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Increase of Endothelaemia in Psoriasis

PETR ARENBERGER[1], LUDMILA BUCHTOVÁ[1] and JOSEF HLADOVEC[2]

[1] Department of Dermatology and [2] Laboratory of Thrombosis, 4th Department of Medicine (JH), Charles University School of Medicine, Prague, Czechoslovakia

Changes of blood vessel endothelium in psoriasis have not been fully documented in the literature. The aim of our study was to ascertain whether in cases of such involvement an increase of endothelaemia could be identified. The counts of circulating endothelial cells were significantly increased in psoriatics compared with healthy individuals and patients with atopic eczema. The explanation of this observation may be based on the endothelial cell replacement stimulation in psoriasis. ***Key words: Blood; Endothelial cells.***

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P. Arenberger, Department of Dermatovenereology, Charles University School of Medicine, CS-100 34 Prague 10, Czechoslovakia.

In psoriasis abnormalities are present in the organs. Recently, we could show that urinary epithelium concentration is significantly higher in psoriatics compared with healthy individuals (1). We have here investigated if there is an increase of endothelial cells in the blood of patients with psoriasis.

METHODS AND PATIENTS

Methods

The method of a quantitative endothelaemia estimation has previously been described (2, 3). In principle, it is the counting of endothelial cells or, even better, of their carcasses in Bürker's chamber after their isolation together with platelets and the removal of the latter by an addition of adenosine-diphosphate. For this purpose, 5 ml of citrated blood is collected with siliconized material and centrifuged at 4°C for 20 min. One milliliter of the supernatant platelet-rich plasma is mixed with 0.2 ml of adenosine-5′-diphosphate sodium (Calbiochem) in concentration of 1 g/l and mechanically shaken for 10 min.

Platelet aggregates are removed by another centrifugation for 20 min. The supernatant is centrifuged for 20 min and the sediment is resuspended in 0.1 ml of 0.9% NaCl. From this suspension two chambers of Bürker's hematocytometer are filled, and the cells in the whole field of each chamber are counted under phase contrast. As the volume of one chamber is 0.9 µl and the material is concentrated 10 times, the results are expressed in terms of the cell number in 9 µl of platelet-rich plasma. Average counts from two chambers were used in the present experiments.

For exact characterization of endothelial cells, the supernatant was analyzed, after the platelet aggregates, had been removed by immunohistochemical methods according to Cordell et al. (4). The characterization of cells in cytospin preparations (Cytospin; Shandon-Elliot, Frankfurt, Germany) was carried out using the monoclonal antibody BMA 120 (Behring Werke, Marburg, Germany).

Patients

Blood samples were collected before the treatment was started and 5 months after it had been finished during the symptom-free period from patients suffering from generalized chronic plaque psoriasis affecting more than 60% of the body surface (11 males; mean age 38.2 years, range 18–79 years; 11 females; mean age 37.6 years, range 19–72 years) and compared with blood samples both from 22 healthy subjects (10 males and 12 females; mean age 41.4 years) and from 20 patients with untreated atopic eczema who showed both acute exudative and chronic lichenified lesions (10 males and 10 females; mean age 37.1 years). No significant difference was observed between the individuals of the three groups as far as the mean blood pressure.

Statistical analysis

The significance of the differences was estimated by Student's *t*-test.

RESULTS

Immunohistochemical analysis of cells obtained with the previously described method (2, 3) showed that over 98% of elements were endothelial cells reacting with the BMA 120 antibody.

There were 3.8 ± 0.42 endothelial cells in 9 µl of platelet-rich plasma in the control group. In untreated psoriatic patients, however, there were 5.01 ± 0.53 endothelial cells in the same plasma volume, which is significantly higher ($p < 0.05$) compared with healthy controls (Fig. 1). Psoriatics 5 months after finishing treatment (during the symptom-free period) showed a significant difference ($p < 0.01$) in the endothelial cell number (3.5 ± 0.29 cells) compared with the same group before starting the topical psoriasis treatment.

In order to test the specificity of the endothelaemia increase in psoriasis, we analyzed the endothelial cell number in 9 µl of platelet-rich plasma in patients with another common inflammatory and hyperproliferative dermatosis, atopic eczema. The cell number in blood samples from these patients was not statistically different from healthy controls (Fig. 1): 4.1 ± 0.36 cells ($p > 0.05$). The values were significantly different from those in psoriasis ($p < 0.05$).

