Original Article

Personalized Medicine In The Treatment Of Cardiovascular Disease.

Kamran Riaz[1],Zia ul Hassan[2],Rafi ullah[3],Zakir Ullah[4],Muhammad Ejaz[5]

1. Trainee Medical Officer HMC Peshawar
2. Trainee Medical Officer Jinnah Medical College, Peshawar
3. FCPS Cardiology, Fellow Interventional Cardiology HMC Peshawar
4. FCPS Cardiology, Fellow Interventional Cardiology HMC Peshawar
5. FCPS Cardiology, Fellow Intervention Cardiology HMC Peshawar

ABSTRACT

Background: focuses on genetic characteristics and patient-specific environmental and behavioral patterns to devise treatment plans. This approach aims to maximize treatment outcomes, minimize side effects, and enhance the prediction of treatment outcomes.

Objectives: to evaluate the role of personalization of treatment in cardiology on the integration of genetic disposition and pharmacological therapies, and the impact on lifestyle modification in improving outcomes and reducing complication risks during the treatment.

Methodology: A total of 200 patients are undergoing diagnostic evaluation for cardiovascular conditions. The mean age of participants was 56.4 ± 11.8 years. Baseline data were collected on clinical presentation, molecular parameters, and pharmacological therapy response, along with assessments of lifestyle and health behaviors. Treatment decisions were guided by advanced imaging, biomarker evaluation, and integrated risk prediction algorithms. Patient outcomes were monitored over a 12-month follow-up period.

Results:200 patients whose Mean age was 56.4 ± 11.8 years. Individualized medical therapies provided patients with specific health benefits by decreasing the adverse responses to medications. Patients who received personalized medication reported a 20% reduction in treatment-related heart complications compared to patients who received standard therapeutic care. Pharmacogenomic approaches empowered specialists to escalate drug regimens, which resulted in 15% reductions in adverse effects. Robust diagnostic tools and genetic tests, combined, provided physicians with precise treatment and care options, which enabled personalized approaches to medicine to statistically outperform the standard care model at $p = 0.03$. The data indicate that specific modifications to the management of progressive cardiovascular disease improve the efficiency of the designed strategies.

Conclusion: Cardiovascular medicine prioritizes effective, patient-centered methods to minimize adverse consequences and improve quality of life.

Keywords: Cardiovascular Diseases, Pharmacogenomics, Quality of Life

Article Metadata

Corresponding Author

Zia ul Hassan

Trainee Medical Officer Jinnah Medical College, Peshawar

Email: ziah3740@gmail.com

https://orcid.org/0000-0002-6489-5603

Article History

Received: 22th January, 2025

Revised: 18th February, 2025

Accepted:27th March,2025

Published 05th April,2025

DOI: https://doi.org/10.64911/qs4rbc66

This article may be cited as:

Gul R, Ullah S, Ullah W. Prevalence Of Iron Deficiency Anemia In Patients With Chronic Infections A Focus On Malaria And Typhoid Patients.**J Pak Int Med Coll. 2025;2(1):55–60.**

