



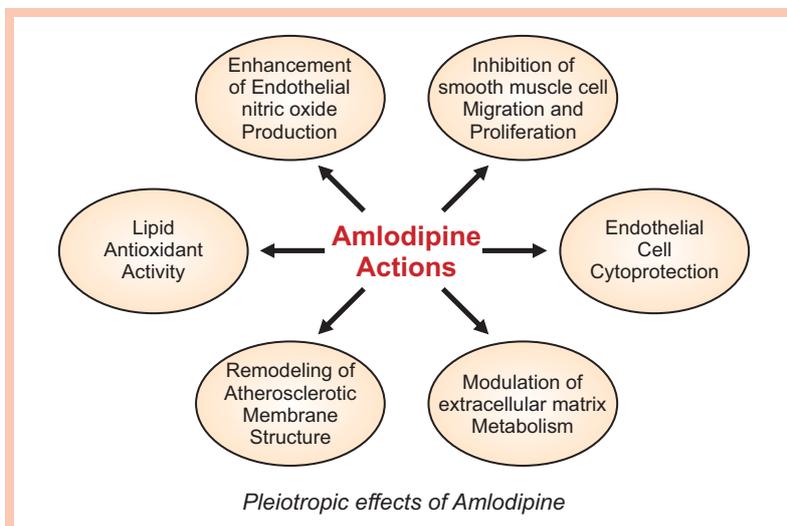
Medical Bulletin

EXCEL Division of Blue Cross Laboratories Pvt Ltd.

PLEIOTROPIC EFFECTS OF AMLODIPINE BENEFICIAL IN CARDIOVASCULAR DISEASES: ADVANTAGES BEYOND BLOOD PRESSURE REDUCTION

Calcium channel blockers (CCBs) are recommended as first-line antihypertensive therapy in major international guidelines owing to their efficacy across diverse age groups, ethnicities, and comorbidity profiles. Amongst all CCBs amlodipine is one of the most prescribed antihypertensive medications, as it possesses a highly favourable pharmacokinetic profile characterized by: Long plasma half-life (30-50 hours) enabling once-daily dosing, high bioavailability (60-80%), smooth & sustained BP reduction without reflex tachycardia, slow onset of action reducing risk of acute hypotension.

Multiple studies demonstrate that amlodipine's beneficial cardiovascular effects extend far beyond simple blood pressure control. Research has shown: Improvement in endothelial nitric oxide (NO) bioavailability, reduction in arterial stiffness and vascular remodelling, anti-inflammatory effects on vascular endothelium, inhibition of low density lipoprotein (LDL) oxidation & atherosclerotic plaque progression, reduction in left ventricular hypertrophy (LVH), lower rates of stroke and coronary outcomes in long term trials. These findings highlight the pleiotropic cardiovascular benefits of amlodipine



Effects on Vascular Tone and Peripheral Resistance:

By reducing calcium influx, amlodipine causes systemic vasodilation, particularly in the arteriolar circulation, which significantly reduces total peripheral resistance, the primary determinant of systolic blood pressure. The drug also lowers afterload, reducing the workload on the heart and improving cardiac energy efficiency, which is beneficial for patients with left ventricular hypertrophy and heart failure with preserved ejection fraction. Unlike nitrates, amlodipine does not cause venous dilation; therefore, it avoids complications like postural hypotension or venous pooling.

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Coronary Artery Dilation and Anti-Ischemic Action:

Amlodipine dilates epicardial coronary arteries and coronary arterioles, improving oxygen delivery to ischemic myocardial tissue. This property is particularly beneficial in patients with chronic stable angina, vasospastic angina (Prinz metal's), and individuals with microvascular ischemia. Enhanced coronary blood flow helps prevent episodes of angina, reduces myocardial oxygen demand, and stabilizes patients with coronary artery disease.

Anti-Atherosclerotic and Endothelial Effects:

Amlodipine reduces vascular oxidative stress by inhibiting lipid peroxidation and reducing free radical formation, contributing to slowed atherosclerotic plaque progression. It improves endothelial nitric oxide bioavailability, which enhances vasodilation and improves vascular health.

CLINICAL BENEFITS OF AMLODIPINE BEYOND BLOOD PRESSURE REDUCTION**Anti-Anginal Effects:**

Amlodipine is highly effective in the management of stable angina, as it improves myocardial oxygen supply by dilating coronary arteries and reducing afterload, thereby lowering oxygen demand. In vasospastic (Prinz metal's) angina, amlodipine suppresses spontaneous coronary artery spasm by relaxing vascular smooth muscle, making it a first line therapy in such cases. Its long action duration ensures overnight protection, a critical time when coronary spasms often occur in susceptible patients.

