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Case Report

Coexistence of Bowen's disease and Pigmented Basal cell carcinoma in a case of chronic arsenicosis

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Abstract Arsenical Keratosis is a premalignant condition of skin, the exposure of which occurs through predominantly drinking water in endemic areas like West Bengal and Bangladesh, predisposing affected individuals to develop Bowen's disease, squamous cell carcinoma and basal cell carcinoma. Here, we describe an interesting case of multiple lesions of Bowen's disease and pigmented basal cell carcinoma coexisting in an adult male who had history of chronic exposure to arsenic through drinking water.

Key words
Arsenic Keratosis; Bowen's disease; Basal cell carcinoma.

Introduction

Chronic exposure to arsenic through drinking water and food can result in arsenical keratosis, which is a premalignant condition of skin, characterised by hyperkeratotic papules and plaques in palms and soles, raindrop pigmentation of skin and Mees lines in nails apart from predisposing the affected individual to develop Bowen's disease, squamous cell carcinoma and basal cell carcinoma. Early identification of arsenical keratosis and preventing further exposure is very much essential as these patients may develop the above said malignant conditions resulting in significant morbidity and mortality. Arsenical keratosis is of public health importance as many cases in the vicinity can be affected and appropriate steps should be taken at community level.

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Case report

A 47 years old male, presented with multiple well demarcated erythematous patches and plaques with few lesions covered with scales and crusts **(Figure 1a)**, few hyperpigmented **(Figure 1b)**, and few ulcerated, which were distributed over face, trunk and both upper and lower extremities with itching as chief complaint. These lesions started before 5 years and were increasing in size and number. Palms and soles showed multiple hyperkeratotic corn like plaques **(Figure 1c)**. There were multiple guttate hypopigmented macules distributed in the background of hyperpigmentation **(Figure 1d)**. Hair and nails were normal, and no regional lymphadenopathy was found. History of several people from same area with similar skin lesions at various points of time over the past 15 years with few mortality have been reported. Patient has stopped taking water from the affected area for the last five years. Routine hemogram, and biochemical profiles were normal. 24 hours urine level of arsenic was found to be 26mcg/L. Highest levels of arsenic from the affected area have been found to be about 1362 µg/L from previous studies.[1] Two incisional biopsies were

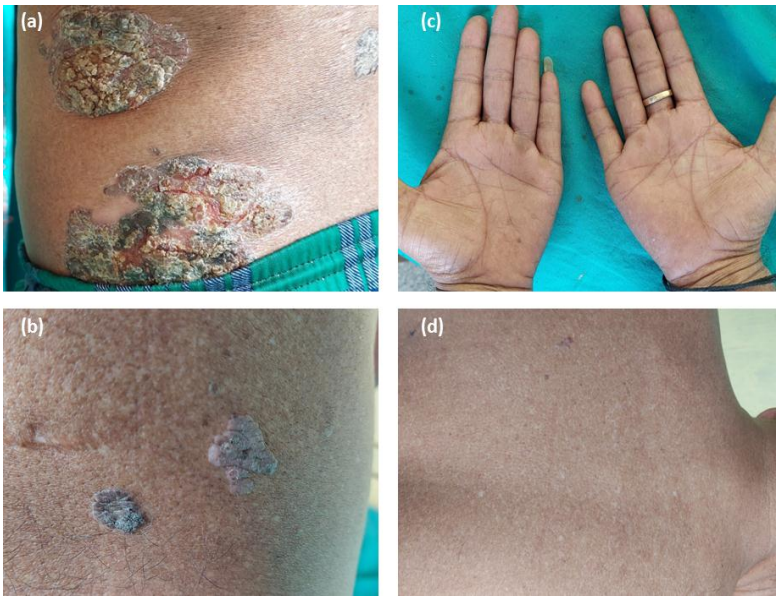

Figure 1

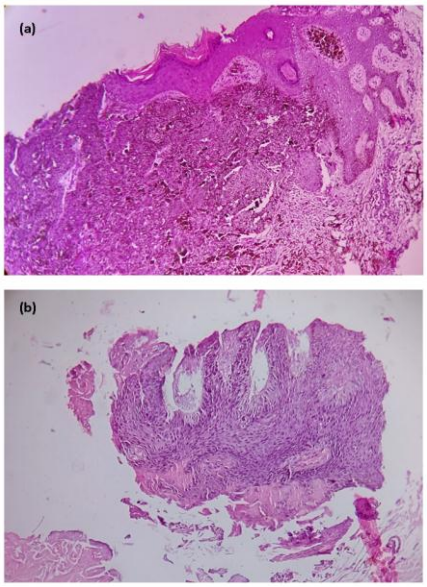

Figure 2

taken, one from a crusted plaque and another from pigmented plaque and sent for histopathological examination.

