Tonic Electromyogram Density in Multiple System Atrophy with Predominant Parkinsonism and Parkinson’s Disease

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Abstract

Background: Both Parkinson’s disease (PD) and multiple system atrophy (MSA) have associated sleep disorders related to the underlying neurodegenerative pathology. Clinically, MSA with predominant parkinsonism (MSA-P) resembles PD in the manifestation of prominent parkinsonism. Whether the amount of rapid eye movement (REM) sleep without atonia could be a potential marker for differentiating MSA-P from PD has not been thoroughly investigated. This study aimed to examine whether sleep parameters could provide a method for differentiating MSA-P from PD.

Methods: This study comprised 24 MSA-P patients and 30 PD patients, and they were of similar age, gender, and REM sleep behavior disorder (RBD) prevalence. All patients underwent clinical evaluation and one night of video-polysomnography recording. The tonic and phasic chin electromyogram (EMG) activity was manually quantified during REM sleep of each patient. We divided both groups in terms of whether they had RBD to make subgroup analysis.

Results: No significant difference between MSA-P group and PD group had been found in clinical characteristics and sleep architecture. However, MSA-P group had higher apnea-hypopnea index (AHI; 1.15 [0.00, 8.73]/h vs. 0.00 [0.00, 0.55]/h, β = 0.024) and higher tonic chin EMG density (34.02 [18.48, 57.18]% vs. 8.40 [3.11, 13.06]%, β = 0.001) as compared to PD patients. Subgroup analysis found that tonic EMG density in MSA + RBD subgroup was higher than that in PD + RBD subgroup (55.04 [26.81, 69.62]% vs. 11.40 [8.51, 20.41]%). Furthermore, no evidence of any difference in tonic EMG density emerged between PD + RBD and MSA - RBD subgroups (P > 0.05). Both disease duration (P = 0.056) and AHI (P = 0.051) showed no significant differences during subgroup analysis although there was a trend toward longer disease duration in PD + RBD subgroup and higher AHI in MSA - RBD subgroup. Stepwise multiple linear regression analysis identified the presence of MSA-P (β = 0.552, P < 0.001) and RBD (β = 0.433, P < 0.001) as predictors of higher tonic EMG density.

Conclusion: Tonic chin EMG density could be a potential marker for differentiating MSA-P from PD.

Key words: Multiple System Atrophy with Predominant Parkinsonism; Parkinson’s Disease; Polysomnography; Tonic Chin Electromyogram Density

Introduction

Both Parkinson’s disease (PD) and multiple system atrophy (MSA) have associated sleep disorders related to the underlying neurodegenerative architecture, such as abnormal sleep architecture, rapid eye movement sleep behavior disorder (RBD), excessive daytime sleepiness, restless legs syndrome, periodic limb movement disorder, and circadian dysfunction.1-3 RBD is common in and strongly correlated with alpha-synucleinopathies (SPs), with the prevalence varying by diseases: 30–50% in PD and 80–95% in MSA.4 Rapid eye movement sleep without atonia (RSWA) is an essential diagnostic feature of RBD on polysomnography (PSG), with either excessive sustained elevation of electromyogram (EMG) tone or excessive phasic EMG.

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activity in the rapid eye movement (REM) stage. Previous research has reported that higher surface EMG activity was associated with longer PD disease duration and greater disease severity and has suggested higher surface EMG activity as a PD biomarker.[9] However, little is known about the clinical correlates of RSWA in MSA. One study found that RSWA percentage was higher in MSA than that in PD.[6] MSA is characterized by prominent autonomic dysfunction with combinations of predominant parkinsonism (MSA-P), predominant cerebellar ataxia, and corticospinal disorders. Clinically, MSA-P resembles PD in the manifestation of prominent parkinsonism. Whether the amount of RSWA could be a potential marker for differentiating MSA-P from PD has not been thoroughly investigated. In this study, we explored the association between RSWA and SPs by manually quantifying chin EMG activity during REM sleep, and then, we compared the results in the MSA-P group to those of the PD group to determine if any significant differences exist and whether these differences could be used to distinguish MSA-P from PD.

