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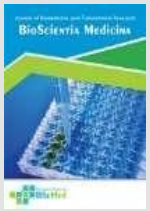

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The Role of Psychological Stress in Psoriasis: A Narrative Literature Review

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A B S T R A C T

Psoriasis is a chronic, persistent skin disease characterized by characteristic lesions in the form of well-defined erythematous patches covered by thick white, shiny scales resembling wax droplets. Psoriasis has a psychological, social, and emotional impact. Psychological stress has also been shown to influence the disease and course of psoriasis. Therapy in psoriasis patients with psychological stress can be given pharmacologically and psychologically. Patients with moderate to severe psoriasis using the Goeckerman regimen for psoriasis showed significant improvements in anxiety and depression scores. Psychological therapies such as relaxation therapy and meditation are able to control the emotions that trigger stress and reduce the appearance and severity of psoriasis. This literature review aims to describe the impact of psychological stress on psoriasis patients so as to increase awareness to detect psychological stress and provide appropriate treatment.

1. Introduction

Psoriasis is a chronic, persistent skin disease characterized by a characteristic lesion in the form of well-defined erythematous patches covered by thick white, shiny scales resembling wax droplets.[1,2] This disorder is generally influenced by the mental state of the sufferer. Psychological stress has been shown to influence the disease and course of psoriasis.[2] Psychiatric disorders due to psoriasis are more common in women. Patients generally suffer from severe psoriasis disorder, decreased quality of life, and positive family history of psoriasis.[3,4] The prevalence of psoriasis in Asia is 0.29-1.18% in Japan and 0.2-1.5% in China.[5] Previous studies have stated that stress plays a role as the most common precipitating factor in psoriasis in as many as 60% of patients.[3,5] Psychiatric symptoms are often found in psoriasis patients in the form of depression and anxiety. Anxiety and depression disorders are generally experienced by patients who have maladaptive personalities such as insecurity, feelings of imperfection, guilty feelings, and feelings of isolation from their surroundings. This literature review aims to describe the impact of psychological stress on psoriasis patients so as to increase awareness to detect psychological stress and provide appropriate treatment.

Definition of psoriasis

Psoriasis is a recurrent disorder of the skin in the form of well-defined erythematous patches with thick white scales and is mostly triggered by trauma to the skin, medication, and infection.[1] Psychiatric disorder or stress is thought to play a role in causing acute exacerbations of this disease. A previous study stated that patients with acute guttate psoriasis experienced psychological trauma 6 months before the diagnosis was made.[3] In another prospective study of 132 psoriasis patients who had recovered for three years, 39% of patients experienced stress. The incubation time from stressful events to psoriasis exacerbations is 2 days to 1 month.[6]

The association of psychological stress with psoriasis

Psychiatric symptoms that occur in psoriasis patients are associated with activation of the hypothalamus-pituitary-adrenal axis (HPA axis).[7] The hypothalamus secretes corticotropin-releasing hormone (CRH) and arginine vasopressin (AVP) into the pituitary portal system. The corticotropin-releasing hormone causes the release of peptides downstream from the pituitary, including adrenocorticotropic hormone (ACTH). Adrenocorticotropic hormone is secreted into the blood and results in the release of glucocorticoids (GC). The corticotropin-releasing hormone stimulates noradrenergic neurons in the central nervous system causing the secretion of norepinephrine (NE) by peripheral sympathetic nerves and NE and epinephrine by the adrenal medulla. CRH can also come from extra-neuronal sources such as keratinocytes, melanocytes, fibroblasts, sebocytes, mast cells, and leukocytes that directly activate skin cells, indicating a functional peripheral HPA axis operating in the skin.[8]

CRH-R1 and CRH-R2 receptors are expressed in the epidermal compartment, and CRH-R2 in the dermal compartment, and both receptors are expressed in skin adnexal structures and mast cells located in the skin.[8] CRH system plays a central role in the skin's stress response and is a component of the skin's immune system. The CRH system activates skin mast cells through a CRH-R1-dependent mechanism and in areas of skin with psoriasis lesions increase, the expression of CRH, sensory nerve tissue, and an increase in the number of mast cells then respond to stress on the skin and the occurrence of an inflammatory response.[9] Increased mast cell degranulation through activation of CRH receptors by urocortin in the skin so that mast cells are located close to nerve endings which can react to stress caused by increased CRH levels by degranulation and then provide a local stress response to the nervous system.[10] Different types of immune cells can be involved in this process and respond to hormonal activation and the release of hormones associated with central nervous system activation, such as catecholamines and GC.[10]

