Journal of Sleep Disorders & Therapy
ISSN: 2167-0277

Xi, J Sleep Disord Ther 2012, 1:2
DOI: 10.4172/2167-0277.1000e107

Editorial Open Access

REM Sleep Behavior Disorder (RBD)-A Potential Predictive Biomarkers of Future Neurodegeneration

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REM sleep behavior disorder (RBD) is a novel type of Parasomnias characterized by abnormal behavioral manifestations during REM sleep [1]. RBD affects 0.4-0.5% of the elderly population with a typical age of onset in 50s-60s [2-5]. RBD presents with a spectrum of dream-enacting behavioral manifestations, including sleep shouting, fisting, kicking or falling out of bed with a high prevalence of sleep-related injuries to themselves or/and bed-partners [2-5].

Accumulating evidences suggests that idiopathic RBD (iRBD) is an integrated part of disease progress in α-synucleinopathy neurodegeneration, such as Parkinson's disease (PD) and dementia with Lewy bodies (DLB) [2-9]. Longitudinal clinical studies suggests that the estimated 5-year risk of developing any neurodegenerative disorder in iRBD patients range from 8.5%-38%, which increase to 38%-65% at around 10-year follow-up [5-9]. Hence, the time gap between RBD and neurodegenerative disorders may potentially provide a therapeutic window for neuroprotective measures in halting the underlying neurodegeneration at an early stage. Thus, research also focused on the identification of subclinical markers of neurodegeneration in iRBD, including color vision deficit and olfactory dysfunction [10-11], which might be construed as the early stages of neurodegenerative process [12]. Additionally, polysomnographic feature of RBD has also been reported as a predictive marker of neurodegeneration [13,14]. Nonetheless, studies were limited by cross-sectional design with relatively small sample size.

Early neurodegeneration in RBD could be demonstrated by brain imaging technologies. Studies suggested that iRBD was associated with dopaminergic neurotransmission abnormalities [15-17], with a progressive decline of the striatal presynaptic dopaminergic transporter from subclinical RBD, iRBD to RBD with PD [15-17]. Up to date, only two studies reported the longitudinal course of dopamine abnormalities in iRBD. Miyamoto et al. [18] found that the dopamine uptake decreased by 4-6% per year. Another prospective case-control study of 20 Spanish iRBD of 3 years reported a significant decline in dopamine uptake in iRBD group, and those developed Parkinson's disease had the lowest dopamine uptake at baseline [19]. Although brain imaging techniques show a high sensitivity in detecting neurodegeneration, they are expensive with limited accessibility for routine screening of early neurodegeneration among RBD patients. Thus, the search of reliable and easily applicable clinical markers is important. Eisensehr et al. [17] correlated the increase in muscle activity during REM sleep to reduction of striatal dopamine transporters. Another study found that there were correlations between cerebral blood flow and color vision and olfactory abnormalities [20]. These studies have provided preliminary results of the possible correlations between clinical markers and neuroimaging findings.

Over past decade, the epidemiological study of RBD in Hong Kong Chinese was conducted by Hong Kong sleep centre [4,9,20-25]. They reported the largest cohort of Chinese RBD patients (n=82) [4]. Recently, prospective follow-up study on this cohort of iRBD patients [n=81, mean follow-up duration=5.6 years (s.d.3.3),] reported that the estimated 5-year and 9-year risk of developing any neurodegenerative disorder was 8.5% and 38.1%, respectively [9], which were comparable to the Caucasian figures [8]. In addition, presence of pre-morbid psychiatric disorder was associated with an increased risk of development of PD [24,26]. Wing and his colleagues found that a single night of vPSG in the presence of suggestive clinical history was adequate in establishing RBD diagnosis among 80-95% of the patients [22]. As there have been limited screening instruments for diagnosis and monitoring of RBD, they developed and validated a novel RBD instrument (RBDQ-HK) with satisfactory validity and reliability [23].

The further research work should be conducted to determine the early clinical and neurobiological markers in predicting an increased risk of neurodegeneration in idiopathic RBD patients, and to prospectively correlate the potential clinical markers with brain imaging abnormalities.

Acknowledgments

I would like to thank Dr. YK Wing for the assistance in the article editing.

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Received February 27, 2012; **Accepted** February 28, 2012; **Published** March 05, 2012

Citation: Xi Z (2012) REM Sleep Behavior Disorder (RBD)-A Potential Predictive Biomarkers of Future Neurodegeneration. J Sleep Disord Ther 1:e107. doi:10.4172/2167-0277.1000e107

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