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Venous Angioplasty and Stenting as a Novel Therapeutic Strategy for Orthostatic Hypotension: A Retrospective Review

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Abstract

Background: Orthostatic hypotension (OH) is identified by a significant decrease in blood pressure upon standing from a seated or supine position. A reduction in systolic blood pressure of 20 mmHg within three minutes of standing meets the criteria for clinical diagnosis. We hypothesized that venous outflow obstruction from jugular valvular dysfunction or extrinsic compression of the left brachiocephalic vein may cause OH. Improving venous return and reducing venous congestion of the autonomic pathways through endovascular intervention could alleviate symptoms. Methods: This retrospective review included six male patients (aged 63–87) with medically refractory OH who underwent venograms revealing jugular, brachiocephalic, or subclavian vein stenosis. Patients were treated with balloon angioplasty and/or stenting. Blood pressure was measured in supine, seated, and standing positions before and immediately after the procedure, with multiple readings per position (total n = 117 for supine-standing comparisons). Statistical analysis used Welch's *t*-test to compare pre- and post-procedural systolic blood pressure disparities. Results: The patients showed improved post-procedural blood pressure and reduced OH symptoms. The average supine-standing systolic disparity decreased from 38.68 mmHg preoperatively to 24.61 mmHg postoperatively ($p = 0.024$). The seated-standing disparity was insignificant, possibly due to autonomic compensation. Patients also reported relief from associated symptoms like headaches, tinnitus, and vertigo. Conclusions: These findings suggest venous outflow obstruction may contribute to OH, and venoplasty/stenting can mitigate symptoms, potentially reducing reliance on medications with adverse effects. Further studies should explore the role of Venous Outflow Obstruction Disorders in neurological conditions.

Keywords: orthostatic hypotension; endovascular intervention; venous stenosis; venous stent

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1. Introduction

Orthostatic hypotension (OH) is characterized by a sharp drop in blood pressure upon standing. OH can be diagnosed clinically by a decrease in systolic blood pressure of at least 20 mmHg or a 10 mmHg reduction in diastolic blood pressure within three minutes of standing [1,2]. OH has become an increasingly prevalent condition, particularly amongst older individuals, impacting 16–30% of adults aged 65 years or older [3]. 50–65% of the institutionalized elderly population is disproportionately affected by OH [4]. Managing

hypertension and OH in the elderly presents a challenging balance [5]. This reduction in blood pressure attenuates cerebral perfusion, resulting in blurred vision, dizziness, lightheadedness, nausea, and syncope. Fainting, in particular, leads to recurring falls, hospitalization, and death for patients with OH [6,7]. Orthostatic hypotension can increase a patient's risk of atrial fibrillation, stroke, venous thromboembolism, and chronic kidney disease [8–10], creating a need to pursue a viable treatment. Current pharmacological treatments include midodrine hydrochloride (Proamatine) [11], fludrocortisone (Florinef) [12], droxidopa (Northera) [13], and even pyridostigmine (Mestinon) [14,15]. However, midodrine is associated with numerous side effects, including adverse urological effects and supine hypertension [16,17], while fludrocortisone is linked to hypokalemia and metabolic alkalosis [18], and congestive heart failure [19]. Droxidopa (Northera) can cause headaches, nausea, fatigue, and palpitations [13], and pyridostigmine can cause fatigue, flatulence, blurred vision, muscle weakness, urinary urgency, increased lacrimation, and excessive belching [20]. The supracardiac venous system comprises several major vessels, including the subclavian, jugular, and brachiocephalic veins [21]. The internal jugular veins and subclavian veins contain one-way valves anchored in the tunica intima, preventing the backflow of blood [22]. Dysfunction of these valves, specifically in the internal jugular veins, can cause cerebral venous outflow obstruction and venous congestion. Additionally, intimal hyperplasia, the growth of tunica intima cells accompanied by an increase in extracellular matrix, is an intrinsic cause of venous stenosis [23,24]. The placement of pacemaker wires, central venous catheters, and hemodialysis catheters, compounded by intimal hyperplasia and fibrosis, leads to venous stenosis in the upper extremities, predominantly affecting the brachiocephalic, subclavian, and jugular veins [25,26]. Extrinsic aortosternal venous compression of the left brachiocephalic vein is also clinically significant, as it can further impair venous outflow and reduce venous return in patients [27]. Venous return is a critical contributor to systemic blood pressure, impacting ventricular preload, which correlates with the systolic pressure and stroke volume of the impending cardiac cycle. We postulated that dysfunctional venous outflow can hamper this venous return, leading to a drop in blood pressure compounded by the effects of gravity, resulting in orthostatic hypotension. This is consistent with findings that pooling in chronic orthostatic intolerance involves arterial vasoconstriction but not venous compliance defects [28]. In addition, poor clearance of metabolites may hamper autonomic structures in the head and neck and contribute to OH. Emerging evidence also points to venous outflow obstructions-such as iliac vein compression [29] or jugular stenosis-as contributors to refractory OH, where impaired venous return diminishes cardiac preload and worsens orthostatic intolerance, particularly in elderly or post-surgical patients. These insights underscore the limitations of current therapies and the potential for targeted vascular interventions to address underlying hemodynamic deficits. We posit that increasing venous outflow through angioplasty or stenting of the large veins will improve systemic blood pressure by enhancing venous return while simultaneously reducing venous congestion in the cervical baroreceptors and autonomic pathways. Endovascular intervention may reduce the reliance on medications with serious side effects and ultimately improve the quality of life for patients with OH. There are no currently available treatments for medically intractable orthostatic hypotension, and these patients have a disabling and often life-threatening situation, justifying endovascular intervention as a means to reduce disability and mortality risk.

