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The Pathway from Skin to Liver: Psoriasis

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ABSTRACT

Psoriasis is a chronic, recurrent, autoimmune, inflammatory skin disease. While psoriasis was considered limited to the skin in the past, it is now recognized as a chronic systemic inflammatory disease accompanied by many comorbidities. The main comorbidities of the disease are non-alcoholic fatty liver disease (NAFLD), mood disorders including depression, cardiometabolic diseases including myocardial infarction, hypertension, type 2 diabetes mellitus, hyperuricemia, dyslipidemia, obesity, metabolic syndrome, and psoriatic arthritis. NAFLD is estimated to have a prevalence of 25% in the general population and is a leading cause of cirrhosis and liver transplantation. Currently, NAFLD has become an escalating epidemic, driven by the rising incidence of obesity, metabolic syndrome, and insulin resistance, as well as the systemic effects of psoriasis itself. This review aims to highlight the adverse effects of psoriasis, a skin disease requiring long-term medication, on the liver, and to emphasize these effects when planning treatment regimens.

Keywords: Skin, Inflammation, Non-alcoholic fatty liver disease, Psoriasis

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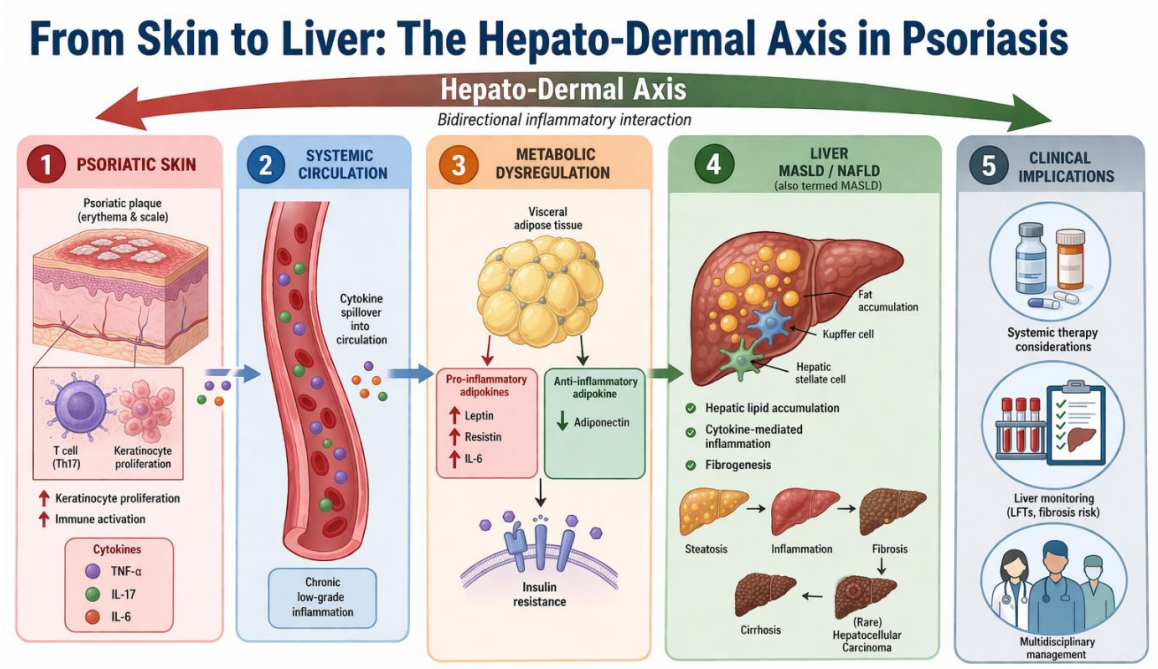

Figure 1. *Visual depiction of the hepato-dermal axis connecting psoriasis and liver disease. Psoriatic skin inflammation, driven by increased TNF-α, IL-17, and IL-6, leads to systemic cytokine spillover and chronic low-grade inflammation. This process contributes to metabolic dysregulation characterised by adipokine imbalance and insulin resistance. Consequently, hepatic lipid accumulation and cytokine-mediated injury promote the progression from steatosis to inflammation and fibrosis within the spectrum of MASLD/NAFLD. The bidirectional interaction between the skin and the liver underscores the importance of liver monitoring, careful selection of systemic therapies, and multidisciplinary management in patients with psoriasis.*

INTRODUCTION

Psoriasis is a chronic, recurrent, autoimmune, inflammatory skin illness characterized by lesions in the form of plaques covered with shiny, white scales [1-3]. While psoriasis was previously considered limited to the skin, today it is regarded as a chronic systemic inflammatory disease accompanied by many comorbidities [4]. The main comorbidities associated with the disease are non-alcoholic fatty liver disease (NAFLD), mood disorders including depression, cardiometabolic diseases including myocardial infarction, hypertension, type 2 diabetes mellitus, hyperuricemia, dyslipidemia, obesity, metabolic syndrome and psoriatic arthritis [2, 5]. NAFLD is estimated to have a prevalence of 25% in the general population and is a leading cause of cirrhosis and liver transplantation. Today, NAFLD has become an increasingly prevalent epidemic, partly due to obesity, metabolic syndrome, and insulin resistance, and partly due to psoriasis [6]. Previous studies have shown a high prevalence rate of NAFLD, varying from 47% to 59%, in patients with psoriasis [7]. This review aims to highlight the adverse effects of psoriasis, a skin disease requiring long-term medication, on the liver, and to emphasize these effects when planning treatment regimens.