Regression of Left Ventricular Hypertrophy (LVH):

Chronic hypertension causes LVH due to increased afterload; amlodipine has been shown to reverse or reduce LVH, independent of blood pressure reduction. It reduces left ventricular mass by improving vascular compliance & decreasing peripheral resistance, lowering the heart's workload long-term. Studies demonstrate that LVH regression with amlodipine correlates with a reduction in future cardiac events such as heart failure & arrhythmias. Compared to non-dihydropyridine CCBs, amlodipine has superior vascular selectivity with minimal negative inotropic effect, making it safer for patients with borderline cardiac function.

Anti-Atherosclerotic and Vascular Protective Effects:

Amlodipine slows the progression of atherosclerosis by inhibiting smooth muscle proliferation, a key step in plaque development. It reduces oxidative modification of LDL, decreasing foam cell formation and plaque vulnerability. The drug improves arterial compliance and decreases stiffness, enhancing microvascular perfusion in hypertensive patients. Evidence shows amlodipine reduces inflammatory markers like c-reactive protein (CRP) and interleukin-6 (IL-6), demonstrating its role in vascular inflammation control.

Reduction of Major Adverse Cardiovascular Events (MACE):

Clinical trials consistently show that amlodipine reduces the risk of stroke, myocardial infarction, and cardiovascular death, even when blood pressure reductions are like other drug classes. The CAMELOT and PREVENT trials showed significant reduction in coronary plaque progression among patients treated with amlodipine compared to other antihypertensives.

The ASCOT-BPLA trial demonstrated a significant reduction in stroke incidence when amlodipine-based therapy was compared to atenolol-based regimens. In the ALLHAT trial, amlodipine showed comparable protection against coronary events while having a lower incidence of new onset diabetes than diuretics. Amlodipine demonstrates superiority in preventing unstable angina hospitalizations compared to ACE inhibitors in high-risk populations. Its consistent control of early-morning blood pressure surges, a major trigger for cardiovascular events, contributes to better long-term outcomes.

Overall, the evidence strongly supports amlodipine as a core component in long-term blood pressure control and cardiovascular risk reduction, making it an indispensable drug in clinical practice.

Source: Bakle A et al; IJFMR, Volume 7, Issue 6, November-December 2025.

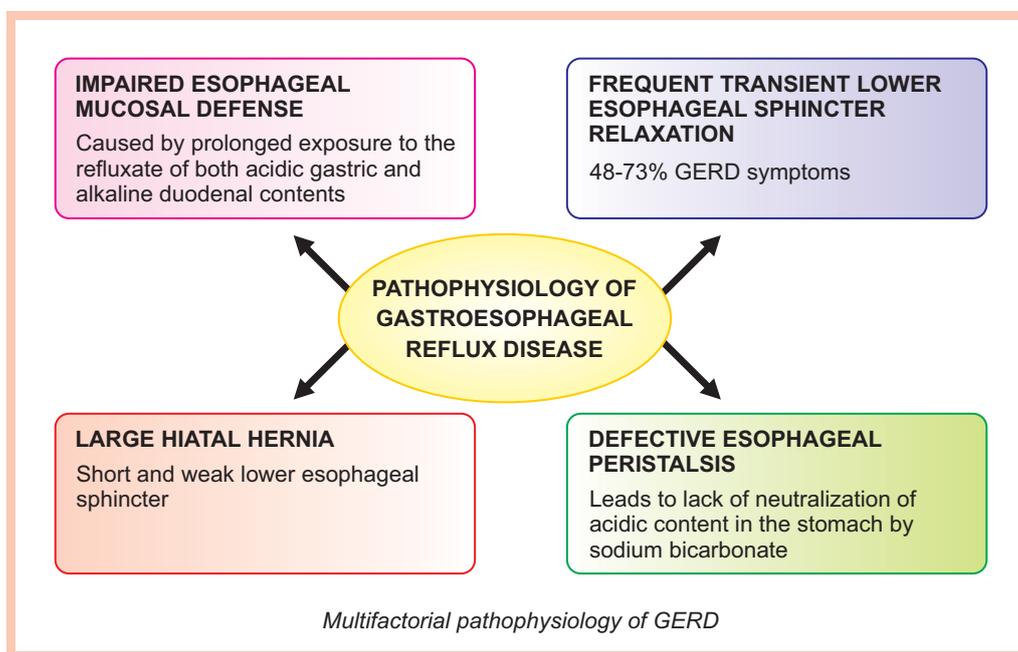
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OPTIMIZING TREATMENT OF GASTROESOPHAGEAL REFLUX DISEASE WITH DOUBLE-DOSE PANTOPRAZOLE

Gastrointestinal (GI) problems affect approximately 18% of the Indian adult population, with the prevalence significantly increasing with age. The prevalence is even higher worldwide with almost 40% of the population reporting these problems. This contributes to an enormous burden on the healthcare system and also affects the patient's quality of life. Commonest gastrointestinal complaints are indigestion, constipation, gastroesophageal reflux disease (GERD), peptic ulcer disease and piles.