2. Methods

Six patients (all male) aged 63 to 87 underwent venograms and were found to have jugular, brachiocephalic, or subclavian vein stenosis and were treated with balloon angioplasty and/or stenting. Each patient was clinically diagnosed with orthostatic hypotension

following standard blood pressure recordings (see Figure 1) and was referred for severely disabling, intractable orthostatic hypotension and orthostatic intolerance that was refractory to medical management. The patients had tried and failed medications or were limited by the side effects of the medications. Post-procedure blood pressures were similarly recorded in the supine, sitting, and standing positions.

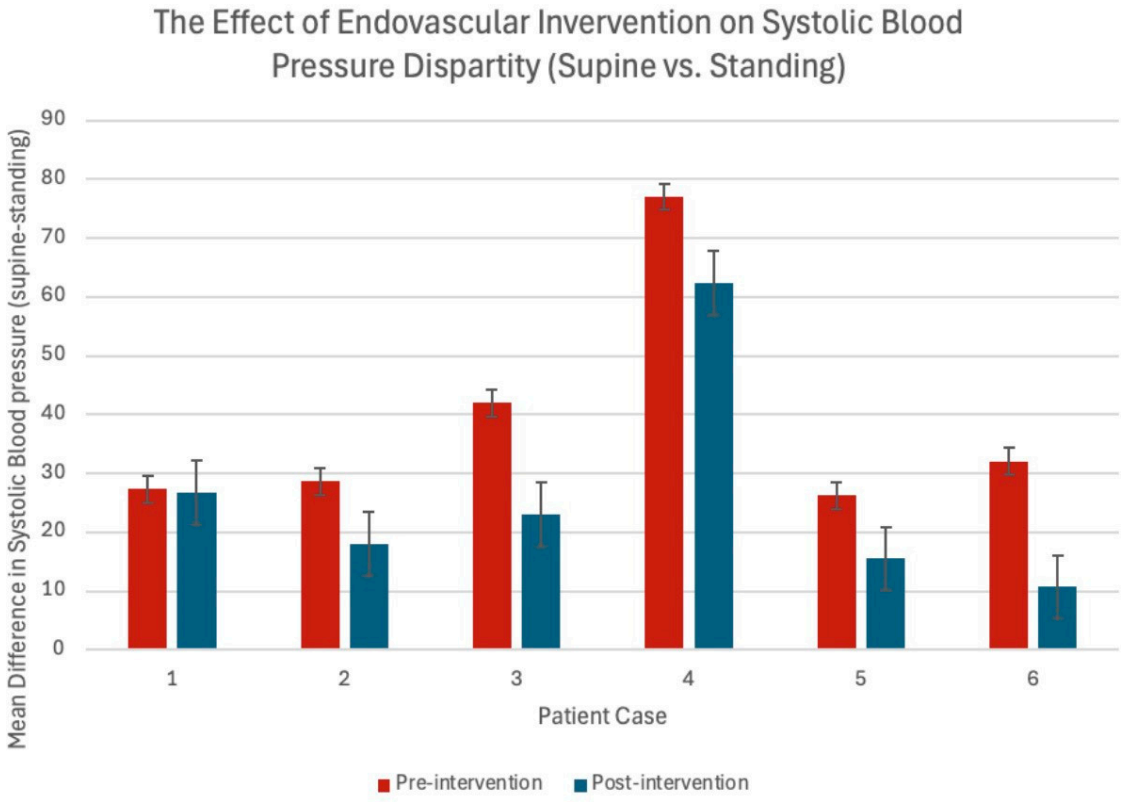

Figure 1. Evaluating the impact of endovascular intervention on the difference between systolic blood pressure in the systolic supine vs. standing positions for all six patients. Statistical analysis (Welch's *t*-test) determined that the disparity between the supine and standing systolic blood pressures significantly decreased following endovascular intervention by 14.07 mmHg (n = 117, $p = 0.024$). Error bars represent standard error of the mean.

Two Example Patient Cases

Patient 1 is an 86-year-old male who presented with severe lightheadedness on standing that forced him to start crawling at home to avoid falls, postprandial hypotension with recurrent syncopal episodes, and imbalance. The patient's medical history was significant for chronic progressively worsening orthostatic hypotension, hyperlipidemia, macular degeneration, and hypertension. The patient was initially on midodrine, and it was ineffective at low doses, and at higher doses, the patient developed severe headaches and severe supine hypertension. He was switched to Florinef but could not tolerate it due to shortness of breath. He was switched to droxidopa, but this was ineffective, and the patient was restarted on Midodrine. Given his severe persistent lightheadedness, he self-increased his midodrine up to doses of 25 to 30 mg a day but developed severe headaches with severe supine hypertension. An angiogram and a venogram at that time revealed bilateral jugular vein stenosis, bilateral subclavian vein stenosis, and left brachiocephalic vein stenosis, which were subsequently angioplastied. The patient responded well for a few months, after which his symptoms began to recur, and he then underwent stenting of the severe recurrent stenosis in the left internal jugular vein and the left brachiocephalic vein. Following stenting, he noticed a remarkable improvement in his blood

