Current Dermatological Therapy

A series of articles on the current dermatological therapy will feature in the pages of the journal. Articles are contributed this year by Dr. P. Sugathan, M.D., Associate Professor, Department of Dermatology & Venereology, Medical College Hospital, Calicut.

NEURODERMATITIS - THE WINDOW TO THE SOUL

Mrs. P. was a healthy 45 years old woman of ample proportions, who may be called 'lucky' by any standard : looks, family background, education and social status. She was married to an intelligent business man who was devoted to her. Of her 3 children 2 were already well settled and the other was studying for a dignified profession. The house she lived in was built to please her, in the choicest locality of the city. She had enjoyed good health all along. But since two years she had been suffering from an itchy, dark patch over the nape of the neck. It was insidious in onset and slowly enlarging. She could not remember any specific reason to account for it. The itching was incessant and intolerable. The lesion responded to treatment initially but promptly reappeared on stopping it. The lesion was a small oval patch of lichenification and on clinical grounds the most obvious diagnosis was lichen simplex chronicus.

It is almost always possible to make a sure, simple, straight-forward diagnosis of lichen simplex chronicus (LSC). But is it enough? Certainly not. That small patch of lichenification is a window through which you can–if you are interested–look into the innermost corners of the patient's mind. LSC is perhaps the only dermatological problem where you have to take an extensive history—personal, familial, and social—to establish or guess the possible aetiological factor. I dare say that if you have the time and patience to probe deeper you will unravel the most bewildering and fascinating situations in human relationship. Compared to it the best of fiction is just pale imitation.

In the case of Mrs. P. the problem was apparently simple. A natural culinary expert, she enjoyed giving occasional dinner parties to her friends and relations. She used to cook with her own hands the choicest food and serve it with meticulous care. After a while her husband pointed out that they were not invited out as often as they give dinner parties. If at all they were invited the dinner consisted of perfunctory frugal meals. He started making subtle objections when she planned such parties, which she ignored. The objections became more vociferous and gradually lead to unplesant exchange of insults. This was the beginning of her neurodermatitis.

That a circumscribed area of lichenification could be an indication of the inner turmoil of an individual is not at all surprising if we recall that the skin participates frequently as a substitute organ of expression for pent-up emotional tension[1]. As the recipient or sounding board of this nervous bombardment it reacts with a wide variety of patterns. They are vascular dilatation

or constriction, increased vascular permeability, modification of glandular activity, sensory radiations and kinetic activities like rubbing, scratching and picking of the skin and its appendages. However it will be fallacious to assume that LSC is always due to deep seated emotional conflicts. At best it is only a reaction pattern of the skin to repeated rubbing and scratching[2]. Therefore it may be initiated by any pruritic dermatosis, when it is best classified as secondary lichenification. Distinction between primary and secondary lichenification is not easy because it has the peculiarity of self perpetuation even after the original cause has subsided.

LSC is almost always situated in a relatively accessible part of the skin[3]. Localization of the lesion is thought by some to have a definite symbolism[4]. Lesions over the nape of the neck indicate family trouble: on the face, back and front of the neck shame and over the knees, ankles and shoulders excessive marital and family responsibility. Localization to the thighs and anogenital region has been related to sexual disorders[5]. There is considerable controversy about these interpretations. The highest incidence of LSC is in the 30–50 age group but it may be seen at any age from adolescence. It is never seen in children perhaps because other forms of traumatic tics related to the skin are more characteristic of childhood such as nail biting, hair pulling and sucking the thumb[6]. The incidence of LSC is high in the caucasian females but among the orientals it is apparently more in the males[7].

The cardinal symptom of LSC is the paroxysm of intense pruritus which is out of proportion to the objective changes. It is often triggered off by emotional stress or even touch. The earliest lesion is an illdefined oedematous patch over which the skin markings are prominent. This is gradually replaced by pigmentation and mild scaling. Surrounding this is a zone of lichenoid papules and beyond that an area of slight thickening which merges with the normal skin. The clinical picture may however be modified by the site and duration of the lesion. For example, over the scalp it is often seen over a birth mark and may appear as a hyperkeratotic nodule with twisted hair. In areas where there is lax subcutaneous tissue, tumour like plaque with warty cribriform surface develops. Prurigo nodularis and giant lichenification of Pautrier are really clinical variants of LSC. A form of discrete lichenoid papules readily developing in certain individuals with pruritic dermatosis is called pebbly lichenification and will resolve in a fortnight with simple treatments[8].

The ability to develop lichenification varies considerably in the population. It is related to the color of the hair and eyes in the caucasians because lichenification is three times more common in brunettes as in blondes[9]. In my experience those who develop intense postinflammatory pigmentation also show a higher incidence of lichenification. Atopics are generally prone to develop lichenification[10,11]. But other workers have failed to confirm this view[12].

