BJCR
Brazilian Journal of Case Reports

PCAA
Publicações Científicas de Acesso Aberto

Case Report

Wide Pulse Pressure in an Elderly Patient with Diabetes: A Case Report and Literature Review

Ana Paula Mendes Monteiro [1], Yasmin Luíse Lucena e Silva [2], Mariana Nascimento Albuquerque Cordeiro [1], Eduardo George Medeiros Araújo das Chagas [3], Rafael Andrade Rego [1,*]

[1] Federal University of Rio Grande do Norte (UFRN), Natal, Rio Grande do Norte, Brazil.
[2] Federal Rural University of the Semi-Arid Region (UFERSA), Mossoró, Rio Grande do Norte, Brazil.
[3] Potiguar University, Natal, Rio Grande do Norte, Brazil.

* Correspondence: rafaelandraderego@gmail.com.

Abstract: Widened pulse pressure (PP) is a marker of reduced large-artery compliance, most often attributed to age- and diabetes-related arteriosclerosis, but its differential includes aortic regurgitation, hyperthyroidism, arteriovenous fistula, and severe anemia. Antihypertensive drug classes may differentially affect PP, with renin-angiotensin-aldosterone system (RAAS) inhibitors and calcium channel blockers showing greater benefit on its reduction. A 67-year-old woman with hypertension and insulin-dependent diabetes presented with polyuria, dizziness, wide PP (70 mmHg), tachycardia, hyperglycemia, and peripheral edema after prolonged discontinuation of losartan and insulin due to public pharmacy shortages. Aortic regurgitation was excluded by absence of a diastolic murmur and by echocardiography, which showed aortic arch atheromatosis and mild valve calcification without regurgitation. Substitution of carvedilol with olmesartan, alongside correction of hyperglycemia-driven dehydration, was associated with progressive narrowing of PP. This case highlights the clinical rationale for favoring RAAS inhibition over beta-blockade, while emphasizing that medication affordability and supply continuity are as clinically fundamental as pharmacologic choice in resource-limited settings.

Keywords: Wide Pulse Pressure; Aortic Stiffening; Arteriosclerosis; Diabetes Mellitus; Medication Access; Renin-Angiotensin-Aldosterone System Inhibitors; Beta-Blockers.

Citation: Monteiro APM, Lucena e Silva YL, Cordeiro MNA, Chagas EGMA, Rêgo R. Wide Pulse Pressure in an Elderly Patient with Diabetes: A Case Report and Literature Review. Brazilian Journal of Case Reports. 2026 Jan-Dec; 06(1):bjcr230.

https://doi.org/10.52600/2163-583X.bjcr.2026.6.1.bjcr230

Received: 17 August 2026
Accepted: 29 September 2026
Published: 5 October 2026

Copyright: This work is licensed under a Creative Commons Attribution 4.0 International License (CC BY 4.0).

1. Introduction

Wide pulse pressure (PP) is defined as a difference between systolic and diastolic blood pressure exceeding 60 mmHg; it is a recognized marker of large-artery stiffening and an established independent predictor of cardiovascular morbidity and mortality [1]. With aging, systolic and diastolic pressures rise in parallel until approximately the sixth decade of life, after which diastolic pressure begins to decline and systolic tension tends to rise, resulting in isolated systolic hypertension, which accounts for roughly 65% of hypertension diagnoses in elderly populations [2]. Beyond age-related degeneration, however, PP is dynamic and can be acutely modulated by hyperdynamic states (anemia, hyperthyroidism), aortic regurgitation, and pharmacologic therapy [3].

Such interplay is clinically important because when systolic pressure increases alongside excessive diastolic lowering, myocardial perfusion may be compromised, particularly in older patients with high cardiovascular risk [4]. This case illustrates how intermittent access to losartan and insulin, alongside empiric substitution of antihypertensive agents with differing hemodynamic profiles, produced a significant change in diastolic pressure, offering a clinical window into the interplay between arterial stiffness, volume status, and antihypertensive drug class selection.

