

Physiopathology of neurogenic detrusor overactivity: Role of neurotrophins, inflammation and extracellular matrix according to the neurological disease

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Introduction & Objectives: The aim of this study was to evaluate the implication of neurotrophines (ngf, bdnf), mediators of inflammation (tgf β -1, pge-2) and the restructuring of the extracellular matrix (timp-2) in the physiopathology of neurogenic detrusor overactivity of patients with spinal cord injury (SCI), spina bifida, multiple sclerosis (MS) or suprapontine lesions (Parkinson, cerebrovascular accidents).

Materials & Methods: A prospective monocentric study was conducted between March 2015 and March 2017. The included patients were more than 16 years old, with neurological disease, who underwent urodynamic examination for the assessment of their urinary tract impairments with detrusor overactivity diagnosed by the urodynamic evaluation. Urine samples from the first void/catheterization of the urodynamic test were collected and frozen at -80°C. At the end of the inclusion period, they were defrost and the urinary levels of ngf, bdnf, timp-2 and tgf-b1 were measured by dedicated ELISA kits. Results were corrected to urine creatinine.

Results: 63 patients were included: 20 with spinal cord injury, 8 with multiple sclerosis, 16 with spina bifida and 19 with suprapontine lesion. Urinary levels of pge-2/cr, timp-2/cr and tgf β -1/cr were similar in the 4 groups of neurogenic diseases (respectively p=0,72, p=0,82 and p=0,64). However, the average urinary level of ngf/cr was significantly higher in multiple sclerosis group compared to other neurologic populations (0,83, vs. 7,06 vs. 0,32 vs. 0,94 pg/ml; p=0,0009), as well for the average urinary level of bdnf/cr (0,05 vs. 0,14 vs. 0,06 vs. 0,03 ng/ml; p=0,048. figure 1). The urinary level of ngf/cr seemed to be inversely proportional to the EDSS score, but did not reached the significance threshold (r = -4,04; p=0,06).

Conclusions: Neurotrophin pathway, known to be damaged in multiple sclerosis, seems to be involved in the physiopathology of neurogenic detrusor overactivity for those patients with significantly higher urinary levels of ngf and bdnf than in other neurologic populations with neurogenic detrusor overactivity. This result, if confirmed, may have diagnostic and therapeutic consequences in the future.