Arthritic psoriasis following natalizumab treatment: a case report and review of the literature

Foschi M 1, *Vacchiano V 1, Sabattini L 2, Scandellari C 2, Mancinelli L 2, Lugaresi A 1*

1) *Department of Biomedical and Neuromotor Sciences – Alma Mater Studiorum – University of Bologna – Bologna.*

2) IRCCS Institute of Neurological Sciences of Bologna – Bologna.

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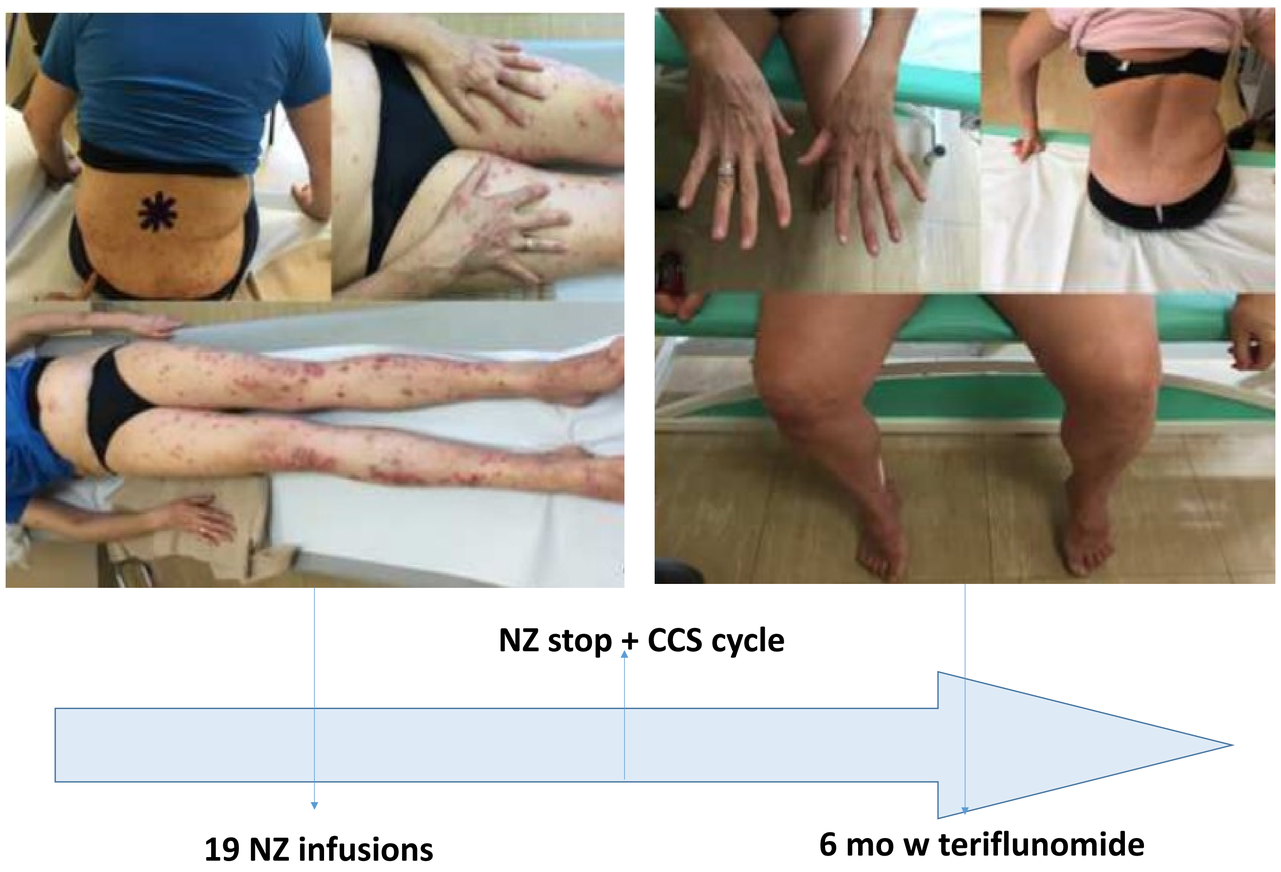
Introduction