Peripheral CRH, ACTH, GC, NE, and epinephrine levels fluctuate throughout the day, influenced by various daily stressors.[11] Increased circulating levels of glucocorticoids and catecholamines act as messengers to the immune system, suppressing the production of antigen-presenting cells of interleukin (IL-12), which functions to induce a Th1-mediated response through the production of interferon-γ (IFN-γ) and tumor necrosis factor (TNF)-α.[12]

Stress has been shown to reduce adaptive immune function and innate natural killer (NK) cell activity. Activation of the HPA axis and automatic nervous system (ANS) in chronic stress leads to a constant upregulation of the pro-inflammatory cytokine profile. Repeated episodes of chronic stress can lead to altered physiological stress response systems and chronic elevations of pro-inflammatory mediators, including IL-1, IL-6, and TNF-α. These cytokines are implicated in major depressive disorder and a number of skin disorders.[13] Patients with psoriasis, especially those with stress exacerbations, exhibit an altered HPA response to social stress.[14]

Dysregulation in the HPA axis and skin cells may influence the production of pro-inflammatory cytokines during stress.[15] Molecular stress mediators and their regulation at the systemic and cutaneous

levels are needed to assess and validate the pathophysiological abnormalities of acute and chronic stress in psoriasis. Neuropeptides such as vasoactive intestinal peptide (VIP) and pituitary adenylate cyclase-activating peptide (PACAP). Both neuropeptides are released from the sensory system but also autonomic neurons, and PACAP receptors are expressed on endothelial cells, keratinocytes, and immune cells, including mast cells and T cells in human skin.[16] In PACAP, the secreted triggers inflammatory vasodilatation, and studies in mice have shown that it may sustain the acute stress response with HPA activation with subsequent release of CRH and corticosterone.[16] Increased PACAP mRNA expression in psoriatic lesions reinforces the hypothesis that neuropeptides may be involved in psoriasis pathophysiology and involvement in the dysregulation process.

Neuropeptide calcitonin gene-related peptide (CGRP) is expressed in the skin and the central nervous system by deltas C and A of the peripheral nervous system. CGRP mediates pain and the proliferation of Schwann cells, endothelial cells, and keratinocytes and acts as a vasodilator. The CGRP peptide was found to be elevated in plasma levels in psoriasis patients when compared to healthy subjects.[17] In psoriasis, patients with high-stress levels were reported to show higher CGRP expression compared to psoriasis patients with low-stress levels. In CGRP with neuronal disorders or psychiatric disorders such as migraine, stress, and major depression can generate CGRP signals that can be the basis for mental health problems in psoriasis patients.[17]

Immune response in psoriasis

In the skin of psoriasis patients, T helper cells (Th1) are activated by the antigen and then secrete IFNγ, which stimulates local antigen-presenting cells to release IL-1 and IL-23, both promoting expansion and IL-17 expressing CD4+ and CD8+ T cells.[18] The maturation of T cells to Th17 cells is mediated by IL-6 and TGF-β, which are derived from regulatory T cells (Treg) and keratinocytes. In one study, the Th17 maturation process in stressed nerves could influence the psoriasis mechanism.[19] Another study noted a marked increase in plasma IL-6 in the stress-exposed psoriasis group.[20]

The increase in IL-6 by external psychogenic stressors may influence the maturation and expansion of Th17 that triggers psoriasis, the pathological expansion of Th17 cells dependent on IL-23. IL-23 expression by dendritic cells is induced by thymic stromal lymphopoietin (TSLP), a proallergic cytokine expressed by keratinocytes and found to be elevated in psoriatic lesions. Keratinocytes respond to cytokines by increasing inflammation mRNA that has the ability to provide immunity in the skin and activate T cells. As many as 75% of dermal dendritic cells in contact with nociceptive neurons pharmacologically ablate and then result in the failure of DCs to produce IL23. Ablation results in a reduction in IL-23-dependent inflammatory cells in the skin.[19,20]

