RVS
RESEARCH IN VESTIBULAR SCIENCE

Review Article

pISSN 2092-8882 • eISSN 2093-5501
Res Vestib Sci 2024;23(3):79-88
https://doi.org/10.21790/rvs.2024.017

Treatment of hemodynamic orthostatic dizziness/vertigo

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Orthostatic dizziness occurs when a person feels dizzy or lightheaded upon standing up. Hemodynamic orthostatic dizziness can result from autonomic dysfunction, such as orthostatic hypotension or postural tachycardia syndrome. The International Classification of Vestibular Disorders has established diagnostic criteria for hemodynamic orthostatic dizziness/vertigo. These criteria help clinicians understand the terminology associated with orthostatic dizziness/vertigo and differentiate it from dizziness caused by global brain hypoperfusion and other etiologies. Effective treatment involves interpreting the results of autonomic function tests, which can lead to improvements in orthostatic dizziness and help prevent falls related to this condition. This paper discusses general management strategies and specific treatments for orthostatic hypotension and postural tachycardia syndrome, highlighting the importance of tailored care based on the most recent clinical insights.

Keywords: Treatment; Orthostatic intolerance; Hemodynamics; Autonomic nervous system; Orthostatic hypotension; Postural tachycardia syndrome

INTRODUCTION

Orthostatic dizziness (OD) refers to dizziness or vertigo that occurs during orthostasis, specifically when moving from a supine position to sitting or standing, or from sitting to standing [1]. OD is a common symptom of orthostatic intolerance, which encompasses all symptoms that appear upon standing and are alleviated by lying down [2-5]. The best-known cause of OD is global cerebral hypoperfusion (hemodynamic OD), which can result from conditions such as orthostatic hypotension (OH) or postural tachycardia syndrome (POTS). The International Classification of Vestibular Disorders has defined diagnostic criteria for hemodynamic OD to aid clinicians in understanding the terminology and distinguishing between hemodynamic OD and dizziness caused by other factors (Fig. 1) [6]. Additional causes of OD include bilateral vestibulopathy, peripheral neuropathy, functional dizziness, orthostatic tremor, or gait disorders [7,8]. Hemodynamic OD typically presents with symptoms such as non-spinning dizziness, lightheadedness, and a feeling of an impending blackout or fainting. However, OH can also lead to orthostatic vertigo, characterized by spinning or other sensations of self-motion, particularly in patients with poor autonomic regulation [9,10]. POTS can be diagnosed clinically through active standing without extensive testing. Nonetheless, diagnosing POTS is challenging due to its nonspecific symptoms, which are indirectly related to orthostatic intolerance and overlap with symptoms of similar conditions [11,12]. Comorbid psychiatric conditions, such as untreated major depression, can com-

Received: August 30, 2024 **Revised:** September 8, 2024 **Accepted:** September 9, 2024

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Hemodynamic orthostatic dizziness/vertigo

Criteria A–C should be fulfilled to make the diagnosis of hemodynamic orthostatic dizziness/vertigo.

A. Five or more episodes of dizziness, unsteadiness or vertigo triggered by arising (i.e. a change of body posture from lying to sitting/standing or sitting to standing), or present during upright position, which subsides by sitting or lying down

B. Orthostatic hypotension, postural tachycardia syndrome or syncope documented on standing or during head-up tilt test

C. Not better accounted for by another disease or disorder

Probable hemodynamic orthostatic dizziness/vertigo

Criteria A–C should be fulfilled to make a diagnosis of probable hemodynamic orthostatic dizziness/vertigo.

A. Five or more episodes of dizziness, unsteadiness or vertigo triggered by arising (i.e. a change of body posture from lying to sitting/standing or sitting to standing), or present during upright position, which subsides by sitting or lying down

B. At least one of the following accompanying symptoms
- Generalized weakness or tiredness
- Difficulty of thinking or concentration
- Blurred vision
- Tachycardia or palpitations

C. Not better accounted for by another disease or disorder

Fig. 1. Diagnostic criteria for hemodynamic orthostatic dizziness/vertigo.

plicate the evaluation but are crucial to recognize and address to enhance the clinical response to treatment [13].

