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TRANSCRIPT

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Understanding Neurogenic Orthostatic Hypotension and the Role of Norepinephrine

Announcer:
Welcome to ReachMD.

This medical industry feature, titled “Understanding neurogenic orthostatic hypotension and the key role of norepinephrine” is sponsored by Lundbeck.

Your guest is Dr. Satish R. Raj. Dr. Raj is Professor of Cardiac Services, Medical Director of Calgary Autonomic Clinic, and Chief of Cardiac Arhythmia Service at the University of Calgary in Canada. He is the past-president of the American Autonomic Society and now serves on the board of directors.

Dr. Raj:
nOH is a rare but distinct subset of orthostatic hypotension caused by autonomic nervous system failure. It occurs frequently in patients with certain neurodegenerative disorders.

nOH reflects an inability to maintain an adequate blood pressure in response to the orthostatic challenge of standing up.

Although nOH presents as a hemodynamic problem, its potentially debilitating symptoms need to be the focus of clinical management. These can include dizziness, lightheadedness, blurry vision, fatigue, neck and shoulder pain, and cognitive impairment.

Still, understanding the underlying pathophysiology may help align management approaches to patient needs.

Normally, following a shift to an upright posture, blood shifts downward, to below the level of the heart, resulting in baroreceptors signaling the brain to initiate a compensatory response. Mediated by the neurotransmitter norepinephrine this signaling sustains blood pressure by promoting peripheral vasoconstriction.

In a setting of autonomic nervous system failure, inadequate norepinephrine release results in impaired vasoconstriction. Plasma norepinephrine levels may serve as one index of sympathetic function.

In a patient with nOH, normal to slightly reduced norepinephrine levels indicate lesions of preganglionic origin, as seen in Multiple System Atrophy, while very low levels of norepinephrine are characteristic of postganglionic lesions, observed in Pure Autonomic Failure. Both pre- and postganglionic lesions can be present in Parkinson’s disease patients with nOH. Their plasma norepinephrine levels typically fall below normal on standing up.

In symptomatic patients with nOH, blood pressure may be modulated by enhancing cholinergic activity in preganglionic sympathetic nerves, or replenishing endogenous norepinephrine in neuronal and non-neuronal tissues to activate both alpha-1 and alpha-2 adrenoreceptors in peripheral arteries and veins, or improving vascular resistance by exogenous stimulation of only the alpha-1 adrenoreceptors, or increasing sodium retention which expands blood volume to improve vascular pressure.

Changes in blood pressure from supine to standing are a hallmark metric of nOH. However, after diagnosis, it is essential that we direct our efforts to addressing the burden of nOH symptoms.

Non-pharmacological methods are a key component of nOH management and may be supplemented with pharmacological measures when symptoms persist.

Announcer:

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