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EDITORIAL

Markers of Skin Inflammation and Wound healing

In this issue several independent groups report their studies on inflammatory mediators and soluble tissue proteins in relation to the progression of skin inflammation and venous stasis ulceration, respectively. Reilly et al (p. 170) analyse the profile of certain cytokines and eicosanoids in suction blister fluid from normal, sensitive (post-eczema) and diseased skin (psoriasis and eczema) of altogether 30 patients and 28 healthy controls. They report that the prostaglandin E_2 concentration (expressed as pg/mg protein) was very closely related to the state of inflammation. Thus, compared to control skin the PGE_2 level was on average 3.8-fold higher in sensitive skin and 4.7-fold higher in diseased skin. Both these differences were statistically highly significant ($p < 0.0002$). Although leukotriene-B_4 and interleukin-1α levels were also markedly increased in diseased skin, no such increase was seen in sensitive skin suggesting that these two mediators affect inflammation at a later stage of the disease process than PGE_2. Provided that variations in the blister fluid protein concentration do not skew a comparison, the presented data might prove useful in a further delineation of the relative importance of different pro-inflammatory mediators as key regulators at various stages of development of skin irritation. The results also agree with previous clinical observations e.g. regarding the higher irritability of dry skin in atopic individual compared to normal healthy skin.

Conti & Seidenari (p. 192) pursue this matter by asking whether the higher skin irritability is related to atopy activity as such or to the coexistence of eczema. For this purpose the authors have investigated the response to irritant stimulation with sodium lauryl sulfate (SLS) in individuals with allergic rhinitis to pollen during pollen-free seasons and during pollen season, respectively. They monitored the trans-epidermal water loss, capacitance and echogenicity over 72 h following SLS exposure. They also made the same measurements in a group of individuals with atopic eczema who non-surprisingly showed a baseline difference compared to skin-healthy individuals and also an exaggerated response to SLS. However, rhinitis patients without skin complaints showed no abnormality at baseline and the skin irritability was not influenced by whether or not their pollen allergy was active or not.

In a paper by Peschen et al. (p. 162) the role of plasminogen activator (PA) is investigated at different stages of stasis-induced skin damage eventually leading to ulceration. Using in situ zymography on tissue sections the authors discriminated the urokinase- and tissue-types of PA activity from non-PA-dependent protease activity in skin biopsies. The biopsies were obtained from the lower leg of 37 carefully informed patients with stasis symptoms ranging in severity from telangiectasias, dermatitis and pigmentation to lipodermosclerosis and ulceration. Total PA activity was demonstrated in virtually all samples but the tissue-type of PA was significantly reduced in the patients with stasis dermatitis and lipodermosclerosis. In contrast, non-PA-dependent activity was absent in normal skin and in the earlier stages of stasis, but markedly increased in nearly all samples from ulcers. The authors conclude that an imbalance of the PA activity might be an important pathogenic factor for the development of venous leg ulcers.

It has also been proposed that extensive proteolytic degradation occur in chronic wounds. In a paper by Schmidtschen (p. 179) the aim was to identify marker molecules for healing activity of venous leg uclers. Selected proteins (inhibitors, matrix molecules, transport proteins, antibodies, complements, etc.) were identified by Western blotting in fluid sampled for 2 h by filter or plastic coverage of venous ulcer. In addition to previously described components, e.g. α_2-macroglobulin, α_1-antitrypsin and fibronectin, several hitherto unrecognized proteins in wound fluid, e.g. C3, inter-α-inhibitor, kinogen, tetranectin and immunoglobulins, were identified. Some of these proteins were to variable extents degraded in the wound fluid. Contact phase activation of proteases during sampling may thus be a compounding factor. In summary, this study forms a basis for future studies of the relationship between the protein level in wound fluid and the prognosis of ulcer healing using different types of therapeutic interventions.

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