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Eccrine Function in Psoriasis Inversus

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THE stimulus to this study was an observation that in obese psoriatic patients the presence of intertrigo appeared to determine the localization of psoriatic lesions. It has been shown that active psoriatic lesions are anhidrotic, that visible sweating cannot be elicited by parasympatheticomimetic or thermal stimuli and that healed sites may remain anhidrotic for many months.¹ Since intertrigo is associated with sweating, an explanation was sought for the moist and macerated clinical appearance of aberrant lesions of psoriasis in skin folds (psoriasis inversus).

Patients were examined by tests for sweating to determine whether psoriatic lesions of the axilla and of the internatal cleft were anhidrotic and to explain the source of the moisture which led to maceration and absence of scaling.

SELECTION OF PATIENTS

Clinically, it is difficult to make a diagnosis of a solitary internatal plaque of psoriasis, and a provisional diagnosis may await confirmation by the appearance of psoriatic lesions elsewhere on the skin surface.

When psoriatic lesions are confined to flexural areas, psoriasis inversus may be difficult to diagnose clinically, and when the clinical diagnosis is in doubt, the histopathological changes are usually non-specific.² The patients included in this study were selected to satisfy the following criteria:

1. They had extensive lesions of psoriasis elsewhere than in the axillae and internatal cleft.
2. The axillary lesions fitted into the general distribution of psoriatic lesions of the trunk and arms. The patients with internatal psoriasis showed a plaque which was typically psoriatic in the lower lumbar and sacral region and confluent with the internatal lesion under study.

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ABSTRACT

Seven patients with psoriatic lesions of axilla and internatal cleft were tested by thermal and intradermal methacholine stimulation using the quinizarin test for sweating to determine the source of moisture causing maceration and absence of scaling in psoriasis inversus.

Histopathological changes were non-specific: slight and patchy hyperkeratosis and parakeratosis, slight elongation of rete ridges and thinning of suprapapillary plates, microspongiosis and vesiculation in variably acanthotic prickle cell layer; dilated capillary loops less prominent than in typical psoriasis.

Lesions clinically and histopathologically consistent with psoriasis inversus were anhidrotic. Maceration was due to spread of sweat from adjacent uninvolved skin, spongiosis and vesiculation, serum exudation and insensible perspiration.

3. The histopathological changes, while not necessarily diagnostic, were consistent with a diagnosis of psoriasis inversus.

It was considered that this selection would provide reasonable probability that the lesions under study were, in fact, psoriatic.

The patients included in the appraisal consisted of seven white males ranging in age from 42 to 82 years. The disease was extensive in distribution, active, and of several years' duration. The patients were under treatment but discontinued application of topical medication for 24 hours before tests were made.

METHOD

Methacholine chloride 0.1 ml., one part in 250 of distilled water, was injected intradermally into

a test site. The lesion was immediately dried by application of Kleenex® tissue with firm pressure and dusted with quinizarin compound powder. The test site was observed for 15 minutes. The ambient conditions varied from 70 to 82° F. and 50 to 70% relative humidity. Thermal sweating was induced by placing the subject in a room at 85 to 98° F. The test sites were dried and dusted with powder as above. Biopsies were obtained with 4 mm. Keyes punches, and the tissues were fixed in formalin and stained with hematoxylin and eosin.

CLINICAL OBSERVATIONS

Tests were first made on persons with clinically normal skin until it was considered that satisfactory and reproducible results were being obtained. An ameboid pattern of sweating with a centripetal extension was observed. This, as noted by Muller and Kierland,³ is thought to represent dispersal of methacholine through the lymphatic channels. Apparent darkening of the quinizarin powder was noted at follicular orifices in some areas immediately after dusting on to the skin.

Tests were made on psoriatic lesions of the trunk and limbs, as in Susskind's study,¹ and then the same method was used on lesions of the axillae and internatal cleft.

In some cases a narrow band, up to 2 mm., of clinically normal skin around the lesion was observed to be anhidrotic. There was evidence of hyperhidrosis around some lesions as described by Sulzberger and Herrmann;⁴ this was not examined quantitatively.

AXILLARY LESIONS

Two patients studied included one with almost universal psoriatic erythroderma. This patient's clinically uninvolved skin showed profuse sweating when the room temperature was raised to 82° F., and this response was enhanced by methacholine injection. The axillae, which were completely involved in the psoriatic process, were anhidrotic. Immediately after the powder was dusted on, a slight colour change was noted at the follicular orifices, of the same degree as that previously noted on normal skin.

Axillary lesions in the second patient were also anhidrotic or showed a very sparse pattern of sweating in contrast to the profuse sweating observed on surrounding normal skin.

INTERNATAL CLEFT

Seven patients were tested by heat and by methacholine injection. The buttocks were taped apart and, after drying of the internatal lesions, thermal or methacholine stimulation was used. With thermal stimulation there was a profuse outpouring of sweat on the clinically normal skin of the lower lumbar region. When the patient was erect (or when lying in some cases), this sweat

poured from above into the internatal cleft. This outpouring of lower lumbar sweat was also noted when patients were at room temperature and being prepared for methacholine injection. It was found that the internatal psoriatic lesions were anhidrotic or showed a very sparse pattern of sweating in comparison with the pattern on the clinically uninvolved adjacent skin. The internatal lesions tested were pruritic, and at some sites crusted abrasions were present; at the site of crusting the powder darkened. As soon as the powder was applied, this discoloration occurred as a smear rather than as the usual pattern of sweating noted elsewhere; it was not exaggerated following the administration of the agent used to elicit sweating. It was found that this smear discoloration from exudate was usually distinguishable from the ordinary sweat pattern.

