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Case Report

Atypical presentation of bowens disease

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Abstract

In-situ squamous cell carcinoma of the epidermis is known as Bowen's disease (BD). BD has a complex aetiology and a significant prevalence in the Caucasian population. Although it can affect other regions of the skin, BD is most common in photo exposed areas. Typically, lesions are isolated. The lesion's age, origin, and level of keratinisation all affect the appearance of BD. Before an overt squamous cell carcinoma, BD is referred to as the "lull before the storm." The most reliable diagnostic technique for confirming the diagnosis is histopathology. Adjuvant techniques for diagnosing BD include immunohistochemistry, dermoscopy, and reflectance confocal microscopy. In this unusual instance of Bowen's disease, we show a single plaque on the thigh, which is not normally exposed to sunlight.

Keywords: Bowens Disease, Squamous cell carcinoma

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1. Introduction

Bowen's disease (BD) is an in-situ squamous cell carcinoma (SCC) of epidermis.[1] The major etiological factors of BD include ultraviolet light exposure, immunosuppression, and Human Papilloma Virus (HPV) infections. BD is common in photo-exposed areas of skin, but other sites can also be involved. The natural course of BD is usually prolonged, needing appropriate treatment. Nevertheless, other diagnostic techniques such reflectance confocal microscopy, dermoscopy, and immunohistochemistry can help with the assessment and diagnosis of BD.

2. Case Report

A 78-year-old male, working as a professional driver and known case of type 2 diabetes mellitus for 10 years, presented to the dermatology outpatient clinic with a painful, burning lesion over the right thigh. He reported that the lesion began as a small red patch approximately one month prior to presentation and had progressively increased in size. It had become increasingly painful with burning sensation, and there was no associated pruritus. The patient denied any history of trauma, topical application, systemic drug intake, photosensitivity, or prior dermatological illness.

Given the erythematous, inflamed appearance of the lesion, an initial clinical impression of photodermatitis was considered, although the location on the anterior thigh—a typically non-sun-exposed area—raised suspicion. The patient reported wearing trousers regularly due to his occupation, which involves long hours of driving. He did not recall any recent change in clothing, exposure to new fabrics, chemicals, or application of herbal or topical remedies. On examination, the patient was moderately built, conscious, cooperative, and oriented. Vitals were stable, and systemic examination revealed no abnormalities. A solitary, well-demarcated, erythematous plaque measuring approximately 4 × 5 cm was present over the anterior aspect of the right thigh. The lesion showed prominent scaling, crusting,

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superficial erosions, and focal areas of haemorrhage. The borders were irregular but clearly defined. The surrounding skin was normal with no signs of photosensitivity (e.g., hyperpigmentation, telangiectasia, or other sun-induced changes). There was no regional lymphadenopathy. Based on the acute presentation, burning sensation, and erythematous nature of the plaque, photodermatitis or irritant contact dermatitis were initially considered in the differential diagnosis. However, the absence of known triggers, non-exposed location, and lack of response to emollients and mild topical steroids, which the patient had used sporadically, prompted further evaluation. A skin biopsy was performed to rule out other inflammatory and neoplastic conditions. Dermoscopy of the lesion showed crusting with vascular structures (**Figure 2**)

The lesion on the anterior aspect of the right thigh was sampled with a 4mm punch biopsy.

Histopathological examination suggested Acanthosis and full thickness hyperplasia, Dysplastic squamous cells show mild to moderate nuclear pleomorphism, Superficial and deeper dermis shows dense stromal perivascular lymphoid cells infiltrate (**Figure 1**), Basement membrane intact and result was concluded as full thickness squamous dysplasia consistent with Bowen's disease.

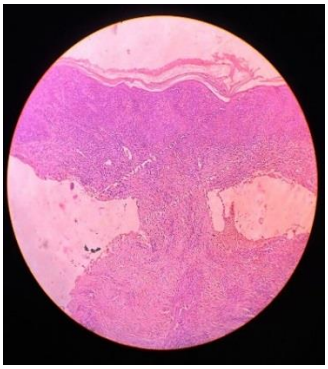

Figure 1: Acanthosis and full thickness hyperplasia, Dysplastic squamous cells show mild to moderate nuclear pleomorphism, Superficial and deeper dermis shows dense stromal perivascular lymphoid cells infiltrate