pressure and no longer complained about dizziness or headaches, and did not need to crawl at home to avoid falling. He now takes 2.5 mg of midodrine at the start of the day only if required.

Patient 4 is an 80-year old male who developed severe, intractable orthostatic hypotension as an inpatient after treatment for severe anasarca from diastolic heart failure with a newly reduced ejection fraction to 25%. His medical history was significant for atrial fibrillation status post-watchman, hypertension, hyperlipidemia, diabetes, and orthostatic hypotension. Midodrine and fludrocortisone were discontinued, given his reduced ejection fraction. He was continued on his outpatient pyridostigmine. He underwent stenting of the left main and proximal circumflex artery and was planned for stenting of the dominant right coronary artery. On Day 9, the patient was noted to have orthostatic hypotension. Midodrine was restarted. His symptoms initially improved, but on Day 14, he had trouble standing for more than a minute and felt lightheaded. He was started on droxidopa 100 mg three times a day and increased to 300 mg three times a day (Day 21) and then to 600 mg three times a day (Day 24) but had no improvement in his symptoms. Midodrine was increased to 20 mg three times a day, and fludrocortisone was added without any improvement. The patient became bedbound and could not even use the bedside commode as he felt lightheaded sitting up. The patient underwent a venogram (Day 35) that showed severe stenosis of bilateral internal jugular veins and bilateral subclavian veins, for which angioplasty was performed. The patient's orthostatic blood pressures improved the next day and he was able to ambulate over a 100 feet without symptoms (Day 37). His right coronary artery was then stented as he was stable, and his blood pressure was improving. His symptoms then slowly began to recur, and he again became bedbound. He then underwent a cerebral angiogram, which ruled out vertebrobasilar stenosis, and a venogram (Day 46) and was noted to again have the severe stenosis of bilateral internal jugular veins and bilateral subclavian veins. The internal jugular veins were stented to maintain patency, and angioplasty of the subclavian veins was performed. The patient once again showed a remarkable improvement in his orthostatic blood pressure and was asymptomatic, able to ambulate in the corridor (Day 49). He was discharged on Day 52 and has remained asymptomatic on follow-up (Day 73).