2. Case Report

A 67-year-old woman with hypertension and insulin-dependent diabetes sought primary care after discontinuing losartan and long-acting insulin for over a week because of shortages at public pharmacies, with subsequent inability to resume therapy for several months. Due to service overcrowding, she was exceptionally referred to our community hospital by the emergency department after prompt evaluation and laboratory testing excluded acute kidney injury (Cr 0.9; BUN 20; Na 136; K 4.1). On admission, she reported polyuria and dizziness. Examination showed blood pressure (BP) 160/90 mmHg, pulse pressure (PP) 70 mmHg, heart rate 110/min, capillary blood glucose (CBG) 263 mg/dL, and 3+ lower extremity edema; cardiac auscultation was unremarkable. To mitigate white coat syndrome (per internal protocol), we measured BP once at the reception desk and once in each arm in the consultation room at the first visit, with an approximately 30-minute wait in line between the two settings. At subsequent visits, we repeated measurements at the reception desk and in the consultation room (one arm only), following the same waiting time.

In initial therapy at visit 1, as shown in Table 1, the patient was prescribed carvedilol 6.125 mg twice daily, amlodipine 5 mg at bedtime, and intermediate-acting insulin 15-0-15 units, with follow-up in two weeks to titrate the beta-blocker as necessary. Notably, she was able to obtain and take carvedilol and amlodipine, as both were available free of charge through public pharmacies. By contrast, losartan remained out of stock, and she declined to purchase it out of pocket at a private institution, citing economic fragility.

Table 1. Summarizes hemodynamic and glycemic trends across visits.

Visit	BP (mmHg)	Pulse pressure (mmHg)	Heart rate	CBG (mg/dL)	Edema	Medications actually taken
Visit 1 (baseline/ 1° Week)	Right Arm 160/90 Left Arm 155/90	70	110	263	3+	None (non adherent)
Visit 2 (3º week)	Desk 150/60 Room 140/60	80–90	83	239	2+	Carvedilol; amlodipine self-discontinued; insulin not started
Visit 3 (4° week–1 week after switch)	Desk 140/90 Room 140/90	50	—	254	2+	Olmesartan 40 mg (replacing carvedilol)
Visit 4 (at 6 months)	Desk 140/90 Room 140/90	50	—	162	None	Losartan and glargine resumed. Lab results received

At visit 2, the patient reported that she had been able to get carvedilol and amlodipine promptly, but self-discontinued amlodipine because of worsening dizziness. Meanwhile, insulin was not started because she was waiting for a new dispatch of the drug at public pharmacies. At this appointment, the patient's blood pressure was 145/60 mmHg. She resisted more aggressive blood pressure control, fearing that combining antihypertensive classes would drop her diastolic tension below a perceived necessary target of roughly 80 mmHg. Given this concern, carvedilol was switched for free hospital samples of olmesartan 40 mg daily. A week later, her pulse pressure had narrowed from the prior 80–90 mmHg range down to 50 mmHg (140/90mmHg).

A requested transthoracic echocardiography (Figure 1) confirmed normal ventricular and septal dimensions, aortic arch atheromatosis, and mild aortic valve calcification

without significant regurgitation. All relevant cardiac measurements are detailed in Supplementary Text 1. At the 6-month follow-up, resumption of losartan and glargine alongside lifestyle counseling led to significant improvement in blood pressure and glycemic control. Laboratory results at this last visit revealed mildly elevated creatinine (1.2 mg/dL), and partially controlled diabetes (HbA1c 7.3%), as summarized in table 2.

Figure 1. Echocardiography with Doppler.

