



Case Report

Bowen’s Disease: A Series of Cases

M R Swaroop¹, Manas S N², Basavaraj H B³, Sathyanarayana B D⁴

¹Associate Professor, ²Junior Resident, ³Professor, ⁴Professor and HOD,
Department of Dermatology, Adichunchanagiri Institute of Medical Sciences, BG Nagara, Bellur, Mandya District,
Karnataka, India.

Corresponding Author: M R Swaroop

Received: 02/12/2014

Revised: 05/01/2015

Accepted: 12/01/2015

ABSTRACT

Bowen’s disease is squamous cell carcinoma in- situ of the epidermis. It commonly presents as solitary psoriasiform plaque involving head, neck or lower limbs. The following case series of Bowen’s disease were studied in Dermatology outpatient department of a Rural Tertiary Care Hospital over a span of 1 year wherein total 5 patients presented with Bowen’s disease. All the patients were male farmers between the age group of 45-80 presented lesions with varied clinical morphology in sun protected areas of the body. The incisional biopsy confirmed the diagnosis of Bowen’s disease in all the cases. Surgical excision was performed and excision biopsy of cases 2, 3, 4 and 5 reconfirmed the diagnosis of Bowen’s disease however, case 1 there was focal invasion to the dermis suggesting squamous cell carcinoma. We hereby are reporting this case series for its varied clinical morphology in different anatomical sites, higher incidence in rural population than usual, and with involvement of sun-protected areas without any evidence of arsenic exposure, multiplicity and rapid progression to invasive Squamous cell carcinoma in case 1.

Key words: Bowen’s disease, squamous cell carcinoma in-situ, multiple, sun-protected areas

INTRODUCTION

Bowen’s disease (BD) is a rare, progressive, intraepithelial carcinoma which was first described by John Bowen in 1912. [1] Classically, it manifests as solitary plaque mimicking psoriasis in sun exposed areas (head and neck), however multiple lesions

have been reported in 10 to 20% of patients especially with arsenic exposure. The risk of transformation to squamous cell carcinoma has been reported to be 8%. Hereby, we are reporting a case series of Bowen’s disease studied over a period of one year in a rural tertiary care center.

CASE REPORTS

CASES OVERVIEW					
	Case 1	Case 2	Case 3	Case 4	Case 5
Age	45	75	70	80	80
Sex	male	male	male	Male	Male
Occupation	farmer	farmer	farmer	Farmer	Farmer
Duration	3 years	3 years	2 years	6 months	One year
Number	four	one	one	One	One
Site	Right buttock	Sternal area	Right infrascapular area	Left chest	Right lower back

On cutaneous examination:

CASE 1



Figure 1: Four well defined plaques with irregular erythematous margins with thick adherent scale and crusting over the right gluteal region.

CASE 2



Figure 2 a&b: Examination revealed well defined erythematous scaly plaque of 10x10 cms over the sternum extending towards the right chest

CASE 3



Figure 3 a&b: A hyperpigmented plaque with scaling and crusting present over the right infra-scapular region

CASE 4



Figure 4a&b: Well defined erythematous scaly plaque with crusting with peri-lesional halo present over the left chest

CASE 5



Figure 5a&b: Hyperpigmented oval plaque with crusting present over the left lower back

Routine investigations in all the patients were within normal limits and patients were sero-negative. Punch biopsy from the lesions [figure 6&9] in common revealed an irregularly acanthotic epidermis with mild nuclear pleomorphism, loss of normal architecture, scattered dyskeratotic keratinocytes, multinucleated giant cells with a varying degree of mitosis with intact basement membrane. Diagnosis of Bowen's disease was confirmed. Patient was referred to the surgeon, wherein, complete excision with wide margin and skin grafting for the largest of plaques was performed in case 1 and complete excision in rest of the cases. Excision biopsy in case 2,3,4&5 reconfirmed the diagnosis of bowen's

disease, however in case 1 [figure 7&8] there was marked acanthosis of epidermis, atypical cells with hyperchromatic nuclei, loss of polarity and focal area of invasion of these pleomorphic cells to the dermis suggesting progression to squamous cell carcinoma.

Histopathology

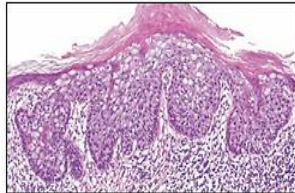


Figure 6: Incisional biopsy reveals irregular acanthosis and loss of polarity of cells with moderately dense lymphohistiocytic infiltrate in dermis

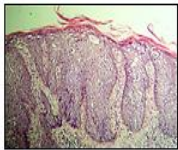


Figure 7: Excision biopsy: Irregular acanthosis, atypia, dysplasia are seen along with moderately dense lymphohistiocytic infiltrate & melanophages in dermis

Case 1

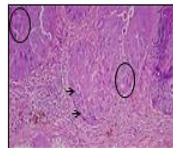


Figure 8: Excision biopsy: Islands of Epidermal cells in dermis showing multinucleate giant cells and atypical mitotic figures

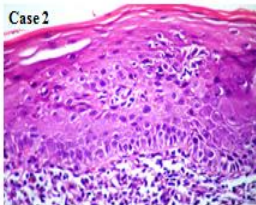
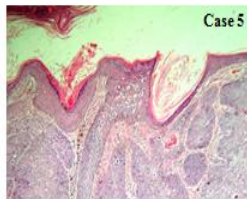
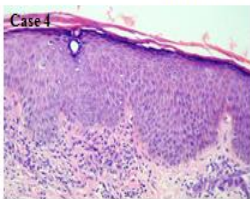
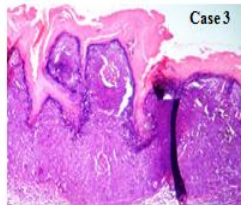


Figure 9: Biopsy revealed acanthosis with loss of polarity and few dyskeratotic cells. On high power, mitotic figures were seen.



