Indian J. Dermatol. Venereol. Leprol. 1989; 55 : 25-29

RESPONSES TO THERMAL STRESSES IN PSORIASIS OF HAND

Soma Mukherjee, A B Gupta and B Haldar

Fifteen subjects having psoriasis on the hand, comprising both sexes, between 22 and 56 years in age and suffering for durations varying from 10 months to 20 years, each with a sex and age-matched control were studied. A thermistor thermometer measured the skin temperature in ambient condition at the finger-tip and at selected sites on the dorsa of hands and fingers, both at lesion site and lesion-free adjacent ones. In all cases except one, the thermistor readings were significantly lower ($p<0.01$) in the psoriatic lesions than in the uninvolved adjacent skin. The hands were next subjected to thermal stresses : first, by immersion in an ice-water bath at 0°C (cold challenge) and later, in a hot-water bath at 45°C (heat challenge), each for a period of 60 seconds. Immediately after each stress period, the finger-tip temperatures were recorded. In psoriatic subjects, the finger-tip temperature dropped on cold challenge, and the amount of drop expressed as percentage of the basal temperature was significantly less ($p<0.01$) than the corresponding value for the control. There was no significant difference ($p>0.05$) in the response to the heat challenge between psoriatic and normal subjects. It appears that there is some fault in the local vasomotor tone in subjects with hand psoriasis.

Key words : Thermal challenge response, Psoriasis.

Psoriasis has an excessive turn-over of epidermis.[1] Previous work in the field indicates that there is a failure of the normal thermo-regulatory homeostatic mechanism in psoriatic subjects.[2] It was in this context that the present investigation was undertaken to study the response of subjects suffering from common plaque type psoriasis of hand to thermal stresses such as cold challenge at 0°C and heat challenge at 45°C.

Materials and Methods

A group of 15 patients having plaque type psoriasis on the dorsum of hand for durations varying from 10 months to 20 years and comprising both sexes in the age range 22 to 56 years constituted the sample population. Majority of them had similar lesions in other usual sites as well. For each patient, a normal subject of the same sex and age was used as a control.

The skin temperature was first measured by a point contact thermistor thermometer (Electromedics Inc, USA) at selected sites on the dorsa of hands and fingers, both at the lesion sites and the normal appearing lesion-free adjacent ones. The measurement was carried out inside a room with no wind velocity and at known ambient temperature and relative humidity. The temperature of the finger-tip was next measured in both the patient as well as the age and sex-matched control by the same thermistor in a similar environment. The time required to take a reading by the thermistor ranged from 20 to 45 seconds.

The hands were thereafter subjected to thermal stresses. First, they were immersed in an ice-cold water bath at 0°C for 60 seconds as cold challenge. Immediately after the cold challenge, the thermistor readings of the finger-tip were taken and the time-temperature data recorded to note the response of the skin to the cold challenge. The same procedure was repeated for the corresponding control.

From the Physics Unit, Indian Statistical Institute, Calcutta-700 035, and Calcutta Skin Institute, Calcutta-700 054, India.

Address correspondence to : Dr. A B Gupta.

Table I. Thermal data in hand-psoriasis in ambient condition.

Number of subjects	Mean temperature (°C) ± SD			
	Lesion site	Lesion-free adjacent site	Finger-tip	
			Patient	Control
15	33.7 ± 1.09	34.2 ± 0.98	33.1 ± 1.69	33.3 ± 1.31
Paired t-test	$t = 5.99$ $p < 0.001$		$t = 0.38$ $p > 0.50$	

Ambient temperature 26-29°C; Humidity = 50-52%

Next the hands were immersed in hot water bath maintained at 45°C for the same duration of 60 seconds. The finger-tip temperatures were read immediately after the stress-period by the same thermistor to obtain the time-temperature data to study the cutaneous response to the heat challenge. Identical procedures were followed for the corresponding normal controls.

The immersion of hands in well-mixed water served as a heat clamp by enveloping and holding the skin at known temperatures of 0°C and 45°C during the cold and heat challenges respectively.