Other cytokines involved in the pathogenesis of psoriasis are IL-13 and IL-1, members of the subfamily IL-1, IL-33, and IL-36. IL-13 synergizes with IL-22, IL-17, and IFN-γ to promote the expression of chemokines that promote the entry of monocytes and DCs into the skin and have been identified as a risk for psoriasis.[21] Cytokines IL-17, IL-23, IL-1, and IL-36γ as the dominant cytokines in psoriasis, especially in generalized pustular psoriasis.[22] IL-17 induces IL-36γ and synergizes with IL-36γ to differentiate keratinocytes, whereas IL-33 expression is increased in psoriatic lesions in experimental mice with psoriasis-like reactions.[23,24]

Until now, the main cytokines that induce stress in psoriasis are unknown, such as IL-17 or IL-23, IL-1, and IL-36. One study reported an increase in T cells and CD3+ in psoriasis patients who were exposed to psychological stress. The role of NKT cells in psoriasis is not fully understood but contributes to cytokines influencing keratinocyte function, with keratinocytes acting as activators and recruiters of immune cells.[25]

Neuropeptide pathogenesis of psoriasis

Skin sensory afferent nerve fibers found in the skin function as transmitters of sensations of pain and

itching in the central nervous system as well as regulating local inflammation by releasing neuropeptides such as nerve growth factor (NGF), substance P (SP), and calcitonin gene-related peptides (CGRP). Neuropeptides influence the immune response and skin defense mechanisms and initially support the acute inflammatory process. Neuropeptides act as signaling molecules that regulate the function of skin cells and immune cells.[26] Mast cells are neuropeptide targets and an important part of neuroimmune communication. The release of neuropeptides from mast cells is an early part of the neurogenic inflammatory process of psoriasis and is one in which more neurons and immune cells are locally involved.

The important role of neuropeptides in the formation of psoriasis can be anticipated from the topical administration of capsaicin. Capsaicin can trigger an inflammatory cascade that causes pain and erythema by releasing pro-inflammatory and vasodilating mediators from nerve endings. The flow of released neuropeptides decreases over time because production cannot compensate for the ongoing neuropeptide loss. Chronic or long-term application of capsaicin can render the nerves in the treated area insensitive to further stimulation due to thinning. The topical application of capsaicin can be a significant therapeutic effect for psoriasis, and the topical application causes a reduction in scaling, erythema, and itching in patients with psoriasis.[25]

Koebner phenomenon is the appearance of psoriatic lesions on the skin of normal psoriasis patients as a result of trauma.[25] Initial trauma causes the proliferation of keratinocytes, fibroblasts, blood vessels, and nerves, as well as the accumulation of inflammatory cells into the skin. The importance of NGF in the development of psoriatic lesions depends on NGF being expressed in increased levels of lesional and non-lesional psoriasis skin, whereas the NGF TrkA receptor was found to be elevated only in psoriatic lesions. NGF inhibition improves disease phenotype in psoriasis rat experimental animals.[27]

Neuropeptides involved in the pathogenesis of psoriasis are regulated by NGF, e.g., calcitonin gene-related peptide (CGRP) and substance P (SP) by mature sensory neurons under NGF control.[27] Other neuropeptides with potential implications in psoriasis development are the tachykinin and SP families. In SP, it is localized at the dermo-epidermal junction of the skin and mediated neutrophil chemotaxis. In rats, it has been shown to stimulate keratinocyte proliferation and degranulation of dermal mast cells. SP stimulates T cell activation in T lymphocytes and mitogenicity of connective tissue and epithelial cells. The immune regulatory functions of the SP, in particular the activation of T cells, mast cells, and macrophages, may contribute to immune imbalance in the early stages of psoriasis.[25]

