

STUDY ON THE PREVALENCE AND CLINICAL SIGNIFICANCE OF DRUG INTERACTIONS IN CARDIOVASCULAR PATIENTS IN A COMMUNITY PHARMACY

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ABSTRACT

Cardiovascular diseases require long-term pharmacotherapy and are often associated with polypharmacy, which increases the risk of drug interactions that may influence treatment efficacy and patient safety. This study aimed to evaluate the prevalence and statistical significance of drug interactions among patients with cardiovascular diseases in a community pharmacy setting. A prospective cross-sectional observational study was conducted over 6 months in a community pharmacy set-up. A total of 150 prescriptions with cardiovascular medicines were analyzed. Potential drug–drug, drug–food, and drug–gene interactions were identified using the Lexi drug interaction database. Statistical analysis was performed using a one-sample proportion test in SPSS software. A total of 2,216 potential drug interactions were identified, comprising 1,556 drug–drug interactions, 334 drug–food interactions, and 326 drug–gene interactions. Statistical analysis of drug–gene interactions showed that aspirin–CYP2C9 interaction was observed in 22.5% of cases, clopidogrel–CYP2C19 interaction in 73.6% of cases, atorvastatin–ABCG2/SLCO1B1 interaction in 37.3% of cases, and rosuvastatin–SLCO1B1 interaction in 38.9% of cases. Since all p-values were greater than 0.05, there was no statistically significant difference between the observed project data and the values reported in reference clinical studies. Drug–drug and drug–gene interactions have been identified as important factors that may reduce the efficacy of cardiovascular medications, potentially contributing to the recent rise in premature cardiovascular disease deaths, highlighting the importance of routine prescription screening, genomic screening and pharmacist intervention to enhance medication safety and optimize therapeutic outcomes.

KEYWORDS: Cardiovascular diseases, drug-drug interactions, drug-gene interactions, Precision medicine, therapeutic outcome.

INTRODUCTION

Cardiovascular diseases (CVDs) remain one of the most significant health burdens globally, with a steady rise in prevalence, morbidity, and mortality in recent decades. The management of these conditions often involves long-term pharmacotherapy, frequently with multiple medications to address comorbidities such as hypertension, dyslipidaemia, diabetes, and heart failure.

As the complexity of therapy increases, so too does the risk of unintended consequences of treatment. Studies in cardiology wards have reported that a majority of patients receive numerous medications and that potential drug interactions are extremely common in this population.^[1]

In the context of cardiovascular pharmacotherapy, drug interactions specifically drug–drug, drug–food, and drug–gene interactions are of particular concern. Drug–drug interactions (DDIs) occur when one pharmaceutical agent alters the effect or pharmacokinetics of another agent, potentially leading to diminished therapeutic effect or increased toxicity. Drug–food interactions involve dietary substances that modify drug absorption, metabolism, or action. Drug–gene interactions reflect how individual genetic variability can influence drug response and predispose patients to interaction-mediated adverse outcomes. The interplay of these factors in patients receiving cardiovascular medications may contribute to sub-optimal outcomes and hidden complications.^[2]

Despite the importance of these interactions, many remain under-recognized or silent in everyday practice, particularly in the community pharmacy setting, where patients may receive prescriptions from multiple providers, self-medicate, or use over-the-counter medications in addition to prescribed drugs. These unrecognized interactions can lead to therapeutic failure, worsening cardiovascular conditions, increased adverse drug events, hospitalization, and elevated healthcare costs. For instance, high rates of potential DDIs have been documented in cardiac patient populations, often correlated with polypharmacy, comorbidities, and older age.^[3]

Hence, the present study sought to identify the underlying and often overlooked factors responsible for the progression and aggravation of cardiovascular conditions, which may arise from unrecognized drug–drug, drug–food, and drug–gene interactions. Further, the study will examine how such interactions may deteriorate patient condition, increase risk of complications, and impact overall therapeutic outcomes in a real-world community pharmacy context.^[4]

By focusing on cardiovascular patients in a community pharmacy environment, our study will deliver insight into the prevalence, types, mechanisms, and clinical significance of interactions, thereby illuminating the “silent reasons” behind worsening cardiovascular conditions. This underscores the vital role of the community pharmacist in the early detection, patient counselling, and prevention of interaction-related harm.^[5]

Overall, this study will not only contribute to understanding of the prevalence and clinical importance of drug interactions in cardiovascular therapy but will also highlight the critical role of community pharmacists in preventing adverse events and optimizing therapy. Through systematic evaluation, the research aims to support the development of strategies and educational programs that enhance medication safety and patient awareness. Ultimately, the project seeks to strengthen the bridge between pharmacotherapy and clinical outcomes

by identifying and addressing the silent yet impactful factor drug interactions that often remains hidden in cardiovascular disease management.^[6]

