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Psoriasis: Role of dietary management in diminution of its symptoms

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ABSTRACT

Psoriasis is a common chronic skin disease mediated by T-cell with atypical proliferation of keratinocytes. The influence of environment and stress as a cause of psoriasis cannot be undermined. The recent research on diet in psoriasis as cause and cure has gained momentum with several studies correlating it with aetiology and pathogenesis of the disease. Various dietary intolerances have added to the cause and concern of the disease. The alcohol dependence of patients with psoriasis has led to its worsening. Trials on Gluten free diet (GFD) in psoriasis patients has established the reason for its avoidance in the diet, the association between celiac disease (CD) and psoriasis probably due to Th1 cytokines is highlighted but in absence of substantial literature cannot be established. Importance of several vitamins and their analogs has also been discussed with insight on vitamin D as a possible medicine in treatment of psoriasis. Likewise, the diet rich in eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) like fish oil was found to improve the clinical condition of patients. The significance of antioxidants and antioxidant therapy to remove the toxic waste and its positive impact on patient with psoriasis has been established.

KEY WORDS: DIET, DIETARY INTOLERANCE, KERATINOCYTE, PSORIASIS, T-CELL

INTRODUCTION

Psoriasis is common chronic, relapsing, disfiguring, inflammatory and proliferative skin disorder where keratinocytes divide and move more quickly from the stratum basale to the stratum corneum. Psoriasis usually varies in duration, periodicity of flares and extent. The interaction between multiple gene susceptibility loci, the immune system, and various environmental factors are held responsible for the pathogenesis of psoriasis. Lowes

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et al., (2008) have discussed the major role of T cells expressing cytokine interleukin 17 in psoriasis. Generally, it is most commonly understood as a T-cell-mediated disease involving interferon-g and tumor necrosis factor-alpha as key pro-inflammatory players. For many years, the impact of nutrition in the treatment of psoriasis has been widely studied. Nutritional aspect of psoriasis off late has been one of the main interests of researchers and the associated comorbid conditions has renewed their concern in nutrition as a mode to recover comorbid conditions as well to underlying skin disease (Ricketts et al. 2010). Araujo et al. 2009 and Wolters, 2005 suggested the importance of diet in the etiology and pathogenesis of psoriasis.

This review aims to highlight the constructive role of various essential dietary contents in diminution of the symptoms of psoriasis and also the adverse impact of dietary habits on severity of psoriasis. This review aims to highlight the constructive role of various essential dietary contents in diminution of the symptoms of psoriasis and also the adverse impact of dietary habits on severity of psoriasis

GLUTEN-FREE DIET IN PSORIASIS

Gluten is a protein commonly present in the cereal crop wheat, rye, barley, triticale, oats, spelt and kamut (Wolters, 2005). It is interesting to note that in gluten sensitive people, intake of gluten sources can lead to mucosal inflammation and villous atrophy with crypt hyperplasia. Baum et al. (2001) and Connon, (1999) have discussed the responsible toxic compounds i.e. gliadin in wheat gluten, secalins in rye, and hordeins in barley grouped under prolamins.

The mechanism of oral tolerance to dietary protein is basically governed by three major underlying principles:

1. Functional Unresponsiveness to the antigen
2. Apoptosis
3. Immune suppression by regulatory T cells

Action of regulatory T-cells varies according to the type of regulatory cells. Firstly, Natural Tregs those act in a contacted approach and express CD25 and transcriptional factor FOXP3. Secondly, Adaptive Treg Type 1 Cells (TR1) unlike Tregs, work in contact independent fashion and may or may not express CD25 AND FOXP3. TR1 and TH3 cells preferentially synthesize immunosuppressive cytokine IL-10 and TGF-B respectively to maintain homeostasis of responses to foreign antigens including gliadin (Vojdani et al. 2008).

In case of intolerance, luminal antigens finds response from gut associated lymphoid tissue that may lead to production of Immunoglobulin like IgA and IgM antibodies, pro-inflammatory cytokines and subsequently tissue damage or autoimmunity (Sollid, 2002). The type of hypersensitivity whether immediate or delayed to gluten is characterized by IgE mediated region or IgG, IgM, IgA plus T-cell reaction to gluten when tolerance to gluten is either not established properly or broken in these conditions (Maloy & Powrie, 2001; Bu et al. 2001 Sollid, 2002, Schwartz, 2003, Matsuo et al. 2005 and Knoechel et al. 2006).