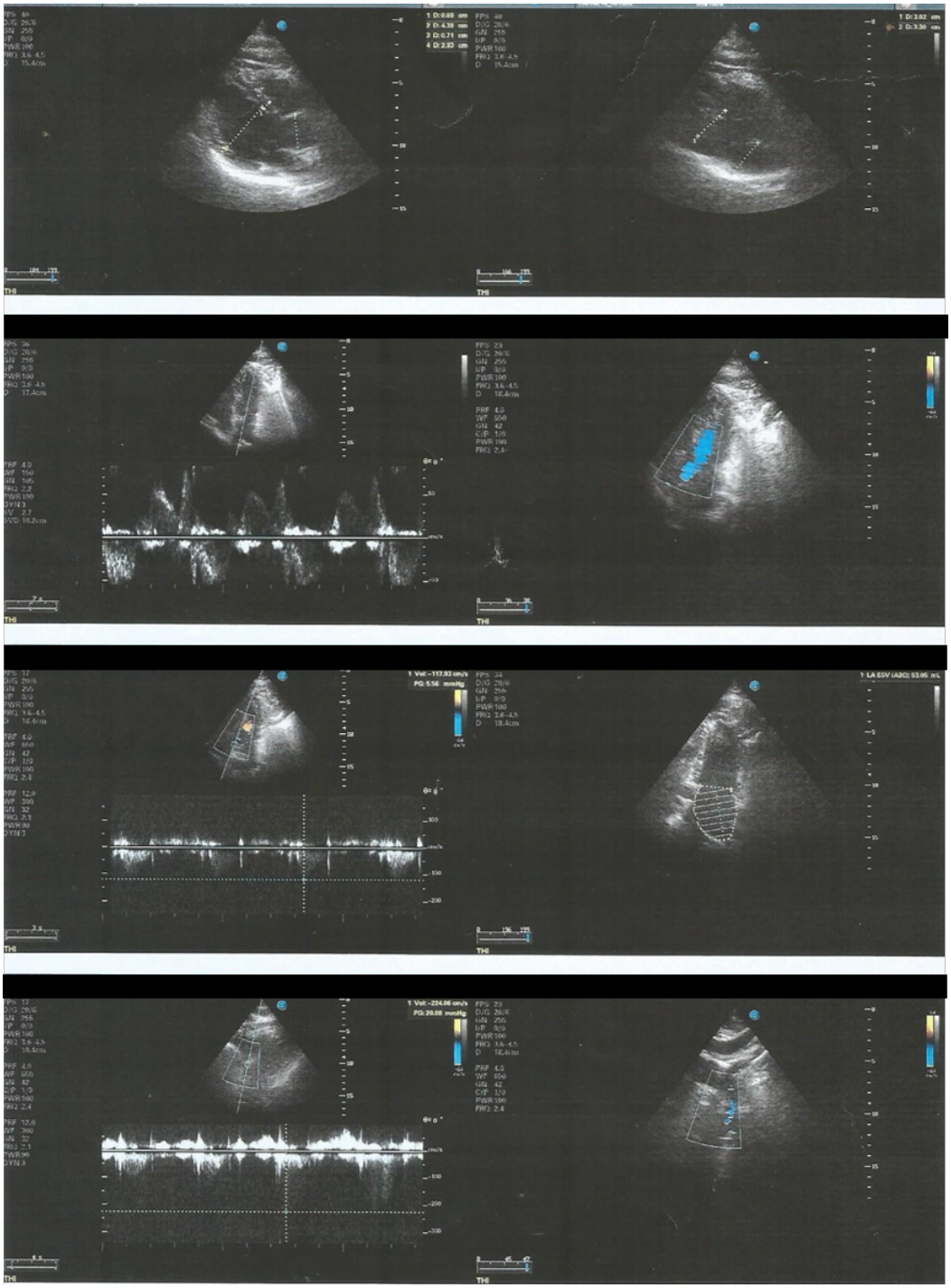


Table 2. Laboratory data on last visit to the community hospital.

