Review paper

Exploring the interplay between psoriasis and inflammatory comorbidities: focus on cardiovascular disease and metabolic syndrome

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Abstract

Psoriasis is a common, chronic skin disorder characterised by chronic inflammation. Because psoriasis lesions usually affect large areas of the skin, it can cause an extension of inflammatory involvement in systemic inflammatory diseases. The current reports indicate that psoriasis is often associated with hypertension, cardiovascular disease, dyslipidaemia, obesity, and diabetes. Psoriasis and its comorbidities have various common pathogenic pathways including genetic connections, chronic inflammatory process, and proinflammatory proteins secreted by adipocytes. Early identification of risk factors and screening would improve patient outcomes through early detection and management of such complications. Patients with psoriasis should be regularly assessed for comorbidities. Numerous recommendations have been suggested to reduce the risk of associated diseases including Mediterranean diet, smoking cessation, and weight loss. Metabolic syndrome is a significant public health problem and a serious concern in the population of patients with psoriasis.

Introduction

Psoriasis vulgaris (PV) is a common, chronic, inflammatory skin disorder that affects around 2% (over 60 million adults and children) of the population worldwide [1, 2]. Psoriasis occurs with equal frequency in women and men, with a mean age of disease onset of 30–40 years. The occurrence of psoriasis depends on gene-environment interactions. The are some triggering factors that can provoke the first lesions, such as stress, infections, alcohol abuse, smoking, exposure to certain drugs, or – very rarely – sunlight [2].

Psoriasis has some different clinical types: chronic plaque psoriasis, guttate psoriasis, inverse psoriasis, pustular psoriasis, and erythrodermic psoriasis. Psoriatic inflammation of the joints causes psoriatic arthritis, which develops in about 40% of psoriasis patients. The most prevalent type (almost 90%) is a plaque psoriasis [1]. It mainly presents as well demarcated, erythematous, raised plaques covered in silvery scales [2, 3].

The most common histological features of psoriasis are epidermal hyperproliferation and parakeratosis with the infiltration of various inflammatory cells: lymphocytes and neutrophils in the dermis [4]. Moreover, inflammatory cells and cytokines can systemically circulate in various organs, which may cause long-term damage to multiple tissues and organs [5]. The immune response in psoriasis is characterised by proliferation of Th1 and Th17 cells, which produce the proinflammatory mediators tumour necrosis factor-α (TNF-α), interferon-γ (INF-γ), and interleukins (IL) 6 (IL-6), IL-22, and IL-17 [6].

Because psoriasis lesions usually affect large areas of the skin, they can cause an extension of inflammatory involvement in systemic inflammatory diseases [4, 6]. Recent studies identified that there are conditions occurring more commonly in patients with psoriasis than in the general population, such as: cardiovascular disease, hypertension, obesity, type 2 diabetes, dyslipidaemia, metabolic syndrome, osteoporosis, chronic obstructive pulmonary disease, asthma, chronic kidney disease, and inflammatory bowel disease [2].

Patients with psoriasis have an increased risk of developing autoimmune thyroid disease, gout, mental health diseases, chronic kidney diseases, and even malignancy, leading to economic problems, poor quality of life, and reduced productivity [7].

In this paper, we focused on common inflammatory comorbidities in psoriasis highlighting cardiovascular diseases and metabolic syndrome. Metabolic syndrome is a significant public health problem and a serious concern in the population of patients with psoriasis.

Cardiovascular diseases

Psoriatic patients are at high risk for cardiovascular disease (CVD), atherosclerosis, and myocardial

infarction, but the precise underlying mechanisms linking psoriasis and CVD are not well defined [4]. Of note, they are at increased risk of developing independent cardiovascular risk factors including hypertension, diabetes, hyperlipidaemia, and obesity. Scientific evidence shows that the chronic inflammation of the skin that occurs in psoriasis promotes vasculitis and the formation of atherosclerotic plaque. Elevated serum proinflammatory cytokines, especially TNF-α and IL-23, are associated with the occurrence and progression of CVD [3].

Interestingly, IL-17A levels were significantly lower in patients with moderate-to-severe psoriasis with CVD compared to patients with moderate-to-severe psoriasis without CVD. Therefore, IL-17A is considered a potential marker of CVD in patients with moderate-to-severe psoriasis [8]. Elnabawi *et al.* [9] reported that biologic therapy in severe psoriasis is associated with beneficial modulation of coronary plaque indices. After 1-year of biologic therapy, a 5% reduction in non-calcified plaque burden on anti-TNF therapy and a 2% reduction on anti-IL-12/23 therapy was recorded. Moreover, in the group of patients treated with IL-17 inhibitors, a 12% reduction was observed.

