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CAN PSORIASIS BE QUANTITATED?*

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The relatively static situation in regard to psoriasis research in recent years is partly due to lack of criteria by which to characterize the disease. Psoriatic lesions differ from normal skin in a number of ways, such as color, temperature, rate of scaling, electrical impedance (7), redox potential (4), and the rate of clearance of injected radioisotopes (1, 6). Some of these characteristics, as well as the total area of involved skin have been used to describe the progression or regression of psoriasis, but never in a satisfactory manner. The reason for this difficulty is partly due to inadequate mensuration techniques and lack of valid criteria regarding the weight to be attached to a change in one variable as compared to a change in another.

More importantly, a change in one variable suggestive of improvement may be accompanied by a concomitant change in another variable which could be interpreted as indicative of exacerbation. This is particularly noticeable when on occasion a striking blanching of all large lesions is accompanied by a no less striking increase in their surface area.

Since clinical evaluation of psoriatic patients is made regularly, and the assessment of the future course of the disease is usually satisfactory, it was thought worthwhile to attempt to isolate those characteristics which consciously or unconsciously determine the clinical impression in the hope that once they are identified they could be given formal expression and their relationships delineated.

This communication presents one such *ad hoc* derivation of what is termed in the body of the paper as status psoriaticus (SP). The derivation rests on the premise that satisfactory assessment of SP can be obtained on the basis of considerations involving only a.) fraction of skin area involved (A), b.) intensity of the process in the lesions (I), and c.) patient risk factor (R).

a. Surface Area Covered by Psoriatic Lesions

This area may be minimal or it may involve in extreme cases the entire skin of the patient. In line with usual clinical practice, the total area involved is considered in relation to the total skin area of the subject. Modified in this manner, this factor assumes the form A_p/A_t, where A_p and A_t refer to the aggregate surface area of the lesions and total surface area of the skin, respectively. Actual numerical evaluation of this ratio could be arrived at by using a scale such as is employed in the assessment of the extent of burned skin area (3).

b. Intensity Factor I

In evaluating the extent of the psoriatic involvement, it appears logical to correct the fraction A_p/A_t by the intensity of the psoriatic process in the lesions, since this varies

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depending on the severity of the disease. One could define it in terms of color or rate of clearance of isotopes, but since it is felt that the rate of scaling is intimately associated with the severity of the process, this marker is chosen in preference to others. The intensity factor is defined by

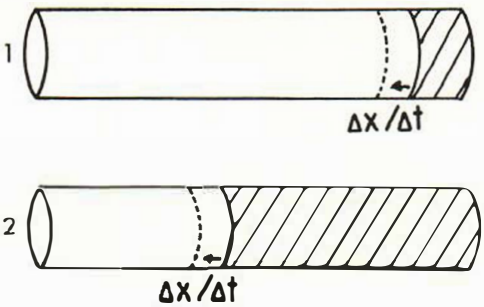
$$1. \qquad I=(S_p-S_n)/S_p,$$

where S_p and S_n represent rates of scaling of psoriatic and normal skin areas.

According to this expression, I tends to one when scaling of the lesions exceeds greatly that of normal skin, and when it reaches that of normal skin, the entire expression

$$(A_f/A_t)\times(S_p-S_n)S_p$$

becomes equal to zero.



Figs. 1 and 2. Mode of enlargement of psoriatic lesions. Cylinder represents a limb and the shaded area its psoriatically involved part. Rate of advance of leading edge is considered independent of surface area of involved skin. Rate of increase of psoriatic area is therefore given by length of leading edge multiplied by its rate of advance.

c. Patient Risk Status R

It is peculiar to psoriasis that exacerbation of the condition is usually associated with an increase in the surface area of the lesions, whereas improvement may occur without any apparent diminution in their size. Patient risk status simply refers to the likely rate of progression of the disease should a quiescent psoriasis become active. Numerical expression of this factor must therefore be based on considerations regarding the spreading process. Quantitative description of the latter can be approximately derived by considering Figs. 1 and 2, in which the cylinder is taken to represent a limb and the shaded area the psoriatically involved part. If spreading of the lesions were a function of the size of the involved area, the leading edge should advance faster in the case depicted in Fig. 2 than in the situation represented in Fig. 1. Clinical impression is against this view; if anything, it suggests that the rate of advance of the leading edge into normal territory is the same in both cases. Consequently, the rate of increase of psoriatic areas is expressed tentatively in the form

$$2. \qquad dA_{p\,(\text{single lesion})}/dt=L\,dx/dt,$$

where L is the length of the leading edge, or in this case the circumference, and dx/dt is its rate of advance in the direction normal to it. Assuming that the leading edge progresses in all lesions at the same rate, we can present the rate of increase of psoriatic areas in the form

$$3. \qquad dA_p/dt=L_{agg.}\,dx/dt,$$

where $L_{agg.}$ represents the aggregate length of all leading edges. Normally, $L_{agg.}$ is a function of time, but is constant in so far as the description of the instantaneous increment of A_p is concerned. dx/dt expresses the propensity of the uninvolved skin to be converted into psoriatic skin; it is largely independent of the intensity factor, since higher than normal scaling still characterizes quiescent psoriasis, and, as mentioned earlier, spreading of the lesions may coexist with their progressive blanching. These considerations suggest that the prevalent notion that in psoriasis small lesions may spread at the time when large lesions remain stationary is a misconception due to the circumstance that the rate of spread is proportional not to the area but to the length of the leading edge of the lesion, which is relatively longer the smaller the lesion.