Methods

Patients

This study was a retrospective study evaluating the medical records of all MSA-P patients and case-matched PD patients from September 2010 to May 2015 in Center of Parkinsonism and Movement Disorder, The Second Affiliated Hospital of Soochow University. All patients meeting the clinical probable diagnostic criteria for MSA-P[7] were enrolled. We then searched for patients that satisfied the UK Parkinson’s Disease Society Brain Bank clinical diagnostic criteria[8] and matched the MSA-P patients for age, gender, and RBD percentage. RBD was diagnosed according to the International Classification of Sleep Disorders (ICSD-II) criteria. It requires the combination of clinical characteristics (either by history or by abnormal REM sleep behaviors captured during video monitoring) and the presence of RSWA during video-polysomnography (vPSG). Patients who were taking selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, melatonin, or clonazepam were excluded from the study as these medications can alter EMG tone.[9,10] Patients continued taking their medications for parkinsonism as usual. Drug dosages were converted to daily levodopa equivalent doses (LEDs) for the purpose of data collection.[11]

All patients underwent a clinical evaluation, including a comprehensive neurological examination, the Mini Mental Status Examination (MMSE), the Montreal Cognitive Assessment (MOCA, Beijing version), the Epworth Sleepiness Scale (ESS), and the Pittsburgh Sleep Quality Index (PSQI). The PD patients were evaluated by the Unified Parkinson’s Disease Rating Scale and Hoehn and Yahr scale during the medication “on” state. All patients provided written informed consent to participate in this study and signed additional consent forms agreeing to the use of their vPSG for scientific purposes. This study was approved by the Ethical Committee of The Second Affiliated Hospital of Soochow University.

Polysomnography

All patients underwent a night of standard vPSG (Compumedics-E series, Australia) monitoring in the sleep center. The basic recordings included electroencephalogram (F3-A2, F4-A1, C3-A2, C4-A1, O1-A2, and O2-A1), electrooculogram (EOG, LOC-A2, and ROC-A1), chin EMG, electrocardiogram, nasal-oral pressure transducer airflow, thermal oronasal airflow, thoracic and abdominal respiratory efforts, oxyhemoglobin saturation, snoring sound, and body position. All the vPSGs were manually scored by experienced technologists according to the American Academy of Sleep Medicine guidelines.

The following vPSG data were obtained and analyzed: awakenings; total sleep time (TST); sleep efficiency (SE); sleep latency (SL); REM sleep latency (REML); wake after sleep onset (WASO); percentage of sleep spent in non-REM sleep stage (NREM) 1, NREM2, NREM3, and REM sleep; arousal index; apnea-hypopnea index (AHI); minimal oxygen saturation (SaO₂); mean SaO₂; and the percentage of time spent at SaO₂ <90% (time – [SaO₂ <90%]).

Analysis of electromyogram activity

The tonic and phasic chin EMG activity was quantified manually in each patient. RSWA was scored according to a previously published method:[12] each 20 s REM sleep epoch was scored as tonic depending on the presence of chin EMG activity for more than 50% of each epoch, with an amplitude of at least twice that of background activity or >10 μV. Phasic EMG activity was scored with 2 s mini-epochs containing bursts of EMG activity lasting between 0.1 s and 5.0 s, with an amplitude of at least four times the background EMG activity. Bursts of phasic activity occurring simultaneously with tonic activity were required to have an amplitude of twice the background tonic EMG activity within the same 2 s mini-epoch to be scored as phasic activity.[13] We calculated separately the percentage of 20 s epochs with tonic and 2 s mini-epochs with phasic EMG activity as tonic chin EMG density and phasic chin EMG density, respectively. All chin EMG activity correlating with arousals or respiratory events was carefully eliminated from the quantification.

Statistical analysis

SPSS software version 17.0 (SPSS Inc., Chicago, IL, USA) was used for the statistical analysis. Data were presented as mean ± standard deviation (SD), median (Q1, Q3), or frequencies (percentages). The qualitative data were analyzed using Chi-squared test or Fisher’s exact test, as appropriate. Comparison of continuous variables in the two groups was conducted using Student’s t-test. If the data were not in a normal distribution, the Mann-Whitney U-test was used. Four subgroups comparisons were performed using one-way analysis of variance or Kruskal-Wallis test. Stepwise multiple linear regression analysis was performed to determine the predictors of higher tonic EMG density. The independent variables included age, gender, BMI, TST, SE, SL, REML,
WASO, NREM1%, NREM2%, NREM3%, REM%, AHI, arousal index, P%, ESS, and MOCA. Statistical significance was defined as $P < 0.05$.