3. Results

Baseline patient characteristics: Orthostatic blood pressure (BP) measurements were recorded for six patients both pre- and post-endovascular intervention. The individual pre- and post-operative BP values in the supine, seated, and standing positions are detailed in Table 1. Before the procedure, the average BP for the cohort decreased with each change in posture. The mean supine BP was 145.2/69.5 mmHg, which fell to 125.3/65.0 mmHg in the seated position and further to 106.3/57.7 mmHg when standing (Table 2). The mean pre-operative systolic difference between the supine and standing positions was 38.8 $\pm$ 19.6 mmHg (Table 2).

Effect of intervention on orthostatic BP: Following endovascular intervention, the average systolic BP disparity between the supine and standing positions significantly decreased by 14.07 mmHg (n = 117, p = 0.024) (Table 2, Figure 1). The mean pre-operative systolic difference of 38.8 $\pm$ 19.6 mmHg (95% CI: 18.3–59.4) was reduced to 28.2 $\pm$ 17.0 mmHg (95% CI: 10.3–46.0) post-intervention (Table 2). This demonstrates that the procedure led to a statistically significant improvement in the patients' orthostatic response when moving from a supine to a standing position.

Effects on seated vs. standing BP: To assess the effect of the intervention on a less extreme postural change, the systolic BP disparity between the seated and standing positions was also evaluated (Figure 2). Following the intervention, the mean difference between seated and standing systolic BPs decreased by 2.35 mmHg. However, a Welch's t-test indicated this decrease was not statistically significant ($p > 0.05$) (Figure 2).

Table 1. Average Pre-operational and post-operational orthostatic blood pressures for each patient (mmHg).

	I	II	III	IV	V	VI
Pre-op BP						
Supine	148/63	147/81	177/65	145/66	131/69	123/73
Seated	134/65	126/81	151/64	111/61	122/53	108/66
Standing	121/56	118/69	135/65	68/46	105/56	91/54
Post-op BP						
Supine	114/62	158/81	165/75	141/70	143/75	140/83
Seated	99/54	143/80	144/69	131/55	139/79	120/70
Standing	87/58	140/78	142/79	79/43	127/70	117/64
Systolic Diff (Sup-Stand)						
Pre-op	27	29	42	77	26	32
Post-op	27	18	23	62	16	23

Table 2. Overall Mean Blood Pressure and Systolic Disparities Across Patients.

Measurement	Systolic (Mean ± SD [95% CI])	Diastolic (Mean ± SD [95% CI])	*p*-Value
Pre-op BP			
Supine	145.2 ± 18.5 [125.7–164.6]	69.5 ± 6.6 [62.5–76.5]	
Seated	125.3 ± 15.8 [108.7–141.9]	65.0 ± 9.1 [55.4–74.6]	
Standing	106.3 ± 24.0 [81.1–131.5]	57.7 ± 8.2 [49.0–66.3]	
Post-op BP			
Supine	143.5 ± 17.7 [125.0–162.0]	74.3 ± 7.6 [66.3–82.3]	
Seated	129.3 ± 17.4 [111.1–147.5]	67.8 ± 11.3 [56.0–79.7]	
Standing	115.3 ± 26.8 [87.2–143.4]	65.3 ± 13.6 [51.1–79.6]	
Systolic Diff (Sup-Stand)			
Pre-op	38.8 ± 19.6 [18.3–59.4]		
Post-op	28.2 ± 17.0 [10.3–46.0]		0.024

Individual patient responses: Analysis of individual patient data revealed varied responses (Table 1). While the mean systolic BP difference between supine and standing positions decreased for most patients, the extent of the improvement differed. The systolic difference decreased for all but one patient, with the greatest reductions seen in patients with the largest pre-operative disparities. For example, Patient IV's supine-to-standing systolic difference decreased from 77 mmHg pre-op to 62 mmHg post-op, representing a substantial improvement in orthostatic BP control (Table 1).

