A case of idiopathic lower limb dystonia treated with combined use of botulinum toxin type A and phenol nerve block

Edoardo Bianchini¹, A. Massimiani¹, J. Bemporad², M. Giovannelli¹

Introduction: Among focal dystonias, lower limb dystonia (LLD) is rather rare [1]. The most effective treatment is botulinum neurotoxin type A (BoNTA), but the evidence for LLD is less robust [2]. Nerve block (NB) is a chemodenervation technique used successfully in spasticity [3], but evidence for dystonia is very scarce [4].

Objective: Here we report a case of focal LLD successfully treated with BoNTA and NB combined.

Methods: A 50-year-old man, former long-distance runner, developed abnormal posture of the right foot and toe in 2014, which initially occurred only during running and was not elicited by other exercises, but over time also affected normal walking and persisted at rest. Physical examination showed inversion, plantarflexion, and internal rotation of the right foot and hyperextension of the toe at rest, with worsening during walking. No other neurological symptoms were found. Instrumental investigations were normal, and other causes of LLD were excluded. The patient underwent Abobotulinumtoxin-A therapy with initial improvement but progressively reduced response since 2018, despite dosage increase. The patient had also tried oral medications and physiotherapy without significant improvement. Ultrasonographic evaluation showed altered echogenic pattern of the right leg muscles.

Results: To test the hypothesis of a structural muscle alteration affecting the effectiveness of BoNTA [5], a diagnostic NB of the tibial nerve with 2.5% lidocaine injection was performed. After the procedure, foot internal rotation and plantarflexion improved and a therapeutic NB with 5% phenol was performed. Toe hyperextension was successfully treated with Abobotulinumtoxin-A. The combined treatment resulted in marked improvement with a lasting response that remained consistent with repeated treatment.

Conclusions: The case presented here suggests that NB might be a viable option, in patients with focal LLD refractory to BoNTA treatment, to assess residual response to chemodenervation and to help achieve effective symptoms control before considering more invasive strategies.

References:

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¹ Department of Neuroscience, Mental Health and Sensory Organs (NESMOS), Sapienza University of Rome, Rome, Italy

² Department of Physical Medicine and Rehabilitation, San Giovanni Battista Hospital, ACISMOM, Rome, Italy

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