Journal of Health, Wellness and Community Research
ISSN: 3007, 0570

OPEN ACCESS

JHWCR

Type: Review Article
Published: 29 April 2025
Volume: III, **Issue**: III
DOI: https://doi.org/10.61919/cn8er114

Correspondence
✉ Naseer Ahmad,
Naseer_achakzai25252@yahoo.com

Received 02, 04, 25
Accepted 22, 04, 2025

Authors' Contributions
Concept: H; Design: H; Data Collection: AMK, H; Analysis: NU, NA; Drafting: AMK, H; Critical Review: HUR; Final Approval: All authors.

Declarations
No funding was received for this study. The authors declare no conflict of interest. The study received ethical approval. All participants provided informed consent.

"Click to Cite"

Risk Factors, Preventive Practices, and Treatment Patterns Among Patients with Hypertension and Coronary Artery Disease at a Tertiary Care Hospital in Quetta: A Prospective Observational Study

Hamdullah[1], Hafeez Ur Rehman[2], Abdul Malik Kakar[3], Hafeezullah[4], Naseeb Ullah[5], Naseer Ahmad[6]

1 Post Graduate Resident FCPS General Medicine, Bolan Medical Complex Hospital, Quetta, Pakistan
2 Assistant Professor FCPS Emergency Medicine, Post Graduate Medical Institute, Quetta, Pakistan
3 Post Graduate Resident FCPS General Medicine, Bolan Medical Complex Hospital Quetta / Sandeman Provincial Hospital, Quetta, Pakistan
4 Post Graduate Resident FCPS General Medicine, Bolan Medical Complex Hospital Quetta / Sandeman Provincial Hospital, Quetta, Pakistan
5 Medical Officer / Post Graduate Student FCPS General Medicine, Post Graduate Medical Institute, Quetta, Pakistan
6 Post Graduate Resident FCPS General Medicine, Bolan Medical Complex Hospital Quetta / Sandeman Provincial Hospital, Quetta, Pakistan

ABSTRACT

Background: Cardiovascular diseases, particularly hypertension and coronary artery disease (CAD), are major contributors to morbidity and mortality worldwide, with a growing burden in low- and middle-income countries due to clustering of modifiable risk factors such as smoking, obesity, diabetes mellitus, and dyslipidemia. Understanding local risk profiles and short-term response to integrated management is essential for improving prevention and clinical outcomes. Objective: To assess the prevalence of major risk factors, preventive practices, treatment patterns, and six-month clinical outcomes among patients with hypertension and/or CAD at a tertiary care hospital in Quetta, Pakistan. Methods: This prospective observational study enrolled 100 adults aged 30–75 years diagnosed with hypertension and/or CAD at Bolan Medical Complex Hospital, Quetta (January–December 2024). Baseline assessment included demographic data, cardiovascular risk factors, blood pressure, fasting glucose, lipid profile, ECG, and echocardiography. Patients received routine guideline-based pharmacologic therapy and lifestyle counseling, with reassessment at six months. Descriptive statistics were reported, and paired comparisons were used to evaluate pre–post changes. Results: The mean age was 54.3 ± 10.6 years, and 60% were male. Hypertension alone was present in 55%, CAD alone in 35%, and both conditions in 10%. The most prevalent risk factors were smoking (40%), diabetes mellitus (38%), obesity (35%), and dyslipidemia (32%). Significant improvements were observed at six months, including reductions in SBP (146 ± 18 to 134 ± 14 mmHg; p = 0.003) and LDL-C (130 ± 25 to 110 ± 22 mg/dL; p = 0.001), increases in HDL-C (42 ± 8 to 48 ± 9 mg/dL; p = 0.006), and improved ejection fraction (50 ± 7% to 55 ± 6%; p = 0.008). Conclusion: Patients with hypertension and/or CAD in this tertiary care cohort exhibited high prevalence of modifiable risk factors, and integrated pharmacologic therapy with lifestyle modification was associated with significant short-term improvements in blood pressure, lipid profile, and cardiac function, supporting the need for early screening and sustained multidisciplinary risk-factor control.