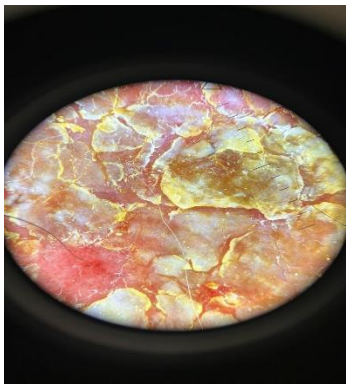

Figure 2: Dermoscopy prior to treatment showing crusting with central vascular structures

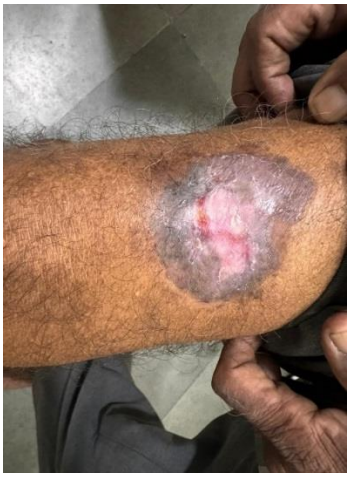

Figure 3: Shows lesion after 2 weeks of initiation of treatment with 5% 5 flurouracil

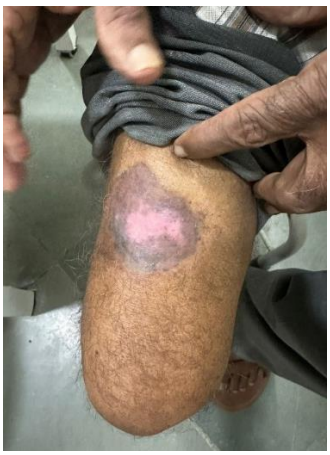

Figure 4: Follow up after 4 weeks

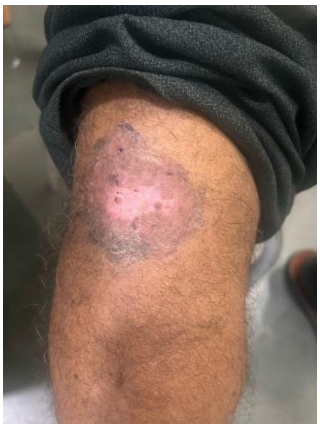

Figure 5: Follow up after 3 months

Following Bowen's disease diagnosis, the patient was started on topical 5% of 5-Flurouracil at night. During a follow-up period of two weeks, the patient developed ulceration at the application site (**Figure 3**) consequently, the frequency of 5-FU was reduced to five days per week, with the addition of topical retinoids on weekends. After this modification, the patient's condition improved clinically, and the ulcers healed (**Figure 4,5**)

3. Discussion

Bowen's disease, an intraepithelial squamous cell carcinoma, was first described by John Bowen in 1912.[2] In 10–20% of cases, numerous lesions may develop, but it usually manifests as a single, asymptomatic plaque. 73.5% of the 617 Bowen's disease cases reported by Thestrup-Pedersen et al. happened over photoexposed regions, such as the hands, neck, and head.[3] According to Kossard et al.'s review of data from 1,001 Bowen's disease patients with histological confirmation, the head and neck area was the most common location (44%) followed by the lower limb (29.8%), upper limb (19.8%), and torso (6.5%). In Cox's series of 128 cases, the most common locations were the lower limbs (75%), followed by the face and scalp. A case report on Bowen's disease arising over a scar of BCG vaccination was also reported.[4]

UV radiation exposure, immunosuppression (corticosteroids, azathioprine), HPV infections (particularly types 16, 18, 31, 33, and 35), and prolonged exposure to arsenic, which damages DNA and dysregulates the immune system, are the primary risk factors.

BD usually manifests as slowly growing, erythematous, scaly patches or plaques. In 10–20% of cases, the lesions may be numerous, but they are typically solitary. Among the variations of BD are Pigmented BD, Perianal, Periungual, Palmar, Nipple, and Erythroplasia of Queyrat (affecting the penis). BD can manifest as widespread lesions on the penis, scrotum, and inguinal region in people with HIV. Lesions of BD vary in size, and the disorder can take two to forty years to fully develop. It also spreads laterally in a sluggish and unpredictable manner. Seborrhoeic keratosis, discoid dermatitis, basal cell carcinoma, SCC, and psoriasis are among the differential diagnosis for BD. Scaly surface and glomerular vessels are the most common dermoscopic characteristics of Bowen's disease.[5] On histopathology, the epidermis shows hyperkeratosis and parakeratosis, marked acanthosis with elongation and thickening of rete ridges. The keratinocytes show atypia, which spans the entire epidermis, not breaching the dermo-epidermal junction. The keratinocytes in BD demonstrate intense mitotic activity, pleomorphism, and very large nuclei. The accompanying loss of maturity and polarity gives the epidermis a "windblown" appearance.[6]

Because there are numerous protocols and the success of a treatment modality depends on multiple factors (e.g., body site, lesion size and thickness, different equipment), it is challenging to compare and evaluate the efficacy of various treatment options and Bowen's disease treatment studies.

