Correlation of Skin Cancer in Psoriatic Patients

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Abstract

A frequent inflammatory, immune-mediated chronic disease with comorbidities is psoriasis. Psoriatic arthritis, cardiovascular disease, metabolic syndrome, inflammatory digestive disorders, and depression are common comorbidities of psoriasis. The link between psoriasis and specific-site malignancies has received less research. The myeloid dendritic cell, which connects the innate and adaptive immune systems and consequently plays a role in the regulation of cancer-prevention mechanisms, is a crucial cell in the pathophysiology of psoriasis. Inflammation has long been known to have a significant role in the formation of neoplastic foci, and this association between cancer and inflammation is not new. Infection causes localized chronic inflammation to emerge, which then fuels the buildup of inflammatory cells. Reactive oxygen species are produced by different phagocytes, which lead to DNA mutations in cells and the survival of cells with changed genomes.

Keywords: • Psoriasis• Skin cancer treatment

Introduction

The elbows, scalp, knees, and lower back are common sites for erythematous, scaly plaques of psoriasis, a chronic, immune-mediated inflammatory disease. However, it can affect any surface of the skin. The condition is more common in some geographic areas than others, with the Scandinavian Peninsula having the highest prevalence (8%), and eastern Asia having the lowest (0.17%).

Recent medical advancements have altered the assumption that psoriasis is only a skin disorder, and systemic involvement, particularly in severe forms, is now proven. Furthermore, it is now a known truth that related comorbidities exist that has a significant negative impact on the patient's quality of life. Psoriatic arthritis, cardiovascular disease, metabolic syndrome, diabetes mellitus, inflammatory digestive disorders, and depression are common comorbidities associated with psoriasis.

Pathogenesis of Psoriasis

There is still much to learn about the precise etiology of psoriasis. Recent studies, however, indicate that the disease must also be triggered by environmental factors, immunological malfunction, and genetic predisposition. Epidermal hyperplasia (acanthosis), dilated blood vessels in the dermis, and inflammatory infiltration of leucocytes, particularly in the dermis, are the three main histological characteristics of psoriasis. The areas of the skin that are unaffected by psoriasis plaques exhibit normal histology. Hyperplastic epidermal changes include the under expression of keratinocyte differentiation markers, a loss of the granular cell layer, parakeratosis (retention of nuclei in stratum corneum cells), the lengthening of rete ridges, and the presence of micro-pustules of Kogoj and micro-abscesses of Munro.With higher levels of IL-2, IL-12, and interferon-, psoriasis can be categorised as a Th1 response illness. IL-1, IL-13, IL-17A, IL-22, IL-23, and TNF- are also present in higher amounts in psoriatic plaques in addition to those cytokines.

The myeloid dendritic cell, a crucial cell in the pathogenesis of psoriasis that connects the innate and adaptive immune systems, is a type of cell. Histological analysis has shown that psoriatic lesions contain an increased proportion of activated dendritic cells. Additionally, the primary source of the cytokine interferon, which has been linked to the development of psoriasis, is the dendritic cell.

Concerns regarding skin cancer

Inflammation and cancer have a long-standing association. An important factor in the formation of neoplastic foci is inflammation. The development of localised chronic inflammation at infection sites results in the buildup of inflammatory cells. Reactive oxygen species are produced by different phagocytes, which lead to DNA mutations in cells and the survival of cells with changed genomes. As a result, cells with damaged DNA will multiply in the inflammatory region, eventually giving rise to tumour cells. Second, it has been discovered that tumour suppressor genes have changed. Inhibiting tumour growth is largely accomplished by the p53 protein, which controls cell division. The gene that makes the p53 protein was shown to contain numerous mutations in patients with chronic inflammation, which causes uncontrolled cell division. The repetition and persistence of this process sustains the growth of tumour cells, which eventually results in the formation of the neoplasm.

One of the malignancies that psoriasis may cause, skin cancer, is one of the most researched. Non-Melanoma Skin Cancer (NMSC) and melanoma skin cancer are two different types of skin cancer. The most common NMSCs are basal cell carcinoma and squamous cell carcinoma, respectively.

Scientists have attempted to determine how much psoriasis can raise the chance of acquiring skin cancer throughout the years. The link between psoriasis and squamous cell cancer appears to be the strongest. The aforementioned claim is supported by a study done in the USA with only female subjects. These findings showed a measurable increased risk of melanoma and squamous cell carcinoma in psoriasis patients. There was no evidence of a causal link between psoriasis and basal cell cancer.

