IgLON5 antibodies are infrequent in patients with isolated sleep apnea

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IgLON5 antibodies were recently identified in patients with a complex syndrome of rapid-eye-movement and non-rapid-eye-movement parasomnias, insomnia, sleep apnea, excessive daytime sleepiness, bulbar symptoms and movement disorders. The prognosis appears to be unfavorable as a considerable number of reported patients died from sudden death during sleep or by respiratory failure [1,2]. Moreover, most patients were unresponsive to immunotherapy [3]. Recent studies, however, may have investigated a higher number of patients in more advanced disease stages where immunosuppressive therapy may have been insufficient due to advanced neuroinflammation and subsequent tau-related neurodegeneration [1,4]. Although the syndrome is typically characterized by a combination of several neurological signs, monosymptomatic or oligosymptomatic disease courses may exist at least in early stages [5]. Moreover, the phenotype of IgLON5 disease may be even broader than previously thought [6]. Thus, screening for IgLON5 antibodies in patients with more uniform phenotypes is warranted. In this regard, a recent screening of 33 patients with tauopathies remained negative [7]. Here the aim was to assess the frequency of IgLON5 antibodies in a sample of patients with obstructive sleep apnea (OSA) as a cardinal sign of IgLON5 disease.

Among 70 screened OSA patients (26 female, age 54.8 ± 16.1 years, range 19–85), sleep disturbances and daytime sleepiness were the main complaints. Eleven patients had a history of stroke (n = 2), Parkinson’s disease (n = 1), restless legs syndrome (n = 1), brain surgery (n = 1), depression (n = 4), borderline personality disorder (n = 2) and restless legs syndrome (n = 1). All patients were diagnosed with obstructive mild to severe sleep apnea (OSA) and apnea–hypopnea indices (AHI) between 10.5 and 57.3/h. Fifty-nine patients were diagnosed using a home sleep test with 22 exhibiting moderate or severe OSA. Continuous positive airway pressure therapy was initiated in them during an in-laboratory titration (group 1, Table 1). The remaining 37/59 patients were initially diagnosed with mild OSA with mild severity using a home sleep test. An additional polysomnography was thus initiated due to suspicious clinical symptoms (group 2, Table 1): fourteen patients finally suffered from mild OSA (AHI ≥ 5 and <15/h), 31 from moderate OSA (AHI ≤ 15 and <30/h) and 14 from severe OSA (AHI ≤ 30/h).

Table 1 Demographic and sleep-related information

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (female/male)</td>
<td>22 (f = 4, m = 18)</td>
<td>37 (f = 18, m = 19)</td>
<td>11 (f = 5, m = 6)</td>
</tr>
<tr>
<td>Age</td>
<td>64.5 ± 13.0</td>
<td>47.9 ± 15.3</td>
<td>58.3 ± 13.6</td>
</tr>
<tr>
<td>AHI</td>
<td>23.0 ± 10.1</td>
<td>23.7 ± 11.9</td>
<td>14.7 ± 20.4</td>
</tr>
<tr>
<td>Min SpO2</td>
<td>81.2 ± 6.7</td>
<td>87.8 ± 4.8</td>
<td>82.1 ± 8.3</td>
</tr>
<tr>
<td>ODI</td>
<td>16.9 ± 10.5</td>
<td>5.1 ± 4.5</td>
<td>13.5 ± 25.8</td>
</tr>
<tr>
<td>ESS</td>
<td>8.4 ± 5.6</td>
<td>9.0 ± 5.2</td>
<td>7.6 ± 3.2b</td>
</tr>
<tr>
<td>BMI</td>
<td>30.9 ± 6.0</td>
<td>30.2 ± 4.8</td>
<td>33.2 ± 7.3</td>
</tr>
</tbody>
</table>

AHI, apnea–hypopnea index; BMI, body mass index; ESS, Epworth Sleepiness Score; min SpO2, blood oxygen saturation; ODI, oxygen desaturation index. ESS scores lower than 11 were considered normal [8]. ESS was available in 4/11 patients.
the window of opportunity to causally treat this serious antibody-related disorder.

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Disclosure of conflicts of interest

The authors declare no financial or other conflicts of interest.

References

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