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THERAPEUTIC POTENTIAL OF AZELNIDIPINE IN CARDIOVASCULAR RISK REDUCTION: A COMPREHENSIVE REVIEW

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ABSTRACT

Azelnidipine is a third-generation dihydropyridine calcium channel blocker (CCB) characterized by high vascular selectivity, long duration of action, and unique dual L-type and T-type calcium channel blockade. It provides effective and sustained antihypertensive action without inducing reflex tachycardia, a common limitation of earlier CCBs. Owing to its high lipophilicity and prolonged membrane binding, Azelnidipine maintains 24-hour blood pressure control and demonstrates cardioprotective, renoprotective, and anti-oxidative properties. The drug has also shown favorable effects on left ventricular hypertrophy, endothelial function, and renal microcirculation. This review summarizes the pharmacological characteristics, pharmacokinetics, and pharmacodynamics, mechanism of action, clinical efficacy, safety profile, and therapeutic role of Azelnidipine in the treatment of hypertension.

KEYWORDS: Azelnidipine, Calcium channel blocker, Dihydropyridine, Hypertension, L-type channel, T-type channel, Cardioprotection.

1. INTRODUCTION

Hypertension is a chronic condition characterized by a persistent elevation in arterial blood pressure and remains a leading modifiable risk factor for cardiovascular morbidity and mortality worldwide. According to the European Society of Hypertension (ESH) and European Society of Cardiology (ESC) guidelines, hypertension is defined as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg.[1] In India, the recommended target blood pressure for adults below 60 years is <130/80 mmHg, and for older adults or patients with comorbidities such as diabetes mellitus, coronary artery disease, or chronic kidney disease, the target is <140/90 mmHg.[2]

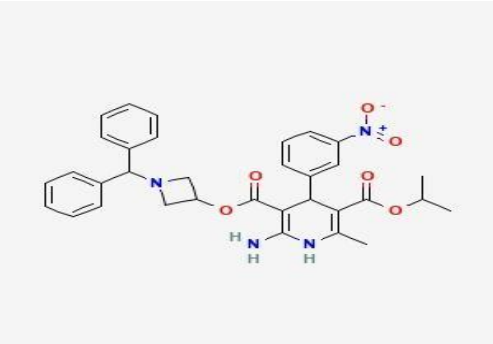
Among antihypertensive agents, calcium channel blockers (CCBs) are widely recommended as first-line therapy due to their efficacy, safety, and metabolic neutrality.[3] CCBs are classified into two main subclasses:

1. Non-dihydropyridines (e.g., Verapamil, Diltiazem) – primarily act on cardiac conduction and contractility.
2. Dihydropyridines (e.g., Amlodipine, Azelnidipine) – mainly act on vascular smooth muscle, causing vasodilation and reduction in peripheral resistance.[4]

Azelnidipine, developed by *Sankyo Co. Ltd., Japan* in 2003 and marketed under the brand name *Calblock*, represents a novel third-generation dihydropyridine CCB.[5] It was approved by the Drug Controller General of India (DCGI) in 2020 and is marketed as *Azusa* by *Ajanta Pharma Ltd.*[6] Unlike earlier DHP-CCBs, Azelnidipine reduces blood pressure gradually and maintains it for a prolonged period without causing reflex sympathetic activation or tachycardia.[7] These unique pharmacological properties make it a promising choice in the long-term management of hypertension.

2. CHEMICAL AND PHYSICOCHEMICAL CHARACTERISTICS

Azelnidipine is a lipophilic dihydropyridine derivative with the chemical formula $C_{33}H_{34}N_4O_6$ and a molecular weight of 582.66 g/mol.[8] Its IUPAC name is *(±)-(3)-(1-diphenylmethyl azetidin-3-yl)-5-isopropyl-2-amino-1,4-dihydro-6-methyl-4-(3-nitrophenyl)-3,5-pyridine dicarboxylate).*[9]



The compound appears as a yellow crystalline powder, practically insoluble in water but soluble in organic solvents like ethanol and methanol.[10] The high lipophilicity contributes to its strong membrane affinity, enabling prolonged interaction with vascular smooth muscle cell membranes and sustained vasodilatory activity.[11]

3. PHARMACOKINETICS

Azelnidipine exhibits favorable pharmacokinetic properties that support once-daily oral administration.