It is believed that gluten sensitive intestinal disease is apparently marked with very few or no gastrointestinal symptoms and laten gluten sensitivity and psoriasis is associated (Duggan, 2004; Leffler et al., 2003; Michaelsson et al. 1993 and Nelson, 2002). Various studies have correlated celiac disease and psoriasis (Wolters, 2005 and Michaelsson et al. 2000).This correlation can be attributed to the fact that both conditions involve Th1 cytokines in the pathogenesis of the disease process. Interleukins, IL-1 and IL-8 released from rapidly dividing keratinocytes are thought to activate the Th1 inflammatory cascade (Ojetti and Aguilar, 2003).

Addolorato et al., 2003 in a case report stated about the significant improvement in patient with celiac disease and psoriasis immediately after starting gluten free diet (GFD) routine. Similarly, in another study reported by Michaelsson et al., 1993, GFD helped in recovering psoriasis with no CD but with IgA and /or IgG AGA. However; some authors Addolorato et al. 2003 and Collin & Reunal, 2003 deny any positive association due to limited literature. Bhatia et al., (2014), in their study concluded that gluten-free diet may potentially be beneficial in celiac antibody positive psoriasis patients, all though insisted on more well-powered studies to confirm this.

HISTAMINE INTOLERANCE

Histamine (2-[4-emidazolyl] ethylamine) was discovered in 1910 by Dale and Laidlaw and identified as a mediator of anaphylactic reactions in 1932 (Santos,1996, Steinhoff et al. 2004). Histamine is synthesized by pyridoxal phosphate (vitamin B6)-containing L-histidine decarboxylase (HDC) from amino acid histidine and are categorised under biogenic amines. Histamine is released by different human cells especially basophils, mast cells, platelets, histaminergic neurons, lymphocytes and enterochromaffin cells and is stored in vesicles or granules released on stimulations The disequilibrium of accrued histamine and capacity for histamine degradation has resulted into histamine intolerance. Various theories have been floored as mechanism leading to the histamine intolerance (Maintz & Novak, 2007). Possible theories for the underlying conditions are:

(a) Endogenous histamine overproduction caused by allergies, mastocytosis, bacteria, gastrointestinal bleeding,

(b) Increased exogenous ingestion of histidine or histamine by food or alcohol,

(c) Biogenic amines like putrescine may also be attributed with the role of displacing histamine from its mucosal mucine linkage, resulting in increase of free absorbable histamine in circulation.

(d) The most important theory credited with the cause of histamine intolerance is an impaired enzymatic histamine degradation caused by genetic or acquired impairment of the enzymatic function of DAO or HNMT.

Enzyme DAO is the main enzyme for metabolism of ingested histamine (Bieganski et al. 1980; Bieganski et al. 1980; Bieganski et al. 1983; Bieganski, 1983 and Sattler et al. 1988). A Reduced DAO activity may lead to impaired histamine degradation resulting in excess of histamine causing numerous symptoms mimicking an allergic reaction. Intake of histamine rich food (Wantke et al. 1994) alcohol (Wantke et al. 1996; Zimatkin & Anichtchik, 1999 and Sattler & Lorenz,1990) or drugs (Wantke et al. 2001; Sattler et al.,1987; Jarisch, 2004 and Jarisch & Wantke,1996) release histamine or block DAO may aggravate diarrhoea, headache, (Wohrl et al. 2004) congestion of the nose, asthmatic wheezing (Wantke et al.1994; Zimatkin & Anichtchik;1999 and Pollock et al. 1991) hypotension, arrhythmia, urticaria, psoriasis, pruritus, flushing, and other skin related conditions in these patients (Schmidt et al. 1990). Because of the multifaceted symptoms, the existence of histamine intolerance is frequently underestimated, or its symptoms are misinterpreted.