INTRODUCTION

The Apo lipoprotein E (APOE) genome focused on identifying potential variations within pathways of lipid metabolism to inform patient-specific therapy [2]. Optimizing therapeutic ranges demonstrates the potential to enhance clinical results while minimizing adverse effects from medications and illustrates the value of individualized pharmacotherapy [3]. In the case of cardiovascular diseases, the constellation of outcome-related factors becomes more complicated, as it involves not only genetic factors but also behavioral and lifestyle determinants, such as diet, physical activity, and stress management. For cardiovascular risk, personalized medicine combines pharmacogenomics, genetic testing, and lifestyle changes. It involves the construction of health profiles based on molecular, clinical, and behavioral variables, which will inform the development of tailored strategies. Integrated approaches alleviate the burden of disease and enhance the quality of life through proactive and preventive care. Rider et al. (2017) demonstrated that genetic therapy, combined with therapeutic lifestyle changes, yielded more favorable outcomes and fewer cardiovascular events compared to conventional therapy [5].Although there has been notable progress in personalized cardiovascular care, the integration remains multifaceted and uneven across patient demographics [6]. Integrating clinical genetics and pharmacology has been qualitatively ranked as the most influential determinant in the outcome’s clinical course alongside the care receiver’s health preferences. Shortages in advanced healthcare technologies, especially genetic assessments, continue to be a barrier. The tools involving systolic high-sensitivity CRP (hs-CRP) and B-type natriuretic peptide (BNP) thresholds have been pivotal in assessing the steps for achieving patient goals, and genetic assessments for modulating warfarin have been documented to limit adverse outcomes [7-9]. Artificial Intelligence (AI) systems, developed around large datasets, have been able to indicate cardiovascular risks and forecast responses to treatment at a higher rate than traditional systems, although gaps in integration remain.This investigation aimed to assess the clinical value derived from personalized cardiovascular care through the integration of pharmacogenomic assessments, genetic markers, and lifestyle data comparisons[10-12].

RESERCH OBJECTIVES

This aimed this Prospective study was implemented through a prospective cohort study, where 200 cardiovascular patients were enrolled for a 1-year study at the study center, a tertiary care facility. Participants had an average age of 56.2 ± 10.5 years. After randomization, participants were placed in either an intervention or a control condition.

METHODOLOGY

This Prospective Study Conducted in the Department of Medical Jinnah Medical College, Peshawar from jan 2023 to jan 2024. 200 patients are undergoing diagnostic evaluation for cardiovascular conditions. The mean age of participants was 56.4 ± 11.8 years. Baseline data were collected on clinical presentation, molecular parameters, and pharmacological therapy response, along with assessments of lifestyle and health behaviors. Treatment decisions were guided by advanced imaging, biomarker evaluation, and integrated risk prediction algorithms. Patient outcomes were monitored over a 12-month follow-up period.

INCLUSION CRITERIA

To qualify, individuals had to be aged between 18 and 80, have a history of cardiovascular disease, and provide informed consent.

EXCLUSION CRITERIA

Exclusions included persons with poorly controlled comorbidities, such as malignancy and chronic renal failure, individuals aged 18 or older who were pregnant and nursing infants, and those who refused or were unable to consent.

DATA COLLECTION

Data were collected with a three-prong approach, which included interviews, clinical examinations, and laboratory investigations. The inclusion of pharmacogenomics, which assesses genetic variants of APOE and CYP450 to determine individualized pharmacological care, particularly with statin and anticoagulant adjustment plans, has been integrated. Assessments of lifestyle factors (diet, physical activity, and stress) were used to develop and integrate care plans. The documentation of patients’ health status was conducted at the beginning and end of the intervention. The data collected were kept confidential.

STATISTICAL ANALYSIS

Analysis was conducted in SPSS Version 22. Descriptive analysis was performed on the quantitative data, which was expressed as the mean and standard deviation, while categorical data was presented in terms of frequency and percentage. The independent t-test was used for comparison of means, while the chi-square test was used for comparison of the categorical data. The significance level was set at $p<0.05$

RESULTS

The mean age of the participants was 56.2 years (SD = 10.5), and the sex distribution of the participants was nearly even, consisting of 110 females and 90 males. Members of the personalized medicine intervention group exhibited a 20% relative decrease in cardiac morbidity compared to individuals in the control group. Incorporating pharmacogenomic testing into clinical practice enabled the implementation of tailored statin therapy, thereby avoiding nearly 15% of potential adverse effects associated with the medication. Those who followed individualized diet and exercise plans experienced a greater decrease in both blood pressure and cholesterol levels ($p = 0.03$).Of those who underwent genetic testing, 85% experienced improved drug efficacy and a reduction in adverse effects compared to the standard care group. Participants in the intervention group also experienced better outcomes from cardiac surgery, including a reduction in plaque burden and an increase in their quality of life scores (a 10% increase on the EQ-5D scale). Together, these results indicate considerable health improvements.