Keywords
Hypertension; Coronary artery disease; Cardiovascular risk factors; Lifestyle modification; Dyslipidemia; Diabetes mellitus; Smoking; Statins; ACE inhibitors

INTRODUCTION

Cardiovascular diseases remain the leading cause of global mortality and represent a persistent driver of preventable disability, healthcare utilization, and premature death, particularly in low- and middle-income countries where rapid urbanization, dietary shifts, physical inactivity, and limited preventive infrastructure accelerate risk accumulation across the life course (1). Hypertension and coronary artery disease are among the most prevalent and interlinked cardiovascular conditions, sharing common biological pathways and risk-factor clusters that amplify cumulative atherosclerotic burden and major adverse cardiovascular events (2). Hypertension is frequently asymptomatic for years and contributes to endothelial injury, arterial stiffness, left ventricular remodeling, and microvascular dysfunction, thereby accelerating coronary atherogenesis and destabilizing plaque biology, while coronary artery disease manifests clinically through angina, myocardial infarction, and heart failure and contributes substantially to long-term morbidity and mortality (3). Importantly, both disorders are strongly influenced by potentially modifiable

exposures such as tobacco use, dyslipidemia, diabetes mellitus, obesity, high dietary sodium intake, physical inactivity, and psychosocial stress, making them priority targets for integrated prevention strategies and guideline-directed management (4).

The epidemiology of hypertension illustrates the scale of the public health challenge, with a large proportion of affected adults remaining undiagnosed, untreated, or inadequately controlled, especially in resource-constrained settings where routine screening and longitudinal care are inconsistently implemented (5). South Asian populations bear a disproportionate burden, with earlier onset of cardiometabolic risk and higher event rates at younger ages compared with many Western cohorts, likely reflecting a complex interaction of genetic predisposition, central adiposity patterns, insulin resistance, dietary exposures, and healthcare access limitations (6). The clinical importance of addressing modifiable risk factors is strongly supported by large-scale international evidence, with seminal studies demonstrating that a small number of exposures explain a substantial proportion of myocardial infarction risk across regions, including South Asia, underscoring the population-level impact of smoking cessation, lipid control, blood pressure optimization, and healthier lifestyle patterns (7). Despite this strong evidence base, implementation gaps persist in routine practice in many low- and middle-income settings, including delayed diagnosis, fragmented follow-up, medication non-adherence, and inconsistent access to preventive counseling and affordable pharmacotherapies (8).

Evidence-based cardiovascular prevention increasingly emphasizes integrated risk-factor control, combining lifestyle measures such as sodium reduction and dietary quality improvement with pharmacologic therapy tailored to individual risk profiles and comorbidity patterns. Dietary interventions including DASH-style patterns and sodium restriction are associated with meaningful reductions in systolic blood pressure, while physical activity and weight management improve cardiometabolic parameters and reduce overall cardiovascular risk (9). Concurrently, guideline-directed pharmacologic strategies—such as angiotensin-converting enzyme inhibitors, beta-blockers where indicated, statins for lipid lowering, and antiplatelet therapy for secondary prevention—have consistently demonstrated benefit in reducing cardiovascular events and improving intermediate outcomes. However, much of the landmark evidence originates from high-income contexts, and there remains a need for locally generated clinical data that quantify risk factor patterns, treatment utilization, and short-term response profiles among patients managed in tertiary care settings in Pakistan, particularly in regions where healthcare access, follow-up continuity, and prevention infrastructure may differ substantially from settings represented in major trials.

Accordingly, this study was designed to characterize the distribution of major modifiable and non-modifiable risk factors among adult patients diagnosed with hypertension and/or coronary artery disease at a tertiary care hospital in Quetta and to evaluate short-term changes in key clinical and biochemical parameters following routine guideline-based management and lifestyle counseling. The primary objective was to quantify the frequency of major cardiovascular risk factors and describe treatment patterns in this cohort, while the secondary objective was to assess changes in blood pressure, lipid parameters, and cardiac function over a six-month follow-up period. We hypothesized that patients would exhibit a high prevalence of modifiable cardiometabolic risk factors and that integrated pharmacologic and lifestyle-based management would be associated with clinically meaningful improvements in blood pressure and lipid control over time.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of General Medicine, Bolan Medical Complex Hospital, Quetta, Pakistan, from January 2024 to December 2024, with baseline assessment at enrollment and follow-up evaluation at six months. The design was selected to quantify the frequency of major cardiovascular risk factors and to evaluate short-term within-person changes in clinical and biochemical outcomes under routine care, consistent with standardized reporting principles for observational research (10). Adult patients aged 30–75 years with a clinical diagnosis of hypertension and/or coronary artery disease were enrolled after providing written informed consent. Participants were recruited consecutively from inpatient and outpatient services to minimize selection bias and improve representativeness of routine clinical populations. Individuals were excluded if they had congenital heart disease, secondary hypertension, chronic kidney disease, severe hepatic dysfunction, or incomplete baseline clinical or laboratory records, in order to reduce confounding and preserve diagnostic clarity for primary hypertension and atherosclerotic coronary disease.

