Psoriasis and Its Correlation with Depression and Body Image: Case Report

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Abstract
Psoriasis is a skin ailment thought to be related to a suppressed immune system with its onset brought on about certain triggers including temperature fluctuations, infections, and stress. Psoriasis manifests as a skin rash but may also express systemic symptoms such as arthritis and nail disfigurement. As there is no cure for the disease treatment is usually found through light therapy, topical ointments, and certain immunosuppressive medications. There are five different variations of psoriasis with plaque psoriasis being the most common and highlighted in our case report. Psoriasis has a genetic component underlying its presentation with mutations along certain gene loci hypothesized to be causal factors but there is still much to be studied in terms of its genetic linkage and manifestation. Psoriasis causes an atypical growth of the epidermal skin layer due to the precocious development of keratinocytes expedited by the dermal inflammatory processes involving T cells, macrophages, and dendritic cells which induce cytokines to proliferate keratinocytes. The purpose of this case report is to look at the depression in our patient being linked to inflammatory processes and hypothesize this finding by analyzing the pathophysiological mechanism.

Background
Psoriasis impacts an individual’s physical, social and mental wellbeing. Psoriatic lesions depending on visibility, appearance and location can be stigmatizing to patients leading to low self-esteem and overt consciousness about their physical appearance and societal perception. Whether the inflammatory pathway linked to Psoriasis correlates with depression or the social ostracization of a sufferer, the resulting depression will be delineated during the course of this report. Our patient was exposed to an extreme stressor during his adolescence that marked the first occurrence of psoriatic plaques on his body. Over the course of the following years, he has had trouble dealing with his body image issues, the systemic symptoms, and adhering to the varying treatment modalities catered to his ailment that lead his symptoms of depression. This case report will address the vitality of the inflammatory pathways and associations between the cognitive and affective aspects of body image in relation with depression in patients with psoriasis.
while the dermatologist went through the treatment management plan with the patient there were social and personal issues that were unaddressed. The dermatologist stressed dietary, lifestyle, and topical hydrocortisone 1% management for the patient, however, the topical ointment and non-compliance with the proposed lifestyle changes led to worsening of the psoriatic rash. These physical symptoms further isolated him socially as his friends abandoned him and people stopped interacting with him in fear of a contagious disease. This led to the patient spiraling further more into a depressive state. This affected his participation in activities, education and social functioning as he regressed into more solitude. The patient’s mother noticed this decline and took him to a child psychiatrist where he was diagnosed with severe depression with body dysmorphic disorder secondary to his psoriasis. Extensive cognitive behavioral therapy was undertaken in adjunct with antidepressant therapy to manage his depressive state. Continued follow ups with his dermatologist resulted in various trials of pharmacotherapy which further aggravated the patient’s depression. Over the ensuing years the patient would cycle through phases of remission and remittance of both his psoriatic and depressive manifestations. He was finally managed with 10 mg IM Methotrexate for his psoriasis and a combination of bi-weekly CBT and Fluoxetine 20 mg daily to treat his depression. The purpose of this case report is to highlight the association of psoriasis with depression and ascertain to the degree at which an inflammatory condition can be attributable to their psychiatric representation.

**Past medical history:** None

**Past family history:** Father has eczema, diabetes, and hepatitis B. Grandfather had asthma. Patient has cousins who have eczema and bullous pemphigoid. Patient’s paternal uncles have diabetes. Patient mother has hypertension, eczema, and is a breast cancer survivor. Patient’s maternal side of the family has a history of coronary artery disease and a strong history of breast and ovarian cancer, with an aunt and cousin passing away from breast cancer.

**Past social history:** Patient drank alcohol and smoked marijuana experimentally in college and has a 15 year cigarette smoking history from the ages of 15 to 27.

**Differential Diagnosis**

1. Psoriasis
2. Eczema
3. Tinea Capitis
4. Atopic Dermatitis
5. Allergic Contact Dermatitis
6. Pityriasis Rosea
7. Seborrheic Dermatitis

**Treatment**

The patient on initial diagnosis for his psoriasis was prescribed 1% hydrocortisone cream to be applied topically and given instructions on how to avoid certain stress triggers and foods. However, the ointment did not help and the patient found it difficult to adhere to the strict lifestyle changes. The patient was able to improve with CBT and after years of trials with anti-depressants, the patient’s symptoms of depression were finally managed with Prozac/Fluoxetine 20 mg daily for his depression along with CBT catering to body image issues and group therapy. The doses of the hydrocortisone ointment had to be increased to 20% due to lack of improvement before the rash was finally managed at 20% topical hydrocortisone and Methotrexate 7.5 mg intramuscularly per month for 3 months. Two weeks post-IM Methotrexate, the patient’s rash disappeared, much to the relief of the patient and he felt better about his appearance and his mood improved significantly as a result. The patient is in currently in remission and is doing well after 6 years of the current regimen.

