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NONCONVENTIONAL REMISSION OF MILIARA RUBRA

DURING HEAT ACCLIMATION: CASE REPORT

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PRECIS

A white male exhibiting <u>miliaria rubra</u> underwent an 8-day laboratory evaluation. In spite of daily periods of exercise-heat exposure $(40^{\circ}C, 48\%$ RH) and profuse sweating, <u>miliaria rubra</u> was no longer visible by day 3. Sweat gland function, thermoregulation and physiological adaptations during heat acclimation were normal. This is the first report to verify that humans can accomplish heat acclimation while <u>miliaria rubra</u> is healing. Climatic and hygienic factors which contributed to this remission of <u>miliaria rubra</u> have been examined in light of current theories regarding the origin of sweat gland obstruction.

Miliaria rubra (prickly heat, lichens tropicalis) is one of the most common skin disorders observed in tropical climates, consisting of small red papules on a mildly erythematous skin. Although usually classfied as a minor skin disorder, miliaria rubra may be the precursor of impetigo, folliculitis, furunculosis, or anhidrotic heat exhaustion (1,2). Severe cases involve diurnal irritation, discomfort and isomnia to a degree that causes a deterioration of overall health. The most commonly affected skin areas are the shoulders, trunk, axillae and waist (2). Histological examinations (3) implicate obstruction of the sweat duct by keratin debris, which forces sweat into periductal squamous epithelium. The origin of the keratin plug remains uncertain, but the predominant theories focus on nonspecfic injury to skin (e.g. maceration, irritating clothing, adhesive tape) (2), microbiological inflammation (4,5,6), hypertonic sodium chloride solutions (7,8), and combinations of the above. The depth of sweat gland obstruction distinguishes miliaria rubra from miliaria crystillina, miliaria pustulosa and miliaria profunda (3).

Most reports of <u>miliaria rubra</u> originated in the Pacific theatre during and after World War II. Sulzberger & Emik, for example, observed that 66 per cent of the Naval personnel studied on Guam in 1945 (n = 261) exhibited <u>miliaria rubra</u> at some time during their first 7 months of duty (1). The incidence of <u>miliaria rubra</u> was not significantly influenced by race, obesity, complexion, place of former habitat, gender, or sun tanning. The mean time of onset was 6 months, although some cases occurred immediately upon landing. Similarly, Sanderson & Sloper (9) observed British Army personnel in Singapore, and reported that the number of <u>miliaria rubra</u> cases reached a 50 per cent incidence at 5 months of exposure, followed by a gradual decline out

to 24 months (10 % incidence); some soldiers were affected within one week of arrival.

The purpose of this case report was to describe clinical and laboratory evaluations of a soldier with a past history of intermittent <u>miliaria rubra</u>. Admitted four times in three years, R.G. was referred for an 8-day laboratory thermoregulatory evaluation because severe <u>miliaria rubra</u> may result in impaired heat tolerance (2,10,15). Interestingly, R.G's skin condition improved in spite of daily exercise-heat exposure and living in non-air conditioned quarters during his 8-day evaluation. Because the most commonly prescribed treatment for <u>miliaria rubra</u> is to avoid heat exposure and heavy sweating (2,3,4,11), this nonconventional remission of <u>miliaria rubra</u> opposes previous recommendations and clarifies the treatment of soldiers stationed in peacetime training assignments.

CASE REPORT

R.G. was a 26 yr old white male (height - 188cm, weight - 92.7kg, surface area - $2.00m^2$) with a history of intermittent skin rash caused by 5-14 days of repeated heat exposure, since the age of 14 yr. R.G. had no previous history of heat stroke, heat exhaustion or heat cramps. Admitted for <u>miliaria rubra</u> four times in three years, the affected skin areas included the abdomen, back, shoulders, chest and lateral surface of the arms to the elbows--pressure areas for wet t-shirts. Treatment consisted of lotions to control mild desquamation, which occasionally accompanied <u>miliaria rubra</u> outbreaks, as well as avoidance of heat and sweating. Immediately prior to referral for laboratory testing, R.G. was stationed in Alabama at a training school which required strenuous daily training and exercise in a hot, humid environment.