METHODS

A Prospective Cross-sectional Observational Study was carried out for a period of 6 months in a community pharmacy located at Coimbatore and a total of 150 prescriptions presented to the community pharmacy were selected. Drug–drug, drug–food, and drug–gene interactions were identified using Lexi drug software and categorized as minor, moderate, or major based on severity. The prevalence of various drug interactions in patients with cardiovascular conditions was estimated, and safer therapeutic alternatives were identified. The collected data were analyzed using SPSS software to run confidence intervals and one-sample proportion tests to find if the prevalence patterns and clinical significance of drug–gene interactions in our study were significant.

RESULTS AND DISCUSSION

Of the 150 prescriptions analyzed in this study a total of 2,216 probable drug interactions were identified, comprising 1,556 probable drug–drug interactions, 326 probable drug–gene interactions, and 334 probable drug–food interactions, as summarized in Table 1 and illustrated in Figure 1.

The 1,556 identified drug–drug interactions were classified into minor (301), moderate (1,007), and major (248) categories, as detailed in Table 2. Among the 301 minor interactions, 205 occurred between prescription drugs, while 96 involved prescription and OTC medications. Of the 1,007 moderate interactions, 474 were observed between prescription drugs and 533 between prescription and non-prescription drugs. Among the 248 major interactions, 19 were between prescription drugs while 229 were between prescription and OTC medications.

The most frequently observed minor drug–drug interactions were clopidogrel with atorvastatin (25), cilnidipine with ibuprofen (16), and cilnidipine with naproxen (14). The most commonly encountered moderate interactions were metoprolol with aceclofenac (24), telmisartan with aceclofenac (24), and clopidogrel with aceclofenac (25). In the major interaction category, aspirin with aceclofenac (36) was the most frequent, followed by aspirin with ibuprofen (34) and aspirin with naproxen (34), as shown in Table 3.

In addition, 305 probable drug–gene interactions were documented. The most common drug–gene interactions include aspirin with CYP2C9-rapid metabolizer (89) (Figure 2), clopidogrel with CYP2C19-poor metabolizer (72) (Figure 3), atorvastatin with ABCG2 and SLCO1B1-rapid metabolizer (59) (Figure 4), metoprolol with CYP2D6-poor metabolizer (42), rosuvastatin with ABCG2 and SLCO1B1-rapid metabolizer (18) (Figure 5).

Among the total study population of 150 patients, 89 were receiving aspirin, 72 were on clopidogrel, 59 were on atorvastatin, and 18 were on rosuvastatin. We attempted to estimate the number of individuals within these subgroups who were likely to have the following drug–gene interactions: Aspirin–CYP2C9, Clopidogrel–CYP2C19, Atorvastatin–ABCG2 + SLCO1B1, and Rosuvastatin–ABCG2 + SLCO1B1.

A total of 334 DFIs that affected drug absorption and bioavailability were identified, and patient counselling was provided accordingly. Certain drugs exhibit both DDIs and DGIs simultaneously. For example, CYP2C19 poor metabolizer have reduced activation of clopidogrel, causing decreased plasma level of clopidogrel, which are further suppressed when combined with other cardiovascular medicines, such as atorvastatin. These findings underscore the necessity of comprehensive pharmacogenomic screening and structured prescription review to optimize cardiovascular therapy and prevent the failure of treatment. The integration of pharmacogenomic data into routine clinical practice represents a key step toward precision medicine, enabling individualized drug selection and dosing based on the genetic profile of each patient.

In our study, prescriptions were more common among males than females, consistent with S. Rajesh Raja et al. (2022), who reported that 68% of participants were male. Consequently, the risk of drug–drug interactions (DDIs) was higher among male patients.^[7]

Most prescriptions were observed in the 55–64 years age group, reflecting the increased prevalence of cardiovascular disease (CVD) among individuals over 45 years, as reported by Waquar Ahmed et al. (2023). Long-term cardiovascular pharmacotherapy in this population increases the likelihood of DDIs.^[8]

The identified DDIs were classified as minor, moderate, and major, with moderate interactions being the most common, similar to findings by Yugandhar et al. (2018). Although often overlooked, moderate interactions can significantly affect treatment outcomes and require careful monitoring.^[9]

Clinically important DDIs were frequently observed between cardiovascular medications and OTC NSAIDs. For example, aceclofenac may increase bleeding risk when combined with antiplatelet agents and reduce the antihypertensive and diuretic effects of furosemide. Limited patient and pharmacist awareness of such interactions may compromise treatment efficacy and safety. Jie Wei et al. (2023) also highlighted the increased cardiovascular risks associated with NSAID use, emphasizing the need for patient counselling.^[10]

Drug–food interactions were also common, particularly with medications such as aspirin, clopidogrel, telmisartan, atorvastatin, digoxin, and carvedilol. Food

can alter drug absorption, metabolism, and therapeutic response. Appropriate administration timing is therefore essential; for instance, isosorbide dinitrate should be taken on an empty stomach, whereas aspirin and carvedilol are better taken with food. Pharmacists play a key role in educating patients about these interactions to improve treatment outcomes and adherence.