Over the years, researchers have tried to figure out how much psoriasis can increase a person's risk of developing skin cancer. The strongest connection between psoriasis and squamous cell cancer seems to exist. A study conducted in the USA with exclusively female participants supports the aforementioned assertion. According to these results, psoriasis sufferers have a measurably higher chance of developing melanoma and squamous cell carcinoma. There was no proof that psoriasis and basal cell carcinoma were related in any way. However, it seems that the severity of psoriasis has little effect on the likelihood of acquiring NMSC. Patients with moderate forms, not requiring systemic medication, have a slightly elevated chance of acquiring NMSC and melanoma, according to a 15-year Danish study including more than 5 million individuals. Another intriguing discovery from this study is that people with severe psoriasis have a higher probability of getting NMSC rather than melanoma. Patients with severe psoriasis had a greater probability of acquiring NMSC It's an intriguing theory, according to a study done in Italy on a sample of 72,000 people, that those with psoriasis may be protected from skin cancer. Regardless of the variables taken into account, psoriasis patients showed a relatively lower risk than the control sample. However, PUVA-treated psoriasis patients had a greater chance of developing NMSC than non-PUVA-treated individuals. In addition, the number of PUVA cycles done seems to be correlated with a higher chance of acquiring NMSC.

A study conducted in Italy on a sample of 72,000 people supports the fascinating hypothesis that persons with psoriasis may be more resistant to skin cancer. Psoriasis patients displayed a relatively lower risk than the control sample regardless of the factors considered. Patients with psoriasis who had PUVA treatment, however, had a higher risk of acquiring NMSC than those who did not. Additionally, a higher likelihood of developing NMSC appears to be associated with the number of PUVA cycles completed.

Additionally, psoriasis has been linked to a higher risk of lymphoma development. Hodgkin's lymphomas and non-Hodgkin' lymphomas, which are also the most prevalent, can be distinguished from each other as lymphomas. B-lymphocyte-origin or T-lymphocyte-origin lymphomas are subcategories of non-Hodgkin's lymphomas. Skinny T-cell lymphoma (CTCL) is the most typical type of non-Hodgkin's T-lymphocyte-origin lymphoma. B. Studying the link between psoriasis and lymphoma is challenging due to the small number of cases; however, it has been noted that psoriasis patients have an increased risk of developing Hodgkin's lymphomas and CTCL, particularly those with severe forms of psoriasis who are receiving systemic therapy. The systemic medications methotrexate and PUVA are thought to enhance the incidence of lymphoma in those with severe psoriasis. According to a study conducted in England, people with psoriasis over 65 have a three times greater risk of acquiring lymphomas than the general population.

Chronic inflammation and epidermal hyperproliferation, which are two characteristics of psoriasis, can both result in the growth of tumour cell clones that ultimately result in cancer. Skin malignancies and lymphomas are more common in psoriasis patients who have more severe cases of the disease. This may be explained by the fact that individuals with more severe psoriasis are receiving systemic medications and/or PUVA treatments, as well as the fact that the systemic inflammation is more intense in these patients. Psoriasis's two hallmarks, chronic inflammation and epidermal hyperproliferation, can both promote the development of tumour cell clones that ultimately lead to cancer. Patients with more severe forms of psoriasis are more likely to develop skin cancers and lymphomas. The systemic drugs and/or PUVA treatments that people with more severe psoriasis are receiving may help to explain this, as well as the fact that the systemic inflammation is more severe in these patients.

There is less clarity when talking about alcohol as a risk factor. Alcohol has not yet been proven to be a risk factor for either getting psoriasis or a more severe type. The fact that psoriasis patients drink more alcohol than the general population has been proven. Immunosuppression brought on by prolonged alcohol use has been linked to more aggressive illness. A significant prospective study linked patients' prolonged alcohol use to a higher chance of getting skin cancer, particularly basal cell and squamous cell carcinoma. The same study, which examined several types of alcoholic beverages, discovered that male patients who mostly drank spirits were more likely to develop basal cell carcinoma and melanoma. It is challenging to evaluate the baseline risk of skin cancer in psoriasis patients. Patients with severe psoriasis must take systemic immunosuppressive drugs and/or undergo phototherapy, which may raise their chance of developing cancer. Patients who have had cyclosporine for more than two years have a six-fold higher chance of developing NMSC, particularly squamous cell carcinoma.

