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DETRUSOR SPHINCTER DYSSYNERGIA OF PUTATIVE PERIPHERAL NERVE ORIGIN

Hypothesis / aims of study

Detrusor sphincter dyssynergia (DSD) has been regarded the symptom of spinal cord lesion between the pontine micturition centre (PMC) and the sacral segment. In contrast, although uncommon, DSD is known to occur in peripheral nerve (PN) lesion. We therefore investigated DSD of putative peripheral nerve origin by both sphincter electromyography (EMG) and urodynamic study (UDS).

Study design, materials and methods

We had 144 subjects who underwent both sphincter EMG and standard UDS. Method of sphincter EMG was in accordance with that by Fowler et al. UDS was performed according to the International Continence Society standards. Nuclear/infranuclear peripheral nerve (PN) dysfunction was indicated as abnormal sphincter EMG.

Results

144patients were classified into 71 with PN dysfunction and 73 without PN dysfunction. DSD was found in 22 of 71 (31.0%) with PN dysfunction and 22 of 73 (30.1%) without PN dysfunction, respectively (Fig.1). After excluding 83 subjects with detrusor overactivity due possibly to central nervous system dysfunction, we further analyzed 61 subjects without detrusor overactivity, 29 with PN dysfunction and 32 without PN dysfunction. DSD was found in 12 of 29 (41.4%) with PN dysfunction and 8 (25%) of 32 without PN dysfunction (Fig.2). Concerning the grade of detrusor contraction in Schäfer nomogram, among 71 subjects with PN dysfunction, the details of 53 subjects enforced PFS were the following; 4 subjects were strong, 20 subjects normal, 22 subjects weak and 7 subjects very weak. Among 64 subjects without PN dysfunction, the breakdown of each group was 4, 31, 29 and no subject. There was significant difference between two groups (p=0.025) (Fig.3). As to the result of Q max and PVR in 53 subjects with PN dysfunction, 5 subjects with DSD (35.7%, 5/14) had the decline of Qmax (<10 ml/sec) and 15 subjects without DSD (38.5%, 15/39) did (Fig.4). Ten subjects with DSD (71.4%, 10/14) had the increase of PVR (>100 ml) and 20 subjects without DSD (51.3%, 20/39) did (Fig.5).

Interpretation of results

We revealed that PN dysfunction can cause DSD, and 31% of subjects with PN dysfunction possessed DSD in this investigation. We suppose the reason is the damage of PN dominate anal sphincter.

Concluding message

PN dysfunction can cause DSD. PN dysfunction patients combined DSD are possible to cause both the decrease of Qmax and the increase of PVR. So we need to be particularly careful to these cases.

Fig.1; Incidence of DSD in 144 subjects

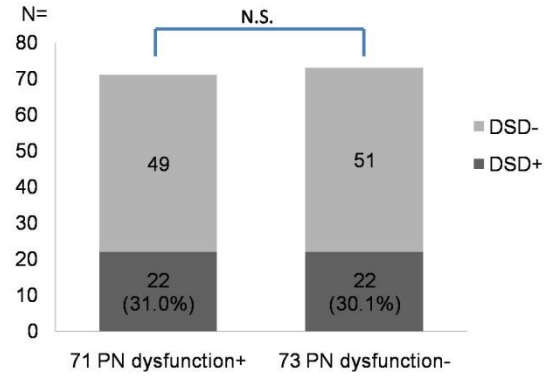


Fig.2; Incidence of DSD in 61 subjects without DO

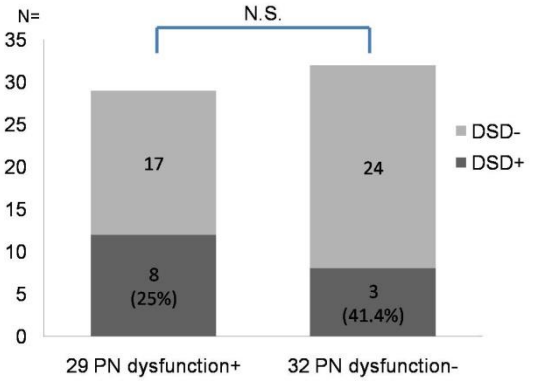


Fig.3; Relationship between PN dysfunction and grade of detrusor contraction

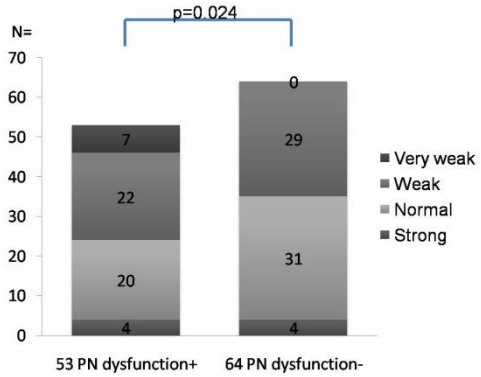


Fig.4; Relationship between PN dysfunction and maximum urinary flow rate (Qmax)

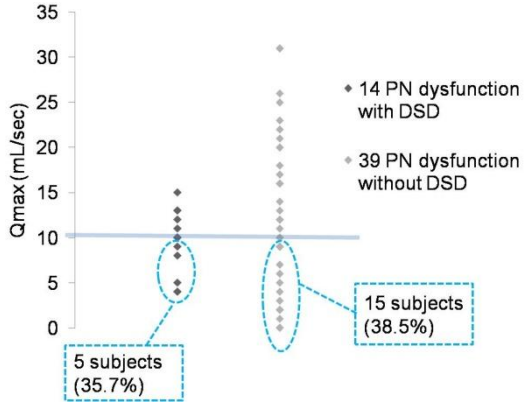
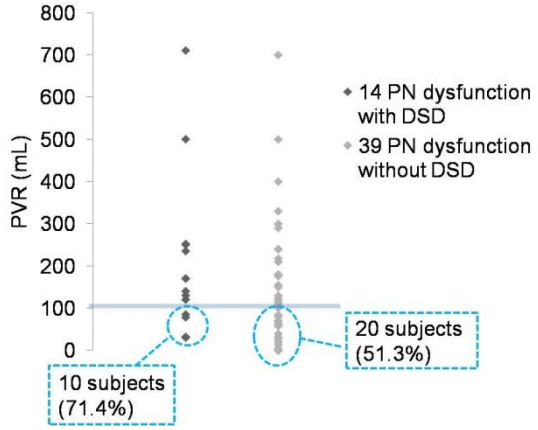


Fig.5; Relationship between PN dysfunction and post void residuals (PVR)



Specify source of funding or grant	**N/A**
Is this a clinical trial?	**No**
What were the subjects in the study?	**HUMAN**
Was this study approved by an ethics committee?	**No**
This study did not require ethics committee approval because	**Our UDS was in force for patients who had urinary symptoms and needed this examination, it is a part of medical examination. This is retrospective study that organized the relationship between DSD and peripheral nerve dysfunction. Of course, we obtained patients' consent in writing to examine UDS.**
Was the Declaration of Helsinki followed?	**Yes**
Was informed consent obtained from the patients?	**Yes**