ATENOLOL-INDUCED PSORIASIFORM PHOTODERMATITIS EVOLVING INTO SEZARY SYNDROME - A CASE REPORT

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Development of sezary syndrome from photodermatitis induced by atenolol in a 74-year-old lady is described

Key Words : Atenolol, Sezary syndrome

Introduction

Sezary syndrome, the leukemic variant of cutaneous T-cell lymphoma (CTCL), was first coined by Sezary and Bouvrain in 1938. Though bewildering clinical presentations have been reported in CTCL, a pattern mimicking photodermatosis is rather unusual. We hereby report such a case.

Case Report

A 74-year-old lady was first seen for photosensitive psoriasiform lesions distributed over the face, upper chest and extensor forearms of 2 months duration. She was hypertensive and was taking atenolol 100mg daily for the past 2 years. Suspecting a drug reaction atenolol was discontinued and she was treated symptomatically. The rash subsided in a few days but recurred after 2 weeks following accidental intake of a tablet of atenolol. Inspite of symptomatic treatment, the rash persisted and it gradually showed a tendency to become lichenoid and progressed to erythroderma thus necessitating hospitalization. Ten months prior to the development of the rash, she was seen for a reticulate pigmentation on the trunk and extremities which was preceded by a generalized asymptomatic erythematous maculopapular rash. She was extensively investigated for an internal malignancy, but there was no evidence for it. The hyperpigmentation gradually subsided within 2 months. She was mildly icteric, had generalized non-tender, firm, discrete and mobile lymphadenopathy and bilateral pitting pedal oedema.

Fig.1. Diffuse infiltration of the face

Dermatological examination showed generalized erythema and scaling interspersed with lichenoid papular lesions and islands of normal skin. Palms, soles, mucosae and nails were unaffected. Systemic examination was normal except for a firm, non-tender hepatomegaly of 3cms. The upper border of the liver was in the (R) 5th intercostal

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space. She was anemic (Hb 7g/dl). TLC was 44,450 cells/ mm^3 and the differential count showed 70% abnormal lymphocytes and 20% neutrophils. RBCs showed poikilocytosis, nucleated and crenated forms.

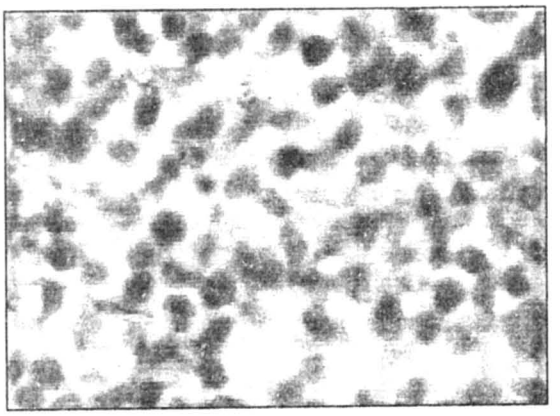

Fig.2. Lymph node biopsy showing atypical lymphocytes

Bone marrow was cellular with decreased erythroid: myeloid forms. Sezary cells were present. The skin biopsy showed dense infiltrate of lymphocytes, histiocytes and sezary cells in the upper dermis. Fine needle aspiration cytology from the (R) inguinal lymph node and the lymph node biopsy from the (L) cervical group showed numerous sezary cells.

Serum bilirubin was raised (3 mg/dl), conjugated being 0.8mg/dl. Albumin-globulin ratio was 2.8:3.5 g/dl. Blood urea, sugar, serum alkaline phosphatase and SGPT were normal. X-ray chest was within normal limits.

From the triad of erythroderma, lymphadenopathy and sezary cells is the peripheral blood, marrow, skin and lymph nodes, a diagnosis of sezary syndrome was made. She was discharged at request as the relatives were unwilling for chemotherapy considering her age.

Discussion

The etiology of CTCL is unknown though genetic, infectious and environmental factors are incriminated in its development. HLA B, Aw 31 and Aw 32 are found with increasing frequency.

Continuous cutaneous antigenic stimulation by chemicals (air pollutants, pesticides, solvents and vapors, detergents and disinfectants) and drugs like analgesics, tranquilizers and thiazides can predispose individuals to the disease.[1,2] Whatever be the antigenic stimulation, the early manifestations of the disease represent a reaction of the host to this particular agent, or to a small number of abnormal cells. The course of the disease is then determined by the host resistance.[3] The progression of the disease occurs either by decrease in host resistance or by an increase in the neoplastic potential of the cells.

The premycotic lesions may mimic a variety of benign dermatoses like psoriasis, eczemas, neurodermatitis, parapsoriasis en plaque, or poikiloderma.[4] Most commonly it occurs as a dull pink, macular or maculosquamous lesion which may be asymptomatic or slightly pruritic. The lesions may wax and wane and may even disappear completely with hyperpigmentation or dyschromia, as noted in our patient 6 months prior to the development of psoriasiform lesions. Since our patient developed psoriasiform rash to atenolol, confirmed by accidental re-exposure which later progressed to CTCL, it is likely that atenolol provoked the onset of the disease.

References

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