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Case Report

Unveiling Bowen's disease on lower limb: a case report of long-term misdiagnosis as dermatitis

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ABSTRACT

Bowen's disease (BD) is a pre-cancerous condition classified as in-situ cutaneous squamous cell carcinoma (CSCC) of the epidermis. Several studies have shown that the condition requires prompt diagnosis and treatment due to its progressive nature. Despite the urgency of treatment, misdiagnoses are common due to similarities to dermatitis or psoriasis. Therefore, this report presents the case of a 69-year-old man with a hyperpigmented erythematous plaque on the right lower leg, which was misdiagnosed as nummular dermatitis for over 10 years. The patient reported the long-term use of corticosteroids, but the lesion persisted. Physical examination showed a 35×40 mm irregular plaque with excoriation. A skin biopsy showed full-thickness epidermal atypia, confirming BD. The lesion was surgically removed with a 4 mm margin, and an O-Z flap was used for closure. After the treatment, no recurrence was observed at 6 months of follow-up. BD often mimics other dermatological conditions, leading to delayed diagnosis. This case underscores the importance of biopsy in chronic skin lesions, especially when treatments fail. Surgical excision remains the most reliable treatment, offering a high success rate and low recurrence. Non-surgical options, such as photodynamic therapy and topical agents, have lower efficacy. In this case, the surgical approach with a 4 mm safety margin ensured complete removal while minimizing recurrence risk. This report showed the significance of early and accurate diagnosis of BD to prevent progression to invasive CSCC. Surgical excision was the preferred treatment for BD, offering high cure rates and minimizing complications.

Keywords: Bowen's disease, In-situ cutaneous squamous cell carcinoma, Diagnosis, Biopsy surgical excision

INTRODUCTION

Bowen's disease (BD), also referred to as in-situ cutaneous squamous cell carcinoma (CSCC) of the epidermis, is a rare skin condition. This condition appears as solitary, slowly enlarging, erythematous, squamous papules or plaques, typically affecting adults over 60 years old, with a higher prevalence in women.[1] In addition, it often occurs in sun-exposed areas, such as the cheeks and lower legs. Although the condition is rare, it carries a 3 to 5% risk of progressing to invasive CSCC, emphasizing the importance of early recognition and treatment. Several studies have shown that BD can be misdiagnosed, particularly in older individuals or those with sun damage, as it is often similar to chronic dermatitis.[2] Therefore, this report aims to explore the clinical, histopathological, and therapeutic aspects of BD, focusing on a case initially

misdiagnosed as dermatitis, resulting in prolonged corticosteroid use.

CASE REPORT

A 69-year-old male patient with Fitzpatrick skin type III presented a hyperpigmented erythematous plaque with excoriation on the right lower leg that had slowly enlarged over more than 10 years. The lesion had previously been misdiagnosed as nummular dermatitis and was treated with topical steroids for several years, but the symptoms failed to resolve. This lesion was asymptomatic, rarely pruritic, and caused cosmetic disturbance. The patient had no family history of disease and denied any previous cutaneous carcinomas. In addition, a history of sunburn, arsenic exposure, previous medication with ultraviolet radiation, organ transplant recipients, HIV, and previous medication of systemic immunosuppressive drugs was denied. The history of HPV infection remained unknown.

Physical examination showed a solitary 35×40 mm lesion, irregular in shape with well-demarcated border hyperpigmented erythematous plaque showing excoriation on the surface and telangiectasia (Figure 1).

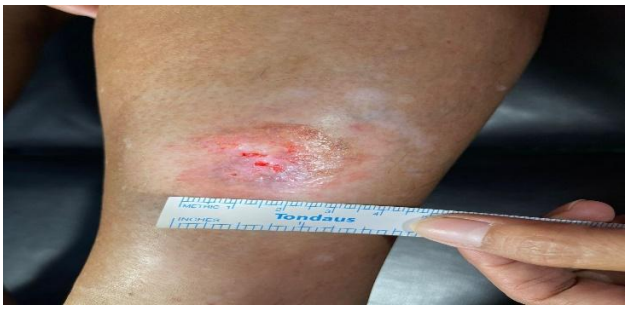

Figure 1: A solitary 35×40 mm lesion, irregular in shape with well-demarcated border hyperpigmented erythematous plaque with excoriation on the surface and telangiectasia.