The most significant histological findings in LSC are hyperkeratosis, parakeratosis, pseudoepitheliomatous hyperplasia and proliferation of Schwann cells; a feature shared by psoriasis and lichen planus[13]. Schwann cell proliferation is maximum in prurigo nodularis when it could be termed Schwannomas. A battery of histochemical studies did not reveal any peculiarities to point towards any specific abnormalities[14]. However Scott[15] demonstrated increased acetylcholine content of the skin in atopics and in patients suffering from LSC.

As the membrane permeability is affected by acetylcholine he postulated that this is significant in the development and course of the disease. Disturbed sterol metabolism was reported by Cooper et al[16] but its significance is not known. Though the rate of epidermal proliferation is similar to or even greater than psoriasis transepidermal transit time in LSC is not as fast as in psoriasis. This is perhaps due to a larger epidermal volume, through which the cells have to travel[17]. Rubbing and scratching produce intraepidermal damage of cells which accounts for the parakeratosis seen in LSC[18]. Efforts to induce lichenification under laboratory conditions with a scratching machine have shown that visible pigmentation occurred on the third day and a total of 6–8 weeks were necessary for visible lichenification[19]. It was also shown that histological lichenification sets in much earliear than any visible changes. Absence of hyaluronidase activity in LSC lesions perhaps accounts for the dry nature of the lesion[20].

The term Neurodermatitis emphasizes only one of the many aetiological factors and hence is considered to be undesirable by some. However large number of studies[1,4,21-24] indicate that emotional factors are of prime importance in the genesis of LSC. Unexpressed rage, guilt, hidden anger as well as unsatisfied craving for love are all mentioned as possible factors in the genesis of neurodermatitis. Any one of these will result in symptomatic expression in the form of scratching which is a conversion reaction. These patients are highly sensitive to life situations which involve or threaten loss of approval, status or love. It is likely that more than one factor may be operative in one particular patient. In other words the genesis of LSC is multifactorial.

Podophyllum[25,26] Thorium-x[27], Shale oil[28] and plaster cast were popular prior to the introduction of topical steroids[29,30]. The outlook now is certainly better with potent topical steroids with or without occlusion and intralesional injections. Successful management of primary LSC requires active co-operation from the patient. A careful psycho-social history is essential to detect the underlying emotional pathology. This could be a tricky problem with a patient who is practically a stranger at the first visit. After prescribing a tranquilizer like diazepam as well as antibiotics and compresses when there are signs of infection or eczematisation, broad hints may be given as to the nature of the disease. Once the patient's confidence is obtained gentle, sympathetic probing will readily bring to light the problems he is facing in his environment. Asking the patient to demonstrate the method of scratching the site will reveal valuable information. In primary LSC the patient always anchors his scratching hand in the vicinity of the lesion as if to conserve the maximum energy solely for the act of scratching. Even if this is not very successful to begin with, as the interview progresses he will resort to the usual accustomed style of scratching without undue muscular movements. Personal insight into the customs, beliefs and rituals of the patient, his caste, community and religion are all helpful in rapidly identifying the reason for his anxiety or tension. For certain patients a chronic stabilized skin disorder may be the best equilibrium they can achieve[31]. Such patients are better left alone after the acute symptoms are controlled, lest they develop neurotic symptoms pertaining to an internal organ.

After obtaining adequate co-operation from the patient and controlling the acute phase, the drug of choice for local use is topical steroids. Two important points have to be kept in

mind while prescribing local steroids. Twenty one per cent of topically applied steroid is absorbed through the normal skin and 35% through an eczematous skin[32]. This is increased nearly 100 fold if administered under occlusion[33]. Prolonged use of topical steroids can induce tachyphylaxis – i. e. tolerance to the action of a drug on constant use[34]. Both these phenomena are faster to occur with the potent topical steroids now available[35].

The introduction of intralesional steroid therapy did indeed revolutionize the treatment of LSC. Originally carried out with a Mantoux syringe it is now done with ease using a dermojet. The dose is 0.1 ml of triamcinalone acetate 5 mgm/ml intralesionally at 1 cm apart once in 6 weeks. The only contra indication to the use of a dermojet is the location of the lesion on the face or over blood vessels where there is the risk of embolism. Improper use of dermojet over the eye brows have resulted in a case of retinal detachment. The troublesome side effect of intra lesional steroid therapy is atrophy and hypopigmentation at the site of injection, which normally disappear after 6 months. Though opinions differ I prefer to use betamethasone for intralesional use on exposed areas as it does not produce any visible atrophy or hypopigmentation. Once the underlying emotional problem is effectively solved and the patient has understood the genesis of the disease and the meaning of the treatment the prognosis is good.

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