3.1 Absorption

After oral administration, Azelnidipine is rapidly and efficiently absorbed from the gastrointestinal tract with an average bioavailability of 62%. Peak plasma concentrations (Cmax) of 1.66–23.06 ng/mL are typically achieved within 2.6–4 hours (Tmax).[12] Food intake enhances absorption; therefore, it is recommended to be taken after meals.[13]

3.2 Distribution

The drug exhibits extensive tissue distribution with a volume of distribution (Vd) of approximately 1749 ± 964 L.[14] It is 90–91% plasma protein-bound, primarily to albumin and α1-acid glycoprotein.[15] The high lipophilicity allows preferential localization in vascular smooth muscle cells, contributing to its long duration of action.

3.3 Metabolism

Azelnidipine undergoes extensive first-pass hepatic metabolism mediated primarily by the cytochrome P450 (CYP3A4) enzyme system to produce inactive metabolites.[16] Co-administration with CYP3A4 inhibitors (e.g., ketoconazole, erythromycin) or inducers (e.g., rifampicin) may alter its plasma concentration.[17]

3.4 Excretion

Approximately 26% of the administered dose is excreted in urine, while 63% is excreted in feces.[18] The elimination half-life (t½) ranges from 16–28 hours, allowing once-daily dosing and sustained blood pressure control throughout 24 hours.[19]

3. PHARMACODYNAMICS

Azelnidipine belongs to the *dihydropyridine* subclass of calcium channel blockers (CCBs) but is distinct from its predecessors due to its dual inhibition of L-type and T-type calcium channels. The L-type channels are primarily located in vascular smooth muscle and myocardium, where they regulate calcium influx during membrane depolarization. Inhibition of these channels by azelnidipine causes vasodilation of peripheral arterioles, resulting in decreased systemic vascular resistance and, consequently, a reduction in arterial blood pressure. However, what distinguishes azelnidipine from older DHPs such as nifedipine or felodipine is its additional T-type calcium channel blockade. T-type channels are expressed in cardiac pacemaker cells and adrenal zona glomerulosa. Their inhibition reduces sympathetic drive, heart rate, and aldosterone secretion, providing a balanced antihypertensive action with less reflex tachycardia.Azelnidipine also demonstrates slow onset and long duration of vasodilatory action. Its lipophilic aromatic structure allows the molecule to partition into lipid bilayers, from which it is slowly released, maintaining a steady calcium channel blockade for over 24 hours. This pharmacodynamic property ensures smooth blood pressure control throughout the day without abrupt hemodynamic changes. Additionally, azelnidipine exhibits anti-inflammatory and antioxidative properties, protecting vascular endothelium from oxidative stress-induced injury and improving nitric oxide bioavailability. Studies indicate that azelnidipine can reduce plasma malondialdehyde and increase superoxide dismutase activity, reflecting its strong free radical–scavenging effect.[20, 21]

