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Preliminary Application of Antihypertensive Gene Detection in the Treatment of Hypertension

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Statistical Analysis C
Data Interpretation D
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Background: Hypertension management is often suboptimal due to interindividual variability in drug response. Pharmacogenomics testing may guide personalized therapy by identifying genetic polymorphisms affecting drug efficacy and safety. This study aimed to evaluate whether pharmacogenomics-guided therapy improves blood pressure control and treatment compliance and reduces rehospitalization in patients with hypertension.

Material/Methods: In this prospective randomized controlled trial, 900 patients with hypertension were assigned to a control group (CG, n=450) receiving conventional care or a study group (SG, n=450) receiving pharmacogenomic-guided therapy. Six gene loci (CYP2D6, CYP2C9, AGTR1, ACE, NPPA, CYP3A5) related to 5 antihypertensive drug classes were tested. Outcomes included blood pressure control rate, compliance, adverse reactions, drug adjustments, and 6-month readmission rate.

Results: The SG achieved significantly higher blood pressure control rates at 3 months (80.7% vs 70.2%, $P<0.001$) and 6 months (78.4% vs 65.1%, $P<0.001$) compared with the CG. Treatment compliance was also higher in the SG (98.0% vs 74.0%, $P<0.001$). angiotensin-converting enzyme inhibitor–related dry cough incidence was lower in the SG (1.6% vs 8.3%, $P<0.001$). The SG required fewer drug adjustments during hospitalization and had a lower 6-month readmission rate (2.4% vs 8.9%, $P<0.001$).

Conclusions: Pharmacogenomic-guided individualized therapy improves blood pressure control, enhances treatment adherence, reduces specific adverse reactions, and decreases rehospitalization. These findings support integrating pharmacogenomic testing into personalized hypertension management.

Keywords: Antihypertensive Agents • Cardio-Renal Syndrome • Hypertension • Polymorphism, Single Nucleotide • Randomized Controlled Trial • src Homology Domains

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Introduction

Hypertension is a common and frequently occurring disease with complex and diverse pathogenic factors, representing a leading global cause of cardiovascular morbidity and mortality [1]. There are many types of clinical medications for treating hypertension, and the sensitivity and metabolic conditions of different drugs also vary significantly [2]. Studies have pointed out that poor control of hypertension is not only related to poor medication compliance and unhealthy lifestyle, but also the lack of individualized medication is a major influencing factor [3]. This "trial-and-error" approach often leads to prolonged periods of uncontrolled blood pressure, suboptimal treatment outcomes, and increased risk of complications in many patients.

Individualized medication is rather difficult, while the development of pharmacogenomics provides a new idea for personalized medication. It focuses on genetic polymorphism, the diversity of drug effects, and their relationships, thereby providing references for clinical rational drug use, personalized drug use, and evaluation of adverse reactions. Single nucleotide polymorphism refers to the DNA sequence differences caused by single nucleotide variations in the genome [4]. It is a key factor influencing the individual differences in the efficacy and safety of antihypertensive drugs. Functional single nucleotide polymorphisms in genes encoding drug-metabolizing enzymes (eg, CYP2C9, CYP2D6), drug transporters, or therapeutic targets (eg, AGTR1, ACE) can significantly alter pharmacokinetics and pharmacodynamics, leading to varied therapeutic responses and adverse event risks among different patients prescribed the same drug [5-7]. Accumulating evidence has established associations between specific genetic polymorphisms and antihypertensive drug outcomes. For instance, CYP2D6 phenotypes influence β-blocker metabolism, while ACE I/D polymorphism is linked to ACE inhibitor efficacy [8,9].

However, much of the existing literature, including studies on specific situations such as preeclampsia, focuses on single gene-drug interactions in select populations or settings [5]. A significant knowledge gap remains regarding the integrated clinical utility of a multi-gene pharmacogenomic panel in guiding initial antihypertensive drug selection and dosing for a broad, general hypertensive population within real-world clinical practice. Furthermore, robust evidence from large-scale, randomized controlled trials evaluating hard clinical endpoints, such as sustained blood pressure control and hospitalization, is still limited.