No overlap of the endothelial cell number was observed

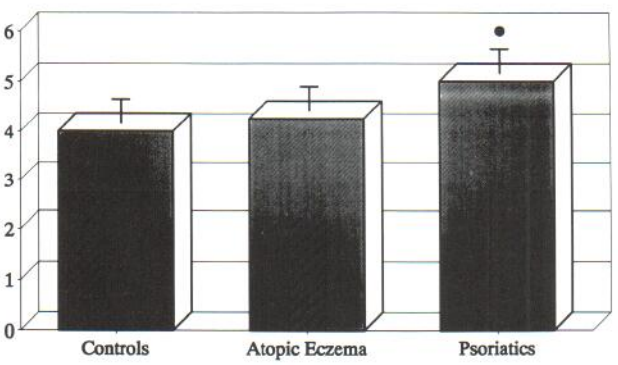

Fig. 1. Endothelial cell counts in 9 µl of platelet-rich plasma found in a group of healthy control subjects and a group of patients with atopic eczema and psoriasis. Values represent mean ± SD.
* $p < 0.05$ compared to healthy controls and patients with atopic eczema.

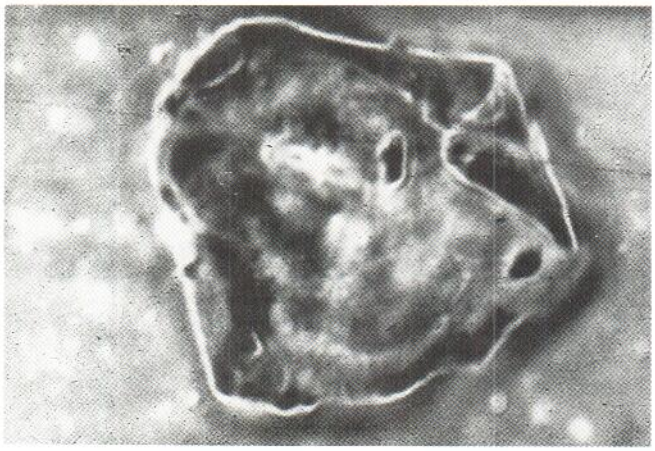

Fig. 2. Phase contrast appearance of a desquamated endothelial cell.

between psoriatics and the atopic eczema group or healthy subjects.

The results suggest that the endothelial cell number increase in blood samples of psoriatic patients is specific and not an epiphenomenon of inflammation present both in psoriasis and in atopic eczema.

DISCUSSION

The first author who attempted to estimate circulating endothelial cells intentionally and purposefully was Bouvier (5). He based his method on the assumption that the cells might be isolated with leukocytes. In 1973 a new method was developed based on the assumed existence in blood of low density anuclear carcasses of endothelial cells (Fig. 2) which can be isolated not with leukocytes but with platelets and can be concentrated and counted in a hematological chamber under a phase contrast microscope (2).

In all probability, the basal level of endothelaemia reflects a continuous replacement of cells. The decrease of the normal endothelaemia level which is observed in clinical transplantation cases (6) is caused by the administration of immunosuppressive drugs. Immunosuppression inhibits the physiological endothelial cell replacement and consequently decreases the circulating cell level. On the other hand, the hyperproliferation in psoriasis could stimulate the physiological cell replacement which we previously showed in an analogous experiment – we could demonstrate that the urinary epithelium concentration was significantly higher in psoriatics compared with healthy individuals (1). The higher replacement of blood vessel endothelial cells and urinary tract epithelial cells causes an increase of the circulating cell level in the blood stream or an increase of the urinary epithelium concentration, respectively.

Furthermore, other experimental findings seem to support the suggestion that psoriatic changes may develop not only in epidermis and mucous membranes – Priestley shows that dermal fibroblasts are hyperactive under culture conditions (7); Pokorny & Mandzárovová-Nohejlová describe psoriatic involvement of the small intestine (8).

An implication of our findings may be suggested. Endothelaemia might serve as an indicator of changes in the blood vessel endothelium in psoriatics, which could be supported by our findings that clinically healthy psoriasis patients after finishing their therapy showed a nonsignificant difference of endothelial cell number compared with the pretherapeutic stage. Family studies may lead to the discovery of a new method for latent psoriasis diagnostics which could be easier than the detection of 12(S)-hydroxyeicosatetraenoic acid receptor defect in keratinocytes (9).

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