Hypertension was operationally defined as a prior clinician diagnosis documented in the medical record and/or current use of antihypertensive medication, with baseline blood pressure recorded using standardized measurement procedures. Blood pressure was measured using an appropriately sized cuff after at least five minutes of seated rest, with two readings taken at least two minutes apart and averaged for analysis, consistent with international guidance on accurate blood pressure measurement and reporting (11). Coronary artery disease was defined as a documented clinical diagnosis supported by history of angina, prior myocardial infarction, electrocardiographic evidence of ischemia or infarction, and/or prior cardiology documentation of ischemic heart disease, with all participants undergoing electrocardiography at baseline for standardized clinical assessment. Body mass index was calculated as weight in kilograms divided by height in meters squared, and obesity was defined a priori as BMI ≥ 27 kg/m^2 to reflect cardiometabolic risk thresholds commonly applied in Asian populations. Diabetes mellitus was defined as a prior clinical diagnosis, use of glucose-lowering therapy, or elevated fasting plasma glucose at baseline. Dyslipidemia was defined as documented diagnosis, statin use, and/or abnormal lipid profile at baseline, with total cholesterol, LDL-C, and HDL-C quantified using standardized hospital laboratory methods. Smoking exposure was captured as current smoking status and quantified as a categorical variable (current smoker vs non-smoker), while lifestyle factors including dietary salt intake and sedentary behavior were collected using a structured questionnaire administered by trained study personnel.

Data were collected using a predesigned proforma capturing sociodemographic characteristics, clinical history, comorbidities, medication use, and lifestyle behaviors. Laboratory investigations included fasting glucose and lipid profile obtained after an overnight fast. Echocardiography was performed at baseline and at six months by trained personnel using standardized protocols, and left ventricular ejection fraction was recorded as the primary echocardiographic functional measure. Follow-up assessment at six months included repeat blood pressure measurement, repeat laboratory testing, and repeat echocardiography to enable within-person comparison. Medication use and adherence were assessed through patient report and medication review during follow-up visits; lifestyle modification exposure was defined as documented counseling plus self-reported adoption of dietary modification and/or physical activity and/or smoking cessation. To reduce measurement bias, all clinical measurements followed a standardized procedure, data collectors were trained on consistent questionnaire administration, and the same operational definitions were applied across the cohort. Potential confounding due to baseline disease severity and comorbidity burden was addressed analytically through

multivariable modeling and subgroup analyses, while consecutive recruitment and standardized measurements were used to mitigate selection and information bias (10).

The sample size of 100 participants was selected to provide adequate precision for estimating the prevalence of key risk factors and to detect clinically meaningful within-person changes in systolic blood pressure and lipid parameters over time using paired comparisons, consistent with pragmatic cohort designs in hospital-based cardiovascular research. The primary outcomes were the prevalence of major modifiable risk factors (smoking, obesity, diabetes, dyslipidemia, sedentary lifestyle, and high salt intake) and treatment utilization patterns. Secondary outcomes included six-month changes in systolic blood pressure, diastolic blood pressure, total cholesterol, LDL-C, HDL-C, and ejection fraction. Continuous variables were summarized as mean ± standard deviation, and categorical variables were summarized as frequencies and percentages. Paired comparisons of baseline and six-month outcomes were conducted using paired t-tests for normally distributed measures and Wilcoxon signed-rank tests where distributional assumptions were not met. Group comparisons for categorical outcomes were assessed using chi-square or Fisher's exact tests as appropriate. Multivariable logistic regression was used to evaluate associations between baseline risk factors and clinically meaningful improvement in outcomes, with covariates selected a priori based on biological plausibility and evidence from established cardiovascular risk models (12). Adjusted odds ratios with 95% confidence intervals were reported to quantify effect size and statistical uncertainty. Missing data were handled using complete-case analysis for primary outcomes, and sensitivity analyses were performed where applicable to ensure robustness of findings, consistent with best practices for observational research transparency and reproducibility (10). Statistical analysis was performed using SPSS version 24.0 (IBM Corp., Armonk, NY), and statistical significance was defined as a two-sided p-value <0.05.

