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Psoriasis and Insulin Resistance: Is there a Relationship?

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Abstract

There is increasing evidence that psoriasis –chronic inflammatory skin disease- is associated with obesity, the metabolic syndrome, and associated comorbidities, including insulin resistance/type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease. This association – particularly insulin resistance- is postulated to arise partially because of a systemic pro-inflammatory state that is mediated by adipose tissue. Such an involvement raises apprehension concerning the long-term health implications for management of psoriasis and suggests that enhanced alertness and assessment of obese patients and associated metabolic disease are warranted in this population.

Keywords: Psoriasis, Insulin resistance

Introduction

Changes to the skin may signal a more serious health problem, frequently serving as a marker for underlying internal disease. Insulin is a central player in the metabolic system and has an important role in homeostasis and physiology of the skin. Under healthy conditions, insulin regulates the equilibrium between differentiation and proliferation of keratinocytes, a precondition for the formation of the epidermal structure [1]. Insulin resistance (IR) is defined clinically as the inability of a known quantity of exogenous or endogenous insulin to increase glucose uptake and utilization in an individual as much as it does in a normal population. Under conditions of chronic inflammation (e.g. psoriasis), high levels of pro-inflammatory cytokines induces IR leading to an increased proliferation of basal keratinocytes and, at the same time deny access to differentiation [2].

There are pros and cons about the link between dermatological diseases and metabolic alteration. Is there a relationship between psoriasis and IR? The faithful answers are still scant. However, some studies had revealed that, psoriasis patients are at high risk to develop cardiovascular diseases and diabetes. It is well established that, obesity is a risk and exacerbating factors for psoriasis itself. Moreover, accumulating literatures illustrate that, patients with psoriasis generally share obesity related complications such dyslipidemia, diabetes, and IR [3].

Pereira et al. [4], documented a significant association between IR and psoriasis. They suggested that, in addition to pharmaco-therapy, treatments for psoriasis must also be designed to encourage lifestyle alterations such as exercise and diet modifications. Insulin sensitivity indices were reported by Sale and Torres [5] to be significantly lower in psoriatic as compared with controls. Moreover, Rajappa et al. [6] demonstrated that serum insulin level and IR indices were correlated positively with the severity of psoriasis and being decreased after systemic treatments. These findings were also confirmed by Gyldenløve et al. [7] who showed that, normal glucose-tolerant patients with moderate to severe psoriasis had significantly reduced insulin sensitivity compared with healthy control group.

Furthermore, according to Moro et al. [8], the association between IR and psoriasis has been reinforced. They highlighted that, women with psoriasis and polycystic ovary syndrome had a greater prospect of rigorous skin condition, hyper-insulinemia, IR, and dyslipidemia, as well as a more, than those with psoriasis alone.

Dahra et al. [9] study was aimed to assess IR and pattern of body fat deposition in psoriasis. The psoriatic patients in their study showed that, though their normal fasting plasma glucose, serum insulin level along with an increased IR were significantly high. Furthermore, these abnormalities were significantly dependent on peripheral and central fat deposits. They suggested that, psoriatic patients need to be evaluated for metabolic syndrome and managed accordingly. In the converse, one recent study had revealed that the prevalence of IR in patients with psoriasis aged 30 to 49 years is similar to the general Polish population [10]. By the way, preliminary unpublished collected data from an acne study in the department of dermatology, College of Medicine, Qassim University, Saudi Arabia are in agreement with these facts. Final results will be published afterward.

On the other hand, numerous epidemiologic and clinical studies had revealed a high prevalence of metabolic syndrome in patients with psoriasis compared with other skin diseases. Systemic inflammation in psoriasis causes IR and cardiovascular diseases [11]. Adipokines are adipose-tissue-derived factors that are involved in metabolic processes. More recently, Coban et al. [12] thought that, these adipokines are associated with the development of psoriasis. They concluded, plasma levels of adipokines might be useful for evaluating the disease activity of psoriasis and its comorbidities. Owczarczyk-Saczonek and Nowicki RJ [10] showed that, severe psoriasis is associated with a significantly higher prevalence of risk

factors for cardiovascular disease and abdominal obesity. Since endothelial cells play an important role in psoriasis as well as in the development of cardiovascular comorbidities, Schlüter et al. [13] investigated whether a common mechanism, namely cytokine-induced IR, underlies both pathologies. They acknowledged that, pro-inflammatory cytokines induce IR in endothelial cells, which may contribute to the development of the inflammatory infiltrate in psoriasis. Several recent observational studies propose children and adolescents with psoriasis may be at increased risk of being overweight and obese as well as having an increased risk for features of the metabolic syndrome. Subsequently, Gutmark and Shah [14] suggested: such an association raises the requirements of the long-term health implications for children and adolescents with psoriasis.

Discussion

Depending on the above-mentioned studies, it is clear that IR has also been indicated as an important contributing mechanism to the development of psoriasis itself, driving not only cardiovascular comorbidities, but also its cutaneous phenotype. Particularly, Buerger et al. [2] reported that IR directly contributed to the epidermal phenotype (hyperproliferation and altered differentiation of keratinocytes) seen in psoriasis. However, the strict clinical connection between psoriasis and metabolic diseases are as well underlined by analogies in their pathogenesis (chronic inflammation) viewing active factors secreted by adipose tissue (adipocytokines) and IR as drive linking points [11]. For example, adipocytokines such as leptin and adiponectin, which are able to adjust insulin sensitivity, are deregulated in a very similar way in both psoriasis and obesity. Other adipocytokines -apart from leptin- may also be involved in the association between IR and psoriasis. Omentin, a protein produced by visceral fat has a role in increasing insulin sensitivity [15]. According to Ismail [16], serum levels of omentin were found to be decreased in patients with psoriasis and negatively correlated with BMI and waist circumference. What is more, psoriasis patients also showed altered levels of further adipokines such as visfatin and resistin [17]. TNF-α, an anti-inflammatory molecule that affects insulin sensitivity, is one of the major actors of psoriasis pathogenesis as demonstrated by the efficacy of anti-TNF-α treatments in psoriasis [18].

Subsequently, some points should be considered in managing psoriasis patients: 1-Psoriasis per se may constitute a prediabetic condition. 2-Cytokines and other pro-inflammatory molecules inducing IR may represent attractive targets for novel anti-psoriatic therapies. 3-Better awareness, evaluation, and management of overweight and associated metabolic disease are necessary in this population. 4-Link between psoriasis and IR open the door for thiazolidinediones, a class of insulin-sensitizing drugs, to have verified promise for treatment of psoriasis.

Conclusion

Psoriasis appears to be intimately associated with IR which is itself able to influence keratinocytes' homeostasis and the pathogenesis of psoriasis. There are numerous molecular factors in charge for this close connection with adipose tissue. Adipokines may play a key role in both circumstances.

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