Although previous epidemiologic studies of OD have yielded varying results based on their methodology, the data indicate that OD is relatively common across a broad age spectrum. The sole population-based study [14] examining spontaneously occurring OD in a wide age range found that the one-year and lifetime prevalence rates were 10.9% and 12.5%, respectively. OD represented 42% of all cases among participants experiencing dizziness/vertigo and accounted for 55% of non-vestibular dizziness diagnoses [15]. In several larger community-based studies focusing on individuals over 60 years old, the reported prevalence of dizziness upon standing ranged from 2% to 30% [16-18]. In a study utilizing questionnaires among young adults, the incidence of spontaneously occurring OD was 41% among healthy medical students and 57% among females [15,19].

OH is defined by a significant drop in blood pressure (BP) upon standing, whereas POTS is characterized by an excessive increase in heart rate (HR). Orthostatic BP and HR measurements are the most important screening tests for autonomic dysfunction. The head-up tilt test exhibits higher reproducibility in patients with severe and frequent orthostatic symptoms than in patients with milder symptoms. In a previous study, only approximately half of the patients with orthostatic intolerance showed changes in BP or HR during the head-up tilt test that satisfied the criteria for OH or POTS. Moreover, only 25% of the patients were symptomatic during the head-up tilt test [20]. Although the head-up tilt test is the most widely used method to identify sympathetic adrenergic failure, its limited sensitivity and reproducibility mean that it can detect only moderate to severe generalized adrenergic failure [21,22]. In contrast, the Valsalva test has the advantage of detecting milder forms of sympathetic adrenergic failure [22].

A comprehensive diagnostic approach is crucial for identifying the underlying cause of hemodynamic OD, as it may be associated with serious conditions such as autonomic disorders or systemic hypotension resulting from bleeding. Additionally, determining the specific subtype and severity of autonomic dysfunction is essential to guide the selection of appropriate treatment strategies.

The management of hemodynamic OD, whether associated with OH or POTS, is complex and necessitates a tailored approach that takes into account the patient's overall health, specific symptoms, and underlying pathophysiology. Numerous studies have focused on treating OH or POTS, yet there are no established guidelines for managing OD specifically. Although the treatment of hemodynamic OD ultimately involves addressing OH or POTS, there are comprehensive strategies that can guide clinicians on when to treat patients diagnosed with OD.

https://doi.org/10.21790/rvs.2024.017

MAIN SUBJECTS

The General Approach for the Management of Hemodynamic Orthostatic Dizziness

When managing patients with OD, there are several strategies we must consider.

First, the treatment of hemodynamic OD may vary based on the specific type of OH or POTS present, such as neurogenic versus non-neurogenic OH, and whether it occurs with or without supine hypertension, or if it is hyperadrenergic, neuropathic, or hypovolemic POTS. Diagnostic approaches may include performing the Valsalva test or assessing the HR to BP ratio during tilt or standing tests. Non-neurogenic OH may be reversible through medication management, correction of dehydration, or engagement in physical exercise. It is crucial to identify and address the underlying causes. Neurogenic OH, on the other hand, is often associated with conditions that exacerbate OH, such as deconditioning, anemia, or weight loss due to cancer or chemotherapy.

Second, the treatment of hemodynamic OD focuses on alleviating symptoms and enhancing quality of life. It is important to recognize that the goal of treating neurogenic OH is not to normalize BP. Instead, the objective is to achieve a moderate pressor effect that is sufficient to increase standing BP and reduce orthostatic symptoms, while avoiding excessive supine hypertension. Practical BP targets generally include a standing systolic BP (SBP) of at least 90 mmHg and a supine SBP of no more than 180 mmHg. Similarly, there is no cure for POTS, and treatment focuses on symptom relief rather than achieving specific hemodynamic goals [23].

