ABSTRACTS FROM CURRENT LITERATURE

Psoriasis: A possible reservoir for HPV-type 5, the virus associated with skin carcinoma of epidermodysplasia verruciformis. Eavre M, Orth G, Majewski S et al, J. Invest Dermatol 1998;4:110.

Epidermodysplasia verruciformis HPV causes widespread latent infection. However their role in skin carcinogenesis in non EV patients remains to be substantiated. This raises the question of the reservoir for HPV5. The authors aimed at finding out whether EV HPV was also present in benign epidermal hyperproliferation taking psoriasis as a model. Sera were obtained from 155 psoriatic patients with a mean age of 49.2+17.9% and sera were also obtained from 44 patients with atopic dermatitis, 14 with EV, 38 renal transplant recipients, 38 with C. acuminata, 60 with no known history of HPV infection. ELISA using purified HPV 5 L1 VLP preparation were used to detect antibodies to HPV serotypes in these patients. Scrapings were taken from single lesions of 37 psoriatic patients and from non-involved skin of 6 patients with plaque psoraisis. Similarly, scrapings were taken from non-involved and original skin of 19 patients with atopic dermatitis. Genotypes of HPV were identified by DNA sequence analysis of PCR amplification products. Seropositivity to HVP 5 was disclosed in 24.5% of other groups including atopic dermatitis. The detection rate was significantly higher for patients with lesions covering more than 50% of body surface. EV HPVS DNA sequence was disclosed in 91.9% of lesional skin and 5 of 6 from uninvolved skin showed the presence of HPV5. In contrast, rested PCR technique could not detect HPV 5 in any of the cases of atopic dermatitis. It was also found that there was frequent multiple HPV infection in psoriasis viz. HPV 36 in 84.2% compared to 22.6% in atopic dermatitis patients. Other types of HPV in psoriasis were HPV 20, 38 and two novel putative EV related genotypes. HPV 12 related RTR X7 and PSOX1 related to HPV 17 and the two novel putative isolated in atopic dermatitis wer AB X1 and AD X2. The presence of antibodies to HPV 5 viral particles and a very high prevalence of HPV 5 DNA in psoriatic epidermis strongly suggests that psoriasis constitutes a reservoir for HPV 5. HPV 5 capsid proteins could be one of the antigens of stratum corneum responsible for the oligoclonal T cell activation observed in psoriatic epidermis and could contribute to the autoimmune pathogenesis of psoriasis.

B. Deepa

Low-dose low-molecular-weight heparin (enoxaparin) is beneficial in lichen planus: a preliminary report. Hodak E, Yosipovitch G, David M, et al. J Am Acad Dermatol 1998;38: 564-568.

Evidence suggests that lichen planus is a T-cell mediated skin disorder and lymphocyte-keratinocyte interaction is the cause for destruction of the epidermis. The ability of activated T-lymphocytes to negotiate through vascular barriers, penetrate extracellular matrix, and migrate to target tissues is related to their expression of a

heparanase enzyme. This enzyme degrades the heparin-sulphate moiety of the proteoglycan of the extracellular matrix. Low dose heparin inhibits expression of T-lymphocyte heparanase and inhibits the production of the key pro-inflammatory cytokine, tumour necrosis factor-alpha. THe immunomodulatory molecules inheparin are the sulphated disaccharides which may not be present in all the batches of low molecular weight heparin preparations. Included in the study were 10 patients with widespread, histopathologically proven lichen planus associated with intense pruritus of several months duration and also refractory to teatment with topical steroids and H1 blockers. Patients were given 3mg enoxaparin, subcutaneously once weekly; three patients received four injections, and seven patients received six injections. In nine patients pruritus disappeared within 2 weeks. Within 4 to 10 weeks in eight of these patients there was complete regression of the eruption with residual postinflammatory hyperpigmentation. Of the four patients, who also had oral LP, only one showed improvement. No side effects were observed in any of the patients. These findings indicate that enoxaparin may be a simple, effective treatment for cutaneous LP.

Jyothy K

Use of topical cyclosporin in oral pemphigus. Gooptu C, Stanghton RCD. J Am Acad Dermatol 1998; 38:860-861.

The use of systemic cyclosporin in the treatment of pemphigus is well recognised. Short use of topical cyclosporin has been reported in a few cases of oral pemphigus. Oral lesions in pemphigus are common but can be very difficult to treat.

