

Does metabolic syndrome influence psoriasis?

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Abstract. – The metabolic syndrome is a combination of diabetes mellitus type 2, hypertension, central obesity and combined hyperlipidemia. The metabolic syndrome and its components have been largely associated with psoriasis.

We report the case of a 66-year-old man affected with metabolic syndrome and psoriasis in which a multidisciplinary approach with endocrinologists and nutritionists led to an improvement of both conditions. After only 4 months of diet and an appropriate therapeutic regimen we observed an improvement of the hyperglycaemia, dyslipidemia, significant lose of weight, BMI switching from obesity to overweight and improvement of plaque psoriasis in absence of other treatments. We report this case to emphasise the need of a major control of the metabolic syndrome and associated comorbidities in psoriatic patients. Moreover we suggest that diet counselling and regular nutritional visits should be recommended in some patients to obtain dual benefits.

Key Words:

Metabolic syndrome, Psoriasis, Multidisciplinary approach.

Introduction

Metabolic syndrome is characterised by several factors including central obesity, atherogenic dyslipidaemia, hypertension and glucose intolerance¹. Currently this syndrome is considered as a strong predictor of cardiovascular diseases, diabetes and stroke²⁻³. The metabolic syndrome and its components have been largely associated with psoriasis¹.

It is possible that the first event that occurs is the onset of psoriasis, followed by lifestyle changes and depression associated with smoking or overeating. These habits can lead to the metabolic syndrome. In contrast it is possible that obesity favours psoriasis in predisposed individuals because of the pro-inflammatory state and release of inflammatory mediators such as adipocytokines. In this way obesity can be considered a risk factor for the development of other inflammatory disease including psoriasis⁴⁻⁵. In accordance with these observations we noticed, in our daily practice, that an adequate control and treatment of the metabolic syndrome can lead to an improvement of psoriasis.

Case Report

We report the case of a 66-year-old man affected with diabetes mellitus type 2 since the age of 60, hypertension in the last 3 years, obesity and psoriasis vulgaris. Onset of psoriasis was referred to be 15 years before and the patient reported a progressive exacerbation of the lesions in the last 2 years in association with worsening of the other comorbidities. The metabolic syndrome was under treatment with glibenclamide 5 mg, metformin 400 mg/3 daily, ramipril 2.5 mg and hydrochlorothiazide 12.5 mg once daily, without a good control. The psoriatic lesions were localised on the extremities and trunk with a psoriasis area and disability index (PASI) score of 5.6. The PASI score combines erythema, scales, and thickness with the size and distribution of the psoriatic lesions. We decided to follow a multidisciplinary approach in collaboration with our endocrinologists and nutritionists with the aim of: 1) improving the metabolic syndrome under a strict diet and nutritional intervention; 2) optimising the glycemic control and skin lesions

with a thiazolidinedione (characterised by euglycaemic and antiproliferative effects); 3) establishing whether the improvement of the metabolic syndrome induces a clinical response of the psoriatic lesions in absence of any other topical drug.

According to these proposals we added rosiglitazone maleate (4 mg/daily for 12 weeks and 6mg/daily for 4 weeks) to control the diabetes and a hypoglycaemic and hypocholesterolemic diet based on his body composition. Rosiglitazone is a synthetic ligand for the peroxisome proliferator-activated receptor-gamma (PPAR-gamma) receptor, insulin-sensitizing drug licensed for use in selected patients with type 2 diabetes mellitus⁶. Several reports suggest that oral thiazolidinediones may be beneficial for moderate chronic plaque psoriasis⁷.

Anthropometric parameters were measured according to standard methods: body weight and height. The patient was instructed to take off its clothes and shoes before performing all the measurements. Body weight (kg) was measured to the nearest 0.1 kg, using a balance scale (Invernizzi, Rome, Italy). Height (cm) was measured using a stadiometry to the nearest 0.1 cm (Invernizzi, Rome, Italy). The BMI was calculated using the formula: $BMI = \text{body weight (kg)} / \text{height (m)}^2$. BMI under 18.5 was considered under-weight; between 18.5 and 24.9 normal-weight, between 25 and 29.9 over-weight and of 30 or more, obese¹¹.