Histopathological examination of the skin biopsy showed full-thickness epidermal atypia with loss of the stratified architecture and disturbed cell polarity. Inflammatory cell infiltration was carefully observed in the dermis, and diagnosis of BD was confirmed based on these results (Figure 2).

The patient was referred to the Department of Surgical Oncology, where the lesion was surgically removed using a wide elliptical excision with a 4 mm safety margin (Figure 3). Pathology confirmed the diagnosis of BD, and complete resection was achieved. Due to the large excision, O-Z flaps were used for reconstruction. Consequently, there were no signs of recurrence after 6 months of observation. Post-surgical wound healing was uneventful, as shown in Figure 3. Another reported cases of BD on the lower limb are shown in Table 1.

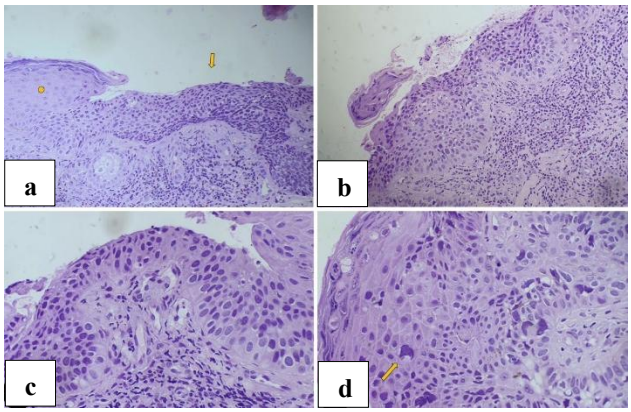

Figure 2: Histopathological examination resulted in HE of the lesion with BD, (a) HE 100x, (b) HE 200x, (c) HE 100x), and (d) HE 400x. Disturbed cell polarity to the surface, full-thickness epidermal atypia with loss of the stratified architecture, inflammatory cell infiltration was presented on the dermis layer.

HE=hematoxylin-eosin

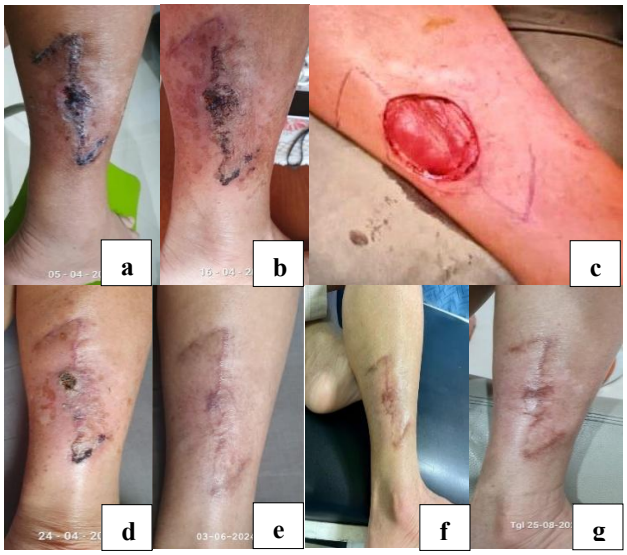

Figure 3: (a-g) Intraoperative procedure and patient's post-surgical wound follow-up after tumor removal.

Table 1: Reported case of Bowen disease on the lower limb.

Author, year, country	Sex	Age (years old)	Size (cm)	Treatment	Recurrence	Follow-up
Chae et al, 2017, South Korea[11]	F	69	3×4	0.05% ingenol mebutate gel	No	2 years
Gold-Olufadi et al, 2015, Nigeria[12]	F	61	5×7	Topical 5% 5-fluorouracil cream	N/A	N/A

Continued.