**Results**

**Demographic and clinical characteristics**

Demographic and clinical characteristics of individuals are presented in Table 1. Among the 24 MSA-P patients, 12 were male. The MSA-P group had a mean age of 64.9 ± 8.0 years (range: 46–77 years), mean body mass index (BMI) of 22.40 ± 3.66 kg/m$^2$ (range: 15.80–33.10 kg/m$^2$), mean disease duration of 38.00 ± 19.75 months (range: 6–72 months), sleep apnea-hypopnea syndrome (SAHS) prevalence of 33.3%, and mean LEDs of 433.77 ± 251.38 mg/d (range: 50–1139 mg/d). According to the ICSD-II criteria, 16 (66.7%) of 24 MSA-P patients were diagnosed with RBD, all of whom had clinical history of abnormal sleep behaviors and definite vPSG-captured dream enactment behavior during REM stage. The remaining eight MSA-P without RBD patients had no clinical history of sleep-related injurious or disruptive behaviors. Thirty PD patients with similar age, gender, and RBD prevalence were recruited. The PD group had a mean age of 65.3 ± 5.5 years (range: 50–79 years), mean BMI of 22.79 ± 3.11 kg/m$^2$ (range: 16.60–28.40 kg/m$^2$), mean disease duration of 43.97 ± 29.37 months (range: 9–128 months), SAHS prevalence of 10.0%, mean LEDs of 372.47 ± 147.05 mg/d (range: 37.50–65.00 mg/d), and 18 (60.0%) of 30 PD patients were diagnosed with RBD according to the ICSD-II criteria. We found no significant differences in BMI ($P = 0.674$), disease duration ($P = 0.398$), LEDs ($P = 0.297$), ESS score ($P = 0.702$), PSQI score ($P = 0.614$), MMSE score ($P = 0.984$), or MOCA score during the medication “on” state ($P = 0.430$) between the two groups.

**Subgroups analysis**

As tonic chin EMG activity and AHI showed significant difference between MSA-P group and PD group, both of them had great associations with RBD, so we divided both groups into with (MSA + RBD; PD + RBD) or without RBD (MSA - RBD; PD - RBD) subgroups to make other comparisons.

The results are displayed in Table 3. Tonic and phasic EMG density subgroup comparisons are shown in Figures 1 and 2. Both disease duration ($P = 0.056$) and AHI ($P = 0.051$) showed no significant differences during subgroup analysis although there was a trend toward longer disease duration in PD + RBD subgroup and higher AHI in MSA - RBD subgroup. Tonic and phasic EMG density showed significant differences between PD + RBD subgroup and PD - RBD subgroup ($Z = −3.979, P < 0.001$; and $Z = −3.726, P < 0.001$), MSA + RBD subgroup and MSA - RBD subgroup ($Z$...)

**Table 1: Demographics and clinical features of patients with MSA-P and PD**

| Characteristics          | PD group ($n = 30$) | MSA-P group ($n = 24$) | Statistical values | $P$  |
|--------------------------|--------------------|------------------------|--------------------|------|
| Age (years)              | 65.3 ± 5.5         | 64.9 ± 8.0             | 0.227*             | 0.821|
| Male                     | 14 (46.7)          | 12 (50.0)              | 0.059*             | 0.808|
| BMI (kg/m$^2$)           | 22.79 ± 3.11       | 22.40 ± 3.66           | 0.422*             | 0.674|
| Disease duration (months)| 43.97 ± 29.37      | 38.00 ± 19.75          | 0.852*             | 0.398|
| RBD                      | 18 (60.0)          | 16 (66.7)              | 0.254*             | 0.614|
| SAHS                     | 3 (10.0)           | 8 (33.0)               | 3.152*             | 0.076|
| ESS score                | 6.50 ± 4.02        | 6.92 ± 4.44            | −0.361*            | 0.720|
| PSQI score               | 8.03 ± 4.00        | 7.43 ± 4.58            | 0.507*             | 0.614|
| MMSE score               | 25.93 ± 3.55       | 25.92 ± 3.23           | 0.021*             | 0.984|
| MOCA score               | 23.20 ± 4.96       | 22.21 ± 3.98           | 0.796*             | 0.430|
| LEDs (mg/d)              | 372.47 ± 147.05    | 433.77 ± 251.38        | −1.059*            | 0.297|
| UPDRS III score          | 21.73 ± 9.40       | NA                     | NA                 | NA   |
| UPDRS total score        | 34.87 ± 13.42      | NA                     | NA                 | NA   |
| Hoehn-Yahr stage         | 2.00 (1.38, 3.00)  | NA                     | NA                 | NA   |