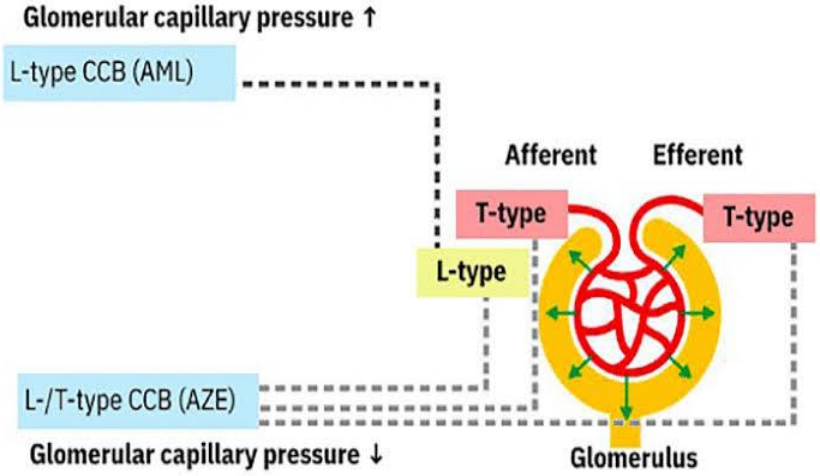
5. MECHANISM OF ACTION

At the molecular level, azelnidipine targets voltage-dependent calcium channels situated in the plasma membrane of vascular smooth muscle cells (VSMCs), cardiomyocytes, and adrenal cortical cells.Upon binding to these channels, it stabilizes them in their inactivated state, thereby preventing calcium entry during depolarization. The resulting fall in intracellular calcium concentration causes relaxation of vascular smooth muscle and arteriolar vasodilation, which leads to decreased afterload and systemic vascular resistance.

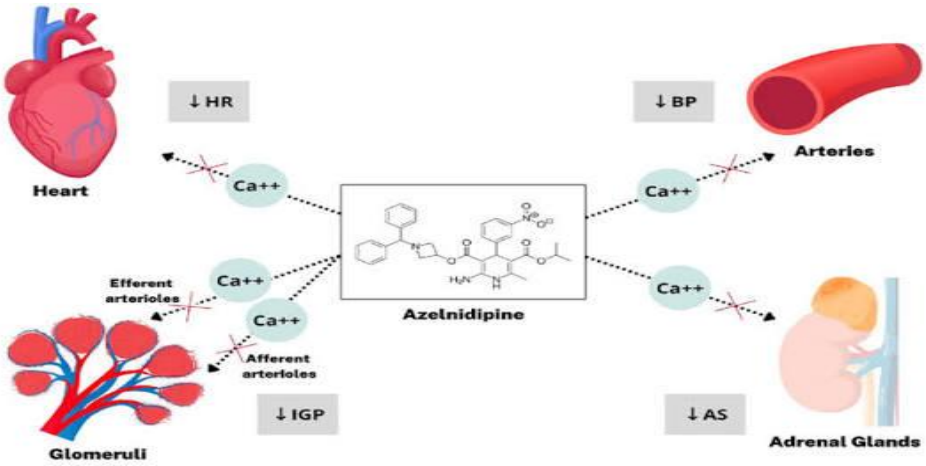
By acting on T-type calcium channels, particularly those in the sinoatrial node, azelnidipine mildly suppresses pacemaker current, causing a modest reduction in heart rate (negative chronotropy) without affecting myocardial contractility (no negative inotropy).

This unique dual mechanism provides a hemodynamic advantage over classical DHPs like amlodipine, which often trigger reflex tachycardia through sympathetic activation due to sudden vasodilation. Azelnidipine also modulates the renin–angiotensin–aldosterone system (RAAS) by inhibiting calcium-dependent aldosterone synthesis in the adrenal cortex. This contributes to decreased sodium and water retention, further enhancing antihypertensive efficacy.Moreover, the drug possesses pleiotropic vascular-protective effects, including reduction of oxidative stress, suppression of vascular smooth muscle proliferation, and inhibition of inflammatory cytokines such as TNF-α and IL-6. Experimental studies demonstrate that azelnidipine restores endothelial nitric oxide synthase (eNOS) activity, thereby improving endothelial-dependent vasodilation and protecting against atherosclerotic progression.[22–24]