A sixty three year old woman with pemphigus vulgaris presented with chronic and painful ulceration of the oral mucosa for twenty three years. She also had perianal and vulval blistering, and lesions on the trunk. Direct and indirect immunofluorescence were both positive.

Treatment with prednisolone (60-10mg) controlled the cutaneous and genital lesions but failed to suppress her oral disease. Various agents like azathioprine, methotrexate, dapsone, chloroquine, intramuscular sodium aurothionate did not prove beneficial. Potent topical steroids were also ineffective. During this period she was maintained on 5-10mg/day of prednisolone.

Topical cyclosporin was started as a five minute mouthwash three times a day (5ml of 100mg/ml solution). Within six months ulcers improvd markedly and she could have a normal diet in twenty years. The frequence of mouthwashes was reduced to once a day on which she remained for five years during which her prednisolone was successfully discontinued.

Circulating IgG was undetectable three years after treatment. No side effects were encountered and blood cyclosporin levels were undetectable. The cyclosporin was subsequently withdrawn without relapse.

In this patient, topical cyclosporin was the first drug to produce a significant improvement in her oral ulceration. As her blood levels were undetectable, the drug is believed

to act locally, inhibiting T cell proliferation and release of lymphokines.

Thus cyclosporine was used not only to induce but also to maintain remission.

C. Lakshmi

Aquadynia-Noradrenergic pain induced by bathing and responsive to clonidine. Shelley WB, Shelley ED. J Am Acad Dermatol 1998:38:357-358.

The authors had first described both aquagenic urticaria and pruritus in which patients develop hives and itching from contact with water. They now extend the spectrum of water-induced skin reactions to include burning pain which they have termed aquadynia, which appears to be a form of noradrenergic pain that can be successfully treated with adrenergic receptor blockade. The authors describe two women in whom bathing was regularly followed by intense widespread burning pain that lasted 15 to 45 minutes. In both cases pain was not relieved by antihistamines. The first patient had a hemoglobin level of 19gm with a hematocrit of 57.6 and a red cell count of 6.82 million/cub mm. She was diagnosed to have polycythemia for which phlebotomies were done. Her blood cell count returned to normal but there was no relief of pain after bathing. Further studies showed elevated serum epinephrine, norepinephrine and catecholamine levels during her attacks of pain. She was treated with clonidine 0.1mg twice daily which gave complete relief. The patient was put on clonidine daily for three years and was asymptomatic. The second patient was put on long acting propranolol in a dose of 80mg daily which helped her significantly. In her, however, the catecholamine levels were not assayed. The pathogenesis of pain in aquadynia appears to involve the sympathetic nerves which can mediate pain, but the authors have no explanation for the mechanism. They believe that the pain is noradrenergic because both systemic clonidine which is an adrenergic inhibitor and propranolol which is a beta adrenergic blocker relieved pain. The authors also surmise that aquagenic pruritus may have a similar noradrenergic origin because this was found to respond to clonidine.

Bindu V

Superpotent topical corticosteroid use associated with adrenal suppression: clinical considerations. Gilbertson EO, Spellman MC, Piacquadio DJ et al. J Am Acad Dermatol 1998;38:318-321.

The super potent corticosteroids are clobetasol propionate, halobetasol propionate, augmented betamethasone dipropionate, augmented diflorasone diacetate and fluorandrenolide. They can cause HPA axis suppression and deaths from Addisonian crisis have been reported. In this article, two cases of profound HPA axis suppression due to inappropriate use of these agents are described.

A 36 - year- old woman with psoriasis for 13 years had recent weight gain, hypertension and Cushingoid features. She was using clobetasol propionate 0.05% ointment, 100g/week for 1 year. Her morning cortisol level was 1 μ g/100ml. Another 21- year- old male with

psoriasis for 8 years had weight gain, worsening of acne and Cushngoid features. He was using augmented betamethasone dipropionate 0.5% 80g/week. His morning cortisol level was 1 μg/100ml. Both patients were treated with oral prednisolone and tapered gradually.

Any patient with inflammatory dematosis with Cushingoid features should be questioned about the use of superpotent topical corticosteroids and investigated to rule out HPA axis suppression. The treatment consists of replacement by oral steroids with gradual tapering. The patients should be educated about the life threatenng consequences of the situation.

Anoop UC