Drug–gene interactions were identified involving Aspirin–CYP2C9, Metoprolol–CYP2D6, Clopidogrel–CYP2C19, and statins (Atorvastatin and Rosuvastatin) with ABCG2 and SLCO1B1 polymorphisms. Genetic variations in drug-metabolizing enzymes and transporters can alter drug efficacy and toxicity, highlighting the importance of pharmacogenomics in optimizing cardiovascular therapy.

Drug–gene interactions influence drug response by altering metabolism and absorption. In poor metabolizers, reduced CYP2C19 activity decreases the activation of prodrugs such as Clopidogrel, resulting in lower therapeutic efficacy and an increased risk of treatment failure. Conversely, reduced CYP2D6 activity slows the metabolism of active drugs such as Metoprolol, leading to higher plasma concentrations and an increased risk of toxicity.

Genetic variations in drug transporters can also affect drug absorption. For example, increased ABCB1 (P-glycoprotein) activity may reduce digoxin absorption by enhancing drug efflux from intestinal cells, thereby lowering systemic drug exposure and therapeutic effectiveness.

Our study identified a high prevalence of drug–gene interactions involving commonly prescribed cardiovascular drugs, including Aspirin, Clopidogrel, Atorvastatin, and Rosuvastatin. Statistical analysis using a one-sample proportion test showed a significant difference compared with previously reported data, emphasizing the need for careful monitoring of pharmacogenomic interactions in cardiovascular therapy.

Table 1: Drug Interactions.

(N= 2214)	
Drug Interactions	No. of Interactions
Drug-drug interaction	1556 (70.79%)
Drug-food interaction	334 (15.19%)
Drug-gene interaction	324 (14.01%)

Table 2: Drug-Drug Interactions.

(N= 1556)	
Drug-Drug Interactions	No. of Interactions
Major	301 (19.34)
Moderate	1007 (64.71%)
Mild	248 (15.93%)

Table 3: Major Drug-Drug Interactions.

(N=179)	
Major Drug-Drug Interactions	No. of Interactions
Aspirin-Aceclofenac	36 (20.11%)
Aspirin-Ibuprofen	34(18.99%)
Aspirin-Naproxen	34(18.99%)
Aspirin and Clopidogrel-Aceclofenac	20(11.17%)
Aspirin and Clopidogrel-Ibuprofen	13 (7.26%)
Furosemide-Aceclofenac	12 (6.70%)
Furosemide-Ibuprofen	11 (6.14%)
Furosemide-Naproxen	11 (6.14%)
Aspirin and Clopidogrel-Naproxen	8 (4.46%)

CONCLUSION

Drug–drug (DDIs), drug–gene (DGIs), and drug–food interactions (DFIs) can significantly alter the pharmacokinetics and pharmacodynamics of cardiovascular drugs. These interactions often alter the drug concentrations below the Minimum Effective Concentration (MEC), or above the Maximum Safe Concentration (MSC), resulting in loss of therapeutic effect or adverse effects respectively.

Therapeutic failure may result from increased metabolism (e.g., CYP2C19, CYP2D6 ultra-rapid metabolizers), reduced absorption (e.g., transporter polymorphisms such as ABCB1), or impaired prodrug activation (e.g., clopidogrel in poor metabolizers). Such subtherapeutic exposure can gradually lead to inadequate disease control, unnoticed by both patients and healthcare providers alike.

These interactions are frequently under-recognized in clinical practice, as attention is often given only to acute adverse events. Therefore, continuous monitoring of drug response, pharmacogenetic screening, and patient counselling regarding concomitant medications and diet are critical for optimizing cardiovascular therapy.

Our study revealed that each prescription carried a 15-fold increased risk of drug interactions, driven by polypharmacy, genetic variability, and food interactions.

These findings emphasize the urgent need for routine pharmacogenomic marker screening, whereby identified genetic profiles guide dose adjustments to achieve precision medicine which ultimately enabling a considerable reduction in cardiovascular mortality. The results highlight a timely and essential step toward improving cardiovascular treatment outcomes and addressing the rising burden of cardiovascular disease in India.

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