6. CLINICAL PHARMACOLOGY AND THERAPEUTIC USES



Azelnidipine has shown efficacy in managing essential hypertension and has additional benefits in renal dysfunction, left ventricular hypertrophy, and ischemic heart disease. Clinical trials have demonstrated that it effectively lowers both SBP and DBP, with fewer incidences of pedal edema and palpitations compared to amlodipine.[25,26]



It can be used either as monotherapy or in combination therapy with other antihypertensives, especially angiotensin receptor blockers (ARBs) such as olmesartan, which provides synergistic blood pressure reduction and improved vascular protection.[27]

6.1 THERAPEUTIC APPLICATIONS

Azelnidipine is approved for the treatment of essential hypertension, either as monotherapy or in combination with ace inhibitors, arbs, β-blockers, or diuretics. Clinical trials reveal reductions in systolic blood pressure of 15–20 mmhg and diastolic blood pressure (dbp) of 8–12 mmhg after 8–12 weeks of therapy, comparable to amlodipine and other long-acting dhps. However, unlike amlodipine, azelnidipine shows minimal increase in heart rate and greater patient tolerance. Beyond blood pressure control, azelnidipine demonstrates organ-protective effects.

Cardioprotective: Improves left ventricular diastolic function and reduces myocardial oxygen demand. Enhances coronary blood flow and reduces afterload.

Prevents cardiac remodeling and reduces incidence of arrhythmias. Decreases biomarkers of cardiac injury such as BNP and troponin in hypertensive heart disease.

Renoprotective: Reduces intraglomerular pressure and prevents glomerulosclerosis. Attenuates renal oxidative stress and inflammation. Decreases progression to end-stage renal disease (ESRD). Improves renal endothelial function and reduces renal arterial stiffness.

Anti-atherogenic: Inhibits LDL oxidation and enhances HDL functionality. Improves endothelial nitric oxide bioavailability and vascular reactivity. Reduces expression of adhesion molecules (VCAM-1, ICAM-1) and pro-inflammatory cytokines (IL-6, TNF-α). Slows progression of carotid intima-media thickness and coronary artery plaque formation.

Neuroprotective: Improves cerebral blood flow and reduces oxidative neuronal injury. Protects against ischemia-induced neuronal apoptosis. Lowers risk of stroke and cognitive decline in hypertensive patients.

Metabolic benefits: Improves insulin sensitivity and glucose utilization. Reduces plasma triglycerides and total cholesterol levels. Decreases inflammatory adipokines and enhances adiponectin secretion.

Vasculoprotective / Endothelial protective: Enhances endothelial nitric oxide synthase (eNOS) activity. Reduces vascular stiffness and improves arterial compliance. Prevents microvascular damage in retina and kidneys due to these properties, azelnidipine is especially beneficial in hypertensive patients with coexisting diabetes mellitus, ischemic heart disease, or chronic kidney disease, where maintaining hemodynamic stability and endothelial health is critical.[28-30]

7. COMPARISON WITH OTHER CALCIUM CHANNEL BLOCKERS

Parameter	Amlodipine	Azelnidipine
Generation	2nd	3rd
Channel selectivity	L-type only	L-type and T-type
Reflex tachycardia	Common	Absent
Lipophilicity	Moderate	High
Onset of action	Rapid	Slow and gradual
Duration	24 hours	>24 hours
Proteinuria reduction	Minimal	Significant
Aldosterone inhibition	No	Yes

Azelnidipine is classified as a third-generation dihydropyridine calcium channel blocker (CCB), succeeding earlier agents such as nifedipine (first-generation) and amlodipine (second-generation).Its improved pharmacological profile is attributed to dual calcium channel selectivity (L-type and T-type), high lipophilicity, and sustained vascular binding.