Therefore, in this randomized controlled trial, we aimed to evaluate whether an individualized medication strategy guided by a 6-gene pharmacogenomic panel could improve clinical outcomes compared with conventional therapy in patients with primary hypertension. We hypothesized that pharmacogenomic-guided therapy would lead to superior blood pressure control, higher treatment adherence, fewer adverse drug reactions, and reduced rehospitalization rates. It is noteworthy that the prevalence of these polymorphisms varies across ethnicities and geographic regions. For instance, the CYP2D6*10 reduced-function allele is highly prevalent in East Asian populations (approximately 50%), whereas the CYP2C9*3 allele frequency is lower in Asians than in Whites [10,11]. Therefore, the clinical utility and cost-effectiveness of such a panel can differ depending on the genetic background of the target population. The present study was conducted in a southern Chinese cohort, and the prevalence estimates for the selected loci in this population are provided in **Table 1**.

Material and Methods

Study Design and Setting

This was a prospective, randomized, open-label, controlled trial conducted at the Department of Cardiology, the Second People's Hospital of Foshan, China. The study aimed to compare pharmacogenomic-guided therapy and conventional therapy in patients with primary hypertension. All outcomes were assessed prospectively according to a predefined schedule.

Participant Enrollment and Criteria

A total of 900 patients with hypertension who visited our hospital from January 2022 to September 2022 were divided into a control group (CG, n=450) or study group (SG, n=450) according to random number table method. The 2 groups were well-balanced at baseline with respect to age, sex, duration of hypertension, baseline blood pressure, and key comorbidities (all $P>0.05$; see **Table 2** for detailed characteristics).

The inclusion criteria were as follows. (1) Diagnosis of primary hypertension was established according to the 2020 International Society of Hypertension Global Hypertension Practice Guidelines. Specifically, patients had a seated, office systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, confirmed by 2 separate measurements on 2 different occasions prior to enrollment, in the absence of antihypertensive medication. For patients already on antihypertensive therapy at referral, the diagnosis was based on documented history and medical records, and their baseline blood pressure was measured while continuing their pre-existing regimen prior to randomization and any study-related intervention. Additional criteria were (2) no vital organ failure and (3) that patients were informed about the details of the research. The exclusion criteria were as follows: (1) serious target organ damage; (2) secondary, malignant, or progressive

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Table 1. Genotype and allele frequencies of the 6 pharmacogenomic loci in the study group (n=450).

Gene (polymorphism)	Genotype	Number of patients (n)	Genotype frequency (%)	Allele	Allele frequency (%)	Notes/ phenotype implication
CYP2D6 (*10)	*10/*10	120	26.7%	*10	51.1%	Reduced metabolizer
	*1/*10	180	40.0%	*1	48.9%	
	*1/*1	150	33.3%			Normal metabolizer
CYP2C9 (*3)	*1/*1	380	84.4%	*1	91.8%	Normal metabolizer
	*1/*3	65	14.4%	*3	8.2%	Reduced metabolizer
	*3/*3	5	1.1%			Poor metabolizer
AGTR1 (A1166C)	AA	260	57.8%	A	75.6%	Reference allele
	AC	160	35.6%	C	24.4%	Associated with drug response
	CC	30	6.7%			
ACE (I/D)	II	110	24.4%	I	48.9%	Lower ACE activity
	ID	220	48.9%	D	51.1%	Higher ACE activity
	DD	120	26.7%			
NPPA (T2238C)	TT	300	66.7%	T	81.1%	Reference allele
	TC	125	27.8%	C	18.9%	Linked to diuretic response
	CC	25	5.6%			
CYP3A5 (*3)	*1/*1	70	15.6%	*1	34.4%	Normal metabolizer
	*1/*3	200	44.4%	*3	65.6%	Reduced metabolizer
	*3/*3	180	40.0%			Poor metabolizer

Genotype and allele distribution of the 6 pharmacogenomic variants in the study group (n=450). Frequencies are expressed as percentages.

hypertension; (3) other serious comorbid diseases; (4) mental disorders resulting in inability to communicate normally; and (5) incomplete clinical data essential for analysis. This was pragmatically defined as missing any 1 of the following key datasets: baseline demographic or vital sign records; results from the allocated pharmacogenomic test (for the SG) or the standard laboratory panel (for the CG); discharge summary including final medication plan; and at least 1 follow-up blood pressure measurement and adherence assessment at either the 3-month or 6-month time point.

The study protocol was reviewed and approved by the Institutional Review Board (Ethics Committee) of the Second People's Hospital of Foshan (approval No. 060-01-(2022)-0069; date of approval: June 23, 2022). The approval explicitly covered all procedures, including pharmacogenomic testing, genetic data analysis, and the use of results to guide clinical management. Written informed consent was obtained from all participants after a detailed explanation of the study, with a specific section dedicated to the nature, purpose, potential benefits, risks, and confidentiality safeguards related to genetic testing. The study was conducted in full compliance with the principles of the Declaration of Helsinki and relevant Chinese national regulations on clinical research and human genetic resources.