Psoriasis and alcohol consumption allocate multifaceted and multi-factorial relation. However, the relation between the alcohol consumption and the disease is not yet significantly clear, however; evidences do suggest psoriasis triggers under alcoholic influence (Ricketts et al. 2010; Jankovic et al. 2009; Kirby et al. 2008; Poikolainen et al. 1999 and Tobin & Kirby, 2009). Several authors (Jankovic et al. 2009 and Gupta et al. 1989) have suggested that men with family history of psoriasis are more susceptible to the skin disorder. Many researchers have established the positive correlation between the disease severity and alcohol abuse (Kirby et al. 2008; Poikolainen et al. 1999 and Tobin & Kirby, 2009), poor diagnosis in psoriasis and less effective treatment (Gupta et al. 1989 and Higgins & Vivier, 1994).

Different studies conducted worldwide have also agreed to the cause of associated hepatotoxicity in psoriasis medications that have been stated more frequently amongst the highly alcohol dependent patient (Gronhoj et al. 2000 and Montaudi et al. 2011). Effect of alcohol dependence on psoriasis patients has elevated alcoholic liver disease, (Tobin & Kirby, 2009) anxiety, depression (Kharaeva et al. 2009), cardiovascular disease (Stücker et al. 2001) and solid tumor risk (Richard et al. 2013). In order to understand whether modification of alcohol intake in patients with psoriasis affects disease course or not a systemic study is required.

VITAMINS IN PSORIASIS

Vitamin A

The effective role of various topical and systemic vitamin A derivatives has been established in the treatment of psoriasis. Retinoid receptors can be divided into retinoic acid receptors and retinoid X receptors, and each family has α, β, and γ subtypes.This receptor helps retinoid to inhibit the growth of hyperproliferative keratinocytes and induce their terminal differentiation. The second generation of aromatic retinoids, etretinate and acitretin, have been established as an effective systemic therapies of psoriasis and other keratinization disorders. Similarly, an increase in squamous cell carcinoma and keratoacanthoma was also reported by (Touraine et al. 1973) in extract of psoriatic lesion. Retinoic acid (RA) and its synthetic analogs, (Peck and DiGiovanna, 1994) have been successful in treating a number of epidermal disorders, including photo-damage, malignancies, acne, and psoriasis and other disorders of keratinisation.

With regard to serum vitamin A level in psoriatic patient conflicting research is reported. Serum vitamin A levels were reported to be decreased in patients with "common psoriasis" (Majewski et al. 1989), severe erythrodermic, and pustular psoriasis (Marrakchi et al. 1994) and in patients with both active and inactive psoriasis (Rocha et al. 2001). Rollman et al., 1985 and Safavi et al., 1992 confirmed no difference in levels of vitamin A in patients with and without psoriasis.

In psoriatic lesions; highly increased concentrations of endogenous retinoids have been implicated (Ricketts et al. 2010), established the increased action of a cytosolic enzyme catalyzing the formation of RA from retinol in extracts from psoriatic skin and Rollman & Vahlquist, (1985) reported a significant increase in 3, 4 didehydroretinol (a precursor of ddRA) in psoriatic lesions. Similarly, an increase in squamous cell carcinoma and keratoacanthoma was also reported in extract of psoriatic lesions (Reichrath et al. 2007 and Bos & Spuls, 2008).

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The epidermal changes in few benign dermatoses like psoriasis quietly resemble the features as observed in the deficiency of Vitamin A so a local abnormal retinoid metabolism has been incriminated in its pathogenesis (Richard et al. 2013).

Vitamin B9 (FOLIC ACID)

Person affected with psoriasis has an increased incidence of folic acid deficiency. This elevated deficiency can be attributed to the elevated homocysteine levels (Kural et al. 2003 and Malerba et al. 2006) decreased intestinal absorption caused by inflammation (Najarian & Gottlieb, 2003; Scarpa et al. 2000 and Schatterman et al. 1995) and increased use by skin epidermal cells (Malerba et al. 2006; Najarian & Gottlieb;2003 and Gisondi et al. 2007). Folate deficiency is also implicated in psoriasis severity. As suggested by Malerba et al. 2006 in a case control study, significantly increased level of plasma homocysteine and diminished plasma folate levels were established among patients with psoriasis compared to healthy controls. Malerba et al. (2006) compared plasma homocysteine levels and folate levels with scores of PASI and a direct correlation was established with plasma homocysteine levels whereas folate levels were inversely linked. Antithrombotic and Cardio protective role of folate supplementation in psoriatic patients has been assumed by Gisondi et al., 2007; however in absence of adequate evidence this claim cannot be supported.

