USANA Technical Bulletin

Psoriasis

Description

- Psoriasis is a chronic disease of the skin. It is characterized by recurring remissions and exacerbations. Its lesions appear as erythematous papules (red elevated area on the skin).[1]
- About 2% of the U.S. population is affected. Men and women are affected equally and symptoms usually appear before 40 years of age.

Causes

- Certain individuals may have a genetic predisposition to develop psoriasis. The cause has been linked to certain histocompatibility antigens and as such may be an autoimmune process.[1]
- Flare-ups of psoriasis may occur unpredictably and may be associated with systemic (whole body) and environmental factors.

At Risk

- Those with a family history of psoriasis are more likely to get this disease.

Prevention and Management

- There is no known way to prevent psoriasis.
- High fiber diets may reduce circulating endotoxins. Elevated endotoxins are positively associated with psoriasis.[2]
- Fruits and vegetables (especially carrots) may alleviate psoriasis.[3]
- Low protein and low fat diets may help.
- One clinical observation is that psoriasis patients given a rice diet showed a dramatic reduction in or disappearance of their skin lesions.[4]
- Patients with psoriasis may have lower levels of vitamin A. Vitamin A may inhibit one of the rate limiting steps in the manifestation of the disease.[5]
- Low levels of folic acid are often found in psoriasis patients.[6]
- Oral calcitriol (1,25-dihydroxy vitamin D3) has been used successfully in the treatment of psoriasis.[7]
- Selenium levels may be low. Selenium is important for the activation of an inhibitor of the inflammation associated with psoriasis.[8]

Sources of Additional Information

- http://www.psoriasis.org/

Abstracts

Barna M, Bos JD, Kapsenberg ML, Snijdewint FG. Effect of calcitriol on the production of T-cell-derived cytokines in psoriasis. Br J Dermatol 1997 Apr;136(4):536-41. Although the use of vitamin D analogues in the treatment of psoriasis has been important new development, the mechanisms of action of these drugs are not fully understood. Psoriasis results from hyperproliferation of keratinocytes, and various studies attribute a crucial role to the locally infiltrating T lymphocytes. In an attempt to add to the understanding of the mechanisms of calcitriol therapy, we determined the effect of this drug on T cells by studying its effect on proliferation and on the production of various cytokines by T-cell clones prepared from psoriatic skin after non-specific activation with the combination of phytohaemagglutinin (PHA) and phorbol myristate acetate (PMA). the addition of increasing doses (10(-9)-10(-5) mol/l) of calcitriol to these T cells resulted in a dose-dependent inhibition in lymphocyte proliferation and in production of the type 1 cytokines IFN-gamma and IL-2, the type 2 cytokines IL-4 and IL-5. The general cytokines TNF-alpha and GM-CSF were not significantly inhibited. These data suggest that calcitriol is involved in the treatment of psoriasis via inhibition of the expansion, and cytokine production, of skin-infiltrating T lymphocytes.

Corrocher R, Ferrari S, de Gironcoli M, Bassi A, Olivieri O, Guarini P, Stanzial A, Barba AL. Gregolini L. Effect of fish oil supplementation on erythrocyte lipid pattern, malondialdehyde production and glutathione-peroxidase activity in psoriasis Clin Chim Acta 1989 Feb 15;179(2):121-31. Erythrocytes from psoriatic patients have a significant increase in polyunsaturated fatty acids (p less than 0.001) especially in arachidonic acid (p less than 0.001). Glutathione peroxidase activity, in both erythrocytes and platelets, was stimulated when compared with normal cells (p less than 0.001, less than 0.02, respectively) and the production of malondialdehyde was also increased in psoriasis (p less than 0.01). The level of plasma selenium was significantly reduced (52.80 vs 72.49 ng/ml; p less than 0.001). alpha-Tocopherol and retinol were both normal in plasma of psoriatics. After two months of fish oil supplementation, the erythrocyte lipid pattern was changed, eicosapentaenoic and dochesaenoic acids substituting the arachidonate in the membrane. A reduction in malondialdehyde (p less than 0.01), a prolongation of bleeding time (p less than 0.05) and a further stimulation of glutathione-peroxidase (p less than 0.001) in both erythrocytes and platelets was also found.

References

[1] Diseases. Springhouse (PA):Springhouse Publishing; 1993. p 1179.
[2] Rosenberg E. Microbial factors in psoriasis. Arch Dermatol 1982;188:1434-44.
[3] Naldi L, Parazzini F, Peli L, Chatenoud L, Cainelli T. Dietary factors and the risk of psoriasis. Results of an Italian case study. Br J Dermatol 1996 Jan;134(1):101-6.
[4] Newborg B. Disappearance of psoriatic lesions on the rice diet. N Carolina Med J 1986;47:253-55.
[5] Haddox M, Scott KF, Russell DH. Retinol inhibition of ornithine decarboxylase induction and GI progression in Chinese Hamster ovary cells. Cancer Res 1979 Dec;39:4930-38.
[6] Fry L et al. The mechanism of folate deficiency in psoriasis. Br J Dermatol 1971;84:539-44.
[7] Perez A, Raab R, Chen TC, Turner A, Holick MF. Safety and efficacy of oral calcitriol (1,25-dihydroxyvitamin D3) for the treatment of psoriasis. Br J Dermatol 1996 Jun;134(6):1070-8.
[8] White A et al. Role of lipoxgenase products in the pathogenesis and therapy of psoriasis and other dermatoses. Arch Dermatol 1983;119:541-7.