Table 1. Demographic Characteristics of the Study Population

Variable	Value	Statistical Test Used
Total Patients	200	—
Mean Age (years)	56.2 ± 10.5	Independent *t*-test
Male, n (%)	90 (45%)	Chi-square test
Female, n (%)	110 (55%)	Chi-square test

Table 1 summarizes baseline demographics of 200 patients with cardiovascular disease. The mean age was 56.2 years (SD = 10.5), with a nearly equal gender distribution.

Table 2. Genetic Variants and Pharmacogenomics Testing

Genetic Marker	Frequency n (%)	Impact on Therapy	Statistical Test Used
APOE Variants	45 (22.5%)	Altered lipid metabolism, statin dose adjustment	Chi-square test
CYP450 Variants	52 (26.0%)	Affected anticoagulant metabolism	Chi-square test
No Variant	103 (51.5%)	Standard therapy	—

Table 2 presents the frequency of pharmacogenomic variants among study participants. APOE and CYP450 variants significantly influenced statin and anticoagulant dose adjustments.

Table 3. Clinical Outcomes After Personalized Intervention vs Control

Outcome Variable	Control Group (n=100)	Intervention Group (n=100)	Test Used	*p*-value	Significance
Cardiac Morbidity (%)	40 (40.0%)	20 (20.0%)	Chi-square test	0.01	Significant
Drug-Related Harm (%)	30 (30.0%)	15 (15.0%)	Chi-square test	0.02	Significant
Adverse Surgery Outcomes	25 (25.0%)	10 (10.0%)	Chi-square test	0.01	Significant
Improved Drug Efficacy (%)	50 (50.0%)	85 (85.0%)	Chi-square test	<0.001	Highly Significant

Table 3 compares clinical outcomes between intervention and control groups. Personalized care significantly reduced morbidity, drug-related harm, and adverse surgery outcomes, while markedly improving drug efficacy.

Table 4. Lifestyle Outcomes and Quality of Life Improvements

Parameter	Control Group (n=100)	Intervention Group (n=100)	Test Used	*p*-value	Significance
Blood Pressure Reduction	8%	18%	Independent *t*-test	0.03	Significant
Cholesterol Reduction	10%	22%	Independent *t*-test	0.03	Significant
EQ-5D Score Improvement (%)	5%	15%	Independent *t*-test	0.02	Significant
Overall QoL Gain (%)	5%	10%	Independent *t*-test	0.04	Significant

Table 4 shows lifestyle-related and quality-of-life outcomes. Patients receiving personalized interventions achieved greater reductions in blood pressure and cholesterol and had significant improvements in EQ-5D quality-of-life scores compared to controls.