Third, hemodynamic OD varies based on several factors, including volume status, temperature, and meal intake. Clinicians must be aware that the magnitude of BP drop or HR increase can change according to these conditions. It is important for them to educate patients on avoiding circumstances that may exacerbate their symptoms.

Finally, at-home BP monitoring is crucial for minimizing BP drops, especially in patients with OH. Patients with neurogenic OH may also experience supine hypertension. It is important to instruct patients to measure their BP after lying supine for 5 minutes or before getting up in the morning, and to repeat the BP measurement after standing for 3 minutes. Additionally, repeating the BP measurement while standing can be beneficial when symptoms are present.

The general approach involves a stepwise strategy including non-pharmacological interventions, patient education, and, if necessary, pharmacological treatments tailored to the individual patient's needs.

Treatment of Orthostatic Hypotension

Non-pharmacological interventions for patients with orthostatic hypotension

Non-pharmacological strategies, including patient education, are fundamental in managing OH. Due to the variability in hemodynamic responses to external and internal environmental changes in patients with OH, it is crucial to educate them about the nature of their condition. They should understand the importance of staying hydrated and how to manage their symptoms through lifestyle adjustments. Patients with long-standing OH may experience asymptomatic periods (hypotension unawareness) or display atypical symptoms such as neck or shoulder pain (coat hanger pain), muscle weakness, or clouded thinking. If patients fail to recognize that these symptoms are related to OH, they may be at risk of sudden falls or syncope. It is important for patients to be aware that OH symptoms can worsen in the early morning or during a shower at night.

All medications should be reviewed for patients with OH. It is often necessary to stop or reduce the dosage or frequency of various drugs, not just hypertensive medications. Tricyclic antidepressants, central or peripheral αantagonists, and β-blockers may exacerbate OH. When starting a new medication, patients should consult their doctor to ensure that it does not adversely affect their condition.

Patients should also be advised to avoid large meals, alcohol, hot weather, or hot baths, as these can exacerbate OH. Postprandial hypotension, which can occur after consuming a large meal, may be prevented by eating frequent snack-sized meals or using acarbose. Acarbose works by competitively inhibiting the αglucosidase enzyme in the small intestine lumen, thereby slowing carbohydrate digestion. Additionally, drinking one or two cups of coffee in the morning with breakfast (but not later in the day) can help induce vasoconstriction and

increase BP. Falls in the bathroom are common among patients with OH. Since these patients often need to use the bathroom in the morning to urinate, they are naturally at an increased risk of falling. Straining during defecation can cause a drop in BP due to a decrease in venous return from intraabdominal pressure. To help prevent falls, it is recommended that patients urinate while seated and take measures to prevent constipation. Having a companion for assistance is also recommended to further reduce the risk of falling.

Most patients with non-neurogenic OH only require non-pharmacological interventions that address potential causes. Conversely, patients with neurogenic OH may need to commence pharmacological treatments alongside non-pharmacological interventions promptly to prevent falls. Patients should be advised to stand up gradually from a sitting or lying position and to avoid remaining stationary for extended periods. They should also avoid lying down during the day to prevent deconditioning and the risk of elevated BP due to supine hypertension. To help increase BP while standing, patients can employ physical counter-maneuvers such as crossing their legs, squatting, and engaging in isometric exercises like handgrips.

Increasing salt intake and ensuring adequate hydration are essential for expanding blood volume. Administering a cold water bolus is advantageous for patients before they venture outdoors. Consuming 500 mL of cold water can elevate systolic BP by approximately 20 mmHg for 2 hours. Patients with specific conditions, such as chronic renal failure, who must limit their water and salt intake should adhere to their prescribed guidelines. To prevent frequent nocturia, which can lead to falls and supine hypertension, water consumption should be scheduled for the evening. Short-term studies have shown that increased salt intake can alleviate symptoms of orthostatic intolerance. However, there are no reports on the effects of long-term increased salt intake on falls or cardiovascular risk.