**Outcome and Follow Up**

While in remission, the patient is still in constant fear of having another outbreak of the rash. The patient now is more cognizant of the stressors but with the passing years fears of recurrent systemic complications stemming from his autoimmune disorder are a viable threat. The patient via CBT is now more aware and equipped to deal with this life-long condition via treatment and coping mechanisms. He is now active and deals with stress through yoga, golf, and meditation. Through the help and guidance of his psychiatrist, the patient has adhered to pharmacotherapy and CBT regime that has allowed him to be a more social and productive member of society. While the years of different medication treatments and their side effects coupled with the autoimmune disorder have taken a toll on the physical appearance of our patient in terms of hair loss, skin patches, nausea, and cyclical infections, our patient remains upbeat and hopeful about his progress moving forward. The patient is continuing his 10 mg intramuscular injections of MTX followed up with regular blood work to keep an eye out on side effects and 20 mg of fluoxetine daily with biweekly CBT sessions with a therapist that now serve as a maintenance therapy to continue helping the patient deal with body image and depressive symptoms that might come up and how he would deal with them.

**Discussion**

Psoriasis has been associated with the release of inflammatory cytokines IL-1β, TNF-alpha, IL-22, IL-6, IL-2, IFN gamma and its associated lesions. The release of cytokines IL-1,6, and TNF-alpha due to dendritic and T cell activity leads to inflammatory pathway initiation that further triggers inflammation with deficiencies in IL-10 and T cells allowing this pathway to occur. BDNF is another correlation between psoriasis and depression as it is reduced in both making the inflammatory pathway involving TNF-alpha, IFN-gamma, and other type-1 cytokines common to both disorders. With depression and its underlying inflammatory pathway we find the roles of IL-17, IL-21, and TGF-Beta are playing a role. Moreover with depression, increased levels of IP-10, CXCL10, anti-inflammatory cytokines, as well as changes in leukocyte mRNA expression, mRNA coding for cyclooxegenase-2, myeloperoxidase, inducible nitric oxide synthase and secretory phospholipase A2 were also found to be prevalent in depression patients. A direct response was found in the association of depression and proinflammatory cytokines such as TNF-alpha, IL-1B, IL-2, IL-6, prostaglandin E2, and CRP. This presence of inflammation in both psoriasis and depression marks the existence of a physiological relationship between the two diseases. Moreover, studies found that specific tandem repeat polymorphisms in intron 2 of the 5 HTR gene and serotonin receptors linked both depression and psoriatic lesions [1]. Direct causal relationships between depression and psoriasis were also observed in that the inflammatory cytokine release associated with psoriasis increased the activation of ideamine 2,3, dihydrogen, the enzyme responsible for converting tryptophan to kynurenine. Tryptophan being a precursor
to serotonin, and kynurenine productions decreasing serotonin concentration, coupled with the metabolism of tryptophan, and would lead to depression. Melatonin also served as a link between depression and psoriasis in that depression disrupts the release of melatonin, which regulates the immune system and helps reduce inflammation by decreasing TNF-alpha, IL-6, and IL-8 concentrations. Melatonin is also noted to be decreased in psoriasis with lowered melanin levels further expounding the symptoms of psoriasis. Treating depression has shown to restore melatonin production and decreases psoriatic symptoms. Another reason why phototherapy is useful for psoriatic patients in that it regulates melanin levels and therefore decreases psoriatic complications and helps lessen depressive feelings. Chronic inflammatory skin conditions such as psoriasis carry an increased risk of being a massive psychological burden with skin inflammation leading to a chronic inflammatory feedback involving cytokines that effects the CNS which perceives this additive stress and leads to a leaky blood brain barrier, increased oxidative stress, a decrease in neurogenesis, a derailment of the HPA axis, and microglial activation [2]. Moreover the deleterious effects of psoriasis on a person’s body image cannot be discounted in terms of their association to the depressive feelings patients feel and distortion of body image is correlated with self-reported depressive symptoms in psoriasis patients. It was established that in the case of both women and men there was a correlation between lowered mood and psoriasis severity (R=0.416), as well as body image (R= –0.282). In relation to individual scales, there was a statistically significant relationship between self-reported depressive symptoms and appearance evaluation (R= –0.519), health evaluation (R= –0.585), satisfaction with body parts (R= –0.462), as well as appearance orientation (R=0.425) [3]. Studies have highlighted the importance of stigmatization in depression and have shown that overcoming the stigma of psoriasis via coping mechanisms is important for a patient’s mental wellbeing [4]. Also a study showed that psoriasis patients are at least one and a half times more likely to manifest signs of clinical depression compared with their healthy peers. More than one-quarter of psoriasis patients show symptoms of depression and approximately one-tenth have signs of clinical depression [5]. Psoriasis has been shown to be stigmatizing and with societal misconceptions about psoriasis being infectious and contagious still prevalent, its now more than ever that we cater to this ostracization to help patients cope with skewed perceptions of themselves mirrored through the public’s maladaptive assumptions about their appearance and help with their self worth and negative body image and associated depression. It is important to take steps to reduce stigma about psoriasis by familiarizing the public with psoriasis as a non-infectious auto-inflammatory condition. This case study show cases a dual effect played by inflammatory sequences and environmental factors contributing to an individual’s depression.

**References**