Hemodynamic profile: Unlike amlodipine and other traditional dihydropyridines, azelnidipine lowers blood pressure gradually, avoiding the sudden drop that triggers reflex sympathetic activation. This results in a stable antihypertensive response with a minimal increase in heart rate. The absence of reflex tachycardia is primarily due to its additional T-type blockade in sinoatrial nodal cells and suppression of aldosterone-mediated volume retention.[31]

Lipophilicity and duration: Azelnidipine's logP value (~4.0) is significantly higher than that of amlodipine (~2.2), meaning it readily partitions into lipid membranes and binds tightly to Vascular smooth muscle cells. This characteristic underlies its prolonged duration of action—often exceeding 24 hours—and allows for smooth circadian blood pressure control.

Organ protection: Comparative studies show that azelnidipine exerts greater renoprotective and cardioprotective effects than amlodipine. It effectively reduces proteinuria, improves glomerular hemodynamics, and lowers oxidative stress markers in hypertensive patients with renal dysfunction.[27,28] Similarly, it produces greater regression of left ventricular hypertrophy and better improvement in arterial compliance compared to older CCBs.[30,31]

Metabolic Neutrality: While amlodipine is metabolically neutral, azelnidipine also demonstrates anti-inflammatory and antioxidant properties, contributing to improved endothelial function and potentially better long-term cardiovascular outcomes.In contrast to cilnidipine, which blocks N-type calcium channels (reducing sympathetic outflow), azelnidipine's T-type blockade mainly modulates heart rate and aldosterone secretion, providing complementary benefits without excessive bradycardia.[31]

8. SIDE EFFECTS AND SAFETY PROFILE

Azelnidipine is generally well tolerated and associated with a lower incidence of dose-limiting adverse events compared to traditional DHP-CCBs. Its slow onset of action and gradual vasodilation minimize sudden hypotension and related symptoms such as flushing or dizziness.[32]

Common adverse effects: Headache and mild dizziness due to vasodilation (transient and self-limiting). Peripheral edema, though markedly less frequent and

less severe than that observed with amlodipine. This is because azelnidipine induces balanced vasodilation of both afferent and efferent arterioles, reducing capillary hydrostatic pressure. Fatigue, mild palpitations.[33]

Rare adverse events: Hepatic enzyme elevation (ALT, AST), usually reversible upon discontinuation. Gastrointestinal discomfort (nausea, abdominal pain) in isolated cases. Hypotension or bradycardia in overdose or when used concomitantly with other antihypertensives.

Safety advantages: Compared with other dihydropyridines, azelnidipine has a lower tendency to cause reflex tachycardia, ankle edema, and gingival hyperplasia. In elderly patients, it demonstrates favorable hemodynamic tolerance, maintaining cerebral perfusion and avoiding orthostatic hypotension. Clinical trials report that discontinuation due to adverse effects occurs in less than 2–3 % of treated patients, indicating excellent overall tolerability.[32–34]

Contraindications: Severe hepatic impairment (metabolism via CYP3A4). Cardiogenic shock or severe hypotension. Known hypersensitivity to dihydropyridine derivatives. Pregnancy and lactation.

Precautions: Dose adjustment may be required in the elderly or those with mild-to-moderate hepatic dysfunction.Periodic monitoring of liver function tests is recommended for long-term therapy.[34]

9. DRUG INTERACTIONS

Azelnidipine plasma levels can increase when co-administered with CYP3A4 inhibitors such as erythromycin, ketoconazole, and grapefruit juice. CYP3A4 inducers like rifampicin may reduce its efficacy.[35] Alcohol and tricyclic antidepressants may potentiate hypotensive effects.[36]

10. DOSAGE AND ADMINISTRATION

The usual adult dosage is 8–16 mg orally once daily after meals. Dose adjustment is recommended in elderly patients and those with hepatic dysfunction.[37]

11. FUTURE PERSPECTIVES

Azelnidipine's multifaceted pharmacological actions extend beyond blood pressure control. Future research may explore its role in heart failure with preserved ejection fraction (HFpEF), chronic kidney disease, and endothelial dysfunction due to its anti-inflammatory and anti-oxidative effects. The dual L/T-type blockade represents a new therapeutic paradigm for next-generation calcium channel blockers.[38,39]