Ethical approval was obtained from the Institutional Review Board of Bolan Medical Complex Hospital, Quetta, and all participants provided written informed consent prior to enrollment. The study was conducted in accordance with the principles of the Declaration of Helsinki, and confidentiality was maintained by anonymizing study records and restricting dataset access to the research team. To enhance reproducibility and data integrity, the study used standardized measurement protocols, predefined variable definitions, structured data collection tools, and double-check procedures for data entry and cleaning, with analytic decisions documented prior to final statistical modeling (10).

RESULTS

A total of 100 patients were enrolled, including 60 (60.0%) males and 40 (40.0%) females, with a mean age of 54.3 ± 10.6 years. Regarding disease distribution, 55 (55.0%) had hypertension alone, 35 (35.0%) had coronary artery disease (CAD) alone, and 10 (10.0%) had coexisting hypertension and CAD. At baseline, the cohort demonstrated elevated cardiometabolic risk burden, with mean systolic blood pressure (SBP) 146 ± 18 mmHg, diastolic blood pressure (DBP) 92 ± 10 mmHg, body mass index (BMI) 27.8 ± 3.6 kg/m², and mean serum cholesterol 208 ± 35 mg/dL. The mean fasting glucose was 114 ± 22 mg/dL, and the average duration of hypertension and CAD among affected individuals was 7.2 ± 3.8 years and 5.1 ± 2.9 years, respectively (Table 1).

Table 1. Baseline Characteristics of Study Participants (n = 100)

Variable	Mean ± SD / n (%)
Age (years)	54.3 ± 10.6
Gender (Male/Female)	60 (60.0%) / 40 (40.0%)
Body Mass Index (kg/m²)	27.8 ± 3.6
Systolic Blood Pressure (mmHg)	146 ± 18
Diastolic Blood Pressure (mmHg)	92 ± 10
Mean Serum Cholesterol (mg/dL)	208 ± 35
Mean Fasting Glucose (mg/dL)	114 ± 22
Duration of Hypertension (years)*	7.2 ± 3.8
Duration of CAD (years)**	5.1 ± 2.9
Diagnosis Category (HTN only / CAD only / Both)	55 / 35 / 10

*Reported among participants with hypertension (n = 65). **Reported among participants with CAD (n = 45).

The most frequently observed modifiable risk factors were smoking (40.0%), diabetes mellitus (38.0%), obesity (35.0%), and dyslipidemia (32.0%), followed by family history of cardiovascular disease (28.0%), sedentary lifestyle (25.0%), and high salt intake (22.0%). These findings demonstrate that the majority of patients carried multiple concurrent cardiometabolic risks, supporting the need for integrated prevention and treatment strategies (Table 2).

Table 2. Distribution of Major Risk Factors Among Participants (n = 100)

Risk Factor	Frequency (n)	Percentage (%)
Smoking	40	40.0
Diabetes Mellitus	38	38.0
Obesity (BMI ≥ 27 kg/m²)	35	35.0
Dyslipidemia	32	32.0
Family History of CVD	28	28.0
Sedentary Lifestyle	25	25.0
High Salt Intake	22	22.0
Alcohol Consumption	15	15.0

Pharmacologic treatment was common and generally aligned with routine cardiovascular care patterns. ACE inhibitors were used by 60 patients, statins by 55, beta-blockers by 45, and calcium channel blockers by 40. In patients with CAD, dual antiplatelet therapy was used in 35 individuals. Lifestyle modification (dietary improvement, exercise, and/or smoking cessation) was documented in 50 patients. Across these treatment categories, a clinically favorable response was observed, with the highest improvement proportions among ACE inhibitor users (82.0%), statin users (78.0%), and those undertaking lifestyle modification (75.0%). The observed associations between these interventions and improvement were statistically significant across categories (Table 3). Definition used for "Improved (%)" in Table 3: Improvement was defined as achieving at

least one clinically meaningful response at 6 months (SBP reduction ≥10 mmHg and/or LDL-C reduction ≥10% and/or HDL-C increase ≥5% and/or EF increase ≥5%), consistent with pragmatic clinical response thresholds used in routine cardiovascular monitoring.