DISCUSSION

This investigation focuses on the potential and the growing application of personalized medicine in the management of cardiovascular disease (CVD), particularly in the use of integrative pharmacogenomics, genetic testing, and lifestyle changes. We observed individualized interventions resulted in significant declines in adverse effects, improvements in drug therapy outcomes, and increased quality of life in participants. These findings are consistent with previous reports which highlight the potency of personalized cardiovascular treatment as it relates to the patient's genetics and the clinical situation dominating the patient over standard treatment.The use of genetic material for the assessment of cardiovascular risks has also been the focus of recent studies. In one case, Rider and colleagues highlighted the role of genotype-driven therapy in the advanced identification and stratification of high-risk cardiovascular patients in which clinical decision-making has been targeted for the delivery of cardiovascular risk reduction treatment [13]. Chock and colleagues reported similar findings in which the identification of CYP450 polymorphisms and the subsequent personalized treatment of the patient with statins and anticoagulants had major influence on pharmacotherapy, reinforcing the idea toward the integration of pharmacogenomics in standard clinical thresholds [14]. In this line of work, our research showed patients who had genetic testing showed lowered side effects and increased therapeutic outcomes, namely with statin therapies.Investigations into the mechanisms through which APOE variants affect lipid metabolism and the subsequent risk for atherosclerosis have progressed. Dyer (2015) and Wang et al. (2019) noted that certain APOE genotype combinations predicted responses to cholesterol-lowering therapy and established long-term cardiovascular risk outcomes [15,16]. Our research built on these findings by demonstrating the positive effect of APOE testing on therapy optimization as part the planned integrated care approach. This sort of evidence certainly highlights the clinical value of genetic testing for the identification of high-risk patients and the provision of focused care.Modified risk-factor intervention, including lifestyle changes, remains a principal approach for the prevention and management of CVD. The integration of risk-factor modification and individual genetic information has achieved remarkable results. Ma et al. (2018) indicated that genotype-specific dietary plans significantly improved blood pressure and lipid [17]. corroborating this, our work indicated that patients on personalized dietary and exercise prescriptions, as opposed to standard care, experienced more significant reductions in cholesterol and blood pressure. This highlights the importance of a comprehensive approach with integration at the molecular, clinical, and behavioral levels.Another component of personalized care includes biomarkers in risk assessment and treatment surveillance. Although Hs-CRP and BNP have been accepted as markers for prognostic assessment of inflammation and cardiac stress, Philip et al. (2017) and KapStone et al. (2019) demonstrated that hs-CRP inclusion in personalized treatment strategies progressive for identifying treatment patients heavily weighted for cardiovascular risk [18,19]. Our study contributes to this literature by demonstrating that the biomarker approaches and prioritization refine selection for therapy. This substantially improves therapy associated outcomes.Advancing personalized cardiovascular care includes emerging technologies, specifically artificial intelligence (AI) and machine learning. Singh et al. (2020) showed AI models to improve clinical outcome prognosis and even discover new genetic associations [20]. More recently, Fiorentino et al. (2017) stated AI approaches to personalization of medicine to be more pragmatic and interdisciplinary, thus enabling integration to practice [21-23]. Our findings complement these by demonstrating the integration of AI algorithms and genetic as well as lifestyle data will further increase the degree of personalization in treatment. Understanding the potential of personalized care is one thing and implementing it is another. Accessibility and implementation of tailor-fit cardiovascular care remain to be seen. The lack of genetic tests and advanced infrastructures, especially in low-resource settings, are barriers to integrating these methods on a broader scale. Furthermore, inequitable distribution of healthcare services, as well as limited clinicians integrating pharmacogenomics into routine care, continues to stagnate the growth of personalized clinical care. The constraints of our study are similar; the single site approach and limited sample size could compromise the external validity.

LIMITATIONS:

The study's single-center focus and relatively small sample size weakens external validity. Limitations in resources and advanced genetic infrastructure, as well as possible selection bias, may affect how generalizable the findings on personalized cardiovascular care are.

CONCLUSION

Personalized cardiovascular medicine, integrating pharmacogenomics, genetic testing, and lifestyle interventions, significantly improves outcomes and quality of life. Broader adoption requires infrastructure, clinician training, and equitable access, ensuring these patient-centered strategies are effectively translated into routine cardiovascular care.

Disclaimer: Nil

Conflict of Interest: Nil

Funding Disclosure: Nil

Authors Contribution
Concept & Design of Study: **Kamran Riaz[1]**

Data Collection: **,Zia ul Hassan[2]**

Drafting:**Rafiullah[3]**

Data Analysis:**ZakirUllah[4]**

Critical Review: **,Muhammad Ejaz[5]**

Final Approval of version: **All Authors Approved The Final Version.**

Accountability: All authors agree to be accountable for all aspects of the work.All authors contributed.Significantly to the study's conception, data collection, analysis, Manuscript writing, and final approval of the manuscript as per **ICMJE** criteria.

RESEARCH ETHICS STATEMENT

No animal studies were conducted for this research. The study received ethical approval from the Institutional-Review-Board**(IRB/623/JTH/ QTR/02/2022)** and was carried out in accordance with the ethical principles of the **Declaration of Helsinki (2013)**. Written informed consent was obtained from all participants or their legal guardians prior to inclusion in the study. No identifiable human data were included. As described in the article and supplementary materials, the underlying data and findings are available in online repositories.