Treatment-related cancer risk

Since psoriasis is a chronic condition, the majority of patients must get therapy for an extended period of time. Patients with psoriasis have a wide range of treatment options. The severity of the condition and any other conditions will largely determine the type of therapy a psoriasis patient will receive. As a result, patients with moderate forms typically receive topical treatment, whilst patients with severe forms typically receive systemic therapy. The patient's potential comorbidities are taken into account while choosing a treatment, as some psoriasis medications have the potential to decompensate the patient's other comorbidities.

1. Topical and Oral Corticosteroids: The most frequently prescribed type of medication for people with psoriasis continues to be topical corticosteroid therapy. This can be taken either by itself or in conjunction with calcipotriol, a synthetic vitamin D analogue. The main objective of a Danish study on mice was to evaluate the carcinogenic risk of topical medicines, and the results revealed that none of the topical medications utilised raised the risk of photocarcinogenesis. The topical medications utilised in the study were calcipotriol, hydrocortisone 17-butyrate, and clobetasol 17-propionate. Unfortunately, this subject has not received much attention in the literature.
2. Phototherapy: UV and psoralen oral The management of psoriasis can be done extremely effectively with light therapy (PUVA). With continued epidermal turnover and reduced symptoms, this therapy slows down epidermal cell division. But concurrently, the p53 protein may have alterations, which could increase the likelihood of developing NMSC.
   Patients who received therapy for less than 150 sessions had a marginally increased chance of developing squamous cell carcinoma. There is no evidence linking bath PUVA to an increased risk of NMSC development. It is significant to note that basal cell carcinoma risk does not directly correlate with the number of therapy sessions.

   The danger is not as high for people who have received more than 350 sessions as it is for squamous cell carcinoma. According to more recent US studies, people who receive PUVA treatment have a higher risk of acquiring melanoma. It has been demonstrated that the risk of developing melanoma appears more than 15 years after the initial exposure to PUVA, with individuals who have undergone at least 250 PUVA sessions showing the highest risk. Even after adjusting for age and gender, the most current follow-up study on PUVA revealed that patients who have had at least 200 treatment sessions have a 2.9-fold greater chance of getting melanoma.

3. Conventional Systemic Therapies: Methotrexate is the traditional immunosuppressant that is most frequently used. Because DNA synthesis is prevented, cell turnover is decreased. Patients with psoriasis often receive less than 25 mg of methotrexate per week, which has an immunosuppressive impact. Patients may be at risk for different neoplasms as a result. However, there isn't enough evidence to conclusively link methotrexate to a higher risk of skin cancer. The patient's chance of getting squamous cell carcinoma increases twofold when methotrexate and PUVA are administered together. The risk of NMSC has also been reported to increase when methotrexate and cyclosporine are combined. When methotrexate is used for more than two years, it develops into a separate risk factor for squamous cell carcinoma.
   Another immunosuppressive medication used to treat severe psoriasis is cyclosporine. Patients with psoriasis receive lower dosages of cyclosporine than those undergoing organ transplants. Treatment plans that are intermittent are typically favoured. A prospective study has demonstrated that taking cyclosporine for longer than two years significantly increases the likelihood of acquiring NMSC.

   It is most likely that the risk of NMSC was significantly raised because all patients who were diagnosed with NMSC had previously undergone PUVA treatment. In a different trial, PUVA-treated patients who also got cyclosporine had a three times higher risk of developing squamous cell carcinoma than psoriasis patients who had never had cyclosporine. It should be noted that patients who had already undergone at least 200 PUVA sessions were at the greatest risk. The most popular oral retinoid drug is acitretin. It has both immunosuppressive and anti-inflammatory properties. It seems that taking PUVA and acitretin together is more efficient than using either medication by itself. Additionally, it appears that using both medications together lessens the toxicity of PUVA, which lowers the likelihood of developing squamous cell carcinoma.

Biologic therapies

Serious cases of psoriasis are increasingly being treated with biologic therapy. Tumour Necrosis Factor Inhibitors (TNFi) (etanercept, infliximab, adalimumab, and certolizumab); interleukin IL-12/23 antagonists (ustekinumab); IL-17A inhibitors and IL-17 receptor antagonists; and anti-IL-23 agents are the four categories of biological drugs currently FDA-approved for use in psoriasis. Although biological therapies only target the overactive portion of the immune system, concerns about the potential for cancer are reasonable. The outcomes of research on the risk of NMSC in psoriasis patients are varied. No higher incidence of NMSC was discovered among individuals receiving biologic therapy in a research involving more than 42,000 psoriasis patients. Another UK study comparing the risk of NMSC in patients receiving biological therapy to those receiving

traditional systemic therapy reported no higher incidence of NMSC in those receiving biological therapy.

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