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Dental changes in psoriasis

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Abstract
Background: Psoriasis is a chronic inflammatory skin condition that imposes a heavy toll on both the skin and the rest of the body. Diseases of the psyche, liver, heart, and blood vessels are all linked to psoriasis, as is rheumatologic illness (psoriatic arthritis). Its association with subpar health-related quality of life (QOL) underscores the need of achieving maximum illness management. The purpose of this article is to examine the prevalence of dental problems in people with psoriasis. Conclusion: Dental caries, pulp and periapical disease, and periodontal disease are all conditions that are more common in patients with psoriasis. Patients who suffer from periodontitis are more likely to get psoriasis.

Keywords: Dental comorbidities, psoriasis, periodontitis, teeth affection.

Introduction

Psoriasis is an autoimmune disorder characterised by chronic inflammation followed by remission, excessive keratinocyte division, and defective differentiation. Both the innate and adaptive immune systems, as well as hereditary factors, have a role in inflammation.

Psoriasis has a large and complicated cytokine profile.

Under the influence of IL-12, Th1 cells secrete IL-2, tumour necrosis factor alpha, and interferon gamma, as well as IFN-y.

In the same way , IL-Iβand IL-6 are responsible for Th17 cell differentiation ,which secrets IL-6,IL-17,IL-21 and Serum levels of these cytokines correspond with the severity of psoriatic disease [2-4].

Psoriasis pathogenesis has been demonstrated to include the IL-1 signalling pathway. Serum levels of LI -1 were significantly greater in psoriasis patients compared to healthy controls [5, 6].

Chronic periodontitis patients had considerably higher mean salivary IL-I levels compared to the periodontally healthy group, according to research by Rangbulla et al., which suggests that these biomarkers might aid in the screening, early diagnosis, and treatment of periodontal disease. Strong salivary indicators for periodontal inflammation, IL-I were discovered [7, 8].

Affection for teeth in psoriasis sufferers is the focus of this review.

Common lesions, such as atrophic candidiasis, stomatitis, diffuse oral erythema, geographic tongue, fissured tongue, erythema migraine, angular cheilitis, Reiter syndrome, erythema circinate, lichen planus, pemphigoid, and leukoplakia, may mimic the clinical presentation of oral psoriasis. Lesions from oral psoriasis are generally not reported since they are asymptomatic and doctors seldom take biopsies to determine cause [9].

Dental caries, pulp and periapical disease, and periodontal disease are all conditions that are more common in patients with psoriasis. Patients with psoriasis have been shown to have a greater need for dental therapies such calculus removal, prosthesis therapy, and caries management due to an increased risk of periodontal disease and dental caries. Even though periodontitis is increasingly being recognised as a comorbidity of psoriasis, the connections between psoriasis and other dental disorders are still not well understood. Psoriasis patients may also have a worse periodontal state, according to some research [10]. This is indicated by indicators such as increased bleeding on probing, probing depth, clinical attachment loss, amount of alveolar bone loss, missing teeth, and reduced remaining teeth. Psoriasis Psoriasis is typically defined by skin and joint symptoms, however it is a chronic immune-mediated inflammatory disease. Psoriatic arthritis, cardiovascular disease, metabolic syndrome (including diabetes, hypertension, obesity, and dyslipidemia), gastrointestinal illness, renal disease, cancer, infection, and mental health issues are only some of the conditions that have been linked to it. It has been hypothesised that psoriasis patients may be at an increased risk of acquiring periodontitis [11], a chronic inflammatory disease of the supporting tissue of the teeth.

Immunopathogenesis

T-cell immunity plays a crucial role in the pathogenesis of psoriasis, which includes a complicated interaction between the innate and adaptive immune systems. Dendritic cells (DCs), macrophages, and neutrophils are examples of innate immune cells; adaptive immune cells (B and T cells) and resident skin

cells also engage in an active interaction (e.g. keratinocytes, melanocytes, and endothelial cells). These interactions seem to exacerbate and prolong chronic inflammation. The autoantigens generated by keratinocytes (KC) activate plasmacytoid dendritic cells, which produce interferon (IFN)-γ, tumour necrosis factor (TNF), and IL-23. After being activated, autoimmune CD4+ T helper (Th) 17 cells and CD8+ T cytotoxic (Tc) 17 cells move to the epidermis, where they detect epidermal autoantigens and produce IL-17 and other inflammatory cytokines. Psoriatic phenotypic formation and inflammation are both fueled by cytokines secreted by Th17 cells [12].

