World Journal of Pharmaceutical Science and Research

www.wjpsronline.com

Review Article

ISSN: 2583-6579
SJIF Impact Factor: 6.916
Year - 2026
Volume: 5; Issue: 11
Page: 420-428

OVERLAPPING ANTITHROMBOTIC THERAPY AND OMITTED SECONDARY PREVENTION IN AN EVOLVED INFERIOR WALL MYOCARDIAL INFARCTION WITH HYPERTENSION: A CLINICAL PHARMACIST-LED CASE ANALYSIS

Seetabai Desai[1], Priya P. Nadamani[1], Aishwarya Siddamallanagouda Patil[1], Boramma B. Hunagund[1], Mallappa Shalavadi[2], Raghavendra Shivaram Hegde*[3]

[1]Pharm.D Intern, Department of Pharmacy Practice, BVV Sangha's Hanagal Shri Kumareshwar College of Pharmacy, Bagalkote – 587101, Karnataka, India.
[2]Associate Professor and Head, Department of Pharmacy Practice, BVV Sangha's Hanagal Shri Kumareshwar College of Pharmacy, Bagalkote – 587101, Karnataka, India.
[3]Assistant Professor, Department of Pharmacy Practice, BVV Sangha's Hanagal Shri Kumareshwar College of Pharmacy, Bagalkote – 587101, Karnataka, India.

Article Received: 20 September 2026 | | ***Article Revised: 11 October 2026*** | | ***Article Accepted: 01 November 2026***

***Corresponding Author: Dr. Raghavendra Shivaram Hegde**
Assistant Professor, Department of Pharmacy Practice, BVV Sangha's Hanagal Shri Kumareshwar College of Pharmacy, Bagalkote – 587101, Karnataka, India. **DOI:** https://doi.org/10.5281/zenodo.23945614 **ORCID**: 0009-0003-7523-6333

How to cite this Article: Seetabai Desai, Priya P. Nadamani, Aishwarya Siddamallanagouda Patil, Boramma B. Hunagund, Mallappa Shalavadi, Raghavendra Shivaram Hegde (2026) OVERLAPPING ANTITHROMBOTIC THERAPY AND OMITTED SECONDARY PREVENTION IN AN EVOLVED INFERIOR WALL MYOCARDIAL INFARCTION WITH HYPERTENSION: A CLINICAL PHARMACIST-LED CASE ANALYSIS. World Journal of Pharmaceutical Science and Research, 5(11), 420-428.

Copyright © 2026 Raghavendra Shivaram Hegde | World Journal of Pharmaceutical Science and Research.
This work is licensed under creative Commons Attribution-NonCommercial 4.0 International license (CC BY-NC 4.0).

ABSTRACT

Inferior wall myocardial infarction is conventionally regarded as carrying a favourable prognosis, yet the antithrombotic regimens used to treat it are among the highest-risk prescriptions written in any hospital ward. We describe a 51-year-old man admitted to the intensive coronary care unit of a tertiary care teaching hospital with typical chest pain of one day's duration and a known but entirely untreated history of hypertension. His admission blood pressure was 160/100 mmHg, two-dimensional echocardiography demonstrated inferior wall hypokinesia with a left ventricular ejection fraction of 41%, and a diagnosis of evolved inferior wall myocardial infarction with hypertension was made. A structured medication review performed by the clinical pharmacy team using the Pharmaceutical Care Network Europe classification identified eight drug-related problems. The most significant were two platelet $P2Y_{12}$ receptor inhibitors on the same chart without a documented switching strategy, a prophylactic-intensity enoxaparin dose overlapping a therapeutic unfractionated heparin infusion, and omission of a beta-blocker, a renin-angiotensin system inhibitor and any antihypertensive agent despite reduced systolic function and uncontrolled blood pressure. The discharge prescription supplied dual antiplatelet therapy and a statin for only seven days. The patient left hospital clinically stable and without bleeding, but with a regimen that was unlikely to protect him beyond the first week. This case illustrates how a single conservatively managed infarction can accumulate multiple interacting, preventable and largely invisible drug-related problems, and argues for structured pharmacist-led prescription review at both the bedside and the point of discharge.

KEYWORDS: Inferior wall myocardial infarction, Drug-related problems, Dual antiplatelet therapy, Clinical pharmacist, Secondary prevention, Prescription review.