In some patients, sometimes on one occasion but not on another, there was discoloration of the powder at the mid-line of the internatal cleft adjacent to the anus. This discoloration was observed immediately after dusting on the powder and was enhanced following thermal stimulation.

MICROSCOPIC FINDINGS

McKee and Foster,² in a study of the histopathogenesis of aberrant lesions of psoriasis, found that lesions of intertriginous areas were clinically erythematous, eczematoid, macerated and often fissured. In their cases the condition in practically all instances had been diagnosed as eczema but had many features of psoriasis. The pathological picture had been altered, so that in 30% of the sections spongiosis and vesiculation were noted. The epidermis was moist in 32% of the lesions, as against 2% of cases of typical psoriasis. The thickness of the suprapapillary layer tended to be normal. Parakeratosis was patchy or sparse. The granular layer was better developed and acanthosis was somewhat more general. Spongiosis was noted in 28% of the sections, as against none in those of typical psoriasis. Vesicles were found in 24% of the sections. The papillary bodies and the subcutis showed less edema and the vessels did not show the marked dilatation so generally found in psoriasis. They found that psoriatic lesions which closely resembled seborrhea had a pathological picture that fitted mid-way between the pattern of typical psoriasis and that of their 26 cases of the eczematous type.

Burks and Montgomery⁵ reviewed the histological changes in psoriasis including the aberrant lesions. They observed that localized neurodermatitis was the most difficult lesion to distinguish histologically. This condition shared with psoriasis *inversus* an eczematoid pattern of reaction characterized by spongiosis and vesiculation. Helwig⁶ noted that the so-called aberrant lesions generally showed more irregular acanthosis with spongiosis. Thinning of the epithelial plaques was less marked and the capillary loops were less prominent.

Our biopsy specimens were obtained to determine that the appearances conformed to the descriptions of the histopathology of psoriasis inversus by these workers. In addition, biopsies were taken from special sites where a smearing pattern of discolouration of the quinizarin powder was noted.

Biopsy specimens of psoriatic lesions in the internatal cleft showed changes which were similar to those described above. There was slight and patchy hyperkeratosis and parakeratosis. There was slight elongation of the rete ridges and slight thinning of the suprapapillary plates. Microspongiosis and vesiculation were present in the variably acanthotic prickle cell layer. The capillary loops in the papillae were dilated, but less prominently than in typical psoriasis. The dermis showed a perivascular infiltration of lymphocytes, plasma cells and occasional macrophages.

Biopsies from sites showing a smearing pattern of discolouration of quinizarin powder which was suggestive of serum exudate revealed erosions of the epidermis. The erosions were covered by keratin debris admixed with numerous degenerating polymorphonuclear leukocytes and some fibrinous material, as well as necrotic cells and occasional red blood cells. The adjacent dermis showed an intense inflammatory cellular reaction and fibroblastic proliferation.

DISCUSSION

In the axillae, follicular discolouration of the powder may have represented an extra thickness of the powder layer in the follicular depression, "follicular eccrine sweat"⁷ or serum exudate which accumulated in the follicular orifices and was not removed by drying. The appearance was not exaggerated by heat. This discolouration was readily distinguished from the pattern of eccrine sweating. The possibility exists that this moisture might represent apocrine sweat. Injections in the axillae are painful, and pain might be expected to elicit apocrine sweating. No increase, however, was noted in this follicular discolouration of the powder during 15 minutes after injection. The problem of follicular discolouration was not encountered in the internatal cleft. After this experience with axillary lesions, the question arose concerning the distinction between sweat and serum exudate as the cause for colour change in the powder in the internatal cleft. It appears that at present this problem is unsurmountable because all the usual tests for sweating, including that for detection of chloride as described for cystic disease of the pancreas,⁸ do not distinguish between serum and sweat. Filter papers, prepared with potassium chromate as in the chloride method, were exposed to serial dilutions of serum and of normal saline. It was found, as may be predicted mathematically, that high

dilutions of both in a concentration equivalent to sweat gave a positive test. This problem did not in fact affect the findings, since the lesions, with the exception mentioned, were free of moisture.

Maceration and absence of scaling of lesions of psoriasis inversus do not arise from delivery of sweat to the surfaces of the lesions themselves. The lesions are anhidrotic, and a combination of the following factors appears to be responsible:

1. Hyperhidrosis of surrounding uninvolved skin.
2. In the case of the internatal cleft, sweat from the lower lumbar and sacral skin pours down the funnel of the cleft. This was noted with patients at room temperatures and is exaggerated by heat and probably by emotion.
3. Spongiosis and vesiculation in the epidermis; this reaction of the skin is intermittent.
4. Serum exudation at sites of erosion of the epidermis.
5. Insensible perspiration. This is known to be increased threefold to tenfold at the site of a psoriatic lesion.⁹
6. Apocrine sweat. No evidence for or against a role played by apocrine sweat is provided in this study.

Some degree of sweat retention with or without miliaria is noted in many dermatoses;¹⁰ anhidrosis in psoriasis appears to be more severe, and miliaria is absent. A clinical distinction between psoriasis and seborrheic dermatitis of the scalp, and between psoriasis and dermatoses affecting the internatal region, may be difficult, so that a simple diagnostic aid would be valuable. Arnold¹¹ discussed anhidrosis as a diagnostic aid in leprosy. The quinizarin powder method has disadvantages as a rapid clinical test, and photographs of the tested sites are required for record purposes. The simpler sweat test described by Muller and Kierland³ produces semi-permanent records, and is being evaluated as a clinical test to aid in the differential diagnosis of aberrant lesions of psoriasis.

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