Skin Keratinocytes and Cells of the Innate Immune System

Dendritic cells, macrophages, and neutrophils, as well as their interactions with keratinocytes, vascular endothelial cells, and adaptive immune cells, have all been connected to the pathogenesis of psoriasis.

Upon damage, keratinocytes produce antimicrobial peptides (LL37, S100 proteins, and -defensins), which are recognised by DCs as professional antigen-presenting cells (APCs). Overexpressed in psoriatic skin, these peptides (mostly LL37) attach to the DNA of cells that have been damaged. Psoriatic plaques may generate IFN due to the activation of plasmacytoid DCs by such binding. Myeloid DCs mature and become activated in response to IFN. By interacting with naive T cells, these activated DCs are converted into APCs and begin secreting large quantities of TNF-, IL-23, IL-12, and IL-6. Those cytokines promote keratinocyte growth and attract neutrophils to areas of inflammation, activating cascades of inflammatory responses. Producing antimicrobial peptides and secreting cytokines (IL-6, IL-1, and TNF-) and chemokines (e.g. CCL20, CXCL5, CXCL8, CXCL9, and CXCL10) [13] are some of the ways in which keratinocytes sustain the inflammatory environment (Fig 1).

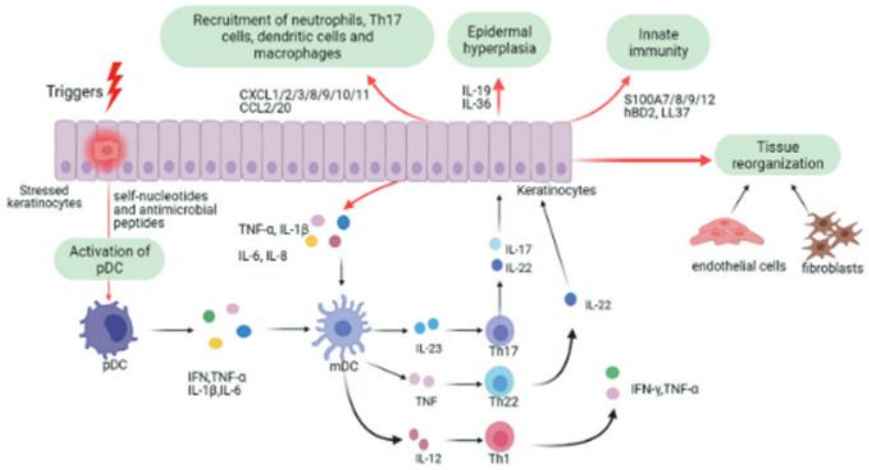

Fig. (1) The function of keratinocytes in the development of psoriasis. The pathogenic development of psoriasis is shown here from the keratinocyte's point of view. The start phase of psoriasis involves keratinocytes being activated by first stimuli and stressed keratinocytes releasing self-nucleotides and antimicrobial peptide, activating pDCs and then mDCs. Activated keratinocytes have a role in psoriasis pathophysiology after being stimulated by cytokines in a variety of ways, including influencing inflammatory infiltration, epidermal hyperplasia, innate immunity, tissue remodelling, etc. plasmacytoid DCs, myeloid DCs, interferons, tumour necrosis factor alpha, interleukin-1 beta, and T helper type 1 (Th1)[14]. Psoriasis-related cytokines.

Interleukin 1 Beta

Interleukin One of the most important proinflammatory cytokines, 1 is released in response to lipopolysaccharide (LPS) and other pathogen- and damage-associated molecular patterns (PAMPs) by monocytes/macrophages and dendritic cells (DCs) (DAMPs). Expression of pro-inflammatory cytokines like IL-1 is stimulated by nuclear factor-kappa B (NF- B) transcription [15].