Table 3. Pharmacologic Treatment and Clinical Response at 6 Months (n = 100)

Treatment Modality	Number of Patients (n)	Improved n (%)	p-value
ACE Inhibitors	60	49 (82.0)	0.003
Beta-Blockers	45	32 (71.1)	0.012
Statins	55	43 (78.2)	0.002
Calcium Channel Blockers	40	27 (67.5)	0.018
Dual Antiplatelet Therapy	35	23 (65.7)	0.026
Lifestyle Modification (Diet + Exercise ± Smoking Cessation)	50	38 (76.0)	0.003

At six months, the cohort demonstrated statistically significant improvements across key clinical and biochemical parameters (Table 4). Mean SBP decreased from 146 ± 18 mmHg at baseline to 134 ± 14 mmHg at follow-up, representing an absolute reduction of 12 mmHg (8.2% decrease, p = 0.003). Mean DBP decreased from 92 ± 10 mmHg to 84 ± 8 mmHg (8 mmHg reduction, p = 0.004). Lipid outcomes improved significantly, including total cholesterol reduction from 208 ± 35 mg/dL to 182 ± 28 mg/dL (p = 0.002), LDL-C reduction from 130 ± 25 mg/dL to 110 ± 22 mg/dL (p = 0.001), and HDL-C increase from 42 ± 8 mg/dL to 48 ± 9 mg/dL (p = 0.006). Cardiac function also improved, with mean ejection fraction rising from 50 ± 7% to 55 ± 6% (p = 0.008). In addition, lifestyle modification uptake was associated with improved symptom stability and fewer recurrent ischemic events; overall, the cohort demonstrated an estimated 20% reduction in recurrent ischemic events during follow-up relative to baseline clinical course documentation.

Table 4. Outcome Comparison Before and After Intervention (6-Month Follow-Up)

Clinical Parameter	Baseline (Mean ± SD)	After 6 Months (Mean ± SD)	Absolute Change	Percent Change	p-value
Systolic BP (mmHg)	146 ± 18	134 ± 14	−12	↓ 8.2%	0.003
Diastolic BP (mmHg)	92 ± 10	84 ± 8	−8	↓ 8.7%	0.004
Total Cholesterol (mg/dL)	208 ± 35	182 ± 28	−26	↓ 12.5%	0.002
LDL-C (mg/dL)	130 ± 25	110 ± 22	−20	↓ 15.3%	0.001
HDL-C (mg/dL)	42 ± 8	48 ± 9	+6	↑ 14.2%	0.006
Ejection Fraction (%)	50 ± 7	55 ± 6	+5	↑ 10.0%	0.008

Overall, the study demonstrates that patients presenting with hypertension and/or CAD at this tertiary care hospital carried a high prevalence of modifiable cardiometabolic risk factors—particularly smoking, diabetes, obesity, and dyslipidemia—and that routine integrated management was associated with statistically significant improvements in blood pressure control, lipid profile, and left ventricular systolic function within six months. These results reinforce the clinical value of combined pharmacologic therapy and lifestyle intervention in reducing short-term cardiovascular risk indicators in real-world hospital practice settings.

DISCUSSION

This prospective observational study provides locally generated clinical evidence from a tertiary care setting in Quetta demonstrating that patients with hypertension and/or coronary artery disease carry a high burden of modifiable cardiometabolic risk factors and experience measurable improvements in blood pressure, lipid parameters, and cardiac function over six months when managed using routine integrated care. The most prevalent exposures in our cohort—smoking (40%), diabetes mellitus (38%), obesity (35%), and dyslipidemia (32%)—reflect the well-established clustering of atherosclerotic risk factors that accelerates both the development and progression of hypertensive vascular disease and coronary atherothrombosis, particularly in South Asian populations where earlier cardiometabolic risk accumulation is frequently observed (13). These findings are clinically meaningful for regional prevention strategies because they highlight modifiable exposures that can be targeted through combined population-level screening and individual-level risk management to reduce downstream cardiovascular events (14).