Vitamin B12

The finding on role of Vitamin B12 in the treatment of this disease is still on a superficial stage. Baker and Comaish, 1962 and Ruedemann, 1962 have adjudged its efficacy in the treatment when levels of vitamin B12 in psoriatic plaques were low. Various studies have established the efficacy with intramuscular and systemic vitamin B12. The benefit in topical vitamin B12 was also demonstrated recently. Stucker et al. 2001 has categorically cited a prospective randomized clinical trial and assessed the effects of topical calcipotriol cream against vitamin B12 cream on a certain group of patients suffering with chronic plaque psoriasis. However the results were not so exciting for Vit.B12 when calculated on PASI score as the beneficial effects in the vitamin B12 group were slower to develop. Even though slower in chronic patients, but, still it can be researched for use in the early stages of the treatment.

Vitamin D

In treatment of psoriasis the effectiveness of topical Vitamin D is well known however the usefulness of oral supplementation of the vitamin is still ambiguous. This vitamin has multiple functions; Vitamin D locally functions as hormones by controlling calcium homeostasis as well have autocrine/paracrine influence on tissues that express CYP27B1 and VDR. Vitamin D3 (Calcitriol) exhibit immunomodulatory properties and through these properties restricts the T-cell proliferation and Th1 development, modulating antigen presenting cell (APCs) function, inducing hypo-responsiveness to antigens, inhibiting production of IL-2, IL-17, IL-8 and interferon, increasing the production of IL-10 and regulatory T cells (Arnson et al. 2007 and Adams & Hewison,2008).

It is involved in the regulation of antimicrobial peptides cathelicidin and human defensin 2 (HBD2), which both participate in the pathogenesis of psoriasis. Vitamin D's role in psoriasis is further supported by studies that confirm the link between VDR polymorphism and psoriasis. Although extra physiologic doses of oral vitamin D may have deleterious effects, supplementation of vitamin D in patients with insufficiency may have a role in psoriasis, still additional research and studies are needed on the vitamin D status in patients with psoriasis, (Okita et al. 2002, Dayangac et al. 2007 and Hollox et al. 2008).

POLYUNSATURATED FATTY ACIDS

Polyunsaturated fatty acids are differentiated into two different categories namely linoleic acid from n-6 fatty acid family and alpha-linoleic acid from n-3 fatty acid family on the basis of the first double bond counted from the methyl end. Sunflower seeds are rich source of linoleic acid and alpha-linolenic acid (C18:3n-3), eicosapentaenoic acid (EPA; C20:5n-3) and docosahexaenoic acid (DHA; C22:6n-3) are the most abundant n-3 fatty acids in food. Linseed and walnut oil are rich source of alpha-linolenic acid whereas oily fishes are rich source of EPA and DHA (Wolters, 2005).

PGE3 and LTB5 are EPA-derived eicosanoids with low inflammatory action as compared to PGE2 or LTB4, both formed from AA (Dayangac et al. 2007). Arachidonic acid (AA) has been found in high amount in psoriatic skin lesions and leukotriene B4, which is assumed to be a mediator of inflammation in psoriasis (Gupta et al. 1989).This is one of the main reason why eicosanoids from AA are thought to aggravate inflammatory responses whereas EPA derived eicosanoids show anti-inflammatory properties (Gil, 2002)Excessive production of these Arachidonic acid (AA) derivatives has been concerned with many inflammatory and autoimmune disorders and also in psoriatic skin lesions.

As discussed earlier that fish oil are excellent source of EPA and DHA , the administration of fish oil in patients with psoriasis leads to elevated plasma and platelet EPA-to-AA ratios and significant decrease in leukotrine B4synthesis by neutrophils is observed that has resulted in clinical improvement of psoriatic patients (Ricketts et al. 2010).

ANTIOXIDANTS IN PSORIASIS

Selenium

Selenium is an essential micronutrient with immune-modulating and anti-proliferative properties, which influences the immune response either by changing the expression of cytokines and respective receptors or by making immune cells more resistant to oxidative stress. Fairris et al. (1989); Roy et al. (1992), Spallholtz et al. (1990) and Matz et al. (2003) have reported the anti-oxidant, anti-inflammatory effects and UVA and UVB protective nature of selenium. Several studies and trials have been done to assess the correlation between selenium status and psoriasis.