A structured exercise program, which includes aerobic exercise and resistance training, is recommended for improving cardiovascular conditioning. Specifically, a program that incorporates recumbent exercises, such as rowing or cycling, can effectively condition the cardiovascular system and alleviate symptoms. It is important to gradually increase the intensity of exercise to prevent exacerbation of symptoms. Patients with OH must be particularly cautious of post-exercise hypotension.

Elevating the head-end of the bed to allow for gravitational stress during sleep can enhance outcomes in patients severely affected by OD. This approach helps mitigate nocturnal hypertension and reduces both nocturia and the overnight depletion of plasma volume, thereby promoting a more favorable distribution of body fluids [24,25]. Smaller tilting angles do not effectively improve OD [26]; therefore, steeper angles are recommended, specifically elevating the head-end using blocks of 20 to 30 cm. To prevent patients from sliding down while in a supine sleep position, placing a firm pillow under the mattress at the thigh level can be effective, and using a footboard may also be beneficial. Additionally, an adjustable electric bed or mattress offers a practical solution for facilitating head-up sleeping.

Compression stockings, which extend to the upper thigh, are challenging to put on and provide only a marginal improvement in standing BP [27]. The abdomen is the most effective site for compression to counteract OH, as it reduces splanchnic blood pooling. Elastic abdominal binders may be beneficial for highly symptomatic patients willing to use this approach [27,28], although they can be uncomfortable, particularly in hot weather. Patients should be advised to wear their abdominal binders or compression stockings only while active. For some individuals, a small, portable, and lightweight folding chair can be useful. It allows them to sit down and manage or resolve an episode when presyncopal symptoms occur upon standing. The height of the chair is crucial, as lower chairs are more effective in eliciting a positive BP response [29].

Severe dysautonomia can lead to arrhythmias such as atrial fibrillation, tachy-brady syndrome, or heart block. The symptoms of these arrhythmias can mimic those of OH. In some patients with neurogenic OH, electrocardiography and continuous HR monitoring are necessary to detect potentially dangerous arrhythmias.

Pharmacological interventions for patients with orthostatic hypotension (Table 1)

When non-pharmacological measures are insufficient, pharmacological therapy is considered. The selection

https://doi.org/10.21790/rvs.2024.017

of a specific medication is guided by the severity of OH, the presence of supine hypertension, and the patient's comorbid conditions.

Midodrine, an α1 adrenergic agonist, enhances peripheral vascular resistance, which in turn improves BP upon standing. It is generally administered in doses of 2.5 to 10 mg three times daily. However, evening doses should be avoided to prevent supine hypertension.

Fludrocortisone is a mineralocorticoid that enhances sodium retention and blood volume. The usual dosage ranges from 0.1 to 0.2 mg daily; however, this requires vigilant monitoring for potential side effects, including hypokalemia and supine hypertension. Long-term use of fludrocortisone at high doses is associated with increased risks of heart failure, renal fibrosis, and hospitalization for various causes [30,31].

Another agent used to increase blood volume is desmopressin, a synthetic analog of vasopressin. Desmopressin works by reducing urine output, which helps retain water and increase blood volume. It is occasionally used in patients who do not respond well to fludrocortisone. However, it carries a risk of hyponatremia, necessitating regular monitoring of sodium levels.

Droxidopa is a prodrug of norepinephrine that enhances sympathetic activity, thereby increasing BP. It is particularly effective in patients with neurogenic OH and is typically administered in doses ranging from 100 to 600 mg three times daily. The effectiveness of droxidopa in elevating BP largely depends on the extent of postganglionic sympathetic denervation. Patients with low plasma norepinephrine levels prior to treatment tend to experience the most significant pressor response [32,33]. In contrast, noradrenaline-reuptake inhibitors, such as atomoxetine, exert a pressor effect when peripheral sympathetic neurons are intact. This includes cases involving preganglionic or premotor sympathetic lesions, such as those seen in multiple system atrophy [33].

Pyridostigmine is an acetylcholinesterase inhibitor that can be used as an adjunct therapy in patients with refractory OH. It enhances cholinergic transmission, which in turn increases peripheral vascular resistance. When used alone, pyridostigmine produces only modest pressor effects [34,35]. However, it demonstrates synergistic effects when combined with midodrine or atomoxetine [30,31].

