Insights in Stem Cells

Extended Abstract
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Stem Cell Congress 2019- Benchmarking Stem Cells and Transplantation in Psoriasis- Damiani G-Study Center of Young Dermatologists Italian Network

Damiani G [1,2,3*], Berti E [2], Pigatto PDM[3], Franchi C[3], Asa'ad F[4], Fiore M[5], Colombo D [6], Gronchi S6, Malagoli P [7] and Piccinno R [8 1] Study Center of Young Dermatologists Italian Network (YDIN), GISED, 24122, Bergamo, Italy [2] Dipartimento di Fisiopatologia Medico-Chirurgica e dei Trapianti, Universita' degli Studi di Milano, Unita' Operativa di Dermatologia, IRCCS Fondazione Ca' Granda, Ospedale Maggiore Policlinico, 20122, Milano, Italy [3] Clinical Dermatology, Department of Biomedical, Surgical and Dental Sciences, IRCCS Galeazzi Orthopaedic Institute, University of Milan, 20126, Milan, Italy [4] Department of Biomedical, Surgical & Dental Sciences, University of Milan, 20122, Milan, Italy [5] Department of Women, Child and General and Specialized Surgery, University of Campania "Luigi Vanvitelli", 80138 Naples, Italy [6] Study Center of Young Dermatologists Italian Network (YDIN) and Private practice, via Livigno, Milan [7] Unit of Dermatology, Azienda Ospedaliera San Donato Milanese, Milan, Italy [8] Servizio di Fotoradioterapia, IRCCS Fondazione Ca' Granda, Ospedale Maggiore Policlinico, 20122, Milano, Italy

Abstract

Psoriasis is a chronic systemic inflammatory disease with several abnormalities in hematopoiesis. During the last 30 years, the science of stem cells has been intensively studied, also in the field of psoriasis, to discover therapeutic modalities by reversing the imbalance of lymphopoiesis. In fact, the results available in the literature have reported a remission of psoriasis after a stem cell transplant. This review summarizes current knowledge on psoriasis, stem cells and transplantation.

Psoriasis is characterized by scaly, infiltrating, erythematous and broad-delimited lesions. Despite the reported prevalence of 3% in the general population, the pathogenesis of psoriasis is still poorly understood. A growing body of evidence has linked psoriasis to multiple comorbidities affecting different tissues, other than the skin, such as: the lungs, the cardiovascular and digestive systems. Although psoriasis is easily diagnosed by clinical examination, associated comorbidities still require less invasive diagnostic instrumental approaches. It is possible to better understand the predisposition to psoriasis by studying the inflammatory microenvironment, in which stem cells seem to play a key role. In fact, various reports and studies over the past 10 years have pointed out that: a) several abnormalities in stem cells are present in psoriatic patients, b) the beneficial role of stem cell transplantation, suggesting the possibility of bone marrow transplant as a future therapeutic modality for permanently curing the disease in refractory cases. In this context, this review aims to summarize the current knowledge and evidence concerning stem cells, their transplantation and their applications in animal models and the future perspectives on their therapeutic role in psoriasis.

Stem Cells in Psoriasis

Sharpe and Ferguson (1988) were the first to suggest the role of stem cells in psoriasis, concluding that embryonic palate cells could differentiate through soluble mesenchymal factors and extracellular matrix molecules. The authors suggested that an imbalance in these signaling molecules could lead to cancer and autoimmune diseases such as psoriasis. By analyzing the genetic profile of stem cells from psoriatic patients, Campanati et al. highlighted a similar imbalance between the Th1 / Th17 and Th2 pathways commonly found in blood and skin samples. In addition, Charruyer et al. describes both in the mouse model and in humans that psoriatic stem cells increase the rate of asymmetric cell division in a manner dependent on IL-17A. These results may suggest that stem cells may play a key role in the initiation and maintenance of psoriasis.

Embryonic stem cells (ESCs)

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Embryonic stem cells (ESC) are immortal cells that make up the basal layer of the epidermis. During stimuli, ESCs transform into self-renewable cells and transient amplification cells (TA) which undergo differentiation after certain other divisions, even if they have a limited proliferation capacity. Characteristically, the epidermis proliferates excessively in psoriasis, causing an increase in the rate of renewal up to 5 times. The inflammatory microenvironment and genetic sensitivity are manifested by altered ESC and TA phenotypes. In particular, the profiled cells Keratin 1 / Keratin10- and β1- Integrin +, namely ESC and TA, showed an increase in the division rate, confirmed by an overexpression of the fatty acid binding protein 5 (FABP5) and Nestin, representing TA and ESC, respectively [19]. Therefore, hyperplasia of the psoriatic epidermis is the result of an increase in the ESC / TA compartment due to TH17-related cytokines, such as interleukin (IL) -17 and IL-22.

Conclusions:

Stem cell abnormalities, their relationship to peripheral blood cells, and the increased number of comorbidities associated with psoriasis highlight the multisystem inflammatory nature of this disease. The dysfunctions of medullary hematopoiesis could explain the abnormal activation of Th1-Th17 previously described in the literature, at the same time offering a possible strategy for treating psoriasis in a safe and effective therapeutic model. However, a better understanding of the pathogenesis of psoriasis is required to better design future trials regarding stem cell transplantation.

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dr.giovanni.damiani@gmail.com

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