BoNT-A on acetylcholine release from parasympathetic nerves.

In this paper, Dr. Silva and coworkers elegantly provide evidence that adrenergic innervation of the prostate might have an effect on prostatic involution after BoNT-A injection [3]. This interesting new finding indirectly corroborates previous evidence indicating that certain alpha 1 blockers can affect the dynamics of prostate growth by changing the balance between prostate cell proliferation and apoptosis, even if the clinical significance of this evidences remain unclear [4].

Of course, there are two major limitations of this work, as recognized by the authors themselves: the rat prostate is widely different from the human prostate, and the study is performed in a nonpathological experimental model. Thus, many issues regarding the botulinum effect on neuronal pathways of the prostate, and more in general on the lower urinary tract, remain to be addressed. Among them, emerging data suggest an important role of BoNT-A in modulating sensory and antinociceptive mechanisms, which affect bladder urgency, pain, and
inflammation. And we are now well aware of a link between inflammation and benign prostatic hyperplasia [5].

What are the real clinical implications of the findings from Silva et al’s paper, then? Well, it is too early to say. But we are on the right path to answering this intriguing question, even though considerable clinical and basic science work still needs to be performed. Currently, all botulinum toxin use for urological conditions is off-label and unlicensed; therefore, caution should be exercised until future studies are reported.

Transforming a lethal poison into a modern, effective, minimally invasive therapeutic weapon remains one of the current exciting challenges of urological research.

References


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