Patient risk status expresses the belief of the clinician that the patient with a single lesion is better off than the patient with 10,000 small lesions of the same aggregate surface area. This impression is consistent

with equation #3, since when a quiescent disease becomes active—dx/dt being considered identical in both bases—dA_p/dt will be 100 times greater in the patient with many small lesions than in the patient having a single lesion, since in the former case the aggregate length of the leading edge is greater by a factor of $\sqrt{10{,}000}$. In line with this reasoning we define the patient risk status by the ratio $L_{agg.}/L_{min.}$ where $L_{agg.}$ represents the total aggregate length of the leading edge actually obtaining, and $L_{min.}$ is the minimal length of the leading edge, were all lesions fused into a single circular plaque. This ratio is numerically equal to $\sqrt{A_p/A_a}$, where A_a is the area of an average lesion, an expression which in some cases may be easier to evaluate.

The foregoing is summarized in the following definition of status psoriaticus:

$$4. \quad SP = A\ I\ R = \frac{A_p}{A_t} \times \frac{S_p - S_n}{S_p} \times \frac{L_{agg.}}{L_{min.}}$$

Changes in the disease status of the patient could be expressed in the form:

$$5. \quad \% \text{ change} = 100\ SP'/SP,$$

where SP' represents the status psoriaticus at some later date.

On the other hand, the effectiveness of a treatment may be expressed by:

$$6. \quad \text{Effectiveness} = (A_p + \Delta A_p) I'/A_p I$$

where the change in psoriatically involved area is given by $\Delta A_p = A'_p - A_p$; I' and A'_p representing I and A_p measured at the conclusion of the treatment.

One of the significant features of expression 4 and 5 is the sharp distinction drawn between the spreading of the lesion and the intensity of the proliferative processes in the lesion itself. The former is the result of one of the most salient feutures of psoriasis, i.e. the conversion of normal skin to psoriatic skin, a process which may have as one of its necessary terminal stages the disruption of the barrier properties of the epidermis. Such a disruption has actually been reported to characterize psoriatic lesions (5) and may as a consequence result in a higher than normal nutritive flux across the basement membrane (2). There are valid reasons to indicate that this situation could result in a new steady state in which both the rate of proliferation of epidermal cells and total number of epidermal cells per unit area of skin surface would assume a value compatible with the hightened nutritive supply (2). Should the increased proliferative pressure in the epidermis lead to the formation of extensive interdigitations at the dermal-epidermal junction, once these are formed, a high level of mitotic activity ought to persist even if the barrier function of the skin were restored. This expectation is based on the likelihood—other things being equal—that the diffusional nutritive flux into the epidermis in this situation would still be a function of the higher than normal ratio of the area of the basement membrane to the area of the overlying skin surface.

Although initiation of hightened proliferative activity appears to involve other factors than its maintenance in the established lesion, both are subject to normalization by interference with the mitotic process. Thus, antimitotic agents arrest the spreading of the lesions in spite of high nutritive flux, as much as they diminish the proliferative activity in the lesions themselves. Conceivably, these agents normalize the diffusional nutritive flux across the basement membrane to the extent to which they are able to reduce the extensive dermal-epidermal junctional surface area. However, if upon the cessation of the medication the water barrier of the skin is still defective, or even further impaired, recurrence of the lesions or a flare-up of the disease may be expected.

Low caloric or otherwise deficient diets may also affect mitotic activity by interfering with the nutritive supply to the epidermis. This mild type of interference may diminish the large nutritive flux of psoriatic areas to a level which still surpasses that of normal skin, allowing the appearance of new lesions or the spreading of old ones to coexist with their diminished scaling activity. The distinction between spreading of the lesions and the extent of scaling

in the lesions themselves is thus in line both with observational evidence and a nutritive hypothesis formulated elsewhere (2).

The various formulations derived in this paper involve a number of stated and unstated assumptions. For instance, it is assumed that the rate of advance of the leading edge and the intensity of the psoriatic process, defined here in terms of scaling, are uniform in all lesoins. In fact, according to the formulation, older lesions and psoriatic areas newly arisen as a result of the advance of the leading edge, are regarded as exhibiting the same rate of scaling. Another feature of the derivation is that the narrow strip adjacent to the border of the lesion in which the essential transformation of normal to psoriatic skin occurs has been replaced by the concept of a linear leading edge. Redness and scaling are usually more intense in these areas than further on towards the center of the lesion, and it is due to this circumstance that small lesions which are almost entirely composed of this region appear more active than larger ones.

The description of the psoriatic state presented in this communication is not unique since it is based on three arbitrarily defined characteristics. In spite of this limitation, or perhaps because of it, the derivation proved of value by providing a conceptual framework through which the complex aspects of the disease could be viewed in a simple and schematic manner.

SUMMARY

An attempt is made to quantitatively describe psoriasis in terms of area, intensity of the process, and patient risk status. Conceptualization of the disease in terms of these variables delineates sharply between spreading of the lesions and their scaling activity and affords an insight into such obscure phenomena as flare-up occasionally observed upon termination of treatment or the coexistence of decreased scaling with enlargement of involved areas. In addition, a theoretical basis is given the impression that many small lesions present a greater threat to the patient than fewer lesions of comparable total surface area. Spreading of psoriasis and its regression is discussed in terms of nutritive hypothesis presented elsewhere. The ad hoc formulation demonstrates the feasibility and potential advantages of this approach.

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