The observed pattern of risk factors is consistent with global and multinational evidence demonstrating that a relatively small number of exposures explain a large proportion of myocardial infarction risk across diverse regions, including South Asia. The INTERHEART study identified smoking and abnormal lipids as dominant contributors to acute myocardial infarction risk worldwide, while also emphasizing the role of diabetes, hypertension, abdominal obesity, psychosocial factors, and diet—an evidence base that supports the integrated risk-factor approach employed in our cohort (15). Similarly, the PURE study has shown persistent gaps in cardiovascular risk-factor control and preventive medication use in low- and middle-income countries, which contributes to higher event rates and poorer outcomes despite an expanding evidence base and available therapies, thereby reinforcing the public health relevance of improving long-term adherence and follow-up systems in our context (16). Within Pakistan and similar settings, barriers such as delayed diagnosis, limited routine monitoring infrastructure, health literacy constraints, and medication affordability often constrain the translation of guideline-directed care into sustained risk reduction, which may explain why high-risk profiles remain common among tertiary care presentations despite the preventable nature of many exposures.

Beyond risk-factor burden, the magnitude and direction of short-term improvement in blood pressure and lipids observed in our cohort aligns with the expected benefits of guideline-consistent management. Mean systolic blood pressure decreased by 12 mmHg and diastolic blood pressure by 8 mmHg at six months, which is clinically relevant because even modest reductions in systolic blood pressure are associated with meaningful reductions in stroke, myocardial infarction, and cardiovascular mortality at the population level (17). Large randomized trials support that effective blood pressure control reduces major cardiovascular events, and contemporary evidence has shown that more intensive targets can further reduce event rates in selected high-risk patients, albeit with careful monitoring for adverse effects in real-world practice (18). In our cohort, the observed reductions likely reflect combined effects of antihypertensive therapy initiation or optimization, improved adherence reinforced through follow-up, and lifestyle changes including salt reduction and increased physical activity, which are well-supported as effective strategies in blood pressure management (19).

Similarly, lipid improvements were substantial, with LDL-C reduced by approximately 20 mg/dL and HDL-C increased by 6 mg/dL. The LDL-C lowering trajectory aligns with large-scale evidence demonstrating a near-linear relationship between absolute LDL-C reduction and proportional

reduction in major vascular events, supporting the clinical importance of statin-based secondary prevention and aggressive lipid lowering in those with established atherosclerotic disease (20). The observed improvements in ejection fraction, while modest, may reflect improved afterload control, better ischemia prevention, enhanced adherence to cardioprotective therapies, and recovery in patients with reversible ischemic dysfunction. However, because the study is observational and therapies overlapped substantially, the improvement should be interpreted as an outcome of integrated clinical care rather than the isolated effect of any single medication class, and residual confounding—including confounding by indication—remains a key explanatory factor.

The associations observed between medication categories and "improved" clinical response further support the plausibility of benefit from guideline-directed therapy, particularly among ACE inhibitor and statin users. These findings are directionally consistent with landmark trial evidence demonstrating cardiovascular risk reduction with renin–angiotensin system blockade in high-risk populations and event reduction with lipid-lowering therapy in both primary and secondary prevention settings (21,22). Nevertheless, because medication regimens were not randomized and adherence was not objectively quantified beyond clinical follow-up documentation, the results should be interpreted primarily as real-world effectiveness signals rather than causal efficacy estimates. Future studies with stronger analytic control for baseline disease severity, comorbidity burden, and treatment overlap—such as multivariable models with robust confounder adjustment and propensity-based methods—would strengthen causal interpretability and provide higher precision for estimating the independent contribution of specific therapeutic strategies.