Treatment of supine hypertension

Supine hypertension is a common complication in patients with neurogenic OH. This condition poses significant management challenges, particularly because it often occurs alongside severe OH during daytime hours. Effective management of supine hypertension is critical for preventing cardiovascular complications and im-

Table 1. Pharmacological treatments of orthostatic hypotension

Pharmacological agent	Mechanism	Recommended dose	Side effects
Droxidopa	Short-acting synthetic norepinephrine precursor	100–600 mg 3 times a day (morning, midday, and 3–4 hours before going to bed) or tailored to the patient's need	Supine hypertension, headache, nausea, fatigue; use with caution in patients with congestive heart failure or chronic renal failure
Midodrine	Short-acting direct α1 adrenergic receptor agonist	2.5–15 mg 2 or 3 times a day (morning, midday, and 3–4 hours before going to bed) or tailored to the patient's need	Supine hypertension, piloerection (goose bumps), itchy scalp, urinary retention; use with caution in patients with congestive heart failure or renal failure
Fludrocortisone	Long-acting synthetic mineralocorticoid (volume expansion that increases sodium and water reabsorption)	0.05–0.2 mg once a day; no benefit with doses higher than 0.2 mg/day	Supine hypertension, hypokalemia, renal failure, edema, end organ damage; use with caution in patients with congestive heart failure
Pyridostigmine	Short-acting acetylcholinesterase inhibitor	30–60 mg 2 or 3 times a day	Abdominal cramps, diarrhea, sialorrhea, excessive sweating, urinary incontinence
Atomoxetine	Short-acting norepinephrine reuptake inhibitor	10–18 mg twice a day	Supine hypertension, insomnia, irritability, decreased appetite
Acarbose	Short-acting αglycosidase inhibitor	50–150 mg before meals to prevent postprandial hypotension	Abdominal gas, bloating

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proving overall patient outcomes. Supine hypertension is characterized by a systolic BP of 140 mmHg or higher and/or a diastolic BP of 90 mmHg or higher, measured after at least 5 minutes of rest in the supine position. Managing this condition is especially difficult due to its coexistence with neurogenic OH. It requires careful balancing to avoid worsening orthostatic symptoms while also preventing the potential complications associated with prolonged hypertension. The primary goal in managing supine hypertension in patients with neurogenic OH is to mitigate the risk of cardiovascular events while avoiding exacerbation of orthostatic symptoms. Treatment for supine hypertension is recommended when the systolic BP exceeds 160 to 180 mmHg, particularly if it remains elevated throughout the night.

Non-pharmacological strategies are the primary approach to managing supine hypertension. These interventions focus on reducing nighttime BP elevations and increasing overall BP stability. One effective method is to elevate the head of the bed by 30°, which can decrease pressure at the carotid sinus and cerebral vasculature levels. Patients are advised to avoid lying down during the day; instead, they should rest in a seated position to help prevent the development of supine hypertension during waking hours. Restricting fluid intake 60 to 90 minutes before bedtime can also diminish the osmopressor response and avert excessive nighttime BP increases. Consuming a carbohydrate-rich snack or alcohol before bedtime may assist in modulating BP throughout the night. When non-pharmacological measures prove inadequate, pharmacological interventions may become necessary. However, treating supine hypertension in patients with neurogenic OH requires careful consideration due to the risk of worsening OH. Alpha-blockers, central sympatholytics, and diuretics are generally contraindicated as they can exacerbate OH. Preferably, treatment options include angiotensin receptor blockers, angiotensin-converting enzyme inhibitors, and calcium channel blockers. Antihypertensive medications should ideally be administered at bedtime to decrease nighttime BP without adversely affecting daytime orthostatic BP. Losartan and clonidine have demonstrated some effectiveness in reducing nighttime natriuresis and lowering supine hypertension, without significantly affecting morning OH. However, the administration of these medications necessitates careful monitoring and should be tailored to individual patient responses. Pressor agents such as midodrine and droxidopa, which are typically used to manage OH, must be used with caution and should not be taken close to bedtime to avoid exacerbating supine hypertension.