INTRODUCTION

India carries one of the heaviest burdens of acute coronary syndrome in the world. The CREATE registry, which enrolled 20,937 patients across 89 centres and 50 cities, found that Indian patients present younger, come disproportionately from lower socioeconomic groups and have a higher proportion of ST-elevation infarction than their counterparts in high-income countries, with thirty-day mortality after ST-elevation myocardial infarction reaching 8.6%.[2] Nearly two decades later, the structural realities behind those figures – delayed presentation, limited access to catheterisation laboratories and heavy reliance on pharmacotherapy – remain familiar to clinicians working in district and teaching hospitals across the country.

Inferior wall myocardial infarction represents roughly 40% to 50% of all myocardial infarctions. In approximately 80% of patients the inferior wall of the left ventricle is supplied by the right coronary artery through the posterior descending branch, and because the same vessel perfuses the atrioventricular node, bradyarrhythmias and conduction block are characteristic accompaniments. Mortality is traditionally quoted at under 10%, which has encouraged a perception of the inferior infarct as the benign member of the family; that perception is misleading when right ventricular extension, posterior involvement or significant left ventricular dysfunction is present.[1]

What is less often discussed is that the drug therapy of an acute coronary syndrome is itself a hazard. Within a few days a patient may receive two antiplatelet agents acting on separate pathways, a parenteral anticoagulant, a nitrate, a statin, a proton pump inhibitor and an antiemetic, prescribed by different clinicians across different shifts and transcribed more than once. Several of these agents converge on the same haemostatic endpoint, and the therapeutic window between protection and haemorrhage is narrow. Contemporary guidelines from the European Society of Cardiology and from the American College of Cardiology and American Heart Association devote substantial space to exactly this balance.[3,4] Studies of cardiovascular inpatients in Indian hospitals have repeatedly shown that drug-related problems (DRPs) in this population are common rather than exceptional, averaging more than two per patient in one prospective series.[12,13]

We report the case of a middle-aged man treated conservatively for an evolved inferior wall myocardial infarction on a background of untreated hypertension, in whom a structured clinical pharmacist review identified a cluster of interacting drug-related problems. The case is presented not as a therapeutic misadventure – the patient recovered uneventfully – but as an illustration of how ordinary, well-intentioned prescribing in a busy coronary care unit can quietly accumulate risk, and of where a pharmacist standing at the chart is best placed to intervene.

CASE PRESENTATION

Presentation and history

A 51-year-old man was admitted to the intensive coronary care unit of a tertiary care teaching hospital in January 2022 with a complaint of typical retrosternal chest pain of one day's duration. The pain had been of acute onset, was non-radiating and had persisted since the previous day. There was no history of breathlessness, palpitations, syncope or orthopnoea.

He was a known hypertensive but had never been established on antihypertensive medication and had not been under regular follow-up. He was not a known diabetic and had no documented history of dyslipidaemia, prior coronary

events, cerebrovascular disease or bleeding disorder. He followed a vegetarian diet, reported a reduced appetite at presentation, slept normally and had regular bowel and bladder habits. Family history was non-contributory.

Clinical examination

On examination the patient was conscious, co-operative and well oriented to time, place and person, and was moderately built and nourished. Blood pressure was 160/100 mmHg and the pulse rate was 78 beats per minute and regular. Cardiovascular examination revealed normal first and second heart sounds with no added sounds or murmurs. The respiratory system showed bilateral equal air entry with clear breath sounds. The abdomen was soft and non-tender, and the central nervous system examination was unremarkable.

Investigations

Laboratory findings on the day of admission are summarised in Table 1. The complete blood count showed a haemoglobin of 14.8 g/dL with a leucocytosis of 14,400 cells/µL and 75% polymorphs. The platelet count and renal function were within normal limits. Serum sodium was mildly reduced at 133 mEq/L and serum chloride at 94 mEq/L, while potassium was normal. Random blood sugar was 134 mg/dL.

Two-dimensional echocardiography demonstrated inferior wall hypokinesia with a left ventricular ejection fraction of 41%. Right-sided precordial leads were not recorded, and coronary angiography was not performed. On the basis of the clinical presentation, the electrocardiographic changes and the echocardiographic findings, a diagnosis of evolved inferior wall myocardial infarction with hypertension was established.

Table 1: Laboratory investigations on the day of admission.