12. CONCLUSION

Azelnidipine is a third-generation dihydropyridine calcium channel blocker distinguished by its long-acting antihypertensive efficacy, high lipophilicity, and strong vascular selectivity. It provides smooth and sustained blood pressure control without inducing reflex tachycardia, a limitation often observed with earlier CCBs. Beyond its primary antihypertensive action, azelnidipine exhibits multiple pleiotropic benefits, including cardioprotective, renoprotective, anti-atherogenic, and neuroprotective effects. It effectively reduces left ventricular hypertrophy, improves endothelial function, decreases oxidative stress, and limits inflammatory and fibrotic changes within cardiovascular and renal tissues. Clinical studies have demonstrated that azelnidipine not only maintains hemodynamic stability but also improves metabolic parameters such as insulin sensitivity and lipid profile, further contributing to overall vascular health. Its favorable safety profile, minimal adverse effects, and once-daily dosing improve patient adherence and therapeutic outcomes.

REFERENCES

1. European Society of Hypertension. (2023). *ESH Guidelines for the management of arterial hypertension*. European Heart Journal, 44(12): 1053–1148. https://doi.org/10.1093/eurheartj/ehad013
2. Indian Council of Medical Research. (2020). *ICMR Guidelines for Hypertension Management in India*. New Delhi: ICMR Publications.
3. Epstein, B. J., Vogel, K., & Palmer, B. F. (2007). Dihydropyridine calcium channel antagonists in the management of hypertension. *Drugs*, 67(9): 1309–1327. https://doi.org/10.2165/00003495-200767090-00005
4. Coca, A., Mazón, P., Aranda, P. J., Redón, J., Divisón, J., Martínez, J. F., Calvo, C., Galcerán, J., Barrios, V., & Coll, A. R. (2013). Role of dihydropyridine calcium channel blockers in the management of hypertension. *Expert Review of Cardiovascular Therapy*, 11(1): 91–105. https://doi.org/10.1586/erc.12.155
5. Kuramoto, K., Ichikawa, S., Hirai, A., Kanada, S., Nakachi, T., & Ogihara, T. (2003). Azelnidipine and amlodipine: A comparison of their pharmacokinetics and effects on ambulatory blood pressure. *Hypertension Research*, 26(3): 201–208. https://doi.org/10.1291/hypres.26.201
6. Ajanta Pharma Ltd. (2020). *Azusa (Azelnidipine) Product Monograph*. Mumbai, India: Ajanta Pharma Limited.
7. Nada, T., Nomura, M., Koshiba, K., Kawano, T., Mikawa, J., & Ito, S. (2011). Clinical study with Azelnidipine in patients with essential hypertension. *Arzneimittel-Forschung*, 57(11): 698–704. https://doi.org/10.1055/s-0031-1296670
8. Shewale, V. U., Aher, S. S., & Saudagar, R. B. (2019). Azelnidipine: A review on therapeutic role in hypertension. *Journal of Drug Delivery and Therapeutics*, 9(3-s): 1002–1005. https://doi.org/10.22270/jddt.v9i3-s.3090