SP expression was increased in lesional and non-lesional skin with higher SP+/CGRP+ nerve fiber density compared to healthy skin and the most abundant neuropeptide in adult lesions. SP+ nerves have more contact with mast cells than fibers containing VIP+ or CGRP+. In SP and cognitive receptors, neurokinin/tachykinin 1 receptors may be increased in expression in peripheral blood cells containing eosinophils in psoriasis patients.[25]

Due to their role in stress, vasoactive intestinal peptide (VIP) and pituitary adenylate cyclase-activating polypeptide (PACAP) may also play a role in the pathogenesis of psoriasis. In VIP, it is localized in blood vessels, sweat glands, and skin nerve endings, signaling via the classic adenylate cyclase pathway. Plasma VIP increases in psoriasis patients and then decreases with the treatment of the disease. VIP's vascular growth function is dependent on the regulation of vascular endothelial growth factor (VEGF), whereas neurogenic inflammation is driven by VIP-dependent release of IL-6, IL-8, and CCL-5 (formerly known as RANTES).[28]

Clinical manifestations of psychiatric disorders in psoriasis

Psychiatric disorders are common comorbidity among patients with psoriasis. Generally, the symptoms shown are alexithymia, which is difficulty recognizing or expressing emotions. These symptoms

are associated with the early onset of psoriasis and increased susceptibility to stress. The stigma associated with skin lesions and unpredictable exacerbations of the disease causes a significant psychological burden. Patients report symptoms of anxiety and depression as an adjustment to their illness and subsequently develop clinically defined psychiatric disorders such as major depressive disorder, persistent depressive disorder, anxiety disorder, and substance abuse disorder. An immature defense mechanism is present in some patients with psoriasis.[6,14] Previous studies have reported an association between psoriasis severity and the risk of depression. This suggests that patients with psoriasis are at a greater risk of depression, regardless of the severity of the disease. Suicidal ideation is common in patients with psoriasis. Research shows that patients with psoriasis are more likely to have ideas about death and commit suicide than those without psoriasis.[6]

Another study discussed the relationship between excessive alcohol consumption and psychological distress in psoriasis patients. Excessive alcohol intake is a risk factor for the development of psoriasis and is known to be associated with a high incidence of anxiety and depression. Routine screening can be a means of identifying patients who abuse alcohol.[29] The visibility of lesions affects body image. More than 40% of psoriasis cases occur before the age of 30 years which causes stigmatization and has a negative impact on psychological function. Stigma is mostly associated with changes in the patient's skin. The patient's hypersensitivity and anticipation of rejection experienced with important and progressive social isolation can lead to decreased self-esteem, feelings of inferiority, and self-confidence.[6]

Psoriasis has consequences in partner relationships due to emotional disturbances suffered by patients, such as feelings of low self-esteem and limitations in activities. In one study, it has been reported that there is a decrease in quality of life associated with limitations in social activities and daily activities such as work and time with family. This requires not only a treatment strategy for the patient but also for the patient's family. Based on the socioeconomic field, it has been observed that psoriasis patients report difficulties in maintaining or getting a job due to their low productivity and lost working days due to exacerbation of the disease and may be financially constrained with low adherence to medication.[30]

Psoriasis also has an impact on decision making, where as many as 66% of patients reported that the disease affects career choices, 58% affects work choices, 52% affects choices in personal relationships, 44% in academics, 22% in decisions to have children and 20% affect on early retirement due to illness. These decisions mostly occur in adolescence and early adult life, so decision-making may be strongly influenced by high rates of disability. One cohort study reported that disease perception and patient strategies might have an impact on the clinical evolution of psoriasis. The highest perception of disease control and adequate emotional expression showed better outcomes when compared to passive and avoidant patients, such as denial, hopelessness, and also impotence, has been associated with higher symptoms and emotional disturbances.[6]

Diagnosis

Several instruments have been developed to evaluate the general quality of life of psoriasis patients. This instrument consists of assessing the patient's perspective on health status, assessing function objectively, subjective health, or both.[31]

Psoriasis area and severity index (PASI)