Of note, not only biologic therapy but also treatment with methotrexate decreases the incidence of vascular disease for patients with psoriasis, probably by modulation of chronic inflammation [10]. These findings highlight the importance of treating systemic inflammation in preventing coronary artery disease. Current reports indicate that systemic therapy is associated with a significant decrease in risk of CVD, compared to no systemic therapy or only topical treatment of psoriasis [11].

Platelets also play a significant role in the pathogenesis of vascular dysfunction in psoriasis. RNA sequencing of platelets in psoriasis reveals an interferon signature and increased expression of cyclooxygenase-1 (COX-1), which correlates with the severity of psoriasis skin symptoms. Furthermore, platelets are predominantly present in psoriatic lesions and trigger a more than 20-fold increase in endothelial cell pro-atherosclerotic transcripts compared to platelets from non-psoriasis patients. This suggests that targeting COX-1 may offer potential benefits in managing psoriasis [12].

The incidence of myocardial infarction (MI), coronary heart disease, and severe vascular accidents is higher in patients with psoriasis compared to patients without psoriasis, and the risk increases with increasing severity of psoriasis. A study from Denmark found that the risk of heart attack was increased only in patients with severe psoriasis – but not in patients with mild psoriasis – compared to the general population [13].

Hypertension

Current reports indicate a higher incidence of hypertension arterialis (HA) in patients with psoriasis [14]. The prevalence of hypertension in psoriasis patients is estimated at 38.8%, while in the general population it is about 29.1% [15]. The mechanism underlying the association of psoriasis and risk of HA has not yet been well defined. Psoriasis can cause stress, depression, more smoking, drinking alcohol, and lack of exercise, and therefore can lead to abnormal blood pressure [16].

Recent studies identified that angiotensin-converting enzyme (ACE) – which is an important component of the renin-angiotensin-aldosterone system – polymorphisms are associated with psoriasis [17]. Moreover, polymorphism of the gene LNPEP [18] and endothelial nitric oxide synthase (eNOS) gene in psoriatic patients may lead to genetic susceptibility for hypertension. Endothelial nitric oxide synthase is involved in the regulation of vascular smooth muscle contraction, due to its involvement in the production of nitrogen oxide [19], while Human Placental Leucine Aminopeptidase (P-LAP encoded by the LNPEP gene) is an essential component in the renin–angiotensin system [19]. Interestingly, the role of endothelin-1, TNF-α, and IL-17 in the pathogenesis of hypertension in psoriasis patients has also been described [3].

There are studies confirming that that some medications used to treat psoriasis (e.g. cyclosporine) can induce or worsen hypertension. The calcium-channel blocker amlodipine is often used to reverse cyclosporine-induced hypertension [20]. It is recommended that all patients with psoriasis undergo screening and management of hypertension [16].

Obesity

The prevalence of obesity in psoriasis patients ranges between 15% and 30% [21]. Several studies showed that there is a multifactorial link between obesity and psoriasis. It is also known that obesity may increase the risk of psoriasis, and vice versa, also in the paediatric population [22]. What is more, increased body mass index (BMI) worsens the course of psoriasis and impairs the treatment response [23].

Dietary habits, lifestyle, certain genetic factors, and the microbiome are indicated as the main factors in the progression of both pathologies, because they are related to a chronic proinflammatory state [24]. Scientific evidence indicates that insulin-like growth factor 1 (IGF-1) as a keratinocyte proliferation factor observed in psoriasis is involved in the development of diabetes and hyperlipidaemia [25].

The relationship between psoriasis and obesity is observed especially in psoriasis patients with a more frequent polymorphism of the Fat Mass and Obesity Associated Gene (FTO), which is associated with increased BMI [26]. Furthermore, Kisielnicka *et al.* [26] described a correlation between 11 genetic polymorphisms and abnormal body weight in psoriatic patients. Moreover, the hypertrophied adipocytes can

produce free fatty acids (FFAs) as well as adipokines and adipocytokines, which leads to a chronic low-grade inflammatory state. These proinflammatory molecules produced by excessive fat tissue activate the innate immune cells (such as neutrophils and macrophages) and then Th1, Th17, and Th22 cells, which leads to epidermal hyperplasia [23].