Results

Table I shows the mean skin temperature at psoriatic lesions and lesion-free adjacent sites of the patients and also the mean finger-tip temperature of both the psoriatic and the control groups in ambient condition. The individual values of the temperature at the lesion site varied from 31.3 − 34.9°C with a mean of 33.7 ± 1.09°C. At the lesion-free adjacent sites, the individual values ranged from 32.0 − 35.2°C having a mean of 34.2 ± 0.98°C. The mean finger-tip temperature in ambient condition for the psoriatic group was 33.1 ± 1.69°C, and the corresponding value for the control group was 33.3 ± 1.31°C. The individual values ranged from 28.2 − 34.8°C for the psoriatic group and 30.0 − 34.5°C for the control group. The lesion temperature (x_1) was always lower than the lesion-free temperature (y_1) except in one case where these were equal. The difference ranged from 0.2-1.1°C with an average value of 0.48 ± 0.28°C. This difference was highly significant ($p<0.001$) and the two sets of data were strongly positively correlated ($r = 0.90$). The regression equation is $y_1 = -0.19 + 0.99x_1$. The analysis of the ambient finger-tip temperature data however, revealed no group difference ($p>0.5$).

Table II shows the individual data giving the thermal response for the psoriatic and the control groups. The response has been shown as the percentage change in the finger-tip temperature on cold and heat challenges with respect to the ambient tip-temperature, the basal temperature. While in the heat challenge, the percentage increase in the temperature varied from 1.1 to 22.7% for the patient, the corresponding range for the control was from 1.2 to 27%. The group difference, on analysis, was found not to be significant ($p<0.50$). The situation however, was different on cold challenge. While both the groups showed a reduction in finger-tip temperature, the percentage decrease in the psoriatic group was always lower than that of the control group and the difference was highly significant ($p<0.01$). A positive correlation ($r = 0.74$) was obtained between the ambient temperature of the finger-tip (x_2) and its percentage reduction (y_2) on cold challenge in the control group only. The regression

Table II. Responses to thermal stresses in hand-psoriasis.

No.	Age/Sex	Percentage change in finger-tip temperature (°C) with respect to basal			
		Cold challenge		Heat challenge	
		Patient	Control	Patient	Control
1	24/F	50.6	53.2	3.2	1.2
2	22/F	46.3	53.2	3.5	1.2
3	32/M	46.1	51.9	2.8	9.9
4	45/M	37.3	40.3	2.0	3.2
5	56/M	32.1	44.6	2.6	3.5
6	22/F	48.5	53.2	3.6	1.2
7	53/M	25.5	52.8	8.6	4.4
8	26/F	44.5	48.1	2.7	10.5
9	27/F	45.7	48.1	5.7	10.5
10	30/M	39.6	42.1	3.9	27.0
11	28/M	28.7	30.6	8.7	21.0
12	29/F	46.0	48.1	1.1	10.5
13	38/M	35.1	50.0	19.5	4.1
14	39/M	36.8	50.1	22.2	3.7
15	40/F	34.0	47.2	22.7	10.9
	Paired t-test	$t = 3.49$ $p < 0.01$		$t = 0.24$ $p > 0.50$	

equation was given by $y_2 = -65.83 + 3.41x_2$. No such correlation ($r = 0.23$) however, was found to exist in the psoriatic group.

Comments

Two distinct observations that stand out from these studies are, that (i) the psoriatic plaques were cooler than the clinically unaffected adjacent skin as determined by point contact thermometry, and (ii) the response to cold challenge was less in psoriatic subjects than in normal ones.

Capillary loops in the dermal papillae of psoriatic lesions were recognised long ago to be dilated and tortuous. In established plaques, there was marked dermal vascular and lymphatic dilation with doubled blood flow. Due to this faster flow and a chronic inflammatory condition, a higher temperature at the lesion site, compared to the adjacent uninvolved skin, was expected. But the observation was contrary to it and it remained unaltered even after removal of some keratin from the plaques. The discrepancy may be resolved if cutaneous radiative and evaporative heat losses are taken into account. At the lesion site, the blood vessels are much closer to the external environment. So, a faster flow rate of vascular blood would increase the radiative heat loss. The proximity of the vessels to cooler air (26°C to 29°C) outside would also result in a rapid evaporative heat loss. An increase in the transepidermal water loss in psoriatic lesions resulting in a considerable evaporative loss has been well documented.[3] These two factors — radiative and evaporative heat loss, could cause a lowering in the skin temperature at lesion sites by a small but significant amount.

A more or less similar study conducted by Warshaw[2] confirmed that the psoriatic plaques were cooler compared to the uninvolved peripheral skin. He also used infrared thermography and found that psoriatic plaques indicated a higher temperature than the surrounding tissue. The apparent reversal could be attributed to an increased emissivity of the

plaque area.