Psoriasis area and severity index is a tool to measure the severity of psoriasis assessed by doctors at four locations, namely the head, upper limbs, trunk, and lower limbs.[1] At each site, a score was assigned, which was calculated based on the severity of psoriasis and the surface area involved. The severity score consists of erythema, induration (thickness), desquamation (scaling) which is graded on a scale from 0 (none) to 4 (very severe), and the sum of the severity scores from 0 to 12. Surface area scores range

from 0 (0%) to 6 (90–100%) where 0 (0%), 1 = 1–9%, 2 = 10–29%, 3 = 30-49%, 4 = 50–69 %, 5 = 70–89% and 6 = 90–100%. Each of the four body location scores was calculated as the total severity score and then multiplied by the surface area score with a probability range of 0 to 72. Each location score was then multiplied by the specific correction score i.e., head = 0.1; upper limb = 0.2; body = 0.3; lower limbs = 0.4 with the adjusted scores added up to produce a total score ranging from 0 to 72.

The PASI score based on Fredricksson and Pettersson is categorized into three, namely the patient is declared to have mild psoriasis if the PASI score is less than < 7, moderate psoriasis if the score is PASI 7 to 12, and severe psoriasis if the PASI score is more than > 12. The PASI score is a scoring system used for research purposes. In clinical trials, the percentage change in PASI can be used as an endpoint for assessing psoriasis therapy.[31] (Table 1).

PASI scores were negatively correlated with health satisfaction, and higher PASI scores were associated with lower health satisfaction (r = -0.32). The higher the PASI score, the stronger the influence of the disease on the choice of food, drink, clothing, make-up, and hairstyle (r = 0.57). The higher the PASI score, the negative effect on social behavior (r = 0.46) and a significant effect on the psychological aspects of the quality of life of psoriasis patients (r = 0.41). Significant positive correlations were shown between PASI scores and perceptions of disease-related consequences (r = 0.30), symptom perception (r = 0.42), concern about disease (r = 0.31) and emotional representation (r = 0.35). Higher PASI scores correlated with stronger anxiety and depression (r = 0.33, r = 0.35, r = 0.35).[32]

Table 1. PASI Score.

Characteristics	Lesions score	Head	Upper Extremities	Chest	Lower Extremities
Erythema	0 = None 1 = Slight 2 = Moderate 3 = Severe 4 = Very Severe				
Induration/thickness					
Desquamation					
Sum up each of the 3 scores for each body part resulting in a separate amount					
Total lesion score (A)					
Percentage of affected areas	Score area	Head	Upper Extremities	Chest	Lower Extremities
Score Area B Degree of involvement as a percentage for each area of the body affected (score each area with a score between 0-6)	0 = 0% 1 = 1% – 9% 2 = 10% – 29% 3 = 30% – 49% 4 = 50% - 69 % 5 = 70% - 89% 6 = 90% - 100%				
Multiply the total lesion score (A) by the area score (B), for each body region to produce 4 individual subtotals (C)					
Subtotals (C)					
Multiply each subtotal (C) by the number of body surface areas, namely: for head x 0.1, for upper extremities x 0.2, the chest area x 0.3, for the lower extremities x 0.4.					
Body surface area		**x 0.1**	**x 0.2**	**x 0.3**	**x 0.4**
Total (D)					
Sum up each score for each body part to give the final PASI score					
PASI score =					

Self-administered PASI (SAPASI)

Self-administered PASI (SAPASI) is designed for patients to be able to objectively quantify the severity of their current psoriasis plaques. SAPASI consists of areas of the body affected by psoriasis measured with three visual analogue scales to assess the erythema, induration, and scalability of an average person's lesions. The third person converts the patient's rating to a scale of intensity and PASI level. SAPASI was well validated and had high test reliability (r = 0.82). This objective score is suitable for epidemiological studies when an assessment cannot be made by a physician.

The psoriasis life stress inventory

The psoriasis life stress inventory (PLSI) consists of 15 items that were developed to measure stress associated with the potential psychosocial problems of psoriasis. The PLSI deals specifically with stress due to anticipatory or avoidant coping behaviors that have an effect on limiting the sociocognitive impairment of psoriasis and stress due to patients' actual experiences or beliefs being evaluated by others only on the basis of their skin. Each patient item was asked to rate the level of stress experienced during the previous month on a 4-point scale, from 0 = not at all to 3 = very much. The PLSI score is calculated by adding up the scores for each question, and then the total score can theoretically range from 0 to 45 (Table 2).