Doses of Selenium have considerate inhibition effect on DNA synthesis and a stimulatory effect on cellular proliferation. Low Selenium levels in psoriatic patients were reported by some researchers (Serwin et al. 2003; Fairris et al. 1987; Matz et al. 2003; Fairris et al. 1989 and Serwin et al. 2002) and Harvima et al. 1993 in their study reported that alone the micronutrient cannot improve psoriasis. The significance of combination antioxidant therapy in severe erythrodermic or arthropathic psoriasis cannot be undermined. In the trial conducted selenium supplementation with coenzyme Q10 (ubiquinone acetate, 50 mg/d), and vitamin E (natural α-tocopherol, 50 mg/d) revealed quick clinical improvement in erythrodermic and arthropathic psoriatic patient. Kharaeva et al. (2009) in a statistical study demonstrated the improvements in measured clinical parameters in the arthropathic and erythrodermic psoriasis groups that received the antioxidants compared with the corresponding groups that received the soy lecithin placebo.

Zinc

Burrows et al. (1994) and Smith et al., (2009) reported the association of zinc deficiency with psoriatic plaque. There is little proof regarding the benefit of oral supplementation However; the approval regarding the dose or administrative ways has not been proposed yet. Still, the ways and advantages of zinc as an antioxidant in psoriasis are needed to be worked upon.

Taurine

Amino acid Taurine in early observation was assumed to be involved in the pathogenesis of psoriasis however number of studies failed to substantiate the assumption that taurine in any quantity can aggravate or improve the medical course of psoriasis. In a series of research conducted thereafter; various researchers have tried to establish the role of taurine in psoriasis. Roe (1962) in an earlier study claimed that 12 patients with chronic psoriasis treated with cholestyramine, a bile-acid sequestrant, all patients experienced clinical improvement and a simultaneous increase in fecal taurine content. This implies that exclusion of the amino acid might be related to clearing of psoriatic skin lesions. In another comparative study, later on by Roe (1965) concluded that high dose of taurine if administered to patient with psoriasis resulted in worsening of skin pruritus, erythema, and scaling within hours of ingestion. However, the same was absent in the patient without the disease.

The presence of taurine in our regular diet was also considered and various researchers tried to postulate whether the dietary intake of amino acid was involved in the pathogenesis of psoriasis. In another research performed on 15 patients with mild to severe psoriasis and low taurine diet was given resulting in complete healing of 9 psoriatic patients and partial cure in case of left over patient was achieved during 3-month period (Roe & Weston,1965). In yet another contrasting study performed by Zackheim and Farber, (1968) it was demonstrated that taurine dose in excess of the amount found in regular diet was given to 13 psoriatic patients but only a few experienced aggravation in the diseased condition.

CONCLUSION

Over the time diet has become an important factor in etiology and pathogenesis of psoriasis. Various systemic dietary intolerance has been responsible for the psoriasis and related skin disorders. Gluten intolerance has been increase in the population and subsequently Gluten free diet has been advised by number of researches. A possible correlation can be linked between psoriasis and celiac disease due to Th1 association. Even Gluten Free Diet helped in recovering psoriasis with no Celiac Disease but with IgA and /or IgG AGA. The disequilibrium of accrued histamine and capacity for histamine degradation has resulted into histamine intolerance. Intake of histamine rich food like alcohol can lead to Psoriasis trigger and has been positively and widely associated by number of researchers. Decrease Vitamin A and Vitamin B9 level has been reported and hence it can be suggested to enhance level of both the vitamins dietary intake. Likewise, Vitamin D can be useful in the treatment of Psoriasis but extra dose of Vitamin D should not be administered as can lead to worsening of the disease. Vegetable diet for minimum 3 months can be helpful to psoriasis patients due to low Arachidonic acid (AA) and Fish oil also seems to improve the clinical symptoms of Psoriasis. The positive role of antioxidants like selenium, zinc and taurine has also been established. However; a condensed research focussing more on pros and cons of dietary intake is needed so that a clear relationship between dietary habits can be established with etiology and pathogenesis of psoriasis.

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