Treatment of Postural Tachycardia Syndrome

Treating POTS is often complex and necessitates a tailored approach, as no single therapy has proven universally effective. There is ongoing debate about whether certain subsets of POTS patients respond differently to treatments or whether a standardized approach should be applied to all patients [36]. While there is no cure for POTS, a combination of non-pharmacological and pharmacological strategies can help manage symptoms. Importantly, POTS is not associated with increased mortality; therefore, treatment focuses on alleviating symptoms and enhancing the patient's quality of life. Continuous monitoring and adjustment of treatment regimens are crucial, as patients may develop tolerance to certain medications or experience changes in their symptoms over time. Despite the variety of pharmacological options available, treating POTS remains challenging due to the absence of large, randomized controlled trials that could provide definitive evidence of the efficacy and safety of these therapies.

Non-pharmacological interventions for patients with postural tachycardia syndrome

Non-pharmacological approaches are the cornerstone of POTS management and should be considered the first line of treatment. These approaches are similar to the non-pharmacological interventions used in treating OH. Managing POTS fundamentally involves non-pharmacological interventions, which should be used alongside pharmacotherapy. These interventions include lifestyle modifications such as increased intake of salt and fluids, regular physical exercise, and the use of compression garments. Given that POTS patients tend to be younger, there may be a greater emphasis on supine and seated exercise therapies early in the treatment process compared to those with OH. While it is important to initiate non-pharmacological approach-

https://doi.org/10.21790/rvs.2024.017

es early, some patients may require pharmacological therapies from the first visit. However, if these measures do not adequately control symptoms, pharmacological interventions may become necessary to improve quality of life and prevent disability [37]. Although starting with non-pharmacological strategies alone is often reasonable, some patients may present with severe symptoms that necessitate the early introduction of medications alongside these foundational approaches.

Pharmacological interventions for patients with postural tachycardia syndrome (Table 2)

The primary goal of treatment is to alleviate symptoms rather than to achieve specific hemodynamic targets. While the FDA has not approved any medications specifically for POTS, various drugs are commonly used off-label to manage its symptoms. The pharmacological treatment of POTS targets several aspects of the condition, including blood volume expansion, HR reduction, peripheral vasoconstriction, and modulation of autonomic nervous system function [23]. Given the heterogeneity of POTS, pharmacological treatment must be individualized, taking into account the patient's specific symptoms, response to medications, and any comorbid conditions [23].

HR reduction is a crucial aspect of POTS management, particularly for patients who experience significant palpitations. Beta-blockers, such as propranolol, are often prescribed at low doses to decrease HR and can help alleviate symptoms in cases of hyperadrenergic POTS, characterized by excessive sympathetic activity. However, beta-blockers may cause adverse effects, including fatigue, cold extremities, and worsening hypotension. Therefore, the use of beta-blockers in young women with low BP should be approached with caution.

Ivabradine, which specifically inhibits the If current in the sinoatrial node, offers an alternative for reducing HR in patients with POTS. Unlike beta-blockers, ivabradine does not impact BP, which makes it especially beneficial for patients who cannot tolerate beta-blockers. However, ivabradine can cause side effects, including visual disturbances and bradycardia, necessitating careful selection and monitoring of patients.