Hematological test	Result	Reference Range (Females)
RBC	4.23 x10^6/μL	3.8 - 5.1 x10^6/μL
Hb	12.9 g/dL	11.5 - 14.9 g/dL
Ht	38.4 %	35 - 45 %
WBC	5060 /μL	2883 - 9969 /μL
Plt	242 x10^3/μL	135.6 x 10^3 - 343 x10^3/μL
Cr	1.1 mg/dL	0.66 - 1.09 mg/dL
HbA1c	7.3 %	<6.5 %
Biochemistry		
Na	137 mEq/L	136 - 145 mEq/L
K	3.7 mEq/L	3.5 - 5.1 mEq/L
TSH	1.590 μUI/mL	0.44 - 6.8 mUI/L
FT4	1.27 ng/dL	0.7 - 2.2 ng/dL
Urinalysis		
Protein	(-)	-
Occult blood	(-)	-
Glucose	(+)	-

3. Discussion

3.1. Rarity of events

Wide PP from arteriosclerotic aortic stiffening is a common finding present in about 30% of patients with isolated systolic hypertension [5], but diastolic hypotension alongside abrupt fluctuation (tracked serially across visits and temporally linked to drug shortages) provides an unusual clinical demonstration of the interplay between adherence, volume status, and arterial hemodynamics.

