

QUANTITATIVE EVALUATION OF CUTANEOUS THERMAL SENSATION IN PSORIASIS, MORPHOEA AND VITILIGO

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A quantitative assessment of cutaneous thermal sensation was undertaken in 12 psoriasis, 7 morphoea and 12 vitiligo patients. The thermal sensory threshold (TST) was determined both at the lesion site and the lesion-free contralateral site (control) by an electronic device fitted with a probe and a temperature sensor. As the differences in TST between the contralateral regions hardly exceed 1°C in normal individuals, a difference in TST \geq 3°C was considered significant. It was observed that about 83% of psoriasis and 71% of morphoea cases showed a significant difference in TST (\geq 3°C), but no such difference existed among the vitiligo patients.

Key words : Thermal sensation, Psoriasis, Morphoea, Vitiligo.

Psoriasis, morphoea and vitiligo may clinically mimic Hanseniasis in certain instances. The converse is also true. Moreover, psoriasis and morphoea may show impairment of cutaneous sensibility.¹ Among the different modalities of sensation, it is the temperature sensation which is usually the first to be lost in leprosy. The temperature sensation was therefore assessed quantitatively in the above three dermatoses.

Materials and Methods

In all 31 patients, 12 psoriasis (8 M, 4 F), 7 morphoea (3 M, 4 F) and 12 vitiligo (5 M, 7 F) constituted the sample population for the study. They were, however, free from any systemic disorder especially psychological or neurological. The mean age of the subjects and the mean duration of the lesions were respectively 39.5 years and 4.5 months in psoriasis, 21 years and 10 months in morphoea and 20 years and 19 months in vitiligo. All the cases were diagnosed clinico-histopathologically. The availability of

disease-free identical contralateral site corresponding to the lesion under study was an important criterion for selecting the patients.

An electronic instrument,² named Thermosense^(R) (designed by Messers Kar electronics, Pune, India), was used for this study. It essentially consists of a testing probe, a temperature sensor and a control unit. The probe can be heated by a heating element to any desired temperature upto 70°C and maintained constant by the control unit. Sensed by a precisional thermocouple, the probe temperature is displayed on a digital meter.

The method consists in heating the probe from the room temperature in steps of 1°C and to make each time an instantaneous (< 3 sec) but firm contact with the affected site till the subject just feels a heat sensation. At this stage, as instructed, the subject gives a signal and the value as displayed in the digital meter is read as the TST of the affected area. The TST of the uninvolved contralateral site is also similarly measured and the two TST values are compared.

As the difference of TST between contralateral regions in normal individuals hardly exceeds 1°C,² a difference of 3°C or more was considered as a significant difference by allowing an additional margin for personal equations and other influencing factors.

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Table I. Thermal sensory threshold (TST) in three dermatoses

Disease	Number of cases	Difference in TST		Number (percentage) of cases showing difference in TST $\geq 3^{\circ}\text{C}$
		Minimum	Maximum	
Psoriasis	12	1 $^{\circ}\text{C}$	18 $^{\circ}\text{C}$	10 (83.3)
Morphoea	7	0 $^{\circ}\text{C}$	5 $^{\circ}\text{C}$	5 (71.4)
Vitiligo	12	0 $^{\circ}\text{C}$	2 $^{\circ}\text{C}$	0 (0.0)

Results

Significant difference in TST values at the lesion site and the lesion-free contralateral site were observed in about 83% of psoriatic patients and 71% of morphoea patients, but there was no such difference among the vitiligo patients (Table I).

No correlation was found between the duration of the lesions and the degree of thermal sensory deficit in morphoea and psoriasis.

Comments

Significantly high TST in psoriatic lesions may be due to thickened parakeratotic horny layer,^{1,3} disrupted cutaneous innervation³ and degeneration of nerve fibres.⁴ Dharmendra¹ observed that if scales are properly removed, sensory impairment would not be found in psoriasis. This is possibly not the whole truth, because cutaneous neural factors³⁻⁵ are also responsible for increased TST. In order to keep the original morphology of the lesions intact, scales were not removed in our study. Weddell et al⁴ studied the cutaneous thermal sensation in psoriasis using test tubes of 0.5 inch diameter containing water upto a depth of 2 inches at a temperature of $15 \pm 2^{\circ}\text{C}$ above or below that of the skin surface. No change in cutaneous thermal sensibility was found by them. But undoubtedly the method used by them was not standardised and lacked precision. They also did not mention if the scales were removed or not.

As regards morphoea lesions, the significant

thermal sensory deficit may be accounted for by the involvement of perineural cells in the sclerotic process,⁶ condensation of collagen and even calcification of deep dermis and subcutaneous tissue³ and neurohistopathological changes.⁷

The probable explanation of having no significant TST changes in vitiligo lesions is that the degenerative neural changes observed in vitiligo occur only in the autonomic nerves and not in the sensory ones.⁸

It appears therefore that the cutaneous sensory impairment even if assessed quantitatively, does not necessarily indicate leprosy unless other cardinal features confirm the diagnosis.

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