Definition of GERD mentioned by American College of Gastroenterology (ACG) is the condition in which reflux of gastric contents into the esophagus results into symptoms and/or complications, and objectively as the presence of characteristic mucosal injury seen at endoscopy and/or abnormal esophageal acid exposure demonstrated on a reflux monitoring study.



The GERD is majorly represented by two symptoms: heartburn and regurgitation. However, the patient may present other symptoms such as chest pain, epigastric pain, water brash, belching, dysphagia, nausea and bloating. In addition, patients may also complain about extra-esophageal symptoms like cough, throat clearing, hoarseness of voice, burning sensation or pain in throat, wheezing and sleep disturbances. The risk of GERD increases with age, higher body mass index (BMI), smoking, anxiety/depression and physical inactivity. The presence of GERD is almost constant with chronic metabolic/lifestyle disorders. Apart from these, even iatrogenic factors like use of non-steroidal anti-inflammatory drugs (NSAID's), disease modifying agents for arthritis, long term steroid therapy for pulmonary and vascular causes contribute to increasing the overall risk of this disease.

The initial approach to managing GERD when alarm symptoms are absent consistently involves lifestyle modifications. These modifications encompass abstaining from foods known to trigger reflux episodes (such as coffee, alcohol, carbonated drinks, chocolate, and spicy foods), achieving weight loss, quitting smoking,

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elevating the head while sleeping, refraining from consuming large meals, and avoiding eating right before bedtime. Proton pump inhibitors (PPIs) have remained the cornerstone of GERD treatment because of their significant and reliable ability to suppress acid & Pantoprazole is the most prescribed drug owing to its efficacy and least interaction with other drugs. Despite, there are numerable causes for failure of PPI therapy including noncompliance, incorrect dose timing, rapid PPI metabolism, hypersecretory state in patients, hiatus hernia, visceral hypersensitivity and non-reflux esophageal causes like dysmotility, eosinophilic esophagitis, pill-induced esophagitis and infectious esophagitis. In cases where signs of esophagitis are seen, the patients are prescribed double dose of PPI for 8 weeks, as per the guidelines given by Indian Society of Gastroenterology and Association of Physicians in India.

Guideline Recommendations for Double Dose PPI

The Indian Society of Gastroenterology (ISG) and Association of Physicians of India (API) recommend increasing the dose of the same PPI to twice daily. The National Institute for Health & Care Excellence (NICE) guideline from UK recommends 8 weeks of the standard dose of PPI for the healing reflux esophagitis. If initial treatment proves ineffective, patients may be transitioned escalating the dose of the initial PPI to double the initial amount. The American College of Gastroenterology Clinical guidelines for the diagnosis and management of GERD also suggest that in patients with both extra-esophageal and typical symptoms of GERD, a trial of twice dose of PPI should be given for 8-12 weeks. It also recommends the use of PPI over H2-receptor antagonist (H2RA) for healing as well as maintenance of healed erosive esophagitis (EE).

The PPIs reach a peak plasma concentration in 2 hours after oral administration but due to its hepatic metabolism, the amount of drug available in the plasma for acid suppression is reduced during the late hours of the 24-hour dosing period. This can lead to recovery of acid secretion in the stomach by uninhibited, restored, or new proton pumps. All these factors show that there is a need of double dose of PPI that can be taken daily, which produces effect for a longer duration so that its clinical efficacy is obtained by the patients.

Patients who have severe symptoms of GERD and are not getting relief on a dose of 40 mg despite 4 weeks of treatment need to be shifted to a higher dose. Patients who have been taking NSAID's for pain (in cases of polyarthralgia and arthritis) and come with symptoms of GERD also are started on a higher dose owing to the increased risk of progression of symptoms. Patients with atypical symptoms of GERD apart from heartburn and waterbrash, like laryngitis, pharyngitis, non-cardiac chest pain and chronic cough need to be started on a higher dose. Overweight/obese patients also tend to report better symptomatic relief when a higher dose of PPI is given to them. Higher dose of PPI is also used in patients who show signs of esophagitis on endoscopy. If there is a positive response in the symptoms, the dose is usually lowered down for maintenance at 40 mg pantoprazole.

Higher dose of pantoprazole is used in patients of GERD with severe symptoms, erosive esophagitis, co-morbidities where NSAID is used, obesity, or in cases of atypical symptoms of GERD like laryngitis, chronic cough, and non-cardiac chest pain. It has shown positive response in terms of symptomatic relief and better quality of life.

Source: Upadhyay R et al; Euroasian J Hepatogastroenterol. 2024 Jan-Jun;14(1):86-91.

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GR = Gastro-resistant.



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