Investigation	Value	Reference range
Haemoglobin	14.8 g/dL	13.5–17.5 g/dL
Total leucocyte count	14,400 cells/µL	4,500–10,500 cells/µL
Polymorphs	75%	45–70%
Lymphocytes	20%	20–40%
Monocytes	3%	0–7%
Eosinophils	2%	0–5%
Red blood cell count	4.79 million/µL	4.7–6.1 million/µL
Platelet count	2,39,000 cells/mm³	1,50,000–4,50,000 cells/mm³
Random blood sugar	134 mg/dL	60–140 mg/dL
Serum sodium	133 mEq/L	135–147 mEq/L
Serum potassium	3.7 mEq/L	3.5–5.2 mEq/L
Serum chloride	94 mEq/L	95–107 mEq/L
Serum creatinine	1.0 mg/dL	0.6–1.2 mg/dL
Blood urea	22 mg/dL	15–39 mg/dL
Left ventricular ejection fraction	41% (inferior wall hypokinesia)	≥ 55%

Treatment received

The inpatient drug therapy is detailed in Table 2. Antithrombotic cover consisted of subcutaneous enoxaparin 40 mg twice daily throughout the admission, oral aspirin 150 mg once daily and oral clopidogrel 75 mg once daily. On two of the treatment days the chart also carried ticagrelor 90 mg twice daily, and on one day an unfractionated heparin infusion of 10,000 units in 100 mL at 10 mL per hour was administered while enoxaparin was continued. Supportive therapy comprised oral nitroglycerine 2.5 mg twice daily, intravenous pantoprazole 40 mg once daily, intravenous ondansetron 4 mg as required and oral atorvastatin 40 mg once daily. No beta-blocker, angiotensin-converting enzyme

inhibitor, angiotensin receptor blocker or other antihypertensive agent was prescribed at any point during the admission, and no thrombolytic therapy or percutaneous intervention was undertaken.

Table 2: Inpatient drug therapy chart.

Brand name	Generic name	Dose	Route	Frequency / days
Inj. Enclex	Enoxaparin	40 mg	S/C	Twice daily, days 1–4
Tab. Ecosprin	Aspirin	150 mg	Oral	Once daily, days 1–4
Tab. Clopivas	Clopidogrel	75 mg	Oral	Once daily, 2 days
Tab. Axcer	Ticagrelor	90 mg	Oral	Twice daily, 2 days
Inj. Heparin	Unfractionated heparin	10,000 U in 100 mL	IV infusion	10 mL/hour, 1 day
Tab. Angiplat	Nitroglycerine	2.5 mg	Oral	Twice daily, days 1–4
Tab. Avas	Atorvastatin	40 mg	Oral	Once daily, days 1–4
Inj. Pan	Pantoprazole	40 mg	IV	Once daily, days 1–4
Inj. Emeset	Ondansetron	4 mg	IV	As required

Hospital course and outcome

The patient remained haemodynamically stable throughout his stay. Blood pressure fell from 160/100 mmHg on the day of admission to 114/88 mmHg on the second day and measured 140/90 mmHg on the third day, with pulse rates between 72 and 82 beats per minute. Serial examination recorded no fresh complaints, and there was no clinical, overt or laboratory evidence of bleeding. Electrolytes and the leucocyte count were not repeated during the admission. The final diagnosis at discharge was unchanged.

He was discharged on the fifth hospital day on pantoprazole 40 mg once daily, nitroglycerine 2.5 mg twice daily, aspirin 150 mg once daily, clopidogrel 75 mg once daily, atorvastatin 40 mg once daily and ondansetron 4 mg as required – each prescribed for a duration of seven days. Ticagrelor was not continued, and no antihypertensive, beta-blocker or renin-angiotensin system inhibitor appeared on the discharge prescription.

PHARMACEUTICAL CARE ASSESSMENT

The clinical pharmacy team reviewed the medication chart daily during ward rounds and again at the point of discharge. Drug-related problems were identified and classified using the Pharmaceutical Care Network Europe (PCNE) Classification version 9.1, which defines a drug-related problem as an event or circumstance involving drug therapy that actually or potentially interferes with desired health outcomes.[11] Eight drug-related problems were documented and are summarised in Table 3. Each was communicated verbally to the treating unit at the time of identification, and the patient received structured counselling before discharge.

Table 3: Drug-related problems identified, classified by PCNE V9.1 domain.

