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HYPO- AND HYPER-ACTIVE LUMBOSACRAL NERVES CAUSE URINARY DYSFUNCTION IN GUILLAIN-BARRÉ SYNDROME

Hypothesis / aims of study
To examine the frequency and pathophysiology of the lower urinary tract (LUT) dysfunction in Guillain-Barré syndrome (GBS).

Study design, materials and methods
Urinary symptoms were observed and neurological examinations made repeatedly during hospitalization of consecutive 65 patients with clinico-neurophysiologically definite GBS.[1] They were 41 men, 24 women; mean age, 41 years old; mean Hughs motor grade, 3; subtypes, acute inflammatory demyelinating polyneuropathy (AIDP), 28, acute motor axonal neuropathy (AMAN)[2], 37. Urodynamic studies consisted of uroflowmetry, measurement of residual urine, medium-fill water cystometry and external sphincter electromyography, which were performed according to the standards of International continence Society.

Results
LUT dysfunction was observed in 27.7% of GBS. LUT dysfunction was related with Hughs motor grade ($p<0.05$) but not with superficial or deep sensory disturbance. LUT dysfunction was related with defecatory dysfunction ($p<0.05$) but not with cardiovascular dysfunction. LUT dysfunction was more common in AIDP (39%) than in AMAN (19%), although frequency of severe LUT dysfunction (retention) was almost the same in AIDP and AMAN (10%). LUT dysfunction was negatively related with serum anti-ganglioside antibodies (GalNAc-GD1a IgG) ($p<0.05$). Urodynamic studies performed in 9 patients revealed urinary retention in 4, post-void residuals in 3 (mean 195 ml) among those who were able to urinate; decreased bladder sensation in one, detrusor overactivity (DO) in 8, low compliance in one (during storage); underactive detrusor in 7 (detrusor hyperreflexia with impaired contractile function [DHIC] in 5), detrusor- external sphincter dyssynergia in one, and detrusor- internal (sympathetic) sphincter dyssynergia in one (during voiding). No evidence of central nervous system involvement was found in these patients.

Interpretation of results
Although bladder paralysis has been regarded a characteristic bladder feature in GBS,[2] in the present study DHIC was also common. Some patients also showed hyperactive urethral sphincter. These abnormalities suggest a combination of both hypo- and hyper-function of the sacral autonomic pathways innervating the LUT. Two mechanisms are postulated for the LUT dysfunction in GBS. The first is damage in the lumbosacral autonomic fibers with irritation or ephaptic transmission, from either bystander inflammation or immune attack of the autonomic fibers. The second is immune attack of the inhibitory spinal cord interneurons, which secondarily leads to lumbosacral autonomic hyperactivity. These notions are in line with the pathology studies showing moderate to severe loss of small myelinated fibers and inflammatory cell infiltration in the lumbosacral spinal roots, sympathetic chain, and spinal cord in GBS.[3]

Concluding message
In our series of 65 GBS cases, 27.7% of the patients had LUT dysfunction including urinary retention. Underactive detrusor, DHIC, and to a lesser extent, hyperactive urethral sphincter are the major urodynamic abnormalities. The underlying mechanisms of LUT dysfunction appeared to involve both hypo- and hyper-active lumbosacral nerves.

References
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