Abstracts from current literature

Neuropeptides - Role in inflammatory skin diseases. Luger TA, Lotti T, J. Eur Acad Dermatol Venereol 1998; 10 : 207-211.

The cutaneous nervous system recently has been demonstrated to interact with multiple target cells in the skin and to mediate actions important in inflammatory conditions. Neuropeptides released by cutaneous neurons such as substance P (SP), vasointestinal peptide (VIP), Calcitonin gene regulated peptide (CGRP), propiomelanocortin (POML) peptides and others modulate the function of immunocompetent and inflammatory cells as well as epithelial and endothelial cells. They have been found to function as mediators of cell proliferation, cytokine and growth production as well as adhesion molecule and cell surface receptor expression. In addition many cells including keratinocytes, fibroblasts, endothelial cells and inflammatory cells have been shown to release several neuroptides and they express their corresponding receptors. Substance P enhances the acitivity of NK cells, stimulates production of IL-1, IL-6, TNFα, IL-2 and IFN and also activates TNFα gene expression in the mast cells. Receptors for substances P have been detected in normal as well as psoriatic skin and in lesional skin of atopic eczema, substance P levels were significantly decreased whereas VIP levels were increased. Neuropeptide Y like immunoreactivity was found to be expressed by langerhans cells in atopic lesions.. CGRP inhibits the antigen presenting function of langerhans cell, inhibits the induction of delayed type hepersensitivity. The POMC peptides regulates cytokine production, inhibits IgE production in higher doses, has a role in the pathogenesis of keloid formation. αMSH inhibits induction and elicitation of contact hypersensitivity, thus may be a significant regulatory mediator in cutaneous immune responses in vivo and may function as one of the signals responsible for anergy or tolerance induction. Since most of these neuropeptides down regulate the function of immunocompetent cells, overexpression may result in systemic immunosuppression may result in systemic immunosuppression. In the future, it probably will be possible to design therapeutic strategies with neuropeptides for various dermatoses.

Deepa B

Follicular mucinosis as a presenting sign of acute myeloblastic leukemia sammer TW, Grichnik MJ, Shea CR, et al. J Am Acad Dermatol 1998; 38: 803-805.

Follicular mucinosis is a nonspecific reaction pattern that occurs in a variety of clinical settings. It is an inflamatory disease of pilose baceous units characterised by accumulatin of follicular popules in plaques with scaling and loss of hair. It was first described by Pinkus as alopecia mucinosa but since alopecia is not evident clinically in areas other than scalop and bear, the term follicular mucinosis has been preferred. The following is the first reported case of follicular mucinosis as a presenting sign of acute myeloblastic leukemia in the absence of mycosis fungoides or leukemia cutis. A 60-year-old white man noted the acute onset of a pruritic papular skin eruption, generalised fatigue and occasional chills. Various topical steroids as well as brief courses of oral corticosteroids were tried. The patient noted

improvement but not complete clearing; new lesions continued to appear and the fatigue and pruritus continued. Four months after onset, an examination revealed numerous erythematous papules and nodules with excoriations on the posterior neck, anterior or central chest, shoulders, forearms and scalp. Many of these lesions were follicular. There was no lymphadenopathy or hepatoslenomegaly. The following investigations were done (1) skin biopsy which showed primary follicular mucinosis with associated perivascular and perilymphocytic infiltrate with marked involvement of follicular epithelium (2) immunophenotyping which revealed primarily a T-cell infiltrate comprising a mixture of CD4 + CD8 subsets 3) bone marrow biopsy and aspirate which were diagnostic of a refractory anemia with excess blasts in transformation. One month later the same investigations were repeated. There was no evidence of mycosis fungoides and the bone marrow biopsy confirmed acute myeloblastic leukemia. The patient was treated with doxorubicin and cytarabin resulting in clearance of his eruption. While the patient was in remission, he received consolidation therapy with cytosine arobinoside. The papular lesions continued to erupt intermittently until sixteen months after his diagnosis of acute myeloblastic leukemia. Now in the fifth year of follow up, both follicular mucinosis and leukemia remain in remission. Follicular mucinosis has been associated with neoplasms like mycosis fungoides, Hodgkin's disease, cutaneous B cell lymphoma, angiolymphoid hyperplasia, lupus erythematous. Good pasteur syndrome melanocytic nevi and lentigo maligna. Acute myeloblastic leukemia should be added to the list of lymphomas and inflammatory disorders associated with follicular mucinosis. The exact aetiology is not known, but immunopathological studies indicate that altered cell mediated mechanisms may play a role in its pathogenesis. Cell-mediated mechanisms can be profoundly altered in patients with leukemia and the cytokines released from T-lymphocytes may stimulate epithelium to secrete mucin.