Sr. No.	Drug-related problem	PCNE domain	Pharmacist recommendation
1	Two $P2Y_{12}$ inhibitors (clopidogrel and ticagrelor) recorded during the same admission without a documented switch or loading dose	Treatment safety – inappropriate drug combination	Confirm a single $P2Y_{12}$ agent; if escalating to ticagrelor, give a 180 mg loading dose and record the rationale
2	Enoxaparin 40 mg twice daily – a prophylactic-intensity dose used in an acute coronary syndrome	Treatment effectiveness – dose too low	Review against weight-based therapeutic dosing or document the deliberate rationale for a reduced dose
3	Unfractionated heparin infusion administered while subcutaneous enoxaparin was continued	Treatment safety – duplicate anticoagulation	Use one parenteral anticoagulant; avoid crossover, which independently increases major bleeding
4	No beta-blocker despite ejection	Treatment effectiveness	Initiate a cardioselective beta-blocker and

	fraction 41%, heart rate 78/min and no documented contraindication	– untreated indication	titrate to tolerance
5	No angiotensin-converting enzyme inhibitor or angiotensin receptor blocker despite reduced ejection fraction and hypertension	Treatment effectiveness – untreated indication	Initiate a low-dose ACE inhibitor with monitoring of renal function and potassium
6	Known hypertension left pharmacologically untreated; discharge blood pressure 140/90 mmHg	Treatment effectiveness – untreated indication	Commence antihypertensive therapy and arrange outpatient blood pressure review
7	Dual antiplatelet therapy and statin prescribed for only seven days at discharge	Treatment effectiveness – treatment duration too short	Specify dual antiplatelet therapy for twelve months and indefinite high-intensity statin therapy on the discharge summary
8	Hyponatraemia, hypochloraemia and leucocytosis not re-checked; no lipid profile or cardiac biomarker trend recorded	Other – monitoring omission	Repeat electrolytes and counts before discharge; obtain a fasting lipid profile to guide statin targets

DISCUSSION

Overlapping P2Y12 receptor inhibition

The most striking feature of this medication chart is the appearance of two oral $P2Y_{12}$ receptor inhibitors during a single admission. Clopidogrel is a thienopyridine prodrug requiring hepatic bioactivation and binds the receptor irreversibly; ticagrelor is a directly acting, reversibly binding cyclopentyltriazolopyrimidine with faster onset and more consistent platelet inhibition. In the PLATO trial, ticagrelor reduced the composite of vascular death, myocardial infarction or stroke from 11.7% to 9.8% compared with clopidogrel over twelve months, without an increase in overall major bleeding but with more non-procedure-related bleeding.[5] Choosing ticagrelor over clopidogrel is therefore defensible; what is not defensible is leaving both on an active chart.

Switching between $P2Y_{12}$ inhibitors is common in practice and has been the subject of a dedicated international expert consensus, which sets out that escalation from clopidogrel to ticagrelor after an acute event should be performed with a 180 mg loading dose irrespective of the timing of the last clopidogrel dose.[6] Neither a loading dose nor a documented rationale for the change appears in this record. In the absence of such documentation, the transition cannot be distinguished from inadvertent therapeutic duplication, and the nursing and dispensing staff who work from the chart have no way of knowing which agent is intended. Duplication of $P2Y_{12}$ blockade on a background of aspirin and two parenteral anticoagulants constitutes a genuine haemorrhagic hazard, not a documentation nicety.

Dose and duplication in parenteral anticoagulation

Two separate problems arise on the anticoagulant side. The first is intensity. Enoxaparin 40 mg twice daily sits awkwardly between the once-daily prophylactic dose and the weight-adjusted therapeutic regimen used in acute coronary syndromes; for a moderately built adult male it is almost certainly sub-therapeutic as treatment. The second is overlap. On one treatment day an unfractionated heparin infusion was administered while subcutaneous enoxaparin continued. Data from the SYNERGY trial are instructive here: crossover between the two anticoagulants raised TIMI major bleeding from 3.7% to 7.8% in patients moving from enoxaparin to unfractionated heparin, and from 2.5% to 8.6% in those moving in the opposite direction.[7] The lesson repeated in every subsequent guideline is to select one

parenteral anticoagulant and remain with it.[3,4] That this patient did not bleed reflects good fortune and a short exposure rather than a safe prescription.