The data are shown as mean ± SD, or n (%). *Student’s t-test; †Chi-squared test. PD: Parkinson’s disease; MSA-P: Multiple system atrophy with predominant parkinsonism; BMI: Body mass index; RBD: Rapid eye movement sleep behavior disorder; SAHS: Sleep apnea-hypopnea syndrome; ESS: Epworth Sleepiness Scale; PSQI: Pittsburgh Sleep Quality Index; MMSE: Mini Mental Status Examination; MOCA: Montreal Cognitive Assessment. UPDRS: Unified Parkinson’s Disease Rating Scale; LEDs: Levodopa equivalent doses; NA: Not applicable; SD: Standard deviation.
Table 2: Polysomnographic data in patients with MSA-P and PD

| Parameters                     | PD group (n = 30) | MSA-P group (n = 24) | Statistical values | P      |
|--------------------------------|------------------|----------------------|--------------------|--------|
| Awakenings (n)                 | 22.00 ± 9.42     | 18.92 ± 7.73         | 1.292*             | 0.202  |
| TST (min)                      | 356.95 ± 101.64  | 334.88 ± 59.02       | 0.943*             | 0.350  |
| SE (%)                         | 68.32 ± 15.78    | 62.75 ± 12.30        | 1.419*             | 0.162  |
| SL (min)                       | 5.25 (0.50, 17.63)| 12.75 (4.50, 26.38)  | −1.630†            | 0.103  |
| REM (min)                      | 126.72 ± 84.28   | 161.50 ± 110.55      | −3.131*            | 0.195  |
| WASO (min)                     | 116.53 ± 81.65   | 134.88 ± 65.88       | −0.892*            | 0.376  |
| NREM1 (%)                      | 21.92 ± 17.22    | 22.29 ± 12.85        | −0.088*            | 0.930  |
| NREM2 (%)                      | 44.83 ± 14.84    | 41.74 ± 15.37        | 0.748*             | 0.458  |
| NREM3 (%)                      | 16.68 ± 10.35    | 16.28 ± 11.54        | 0.135*             | 0.893  |
| REM (%)                        | 16.58 ± 7.17     | 19.68 ± 11.96        | −1.180*            | 0.243  |
| Arousal index                  | 5.60 (2.10, 9.38)| 7.00 (3.85, 11.85)   | −1.297†            | 0.195  |
| AHI (h)                        | 0.90 (0.00, 0.55)| 1.15 (0.00, 8.73)    | −2.261†            | 0.024  |
| Minimal SaO2 (%)               | 91.60 ± 2.93     | 89.33 ± 3.81         | 2.473*             | 0.017  |
| Mean SaO2 (%)                  | 95.77 ± 1.61     | 94.79 ± 1.91         | 2.034*             | 0.047  |
| Time spent at SaO2 <90% (%)    | 0.00 (0.00, 0.00)| 0.05 (0.00, 3.43)    | −2.674†            | 0.007  |
| Tonic EMG density (%)          | 84.0 (3.11, 13.06)| 34.02 (18.48, 57.18) | −4.169†            | <0.001 |
| Phasic EMG density (%)         | 4.85 (1.67, 9.36)| 5.78 (1.12, 17.99)   | −0.470†            | 0.638  |

The data are shown as mean ± SD or median (Q1, Q3). *Student’s t-test; †Mann-Whitney U-test. PD: Parkinson’s disease; MSA-P: Multiple system atrophy with predominant parkinsonism; TST: Total sleep time; SE: Sleep efficiency; SL: Sleep latency; REML: Rapid eye movement sleep latency; WASO: Wake after sleep onset; REM: Rapid eye movement; NREM: Non-REM; AHI: Apnea-hypopnea index; SaO2: Oxygen saturation; EMG: Electromyogram; SD: Standard deviation.