Protein 17 (IL-17)

Because of its central role in the pathogenesis of psoriasis and other inflammatory and autoimmune diseases like rheumatoid arthritis, inflammatory bowel disease, and systemic sclerosis, as well as responses against infection by extracellular pathogens like Gram-negative bacteria and fungi, interleukin-17 has been the subject of extensive study.

IL-17A (also known as IL-17), IL-17B, IL-17C, IL-17D, IL-17E (also known as IL- 25), and IL-17F, which operates via an IL-17 receptor heterodimer, are the six members of the IL-17 family cytokine. Psoriasis pathogenesis research has focused mostly on

IL-17A since it is a significant downstream cytokine of IL-23 [17].

In a nutshell, when IL-17A binds to its receptors on keratinocytes, it activates innate immunity by inducing the production of keratinocyte-derived antimicrobial peptides like S100A7, LL37, and DEFB4A; it also recruits leukocytes like neutrophils, Th17 cells, mDCs, and macrophages; and it upregulates multiple pro-inflammatory genes like IL-1 On theother hand, IL-17A might indirectly cause epidermal hyperplasia via increased production of IL-19 and IL-36 by keratinocytes [18].

Called IL-22, it is an interleukin.

CD4+ T cells and group 3 innate lymphoid cells are the primary sources of the IL-22 cytokine (ILC3). Non-hematopoietic cells including keratinocytes, epithelial cells, and hepatocytes express its receptor IL-22R [19].

Five primary keratinocyte activities are regulated by IL-22. First, IL-22 promotes the production of antimicrobial peptides (AMPs); second, IL-22 prevents keratinocytes from reaching their terminal differentiation; third, IL-22 causes epidermal hyperplasia; fourth, IL-22 increases the production of matrix metalloproteinases 1 and 3; fifth, IL-22 causes keratinocytes to produce IL-20; and finally, IL-22 increases cellular mobility. Chemokines that attract neutrophilic granulocytes may be induced by IL-22 [20].

Medical Categorization

Psoriasis comprises a wide range of phenotypes, each with its own unique phenotype-specific appearance, distribution, severity, and course. Once diagnosed, the condition often progresses chronically, while some people experience remission on their own [21].

Psoriasis plaquing

Plaque psoriasis, or psoriasis vulgaris, is the most prevalent kind and accounts for 90 percent of cases. It manifests on the head, trunk, buttocks, skin folds, nails, or palmoplantar areas as well-demarcated, erythematous, symmetric plaques with an underlying silvery-white scale [21]. Dermatitis inversa Because of the friction and moisture in these regions, the skin of the underarms, buttocks, and genitalia may not have scales from inflammation caused by inverse psoriasis[22].

Psoriasis guttata

Droplet-shaped pink papules 1–10 mm in diameter with a fine scale define individuals younger than 30 with acute guttate psoriasis. The guttate form often manifests after an infection with ß-hemolytic or group-streptococci, tonsillitis, pharyngitis, or a virus[23].

Psoriasis Pustular

Neutrophil-rich, white, consolidating pustules define pustular psoriasis, which may affect the whole body or only the palms, soles, nails, and tips of the fingers and toes. Erythrodermic psoriasis is an uncommon but potentially fatal condition that causes severe skin redness and layer-by-layer skin loss [24].

Involvement of Nails

Psoriatic arthritis sufferers have a 90% lifetime risk of developing nail psoriasis, while those with skin psoriasis have a 45% risk. Both the matrix and the nail bed, or both, may be involved in nail psoriasis. Nail changes include pitting, nail whitening, splinter haemorrhages from capillaries under the nail, thickening of the skin under the nail (subungual hyperkeratosis), yellow-reddish discoloration of the nail (oil drop or salmon spot), loosening and detachment of the nail (onycholysis), and disintegration [25].

Psoriasis Severity Ratings

Psoriasis severity may be measured using a number of different measures. The Psoriasis Area Severity Index (PASI) is the gold standard for quantifying the extent of psoriasis. Fredriksson and Pettersson created the score in 1978 to evaluate the efficacy of retinoid therapy for patients with persistent plaque-type psoriasis. Plaque intensity and area are determined independently for the scalp, trunk, upper and lower extremities [26].

