Asymptomatic erythematous lesions on dorsal aspect of both hands

AK Douieb,[1] MD, Bayoumi Eassa,[2] MD

[1]*Consultant, Department of Dermatology, HPLM, Larache, Morocco*
[2]*Department of Dermatology, Venereology & Andrology, Faculty of medicine, Al-Azhar University, Cairo, Egypt*

CLINICAL FINDINGS

A 48-year-old male presented with occasionally itchy, progressively enlarging, irregularly shaped, eczematous plaque of 3 years' duration measuring 4×4 cm on the dorsum of the left hand [Fig. 1, 2]. Another new similar looking but smaller lesion developed 1 year ago on the right hand. It had not responded to topical steroids, salicylic acid, and antifungal treatment. Cryotherapy was tried but with minimal and transient improvement. According to the patient it started as a small papule following blood collection at the site and gradually increased in size and started to form hypertrophic crust. He was a known diabetic, controlled on insulin for the past 11 years. The patient did not complain of any systemic illness and there was no family history of similar lesions.

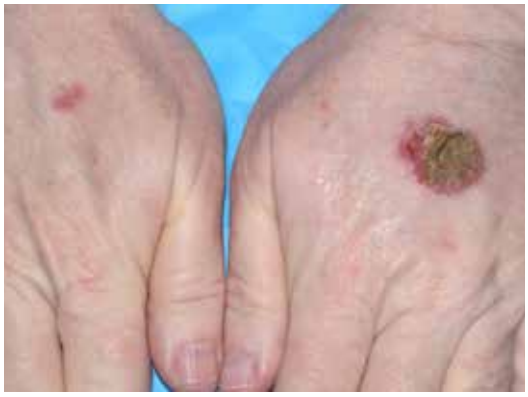

Fig. 1 Erythematous scaly patch on dorsum of right hand and scaly, crusted, erythematous plaque in left hand.

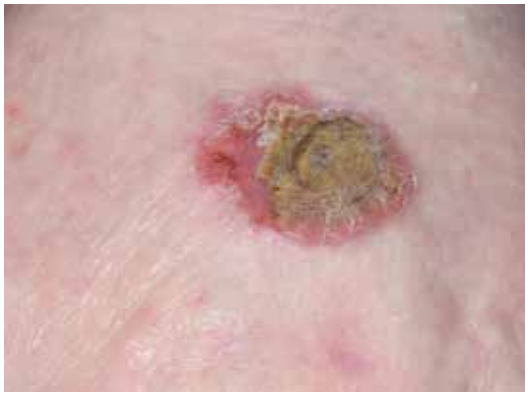

Fig. 2 Crusted scaly erythematous plaque on dorsum of left hand.

On clinical examination, there was a well-defined erythematous plaque and overlying scale and crusts on dorsal aspect of both hands. There was no tenderness or pain on palpation. Rest of the dermatological and systemic examination of the patient was normal. Nails and mucous membranes were not affected and showed no significant abnormalities. Routine laboratory investigations including CBC, blood sugar, hepatic and renal profile revealed no abnormal findings. X-ray of both hands showed no attachment of the lesion to underlying bones.

What is your clinical differential diagnosis?

1. Bowen's disease
2. Squamous cell carcinoma
3. Superficial spreading basal cell carcinoma
4. Actinic Keratosis
5. Deep mycosis

Correspondence: *Dr. AK Douieb, Consultant, Department of Dermatology, HPLM, Larache, Morocco, Email: douieb@menara.ma*

Punch (5mm) skin biopsy was done and histological analysis revealed parakeratosis and acanthosis with full thickness dysplasia. Loss of polarity, atypical cells with hyperchromatic nuclei, atypical mitoses and dyskeratotic cells were also seen. The dermis shows superficial perivascular inflammatory infiltrate formed of lymphohistiocytic cells admixed with melanophages [Fig. 3, 4].

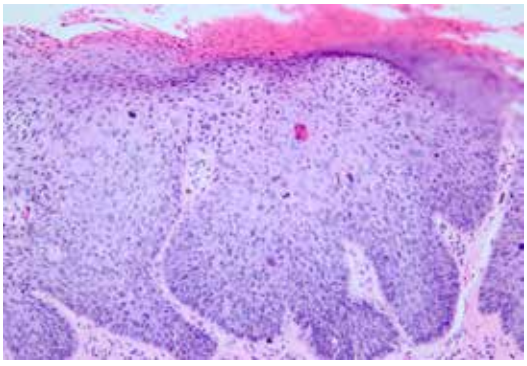

Fig. 3 Parakeratosis and acanthosis with full thickness dysplasia.

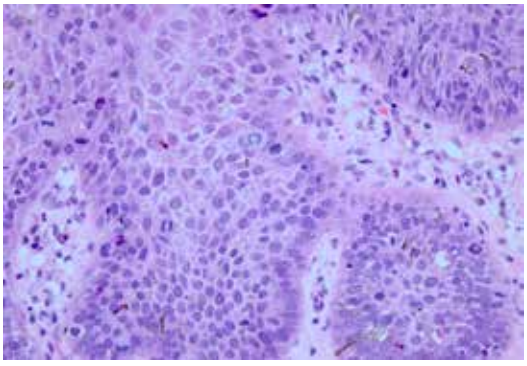

Fig. 4 loss of polarity, atypical cells with hyperchromatic nuclei, atypical mitoses and dyskeratotic cells.

DIAGNOSIS

Bowen's disease

DISCUSSION

BD was first described by an American Dermatologist John T. Bowen in 1912. It is a squamous cell carcinoma in situ with potential for significant lateral spread.

It can affect the skin and mucous membranes. The involvement of sun-exposed sites is more common in the whites, whereas that of the unexposed sites is more common in the pigmented skin.[1] It is more common on the head, neck, and extremities in men and lower limbs and cheeks in females. It ranges from very few millimeters to several centimeters in diameter. Lesions are usually solitary, but multiple lesions are seen in 10-20% of patients.

Significant sun exposure, ionizing radiation, arsenic exposure,[2] immunosuppression,[3] and certain types of human papillomavirus[4] are predisposing factors for BD. Genetic factors, trauma, chemical carcinogens, and X-ray radiation are other factors implicated in the pathogenesis.

Patients usually present with an asymptomatic slowly enlarging erythematous scaly patch or plaque. Histopathology shows full-thickness anaplasia of the epidermis with loss of normal maturation, although the basement membrane remains intact. Parakeratosis and hyperkeratosis, acanthosis with complete disorganization of the epidermal structure is present. Throughout the epidermis are numerous, atypical, pleomorphic hyperchromatic keratinocytes producing the windblown appearance. These cells are sometimes vacuolated and have a pale-staining cytoplasm. Loss of maturation and polarity of the cells, numerous mitotic figures, individually keratinized cells, multinucleated cells and atypical cells are seen throughout the pilosebaceous unit, within the acrotrichium, follicular infundibula, and sebaceous glands.

The chances of development of squamous cell carcinoma (SCC) in a case of BD is 3%-5% and there are 33% chances of metastasis from a case of SCC that has evolved from BD.[3] Therapy is guided by size and location of BD in addition to individual

patient characteristics, such as age and healing capacity. Surgical excision is generally regarded as the treatment of choice for most BD lesions, if the lesions size and location permit such a procedure. Mohs micrographic surgery, electrodessication and curettage, cryosurgery, topical chemotherapy with 5-fluorouracil, topical immune response modifiers such as imiquimod, laser therapy, radiotherapy, and photodynamic therapy are the known modalities of treatment.[5]

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