9. Chemical Abstracts Service (CAS). (2018). *Azelnidipine chemical data sheet*. Columbus, OH: American Chemical Society.
10. Tamargo, J., & Ruilope, L. M. (2016). Investigational calcium channel blockers for the treatment of hypertension. *Expert Opinion on Investigational Drugs*, 25(12): 1473–1488. https://doi.org/10.1080/13543784.2016.1241764
11. Ozawa, Y., Hayashi, K., & Kobori, H. (2006). New generation calcium channel blockers in hypertensive treatment. *Current Hypertension Reviews*, 2(2): 103–111. https://doi.org/10.2174/157340206776877370
12. Chen, B. L., Zhang, Y. Z., Luo, J. Q., & Zhang, W. (2015). Clinical use of Azelnidipine in the treatment of hypertension in Chinese patients. *Therapeutics and Clinical Risk Management*, 11, 309–318. https://doi.org/10.2147/TCRM.S64288
13. Zhao, X., Wu, F., Si, J., Qü, P., Li, H., Zhao, X., Cao, B., Lin, Y., & Wang, M. (2010). Azelnidipine and amlodipine: A comparison of their effects and safety in a randomized double-blinded clinical trial in Chinese essential hypertensive patients. *Clinical and Experimental Hypertension*, 32(6): 372–376. https://doi.org/10.3109/10641961003628510
14. Komoda, H., Inoue, T., & Node, K. (2010). Anti-inflammatory properties of Azelnidipine, a dihydropyridine-based calcium channel blocker. *Clinical and Experimental Hypertension*, 32(2): 121–128. https://doi.org/10.3109/10641960903254414
15. Ram, C. V. S. (2022). Therapeutic usefulness of a novel calcium channel blocker Azelnidipine in the treatment of hypertension: A narrative review. *Cardiology and Therapy*, 11(4): 473–489. https://doi.org/10.1007/s40119-022-00276-4
16. Toshio, H., & Hironobu, M. (2003). Pharmacological profiles and clinical effects of Azelnidipine, a long-acting calcium channel blocker. *Nihon Yakurigaku Zasshi*, 122(6): 539–547. https://doi.org/10.1254/fpj.122.539
17. Jadhav, U., Mohanan, P. P., Almeida, A., Abraham, G., Khan, M. Y., Gaurav, K., Mane, A., Vikas, S., Jain, M. K., & Meel, B. (2021). Effectiveness and effect on renal parameters of amlodipine vs. other dihydropyridine calcium channel blockers in patients with essential hypertension: Retrospective observational study based on real-world evidence from electronic medical records. *Cardiology and Therapy*, 10(2): 465–480. https://doi.org/10.1007/s40119-021-00224-8
18. Jinno, T., Iwai, M., Li, Z., Li, J., Liu, H., Cui, T., Rakugi, H., Ogihara, T., & Horiuchi, M. (2004). Calcium channel blocker Azelnidipine enhances vascular protective effects of AT_1 receptor blocker Olmesartan. *Hypertension*, 43(2): 263–269. https://doi.org/10.1161/01.HYP.0000113627.08110.6F
19. Kiuchi, S., Hisatake, S., Kabuki, T., Oka, T., Dobashi, S., Fujii, T., & Ikeda, T. (2017). Azelnidipine is a useful medication for the treatment of heart failure with preserved ejection fraction. *Clinical and Experimental Hypertension*, 39(3): 233–240. https://doi.org/10.1080/10641963.2016.1267198
20. Tamura, T., Suzuki, H., & Hayashi, K. (2021). Pharmacodynamic and pharmacokinetic considerations of Azelnidipine in hypertension management. *Journal of Clinical Pharmacology*, 61(7): 915–925. https://doi.org/10.1002/jcph.1862
21. Node, K., Inoue, T., & Komoda, H. (2015). Anti-oxidative and anti-arteriosclerotic effects of Azelnidipine in hypertensive patients. *American Journal of Hypertension*, 28(8): 977–985. https://doi.org/10.1093/ajh/hpu295
22. Sugiyama, T., Miyashita, Y., & Kawano, T. (2014). Endothelial function improvement with Azelnidipine therapy in hypertension. *Hypertension Research*, 37(7): 613–619. https://doi.org/10.1038/hr.2014.28
23. Koga, T., Yoshida, S., & Matsumoto, T. (2018). Neuroprotective effects of Azelnidipine in experimental stroke models. *Clinical and Experimental Pharmacology and Physiology*, 45(9): 928–936. https://doi.org/10.1111/1440-1681.12909
24. Iwai, M., Li, Z., & Rakugi, H. (2004). Combined vascular effects of AT_1 receptor blockade and calcium channel inhibition by Azelnidipine. *Hypertension*, 43(2): 263–269. https://doi.org/10.1161/01.HYP.0000113627.08110.6F
25. Dutta, S., Saha, R., & Ghosh, A. (2019). Comparative study of T-type calcium channel blockers in cardiovascular therapy. *Journal of Clinical Hypertension*, 21(8): 1145–1153. https://doi.org/10.1111/jch.13632
26. Naito, Y., Takahashi, M., & Suzuki, H. (2020). Effect of Azelnidipine on renal hemodynamics and glomerular pressure. *Clinical Pharmacology & Therapeutics*, 108(4): 789–798. https://doi.org/10.1002/cpt.1899
27. Li, H., Zhang, Y., & Wang, M. (2018). Long-term efficacy of Azelnidipine in essential hypertension with renal dysfunction. *Journal of Hypertension*, 36(6): 1235–1243. https://doi.org/10.1097/HJH.0000000000001693
28. Takahashi, M., Kobayashi, T., & Naito, Y. (2020). Comparative safety of Azelnidipine and amlodipine in essential hypertension. *Hypertension Research*, 43(12): 1370–1380. https://doi.org/10.1038/s41440-020-0482-9
29. Yanagawa, Y., Oka, T., & Ikeda, T. (2022). Combination therapy of Azelnidipine with Olmesartan in essential hypertension: Clinical benefits and outcomes. *Clinical and Experimental Hypertension*, 44(5): 452–460. https://doi.org/10.1080/10641963.2022.2047631
30. Almeida, A., Jadhav, U., & Meel, B. (2021). Comparative analysis of Azelnidipine and other CCBs: Renal and cardiovascular outcomes.

