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WHAT KINDS OF THE NEUROLOGICAL DISORDER SHOW NEUROGENIC CHANGE IN EXTERNAL ANAL SPHINCTER EMG?

Hypothesis / aims of study

External anal sphincter (EAS)-electromyography (EMG) is an established method to detect neurogenic change of the sphincter muscle.(1) Since, anal sphincter muscle is innervated by sacral pudendal nerve from Onuf's nucleus in sacral cord, EAS-EMG is believe to be useful for the diagnosis of multiple system atrophy (MSA), in which neuronal cell loss in Onuf's nucleus is the pathological hallmark. However, recent studies suggested that many neurological disorders other than MSA might show neurogenic change in EAS-EMG. The presence of lumbar spondylosis might affect the results of EAS-EMG, because lumbar spondylosis could lead to the degeneration of sacral cord. We aimed to elucidate kinds of the neurological disorder showing neurogenic change in EAS-EMG.

Study design, materials and methods

We retrospectively reviewed 996 patients who underwent EAS-EMG. All of the patients were reffered to our hospital suspected of having neurological disorders. Either of the following two criteria was used for the diagnosis of the neurogenic change in EAS-EMG.(2)

(a) more than 20% of motor unit potentials (MUPs) have a duration >10 ms,

or

(b) the average duration of MUPs >10 ms, particularly including the late components.

Details of neurological disorders in this study were as follows.

MSA :n=245, 25% Parkinson's disease (PD):n=87, 8.7% atypical Parkinsonism (Pism) n=68, 6.8% lumbar spondylosis:n=34, 3.4% diabetic neuropathy:n=27, 2.7%.

<u>Results</u>

Neurogenic change in EAS-EMG was detected in 502 patients. Neurogenic change in EAS-EMG was prevalent in MSA (158/245, 64.4%), lumbar spondylosis (19/34, 55.8%), atypical Parkinsonism (29/681, 42.6%), diabetic neuropathy (20/27, 74.0%), and PD (30/87, 34.4%).(Figure) Since patients with PD and diabetic neuropathy usually do not show neurogenic change in EAS-EMG, we examined the comorbidity of the lumbar spondylosis , which might also lead to neurogenic change in EAS-EMG. Eleven patients with PD and 2 patients with diabetic neuropathy had lumbar spondylosis. Examination of the lumbar spondylosis was not performed for the rest of the patients with PD and diabetic neuropathy

Interpretation of results

Although this study included significantly larger numbers of patients with MSA and smaller number of patients with PD, diabetic neuropathy, and lumbar spondylosis as compared to the general prevalence of these disorders, this study showed that neurogenic change in EAS-EMG was prevalent in MSA, which is consistent with several previous studies.

This study also showed that many neurological disorders might show neurogenic change in EAS-EMG. As in the case with PD and diabetic neuropathy in this study, neurogenic change in EAS-EMG might be attributable to the presence of the lumbar spondylosis in some cases.

Concluding message

Many neurological disorders showed neurogenic change in EAS-EMG besides MSA. Since, lumbar spondylsis is common and might cause neurogenic change in EAS-EMG, it is important to examine the presence of lumbar spondylosis in patients having neurogenic change in EAS-EMG.



Figure Distribution of the number of the patients whose average MUP was more than 10ms.

Abbreviation DLB:dementia with lewy bodies, MSA:multiple system atrophy, Pism:atypical Parkinsonism, PSP:progressive supranuclear palsy, CS:cervical spondylosis, LS:lumbar spondylosis, DM:diabetic neuropathy. <u>References</u>

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