Review Article

A Review Study- Dietary Management of Psoriasis

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Abstract

Psoriasis is a common, chronic, inflammatory scaly rashes associated with T-cell medicated immunological disorder. The cause of psoriasis is unknown but molecular mechanism suggest an immune disorder and the role of genetic and environmental influence. There are activation of T-cells that releases pro-inflammatory cytokines and causes an inflammatory reaction in the affected skin. The inflammatory nature of psoriasis itself is associated with an increased cardio-vascular morbidity. Modern trends of diet consisting high amounts of omega-6 fatty acid and low amounts of omega-3 fatty acid which is associated with a pro-inflammatory state. Dietary modification in the treatment of psoriasis may reduce the need of aggressive approaches and improves the cardio-vascular risk factors associated with psoriasis. This review summarizes the understanding of dietary modification in the management of psoriasis and to achieve a pathogenesis directed dietary approach in the treatment of psoriasis.

Introduction:

Psoriasis is a common, chronic skin disease associated with T-cell mediated immunological disorder with a high degree of morbidity affecting approximately 2% of the population.[1] Psoriasis most commonly affects the caucasians about 60 cases per 100,000 year in this population. Its prevalence in the US is 2-4 percent. In Asia it is common in Japan but much less common in China, about 0.3 percent. The incidence is lower in warmer, sunnier climates, most likely related to the beneficial effect of sunlight in latitude.[2]

The disease is characterized by circumscribed, raised, red, thickened plaques with overlying adherent silvery white scales. The morbidities associated with psoriasis is psychological impact of skin lesion, side effects of medication, reduced levels of employment with a decreased quality of life form disease itself and related complications. [3,4]

Etiology and Pathogenesis:

The primary cause of psoriasis remains unknown. It was considered a disorder of keratinocytes, recently it is recognized as a chronic inflammatory skin disease with a strong genetic basis and environmental influence, characterized by alteration in epidermal growth and differentiation and multiple biochemical, immunologic and vascular abnormalities. There are hyper proliferation of epidermis with premature maturation of keratinocytes with incomplete cornification and parakeratosis. The mitotic rate of basal keratinocyte of affected skin is increased. The epidermis is thickened. The inflammatory infiltrate in the dermis consists mainly of dendritic cells, macrophages, and T cells and neutrophils with T cells in the epidermis. The epidermis became thinned markedly and increased number of tortuous capillaries make the lesion erythematous.

About 30% of individuals with psoriasis have a family history of the disease in a first or second degree relatives. The risk of psoriasis in an offspring has been estimated to be 41 percent if both parent is affected, 14 percent if one parent is affected and 6 percent if one sibling is affected. Evidence suggests that UV exposure may be a major environmental factor interacting with genetic factors in psoriasis. HLA-Cw6 is a major determinant of disease expression with a tendency for early onset of skin lesions.

Skin has circulating T lymphocytes, antigen presenting cells, cytokine synthesizing kerationcytes, capillary endothelial cells, mast cells, tissue macrophages, granulocytes, fibroblasts, and non-Langerhans cells. The T cells are mainly of the memory phenotype and express the cutaneous lymphocytic antigen (CLA) which bind with E- selectin on skin capillaries. The cytokine profile of psoriasis is rich in interferon γ (IFN-γ). There is impaired inhibitory function and failure of regulatory T-cell to suppress effector T-cell proliferation.[5] Natural killer cells are present in psoriasis and are major producers of IFN-γ. These cells communicate by means of cytokine secretion and in response to antigen the primary cytokine released is tumor necrosis factor-alpha (TNF-α). The cytokine profile in psoriasis proposed a central role of proinflammatery cytokines, including TNF-α. The T cells in psoriasis

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predominantly secrete IFN-γ and interleukin-17, also interleukin-22 (IL-22)[6,7] IL-22 are involved in epithelial dysregulation and production of antimicrobial peptides human β defensin-2 (HBD-2), cathelicidin (LL-37) and chemokines.

