Psoriasis – an inflammatory skin disease

Psoriasis is a serious global health problem, with at least 100 million individuals affected worldwide (according to the World Health Organization Global Report on Psoriasis[1]).

The incidence varies between 0.09% and 11.43%, depending on geography and ethnicity. In Europe, Nordic populations are more affected than Medi-terranean, and a higher prevalence is observed in Cauca-sians than in Asians and African Americans[1]. Since the famous Greek doctor Hippocrates described the disease for the first time around 460 BC, doctors have been trying to unravel the mystery of its origins.

Today, they still don't know everything about the causes of psoriasis. The tendency to become ill lies in a genetic predisposition, but external/internal triggers or risk factors cause an outbreak. Only when they come into play, the skin cells are affected by inflammatory processes and begin to multiply uncontrollably. The dermatologic manifestations of psoriasis are varied and psoriasis vulgaris (also called plaque-type psoriasis) is the most prevalent type. Usually, the affected skin areas are outside of the elbows, knees or scalp, but can also appear on other locations – such as the eyelids, ears, mouth and lips, hands, feet and nails.

Clinical features, especially size and distribution of the psoriatic lesions, allow classification of psoriasis into plaque, guttate, pustular, and erythrodermic types[2–5]. The quality of life due to psoriasis can be very much decreased, causing reduced work productivity, physical disability, depression and impaired social relations[6,7].

Psoriasis – an inflammatory skin disease

Environmental factors can negatively influence the onset of symptoms and the severity of the disease. Usually, several risk factors trigger the onset of psoriasis, such as psychological stress, skin injuries, infections (e.g. Staphylococcus aureus, Helicobacter pylori, Candida sp. Streptococcus sp. or HIV), hormone fluctuations, medication (e.g. lithium, TNF inhibitors), smoking or alcohol consumption. Overweight – especially excessive abdominal fat that promotes inflammatory processes, is another important risk factor for psoriasis[8]. Obesity promotes a lowgrade inflammatory condition, whereby the adipose tissue is an active endocrine organ that has a key role in inflammation, glucose and lipid metabolism or insulin-mediated processes. The relationship between the two conditions seems to be bidirectional, with obesity predisposing to psoriasis and psoriasis favouring obesity[9].

Inflammation is not limited to the psoriatic skin, and has been shown to affect different organ systems. Psoriasis should therefore be regarded as a systemic entity rather than a solely dermatological disease. It is not surprising that doctors diagnose at least one accompanying disease in most of the patients. Individuals with psoriasis are very often affected by increased hyperlipidemia, hypertension, coronary artery disease, type 2 diabetes, increased BMI and metabolic syndrome. Diabetes and cardiovascular disease correlate with the severity of psoriasis and it seems that psoriasis increases risk for myocardial infarction and stroke[10–13]. Up to 35% of individuals with psoriasis develop chronic, inflammatory arthritis (psoriatic arthritis) leading to joint deformations and disability[14].

By adopting an appropriate anti-inflammatory lifestyle, psoriasis patients can themselves contribute to less frequent and less severe relapses. These include an appropriate diet, overweight reduction, avoidance of alcohol and cigarettes and the ability to cope with stress.

What happens in psoriatic skin lesions?

The hallmark of psoriasis is sustained inflammation that leads to uncontrolled keratinocyte proliferation, dysfunctional cell differentiation and an accelerated processes of skin renewal. In psoriasis, the immune system mistakenly targets the body's own cells, which causes an immunological overreaction, as it occurs in many other autoimmune diseases. An excessive number of new skin cells are constantly formed. Normally, the epidermis renews itself within four weeks, but in psoriatic patients this occurs in only three to four days.

What happens in the affected skin, however, can be observed thanks to modern scientific methods. Psoriasis is considered as a T-cell-mediated inflammatory skin disease. The T cells of the immune system (specialized white blood cells) and the socalled Langerhans cells (immune cells of the skin that are located in the lowest layer of the epidermis) play a key role. Langerhans cells sense any kind of "danger" and constantly monitor the environment of the skin for "unsafe" situations. Immune cells are then sent out to collect information about any "trespasser" and the body can decide to produce inflammatory signals to fight off the "attack" by creating an allergic reaction or forming scar tissue.

In a psoriatic patient, Langerhans cells take up the body's own cells, which are erroneously classified as dangerous (autoantigens). Then they migrate from the epidermis via the lymph stream to the lymph nodes and present the autoantigen to the T-cells. As soon as the T-cells have recognised the autoantigens via special receptors, they become active and release a whole series of cytokines as messenger substances. The result is an inflammation of the skin, with the typical symptoms of skin redness, swelling and itching, which is kept going by the constant activation of immune cells and messenger substances

Psoriasis – an inflammatory skin disease

(Interleukins such as IL1β, IL17, IL22 IL23, and TNF-α). When the body attempts to heal the inflamed area, the cells of the skin (keratinocytes) multiply excessively. They migrate too fast from the lowest to the uppermost skin layer. This excessively rapid cell renewal leads to an accumulation of immature cells on the skin surface and the formation of a large number of scales that appear as patches or plaques[2,10,13,15].

