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The implications of poor sleep quality on arterial health in persons with multiple sclerosis

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Abstract *Background:* Multiple sclerosis (MS) is associated with increased risk of cardiovascular disease (CVD) and approximately 25–54% of patients report poor sleep quality. There is evidence from the general population of an association between poor sleep and increased CVD risk, but this is poorly understood in MS.
Purpose: This study examined the association between self-reported sleep quality and arterial health in persons with MS.
Methods: MS subjects (n = 31) and control subjects (n = 23) were recruited. Control subjects were age and body size matched. All subjects were administered the Pittsburgh Sleep Quality Index (PSQI) to assess self-reported sleep quality. Subjects with a global score >5 were classified as "poor sleepers". Blood pressure, arterial stiffness, and the forearm blood flow responses following 5-min ischemic occlusion (endothelial function) were measured.
Results: Nineteen MS subjects and 5 control subjects were classified as "poor sleepers". AIx was significantly higher in MS subjects who had poor sleep quality (32.5 ± 8.8 vs 22.0 ± 13.2; $P < 0.05$).
Conclusions: Markers of arterial dysfunction were significantly higher in MS subjects with poor sleep quality compared to those with good sleep quality. This study suggests novel evidence for the association of CVD risk and sleep quality in MS.

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Introduction

Multiple Sclerosis (MS) is a chronic, autoimmune disease with an estimated prevalence of 2.5 million people worldwide.[1] The disease is characterized by demyelination and transection of axons in the central nervous system.[1,2] The pathophysiology directly and indirectly results in heterogeneous outcomes among those with MS. We are particularly interested in two of these outcomes, namely cardiovascular disease (CVD) and poor sleep quality.

Comorbidity is common in MS and there is an increased risk of cardiovascular disease (CVD) and early mortality[3] in this population. Furthermore, patients with MS report sleep disturbances more frequently compared to the general population.[1,4] Approximately 25%—54% of patients with MS report sleep related problems,[1,2] with sleep quality being associated with disease severity.[2] Shorter sleep duration and poor sleep quality have been associated with a higher risk of coronary heart disease, CVD, and overall mortality among healthy populations,[5—8] but this association is poorly understood in MS.

The pathophysiology of CVD and poor sleep quality is unknown in MS, but the inflammatory nature of the disease could be associated with both outcomes.[1] For example, reductions in sleep duration and sleep quality have been associated with a proinflammatory environment and disrupted immune function.[9—11] Acute increases in systemic inflammation result in temporary increases in large artery stiffness[12] and reduced endothelial function.[13] Increased aortic stiffness is a strong predictor of future cardiovascular events[14] and vascular dysfunction is a common comorbidity observed in MS patients.[15] Recently, it has been reported that patients with MS demonstrate altered arterial function compared to matched controls.[15] It is possible that the disruption of various regulatory systems associated with poor sleep could have detrimental effects on arterial function. However, the extent to which poor sleep habits in patients with MS affect arterial structure and function is unknown.

This study examined the relationship between sleep quality and arterial health among patients with MS and a matched control group. We hypothesized that sleep quality would be lower in individuals with MS and that poor sleep would be associated with worse arterial function.

Methods

The information presented is a secondary analysis on data previously published.[15] The study protocol was approved by the Institutional Review Board at the University of Illinois and all participants signed an informed consent document.

Participants

Thirty-three persons with MS were recruited from central Illinois. Age, sex, and body size matched control group (n = 33) were recruited from the community. All subjects were between the ages of 18—64 and both groups had equal number of women (n = 27) and men (n = 6). The current study includes subjects with complete arterial function and sleep quality data (MS n = 31; controls n = 23). Eligibility criteria included being ambulatory with or without single-point assistance, having the visual ability to read 14-point font, and having abstained from smoking for a minimum of six months. Additionally, MS subjects were included if they had been relapse free for at least 30 days prior to participating in the study. Persons who did not meet those criteria were excluded. Twenty-five of the subjects with MS were taking disease-modifying medications. Details regarding subject medications and disease severity have been described elsewhere.[15] All subjects abstained from caffeine and food for at least 4 h before the testing visit.

Sleep quality

Sleep quality was assessed with the Pittsburgh Sleep Quality Index (PSQI). The PSQI evaluates subjective sleep quality during the preceding month through 19 self-rated questions and five questions rated by a bed partner or roommate. The last five questions are used for clinical information and do not influence the final scoring of the PSQI. The 19 questions are grouped into seven component scores: sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction. The seven component scores are summed up to a global PSQI score ranging on a scale from 0 to 21. Higher scores indicate worse sleep quality, with a PSQI more than five discriminating between poor sleep (PSQI >5) from good sleep (PSQI ≤ 5).[16]

Brachial blood pressure

Subjects rested in a supine position for 10 min before resting measurements were obtained. Systolic BP (SBP) and diastolic BP (DBP) were measured using an automated oscillometric cuff (HEM-907XL; Omron Corporation, Kyoto, Japan). All BP measurements were obtained twice and the average of the two values was recorded.

Augmentation index

Applanation tonometry was performed to attain radial artery pressure waveforms using a high fidelity pressure transducer (SphygmoCor; AtCor Medical, Sydney, Australia). A central aortic pressure waveform was reconstructed from the radial artery pressure waveform using a generalized validated transfer function.[15] Augmentation index (AIx) was calculated as the difference between the early and late systolic peaks of the arterial waveform divided by the total pulse pressure. AIx was expressed as a percentage of the central pulse pressure. Because of the influence of HR, AIx was also normalized to a HR of 75 bpm. Central artery pressure waveforms were calibrated against brachial mean arterial and diastolic pressure.

Pulse wave velocity

To measure carotid-femoral pulse wave velocity (cf-PWV) we used a single high-fidelity pressure transducer to sequentially obtain pressure waveforms between the right common carotid and the right femoral artery, using a commercially available system (SphygmoCor, Atcor