Mild, moderate, and severe are all interpreted from PASI scores. A PASI score between 5 and 10 indicates moderate illness, whereas a score more than 10 indicates severe disease. The current standard for most clinical studies in psoriasis is a 75% decrease in the PASI score (PASI 75), and this is also the requirement for effectiveness of novel psoriasis medicines authorised by the FDA [27].

Psoriasis outlook

Although psoriasis seldom causes serious complications, it is a chronic condition that may worsen and improve over the course of a person's lifetime. Psoriasis in its mildest forms does not seem to raise mortality risk. Patients with more advanced psoriatic skin disease are at increased risk for cardiovascular disease, diabetes, chronic lung disease, renal disease, and other significant medical complications. It is crucial to identify and treat psoriasis's comorbidities as soon as possible. Psoriasis therapy shouldn't conflict with co-morbid disease care, and vice versa, hence an integrated strategy is necessary. [28].

Periodontitis:

Periodontal disease is an ongoing infection that may destroy the gums, periodontal ligament,

and/or alveolar bone, resulting in tooth loss. Tooth loss is a result of periodontitis, an inflammatory condition that causes infection of the tissues that support your teeth (Fig.2). Periodontitis is a chronic inflammatory illness of the gingiva that begins in the gingival fissure, spreads to the surrounding alveolar bone, and ultimately causes a loss of mineral density, loosening of the teeth, and the possibility of permanent tooth loss. Periodontitis may be traced back to a combination of host and oral microbial dysbiosis in its aetiology.[29].

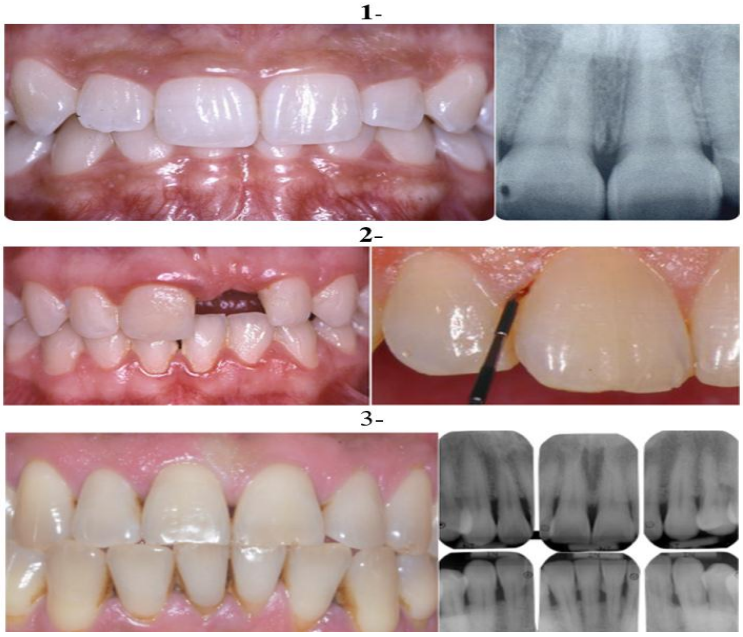

Fig. (2) 1. Periodontal health is defined by clinically and radiographically intact periodontium. Erythematous and swollen gingivitis are clinical symptoms of gingivitis caused by the presence of supragingival biofilm and calculus.

Radiographic signs of horizontal alveolar bone loss and clinical evidence of supra- and subgingival calculus indicate the presence of periodontitis [30].

Psoriasis and Interleukin 1

Secukinumab, by inhibiting IL-17A, normalises levels of dysregulated proteins, including IL-1, in individuals with psoriasis [31].

Psoriasis aetiology relies heavily on IL-23 and IL-17A, two inflammatory cytokines. Effector cytokines like IL-17A and IL-22 are generated in response to IL-23's stimulation of Th17 cell differentiation, activation, proliferation, and survival. However, IL-17 may also be produced in the absence of IL-23. IL-17A promotes inflammation, coagulation, and bone/joint injury in a variety of cellular targets inside the skin and joints. Keratinocytes, endothelial cells, and innate immune cells are primary IL-17 targets in psoriasis. Antimicrobial peptides (lipocalin 2, S100A proteins (S100A7, psoriasin, and beta defensins), proinflammatory cytokines and chemokines (including IL-1), and IL-17 are all produced by keratinocytes in response to IL-17. Secukinumab, by inhibiting IL-17A, normalises levels of dysregulated proteins, including IL-1, in individuals with psoriasis [32].