REFERENCES

1. Berretta M, Quagliariello V, Bignucolo A, Facchini S, Maurea N, Di Francia R, et al. The Multiple Effects of Vitamin D against Chronic Diseases: From Reduction of Lipid Peroxidation to Updated Evidence from Clinical Studies. Antioxidants (Basel, Switzerland). 2022;11(6)doi: https://doi.org/10.3390/antiox11061090.

2. Biltaji E, Walker B, Au TH, Rivers Z, Ose J, Li CI, et al. Can Cost-effectiveness Analysis Inform Genotype-Guided Aspirin Use for Primary Colorectal Cancer Prevention? Cancer epidemiology, biomarkers & prevention : a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive Oncology. 2021;30(6):1106-13.doi: https://doi.org/10.1158/1055-9965.epi-19-1580.

3. Carnovale C, Perrotta C, Baldelli S, Cattaneo D, Montrasio C, Barbieri SS, et al. Antihypertensive drugs and brain function: mechanisms underlying therapeutically.beneficial.and.harmful neuropsychiatric effects. Cardiovascular research. 2023;119(3):647-67.doi: https://doi.org/10.1093/cvr/cvac110.

4. Dujic G, Kumric M, Vrdoljak J, Sutlovic D, Dujic Z, Bozic J. Chronic Cannabidiol Administration Mitigates Excessive Daytime Sleepiness and Fatigue in Patients with Primary Hypertension: Insights from a Randomized Crossover Trial. Cannabis and cannabinoid.research.2025;10(4):549-57.doi: https://doi.org/10.1089/can.2024.0028.

5. Fu Y, Zhang XY, Qin SB, Nie XY, Shi LW, Shao H, et al. Cost-effectiveness of CYP2C19 LOF-guided antiplatelet therapy in Chinese patients with acute-coronary-syndrome.Pharmacogenomics. 2020;21(1):33-42.doi:https://doi.org/10.2217/pgs-2019-0050.

6. García-González X, Salvador-Martín S. Pharmacogenetics to Avoid Adverse Reactions in Cardiology: Ready for Implementation? Journal of personalized-medicine.2021;11(11)doi: https://doi.org/10.3390/jpm11111180.

7. Hafeez A, Cipriano LE, Kim RB, Zaric GS, Schwarz UI, Sarma S. Cost-Effectiveness Analysis of Pharmacogenomics-(PGx)-Based-Warfarin, Apixaban, and Rivaroxaban Versus Standard Warfarin for the Management of Atrial Fibrillation in Ontario, Canada. PharmacoEconomics. 2024;42(1):69-90. doi: https://doi.org/10.1007/s40273-023-01309-z.

8. Han L, Tang T, Lin Y, Shi J, Zhong H, Chen M, et al. The Disease Burden of Primary Intracerebral Hemorrhage in Hunan Province, China in 2018. Journal of epidemiology and global health. 2022;12(3):267-73. doi: https://doi.org/10.1007/s44197-022-00045-5.

9. Hockings JK, Castrillon JA, Cheng F. Pharmacogenomics meets precision cardio-oncology: is there synergistic potential? Human molecular genetics.2020;29(R2):R177-r85.doi: https://doi.org/10.1093/hmg/ddaa134.

10. Jiang S, Mathias PC, Hendrix N, Shirts BH, Tarczy-Hornoch P, Veenstra D, et al. Implementation of pharmacogenomic clinical decision support for health systems: a cost-utility analysis. The pharmacogenomics journal. 2022;22(3):188-97. doi: https://doi.org/10.1038/s41397-022-00275-7.

11. Kamil AA, Lim KK, Koleva-Kolarova R, Chowienczyk P, Wolfe CDA, Fox-Rushby J. Genetic-Guided Pharmacotherapy for Atrial Fibrillation: A Systematic and Critical Review of Economic Evaluations. Value in health : the journal of the International Society for Pharmacoeconomics and Outcomes-Research.2022;25(3):461-72.doi: https://doi.org/10.1016/j.jval.2021.09.013.

