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REVIEW ARTICLE OPEN ACCESS

BOTULINUM TOXIN IN PSORIASIS: A REVIEW

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ABSTRACT

Psoriasis is a chronic inflammatory disease involving the exacerbated production of keratinocytes in the affected areas, in response to the activation of the immune system. Some neurotransmitter channels are involved in the pathogenesis of psoriasis and may act as a trigger for the pathology. This article aims to demonstrate the use of Botulinum Toxin A as a substance capable of interfering in the immunopathogenesis of the disease.A bibliographic review was carried out using articles indexed in the MEDLINE AND LILACS platforms, in both English and Portuguese. Review articles were not included. Cutaneous nerves in lesions can activate the production of IL-23 by dermal dendritic cells, triggering the expression and release of interleukin (IL)-17 – the major inflammatory response factor – by T cells. Botulinum Toxin A, acts directly in this tract.Improvement reports show the applicability of botulinum toxin as a safe and practical treatment option for daily clinic practice.

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INTRODUCTION

Psoriasis is a chronic inflammatory disease, characterized by the presence of erythematous-scaly plaques and joint changes of varying degrees of intensity that affects a large part of the world population, about 3%. (Ji; Liu, 2019). Histologically, it displays exacerbated proliferation of keratinocytes in affected areas, as a response to the immune system activation by the action of T lymphocytes. (Chiricozzi, *et al*., 2018). In addition, other factors, such as environmental and genetics, may be intrinsically involved in the process of disease development and the formation of inflammatory plaques.(Pen Chen, *et al*., 2012). It is possible to verify the presence of an intense inflammatory process in the affected area, with accumulation of immune system cells; this hinders the healing process and impairs the skin's barrier function.(Baumbauer, *et al*., 2015). Also noteworthy are the neurocutaneous pathways in the pathogenesis of psoriasis and with dermatome improvement of plaque psoriasis after lesions that compromised some branches of the peripheral nervous system (González, *et al*., 2020). An increasing amount of evidence points to the critical role of the cutaneous nervous system in the initiation and maintenance of psoriatic skin lesions from neurogenic inflammation. However, the molecular mechanisms that affect cutaneous neurons are largely uncharacterized. : Cutaneous nerves present in the lesions can activate the production of IL-23 from dermal dendritic cells, triggering the expression and release of interleukin (IL)-17 – the major inflammatory response factor – by T cells (Romhányi *et al*., 2022). Several treatments have been instituted to effectively improve psoriasis. Currently, the role of botulinum neurotoxins (BoNTs), which have effects on different types of human cells, is described as effective, opening a wide spectrum for treatments. (Szlavicz *et al*., 2017) Thus, this article aims to demonstrate the applicability of botulinum toxin as a therapeutic alternative capable of interfering in the immunopathogenesis of the disease in question.

METHODS

A bibliographic review was carried out in MEDLINE, LILACS indexed databases, using the following as descriptors: Botulinum Toxin; Neurotransmitters; Psoriasis. The search languages were English and Portuguese, review articles being the basic exclusion factor. Articles were included regardless of year of publication, as kibg as they were relevant to this review.

DISCUSSION

Psoriasis is a chronic inflammatory skin disease where interleukin (IL)-17 is the major factor in the inflammatory response. Cutaneous

nerves can activate the production of IL-23 from dermal dendritic cells, and IL-23 triggers the expression and release of IL-17 by T cells (Riol-Blanco, *et al*., 2014).Therefore, abnormalities related to the peripheral nervous system may be important to understand the pathological mechanism of psoriasis (Furue, *et al*., 2019). Patients with psoriasis have macroscopically healthy skin in non-injured areas, however, these areas already carry changes that, in combination with other stimuli lead, to the appearance of symptoms (Szlavicz, *et al*., 2017). Thus, one of the widely known characteristics of NL skin is the Köbner phenomenon, the development of lesions in response to mechanical provocations or stress. (Ji; Liu, 2019) due to heightened immune response and increased proliferation of keratinocytes (Szabó, *et al*., 2014). The largest search for a significant treatment for this pathology is associated with the quality of life of its sufferers. The impact of psoriasis on patients' quality of life is profound and is well documented in the scientific literature. (Bulat *et al*., 2020) New treatments, whether associated or not, have been reported. This is the case of BTxBoA, whose efficacy is attributed to the role of the nervous system in psoriasis, which demonstrated a high concentration of nerve fibers in the psoriatic skin and an increase in the level of CGRP and SP derived from sensory nerves (Sandoval-Talamantes *et al*., 2020).

BoNT-A inhibits nerve-derived CGRP and SP release, and this likely explains the subjective clinical observation of disease improvement in psoriasis after BoNT-A administration by Zanchi *et al*., (2008). Therefore, the clinical observation that psoriasis remits after loss of innervation, nerve function, or nervous system injury supports this hypothesis (Ward *et al*., 2012). In 2008, Zanchi *et al*., used ONA (50-100 units, depending on the extent and severity of psoriasis) to reduce sweating and inflammation in 15 patients with inverse psoriasis (Zanchi *et al*., 2008). From the results obtained, treatment was successful in 87% of cases. Ward *et al*., (2012) demonstrated that botulinum toxin can induce psoriasis remission in the transgenic keratinocyte-Tie2 (KC-Tie2) mouse model of psoriasis. As early as 2014, Gilbert, *et al*., demonstrated local clearance of plaque psoriasis after an off-label intradermal injection of BoNT-A in a single patient with recalcitrant disease, with complete elimination of plaques. From these studies and on the basis of current findings, the use of botulinum toxin BoNT-A has become a practical approach for patients with small lesion psoriasis or with focal lesions that are recalcitrant to standard therapy.(Schlessinger, *et al*., 2017). In 2020, Gonzalez *et al*., found similar data, confirming the results of Gilbert, *et al*., (González, *et al*., 2020). Li, *et al*., (2021) reiterated blocking action. Accumulating evidence suggests that botulinum neurotoxins (BoNTs) that inhibit the release of acetylcholine may be used in the treatment of plaque psoriasis. (Li Q, *et al*., 2021). Botulinum toxin is currently being used as a new, safe, single-injection, and effective therapy for plaque-type psoriasis. (Khattab, Samir, 2021)

CONCLUSION

Some studies have shown that neurotransmitters are involved in the pathogenesis of numerous skin diseases, including psoriasis. Psoriasis improvement from the use of botulinum toxin has been reported as a safe and practical treatment, and so is an option for the daily clinical practice.

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