In this reasoning, treatment of psoriasis must consider the underlying causes and eliminate or alleviate the body's systemic inflammatory load. This can be achieved by nutritional adjustments, for example according to the results of the ImuPro test.

Nutrition plays a crucial role in psoriasis.

Nutritional and lifestyle counselling must play a central role in an integrated therapeutic approach for psoriatic patients. They should actively participate to prevent or reduce overweight, diabetes, cardiovascular and metabolic diseases or other conditions related with chronic inflammations. Hypocaloric diets were shown to be helpful to patients with psoriasis who are overweight or obese, leading to significant improvement in psoriasis severity[16–24]. However, the consensus regarding the nutritional strategies to be adopted still lacks in clinical settings.

A gluten-free diet can improve psoriatic symptoms.

A number of studies have examined the effect of a gluten-free diet (GFD) on psoriasis severity. The impact of a 3 month gluten free diet was evaluated in 33 psoriasis patients with elevated antigliadin antibodies (AGA), compared to 6 psoriasis patients without elevated AGA. Seventythree percent of the AGA-positive psoriasis patients showed an improvement in their psoriasis area and severity index (PASI), compared to none of AGA-negative psoriasis patients. After the GFD, AGA values were lower in 82% of the psoriasis patients who improved[25].

In another clinical trial with 28 patients, a gluten-free diet was shown to decrease the expression of tissue transglutaminase in psoriasis patients with AGA positivity[26]. Numerous other reports document the rapid resolution of skin lesions and clearance of the skin in psoriatic patients following a gluten-free diet[27–32].

In addition, many health care providers agree that a diet based on a test for delayed food hypersensitivities – such as ImuPro – is helpful for the treatment of patients suffering from psoriasis.

A few years ago, ImuPro undertook a clinical observation study, to collect evidence on the efficacy of ImuPro300 in patients with very different conditions that may indicate food intolerance, including psoriasis, neurodermatitis, headaches/migraines, overweight/obesity, fatigue, rheumatic diseases or gastroenterological complaints.

A total of 938 patients participated in this trial and eliminated IgG positive foods for a period of 8 weeks. Initially, 201 patients indicated that they suffered from psoriasis. In the control documentation, which took place after approximately 8 weeks, the severity of psoriatic symptoms was significantly lower in 118 of the 201 patients (59.2%). Comparable improvements were also found for other inflammatory skin diseases such as acne, neurodermatitis or itchiness (60.8%, 66.9% and 72.5%, respectively)[33].

The results of the ImuPro observational study show that an individualized anti-inflammatory nutrition should be a considered as first line intervention, affecting disease severity and management of patients with psoriasis.

ImuPro
Right Food. Better Health.