Table 3: Comparisons of demographics and chin EMG density among the four subgroups

| Parameters                     | PD + RBD subgroup (n = 18) | PD - RBD subgroup (n = 12) | MSA + RBD subgroup (n = 16) | MSA - RBD subgroup (n = 8) | Statistical values | P      |
|--------------------------------|-----------------------------|----------------------------|-----------------------------|-----------------------------|--------------------|--------|
| Age (years)                    | 65.9 ± 6.4                  | 64.4 ± 3.9                 | 64.5 ± 9.2                  | 65.8 ± 5.0                  | 0.198*             | 0.897  |
| Male                           | 9 (50.0)                    | 5 (41.7)                   | 7 (43.8)                    | 5 (62.5)                    | 1.075†             | 0.800  |
| BMI (kg/m²)                    | 23.46 ± 3.02                | 21.80 ± 3.10               | 22.63 ± 3.62                | 21.95 ± 3.95                | 0.713*             | 0.549  |
| Disease duration (months)      | 53.67 ± 32.19               | 29.42 ± 16.96              | 39.31 ± 20.53               | 35.38 ± 19.16               | 2.691*             | 0.056  |
| AHI (h)                        | 0.00 (0.00, 1.75)           | 0.00 (0.00, 0.43)          | 0.65 (0.00, 4.30)           | 14.20 (0.08, 36.48)         | 7.789§             | 0.051  |
| Tonic EMG density (%)          | 11.40 (8.51, 20.41)‡         | 1.82 (0.63, 5.51)          | 55.04 (26.81, 69.62)‡        | 11.19 (2.94, 28.88)‡         | 35.262‡            | <0.001 |
| Phasic EMG density (%)         | 8.30 (5.15, 15.19)†          | 1.67 (1.26, 2.59)          | 8.08 (3.57, 25.18)†          | 0.57 (0.00, 8.36)†           | 17.424†            | 0.001  |

The data are shown as mean ± SD or median (Q1, Q3). *One-way analysis of variance; † Chi-squared test; §Kruskal-Wallis test followed by Mann-Whitney U-test, with Bonferroni correction α' = 0.05/(4×[4−1]/2) = 0.0083; ‡p<0.05 versus PD-RBD subgroup; †P<0.05, versus MSA + RBD subgroup; PD: Parkinson’s disease; MSA: Multiple system atrophy; RBD: Rapid eye movement sleep behavior disorder; BMI: Body mass index; AHI: Apnea-hypopnea index; EMG: Electromyogram; SD: Standard deviation.

Regression analysis

To further explore the associations of clinical factors and the presence of higher tonic EMG density, the data including age, gender, BMI, disease type, disease duration, TST, SE, SL, REML, WASO, NREM1, NREM2, NREM3, REM, arousal index, AHI, phasic EMG density, ESS score, MOCA score, and LEDs were subjected to regression analysis. Table 4 shows that the presence of MSA-P (β = 0.552, P < 0.001) and RBD (β = 0.433, P < 0.001) was associated with higher tonic EMG density.

Discussion

The main finding was that the MSA-P group had higher tonic tone than the PD group, irrespective of whether there was clinical RBD or not. In addition, we identified the presence of MSA-P and RBD as independent predictors of higher tonic EMG density. Subgroup analysis showed that the disease duration had a trend toward being significant, and we might see a longer disease duration in PD + RBD subgroup, which could...
be attributed to the fact that the RBD symptoms seem to gradually increase during the course of PD.\textsuperscript{14}

Only a few studies have evaluated the difference in RSWA between MSA and PD. In a study of 26 MSA and 45 PD patients with a mean disease duration of 4.5 ± 2.3 years and 9 ± 5.3 years ($P < 0.001$), respectively, Iranzo \textit{et al.}\textsuperscript{6} found that the MSA group, as compared to the PD group, exhibited increased RSWA (68.8 ± 29.3\% vs. 39.4 ± 31.6\%, $P < 0.001$) and submental phasic EMG activity (30.6 ± 20\% vs. 22.1 ± 11.2\%, $P > 0.05$). Their finding of a higher RSWA percentage in MSA patients was consistent with our results, but their values of RSWA were higher than those found in this study.