In response to dendrite cell derived and T-cell derived cytokines keratinocytes produces proinflammatory cytokines (IL-1, IL-6, and TNF-α) and chemokines (IL-8).[8] Dendrite cells are antigen presenting cells that after recognization and capturation of antigen migrates to local lymph nodes and presents them to T cells. They induce autoproliferation of T cells and production of type 1 helper T cell (Th1) cytokines, TNF-α and nitric oxide synthase. The increased IL-2 from activated T cells and IL-12 from Langerhans cells ultimately regulate genes that code for IFN-γ, TNF-α and IL-2. These cytokins are responsible for differentiation, maturation, and proliferation of T cells into memory effector cells. The endothelial cells in psoriasis releases vascular endothelial growth factor (VEGF). Interleukin-8 released from keratinocytes, VEGF and angiopoietins are responsible for vascular changes in psoriasis.[9]

Management of Psoriasis:

Psoriasis is characterized by the presence of proinflammatory cytokines and activation of endothelium and there is associated co-morbidities including cardiovascular disease.[10,11,12] Inflammation is common in the aetio-pathogenesis of both psoriasis and cardiovascular disease. Evidence suggest the possible role of omega-3 fatty acid, folate and deficiency of vitamin B12. Severity of psoriasis are associated with high homcysteine and low folate levels. Increase utilization of folate with subsequent deficiency results from a rapid turnover of keratinocytes.[13] In psoriasis levels of free arachidonic acid, leukotriene-B4, 12-hydroxyeicosatetraenoic acid, and 15- hydroxyeicosatetraenoic acid, prostaglandin E and F2 are increased. Study suggest dietary supplementation of folic acid, B6, and B12 are reasonable in patent with psoriasis particularly those with elevated homocysteine, low folate levels.[14]

Dietary Modification:

Prostaglandin E2 (PGE2) derived from omega-6 fatty acid which can contribute to an inflammatory response. PGE2 are responsible for increased pain sensitivity, swelling and constriction of blood vessels. Excess consumption of omega-6 rich vegetable oil and arachidonic acid rich food (meat, eggs, dairy and dairy product) provides synthesis of PGE2.[15] Prostaglandin E3 (PGE3) improves the circulation and promotes anti-inflammatory response. Prostaglandn E3 derived from the omega-3 fatty acid, eicosapentaenoic acid (EPA). EPA is thought to act by competing with arachidonic acid for binding site on cyclooxygenase-2, producing a less potent inflammatory mediators and reduces inflammation.[16]

Processed food contain large amount of omega-6 rich vegetable oil and livestock, poultry and farmed fish are being fed cornmeal and soy-based feed, which raises the omega-6 content of the meat and fish. Modern trends of food raises the ratio of omega-6: omega-3 as high as 11:1. This dietary trends of eating high amounts of omega-6 fatty acid and low amount of fish (rich in omega-3 fatty acid) push the balance toward a pro-inflammatory state. Consumption of omega-3 EPA rich fatty fish can benefit the patient with psoriasis.

Leucotriene B5 (LTB-5), an omega-3 fatty acid derivatives reduces IL-1 and TNF-α, decreases chemo-attractant protein platelet derived growth factor (PDGF), reduces expression of adhesion molecule and increases the level of nitric oxide. The resultant effect is prevention of new blood vessel formation and improvement of circulation. Omega-3 fatty acid also act as immunomodulators by suppression of proinflammatory cytokine production.[17] Spices in the diet (turmeric, red pepper, cloves ginger, garlic) can prevent the activation of inflammatory cytokines.[18]

Vitamin D has antiproliferative effect on keratinocytes and keratinocytes convert 7-dehydrocholesterol to vitamin D3 in presnce of UVB. So, supplementation of vitamin-D is important in the dietary management of psoriasis.19,20. Milk whey (XP-828L) inhibits the production of IFN-γ and IL-2. It has a considerabhle immunoregulatory effect.[21]

Conclusions:

Psoriasis is an immune mediated disorder, characterized by inappropriate activation of T-cells that releases proinflammatory cytokines such as TNF-α, IL-2 and interferon (IFN- γ) and causes an inflammatory reaction in the affected skin. The cytokines leads to the recruitment of other immune cells and expression of VEGF, leading to vascular proliferation. The inflammatory nature of psoriasis itself is associated with an increased cardio-vascular morbidity.

There are many treatment options which are highly effective in psoriosis but there are considerable risk of long term use. The patho-physiology of psoriasis suggest dietary modification in the management of psoriasis which may be safe and

effective. Dietary modification would be extremely helpful and possible in treating psoriasis as well as may reduce cardio-vascular morbidity.

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