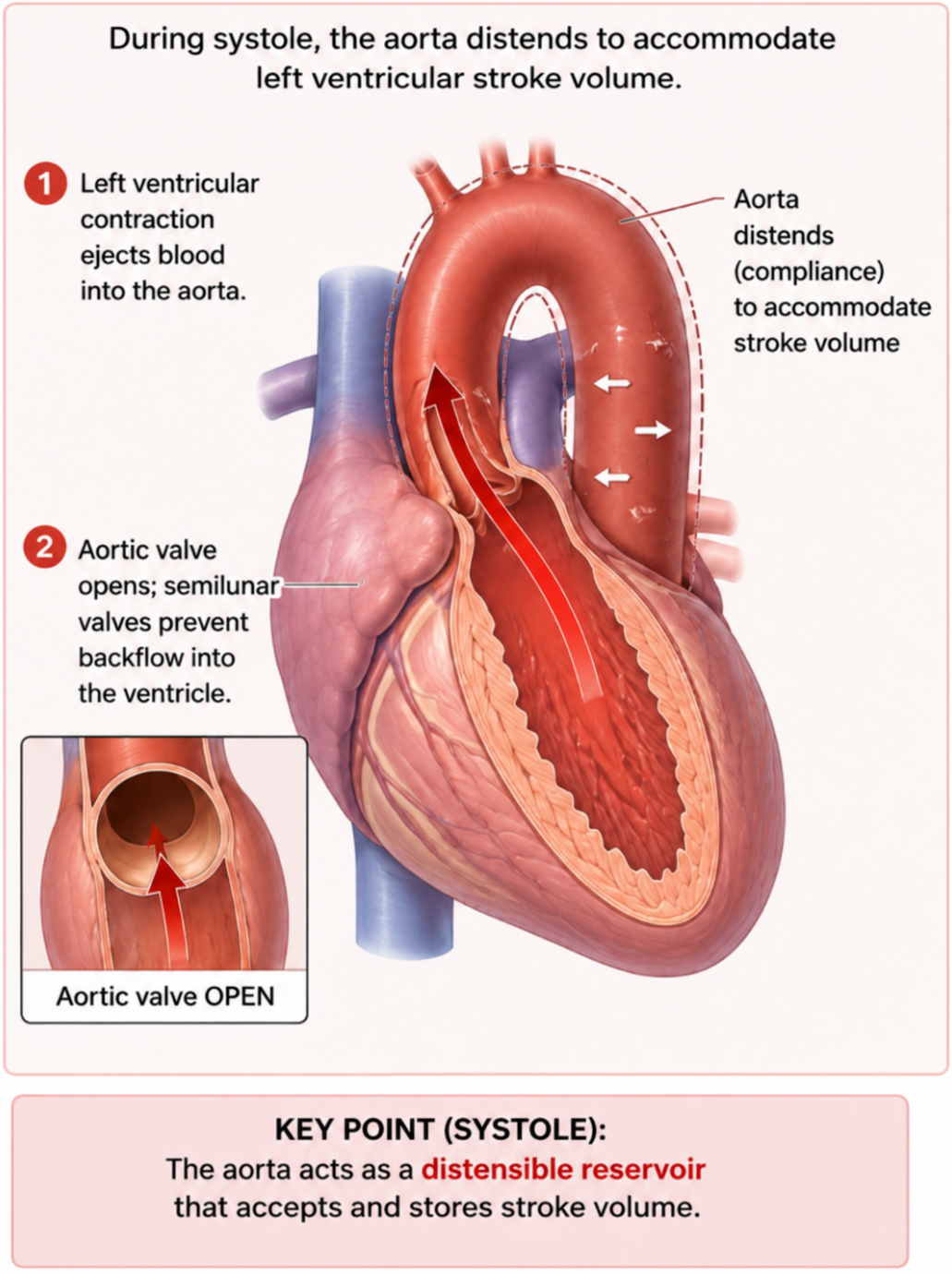
3.2. Pathophysiology

During systole, the aorta expands to accommodate the left ventricular stroke volume (Figure 2); during diastole, elastic recoil propels this stored volume forward into the systemic circulation and backward into the coronary arteries (Figure 3). Aortic stiffening, whether from age-related collagen deposition or from calcification, as seen on this patient's echocardiogram, reduces vessel dispensability. This forces systolic pressure upward to overcome afterload, while diastolic blood pressure (DBP) declines because rigid vessels recoil poorly and can no longer sustain diastolic flow. Since coronary filling occurs predominantly during this recoil phase, aggressively lowering an already reduced DBP risks compromising myocardial perfusion. In this context, a 2021 JAMA Network Open analysis restricted to patients with well-controlled systolic readings (<130 mmHg) found the lowest risk of mortality and nonfatal myocardial infarction among those maintaining a range of 70–80 mmHg DBP, whereas the hazard for these outcomes rose by up to 2.5-fold once values dropped below roughly 60–65 mmHg [6].

Another relevant cause of DBP alteration is aortic regurgitation. Because this condition causes backflow to the left ventricle during relaxation, less effective diastolic pressure is available for systemic circulation, which widens PP. In this patient, however, we reasonably deprioritized aortic regurgitation based on the absence of a diastolic murmur and the echocardiogram showing no significant regurgitant lesion. At last, thyroid dysfunction and hemoglobin status can independently modulate hemodynamics and should be considered in the differential of a widened or fluctuating PP. Either hyperthyroidism

or anemia produces a hyperdynamic circulatory state (increased heart rate, stroke volume, blood volume, and myocardial contractility combined with reduced systemic vascular resistance) that raises systolic pressure while lowering diastolic pressure, thus altering PP [7]. For this analysis, we did not have thyroid function tests at admission, but since TSH/fT4 and hemoglobin were within the expected range after 6 months follow-up (Table 2), we ruled out hypothyroidism and severe anemia as determining factors in this case.

Figure 2. Systolic dynamics in cardiac cycle.