Recreational sports such as softball and flag football were also a part of his daily schedule. R.G. commented that he rarely had time for showers during field maneuvers and that he spent many hours in a sweat-soaked uniform. R.G. was referred to Massachusetts for laboratory thermoregulatory evaluation because of his individual susceptibility to <u>miliaria rubra</u> and his unknown potential for heat stress injury.

The purposes of laboratory testing were to evaluate (a) sweat gland function (b) heat acclimation and (c) thermoregulation in a hot environment. All tests were conducted during the month of June in an environmental chamber maintained at 40°C, 48% RH. R.G. wore only shoes, socks and nylon shorts during heat exposures. Days 1 and 8 involved an exercise step test (30 cm high, 12 steps/min,120min). Days 2-7 consisted of a 90 min treadmill walk (3.5 mi/hr, 5% grade). Blood samples were taken from an antecubital vein before and after exercise on days 2, 5, 6 and 7. Heart rate was monitored via ECG telemetry system. Rectal and skin (chest, arm, leg) temperatures were measured using thermistors. Whole body sweat rate was calculated by using body weight differences (+ 10g), and local sweat rate was measured at five locations using a dew point sensor. Sweat electrolyte concentration was evaluated via a whole-body washdown procedure. The number of active sweat glands (per cm^2) were counted on microphotographs of the skin surface, taken 45 sec after application of a thin layer of petroleum jelly (Fig. 1). Trials were terminated if heart rate exceeded 180 beats/min or if rectal temperature exceeded 39.5°C.

On days 1 and 2, the <u>miliaria rubra</u> was visible on the back, upper arms, shoulders and chest. R.G. reported a mild stinging sensation occasionally, but was not bothered by itching. Miliaria rubra was not visible on days 3-8, in spite of daily standing (40 - 50 min), exercise (66 - 90 min), and heavy sweating (1.4 - 2.6 L/hr) in the heat. A skin biopsy (right scapula) on day 8 was negative for miliaria rubra but was positive for chronic folliculitis, which was sparsely distributed over the skin. This finding supported previous reports that miliaria rubra may be a precursor of folliculitis (1,2). Daily entering body weight was stable, urine volume ranged from 1.572 - 4.867 L/24h, and daily entering urine specific gravity ranged from 1.014 - 1.027, reflecting R.G.'s attempts to thoroughly rehydrate during off-duty hours (days 1-8). Thermoregulation and the ability to acclimate to heat were judged to be normal, based upon changes in day 2 vs day 7 heart rate (170 vs 155 beats/min), rectal temperature (39.5 vs 39.3°C), trial duration (71 vs 90 min), sweat Na+ concentration (57 vs 23 mEq/L), 24-hr urine Na+/K+ ratio (2.6 vs 1.5) and whole-body sweat rate (1.350 vs 2.155 L/hr). Local sweat rate (Table I) also uniformly increased (day 2 vs day 7) at the forehead, chest, back, arm and thigh. Heat acclimation was incomplete because full adaptation requires 10-14 days. The mean number of active sweat glands (Table I) were in accord with previous observations of normal males and were not statistically different on days 2 and 7, as expected. Pre- vs post-exercise venous blood analyses indicated no remarkable changes in eight hematological measures and 15 chemical analyses, including the blood enzymes ALT, AST, CPK, and LDH.

DISCUSSION

The 8-day evaluation of R.G. was similar to many modern military training scenarios in that periods of heavy sweating, rectal temperature elevations and exercise in the heat were combined with garrison living. Our observations indicated that miliaria rubra subsided on this schedule of heavy sweating and

exercise-heat exposure, contrary to many previous recommendations (2,3,4,11). A comparison of physiological measurements on days 2 vs 7 indicated normal heat acclimation adaptations. This finding has important military implications because it is the first recognition that soldiers may successfully undergo heat acclimation training (e.g. 90 min of exercise in heat) while being treated for <u>miliaria rubra</u>.

Considering the current theories regarding the origin of sweat gland obstruction via hypertonic sodium chloride solution (7,8) or microbiological inflammation (4,6), daily showers after periods of heavy sweating appear to be prudent. Evaporating sweat exposes the skin to hypertonic salt solutions, but this may not present a problem if the skin is cleaned regularly. In fact, Lowenthal et al. verified that salt water bathing was a predisposing factor in the development of <u>miliaria rubra</u> and that fresh water showers taken immediately after ocean bathing tended to prevent or minimize this disorder (12). Even a high sodium chloride concentration in sweat (12) and an increased dietary intake of salt (13) have been implicated as etiological factors in the onset of <u>miliaria rubra</u>. Although modern training scenarios typically do not involve water constraints, it is significant that one recent report (14) recommended only one field shower (4.5 gal each) every 84 hr in water-restricted, arid, battlefield environments.