Psoriasis is characterised by the overproduction of pro-inflammatory cytokines as tumour necrosis factor alpha, interferon gamma, interleukin 1 beta, interleukin 8 and interleukin 12. The inflammatory responses to different illnesses are affected by variations in the genes that code for cytokines. Some significant cytokines that are known to possess polymorphism areas include genes of IL-1 cluster and TNF-α. One of the essential variables in the pathophysiology of the illness is NF-kappa B [33], one of the most significant regulators of pro-inflammatory gene expression.

The link between psoriasis and gum disease:

Chronic inflammatory illnesses such as psoriasis and periodontitis have similar inflammatory pathways. Immune activation is driven by macrophages, -T cells, T helper (Th) 17 cells, and dendritic cells in both illnesses, with adaptive immunity playing a supporting role. Target areas of illness have been shown to contain elevated

amounts of cytokines such tumour necrosis factor, interleukin (IL)-1ß, and IL-17. As an added bonus, chemokines entice neutrophil granulocytes to move into the crevice of the gingiva and psoriatic skin lesions [34].

Periodontal inflammation and tissue degeneration are both strongly stimulated by interleukin-1. Bone resorption and the generation of tissue-degrading proteinases are two of IL-1's features. Potentially using IL-1 as a therapeutic target for periodontitis has been proposed [35].

How IL-1 contributes to gum disease

Bone resorption, immunological modulation, and inflammation are all processes in which IL-1 plays a role as a pro-inflammatory cytokine in periodontitis. Patients with periodontitis have been shown to have higher IL-1 levels in their saliva and GCF than healthy controls. [36].

The secreted IL-1 accumulates and then plays a role in the pathophysiology of periodontitis by setting off a chain reaction of inflammatory responses. IL-1 is a prominent cytokine in periodontitis because it stimulates bone resorption, increases blood flow to the inflammatory region, and recruits leukocytes and neutrophils. Increased expression of collagenolytic enzymes, matrix metalloproteinases (MMPs), upregulation of receptor activator of nuclear factor kappa-B ligand (RANKL), and PGE2 synthesis in fibroblast cells all contribute to IL-1's promotion of osteoclastogenesis, which in turn promotes extracellular matrix degradation and leads to bone resorption and tissue destruction.[17] (Fig. 3).

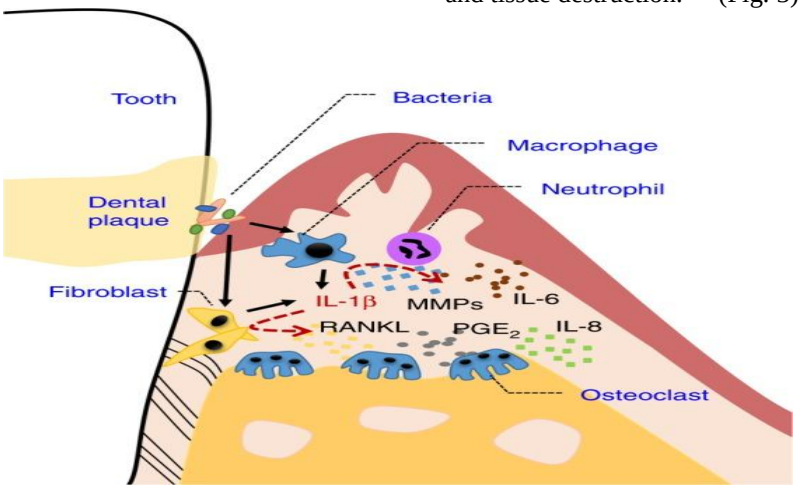

Fig. (3) IL-Dental caries, pulp and periapical disease, and periodontal disease are all conditions that are more common in patients with psoriasis, leading us to conclude that they provide an elevated danger for these patients. Patients with periodontitis are at a much higher risk of developing psoriasis.

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