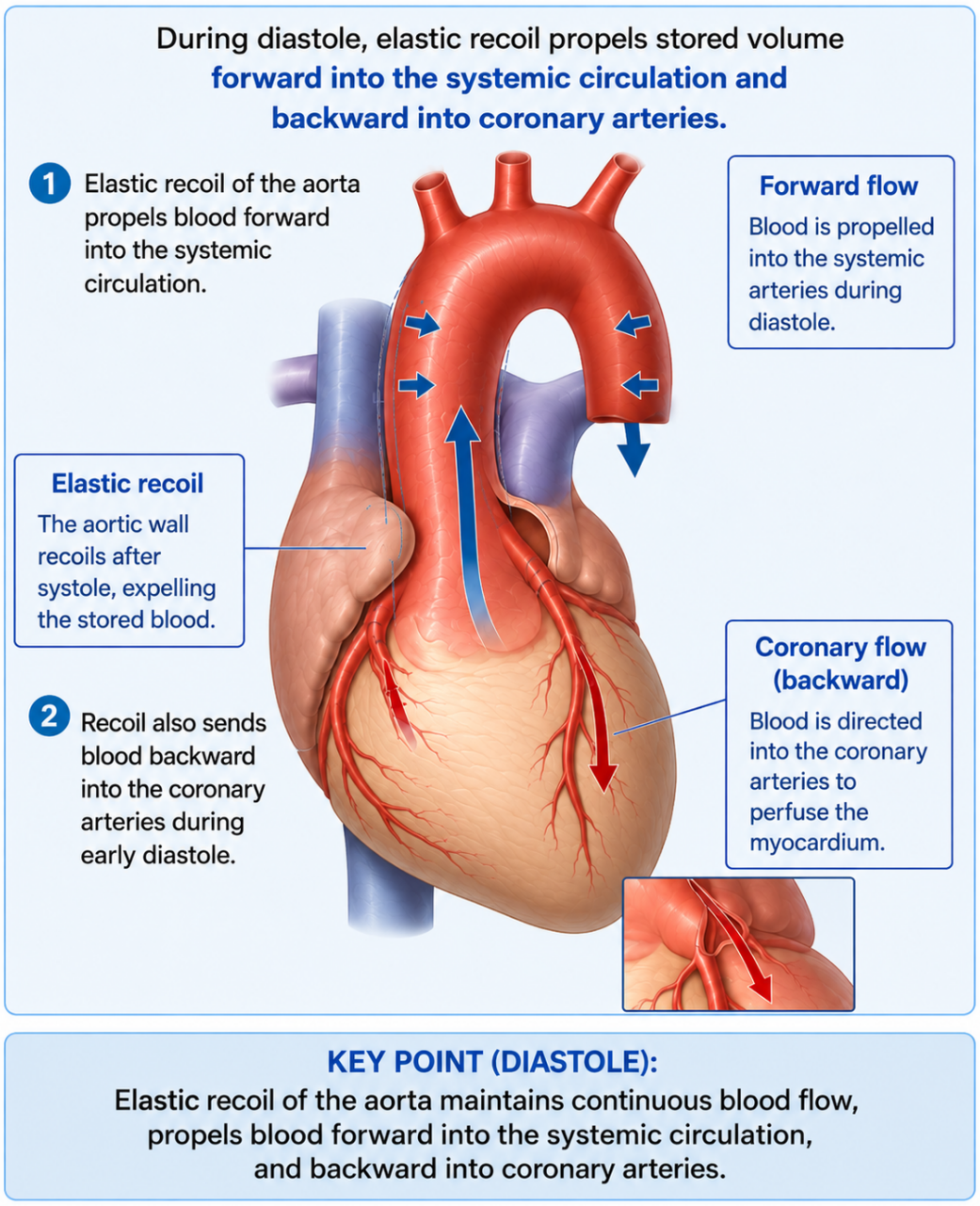


3.3 Cuff pressure and Central Pressure

As the pressure wave generated by left ventricular ejection travels distally through the arterial tree, its waveform morphology changes. Systolic pressure and pulse pressure

rise as the wave moves distally through narrower peripheral arteries (up to 10–15 mmHg aorto-brachial increase in young individuals), while diastolic tension tends to keep nearly constant (falling about 1 mmHg) along the same course [8]. Such an effect makes brachial PP higher than central (aortic) PP when measured, so that cuff BP is not an accurate stand-in for central hemodynamic load; a given brachial reading may overestimate PP, and for this reason this case and discussion are estimates for hypothesis generation rather than a precise measure of central aortic hemodynamics.

Figure 3. Diastolic dynamics in cardiac cycle.



3.4. Contribution of dehydration and diabetes

Polyuria with volume depletion from poorly controlled diabetes can reduce effective circulating volume and, therefore, impair aortic filling. By this mechanism, dehydration may further widen PP in patients with baseline arteriosclerosis and aortic calcification, which coexisted as comorbidities in this patient [9]. We believe that, in this case,

Olmesartan may have reduced polyuria by lowering glomerular filtration rate, indirectly improving hydration and consequently hemodynamics, though this is limited as hypothesis because we were not able to measure urine volume logs. The observed creatinine rise from 0,9 to 1,1 mg/dL was somewhat expected since ACEi class can raise baseline values up to 30%.

