

“Amlodipine Induced Pedal Edema: A Clinically Overlooked Adverse Effect Managed With Telmisartan – A Case Report”

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ABSTRACT: -

Background: -Calcium Channel blockers inhibit L-type calcium channels in vascular smooth muscle and cardiac tissue, causing vasodilation and reduced cardiac workload. They are used in hypertension, angina, and arrhythmias, and include dihydropyridines (Amlodipine, Nifedipine, Felodipine) and non-dihydropyridines (Verpamil, Diltiazem).

Case report: A 52-year-old female with type 2 diabetes mellitus and hypertension presented with acute gastroenteritis and bilateral pitting pedal edema. She was on amlodipine for hypertension, and drug induced pedal edema was suspected. Investigations were normal. Amlodipine was discontinued and replaced with telmisartan, leading to improvement in edema. This case highlights amlodipine induced pedal edema as a reversible adverse drug reaction and emphasizes the importance of timely antihypertensive modification.

Key words: Calcium channel blocker, Amlodipine, Pedal edema, Adverse drug reaction, Telmisartan

I. INTRODUCTION: -

Calcium channel blockers (CCBs) include two subclasses: dihydropyridines (DHPs) and non-dihydropyridines (non-DHPs). Both share the same basic mechanism but differ in their effects. DHP CCBs are stronger vasodilators, while non DHP CCBs have greater negative inotropic action. Although both effectively reduce blood pressure, non DHP CCBs may provide added benefits in patients with chronic kidney disease and diabetic nephropathy.¹ Amlodipine is a third-generation calcium channel blocker and 1,4 dihydropyridine and also act as cardiovascular agent. Amlodipine is mainly indicated for the treatment of coronary atherosclerosis, stable angina and hypertension in adults and paediatrics above 6 years. Calcium channel blockers act by blocking voltage- sensitive

L- type calcium channels at the alpha-1 subunit, reducing calcium entry into cardiac and vascular smooth muscle. This lowers peripheral vascular resistance and blood pressure. Their benefit in angina is due to decreased vascular resistance and prevention of coronary artery spasm. These drugs have consistent pharmacokinetic and pharmacodynamic properties, including long duration of action good bioavailability, long half-life, and sustained therapeutic effect, and are generally well tolerated. The most frequently occurring adverse effects of amlodipine is pedal edema, abdominal pain, nausea, fatigue, dry mouth, constipation, hypotension, palpitation, flushing.² Here is a case report on 52 years- old female with pedal edema after treating with amlodipine for hypertension.

II. CASE REPORT: -

A 52 years old female patient was admitted to Ballari Medical College and Research Centre (A tertiary care teaching hospital), Ballari, Karnataka, India with a diagnosis of Acute gastroenteritis, type 2 diabetes mellitus, systemic hypertension.

She presented to the emergency department with the complaints of abdominal pain in the last 15 days, watery loose stools, and multiple episodes of vomiting since the morning of admission, swelling of lower limbs for 5 days.

History of present illness: The abdominal pain was described as a dull aching type, localized diffusely, with no radiation. The loose stools were watery in consistency, non-bloody, and not associated with mucus. The vomiting was non bloody, non-bilious, and non-foul smelling. Swelling of lower limbs which gradually developed and was more noticeable around the ankles. The swelling was pitting in nature and partially relived with rest and elevation. She had no complaints about pain, redness or warmth. Given

her history of hypertension and Amlodipine use, drug induced pedaledema was considered as a possible cause.

Past medical history: The patient had a known history of Type 2 Diabetes Mellitus and Systemic Hypertension in the last 6 years. She was

on regular medication including, Amlodipine 10mg, combination of Glimepride+ Metformin and Vildagliptin + Dapagliflozin. She reported good medication compliance and denied any past episodes of similar gastrointestinal complaints.