The required sample size was estimated based on the primary outcome: the proportion of patients achieving blood pressure control at 6 months. We assumed a baseline control rate of 65% in the CG, based on local historical data, and hypothesized an absolute improvement of 15 percentage points in the SG with pharmacogenomic guidance, corresponding to a control rate of 80%. With a 2-sided alpha of 0.05 and 80% power, the calculated sample size was 392 patients per group (784 total)

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Table 2. Detailed baseline characteristics of the study participants.

Characteristic	Study group (n=450)	Control group (n=450)	*P* value
Demographics			
Age, years, mean±SD	59.64±3.75	59.62±3.73	0.942
Male, n (%)	280 (62.2)	270 (60.0)	0.521
Body mass index, kg/m^2, mean±SD	26.1±3.2	25.9±3.4	0.401
Clinical history			
Duration of hypertension, years, mean±SD	17.69±2.53	17.67±2.51	0.911
Family history of hypertension, n (%)	278 (61.8)	265 (58.9)	0.376
Baseline blood pressure			
Systolic blood pressure, mmHg, mean±SD	152.3±10.4	151.8±10.1	0.480
Diastolic blood pressure, mmHg, mean±SD	94.2±6.8	93.9±6.6	0.531
Comorbidities, n (%)			
Diabetes mellitus	98 (21.8)	102 (22.7)	0.752
Dyslipidemia	156 (34.7)	148 (32.9)	0.584
Coronary artery disease	67 (14.9)	72 (16.0)	0.635
Chronic kidney disease (stage 1-3)	45 (10.0)	48 (10.7)	0.735
Previous antihypertensive use, n (%)*	392 (87.1)	385 (85.6)	0.502
Smoking status, n (%)			
Current smoker	105 (23.3)	98 (21.8)	0.581
Former smoker	120 (26.7)	115 (25.6)	0.719
Never smoked	225 (50.0)	237 (52.7)	0.431

Previous use refers to patients who were on at least 1 antihypertensive medication prior to enrollment. Continuous variables compared using the independent *t* test; categorical variables using the chi-square test.

using the chi-square test for 2 proportions. To account for potential loss to follow-up (estimated at 10%) and to ensure adequate representation of key polymorphisms (eg, CYP2D6*10 prevalence of approximately 50% in our population), we increased the sample size to 450 patients per group (900 total).

Randomization, Allocation Concealment, and Blinding

Eligible patients were randomly assigned in a 1: 1 ratio to either the SG or the CG. The randomization sequence was computer-generated using block randomization (block size of 6) by an independent statistician not involved in patient recruitment or outcome assessment.

The allocation sequence was concealed using sequentially numbered, sealed, opaque envelopes. The envelopes were prepared by the independent statistician and kept securely by the study coordinator. After a patient completed all baseline assessments and signed informed consent, the study coordinator opened the next sequentially numbered envelope to reveal the group assignment. This procedure ensured that the treatment allocation was unknown before enrollment.

Given the nature of the intervention (availability of genetic test results), participants and treating physicians were not blinded to group assignment (open-label). However, to minimize bias in outcome assessment, the research nurses who performed all follow-up blood pressure measurements, conducted the structured adherence interviews, and collected adverse event data were blinded to the patient's group allocation. Furthermore, personnel in the laboratory performing the genotyping assays were blinded to all clinical data and group assignments. The statistician performing the final data analysis was also blinded to group codes until the primary analysis was complete.

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Table 3. Individualized medication for hypertension under pharmacogenomics guidance.