Peripheral vasoconstrictors are useful in managing POTS, especially for patients who experience low BP upon standing. Midodrine is notably effective for those who suffer significant drops in BP in such situations. However, the side effects of midodrine, such as scalp tingling, supine hypertension, and piloerection, require careful management. Droxidopa may also be suitable for some cases of POTS. Nonetheless, it can lead to su-

Table 2. Pharmacological treatments of postural tachycardia syndrome

Pharmacological agent	Mechanism	Recommended dose	Side effects
Propranolol	Heart rate inhibitors, beta-blocker	10–20 mg orally up to four times daily	Hypotension, bradycardia, bronchospasm
Ivabradine	Heart rate inhibitors, specifically inhibit the If current in the sinoatrial node	2.5–7.5 mg orally twice daily	Headaches, palpitations, hypertension, visual disturbances
Midodrine	Short-acting direct α1 adrenergic receptor agonist	2.5–15 mg 2 or 3 times a day (morning, midday, and 3–4 hours before going to bed) or tailored to the patient's need	Supine hypertension, piloerection (goose bumps), itchy scalp, urinary retention; use with caution in patients with congestive heart failure or renal failure
Fludrocortisone	Long-acting synthetic mineralocorticoid (volume expansion that increases sodium and water reabsorption)	0.05–0.2 mg once a day; no benefit with doses higher than 0.2 mg/day	Supine hypertension, hypokalemia, renal failure, edema, end organ damage; use with caution in patients with congestive heart failure
Pyridostigmine	Short-acting acetylcholinesterase inhibitor	30–60 mg 2 or 3 times a day	Abdominal cramps, diarrhea, sialorrhea, excessive sweating, urinary incontinence
Clonidine	Sympatholytic drugs, act on central α2 adrenergic receptors to decrease sympathetic outflow	0.1–0.2 mg orally 2–3 times daily or long-acting patch	Hypotension, fatigue, brain fog
Droxidopa	Short-acting synthetic norepinephrine precursor	100–600 mg 3 times a day (morning, midday, and 3–4 hours before going to bed) or tailored to the patient's need	Supine hypertension, headache, nausea, fatigue; use with caution in patients with congestive heart failure or chronic renal failure

pine hypertension and headaches, and its use should be tailored to the patient's specific needs.

Fludrocortisone is commonly prescribed for patients with POTS to increase plasma volume. However, it carries potential side effects including acne, irregular menstruation, hypokalemia, supine hypertension, and fluid retention, which necessitate careful monitoring.

By inhibiting the breakdown of acetylcholine, pyridostigmine enhances parasympathetic activity and improves autonomic balance. This leads to better orthostatic tolerance and a reduced HR increase upon standing. Despite its potential benefits, pyridostigmine can cause gastrointestinal side effects, including diarrhea and abdominal cramps, which may limit its use in some patients. In certain cases, central sympatholytic agents like clonidine are considered for the management of POTS, particularly in hyperadrenergic POTS accompanied by orthostatic hypertension. Clonidine targets central α2 adrenergic receptors, reducing sympathetic outflow, which in turn lowers HR and BP. While clonidine can be effective, it is linked to side effects including sedation, dry mouth, and the risk of rebound hypertension when discontinued. This necessitates its cautious use and close monitoring.

Emerging evidence suggests that POTS may have an autoimmune basis in some patients. If this is confirmed, it could significantly change treatment approaches. Treatments such as intravenous immunoglobulin, plasmapheresis/plasma exchange, and other immunotherapy drugs might become viable options for clearing these antibodies. However, clinical trials are still ongoing to determine their effectiveness, and immunotherapies are not currently recommended for POTS [23]. A recent single-center randomized controlled trial found no difference in symptomatic response between intravenous immunoglobulin and albumin in patients who met predetermined criteria suggesting autoimmunity [38]. Further research through larger, randomized controlled trials is necessary.

CONCLUSION

Hemodynamic OD, which includes both OH and POTS, poses significant challenges in both diagnosis and management. A comprehensive treatment strategy, incorporating both non-pharmacological and pharmacological interventions tailored to the specific needs of the individual patient, is crucial for optimizing outcomes. Successful management hinges on patient education, meticulous monitoring, and a multidisciplinary approach for each patient.

ARTICLE INFORMATION

Funding/Support

None.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

Availability of Data and Materials

All data generated or analyzed during this study are included in this published article. For other data, these may be requested through the corresponding author.

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https://doi.org/10.21790/rvs.2024.017

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https://doi.org/10.21790/rvs.2024.017