Omission of guideline-directed secondary prevention

Errors of omission are quieter than errors of commission and are consequently harder to catch. This patient left hospital without a beta-blocker, without a renin-angiotensin system inhibitor and without any antihypertensive agent, despite an ejection fraction of 41% and a documented history of hypertension that had never been treated. The REDUCE-AMI trial, which has reasonably prompted clinicians to reconsider routine beta-blockade after infarction, enrolled only patients with a left ventricular ejection fraction of at least 50% and found no benefit in that group over a median 3.5 years.[8] Its findings do not extend to a patient with an ejection fraction of 41%, in whom beta-blockade and renin-angiotensin system inhibition remain cornerstones of guideline-directed therapy.[3,4] The omission here appears to be oversight rather than a reasoned decision, since no contraindication is recorded and the heart rate and blood pressure both permitted initiation.

Equally consequential is duration. Dual antiplatelet therapy and a high-intensity statin were dispensed for seven days. Current European guidance recommends dual antiplatelet therapy for at least twelve months after an acute coronary syndrome and explicitly advises against de-escalation within the first thirty days.[3] A seven-day supply in a health system where many patients will not return unless symptomatic is effectively a one-week course of secondary prevention after a myocardial infarction. The discharge prescription, more than any inpatient order, determines what happens to this man over the next year, and it is precisely the document that receives the least scrutiny.

Nitrate therapy in inferior wall infarction

Oral nitroglycerine was prescribed twice daily without right-sided precordial leads having been recorded. Right ventricular involvement complicates a substantial proportion of inferior infarctions, and such patients are preload-dependent and may become profoundly hypotensive when venodilated. It should be acknowledged that the strength of this traditional prohibition has been questioned: a systematic review and meta-analysis of five studies involving 1,113 patients found no statistically significant excess of adverse events with sublingual nitroglycerine in right ventricular infarction, and concluded that the adverse events observed were transient and readily managed.[9] The point for practice is therefore not that nitrate use was wrong, but that the assessment on which the decision should rest – a V4R recording – was never made. Prescribing a preload-reducing agent in an inferior infarct without excluding right ventricular extension leaves the clinician without the information needed to anticipate a hypotensive response.

Counselling, monitoring and the discharge interface

Counselling delivered before discharge covered the nature of the infarction, the importance of not missing doses, administration of pantoprazole thirty minutes before food, dietary modification towards whole grains, vegetables, fruit and lean protein, avoidance of refined sugar and processed food, adequate rest, and regular medical review. Advice to avoid grapefruit juice while taking clopidogrel is pharmacologically well-founded: grapefruit constituents inactivate intestinal CYP3A4 and inhibit CYP2C19, and in a randomised crossover study grapefruit juice reduced the peak plasma concentration of the active metabolite of clopidogrel to 13% of control values with a measurable fall in platelet inhibition.[10] The same counselling session would have been strengthened by explicit instruction on recognising bleeding, since the patient was leaving on two antiplatelet agents.

Monitoring gaps were also evident. The mild hyponatraemia, hypochloraemia and leucocytosis present on admission were never re-checked, no lipid profile was obtained despite dyslipidaemia being listed among the patient's risk factors and a high-intensity statin being prescribed, and no cardiac biomarker trend was recorded. None of these omissions harmed this patient, but each represents a lost opportunity to individualise therapy.

The clinical pharmacist at the chart

None of the problems described here required sophisticated pharmacological insight. They required somebody to read the whole chart at once, in sequence, with the discharge prescription alongside the inpatient orders. That is the characteristic contribution of a ward-based clinical pharmacist, and it is reflected in the Indian literature: a prospective study of 189 cardiovascular inpatients identified 416 drug-related problems, an average of 2.2 per patient, with the majority of pharmacist recommendations accepted by the treating team.[12] A further Indian series of cardiovascular inpatients using the same PCNE framework reached similar conclusions about the frequency of such problems and the value of structured intervention.[13] The present case, with eight drug-related problems in a single uncomplicated admission, sits comfortably within that range.

It is worth emphasising that this patient recovered and was discharged stable. The case is reported precisely because the outcome was good. Drug-related problems that produce no visible harm are the ones that persist, because nothing in the clinical course prompts anyone to look for them. A documented, structured review is what converts an invisible risk into a correctable one.

Limitations

This is a single-patient report from one centre, managed conservatively without coronary angiography, and the drug-related problems described were identified by retrospective structured review of the medication chart rather than by prospective prescriber-acceptance recording. The clinical significance of each problem is therefore assessed against guideline standards rather than against observed harm, and the findings are not generalisable in a statistical sense. They are offered as an illustrative account of patterns that are familiar to clinical pharmacists working in coronary care.