The significant positive correlation between the temperature of the psoriatic plaque and the uninvolved adjacent skin is rather expected for the two variables are not quite independent as these relate to the same individual.

In the extremities such as finger-tip, peripheral vasomotor tone is a response to local ambient temperature. This is not a thermoregulatory response through the central nervous system. Open-loop local control can alter the diameter and the volume of blood vessels to maintain a constant internal temperature.[4] Since the finger-tip of psoriatic subjects do not get as cold as their counterparts do, it appears that the local vasomotor tone in psoriatic subjects is defective The significant correlation between the tip temperature and its percentage reduction on cold challenge in the control group, and its absence in the psoriatic one also lends indirect support to this view. Such investigations conducted by Warshaw et al[5] with a smaller sample size showed more or less similar results. They also performed Doppler venous flow studies for both the groups but the results were inconclusive.

The thermoregulatory physiology is complex and the skin behaves rather variably in its several roles in temperature regulation. Thus a simple explanation of skin disorder in thermoregulation appears highly unlikely. Ryan[6] reported that limb elevation causes pallor of normal skin but not of an untreated plaque of psoriasis and that a greater manual pressure is needed to empty the capillaries in involved psoriatic skin. Thus an abnormal obstruction to outflow may exist at the junction of the capillaries with the subpapillary venous plexus in involved psoriatic skin. Klemp[7-9] studied the local regulation of cutaneous blood flow (CBF) and subcutaneous blood flow (SBF) in psoriasis by using Xe^{133} labeling wash out technique. In involved psoriatic skin areas, he observed a deviation from normal local autoregulation and a concomitant increase in both CBF and SBF. While the morphologic abnormalities in cutaneous microvasculature are well known, no such anatomic abnormalities in the subcutaneous tissue have been reported. He attributed the increase in CBF to the abnormal morphology of papillary capillaries and the increase in SBF as a secondary phenomenon due to increased CBF rather than to physiologic abnormalities in the vascular tree of cutaneous and subcutaneous tissues.

Recently, Braverman[10] reported that during the healing process of psoriatic lesions by common treatment modalities (methotrexate, topical steroid), the histopathologic features of psoriatic epidermis reverted almost completely without any accompanying changes in the microvasculature. This lack of response of dermal capillaries to treatment during the initial clinical improvement raises the possibility of prompt clinical relapses in psoriasis on withdrawal of therapy. Topical corticosteroids produce vasoconstriction. It is thus likely that a decreased blood flow would supply less nutrients to the epidermis and this might initiate epidermal normalisation.

It appears that the contribution of microcirculation is important for both the development and regression of psoriatic lesions and further studies of the phenomenon involving measurements of the capillary pressure, skin perfusion pressure etc are necessary. But morphologic studies alone may not be sufficient in this regard.

References

1. Fitzpatrick TB, Eisen AZ, Wolff K et al: Dermatology in General Medicine, McGraw Hill, New York, 1979; p 233.
2. Warshaw TG: Thermal studies in psoriasis, J Invest Dermatol, 1973; 60 : 191-193.
3. Shaidullah M, Raffle ET and Frain-Bell W: Insensible water loss in dermatitis, Brit J Dermatol, 1967; 791 : 589-597.
4. Bligh J, Cottel D and Maskrey M: Influence of ambient temperature on thermoregulatory response, J Physiol, 1971; 212 : 377-392.
5. Warshaw TG, Alexander H and Curtis G: Quantification of thermoregulatory mechanisms in relation to psoriasis, in: Bioengineering and the Skin, First ed, MTP Press, Hague, 1981; p 311.

6. Ryan TJ: Microcirculation in psoriasis: blood vessels, lymphatic and tissue fluid, Pharmacol Therap, 1980; 10 : 27-64.
7. Klemp P : Local regulation of cutaneous blood flow in psoriasis, J Invest Dermatol, 1984; 83 : 421-425.
8. Klem P : Subcutaneous blood flow in psoriasis, J Invest Dermatol, 1985; 84 : 187-190.
9. Klemp P : Regulation of local subcutaneous blood flow in patients with psoriasis and effects of anti-psoriatic treatment on subcutaneous blood flow, J Invest Dermatol, 1985; 85 : 175-178.
10. Braverman IM and Sibley J : The response of psoriatic epidermis and microvessels to treatment with topical steroids and oral methotrexate, J Invest Dermatol, 1985; 85 : 584-586.