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TURN-OVER OF STRATUM CORNEUM IN LEPROSY

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Stratum corneum showed increased proliferative activity on the patches of leprosy as evidenced by a significantly fast stratum corneum turn-over time ($p < 0.001$) measured by fluorescent staining technic with dansyl chloride. These findings suggest imperfect keratinization on the patches of leprosy leading to formation of structurally weak stratum corneum.

Key words : Stratum corneum, Turn-over, Leprosy.

Dryness of the involved areas of skin in leprosy has been attributed to destruction of the autonomic nerve fibres impairing sweat gland function.[1,2] It is now well recognised that dryness of the skin is largely dependent upon the water-binding/water-holding power of the stratum corneum (SC), and any condition, inherent or acquired, where SC is damaged or imperfectly keratinized, it tends to get dry, more so in the months of winter when cracks and fissures appear.[3,4] Such damaged SC may be associated with an altered proliferative activity of the integumentary epithelium.

Although, counting mitosis or labelling the nucleus or cytoplasm with radio-active substances, or in recent years pulse cytophotometery are the recognized research methods, measurement of the SC turn over time (SCTT) is a simple but reasonably precise method to estimate the proliferative activity of the epidermis.

In the course of study of different parameters to assess the functional ability of the SC in leprosy, poor hydration power of the affected SC and poor cutaneous insensible perspiration on the patches of leprosy have been recorded.[5,6] In continuation with the same study, the estimation of SCTT has been undertaken in leprosy by fluorescent staining technic with dansyl chloride.[7]

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Materials and Methods

The subjects were divided into two groups. Group A comprised of 12 lepromatous and borderline lepromatous (L and BL) cases,[3] all males, age group 30-40 years having the disease for the last 5 to 10 years, mostly under anti-leprotic treatment, slit and scrape skin smear showing AFB positive (+ to +++), and lepromin-A test negative. Fourteen healthy volunteers in the same age-group served as controls. Group B consisted of sixteen tuberculoid, borderline tuberculoid and pure neuritic (T, BT and N), all males, age-group 15-45 years, duration of illness 1 to 30 years, most of them without any anti-leprosy drugs, skin slit and scrape smear negative for AFB and lepromin-A test positive (+ to ++). In this group, the adjacent unaffected skin of the same patients served as control.

The method for measurement of SCTT was adopted from Janson et al.[7]

Results

Tables I and II show the SCTT in days in group A (L and BL) and group B (T, BT and N) patients respectively. The SC turn-over is much faster in both the groups as compared to the controls ($P < 0.001$).

Comments

The increased proliferative activity of epidermal tissue in leprosy may lead to formation of imperfect and structurally weak horny layer which may fail to provide the first line of defence

Table I. Stratum corneum turn-over time (SCTT) in days in L and BL patients and controls.

Mean (±SD) values in	
L and BL patients (12 patients)	Controls ((14 subjects))
9.25±1.479	13.214±1.612
t—Test : T=6.279, DF=24, p<0.001 (highly significant)	

Table II. Stratum corneum turn-over time in days in T, BT and N patients and controls.

Mean (±SD) values in	
T, BT and N patients (16 patients)	Controls (16 subjects)
9.625±1.218	11.625±1.495
Paired t—test : T=6.076, DF=15, p<0.001 (highly significant)	

against the varied environmental assaults. Poor hydration power of the SC and poor cutaneous insensible perspiration on the patches, make the SC in leprosy dry and brittle which becomes more pronounced in winter months as the hydration of SC is known to have temperature dependence.[9] Such SC is liable to form minute cracks or fissures especially on the dependant parts of the body. Secondary infection may supervene through these cracks and fissures which is considered important in causing trophic ulcerations.[10]

There is one more aspect of this increased epidermal proliferative activity recorded in leprosy. In spite of rapid advances made in the understanding of structural and chemical characteristics of epidermal cell differentiation and keratinization, not much is known of the exogenous and endogenous factors which control or influence the keratinization process.[11] Petrone[12] reported accentuation of ichthyosis vulgaris in one leg of a patient after accidental denervation of that limb. He also reported increased SCTT in the affected limb of the patient. Whether destruction of peripheral nerve fibres as present in leprosy could disturb the keratinisation process remains a question warranting further study and elucidation.

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