The effectiveness of the treatment, the size and location of the BD, the number of lesions, the therapy's availability, the clinician's experience, the patient's characteristics (age, immune state, concurrent medication, comorbidities, and compliance), the cosmetic result, and the patient's preferences should all be taken into consideration. Cryotherapy, curettage with cautery, excision, 5-fluorouracil (5-FU), radiation, laser, photodynamic therapy (PDT), imiquimod, and a few more treatments that have been reported in a few case studies or small patient populations are among the several treatment possibilities for BD. None of the available treatments have been conclusively shown to be better than the others thus far.[7]

More recent options for treatment includes Diclofenac which reduces downstream byproducts of arachidonic acid (AA) metabolism by inhibiting cyclooxygenase enzymes. These AA metabolites have been demonstrated to be crucial in promoting the formation of epithelial tumours through a number of processes and pathways (such as immune surveillance and apoptosis suppression, angiogenesis stimulation.[8]

The majority of pharmaceutical markets have approved imiquimod 5% cream, a topical immune response modifier, for the treatment of superficial basal cell carcinoma, actinic keratoses, and anogenital warts. Heterocyclic imidazoquinoline imiquimod has antiviral and anticancer properties. Imiquimod's effectiveness is attributed to the promotion of innate and acquired immunity; it lacks antiviral or antiproliferative properties. The production of cytokines such as interferon (IFN) alfa, tumour necrosis factor alfa, interleukin (IL)-1α, IL-1 receptor antagonist, IL-12, and IFN-γ is induced when imiquimod binds to toll-like receptors (TLR)-7 and -8. This results in a type 1 coordinated cell-mediated immunity.[9]

A well-established treatment option for actinic keratoses, superficial and nodular basal cell carcinoma, and BD is photodynamic therapy (PDT).

4. Conclusion

We are bringing up this case to draw attention to two important points:

1. The uncommon incidence of the illness at a non-photo exposed place, which contributes to the scant research on its occurrence there.
2. The therapeutic response in which topical retinoid was added to a combined regimen that significantly sped up wound healing after initial monotherapy with 5% 5 fluorouracil caused ulceration. This innovative therapeutic technique might provide insightful information for improving management tactics in circumstances like this.

5. Conflict of Interest

None.

6. Source of Funding

None.

References

1. Cox NH, Eedy DJ, Morton CA. Guidelines for management of Bowen's disease: 2006 update. *Br J Dermatol*. 2007;156(1):11–21.
2. Ragi G, Turner MS, Klein LE, Stoll Jr HL. Pigmented Bowen's disease and review of 420 Bowen's disease lesions. *Dermatol Surg*. 1988;14(7):765–9.
3. Pravin A, Kulkarni OD, Thomas J. Bowen's disease arising over a scar of BCG vaccination: a rare case report. *Int J Sci Res*. 2019;8(9):55–6.
4. Maria Wozniak-Rito A, Rudnicka L. Bowen's disease in dermoscopy. *Acta Dermatovenerologica Croatica*. 2018;26(2):157–61.
5. Palaniappan V, Karthikeyan K. Bowen's disease. *Indian Dermatol Online J*. 2022;13(2):177–9.
6. Neubert T, Lehmann P. Bowen's disease–a review of newer treatment options. *Ther Clin Risk Manag*. 2008;4(5):1085–95.
7. Gately S. The contributions of cyclooxygenase-2 to tumor angiogenesis. *Cancer Metastasis Rev*. 2000;19(1-2):19–27.
8. Hengge UR, Benninghoff B, Ruzicka T, Goos M. Topical immunomodulators—progress towards treating inflammation, infection, and cancer. *Lancet Infect Dis*. 2001;1(3):189–98.
9. Braathen LR, Szeimies RM, Basset-Seguin N, Bissonnette R, Foley P, Pariser D, et al. Guidelines on the use of photodynamic therapy for nonmelanoma skin cancer: an international consensus. *J Am Acad Dermatol*. 2007;56(1):125–43.

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