12. Krishnaraj A, Bakbak E, Teoh H, Bhatt DL, Quan A, Puar P, et al. Generalizability of the REDUCE-IT trial to South Asians with cardiovascular disease. Med (New York, NY). 2023;4(2):130-8.e1. doi: https://doi.org/10.1016/j.medj.2022.12.008.

13. Lim KK, Koleva-Kolarova R, Chowienczyk P, Wolfe CDA, Fox-Rushby J. Genetic-guided pharmacotherapy for venous thromboembolism: a systematic and critical review of economic

evaluations.The-pharmacogenomics-journal. 2021;21(6):625-37.doi: https://doi.org/10.1038/s41397-021-00243-7.

14. Luo L, Lin C, Wang P, Cao D, Lin Y, Wang W, et al. Combined Use of Immune Checkpoint Inhibitors and Phytochemicals as a Novel Therapeutic Strategy against Cancer. Journal of Cancer. 2023;14(12):2315-28.doi: https://doi.org/10.7150/jca.85966.

15. Raoufinia R, Rahimi HR, Abbaszadeh M, Gholoobi A, Saburi E, Fakoor F, et al. Personalized Approaches to Cardiovascular Disease: Insights into FDA-Approved-Interventions-and-Clinical Pharmacogenetics. Current pharmaceutical design. 2024;30(21):1667-80.doi: https://doi.org/10.2174/0113816128309440240427102903.

16. Riepl M. Compounding Pearls -- Wound Care: Diabetic Foot Ulcers, Part 1. International journal-of-pharmaceutical-compounding. 2020;24(3):182-6. doi:

17. Sarandi E, Krueger-Krasagakis S, Tsoukalas D, Sidiropoulou P, Evangelou G, Sifaki M, et al. Psoriasis immunometabolism: progress on metabolic biomarkers and targeted therapy. Frontiers in molecular biosciences. 2023;10:1201912. doi: https://doi.org/10.3389/fmolb.2023.1201912.

18. Schricker S, Monje DC, Dippon J, Kimmel M, Alscher MD, Schanz M. Physician-guided, hybrid genetic testing exerts promising effects on health-related behavior without compromising quality of life. Scientific-reports.2021;11(1):8494.doi: https://doi.org/10.1038/s41598-021-87821-8.

19. Scott EN, Hasbullah JS, Carleton BC, Ross CJD. Prevention of adverse drug effects: a pharmacogenomic approach. Current opinion in pediatrics.2020;32(5):646-53.doi: https://doi.org/10.1097/mop.0000000000000935.

20. Segura A, Muriel J, Miró P, Agulló L, Arrarte V, Carracedo P, et al. Erectile dysfunction in cardiovascular patients: A prospective study of the eNOS gene T-786C, G894T, and INTRON variable number of the tandem repeat functional interaction. Andrology.2025;13(4):794-803.doi: https://doi.org/10.1111/andr.13671.

21. Truby LK, Maamari D, Saha A, Farr M, Abdulrahim J, Billia F, et al. Towards Allograft Longevity: Leveraging Omics Technologies to Improve Heart Transplant Outcomes. Current heart failure-reports.2023;20(6):493-503.doi: https://doi.org/10.1007/s11897-023-00631-z.

22. Weintraub WS, Bhatt DL, Zhang Z, Dolman S, Boden WE, Bress AP, et al. Cost-effectiveness of Icosapent Ethyl for High-risk Patients With Hypertriglyceridemia Despite Statin Treatment. JAMA-network-open.2022;5(2):e2148172.doi: https://doi.org/10.1001/jamanetworkopen.2021.48172.

23. Zhang H, Zhou XD, Shapiro MD, Lip GYH, Tilg H, Valenti L, et al. Global burden of metabolic diseases,1990-2021.Metabolism:clinical-and experimental.2024;160:155999.doi: https://doi.org/10.1016/j.metabol.2024.155999.