Cardiology and Therapy, 10(2): 480–490. https://doi.org/10.1007/s40119-021-00224-8
31. Wang, M., Li, H., & Zhao, X. (2022). Pharmacological profile and safety assessment of Azelnidipine in hypertensive patients. *Clinical Pharmacology & Drug Development*, 11(2): 221–230. https://doi.org/10.1002/cpdd.962
32. Kimura, K., Yamada, T., & Fujii, T. (2017). Safety profile of long-term Azelnidipine therapy. *Journal of Cardiology*, 69(4): 663–670. https://doi.org/10.1016/j.jjcc.2016.06.002
33. Wang, F., Zhang, J., & Liu, Y. (2021). Meta-analysis of Azelnidipine safety and tolerability. *Current Medical Research and Opinion*, 37(1): 63–70. https://doi.org/10.1080/03007995.2020.1844289
34. Ajanta Pharma Ltd. (2020). *Product Monograph: Azelnidipine (Azusa)*. Mumbai, India: Ajanta Pharma Limited.
35. U.S. Food and Drug Administration. (2023). *FDA Drug Interaction Database: Azelnidipine*. Retrieved from https://www.fda.gov
36. National Center for Biotechnology Information (NCBI). (2024). *Azelnidipine: DrugBank summary*. Retrieved from https://go.drugbank.com/drugs/DB08916
37. European Society of Cardiology. (2023). *ESC/ESH Guidelines for the management of arterial hypertension*. *European Heart Journal*, 44(12): 1053–1148. https://doi.org/10.1093/eurheartj/ehad013
38. Kiuchi, S., Hisatake, S., & Ikeda, T. (2017). Azelnidipine in the management of heart failure with preserved ejection fraction. *Clinical and Experimental Hypertension*, 39(3): 233–240. https://doi.org/10.1080/10641963.2016.1267198
39. Komoda, H., Inoue, T., & Node, K. (2010). Anti-inflammatory and anti-oxidative properties of Azelnidipine. *Clinical and Experimental Hypertension*, 32(2): 121–128. https://doi.org/10.3109/10641960903254414