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COMMENTARY

Therapy for Psoriasis Signs and Symptoms

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Description

A chronic, non-contagious autoimmune illness called psoriasis is characterised by elevated, abnormal skin patches. These areas are dry, itchy, and scaly, with red or purple colouring on some persons with darker skin. The extent of psoriasis can range from small, localised spots to total body coverage. The Koebner phenomenon describes how skin damage might result in psoriatic skin alterations there.

The five main kinds of psoriasis are erythrodermic, pustular, guttate, inverse, and plaque. About 90% of instances of psoriasis are plaque psoriasis, commonly known as psoriasis vulgaris. It often appears as white scales on top of crimson areas. The backs of the forearms, shins, navel region, and scalp are the body parts most frequently impacted. Lesions in guttate psoriasis are formed like drops. Pus-filled, tiny blisters with no infection are the first sign of pustular psoriasis. Red areas appear in skin creases due to inverse psoriasis. Erythrodermic psoriasis can arise from any of the other kinds and manifests as a very extensive rash. Most psoriasis sufferers eventually have problems with their finger and toenail nails. Pits in the nails or variations in nail colour may be examples of this.

Most people believe that psoriasis is a genetic condition that is brought on by environmental factors. If one twin has psoriasis, the second twin has a threefold greater chance of developing it if they are identical than if they are not. This shows that psoriasis is predisposed by hereditary factors. Wintertime and certain drugs, such as beta blockers or NSAIDs, sometimes make symptoms worse. Stress and psychological conditions might also contribute. The immune system reacting to skin cells is the fundamental process. The signs and symptoms are often used to make a diagnosis.

Psoriasis does not have a proven cure, although a number of therapies can help manage the symptoms. These remedies include of immunosuppressive medicines such methotrexate, steroid creams, vitamin D3 cream, UV light, and ointments. With just creams, about 75% of skin involvement gets better. Between 2 and 4 percent of people have the condition. Equal numbers of men and women are affected. Although the disease can start at any age, it usually does so in adulthood. Psoriasis is linked to a higher incidence of depression, lymphomas, Crohn's disease, cardiovascular disease, and psoriatic arthritis. Of those with psoriasis, up to 30% develop psoriatic arthritis.

Signs and symptoms

The skin's epidermal layer grows abnormally quickly and excessively during psoriasis. The series of pathological events in psoriasis lead to abnormal skin cell synthesis (particularly during wound repair) and an overpopulation of skin cells. An event (such as skin damage, an infection, or medication) is hypothesised to trigger the immune system to activate the pathogenic events in psoriasis. This is followed by the maintenance phase, which is characterised by the persistent progression of the illness. In contrast to the typical 28–30 days, skin cells are changed every 3-5 days in people with psoriasis.

The inflammatory chemical signals (cytokines) interleukin-36, tumour necrosis factor, interleukin-1, interleukin-6, and interleukin-22 are secreted by these immune cells as they migrate from the dermis to the epidermis. It is thought that these released inflammatory signals encourage the proliferation of keratinocytes. One theory is that regulatory T cells and the regulatory cytokine interleukin-10 are defective in psoriasis. Inflammatory cytokines are identical to those in psoriatic skin lesions

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and joints (in the case of psoriatic arthritis), suggesting a same inflammatory mechanism.

Psoriasis susceptibility markers have been shown to be gene variants of proteins involved in the skin's capacity to act as a barrier. In psoriasis, Deoxyribonucleic Acid (DNA) produced by dying cells triggers the receptors on specific dendritic cells, which in turn causes them to create the cytokine interferon.

Keratinocytes also release cytokines including interleukin-1, interleukin-6, and tumour necrosis factor in response to these chemical signals from dendritic cells and T cells, which alert downstream inflammatory cells to arrive and promote more inflammation. The innate immune system and the adaptive immune system are connected by dendritic cells. They are more prevalent in psoriatic lesions and encourage T cell and type 1 helper T cell growth (Th1). Targeted immunotherapy can decrease the amount of dendritic cells and favours a Th2 cell cytokine secretion pattern over a Th1/Th17 cell cytokine profile. This is also true of psoralen and ultraviolet A (PUVA) therapy. Psoriatic T cells release interferon and interleukin-17 when they migrate from the dermis into the epidermis. It is well known that interleukin-23 stimulates the synthesis of interleukin-17 and interleukin-22. Keratinocytes are induced to release cytokines that attract neutrophils by interleukin-22 and interleukin-17.