CONCLUSION

A conservatively managed inferior wall myocardial infarction in a previously untreated hypertensive man accumulated eight drug-related problems over a five-day admission: overlapping $P2Y_{12}$ receptor inhibition without a documented switching strategy, a sub-therapeutic enoxaparin dose overlapping a heparin infusion, omission of beta-blockade and renin-angiotensin system inhibition in the presence of reduced systolic function, continued non-treatment of hypertension, and a discharge prescription that supplied seven days of therapy after an event requiring twelve months of it. The patient came to no harm, which is exactly why such problems are so durable. Structured medication review by a clinical pharmacist at the bedside and, critically, at the point of discharge offers a practical and low-cost safeguard in settings where acute coronary syndromes are managed largely with drugs. Embedding prescription review and clinical audit into routine coronary care is likely to yield benefits out of proportion to its cost.

ETHICAL APPROVAL AND CONSENT

This report describes a single patient and did not require institutional ethics committee approval under the institutional policy applicable to individual case reports. Written informed consent was obtained from the patient for publication of the clinical details. All identifying information has been removed, and the patient is referred to by initials only.

ACKNOWLEDGEMENT

The authors thank the faculty of the Department of Pharmacy Practice and the staff of the Department of Cardiology for their support during the preparation of this report.

FUNDING

This work received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

AUTHOR CONTRIBUTIONS

All authors participated in case data collection, medication review, drafting and critical revision of the manuscript, and all approved the final version for submission.

REFERENCES

1. Warner MJ, Tivakaran VS. Inferior myocardial infarction. In: StatPearls, Treasure Island (FL); StatPearls Publishing, 2023.
2. Xavier D, Pais P, Devereaux PJ, Xie C, Prabhakaran D, Reddy KS, et al. Treatment and outcomes of acute coronary syndromes in India (CREATE): a prospective analysis of registry data. Lancet, 2008; 371(9622): 1435-42.
3. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J, 2023; 44(38): 3720-826.
4. Rao SV, O'Donoghue ML, Ruel M, Rab T, Tamis-Holland JE, Alexander JH, et al. 2025 ACC/AHA/ACEP/NAEMSP/SCAI guideline for the management of patients with acute coronary syndromes. J Am Coll Cardiol, 2025; 85(22): 2135-237.
5. Wallentin L, Becker RC, Budaj A, Cannon CP, Emanuelsson H, Held C, et al. Ticagrelor versus clopidogrel in patients with acute coronary syndromes. N Engl J Med, 2009; 361(11): 1045-57.
6. Angiolillo DJ, Rollini F, Storey RF, Bhatt DL, James S, Schneider DJ, et al. International expert consensus on switching platelet P2Y12 receptor-inhibiting therapies. Circulation, 2017; 136(20): 1955-75.
7. White HD, Kleiman NS, Mahaffey KW, Lokhnygina Y, Pieper KS, Chiswell K, et al. Efficacy and safety of enoxaparin compared with unfractionated heparin in high-risk patients with non-ST-segment elevation acute coronary syndrome undergoing percutaneous coronary intervention in the SYNERGY trial. Am Heart J, 2006; 152(6): 1042-50.
8. Yndigegn T, Lindahl B, Mars K, Alfredsson J, Benatar J, Brandin L, et al. Beta-blockers after myocardial infarction and preserved ejection fraction. N Engl J Med, 2024; 390(15): 1372-81.
9. Wilkinson-Stokes M, Betson J, Sawyer S. Adverse events from nitrate administration during right ventricular myocardial infarction: a systematic review and meta-analysis. Emerg Med J, 2023; 40(2): 108-13.
10. Holmberg MT, Tornio A, Neuvonen M, Neuvonen PJ, Backman JT, Niemi M. Grapefruit juice inhibits the metabolic activation of clopidogrel. Clin Pharmacol Ther, 2014; 95(3): 307-13.
11. Pharmaceutical Care Network Europe Association. PCNE classification for drug related problems V9.1. Zuidlaren; PCNE: 2020. https://www.pcne.org/upload/files/417_PCNE_classification_V9-1_final.pdf

12. Movva R, Jampani A, Nathani J, Pinnamaneni SH, Challa SR. A prospective study of incidence of medication-related problems in general medicine ward of a tertiary care hospital. J Adv Pharm Technol Res, 2015; 6(4): 190-4.
13. Biradar SM, Kohima B, Nayak V, et al. Assessment of drug related problems and pharmacist interventions in inpatients with cardiovascular disease. Ration Pharmacother Cardiol, 2022; 18(5): 536-43.