The RSWA metrics in the research seemed quite low for SPs, especially for the Montreal 20S scoring approach. In our previous study, Gong \textit{et al.}\textsuperscript{15} identified the tonic EMG density in PD + RBD to be 13.88 (3.07–39.96\%). Some other studies analyzing Asian patients have shown similar results. Zhou \textit{et al.}\textsuperscript{16} showed tonic EMG density in Chinese RBD patients was 20.6 ± 16.9\%, and in their previous study, they found significant lower values of EMG activity as well.\textsuperscript{17} Therefore, we speculated the lower EMG activity in Chinese patients with RBD might be attributed to ethnic differences. We further grouped both groups into with and without RBD to make subgroup comparisons to eliminate the influence of RBD on tonic EMG density. Moreover, we showed that not only MSA + RBD subgroup had a higher tonic EMG density than PD + RBD subgroup but also the MSA - RBD subgroup had the tonic EMG density close to that of PD + RBD subgroup. That was MSA-P group had higher tonic tone than the PD group, irrespective of whether there was clinical RBD or not.

It has demonstrated that lesions in the sublaterodorsal nucleus (SLD) cause RSWA. The proposed pathophysiology of RBD in humans suggested by Boeve \textit{et al.}\textsuperscript{18} included lesions in the SLD and altered locomotor drive. As loss of REM atonia is the essential feature of RBD, some researchers believed that a higher tonic chin EMG density during REM sleep could indicate a greater RBD severity\textsuperscript{19} and resulted from more severe neurodegeneration of the associated brainstem structure. The study showed higher tonic chin EMG density in the MSA-P group, which could be attributed to the more rapid and widespread neurodegenerative pathology in MSA-P as compared to that in PD.

Moreover, the tonic chin EMG activity might be partially due to the RBD, and it might also reflect the severity of basal ganglia (BG) dysfunction. Takakusaki \textit{et al.}\textsuperscript{20} demonstrated that the GABAergic substantia nigra pars reticulata (SNr)-pedunculopontine nucleus (PPN) projections could control the muscle tone of movements during wakefulness and modulate REM-sleep atonia by activating the cholinergic PPN neurons. Studies have shown that loss of cholinergic neurons in the PPN occurs in both PD and MSA and the degree of cholinergic neuron loss correlates with the severity of motor symptoms in PD. In parkinsonism, globus pallidus interna hyperactivity results in more inhibitory GABAergic input to the PPN, leading to reduced activity of the glutamatergic PPN neurons, which project to the reticulospinal neurons of brainstem and spinal cord, causing abnormal changes in locomotion and muscle tone.\textsuperscript{21} MSA has more rapid and widespread pathology than PD. We suspected that the SLD, PPN, and BG are affected earlier and more severely in MSA-P than PD, resulting in a higher REM sleep tonic chin EMG density in MSA-P. We anticipate future research focusing on the
relationship between abnormal BG output and tonic EMG activity during REM sleep.

We found a significant difference in tonic chin EMG density, but not in phasic chin EMG density between the two groups. In our previous study, we found tonic RSWA was more closely correlated with PD severity than phasic RSWA,[20] which was consistent with the results of this study. Similarly, a case report by Tachibana and Oka[21] described a 60-year-old MSA patient, whose PSGs indicated tonic chin EMG activity that increased as the patient’s disease progressed, whereas the patient’s phasic chin EMG activity remained relatively suppressed. Postuma et al. analyzed[19] the baseline PSG data of the patients who first presented with RBD and later developed neurodegenerative diseases; the researchers found that the severity of tonic chin EMG density on baseline PSG predicted the development of PD. Researchers have suggested that tonic RSWA might reflect degeneration of the SLD,[24-26] whereas phasic RSWA results from activation of locomotor generators during sleep and alterations of the intermediate ventromedial medulla pathways.[18] Dysfunction of brainstem structures, such as the PPN or BG, can also alter muscle tone. Because of the different mechanisms underlying tonic and phasic chin EMG activities, we found no contradiction in the existence of a strong correlation between tonic chin EMG activity and neurodegenerative pathology of SPs in the brainstem and the absence of a correlation between phasic chin EMG activity and SPs.

There were some limitations of this study. First, the sample size was not sufficiently large enough to determine the cutoff value for tonic chin EMG activity. Second, recognizing MSA early in the disease progression is difficult. The patients in this study were not in the earliest stages of disease progression. Future studies should focus on patients who first manifest idiopathic RBD and are subsequently diagnosed with an SP, to examine the evolution of RSWA in search of conversion predictors.

In conclusion, the results showed that the MSA-P patients had higher AHI and higher tonic chin EMG density as compared to PD patients. Moreover, the presence of MSA-P and RBD were associated with higher tonic EMG density. Given these results, the tonic EMG density could be a potential marker for differentiating MSA-P from PD.

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**Conflicts of interest**

There are no conflicts of interest.

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