Psoriasis – an inflammatory skin disease

References

1. World Health Organization. Global report on Psoriasis. Glob. Rep. Psoriasis (2016).
2. Ogawa, E., Sato, Y., Minagawa, A. & Okuyama, R. Pathogenesis of psoriasis and development of treatment. J. Dermatol. 45, 264–272 (2018).
3. Lee, E. B., Wu, K. K., Lee, M. P., Bhutani, T. & Wu, J. J. Psoriasis risk factors and triggers. Cutis 102, 18–20 (2018).
4. Brandon, A., Mufti, A. & Gary Sibbald, R. Diagnosis and Management of Cutaneous Psoriasis: A Review. Adv. Skin Wound Care 32, 58–69 (2019).
5. Christophers, E. & van de Kerkhof, P. C. M. Severity, heterogeneity and systemic inflammation in psoriasis. J. Eur. Acad. Dermatol. Venereol. 33, 643–647 (2019).
6. Korman, N. J., Zhao, Y., Pike, J. & Roberts, J. Relationship between psoriasis severity, clinical symptoms, quality of life and work productivity among patients in the USA. Clin. Exp. Dermatol. 41, 514–521 (2016).
7. Lim, D. S., Bewley, A. & Oon, H. H. Psychological Profile of Patients with Psoriasis. Ann. Acad. Med. Singapore 47, 516–522 (2018).
8. Fleming, P., Kraft, J., Gulliver, W. P. & Lynde, C. The Relationship of Obesity With the Severity of Psoriasis. J. Cutan. Med. Surg. 19, 450–456 (2015).
9. Carrascosa, J. M. et al. Obesity and psoriasis: inflammatory nature of obesity, relationship between psoriasis and obesity, and therapeutic implications. Actas Dermosifiliogr. 105, 31–44 (2014).
10. Boehncke, W.-H. & Schön, M. P. Psoriasis. Lancet (London, England) 386, 983–94 (2015).
11. Augustin, M. et al. Epidemiology and Comorbidity in Children with Psoriasis and Atopic Eczema. Dermatology 231, 35–40 (2015).
12. Vena, G. A. et al. Incidence of psoriasis and association with comorbidities in Italy: a 5-year observational study from a national primary care database. Eur. J. Dermatol. 20, 593–8
13. Rendon, A. & Schäkel, K. Psoriasis pathogenesis and treatment. Int. J. Mol. Sci. 20, 1–28 (2019).
14. Pariser, D. et al. A multicenter, non-interventional study to evaluate patient-reported experiences of living with psoriasis. J. Dermatolog. Treat. 27, 19–26 (2016).
15. Boehncke, W.-H. & Brembilla, N. C. Unmet Needs in the Field of Psoriasis: Pathogenesis and Treatment. Clin. Rev. Allergy Immunol. 55, 295–311 (2018).
16. Di Minno, M. N. D. et al. Weight loss and achievement of minimal disease activity in patients with psoriatic arthritis starting treatment with tumour necrosis factor α blockers. Ann. Rheum. Dis. 73, 1157–62 (2014).
17. Del Giglio, M., Gisondi, P., Tessari, G. & Girolomoni, G. Weight Reduction Alone May Not Be Sufficient to Maintain Disease Remission in Obese Patients with Psoriasis: A Randomized, Investigator-Blinded Study. Dermatology 224, 31–37 (2012).
18. Jensen, P. et al. Long-term effects of weight reduction on the severity of psoriasis in a cohort derived from a randomized trial: a prospective observational follow-up study. Am. J. Clin. Nutr. 104, 259–65 (2016).
19. Naldi, L. et al. Diet and physical exercise in psoriasis: a randomized controlled trial. Br. J. Dermatol. 170, 634–642 (2014).
20. Guida, B. et al. Energy-restricted, n-3 polyunsaturated fatty acids-rich diet improves the clinical response to immuno-modulating drugs in obese patients with plaquetype psoriasis: a randomized control clinical trial. Clin. Nutr. 33, 399–405 (2014).
21. Al-Mutairi, N. & Nour, T. The effect of weight reduction on treatment outcomes in obese patients with psoriasis on biologic therapy: a randomized controlled prospective trial. Expert Opin. Biol. Ther. 14, 749–756 (2014).

Psoriasis – an inflammatory skin disease

22. Debbaneh, M., Millsop, J., Bhatia, B., Koo, J. & Liao, W. Diet and Psoriasis: Part I. Impact of Weight Loss Interventions. J. Am. Acad. Dermatol. 1, 133–140 (2014).
23. Zuccotti, E. et al. Nutritional strategies for psoriasis: current scientific evidence in clinical trials. Eur. Rev. Med. Pharmacol. Sci. 22, 8537–8551 (2018).
24. Barrea, L. et al. Environmental risk factors in psoriasis: The point of view of the nutritionist. Int. J. Environ. Res. Public Health 13, (2016).
25. Michaëlsson, G. et al. Psoriasis patients with antibodies to gliadin can be improved by a gluten-free diet. Br. J. Dermatol. 142, 44–51 (2000).
26. Michaëlsson, G., Ahs, S., Hammarström, I., Lundin, I. P. & Hagforsen, E. Gluten-free diet in psoriasis patients with antibodies to gliadin results in decreased expression of tissue transglutaminase and fewer Ki67+ cells in the dermis. Acta Derm. Venereol. 83, 425–9 (2003).
27. Addolorato, G. et al. Rapid Regression of Psoriasis in a Coeliac Patient after Gluten-Free Diet. Digestion 68, 9–12 (2003).
28. Frikha, F., Snoussi, M. & Bahloul, Z. Osteomalacia associated with cutaneous psoriasis as the presenting feature of coeliac disease: a case report. Pan Afr. Med. J. 11, 58 (2012).
29. De Boer, W. A. & Tytgat, G. N. A patient with osteomalacia as single presenting symptom of gluten-sensitive enteropathy. J. Intern. Med. 232, 81–5 (1992).
30. Pona, A., Haidari, W., Kolli, S. S. & Feldman, S. R. Diet and psoriasis. Dermatol. Online J. 25, (2019).
31. Wolters, M. [The significance of diet and associated factors in psoriasis]. Hautarzt. 57, 999–1004 (2006).
32. Wolters, M. Diet and psoriasis: experimental data and clinical evidence. Br. J. Dermatol. 153, 706–714 (2005).
33. Observational study ImuPro300: Gewichtsprobleme und Nahrungsmittelunverträglichkeiten [German]. Evomed MedizinService GmbH, Darmstadt (2008).

More case reports on our website: http://imupro.com