MACULAR AMYLOIDOSIS AND HYPOTHYROIDISM

Adarsh Chopra, Komal Pahuja, Dimple Chopra

A 53 -year -old woman presented with extensive pruritic hyperpigmented macules in interscapular area and extremities of four years duration. She was an established case of hypothyroidism on treatment for last four years.

Key Words : Macular amyloidosis, Hypothyroidism

Introduction

Primary localised cutaneous amyloidosis (PLCA) is a disorder in which amyloid deposition occurs in the skin in the absence of evidence of amyloidosis in other organs.[1] PLCA comprises of macular, papular (lichen amyloidosis) and nodular (tumefactive) forms.

Macular amyloidosis is a less common variant. It is a pruritic eruption of small brownish macules distributed in a rippled, symmetrical fashion in the interscapular area but more commonly the lesions are more extensively distributed over the back or chest. It is usually ideopathic but occasionally has been reported in association with autoimmune disease.[3]

Case Report

A 53-year-old woman presented in our OPD with complaints of pruritic hyperpigmented lesions in interscapular area and extremities for the last four years. Dermatological examination revealed pruritic eruption on the back, arms and legs comprising of small brown macules which were coalescing to form plaques with rippled pattern. On further exploring she came out to be a case of hypothyroidism on treatment for last four years.

Laboratory investigations showed an elevated ESR (60mm 1st h). Full blood count and urine biochemistry were within normal limits. Thyroid function tests were highly deranged (T_3 = 0.19 Ngm/ml; T_4 = 1.24 mgm/dl; TSH = 110.7 ul/ml). The serum cholesterol levels were also high (323 mg%).

Ultrasonography of thyroid revealed hyperechoic lesions in right lobe which gave the impression of adenoma. There was no lymphadenopathy. Serum calcitonin level was within normal limits.

Histopathological examination of elliptical biopsy taken from a small brown macule from the leg stained with haematoxylin and eosin showed marked pigmentary incontinence with drooping of melanin pigment in upper dermis associated with a sparse amount of lymphohistiocytic cell infiltrate in relationship to vessels of sub papillary plexus. The congo red stain revealed congo red positive areas in upper dermis confirming amyloidosis.

Discussion

The patient fulfilled the criteria for being labelled

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as a case of hypothoyroidism.[4] The histopathological examination and dermatological examination were suggestive of the case being of macular amyloidosis. The association of hypothyroidism and macular amyloidosis has not been reported as a discrete case. The familial association of hereditary cutaneous lichen amyloidosis and MEN IIa (Multiple endocrine neoplasia IIa) have been described.[2] But the present case is a discrete case of hypothyroidism with macular amyloidosis.

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DERMATITIS HERPETIFORMIS INTOLERANT TO DAPSONE IN AIDS

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A 35-year-old man with AIDS and pulmonary tuberculosis presented with lesions suggestive of dermatitis herpetiformis and intolerance to dapsone. He was managed successfully with a combination of nicotinamide 200 mg/day and indomethacin 75 mg/day, topical steroids and gluten free diet.

Key Words : Dermatitis herpetiformis, Dapsone, AIDS

Introduction

Dermatitis herpetiformis is a chronic blistering skin disease, presumed to have an autoimmune etiology with associated gluten sensitive enteropathy. It is associated with thyroid disorders. Other autoimmune diseases (SLE, rheumatoid arthritis, Sjogren's syndrome, ulcerative colitis) are associated due to high frequency of HLA B8.[1] Raynaud's phenomenon, glomerulonephritis, vitiligo, diabetes mellitus and atopic dermatitis also occur more frequently in patients with dermatitis herpetiformis than in normal population.[2] Dermatitis herpetiformis is probably a new association of AIDS, hitherto undescribed.

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Case Report

A 35-year-old man diagnosed to have HIV infection with pulmonary tuberculosis presented with grouped, bilaterally symmetrical, intensely pruritic, excoriated papular and papulo-vesicular lesions over the posterior axillary folds, buttocks, extensor forearms, knees, shins and ankles. The lesions left behind hyperpigmentation and scarring on involution. There was mild hepatosplenomegaly and generalised, non tender, lymphadenopathy on systemic examination, and occasional episodes of diarrhoea.

Routine hemogram, urinalysis, blood sugar level were within normal limits. ELISA and Western Blot for HIV were strongly positive. Chest X-ray showed infiltrates suggestive of pulmonary tuberculosis. The patient refused