Depending on the severity of psoriasis, a spectrum of disease can be observed in the liver, ranging from simple fatty infiltration to fibrosis progressing to NAFLD, followed by cirrhosis and, rarely, hepatocellular carcinoma [4]. It is hypothesized that liver damage in psoriasis is partly due to the release of cytokines from skin-derived cells. Cytokines are immune components that mediate the inflammatory response of psoriatic plaques and play a mechanistic role in the development of fatty liver disease and insulin resistance. However, their role as biomarkers of NAFLD in psoriatic patients is not well determined [6]. It is hypothesized that lymphocytes and keratinocytes of psoriatic skin origin, forming a so-called hepato-dermal axis, produce inflammatory cytokines such as interleukin 6 (IL-6), Tumor Necrosis Factor-alpha (TNF-α), and interleukin 17 (IL-17), which then reach the liver systemically, causing a series of metabolic disorders that increase insulin resistance, and that this is a distinctive feature of NAFLD pathogenesis [8].

NAFLD accompanying psoriasis is characterized by hepatic fat in the absence of excessive alcohol consumption and is strongly associated with metabolic syndrome, type 2 diabetes mellitus, and obesity [5, 9]. NAFLD, the most widespread liver disease in the general population, is an

immune cell-mediated inflammation that carries a risk of progression to cirrhosis [2, 9]. The incidence of this comorbidity in patients with psoriasis is 1.5-3 times higher than in the general population [5]. Psoriasis and NAFLD are multifactorial diseases [10]. Although the underlying mechanisms of both are not fully understood, they are strongly associated with increased oxidative stress, low-grade chronic inflammation, and peripheral insulin resistance [5]. Inflammation associated with psoriasis can initiate the development of NAFLD [6]. Both conditions are associated with an increase in pro-inflammatory adipokines and hepatokines, and a decrease in anti-inflammatory adipokines and adiponectins. This imbalance in inflammatory mediators results in insulin resistance, thus facilitating the development and progression of NAFLD [11].

Adipose tissue functions as an endocrine organ generating adipokines and adipocytokines that play significant roles in both psoriasis and NAFLD [10]. These include proinflammatory adipokines leptin, resistin, IL-6 and adiponectin, which are important in insulin sensitivity, lipid and glucose metabolism, energy balance, blood pressure, and angiogenesis [10,11]. TNF-α and IL-6, which are vital for inflammatory processes [11]. These mediators enable interaction between adipose tissue and other organs, such as the liver, and the liver responds by producing hepatokines such as fibroblast growth factor 21, CRP, fetuin-A, IL-6, and TNF-α [11].

Proinflammatory cytokines like TNF-α and IL-17, formed in psoriatic tissues, reach the liver via the circulation, causing insulin resistance and hepatic inflammation [5].

TNF-α plays a significant role in the pathogenesis of psoriasis [12]. Studies have shown that patients with NAFLD have higher inflammatory activity, as determined by the cytokines TNF-α and TGF-β, and this activity was found to be higher in patients with moderate to severe NAFLD [6]. In psoriasis, TNF-α increases keratinocyte proliferation, the production of pro-inflammatory cytokines, the expression of vascular endothelial cell adhesion molecules, and angiogenesis [12].

T cells in adipose tissue synthesize IL-7, which regulates adipogenesis and glucose metabolism [12]. IL-17 (Th17 linkage), secreted from T helper cells, plays a central role in the pathogenesis of psoriasis. [7]. In NAFLD, IL-17 and IL-17-secreting T helper type 17 cells facilitate the progression from simple steatosis to steatohepatitis [10]. IL-17 contributes to the pathogenesis of both psoriasis and NAFLD [7]. The liver is one of the main producers of IL-17 [13]. Elevated IL-17 levels have also been shown to activate the IL17A receptor in hepatocytes, sinusoidal cells, Kupffer cells, biliary cells and hepatic stellate cells [7,13]. This cytokine increases lipid uptake by hepatocytes, causes liver fibrosis, and is associated with an increased risk of hepatocellular carcinoma. In some studies, which inhibit the effects of Th17 differentiation, a reduction in pro-inflammatory molecules, steatosis, and liver damage has been demonstrated [7].

Ultimately, in psoriasis, IL-17 triggers IL-6 expression in keratinocytes [12]. As we have previously emphasized, IL-6 is a pro-inflammatory cytokine involved in the pathogenesis of both psoriasis and NAFLD, and is known to be associated with hypertension, type 2 diabetes, insulin resistance and abdominal obesity [12,14]. IL-6 induces TNF-α and CRP synthesis in the liver, and can change insulin sensitivity via the insulin signaling pathway [14].

TNF-α and IL-6 not only affect keratinocyte proliferation and differentiation, but also increase insulin resistance and promote the release of pro-inflammatory cytokines [15].

TNF-α, IL-17, IL-6, and CRP, manufactured by the liver (hepatokines) and psoriatic skin, have mutual direct effects on these organs. There may be a bidirectional association between psoriasis and NAFLD through proinflammatory pathways referred to as the hepato-dermal axis [5]. The axis between psoriasis and the liver, and the contributing adipose tissue, is schematically shown (**Figure 1**).

Conclusion and Recommendations

In conclusion, it is now accepted that psoriasis is a systemic inflammatory disease. In light of this literature, clinicians should not focus solely on the skin in patients with psoriasis, but should also consider other organs and systems, particularly the liver, taking a multidisciplinary approach, given that the disease is a systemic inflammatory disease. We believe this approach is more suitable for improving treatment efficacy and reducing potential side effects in psoriasis patients who require long-term treatment affecting the liver.

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version.

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