Table 2. Psoriasis life stress inventory (PLSI).

Question:	
1.	Uncomfortable with exfoliating your skin.
2.	Feeling self-conscious among strangers.
3.	Feeling that you have to devote most of your time to treating your psoriasis.
4.	Not going to public places (e.g., swimming pools, clubs, health care, restaurants) when you want to.
5.	Wear unattractive or uncomfortable clothing to cover certain areas of your body.
6.	Should avoid sunbathing with other groups of people.
7.	Fear of serious side effects from medical treatment.
8.	People treat you as if your skin condition is contagious.
9.	Avoid social situations
10.	Strangers (children or adults) make rude or insensitive comments about your appearance.
11.	Not enough money to pay medical bills
12.	Often feel like an "outcast" or " social incompatibility."
13.	People consciously try not to touch you.
14.	Hairdressers seem reluctant to cut your hair.
15.	People imply that your skin condition may be due to AIDS, leprosy, or venereal disease.

Dermatology life quality index (DLQI)

The dermatology life quality index (DLQI) is a questionnaire on health-related quality of life (HRQoL), especially in the field of dermatology. With 10 years of experience with more than 85 peer-reviewed research articles and 52 published abstracts describing their use, there are many current studies around the world using the DLQI as a measure of outcome. The DLQI consists of 10 questions about symptoms and feelings, daily activities, vacations, work, school, and personal relationships. Each question is scored from 0 to 3, and then the scores are added up, giving a range from 0, i.e., there is no decrease in quality of life, to 30, which means there is a maximum decrease.

The DLQI questions are specially designed for skin diseases, with 10 questions mentioning the skin. DLQI is designed for use in adults over the age of 18. There is a very high specificity of the DLQI when compared with the normal population. The mean DLQI score with a maximum of 30 in the normal population ranges

from 0 to 0.5. The correlation coefficient between the overall DLQI scores was very high. The interpretation of the DLQI score is as follows; score 0-1; no effect at all on the patient's life; score 2-5: stress has minimal effect on the patient's life; score 6-10; stress has a moderate effect on the patient's life; score 11-20; stress has a huge impact on the patient's life; score 21–31; Stress has a huge effect on the patient's life.[33]

Psoriasis disability index

The psoriasis disability index (PDI) is a self-validated psoriasis-specific questionnaire that initially consists of 10 questions about aspects of the patient's functional disability over the previous 4 weeks. A PDI version that uses 15 questions has also been used. The questions reflect on daily activities, work, personal relationships, and daily treatment. Answers are recorded on a 4-point scale, which indicates a score from 0 = not at all to 3 = very much.

Dermatology-specific quality of life

Dermatology-specific quality of life (DSQL) is a questionnaire designed to discuss the effects of skin diseases and their treatment on physical and social functioning and self-perception. DSQL to assess 5 dimensions of quality of life, namely skin conditions, personal choices, behavior, social aspects, and psychological aspects of quality of life. SQL is rated on a 5-point Likert scale from 0: never to 4, indicating all the time or almost always.[32]

The state-trait anxiety inventory

The state-trait anxiety inventory consists of two questionnaires with 20 questions. State anxiety predicts tension, anxiety, and stress. Trait anxiety measures the level of anxiety as a relatively stable personal characteristic and long-term anxiety not related to a particular situation. Scores on each questionnaire are given on a 5-point Likert scale. The minimum result is 0, and the maximum result is 80. Interpretation of the STAI score is a score of less than 35 that is a low level of anxiety, a score of 36 to 45 is mild anxiety, a score of 46 to 55 is moderate anxiety, a score of 56 to 65 is moderate-high anxiety, a score of more of 65 is a high level of anxiety.[32]

The Beck depression inventory-II

The Beck depression inventory-II (BDI-II) is a 21-item self-report instrument intended to assess the presence and severity of depressive symptoms. Each of the 21 items corresponding to depressive symptoms was summed to provide a single score for the BDI-II. The scoring guidelines for the BDI-II are provided with a recommendation that the threshold is adjusted based on the characteristics of the sample and the intended use of the BDI-II. A total score of 0-13 is minimal depression, 14-19 is mild depression, 20-28 is moderate depression, and 29-63 is major depression.[34]