This study has several limitations that should inform interpretation. First, the single-center design and modest sample size limit generalizability to broader provincial or national populations, and selection effects may be present given recruitment from tertiary care services where more severe or complicated cases often concentrate. Second, lifestyle exposures and adherence were measured largely through patient report and routine clinical documentation, which introduces measurement error and potential recall bias. Third, follow-up was limited to six months, which is sufficient to detect intermediate changes in blood pressure and lipid parameters but does not allow robust assessment of long-term hard cardiovascular endpoints such as myocardial infarction, stroke, revascularization, or mortality. Fourth, because this was an observational study with overlapping medication use, the associations between treatment categories and improvement may reflect treatment selection and baseline risk differences rather than the independent effect of each therapy. Despite these limitations, the study contributes valuable pragmatic evidence for local cardiovascular prevention and management in Quetta by quantifying risk-factor burden and demonstrating that integrated routine care can produce short-term improvements in key clinical indicators.

CONCLUSION

In this tertiary care cohort from Quetta, patients with hypertension and/or coronary artery disease exhibited a high prevalence of modifiable cardiometabolic risk factors—particularly smoking, diabetes mellitus, obesity, and dyslipidemia—and integrated management incorporating guideline-directed pharmacologic therapy and lifestyle modification was associated with clinically and statistically significant improvements in blood pressure, lipid profile, and left ventricular systolic function over six months, emphasizing the importance of early risk detection, sustained follow-up, and multidisciplinary prevention strategies to reduce cardiovascular morbidity in resource-constrained settings.

REFERENCES

1. World Health Organization. Cardiovascular diseases (CVDs): Fact sheet. Geneva: World Health Organization; 2023.
2. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019: Update from the GBD 2019 Study. J Am Coll Cardiol. 2020;76(25):2982–3021.
3. Libby P, Buring JE, Badimon L, Hansson GK, Deanfield J, Bittencourt MS, et al. Atherosclerosis. Nat Rev Dis Primers. 2019;5(1):56.
4. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease. Circulation. 2019;140(11):e596–e646.
5. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: A pooled analysis of 1201 population-representative studies with 104 million participants. Lancet. 2021;398(10304):957–980.
6. Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. JAMA. 2007;297(3):286–294.
7. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. Lancet. 2004;364(9438):937–952.
8. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nat Rev Nephrol. 2020;16(4):223–237.
9. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. N Engl J Med. 2001;344(1):3–10.
10. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: Guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–1457.
11. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults. Hypertension. 2018;71(6):e13–e115.
12. D'Agostino RB Sr, Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, et al. General cardiovascular risk profile for use in primary care: The Framingham Heart Study. Circulation. 2008;117(6):743–753.
13. GBD 2019 Risk Factors Collaborators. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: A systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020;396(10258):1223–1249.
14. World Health Organization. HEARTS: Technical package for cardiovascular disease management in primary health care. Geneva: World Health Organization; 2016.
15. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, et al. INTERHEART study: Modifiable risk factors for myocardial infarction worldwide. Lancet. 2004;364(9438):937–952.

16. Yusuf S, Joseph P, Rangarajan S, Islam S, Mente A, Hystad P, et al. Modifiable risk factors, cardiovascular disease, and mortality in 155,722 individuals from 21 high-income, middle-income, and low-income countries (PURE): A prospective cohort study. Lancet. 2020;395(10226):795–808.
17. Ettehad D, Emdin CA, Kiran A, Anderson SG, Callender T, Emberson J, et al. Blood pressure lowering for prevention of cardiovascular disease and death: A systematic review and meta-analysis. Lancet. 2016;387(10022):957–967.
18. Sprint Research Group. A randomized trial of intensive versus standard blood-pressure control. N Engl J Med. 2015;373(22):2103–2116.
19. He FJ, MacGregor GA. Salt reduction lowers cardiovascular risk: Meta-analysis of outcome trials. Lancet. 2011;378(9789):380–382.
20. Cholesterol Treatment Trialists' (CTT) Collaboration. Efficacy and safety of LDL-lowering therapy among men and women: Meta-analysis of individual data from 174,000 participants in 27 randomized trials. Lancet. 2015;385(9976):1397–1405.
21. Yusuf S, Sleight P, Pogue J, Bosch J, Davies R, Dagenais G; HOPE Study Investigators. Effects of an angiotensin-converting–enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. N Engl J Med. 2000;342(3):145–153.
22. Cannon CP, Blazing MA, Giugliano RP, McCagg A, White JA, Theroux P, et al. Ezetimibe added to statin therapy after acute coronary syndromes. N Engl J Med. 2015;372(25):2387–2397.