RESEARCH ARTICLE

A case of Lichen Planus at a glance.

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56 years old female, with BMI 24.76 kg/m², Hypertensive since one year on ARB inhibitor workacoholic reported in Dermatology OP attached to Madurai Medical College with hyperpigmentation over right frontal region and bluish black pigmentation in the tongue and oral cavity sudden in onset.

Proviosionally diagnosed as Lichen Planus.

The following investigations were done:-
1. Urine analysis- nil relevant
2. Complete Hemogram- RBC-4.68 millions/ cu mm normocytic normochromic
   a. WBC- 8,300 cells/ cu mm
   b. DC- P41%, L52% E7%
   c. Hb 12.1Gms %
   d. ESR 10 mm at the end of 1 hour.
   e. Platelet 2.36 lakhs / cu mm
   f. PCV 40.6 %
   g. MCV 86.8 fl
   h. MCH 25.8 pg
   i. MCHC 29.8 g/dl
4. Creatinine 0.81 mg/dl.
5. Lipid profile revealed Hypertriglyceridemia and hyper VLDL cholesterolemia with fasting sample.
6. G6PD Activity, (Qualitative ) EDTA whole blood Indophenol Dye Decolourisationmanuel Method -20 minutes (0-60)
7. G6PD Activity, Quantitative blood by Kinetic Assay, Modified method of Kornberg and Horecker 9.7 U /g Hb (4.6-13.5)
8. X ray Chest PA view- normal
9. ECHOcardiogram had adequate LV function.

Epidemiology:-
The exact incidence and prevalence is unknown. Worldwide occurrence is less than 1 % Le Cleach. No racial and sexual predilection has been observed. Females are usually affected in their 50s and 60s due to seasonal or environmental factors Boyd. There’s no familial predisposition and is non infectitious.
Lichen Planus in Greek leichen means, ‘tree moss’, in Latin planus means ‘flat’ is a unique, idiopathic common inflammatory disorder that affects the skin and mucous membrane.

The term lichen planus was introduced by Erasmus Wilson in 1869.

The 4 Ps- purple, polygonal, pruritic and papule is the mnemonic device used to recall the constellation of symptoms and skin findings of Lichen planus.

Aetiology:-
Viral infections, autoimmune diseases, medications, vaccinations, restorative dental materials and immunological mechanisms trigger this disease.

Pathogenesis:-
Cell mediated immunity triggers the expression of this disease. CD4 and CD8 T cells are found in skin lesions of Lichen planus. The epithelium –lymphocyte interaction can be divided into 3 stages---

i. Specific Antigen recognition
ii. Cytotoxic lymphocyte activation and
iii. Keratinocyte apoptosis.

LP triggers T cell mediated autoimmune damage to basal keratinocytes that manifests as altered self antigens on their surface.

Clinical findings:-
Initial lesions always appear bilaterally and symmetrically on the extremities, tends to be pruritic and spreads within 1-4 months from onset. Face is usually spared. Oral involvement is generally asymptomatic but sometimes extremely painful.Scratching, injury or trauma induces Koebner response. Oral involvement occurs in 60-70% of patients with Lichen planus. Surface of the lesion is usually smooth and shiny. Lesions are 3-5 mm in diameter and may have whitish criss-cross streaks on their surface (Wickhamsstriae). Diabetes mellitus is a possible association.

Laboratory tests:-
The Total count and Lymphocytes are reduced. Dyslipidemia is more common. Diabetes has to be ruled out. Histology of skin lesions present with irregular acanthosis of the epidermis with hypergranulosis and compact hyperkeratosis.

Treatment:-
Its always challenging and discouraging for both patients and physician. Good oral hygiene and regular personal and professional dental care needs to be encouraged. Topical steroids are the first line therapy in mucosal lesions. Dapsone 100-200 mg daily has proved effective in 2/3 rds of patients with cutaneous and oral disease. It is safe to be alert about anaemia, methaemoglobinemia, liver damage with jaundice while using Dapsone.

Conclusion:-
Lichen planus is an inflammatory, T cell mediated autoimmune disorder affecting the skin, hair, nails and mucous membrane. Onset is insidious. Papules flatten after a few months and there may be a gradual change in colour from pink to blue to black. Dermoscopy has to be done periodically. Direct immunofluorescence shows globular deposits of Ig M and occasionally Ig G and Ig A. Management depends on the localisation, clinical form and severity Peramiquel.
In this patient, when oral Hypoglycemic agents with topical and systemic steroids were started, lesion is getting gradually subsiding along with lifestyle modification. As G6PD levels are normal, Dapsone therapy may be recommended provided there’s a recurrence.

Anyhow patient is asked to report regularly for follow up to avoid remission of disease.

Bibliography:
3. Fitzpatrick’s Dermatology in General Medicine-Page 296-312.
5. IADVL’s Concise Textbook of Dermatology by VisalakshiViswanath page 213-218