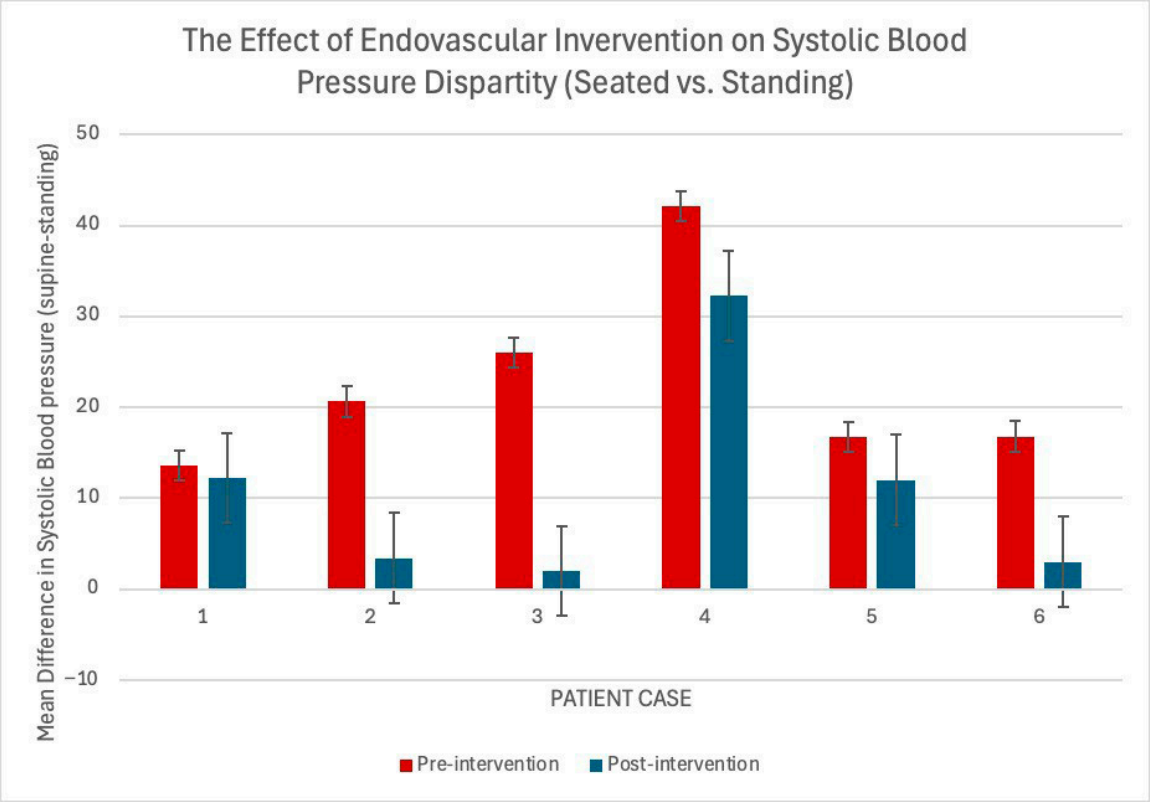

Figure 2. Evaluating the impact of endovascular intervention on the difference between systolic blood pressure in the systolic seated vs. standing positions for all six patients. Statistical analysis (Welch's *t*-test) determined that the disparity between the seated vs. standing systolic blood pressures decreased following endovascular intervention by 2.35 mmHg, but the results were insignificant (n = 116, $p > 0.05$). Error bars represent standard error of the mean.

