A Case of Pigmented Bowen’s Disease

Hyun Jeong Lee, M.D., Shin Taek Oh, M.D., Seog Jun Ha*, M.D., Jin Wou Kim, M.D.

Department of Dermatology, St. Paul’s Hospital, College of Medicine,
The Catholic University of Korea, Seoul and *Headquarter of Navy Education, Jinhae, Korea

A 38-year-old woman was presented with a dark brown plaque on the abdomen. Clinically, the tumor was simulating the appearance of dysplastic nevus. Microscopically, the cells of the stratum malphighiii lay in a disordered pattern. Many cells in the epidermis were atypical and melanin pigment was mainly in the basal layer of the epidermis and the upper dermis. Diagnosis of pigmented Bowen’s disease was made. Pigmented Bowen’s disease is rarely found at body sites other than the anogenital area. (Ann Dermatol 12(1) 68-70, 2000).

Key Words : Bowen’s disease, Increased pigment

Bowen’s disease is a squamous cell carcinoma in situ of the skin and mucous membrane. It commonly involves sun exposed areas and proximal extremities, but it can involve any other parts of the body. Pigmentation in Bowen’s disease is rare as compared to other epithelial neoplasms. The occurrence of pigmented Bowen’s disease has been reported mainly in the anogenital area. Lloyd reported the pigmented tumor in the groin as multicentric pigmented Bowen’s disease of the groin, but this is regarded as an earlier description of Bowenoid papulosis. Pigmented Bowen’s disease differs from Bowenoid papulosis clinically in the age of onset, location, number of lesions, size, appearance, possibility of spontaneous regression, and prognosis, and histologically in epithelial maturation, presence of dyskeratotic keratinocyte, and involving acroxyringia or acrotrichia.

In our review, no pigmented Bowen’s disease was found in the Korean literature. We report a rare case of pigmented Bowen’s disease.

CASE REPORT

A 38-year-old woman presented with a dark-brown plaque of about 1cm in diameter on her abdomen. She noticed the lesion 5 months before, incidentally. She reported that the lesion showed a slight increase in size without notable changes in pigmentation. Examination disclosed a relatively well demarcated, slightly elevated plaque with fine scales (Fig. 1). The medical evaluation of this patient disclosed no evidence of internal malignancy. No history of arsenic exposure or ingestion could be obtained. Initially the lesion was considered as dysplastic nevus.

Microscopic examination of a biopsy specimen taken from the plaque of the abdomen disclosed a thickened epidermis. The cells of the stratum malphighiii lay in a disordered pattern, and many cells were atypical (Fig. 2A,B). Individual cell keratinization was found. Increased melanin pigment was noted in the basal layer, and melanophages were scattered throughout the upper dermis (Fig. 3). A diagnosis of pigmented Bowen’s disease was made. The patient underwent treatment with surgical excision with primary closure. No recurrence was observed for six months.

DISCUSSION

Pigmented Bowen’s disease is characterized by microscopically increased melanin pigment in the
epidermis and/or papillary dermis in addition to typical findings of Bowen's disease. It may not be a pigmented lesion in clinical appearance.\(^1\)

Pigmented Bowen's disease is very rare. Ragi et al found 7 lesions (1.67%) by reviewing the records of 420 cases of Bowen's disease.\(^1\) In their report, involved sites were the trunk (3 cases), thigh (2 cases), head and neck (2 cases). In other literature, pigmented Bowen's disease was observed on various sites such as the perianal area, umbilicus, lip, thumb, feet, scrotum, neck, nail and arm.\(^5\) In contrast to Bowen's disease in which the role of chronic sun damage is important in its evolution, the site of pigmented Bowen's disease is mainly a non-sun exposed area.\(^1,10\) Pigmented Bowen's disease usually affects elderly persons.\(^5,9\) Analysis of previous reports revealed peak incidence in the seventh or eighth decade. Occurrence under the age 40 is rare in pigmented Bowen's disease as in Bowen's disease which showed less than 2% of incidence under the age 40.\(^4,2,10\)

In our case, the tumor was located in the non-sun exposed abdomen. Clinically the tumor was presented as a dark brown plaque simulating the ap-

---

**Fig. 1.** A dark-brown, well demarcated, flat plaque on the abdomen.

**Fig. 2.** (A). The epidermis is thickened. The cells of the stratum malphighii lay in a disordered pattern, and many cells were atypical (H&E stain, \(\times 40\)).

(B). Higher magnification of Fig. 2A. Atypical cells showing large or hyperchromatic nuclei in a disordered array of "windblown appearance" (H&E stain, \(\times 400\)).

**Fig. 3.** Increased melanin pigment was noted in the basal layer, and melanophages were scattered throughout the upper dermis (H&E stain, \(\times 100\)).
pearance of a seborrheic keratosis or a dysplastic nevus. But, the lesion could be differentiated from dysplastic nevus by the clinical findings of regular border and rather even pigmentation and histologic pictures. Pigmented seborrheic keratosis was also easily excluded by the histologic findings. It was found in the patient’s late thirties, and this is a relatively early occurrence compared with the age of peak incidence of pigmented Bowen’s disease.

Pigmentation was mainly in the basal layer of the epidermis and upper dermis in this case as in previous reports. Histologically, increased pigmentation is due to melanocyte proliferation with its transfer to keratinocytes, mainly of the basal layer and increased melanin pigment in the upper dermis. The cause of increased pigmentation is considered to be related to the hyperplasia of keratinocyte which may affect the growth of melanocytes or the melanin transfer to the keratinocytes. The amount of melanin is variable within the tumor, and there has been no evidence of association between the degree of pigmentation and poor prognosis or malignant behavior.

Possible etiologic factors of Bowen’s disease includes chronic sun exposure, arsenic, trauma, human papilloma virus and ionizing radiation. In Oriental populations, hyperpigmentation and pigmented Bowen’s disease has been reported in association with chronic arsenic ingestion, but, no difference was documented in the pathogenesis of pigmented and classical Bowen’s disease.

This is a rare case of pigmented Bowen’s disease which developed on the abdomen in a young woman with an unusual clinical appearance similar to a seborrheic keratosis or a dysplastic nevus.

REFERENCES