Multiple Sclerosis Put On Pause: cardiogenic syncope with prolonged sinus pauses secondary to demyelinating brainstem lesions

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Cardiac abnormalities have been reported as a rare complication of multiple sclerosis (MS), possibly secondary to autonomic dysfunction. Neurogenic pulmonary oedema and systolic dysfunction are most common; symptomatic sinus bradycardia has only been described once. We present a young patient with newly-diagnosed MS and recurrent syncope associated with sinus pauses in the presence of multiple brainstem lesions.

A twenty-one year old male, presented with sudden onset left hemisensory loss. He reported an episode of sensory symptoms two years ago and another of persistent nausea and vomiting two months prior to his presentation; he had not sought medical attention. On examination he had a right-sided Brown-Sequard syndrome at T10 level; MRI revealed multiple hyperintense non-enhancing lesions in the periventricular and subcortical white matter, pons, corpus callosum, left middle cerebellar peduncle and medulla, as well as a lesion in the cervical cord(C6). After mimics were excluded, a diagnosis of Relapsing Remitting Multiple Sclerosis (RRMS) was made. In addition to his neurological symptoms, for 18 months prior to his presentation, he was under investigation for syncopal episodes; a 24h ECG showed 5.5 second pauses and a pacemaker was advised by cardiology. Thyroid function and autonomic function tests were normal. Given the MRI findings and absence of cardiac cause, the bradycardia was thought to be secondary to the brainstem lesions; a decision was made to delay pacemaker insertion due to the likelihood of improvement after acute relapse resolution. He was promptly started on Alemtuzumab; 6 months after his first infusion he had no further syncopal episodes and repeat imaging showed no new lesions and reduction in size of the brainstem lesions.

Conductive cardiac abnormalities are an extremely rare but important presentation of MS, particularly in patients with brainstem lesions. Symptoms seem to improve with resolution of relapse, thus delaying cardiac treatment, prompt acute relapse treatment and aggressive disease modifying treatment might be preferable in these patients.

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