4. Conclusions and Future Research

All patients noticed an improvement in their orthostatic intolerance symptoms and post-procedural orthostatic blood pressure. The difference between supine and standing systolic blood pressures for all six patients was averaged (38.68 mmHg for preoperational; 24.61 mmHg for post-operational), producing a 36.38% decrease in blood pressure disparity. The difference was insignificant when comparing the seated vs. standing differences, possibly due to the increasing effects of gravity. We used SPSS (Version 30) Statistics Independent two-tailed *t*-test with equal variances not assumed to evaluate the disparity difference between the mean preoperational and post-operational systolic blood pressures, which proved to be statistically significant for the standing vs. supine data in accordance with a 95% confidence ($t = 2.43(115)$; $p = 0.024$). These results indicate that the patient's orthostatic hypotension lessened in severity when attempting to stand from a supine position. This demonstrates that endovascular intervention, by improving venous outflow, can offer a therapeutically effective adjunct and, in some cases, even a substitute for pharmacotherapy. This is particularly important given the dependence, potency, and serious side effects of the currently available medications that may, in addition, be ineffective. The improvement in standing blood pressure supports the idea that orthostatic hypotension may be heavily influenced by venous outflow obstruction of the large supra-cardiac veins (internal jugular, subclavian veins, and the left brachiocephalic vein). We acknowledge the significant limitations of the study. All patients in this retrospective review were older males (aged 63–87), reflecting referral patterns for severe, refractory OH in our practice. The absence of female patients limits generalizability, as OH prevalence and venous stenosis may differ by sex due

to hormonal or anatomical factors. Future studies should include diverse demographics to assess broader applicability. The small sample size (n = 6) was justified by the exploratory nature of this novel intervention in a rare, refractory OH cohort. This limits statistical power and generalizability; larger, prospective studies are needed. Tilt table testing was not conducted due to the established OH diagnosis via standard clinical protocol; future studies should consider this to evaluate cardiodepressive contributions. The lack of a control group (e.g., patients managed medically without intervention) is a key limitation, potentially introducing bias. Future research should incorporate randomized controls to validate these findings. The intervention appears durable in this small cohort without any recurrence or need for medication escalation.

Future research should continue to investigate the long-term outcomes of venous angioplasty and stenting of the large supracardiac veins. Additionally, further investigation into the underlying mechanisms that may account for the observed improvements in patients is warranted. Several patients also reported notable improvements in symptoms beyond orthostatic hypotension, including chronic headaches, tinnitus, imbalance, brain fog, and vertigo. These observations support the hypothesis that impaired venous return may contribute to various neurological problems in this patient population. The term "Venous Outflow Obstruction Disorders" is proposed as a potential classificatory framework to encompass a spectrum of disorders stemming from venous stenosis that may affect neurological function [30]. This aligns with previous reports that have described similar clusters of symptoms in patients with internal jugular vein outflow obstruction, including ophthalmological disorders, cognitive and behavioral issues, headaches, and dizziness [31]. Our findings suggest that neurological dysfunction may arise from venous outflow obstruction occurring anywhere from the nervous system's venous drainage up to and including the heart. This raises the possibility that a broad range of neurological and neurovascular disorders could be linked to this type of obstruction, an area that merits further exploration given its potential clinical significance.

Author Contributions: K.M.A. conceptualized the study. J.T., A.M., C.K. and K.M.A. performed material preparation, data collection, and analysis. J.T. and K.M.A. wrote the first draft of the manuscript. V.S.D., K.M.A., C.K. and N.G. authored the patient cases. J.T., A.M. and K.M.A. created tables and figures, wrote the conclusions, and outlined future research directions. All authors critically reviewed and edited previous versions of the manuscript. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: Institutional Review Board approval was obtained from the St. Francis Institutional Review Board (IRB) for this retrospective review.

Informed Consent Statement: Informed consent was obtained from all individual participants included in the study. Potential risks of venous stenting including hematoma, infection, stent migration/fracture, thrombosis, and bleeding (rates < 15% per literature) were discussed in detail when consent was obtained for the procedure. None of the members in this cohort experienced any of the above complications. The participants have consented to the submission of the case report to the journal.

Data Availability Statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding authors.

Conflicts of Interest: There are no conflicts of interest related to this paper.

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