Short Communication

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Parkinson Disease and Orthostatic Hypotension

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Introduction to Parkinson's disease and Orthostatic Hypotension:

Parkinson 's disease (PD) is a chronic and often progressive neurodegenerative disease, in its advanced stages often accompanied by postural instability and gait disturbance. [1] The main risk factor for PD is advancing age, with worldwide prevalence estimates ranging from 0.43% for those aged 60 to 69 years, 1% for those aged 70 to 79 and 1.9% for those over 80 years old. [1]

Pathophysiology of Orthostatic Hypotension in Parkinson's Disease:

Orthostatic hypotension (OH) could be observed in every third patient with Parkinson's disease during the course of the disease and it is mostly neurogenic (nOH). [2] It is thought to result from degeneration of the peripheral autonomic nervous system as the disease progresses, resulting in an inadequate response to gravity acting on effective circulatory volume on standing due to defective vasoconstriction and excessive venous accumulation of blood. Symptoms of OH are mainly related to cerebral and retinal hypoperfusion. [3] OH is defined as a decrease in systolic blood pressure of ≥20 mmHg or diastolic blood pressure of at least ≥10 mmHg within 3 minutes of standing.[4] It presents with syncope, unexplained falls, dizziness, cognitive impairment, blurred vision, dyspnea, fatigue, and shoulder, neck, or lower back pain that develops with standing and decreases with lying down. [2]

Prevalence and Impact of Orthostatic Hypotension in PD Populations:

The condition of people with a neurodegenerative disease that is the cause of OH will gradually worsen and the rate of progression will depend on the subtype of degenerative disease (Parkinson disease [PD] and dementia with Lewy bodies [DLB]progress more rapidly, multiple system atrophy[MSA] progresses most rapidly while pure autonomic failure progresses least slowly). [5] In a study to investigate the prevalence of orthostatic hypotension and its symptoms associated with the fall in blood pressure in patients with Parkinson's disease, a fall of at least 20 mm Hg of systolic blood pressure was found in 58.2% of the patients, postural fall in blood pressure was asymptomatic in 38.5% and was associated with postural events in 19.8% of the patients. Another study done aiming to determine the prevalence of OH in the PD patient population and to determine the demographics of patients with and without OH concluded that

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Forty-two (47%) patients met the OH criteria. People with OH were older than those without OH, but there was no difference in the duration or severity of PD. [7]

Screening and Diagnosis of Orthostatic Hypotension in PD:

It is recommend screening the patients at high risk for OH, especially those with suspected or diagnosed neurodegenerative disorder associated with autonomic dysfunction such as PD as per the guidelines issued by American Autonomic Society and the National Parkinson Foundation consensus panel. [8] OH screening begins with questioning to identify symptoms of OH, patients should be inquired about the main symptoms of OH, their frequency, and the impact of those symptoms on their everyday life, followed by blood pressure measurements in supine (or sitting) and standing positions. [8] Other factors that may cause patient to fall frequently such as cognitive impairment, postural instability, gait disturbances, poor vision, impaired proprioception and physical environment factors should also be considered by the physicians while asking about the symptoms. [1] The recommended gold standard for measuring OH is having the patient lie on his or her back for at least 5 minutes, then stand for 3 minutes, and measuring blood pressure just before standing up, and 1 and 3 minutes after standing. [8] When BP measurement in the supine position is not possible, BP measurement in the seated to standing position may be a suitable alternative. [8]

Management and Treatment Strategies for Orthostatic Hypotension:

In OH, the primary goal of treatment is not to reduce the degree of hypotension caused by changes in standing, but to alleviate symptoms. [9] Patients with PD+OH can often tolerate a significant drop in blood pressure without any symptoms.[10] A proposed OH treatment algorithm consists of a 4-step hierarchical process:(1)evaluating and adjusting existing drugs, (2) using non- pharmacological approaches, (3) using single drug treatments, and (4) use multi drug therapy with extreme caution , caution.

At each stage, the patient should undergo a 2-week evaluation to determine whether adequate improvement in symptoms has been achieved before proceeding to further steps. [8,9]

Non-pharmacological treatment:

Orthostatic hypotension should be treated primarily with non-pharmacological methods such as patients should be instructed to eat small, frequent meals, drink plenty of fluids, and avoid rapid position changes or prolonged standing, use lower body–only compression garments such as knee-length compression stockings and water bolus ingestion (rapid ingestion of 500ml of tap water over 3 to 4 minutes) and do physical counter maneuvers (leg crossing, squatting, stooping and tensing muscles of whole body, arms, legs, abdomen or buttocks). [10]

If non-pharmacological measures do not lead to an adequate improvement in the symptoms of OH, then drug therapy should be initiated especially for patients with syncope, presyncope , or falls, the potential consequences are so severe that some clinicians consider it appropriate to prescribe drug therapy at the start of treatment. [8]

Pharmacological treatment:

A norepinephrine prodrug, droxidopa has been approved by the U.S. Food and Drug Administration (FDA) for the treating symptoms of nOH with an underlying disautonomia.[11] Droxidopa is a short-acting agent, its peak response occurs within approximately 3.5 hours after taking orally. The recommended dosage varies from 100 mg to 600 mg up to 3 times a day. [9] Midodrine which is a short acting drug that causes standing systolic BP to rise by 10 mmHg to 30mmHg, 1 hour after a 10 mg dose with its effect persisting for up to 3 hours, 10mg dose can be then increased up to 10 mg thrice a day, has also been approved for treatment of symptomatic OH, including nOH.[9,11] Fludrocortisone is a synthetic mineral corticoid that causes the plasma volume to increase by mechanism of sodium retention. It is used alone

or in combination with midodrine, but no studies have compared the efficacy and safety of long-term monotherapy and combination regimens. [12] Erythropoietin in combination with iron preparations may have a beneficial effect on OH in patients with concomitant anemia, however, the risk of polycythemia vera and thrombotic complications should be taken into account. [13] In severe cases where symptoms are persistent or are related to injury, treatment with fludrocortisone, desmopressin, octreotide, methylphenidate or yohimbine may be required. [10] Supine hypertension can present in up to 50% of patients with nOH. Non-pharmacological interventions include elevating the head of the bed during night, snacking to induce postprandial hypotension, and avoiding lying down unless sleeping. In rare cases, drug therapy is required. The drugs used include short-acting, low-dose angiotensin-converting enzyme inhibitors (e.g. captopril), short-acting calcium channel blockers (e.g. nifedipine), and nitroglycerin delivered by a patch.[14]

Challenges and Considerations in Treating OH in PD Patients: About 30% of people with Parkinson's disease (PD) have neurogenic OH, a frequent and potentially dangerous non-motor illness characterized by a persistent drop in blood pressure upon standing and an insufficient compensatory heart rate increase. [11] OH is caused by the autonomic nerve system degeneration and can affect a patient's daily activities. Patients with Parkinson's disease should be screened for OH using orthostatic symptom questionnaires, orthostatic blood pressure readings, and (if required) specialized autonomic testing. This will help in identifying symptomatic and asymptomatic cases and help in starting prompt treatment. Both non-pharmacologic treatments and pharmaceutical ones are effective for OH symptoms. Supine hypertension can occur in OH patients, which affects medical therapy choices. Adrenoceptor agonists should be taken into consideration early on in the pharmacological therapy of OH in PD due to denervation supersensitivity. Short-acting medications such nitroglycerin, clonidine,or angiotensin receptor blockers should be used to treat nocturnal SH. Future studies are required to better comprehend and reduce blood pressure variations by enhancing baroreflex sensitivity.

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