

BOTULINUM TOXIN-TYPE-A (BoNT-A) IMPROVES HORMONAL INDEX OF PHYSICAL PERFORMANCE IN WOMEN AFFECTED BY CHRONIC MIGRAINE (CM)

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BoNT-A is a focally acting neurotoxin that inhibits the release of acetylcholine and other neurotransmitters from presynaptic nerve endings. There is an emerging scientific and clinical evidence to support advantages and limitations of BoNT-A use in the pharmacological prevention of CM. The connection between CM and stressful events highlights a real need of scientific data regarding stress hormones variations and their regulation inside of this very disabling disorder (1). Since it has been recently postulated that DHEA-S might moderate the effect of glucocorticoids and protect neurons from their neurotoxicity, we measured salivary cortisol level both as absolute marker of hypothalamus-pituitary-adrenal (HPA) axis activity, and in its relation with the concomitant release of salivary DHEA-S and testosterone in 20 women with chronic migraine in comparison to 20 healthy women. Moreover, 10 women with chronic migraine were treated subcutaneously with 100 IU BoNT-A. The present study shows that injection of BoNT-A counteracted the CM-induced cortisol increase. Neither Testosterone nor DHEA-S were modified during CM, while both hormone levels were significantly lower than placebo one month after BoNT-A. The increased Cortisol to DHEA-S ratio observed in CM patients and not modified by BoNT-A treatment, suggested that the antiglucocorticoid action exerted by DHEA-S is not working in CM disease. Moreover, the lower Testosterone to Cortisol ratio measured in CM patients in comparison with controls might account for the tiredness, a symptom frequently reported by the CM patients.

Botulinum toxin injections, already hugely popular for removing wrinkles, could perhaps one day become a novel complementary therapy for migraines. Here we report that BoNT-A could counteract the CM-induced increase of cortisol, and consequently reduce the potentially damaging overexposure to stress hormones. It is difficult to give a clear account of the mechanism of this action, we can only argue that BoNT-A could act by altering the working of pain-carrying neurotransmitters as well as relaxing muscles. Moreover, further studies are advised to establish whether the HPA derangement in CM has not only a pathogenetic relevance but can also be considered as a target of novel treatment strategies more oriented to improve well-being of the migraineur not only by symptomatic therapies.

1. Patacchioli F.R. Monnazzi P., Simeoni S., De Filippis S., Salvatori E., Coloprisko G. and Martelletti P. (2006) Salivary cortisol, DHEA-S and Testosterone in Chronic Migraine. *J. Headache & Pain* 7: 90-94.