Depression anxiety stress scale-42 (DASS)

The depression anxiety stress scale 42 is a questionnaire designed to measure the magnitude of unemotional states such as depression, anxiety, and stress. Depression in the DASS focuses on reports of decreased mood, motivation, and self-esteem. Anxiety in the DASS focuses on panic and fear, while stress in the DASS focuses on tension and irritability. The score on the DASS 42 instrument is classified into 5, namely normal, mild, moderate, severe, and very severe. The scores for each item for depression were normal (0-9), mild (10-13), moderate (14-20), severe (21-27), and very severe (≥ 28). Scores for anxiety were divided into normal (0-7), mild (8-9), moderate (10-14), severe (15-19), and very severe (≥ 20), while the stress score was normal (0- 14), light (15-18), moderate (19-25), heavy (26-33) and very heavy (≥ 34).[35]

Taylor manifest anxiety scale (T-MAS)

Taylor manifests anxiety scale (T-MAS) is a measuring tool to determine the degree of anxiety. Statements in T-MAS consist of favorable and unfavorable items. Favorable statements are statements that support or indicate the attributes measured by statements number 1 to number 49, while unfavorable statements are statements that do not support them. The answer "yes" to the favorable item gets a value of 1, the answer "no" gets a value of 0, then the answer "yes" on the unfavorable item gets a value of 0, while the answer "no" gets a value of 1.

Anxiety, according to T-MAS, a person is said to be anxious if the score is more than 22, and if the score is less than or equal to 22 then it is said not to be anxious.

Therapy of psychoriasis

The impact of mental and physical health on patients' quality of life is an important component of treatment in psoriasis patients. Active mental illness often impairs the ability of patients to proactively seek interventions for their psoriasis, so clinicians should be aware of the relationship between depression and anxiety with psoriasis so that appropriate patient education, resources, referrals, and treatment can be offered.[6]

Pharmacological therapy of psoriasis

In a prospective clinical study of patients with moderate to severe psoriasis, in which patients received a modified Goeckerman regimen for psoriasis, the results of this study demonstrated significant improvements in anxiety and depression scores compared with the group receiving only conventional therapy.[13] A previous study showed a significant increase in DLQI at week 16 in psoriasis patients treated with adalimumab compared with placebo-treated. In another study, more than 300 psoriasis patients showed significant improvement in the Beck Depression Inventory and Hamilton Depression Rating Scale scores at 12 weeks in patients with psoriasis treated with etanercept compared with placebo.[35]

Treatment with denileukin diftitox, a drug that targets T cells with the agent DAB389IL-2, results in the apoptosis of activated T cells expressing the IL-2 receptor. T cell depletion in the skin of the lesion results in histologic and clinical resolution. Patients treated with DAB389IL-2 reported a mean reduction of 32% on the PASI scale. Another treatment agent of alefacept, lymphocyte function-associated antigen (LFA)-3-Ig fusion protein, can block CD2-mediated T-cell activation. Patients responded well to alefacept with a mean overall reduction on the PASI scale of 50%. Improvements that occurred at the end of treatment for psoriasis lesions were also followed by improvements in symptoms of mental disorders experienced by psoriasis patients.

Psychological therapy

Psychotherapy is used to improve thought patterns and from this mind is able to control the condition of the body. Relaxation therapy, such as meditation, can be used to control emotions that can trigger stress and suppress the emergence of the severity of psoriasis. In addition, cognitive behavior therapy (CBT) is also effectively used to change the patient's negative thought patterns by presenting new views and thoughts that sufferers do not experience more severe pain than themselves.[6,14]

2. Conclusion

Psoriasis has a major impact on the patient's life so that the measurement of disease status alone is generally considered insufficient to describe the burden of the disease, so it is necessary to consider the patient's assessment of the disease because it affects the patient's quality of life physically, psychologically and socially. Therapy in psoriasis is comprehensive, and this therapy involves screening, treatment, and evaluation of the mental health of psoriasis patients.

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