Sunil Menon

Glycolic acid peeling in the treatment of acne. Atzori L, Brundu MA, Orru A et al. J Eur Acad Dermatol Venereol 1992;12:119-122.

An important objective of the treatment of acne is to induce rapid initial improvement so that the patient becomes confident in the therapy. To this purpose the authors propose treatment with glycolic acid, an alpha hydroxy acid in a concentration of 70%. The technique produces a partial, controlled cutaneous wound that induces first removal and then regeneration of part of the epidermis and/or dermis depending on the concentration of the acid and exposure time. The advantages of glycolic acid over some of the other peeling agents are its stability, the close correlation between application times and depth of peeling, and the fact that it is easily neutralised. Its therapeutic value in acne is mild epidermolysis, with dislodgement of comedones and unroofing of pustules that affect the follicular epithelium at the sebaceous gland level while excess keratinisation of the pilosebaceous duct is avoided.

Eighty females, aged 13-40 years were selected for the study. They were given a preparatory treatment with topica antibiotic and cosmetic formulations containing glycolic acid in concentrations varying from 8 to 15% at home twice daily. This preparatory treatment was given for two weeks, was continued during the study, except for 2 days before and after the peeling treatment with glycolic acid, during which period a specific moisturing cream and cleansing agent were suggested. The chemical peeling was performed with 70% glycolic acid solution applied for two to eight minutes. The number and frequency of application depend on the clinical response. The main clinical forms were comedonic acne in 32 cases, papulopustular forms with comedonic acne in 32 cases, papulopustular acne in 40 cases and nodular-cystic acne in the remaining eight cases. The most rapid improvement was in comedonic acne. In papulo-pustular forms an average of six applications were required. Although nodular - cystic forms required eight to ten applications, a significant improvement of the co-existing post-acne superficial scarring was noted. The procedure was well tolerated and patient compliance was excellent.

Bindu.V.

Treatment of granuloma annulare by local injections with low-dose recombinant human interferon gamma. Weiss JM, Muchenberger Silke. J Am Acad Dermatol 1998;39:117-119.

The authors describe three patients with granuloma annulare who were treated with intralesional rhu-IFN- γ injections, after informed consent, on seven consecutive days and thereafter 3 times / week for an additional 2 weeks. As a control, two similar GA lesions were treated by injection with solvent [0.09 % Normal saline] or by a corticosteroid under occlusion. Biopsy specimens were obtained before and at the end of the treatment. The follow-up period was 12 months. None of the patients has a history of other concomitant illness. All had previously been treated unsuccessfully with one or several modalities. Therapy was well tolerated by all patients. After 23 days a moderate inflammatory response was noted in GA lesions injected with rhu-IFN- γ but not in control lesions. The lesions began to involute after five to seven injections of rhu-IFN- γ and the control lesions remained unchanged. Specimens taken from the same lesions after rhu-IFN- γ treatment revealed a infiltrate. One month after the remission of the lesions treated with rhu-IFN-γ the control lesions were also treated with rhu-IFN-γ injections. In a follow-up period of 12 months all patients remained disease-free. Spontaneous involutions are a frequent observation in GA. The authors consider it unlikely that clearing resulted from spontaneous involution in their patients because the control lesions did not improve.

Smitha Prabhu S

Topical imiquimod therapy for chronic giant molluscum contagiosum in a patient with advanced immunodeficiency virus I disease. Buckley R, Smith K. Arch dermatol 135; 1167-1169.

A 32-year-old woman with HIV-1 infection had history of extensive facial lesions of molluscum contagiosum which were treated with multiple therapies with no improvement. She was no antiretroviral multidrug therapy for past 5 months and Bactrim DS and fluconazole daily for past 2 years. On examination there were papules and large nodules and plaques, predominantly on the face with areas of depressed scarring and post-inflammatory hyperpigmentation. She was started on imiquimod 5% cream, 25-mg single-use packets, 2.5 packets 3 times weekly for the first month, 2 packets for the second month and 1 packet for the third and last month. By the end of first month, 25-50% of her lesions had resolved, by the end of second month the lesions nearly resolved clinically with stable post-inflammatory hyperpigmentation. Fiver months after therapy genital lesions occur commonly in HIV patients, most respond to antiretroviral therapy. Here, the authors state that the unresponsive lesions were cured with imiquimod, an immunomodulator drug with no direct antriviral effects. The mechanism of action being mediated via production of pro inflammatory cytokines by stimulating the epidermal keratinocyes to produce increased levels of IL-6, IL-8, IFN-alpha and TNF-alpha, which in turn potentiate the effects of other cytokines. Because MCV is not capable of latency the clearing of lesions is long term.

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