Dyslipidaemia

Dyslipidaemia is an important, modifiable risk factor for coronary heart disease and myocardial infarction. Treatment of dyslipidaemia is based on lifestyle modification and lipid-lowering drugs therapy (including statin therapy as the first-line medical treatment for dyslipidaemia) [27]. Psoriasis and dyslipidaemia are deeply intertwined conditions, as demonstrated by several studies. Moreover, dyslipidaemia was found to be an independent risk factor for the development of psoriasis [3].

Scientific evidence indicates that patients with psoriasis have significantly increased mean levels of cholesterol, triglyceride, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) and decreased levels of high-density lipoprotein (HDL) [28]. In chronic inflammation, TNF-α induces the production of LDL and oxLDL, but at the same time reduces the level of HDL-C [25]. In patients with severe inflammatory diseases, it is recommended to increase the calculated risk by approximately 50%. The treatment of dyslipidaemia in psoriatic patients is similar to that in patients without inflammatory disorders [28]. Moreover, it has been demonstrated that statins reduce the severity of psoriatic lesions [29]. To improve the effectiveness of treatment, it is necessary to expand knowledge about the relationship between the lipid profile and psoriasis.

Type 2 diabetes

Systemic inflammation that is present in psoriasis may increase the risk of type 2 diabetes (T2D) and insulin resistance. The association may even be strongest among patients with severe psoriasis [30]. The prevalence of T2D in psoriasis patients is estimated between 10% and 20% [21], while in the general population it is about 5% to 6% [31]. The risk of developing T2D increases with increasing body surface area (BSA) affected by skin lesions [32].

It has been discovered that there is a significant association in the PTPN22, ST6GAL1 and JAZF1 gene loci in patients with diabetes and psoriasis [33]. Additionally, some studies have shown that the STING-IRF3 pathway is associated with the inflammatory response involved in diabetes in psoriasis patients [34]. Of note, medications used in the treatment of psoriasis, including cyclosporine, may worsen serum lipid and glucose levels, which can be explained by inhibition of insulin secretion [35]. Some studies have implicated that psoriasis is associated with higher rates of microvascular and macrovascular complications of diabetes [36]. In patients with psoriasis affecting > 10% BSA, diabetes prevention measures should be taken [32].

Conclusions

Until recently, psoriasis was considered as a disease affecting only the skin, but now it is known that systemic inflammation that is present in psoriatic disease may increase the risk of cardiovascular disease, hypertension, obesity, T2D, dyslipidaemia, and metabolic syndrome. Therefore, it is necessary not only to properly treat psoriasis, but also to take care of the patient holistically.

Early identification of risk factors and screening would improve patient outcome through early detection and management of those complications. Psoriatic patients should be regularly evaluated by measuring blood pressure, waist circumference, fasting blood glucose and/or haemoglobin A1C, and fasting lipid levels. Moreover, it is very important to educate patients about modifiable risk factors for comorbidities and a healthy lifestyle.

Overlapping inflammatory pathways and genetic predispositions may link the pathophysiology of cardiovascular diseases and metabolic syndrome and psoriasis. Given this overlap, it is reasonable to conclude that the conditions are related and that action in 1 case may be accompanied by action in the other; similarly, effective treatment of one condition may result in improvement of the other.

Although psoriasis is not usually life-threatening, patients may incur lifelong costs due to the chronic nature of the disease. In addition to the direct medical costs incurred in treating the disease, psoriasis patients often suffer from lost productivity at work, sick leave, job losses, or lower wages. Furthermore, patients may experience a reduced quality of life due to psoriasis-related discomfort or disability or social stigma [37].

Optimal lifestyle modifications in psoriasis are fundamental strategies aimed at reducing cardiovascular disease risk and improving skin severity. In overweight or obese individuals with psoriasis, the National Psoriasis Foundation recommends a hypocaloric diet. A meta-analysis of seven randomised controlled trials involving approximately 900 overweight or obese patients with psoriasis revealed that weight loss achieved through caloric restriction leads to improvements in psoriasis skin severity. Specifically, there was a threefold higher rate of skin clearance when combining diet with psoriasis treatment compared to psoriasis treatment alone.

The National Psoriasis Foundation weakly recommends adopting a Mediterranean diet, as patients who adhere to it demonstrate improvements in pso-

riasis skin severity, reduced fat mass, and lower levels of high-sensitivity C-reactive protein. Additionally, smoking cessation is encouraged because it not only reduces CVD risk but may also improve skin psoriasis severity [38].

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Ethical approval

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Conflict of interest

The authors declare no conflict of interest.

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