Artifical <u>miliaria rubra</u> (polyethylene wrapping for 48-72 hr) induces acute hypohidrosis which is lineraly related to bacterial numbers at the affected site (4). This hypohydrosis is not resolved for 9-21 days, and coincides roughly with the 14 day turnover time of the stratum corneum (4,15). Impaired heat tolerance also results from severe artifical <u>miliaria rubra</u>. The critical surface area which produces heat intolerance is related to the

affected region of the body (10). For example, smaller rashed areas of the trunk may produce responses to heat stress similar to larger rashed areas of the limbs, because the trunk has a greater sweat production potential. Although R.G. exhibited <u>miliaria rubra</u> primarly on the shoulders and trunk, the sweat gland function measurements (Table I) indicated neither hypohidrosis nor obvious abnormalities. In addition, R.G. utilized none of the previously recommended topical treatments for <u>miliaria rubra</u>, such as neomycin lotion (5), dusting powder (16), keratolytic agents (1), or anticholinergic agents (17).

We believe that two factors were primarily responsible for the observed healing of <u>miliaria rubra</u>. First, R.G. showered after each heat exposure. Second, his increased sweat rate coincided with the reduction of sweat Na⁺ concentration, as normal sweat gland adaptations during heat acclimation. Both of these factors involved the reduction/removal of residual Na⁺ from the skin surface. In addition, both factors argue in favor of regular field showers as well as heat acclimation procedures prior to deployment to hot climates.

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Human subjects participated in these studies after giving their informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 for use of volunteers in research.

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TABLE I

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EVALUATION OF SMEAT GLAND FUNCTION

		WHOLE-BODY	П	OCAL SWE	AT RATE	*		NO. ACTIVE	SWEAT Na
VNO	MODE CF EXERCISE	SMEAT RATE (L/hr)	FOREHEAD	(mg/cm ² /min) CHEST BACK /	2/min) BACK	ARM	THICH	SMEAT GLANDS (per cm ²)	CONCENTR. LFION (mBg Na+/L)
	step test	1.238	3	Э	2	2	e	57 ± 3	
7	treadmill	1.350	m	2	m	e	7		57
m	treadmill	1.761							
4	treadmill	1.671							
S	treadmill	1.428							43
9	treadmill	2.555							
٢	treadmil1	2.155	4	9	4	9	S		23
80	step test	1.316	4	Ś	5	4	4	52 ± 4	

* - taken after 40-50 min standing in heat (pre-exercise)

** - represents the mean (±SD) of 10-15 counts (see Fig. 1)

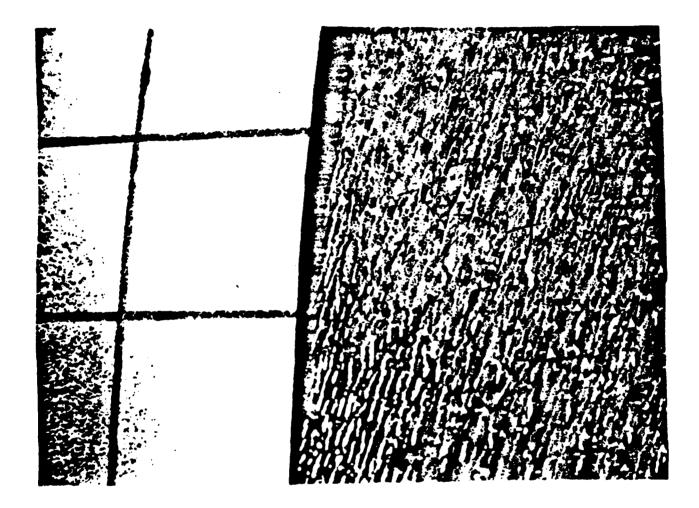


FIG. 1. Microphotograph of skin surface of patient RG (day 1), taken 45 sec after application of a thin layer of petroleum jelly. Note beads of sweat on skin surface. Grid squares are 1 cm^2 each.