Psoriasis is a chronic immune-mediated dermatosis, affecting approximately 2% of the world's population. Several studies have shown an higher incidence of psoriasis in Multiple Sclerosis (MS) patients. It is already estabilished that some MS drugs can trigger new-onset psoriasis or the exacerbation of pre-existing skin psoriatic lesions. The role of interferon beta in new-onset or exacerbation of cutaneous psoriasis have been reported in the past, whereas only a case of severe disseminated psoriasiform eruption has been described during natalizumab, to our knowledge.

Case report

We report the case of 41-years-old patient affected by Relapsing Remitting Multiple Sclerosis (RRMS) and with a positive family history for psoriasis, who presented with new onset psoriatic arthritis after 19 natalizumab (NZ) infusions. Possible alternatives to natalizumab treatment active on psoriatic arthritis as well, are represented by methotrexate and teriflunomide, whereas the use of rituximab is still controversial. Ponesimod might represent a future treatment for both diseases. The use of ocrelizumab, an anti-CD20 monoclonal antibody, ustekinumab, a human anti-interleukin-12/23 monoclonal antibody, secukinumab, a monoclonal antibody against IL-17A, or other S1P inhibitors, such as fingolimod and siponimod should also be explored, as several case reports suggest efficacy on both psoriasis and MS.



Litterature review

DMD (MS)	Authors	Cutaneous AE	Pathophysiological mechanism
Interferon beta	- *Lòpez-Lerma L et al (2009)a*	- scaly and erythematous plaques on trunk and limbs.	↑ IL-23 Th17 pathway, activating granulocyte recruitment and the secretion of pro-inflammatory factors in dermis. ↓ IL-17 enhances keratinocyte proliferation and inhibits keratinocyte differentiation. Then they in turn promote Th17 cell recruitment.
	- *La Mantia L et al (2010)b*	- exacerbation of cutaneous psoriasis and new appearance of arthritis.	
	- *Kolb-Mäurer A et al (2015)c*	- new onset of cutaneous psoriasis (family history +)	
Natalizumab	- *Piqué-Duran E et al (2013)d*	- acquired perforating dermatosis (APD, mummular eczema with umbilicated papules)	***Unknown???*** alfa4 integrin blocking could cause a paradoxical exacerbation of flogosis *(animals models of chronic inflammatory bowel disease)*f
	- *Millán-Pascual J et al (2012)e*	- severe disseminated psoriasis (history of mild cutaneous psoriasis)	

References

DMD = disease modyfing drugs; **MS** = multiple sclerosis; **AE** = adverse event.

a = López-Lerma I, Iranzo P, Herrero C (2009) New-onset psoriasis in a patient treated with interferon beta-1a. Br J Dermatol. Mar; 160(3):716-7. **b** = La Mantia L, Capsoni F (2010) Psoriasis during interferon beta treatment for multiple sclerosis Neurol Sci. Jun; 31(3):337-9. **c** = Kolb-Mäurer A, Goebeler M, Mäurer M (2015) Cutaneous Adverse Events Associated with Interferon-β Treatment of Multiple Sclerosis. Int J Mol Sci. Jul 2;16(7):14951-60. **d** = Piqué-Duran E, Eguía P, García-Vázquez O (2013) Acquired perforating dermatosis associated with natalizumab. J Am Acad Dermatol. Jun;68(6):e185-7. **e** = Millán-Pascual J1, Turpín-Fenoll L, Del Saz-Saucedo P, Rueda-Medina I, Navarro-Muñoz S (2012) Psoriasis during natalizumab treatment for multiple sclerosis. J Neurol. Dec;259(12):2758-60**. f**= Bjursten M1, Bland PW, Willén R, Hörnquist EH (2005) Long-term treatment with anti-alpha 4 integrin antibodies aggravates colitis in G alpha i2-deficient mice. Eur J Immunol. Aug;35(8):2274-83.