Author, year, country	Sex	Age (years old)	Size (cm)	Treatment	Recurrence	Follow-up
Dudani et al, 2020, India[13]	M	58	0.5×1	Topical 5% 5-fluorouracil cream	N/A	N/A
Sirka et al, 2021, India[3]	M	60	4×6	Topical 5% imiquimod cream for 12 weeks	N/A	8 months

M=Male; F=female; N/A=not available

DISCUSSION

BD refers to a rare pre-cancerous cutaneous disease characterized by the in-situ proliferation of atypical keratinocytes within the epidermis, serving as a precursor lesion to CSCC.[1] The prolonged delay elevates the risk of disease progression, potentially increasing the likelihood of developing invasive squamous cell carcinoma, which is associated with significantly higher morbidity and mortality rates.[3,4]

The 60-year-old patient presented with a 10-year history of clinical signs of BD lesions. Before referral to the center, the respondent had been misdiagnosed with dermatitis and was treated with topical corticosteroids for an extended period. This respondent's clinical presentation also included persistent pruritus, which occurred in a body area subjected to sunlight. The clinical manifestations of BD can closely mimic various dermatological conditions, complicating its identification and increasing the risk of diagnostic error. Misdiagnosis can occur in diagnosing BD such as dermatitis, psoriasis, actinic keratosis, and superficial basal cell carcinoma due to their overlapping clinical features and presentations.[1] Takada et al reported that chronic repetitive friction served as a potential etiological factor for BD. Persistent stimulation from friction could lead to chronic wound formation and scarring, which represented a progression to CSCC.[5]

Scar tissue formation could disrupt the immune response, potentially developing of tumors. This mechanism was linked to the onset of BD formation. Consequently, there have been reported cases of BD arising from long-standing scars, suggesting a possible association between chronic skin injury and the onset of this condition.[5] American Osteopathic College of Dermatology stated that BD could be mistaken as dermatitis due to overlapping clinical features, including pruritus, erythema, crusting, and scaling. Therefore, a biopsy had to be performed to confirm BD diagnosis.[6] A punch biopsy was also performed to rule out differential diagnoses, showing atypical keratinocytes, which proliferated throughout the full thickness of the epidermis, thereby confirming diagnosis of BD.[7]

Treatments for BD included surgical excision, destructive therapies, topical treatments, and photodynamic therapy (PDT). Surgical excision was considered the most effective, specifically for solitary lesions. Previous studies had shown that excision had a higher clearance rate than non-invasive treatments, with a 97.4% clearance rate compared to 85.7% for 5-fluorouracil and 82.1% for methyl aminolevulinate (MAL)-PDT. Both non-invasive treatments were less effective compared to surgical options. Patients in the MAL-PDT group experienced frequent pain and burning sensations, meanwhile in the 5-fluorouracil group, severe skin reactions led to treatment discontinuation within 3 weeks. Although PDT is a promising non-invasive treatment but it was not considered in this case due to the lesion's characteristics, the higher recurrence rate than the surgical approach, and the patient's preference for definitive surgical management.[8]

Surgical excision was often considered the first-line treatment modality for BD, particularly for solitary and small lesions, due to its efficacy and cost-effectiveness. Complete excision of the BD lesion could significantly reduce the risk of progression to invasive CSCC.[9] In our case, surgical excision with a 4 mm margin was performed to minimize recurrence risk. While European guidelines did not specify margins, the American NCCN recommended 4 to 6 mm.[10] The patient had a 4 mm margin due to the lesion's low risk of progression, ensuring clear margins while minimizing complications such as bleeding and scarring. The O to Z wound closure technique was used, which promoted rapid healing, aesthetically pleasing results, and low postoperative care costs. The patient showed complete healing within 30 days, consistent with typical outcomes for this technique.

CONCLUSION

This case report presents a patient with BD who was initially misdiagnosed as nummular dermatitis. The patient experienced a prolonged delay in diagnosis, leading to unnecessary treatment and potential complications. A delay in diagnosis can have significant consequences, including increased risk of progression to invasive CSCC. Further research into the long-term effects of delayed or incorrect treatment for BD is warranted.

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