3.5. Preferable Antihypertensive Choice

The comparative evidence on drug class and arterial stiffness/PP is mixed and should be presented with appropriate caution rather than as a settled mechanism. It is reasonable to consider that drugs with a vasodilatory effect somewhat improve aortic distensibility, and that combining classes may produce a synergistic effect. In the SELECT study [10], a clinical trial of 2005 comprising 443 outpatients with severe systolic hypertension, amlodipine/benazepril combination reduced 24-hour ambulatory PP by 10.5 mmHg, significantly more than amlodipine (−6.7 mmHg) or ACE inhibitor alone (−5.0 mmHg; $p<0.0001$). Additionally, the CAFE analysis (ASCOT substudy from 2023) provided head-to-head central hemodynamic data of 2,199 patients using amlodipine ± perindopril produced substantially lower central aortic pressures, and central PP independently predicted cardiovascular and renal outcomes in the cohort [11].

By contrast, beta-blockers appear to underperform for PP reduction. A meta-analysis of 20 RCTs confirmed RAAS inhibitors and CCBs reduce central PP and augmentation index more than beta-blockers or diuretics, even when peripheral PP reductions are similar [12]. Meanwhile, A Cochrane review found that beta-1 selective blockers have essentially no net effect on wide PP, compared with average reductions of −5 mmHg with thiazides and −3 mmHg with RAAS inhibitors (ACE inhibitors/ARBs). The authors considered that beta-blockers lowered diastolic pressure to nearly the same degree as systolic tension, thereby leaving PP largely unchanged [13]. This pattern is consistent with the approach taken in this case, in which carvedilol was discontinued in favor of an ARB.

Regarding Nitrates, though NO-mediated vasodilation is proposed to improve diastolic function due to vessel stiffening, available trial data are limited and do not support a clinically meaningful benefit. In a 2022 phase III randomized trial of elderly patients with isolated systolic hypertension, isosorbide mononitrate produced no significant reduction in PP compared with placebo (5.28 vs 7.49 mmHg, $p = 0.79$) [14]. It is possible, however, that subgroups with decreased diastolic tension at baseline could benefit differently than in this population. Based on limited evidence, nitrate- and PDE5-based therapy remains an area of emerging interest rather than an established therapeutic option.

4. Patient Perspective

When I couldn't get my medication at the pharmacy, I felt scared and lost. After starting new drugs for hypertension, I became very concerned because the smaller number in my blood pressure reading was dropping too low (diastolic tension - 150/60mmHg). I am grateful that my physician listened to my fears and made treatment in a way that respected my fears and financial reality.

5. Conclusion

This case highlights wide PP as a challenging complication of uncontrolled diabetes in a senior, which was decompensated here by intermittent medication access. Aortic regurgitation or other high-output causes were reasonably excluded by examination, lab results, and echocardiography. Available evidence suggests that RAAS inhibitors and calcium channel blockers may reduce wide pulse pressure more effectively than beta-blockers, either in central measures or peripheral cuff, though it's important to keep in mind that this case and discussion are estimates for hypothesis generation rather than a

precise measure of central aortic hemodynamics. Ultimately, hemodynamic and glycemic control were restored only after access to losartan and insulin was reestablished; this points out that medication affordability and continuity of treatment are as paramount as pharmacologic class selection in resource-limited areas.

Funding: None.

Research Ethics Committee Approval: Written informed consent was obtained from the patient's legal representative, and the study was conducted in accordance with the ethical principles established in the Declaration of Helsinki.

Acknowledgments: The Authors thank MD Larissa Lucena for advice on research publishing. Special thanks to UFRN (Universidade Federal do Rio Grande do Norte) for advice about consent for publication.

Conflicts of Interest: All other authors declare no conflicts of interest.

