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Case Report OMICS International

Treating Multiple Sclerosis with an Anticholinergic Drug Causes Changes in the Skin

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Abstract

Benztropine, an anticholinergic drug, caused a number of skin changes only in the areas of the body that previously had demonstrated clinical symptoms of multiple sclerosis (MS). These changes included erythema, telangiectasias, non-pitting edema and flaky/scaly skin. Despite continuation of the benztropine, the skin changes eventually resolved. However, a few months later, minimal erythema and swelling of the joints recurred. The pathophysiological events that could be inducing these changes are discussed.

Keywords: Multiple sclerosis; Skin changes; Treatment; Remyelination

Introduction

Multiple sclerosis is a chronic autoimmune disease of the central nervous system characterized by demyelination [1]. Transient repair of the myelin sheath can occur during the course of this disease [2] and is thought to be due to promotion of oligodendrocyte precursor cell differentiation [3]. Recently, it was documented that benztropine (Cogentin, Qualitest Pharmaceuticals, Huntsville, AL), an anticholinergic drug, can significantly decrease the clinical severity of experimental autoimmune encephalomyelitis (EAE), an animal model for MS. Benstropine can improve the symptoms of EAE by inducing remyelination [4]. To determine whether benztropine also can lead to remyelination in humans, this drug was prescribed for an individual with recurrent remitting MS. Since this drug currently is FDA approved for the treatment of Parkinson's disease, human subject's committee approval was not necessary.

Observation

The proband, which has a 34 year history of MS, has been treated with benztropine since 2013. Treatment with benztropine caused remarkable changes in the skin but only in those areas previously affected by the MS. These changes followed a clear pattern. First, the skin became erythematous and demonstrated telangiectasia. A month or two later, the erythematous areas became purple (Figure 1). In the areas most affected by the MS, oozing of serum through the stratum corneum occurred. After a few more weeks, some of the purple patches became white and these areas were cold both subjectively and objectively, indicating vasoconstriction. This was followed by non-pitting edema. During or immediately after the purple phase, some of these areas developed fine, white flakes/scales (Figure 2). The growth of the nails and hair in these areas also was inhibited. Finally, the purple patches turned blue. Rarely, in areas of the body affected minimally by the MS, the skin did not demonstrate the early changes described above, but only showed the blue patches. After the blue phase, all of the skin changes resolved. The severity of the skin changes mimicked the severity of the nerve damage/impairment the MS had caused.

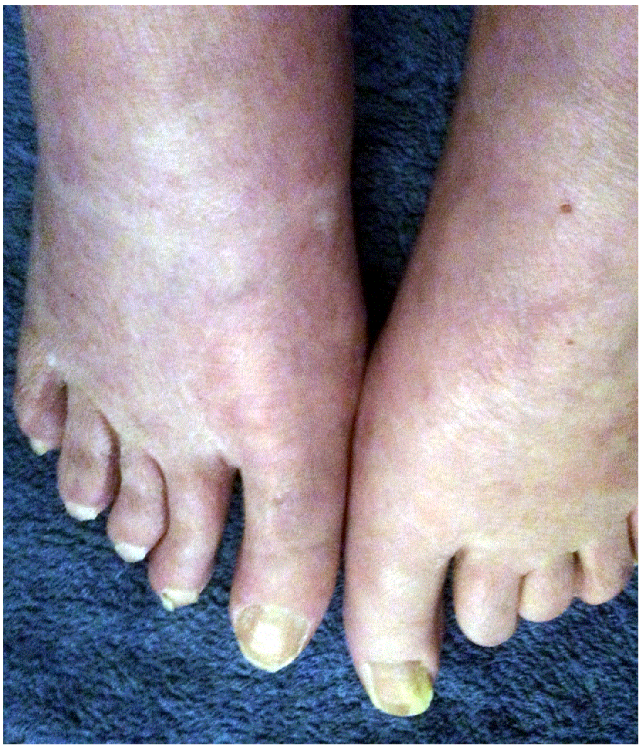

Figure 1: The feet demonstrate purple discoloration, fine scale and edema.

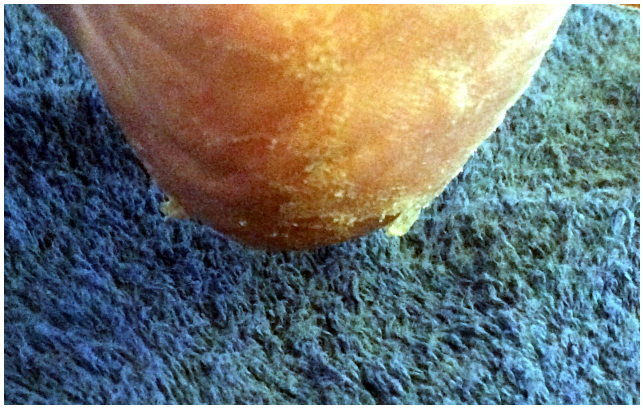

Figure 2: Flakes of skin on the heel

Discussion

Several factors could be playing a role in the pathophysiology of the changes in the skin documented above. For example, benztropine could stimulate keratinocytes that are known to express acetyl choline receptors to synthesize vascular endothelial growth factor which, in turn, could account for the vascular changes seen in the affected areas of the integument [5,6]. Keratinocytes also could be responsible for synthesizing and releasing nitric oxide, a substance that could cause vasodilation and production of epithelial growth factor.

References

1. Steinman L (1996) A few autoreactive cells in an autoimmune infiltrate control a vast population of nonspecific cells: A tale of smart bombs and the infantry. Proc Natl Acad Sci 93: 2253-2256.
2. Patel JR, Klein RS (2001) Mediators of oligodendrocyte differentiation during remyelination. FEBS Lett 23: 3730-3737.
3. Patel JR, McCandless EE, Dorsey D, Klein RS (2010) CXCR4 promotes differentiation of oligodendrocyte progenitors and remyelination. Proc Natl Acad Sci 107: 11062-11067.
4. Deshmukh VA (2013) A regenerative approach to the treatment of multiple sclerosis. Nature 502: 727-732.
5. Grando SA (2012) Muscarinic receptor agonists and antagonists: effects on keratinocyte functions. Handb Exp Pharmacol 208: 429-450.
6. Adase CA, Borkowski AW, Zhang LJ, Williams MR, Sato E, et al. (2016) Non-coding double-stranded RNA and antimicrobial peptide LL-37 induce growth factor expression from keratinocytes and endothelial cells. J Biol Chem 291: 11635-11646.