Systemic examination	Clinical examination
BP: 159/90mmHg	CVS: S1, S2, heard no murmur
PR: 91bpm	CNS: conscious and oriented
SPO ₂ : 97%	RS: B/L NVBS no added sounds
PICCLE: Edema positive	P/A: Soft and non-tender

On General Physical Examination the patient appeared moderately built and nourished. She was conscious, alert, oriented to time, place and person. As part of the diagnostic and management plan the following investigation were advised CBC, Electrolytes, Liver function test,

Renal function test, Electrocardiogram, Biochemistry, and were found to be normal.

Treatment:

The patient was initiated on the following management upon admission:

Sl. No	Name of the medication	Dose	Frequency	Route	Days
1	Inj. Pantoprazole	40mg	1-0-0	IV	D1-D5
2	Inj. Ondansetron	4mg	1-0-1	IV	D1-D5
3	Inj. Hyoscine Butyl bromide	10mg	1-0-1	IV	D1-D5
4	Inj. Ciprofloxacin	500mg	1-0-1	IV	D1-D5
5	Inj. Metronidazole	400mg	1-1-1	IV	D1-D5
6	IV Fluids	One pint RL	1-0-0	IV	D1-D5
7	Tab. Sporolac	120M spores	1-1-1	PO	D1-D5
8	Tab. Telmisartan	40mg	1-0-1	PO	D1-D5

III. DISCUSSION: -

Calcium channel blocker (CCBs) is a class of drugs widely used in the management of cardiovascular disorders such as hypertension, angina pectoris, and certain cardiac arrhythmias. They act by inhibiting L-type of voltage-gated calcium channels, thereby reducing the influx of calcium ions into vascular smooth muscle and cardiac cells. This results in vasodilation, decreased myocardial contractility, and slowed atrioventricular conduction. Based on the pharmacological effects, CCBs are broadly classified into dihydropyridines and non-dihydropyridines. Due to their efficacy and favourable safety profile, CCBs play an important role in cardiovascular therapeutics.

Amlodipine, a dihydropyridine calcium channel blocker used as an anti-hypertensive agent, commonly causes pedal edema due to selective action on vascular smooth muscle. It blocks the L-type calcium channels leading to decrease permeability of precapillary venules. This

imbalance increases capillary hydrostatic pressure, resulting in the movement of fluid from the intravascular compartment into the interstitial spaces, particularly in gravity dependent areas such as the ankles and feet. The edema is therefore caused by fluid redistribution rather than sodium or water retention, which explains why diuretics are usually ineffective and cardiac, renal and hepatic functions remain normal. Pedal edema is dose dependent, more common at higher doses, and frequently observed in elderly patients and females. It tends to worsen in the evening and improves with leg elevation or during sleep. Clinically this adverse effect can be managed by reducing the dose of amlodipine, switching to an alternative antihypertensive, or combining it with an ACE inhibitor or angiotensin receptor blocker such as telmisartan, which promotes vasodilation and helps reduce capillary pressure.

IV. CONCLUSION: -

This case highlights amlodipine induced pedal edema as a clinically significant, predictable, dose related, and reversible adverse reaction that is often under recognized in patient receiving long term antihypertensive therapy. Failure to identify this condition in a timely manner may result in progressive discomfort, reduced mobility, impaired quality of life, compromised skin integrity, secondary infections, and poor medication adherence, ultimately leading to suboptimal blood pressure control and adverse cardiovascular outcomes. Clinically, distinguishing drug induced edema from edema of cardiac, renal or hepatic origin is essential to prevent unnecessary diagnostic evaluations and inappropriate therapeutic escalation. In susceptible population, particularly elderly females and patients receiving higher doses, heightened clinical vigilance is warranted. Prompt withdrawal of amlodipine and initiation of an alternative antihypertensive agent, such as telmisartan, resulted in complete resolution of symptoms in patient, confirming the reversibility of adverse effect. This case highlights the importance of early recognition, individualized antihypertensive selection, regular medication review, and active pharmacovigilance, emphasizing the critical role of clinicals and clinical pharmacists in ensuring safe, effective, and patient centred cardiovascular care.

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