References

1. Boutouyrie P, Chowienczyk P, Humphrey JD, Mitchell GF. Arterial stiffness and cardiovascular risk in hypertension. Circ Res. 2021;128(7):864-886. doi:10.1161/CIRCRESAHA.121.318061.
2. Tang KS, Medeiros E, Shah AD. Wide pulse pressure: A clinical review. J Clin Hypertens (Greenwich). 2020;22(11):1960-1967. doi:10.1111/jch.14051.
3. Chirinos JA, Segers P, Hughes T, Townsend R. Large-artery stiffness in health and disease. J Am Coll Cardiol. 2019;74(9):1237-1263. doi:10.1016/j.jacc.2019.07.012.
4. Khan NA, Rabkin SW, Zhao Y, McAlister FA, Park JE, Guan M, et al. Effect of lowering diastolic pressure in patients with and without cardiovascular disease. Hypertension. 2018;71(5):840-847. doi:10.1161/HYPERTENSIONAHA.117.10177.
5. Madan N, Lee AK, Matsushita K, Hoogeveen RC, Ballantyne CM, Selvin E, et al. Relation of isolated systolic hypertension and pulse pressure to high-sensitivity cardiac troponin-T and N-terminal pro-B-type natriuretic peptide in older adults (from the Atherosclerosis Risk in Communities Study). Am J Cardiol. 2019;124(2):245-252. doi:10.1016/j.amjcard.2019.04.030.
6. Li J, Somers VK, Gao X, Chen Z, Ju J, Lin Q, et al. Evaluation of optimal diastolic blood pressure range among adults with treated systolic blood pressure less than 130 mm Hg. JAMA Netw Open. 2021;4(2):e2037554. doi:10.1001/jamanetworkopen.2020.37554.
7. Klein I, Ojamaa K. Thyroid hormone and the cardiovascular system. N Engl J Med. 2001;344(7):501-509. doi:10.1056/NEJM200102153440707.
8. Putowski Z, Teboul JL, Castro R, Chemla D, Oras J, Morales S, et al. Can we reliably use pulse pressure as a surrogate for stroke volume? Physiological background and potential clinical implications for shock resuscitation. Crit Care. 2025;29(1):249. doi:10.1186/s13054-025-05490-9.
9. Tang KS, Medeiros E, Shah AD. Wide pulse pressure: A clinical review. J Clin Hypertens (Greenwich). 2020;22(11):1960-1967. doi:10.1111/jch.14051.
10. Neutel JM, Smith DH, Weber MA, Schofield L, Purkayastha D, Gatlin M. Efficacy of combination therapy for systolic blood pressure in patients with severe systolic hypertension: The Systolic Evaluation of Lotrel Efficacy and Comparative Therapies (SELECT) study. J Clin Hypertens (Greenwich). 2005;7(11):641-646. doi:10.1111/j.1524-6175.2005.04615.x.
11. Schettini IVG, Rios DRA, Figueiredo RC. Effect of different classes of antihypertensive drugs on arterial stiffness. Curr Hypertens Rep. 2023;25(5):61-70. doi:10.1007/s11906-023-01238-4.
12. Cheng Y, Xia J, Li Y, Wang J. Antihypertensive treatment and central arterial hemodynamics: a meta-analysis of randomized controlled trials. Front Physiol. 2021;12:762586. doi:10.3389/fphys.2021.762586.
13. Wong GW, Boyda HN, Wright JM. Blood pressure lowering efficacy of beta-1 selective beta blockers for primary hypertension. Cochrane Database Syst Rev. 2016;2016(4):CD007451. doi:10.1002/14651858.CD007451.pub2.
14. Pérez DA, Polo IG, Salvanés FJR, Roncal SB, Sanz PI, Fernández CS. Sustained-release isosorbide mononitrate as adjuvant treatment in isolated systolic hypertension in the elderly. J Hum Hypertens. 2022;36(2):163-170. doi:10.1038/s41371-021-00498-4.