ORIGINAL CONTRIBUTIONS

TREATMENT OF LICHEN PLANUS WITH PROPRANALOL

K Pavithran

Accidental observation of early cure of lichen planus in a female patient who received propranalol for the treatment of associated hyperthyroidism made us to try this drug in 20 patients with lichen planus. The cutaneous lesions responded very well to the treatment in 90% of the cases. The mucous membrane lesions responded only partially and slowly. Another group of 20 patients with lichen planus who received only oral pheniramine maleate showed persistence of skin lesions even after 3 months. There were no serious side effects with propranalol, though one patient with an atopic background, developed bronchial asthma for the first time at the age of 21. The excellent therapeutic response to propranalol—a beta-adrenergic receptor blocker, in these patients indicates further study.

Key words : Lichen planus, Propranalol, Treatment.

The cause of lichen planus is still a subject for debate. The principal hypotheses have been based on infectious origin, immunological abnormalities, neurologic changes and emotional stress.[1] Numerous remedies that have been advocated include heavy metals, calcium gluconate, aspirin, antihistamines, tranquillizers,[2] griseofulvin,[3] isoniazid,[4] retinoic acid[5] and benzathine penicillin. Until the introduction of topical or systemic corticosteroids, the treatment had little effect on the duration of the disease. Observation of dramatic relief from acute lichen planus in a 32-year-old female who received propranalol for the treatment of associated hyperthyroidism, made us to try this drug in 20 patients with lichen planus.

Materials and Methods

Ten male and ten female patients with lichen planus (Group 1) who attended this hospital were randomly selected for the study. They were normo-tensive and there was no evidence of any cardiac disease, bronchial asthma, diabetes mellitus, psychic depression or other systemic diseases. Histopathological study of the skin lesions and routine laboratory tests on blood, urine and stools were performed in all the cases. Propranalol in a dose of 20 mg, thrice daily orally was given to each patient and followed up every two weeks. Depending on the clinical response, the dose of propranalol was gradually reduced and finally withdrawn at the end of the third month. They were not prescribed any other topical or systemic medicines. Another group of 20 patients with lichen planus (Group 2) who were given only oral pheniramine maleate, 22.5 mg thrice a day also were observed periodically.

Results

The ages of the patients in group 1 varied from 16 to 42 years (mean 32 years), and the duration of the skin lesions varied from 12 days to 8 weeks. Twelve patients had chronic localised form of lichen planus, while the rest had the acute widespread type. Mucous membrane of the cheeks was involved in 6 patients and pruritus was predominent in all the cases.

From the Department of Skin and VD, Medical College Hospital, Kottayam-686008, India.

Dramatic relief of pruritus and flattening of the skin lesions after propranalol therapy were observed in 14 patients at the end of one month, after which the dose was reduced to 10 mg thrice daily and finally withdrawn at the end of 3 months. In 4 patients therapeutic response was very slow and the dose of propranalol could be reduced only after 2 months. Two patients did not show any response at all and the drug was withdrawn after two months. Two to three months after stopping the drug, four patients experienced a mild relapse of the skin lesions which disappeared on reintroduction of propranalol for a further period of two months. There was no evidence of regression of the mucosal lesions in four cases at the end of three months No serious side effects were noted after propranalol therapy. Five patients however, felt increased fatigability and one with a family history of atopy, developed bronchial asthma for the first time at the age of 21. None of the patients in group 2 showed evidence of regression of the skin lesions even after 3 months of treatment.

Comments

The cause of lichen planus remains uncertain. Rarely, it has been associated with neurologic diseases such as syringomyelia, bulbar paralysis and peripheral neuritis. This has led to support a neurogenic cause for lichen planus. Cases of lichen planus have also developed simultaneously with tumours of paravertebral localization thought to be exerting pressure on the sympathetic pathways.[1] Midana and Ormea[6] found proliferation of the peripheral nerve tissue in the early papules and an 'irritative condition' of the sympathetic ganglia. He concluded that the autonomic nervous system from the beginning takes an active part in the pathological process. Hypertension coincident with lichen planus has also been found in a small percentage of patients.[1,7] Owing to the pronounced adrenergic component in hyperthyroidism, various adrenergic antagonists have been employed in the management of this disease. Our patient developed acute lichen planus along with the early signs and symptoms of hyperthyroidism. Dramatic relief of lichen planus and subsidence of the adrenergic manifestations of hyperthyroidism such as sweating, tremor and tachycardia after propranalol therapy, in this patient, further support the view that lichen planus may be of neurogenic origin. Majority (90%) of our patients who received propranalol, had symptomatic relief of pruritus and regression of the skin lesions within a short time even though the mucous membrane lesions persisted. Only 4 had a mild relapse of the skin lesions which disappeared on reintroducing the drug. Spontaneous resolution of the skin lesions in lichen planus can be expected in 15 months.[8] So propranalol seemed to have caused an early cure of lichen planus in more than 90% of our patients. This suggests that sympathetic part of the autonomic nervous system possibly plays some role in the pathogenesis of lichen planus. Betablockers antagonize the effects of catecholamines and sympathetic nerve stimulation on heart. A central mechanism of action has been suggested to explain the beneficial effect of propranalol in various anxiety states.[9] Propranalol is the most widely used and least toxic among this group of drugs. No serious side effects were noted in our patients though one patient who had a family history of atopy, developed bronchial asthma for the first time during this treatment. This was probably induced by propranalol and thus supports the betablockade theory of atopy.[10] Personal or family history of asthma is a contraindication for propranalol therapy. It is also contraindicated in cardiac diseases, diabetes mellitus, psychic depression and psoriasis.[11]

References

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