Early morning blood sample was taken and plasma was obtained by centrifugation at 1600 x g for 10 min at 4°C. The lipid profile, included plasma total cholesterol, high density lipoprotein-cholesterol (HDL), low density lipoprotein-cholesterol (LDL), and triglyceride concentrations, was determined through standard enzymatic colorimetric techniques (Roche Modular P800, Roche Diagnostics, Indianapolis, IN, USA), according to the manufacturer's procedures, with reagents provided by the same company. Analyses were carried out by the accredited Clinical Chemical Laboratories of the "Tor Vergata" Polyclinic of Rome, Italy.

After only 4 months of diet and the new therapeutic regimen we observed: 1) glycemic control improvement (HbA1c decreased from 9.5% to 5.6%); 2) improvement of dyslipidemia (cholesterolemia from 244 mg/dl to 189 mg/dl, triglyceridemia from 182 mg/dl to 115 mg/dl); 3) improvement of skin lesions achieving a PASI 50 (from a PASI score 5.6 to 2.1) in absence of oth-

er treatments; 4) significant lose of weight and BMI switching from obesity to overweight (from 97 kg to 87 kg and from 31.67 to 28.40 of BMI); 5) absence of side effects.

Discussion

We report the case of a patient affected with psoriasis in which an appropriate management of the associated metabolic syndrome improved significantly psoriasis in absence of any other treatment.

The metabolic syndrome is a combination of diabetes mellitus type 2, hypertension, central obesity and combined hyperlipidemia. It is not clear whether metabolic syndrome and its components are cause or consequence of psoriasis considering that patients with psoriasis change their lifestyle habits including nutrition and smoking⁹. It is possible that the first event that occurs is the onset of psoriasis, followed by lifestyle changes that include smoking and overeating, which leads to diseases that belong to the metabolic syndrome⁹. In contrast it is possible that obesity favours psoriasis in predisposed individuals because of the pro-inflammatory state and release of inflammatory mediators such as adipocytokines. In particular, obesity that is generally characterised by intra-abdominal fat, is not merely an inert mass but a vigorous endocrine organ capable of secreting multiple adipocytokines promoting inflammation and affecting glucose metabolism and vascular endothelial biology. Primary adipocytokines include interleukin (IL)-6, tumour necrosis factor (TNF)- α . The latter plays an important role in psoriasis. Pathogenesis of psoriasis is associated with an increased expression of TNF- α . Level of both TNF- α and TNF- α receptors are higher and more widely expressed in skin and serum of patients affected with psoriasis¹⁰. This cytokine has numerous effects on the immune response, driving activation and recruitment of other inflammatory cells, amplifying production of IL-1, IL-6, and IL-8, and activating nuclear transcription factors such as NF- κ B to propagate and maintain an inflammatory response¹⁰. The important role of TNF- α in psoriasis has been largely demonstrated by the efficacy of the anti- TNF- α biological therapy¹⁰. Serum TNF- α level increases with increasing BMI, induces insulin resistance, and causes endothelial cells to produce adhesion mol-

ecules with the subsequent adherence of monocytes. Thus, we can speculate that the lose of weight and the lower BMI herein observed was related to reduced levels of circulating TNF- α . The lower serum TNF- α could be responsible of the improvement of the psoriatic lesions in absence of any other treatment.

In conclusion, we observed that a reduction of the risk factors associated with the metabolic syndrome led to an improvement of psoriasis. Moreover we suggest that diet counselling and regular nutritional visits should be recommended in some patients. Finally although the efficacy of rosiglitazone is controversial in psoriasis⁷⁻⁸, its use should be considered in patients in which diabetes type 2 coexists with psoriasis to obtain dual benefits.

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