Biopsy from pigmented plaque showed unremarkable epidermis except for loss of rete ridges and superficial dermis showed proliferation of undifferentiated basaloid cells in nest and lobules with prominent palisading in the periphery **(Figure 2a)**. The cells showed scanty cytoplasm and frequent mitosis; Increased density of melanocytes and melanin pigmentation was noted with pigment incontinence and the stroma showed infiltration of inflammatory cells along with collection of melanophages, the findings of which were consistent with pigmented basal cell carcinoma.

Biopsy from crusted plaque with verrucous surface showed epidermis of skin with central ulceration and the adjacent epidermis showed full thickness keratinocyte dysplasia **(Figure 2b)** along with areas showing proliferation of basaloid keratinocytes with downward extension of proliferating cells; underlying dermis showed focal dense infiltration of chronic inflammatory cells and pigment incontinence, the findings of which were consistent with Bowen's disease. Hence, final diagnosis of coexisting Bowen's disease and Pigmented basal cell carcinoma in a case of Chronic Arsenicosis was made. Topical 5-Fluorouracil once daily was advised for smaller plaques without ulceration and patient was started on oral acitretin, while the larger ones were surgically excised.

Discussion

Chronic arsenicosis occurs following ingestion of arsenic for a period of more than 6 months, which can occur through contaminated ground water, whole grains, tobacco, occupational exposure as in workers involved in smelting and mining.[2] Several cases have been reported from Eastern India and Bangladesh. Biomethylation of arsenic occurs following ingestion, rendering S-adenosylmethionine unavailable for methylation of DNA resulting in altered gene expression. Cellular stress also occurs due to reactive oxygen species production due to uncoupling of oxidative phosphorylation caused by arsenic[3] and several genetic polymorphisms have been identified in DNA repair pathways leading to carcinogenesis associated with arsenic exposure.

Cutaneous manifestations of chronic arsenicosis include diffuse hyperkeratotic papules and plaques in palm and soles, and pigmentary changes in the form of 'rain drop pattern' and 'raindrop on a dusty road pattern'. Transverse white lines of finger or toenails called Aldrich – Mees lines are also not uncommon. Systemic involvement in the form of chronic liver and lung disease, sensory and motor neuropathy, peripheral vascular diseases can occur. Histologically, arsenical keratosis is characterised by hyperkeratosis, parakeratosis, and atypia of the keratinocytes.

Measurement of arsenic in 24 hour urine and levels of more than 50 mcg/L is suggestive of recent exposure to arsenic. According to WHO guidelines, maximum permissible concentration of arsenic in drinking water 10 mcg/L.[4] Moreover, arsenic estimation in hair and nails may be helpful.

Preventing further exposure to arsenic is the mainstay of treatment. Localised lesions can be treated with electrodessication, cryotherapy, topical 5–Fluorouracil, 5% Imiquimod, topical retinoids and keratolytics whereas systemic retinoids are advocated in disseminated lesions with surgical excision of advanced lesions. Since malignant transformation is a dreaded complication, subsequent follow up visits should be planned, and it must be individualised according to the presentation of the patient and biopsies should be performed for earlier diagnosis and interventions.

References

1. 1. Mazumder DN, Ghosh A, Majumdar KK, Ghosh N, Saha C, Mazumder RN. Arsenic contamination of ground water and its health impact on population of district of nadia, west Bengal, India. *Indian J Community Med*. 2010;**35(2)**:331-8.
2. 2. Shajil C, Mahabal GD. Arsenical Keratosis. [Updated 2022 Aug 8]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2022 Jan-
3. Tchounwou PB, *et al*. Arsenic toxicity, mutagenesis, carcinogenesis - a health risk assessment and management approach. *Mol Cell Biochem*. 2004;**255**: 47
4. Kapaj S, Peterson H, Liber K, Bhattacharya P. Human health effects from chronic arsenic poisoning--a review. *J Environ Sci Health A Tox Hazard Subst Environ Eng*. 2006;**41(10)**:2399-428.