DISCUSSION

Bowen's disease is a rare, premalignant progressive intra-epidermal carcinoma affecting skin and / or mucous membrane with potential to progress to squamous cell carcinoma. [1] The incidence is around 1.42 per 1000 population. [2] It can occur at any age but mostly seen in elderly having equal sex preponderance. Although

the exact etiology is unknown, chronic sun exposure plays a major role when lesions are present over the head, neck, dorsae of hands and lower limbs. When lesions are seen over palms, soles, mucosa and unexposed areas, arsenic exposure and *Human papilloma virus* (2, 16, 18, 34, and 35) are implicated. [1,3] Clinically, it presents as a solitary, erythematous, well demarcated, crusted or scaly plaque. [1] Multiple lesions are seen in 10-20% of cases with history of arsenic exposure. [3] The other uncommon clinical variants are pigmented, intertriginous, verrucous, hyperkeratotic, atrophic, palmar, plantar, genital, periungual and subungual type. [1,4] Histological examination of BD reveals hyperkeratotic epidermis with variable degree of parakeratosis. There is elongation and thickening of rete ridges. The cells throughout the epidermis lie in complete disorder resulting in a windblown appearance showing atypia with large hyperchromatic nuclei. Numerous vacuolated atypical cells are especially seen in arsenical Bowen's disease. The basement membrane is intact. The upper dermis shows a moderate amount of chronic inflammatory infiltrate. [5] Other histological variants namely, psoriasiform, atrophic, acantholytic, epidermolytic and pagetoid pattern have also been reported. In our case, the histological findings were similar; however, excision biopsy revealed marked acanthosis of epidermis, atypical cells with hyperchromatic nuclei, loss of polarity and focal area of invasion of these pleomorphic cells to the dermis thereby indicating progression towards squamous cell carcinoma. On dermoscopy, Bowen's disease reveals glomerular vessels (coiled vessels), scaly surface and linear arrangement of brown or grey dots (pigmented variant). [3,6] Based on above discussed, clinical and histopathological findings, we arrived at the diagnosis and thereby excluded differential diagnosis like herpetic granuloma, botryomycosis,

psoriasis, seborrhoeic keratosis, dermatophytic granuloma and lupus vulgaris. The progression of Bowen's to squamous cell carcinoma is 3-10% and average duration is 3-6 years, however in our case 1, it had progressed within 1 year of duration. [7] Various treatment modalities are surgical excision, Moh's micrographic surgery, cryotherapy, curettage, cautery, chemotherapy with topical 5-fluorouracil, 5% imiquimod cream, lasers (CO₂ laser, Er: YAG ablative fractional laser), radiotherapy and more recently photodynamic therapy with a topical photosensitizer δ-aminolevulinic acid. [1,8] In all patients, surgical excision with wide margin was performed. Excision biopsy of cases 2,3 4 and 5 reconfirmed the diagnosis of Bowen's disease however, case 1 there was focal invasion to the dermis suggesting squamous cell carcinoma. It has a recurrence rate of 10-15%. [1]

CONCLUSION

We hereby are reporting this case series for its varied clinical morphology in different anatomical sites, higher incidence in rural population than usual and uniqueness of individual cases namely

- a. Involvement of sun-protected areas in all cases
- b. No evidence of immunosuppression or arsenic exposure in all cases
- c. Multiplicity of lesions and rapid progression to invasive Squamous cell carcinoma in case 1.

REFERENCES

1. Singh S, Khaitan BK, Sharma MC, Seenu V, Kumavat M, Chatterjee P. Bowen's disease on finger: A diagnostic and therapeutic challenge. *Indian J DermatolVenerolLeprol* 2013;79:227-30.
2. Neubert T, Lehmann P. Bowen's disease – a review of newer treatment options. *TherClin Risk Manag* 2008;4:1085–95.
3. Gahalaut P, Rastogi MK, Mishra N, Chauhan S. Multiple Pigmented Bowen's Disease: A Diagnostic and Therapeutic Dilemma. *Case Rep Oncol Med* 2012; 2012: 342030.
4. Shyam B, Verma D. Hyperkeratotic Bowen's disease- A case report. *Dermat online Journal* 2008; 14(6):24
5. Rai VM, Balachandran C, Bhat S, Geetha V. Scaly erythematous plaque on the chest. *Indian J Dermatol Venereol Leprol* 2007;73:447-8.
6. Cameron A, Rosendahl C, Schandl PT, Riedl E, Kittler H. Dermatoscopy of pigmented Bowen's disease. *J Am AcadDermatol* 2010;62:597-604.
7. Hassan I, Sajad P, Reshi R. Histopathological Analysis of the Cutaneous Changes Due to kangri use in Kashmiri Population: A Hospital Based Study. *Indian J Dermatol* 2013;58 (3):188-90
8. Patel KB. Bowen's Disease Treated with Imiquimod and Cryotherapy. *Indian Journal of Dermatology* 2012; 57(3):239-41

How to cite this article: Swaroop MR, Manas SN, Basavaraj HB et. al. Bowen's disease: a series of cases. *Int J Health Sci Res.* 2015; 5(2):488-491.
