and cutaneous hyperpigmentation with "raindrop" depigmentation, were absent. There was also no peripheral lymphadenopathy. Routine laboratory examinations were within normal limits. The biopsy of the lesions over the back and abdomen both revealed epidermal thickening with hyperkeratosis, dyskeratosis, loss of polarity, cellular derangement with atypia and many mitoses, along with variable degrees of acanthosis and chronic inflammatory infiltrates in the upper dermis [Figure 1b]. The immunohistochemical stains for human papillomavirus (HPV) types 16 and 18 were negative [Figure 1c]. Electron microscopy examination revealed intercellular inflammatory infiltrates in the epidermis with mild edema. The basement membrane was intact with the absence of viral particles [Figure 1d].

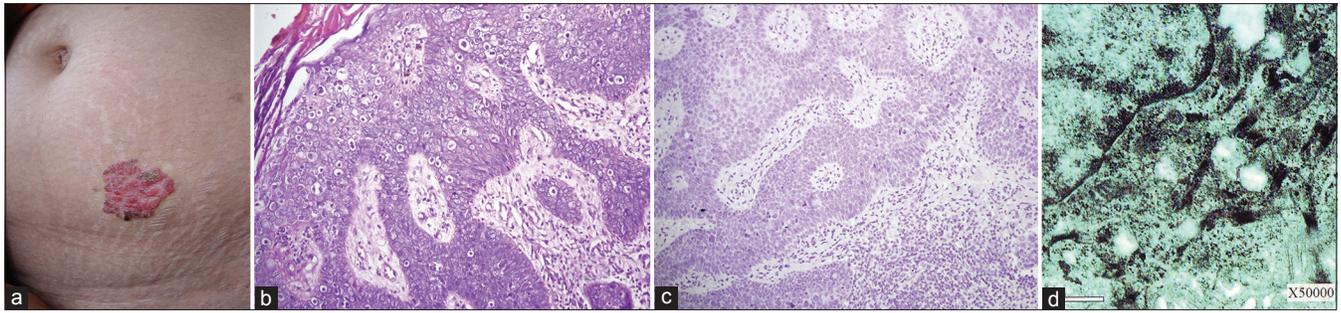
The father of the patient (mentioned in the first case) complained of an asymptomatic salmon-red plaque on his left upper arm for 3 years. He is a farmer by occupation and both patients denied the pre-existence of freckles/tendency to sunburn. Physical examination revealed an erythematous, dome-shaped plaque with crusting on his left upper arm [Figure 2a]. Laboratory examinations were normal. The biopsy of the lesions revealed the same results as the first case [Figure 2b]. The immunohistochemical staining for HPV type 16 and 18 and electron microscopy examination also revealed the same results as the first case [Figure 2c].

The exact cause of Bowen's disease remains unclear, although arsenic poisoning, sun exposure, and HPV infection are likely to be responsible for it. The incidence of Bowen's disease in the Chinese population

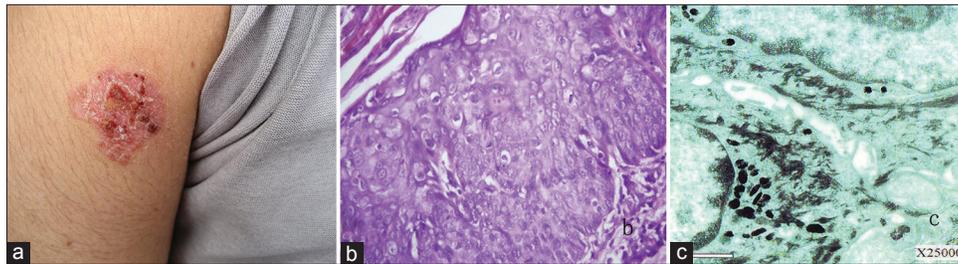
## Familial Bowen's disease

Sir,

Bowen's disease has its highest incidence in the older age groups, while it rarely occurs in younger



**Figure 1: (a) Dark-red, hyperkeratotic flat scaling papules coalescing to form a papillomatous hyperplastic plaque with a discrete border and overlying crust. (b) Cellular derangement with atypia and many mitoses, multinucleated oncocyte (H and E,  $\times 200$ ), (c) The immunohistochemical stains for human papillomavirus (HPV) types 16 and 18 were negative ( $\times 200$ ), (d) No viral particles seen at  $\times 50,000$  magnification under electron microscopy**



**Figure 2: (a) Salmon-red hyperkeratotic flat scaling papules coalescing to form a hyperplastic plaque with a discrete border and overlying crust that measures 2 cm by 2 cm in size, (b) Epidermal thickening with hyperkeratosis, dyskeratosis, papillary hyperplasia, loss of polarity, cellular derangement with atypia and many mitoses, along with chronic inflammatory infiltrates in the upper corium (H and E,  $\times 400$ ), (c) No viral particles were seen at  $\times 25,000$  magnification under electron microscopy**

remains unclear. We report a father and daughter pairing with lesions occurring mainly on the trunk and extremities which were not sun exposed. The patients presented sequentially. The daughter presented earlier than her father. Till the time of reporting, no other family members had been found with the same lesions. Arsenic exposure has demonstrated an increased risk of cancer of the skin in humans.<sup>[2,3]</sup> Both our patients denied a history of cryptic arsenic ingestion. More than 70% of all HPV-related anogenital cancers contain DNA from either HPV-16 or HPV-18.<sup>[4]</sup> We employed immunohistochemical methods to detect the presence of HPV-16 and HPV-18 DNA in the lesions, which turned out negative. Electron microscopy examination was also negative for viral particles in the lesions at  $\times 40,000$  magnification. We, therefore, conclude that there is no evidence to support an oncogenic viral cause for the occurrence in our two patients. It has been reported that the standardized incidence ratios for squamous cell carcinoma and Bowen's disease increased to 1.95-4.30 times for individuals with affected siblings or parents.<sup>[5]</sup> The characteristics of our two patients include an early age of disease onset, and multiple and widely distributed lesions on the body. We therefore, postulate the possibility of

a familial genetic susceptibility playing a part in the pathogenesis of the lesions in our reported patients. However, due to the fact that molecular and genetic studies were not performed, we could not completely exclude the possibility that this reported concurrence may be a coincidental finding. Future studies should explore hereditary, molecular, and genetic factors in the familial clustering of this disease.

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