Detection site	Genotype	Phenotype	Expected effect	Drug types	Related drugs
CYP2D6 [25]	*1/*1	Metabolic function was normal	Normal level	β-blocker	Labelolol, aprolol, metoprolol, carvedilol, bisoprolol
	*1/*10	Metabolic function was slightly lower	Slightly higher		
	*10/*10	Metabolic function was lower	Higher		
CYP2C9 [26]	*1/*1	Metabolic function was normal	Normal level	Angiotensin II receptor antagonist	Valsartan, losartan, irbesartan, candesartan, irbesartan
	*1/*3	Metabolic function was slightly lower	Slightly higher (losartan was slightly lower)		
	*3/*3	Metabolic function was lower	Higher (losartan was lower)		
ACE [27]	II	Enzyme activity was normal	Normal level	Angiotensin converting enzyme inhibitors	Benazepril, enalapril, fosinopril, pedoapril, ramipril
	ID	Enzyme activity was slightly higher	Slightly higher		
	DD	Enzyme activity was higher	Higher		
NPPA (2238T>C) [28]	TT	Sensitivity was normal	Normal level	Diuretic	Chlorothiazide, hydrochlorothiazide, benzyl fluthiazide, chlorothiazone
	TC	Sensitivity was slightly higher	Slightly higher		
	CC	Sensitivity was higher	Higher		
CYP3A5 [29]	*1/*1	Metabolic function was normal	Normal level	Calcium ion antagonist	Nifedipine, felodipine, racidipine, amlodipine, cinidipine
	*1/*3	Metabolic function was slightly lower	Slightly higher		
	*3/*3	Metabolic function was lower	Higher		

Methods

This study was conducted within a hypertension specialty care pathway. All enrolled patients were admitted to the hospital for the initial phase of treatment optimization and monitoring. Following discharge, patients entered a structured outpatient follow-up program for continued management and outcome assessment.

The inpatient phase focused on diagnostic confirmation, pharmacogenomic testing (for SG), initiation/titration of the guided antihypertensive regimen, and patient education. The outpatient phase involved regular clinic visits and telephone consultations to assess adherence, monitor blood pressure, manage adverse effects, and prevent rehospitalization.

Patients in the CG were treated using the traditional diagnosis and treatment mode. Patients in the SG were given individualized medication under pharmacogenomics guidance. Using the ARMs-PCR+ sequencing method, 6 polymorphic gene loci (CYP2D6, CYP2C9, AGTR1, ACE, NPPA, and CYP3A5) related to 5 types of antihypertensive drugs (diuretics, β-blockers, angiotensin converting enzyme inhibitors, angiotensin II receptor antagonists, and calcium antagonists) were detected.

The specific 6 gene loci were selected through a multi-step process to ensure clinical relevance and actionability. First, a comprehensive literature review was conducted to identify gene variants with Level A or B evidence (strong or moderate) for affecting antihypertensive drug response, as defined by major pharmacogenomics consortia such as the Clinical Pharmacogenetics Implementation Consortium and the Dutch Pharmacogenetics Working Group. Second, priority was given

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to variants influencing the 5 first-line antihypertensive drug classes recommended by major hypertension guidelines (diuretics, β-blockers, ACE inhibitors, angiotensin II receptor blockers [ARBs], and calcium channel blockers). Third, the selection was limited to polymorphisms for which reliable and clinically validated ARMs-PCR assays were available in our laboratory. All laboratory procedures were performed by licensed medical laboratory technicians under the supervision of a board-certified clinical pathologist, following standardized operating procedures with internal quality controls. The final panel (CYP2D6*10, CYP2C9*3, AGTR1 A1166C, ACE I/D, NPPA T2238C, and CYP3A5*3) represents a consensus set with well-documented effects on drug metabolism (CYP2D6, CYP2C9, and CYP3A5), drug targets (AGTR1 and ACE), and diuretic response (NPPA), thereby covering the major pharmacokinetic and pharmacodynamic pathways of the included drug classes. The individualized medication regimen was guided according to the determination results, as shown in **Table 3**.

All enrolled patients were admitted to the hospital for the initial phase of treatment optimization and monitoring (median length described in results). Following discharge, all patients entered a structured outpatient follow-up program with scheduled visits at 1, 3, and 6 months.

Outcome Measures and Assessments

Blood Pressure Measurement and Control Definition

Blood pressure was measured by trained nursing staff using a validated, automated upper-arm oscillometric device (brand: OMRON; model: HEM-7124), which was regularly calibrated. All measurements were taken in a dedicated, quiet examination room with controlled temperature. Patients were instructed to avoid caffeine, exercise, and smoking for at least 30 minutes prior to measurement. They were seated in a chair with back support, feet flat on the floor, and the cuff placed on the bare upper arm at heart level. After a 5-minute rest period, 3 consecutive blood pressure readings were taken at 1-minute intervals. The average of the second and third readings was recorded as the representative value for that time point. This protocol was followed to obtain baseline blood pressure at admission, and subsequently daily at 8: 00 AM and 4: 00 PM throughout the hospitalization. Heart rate was automatically recorded by the device alongside each blood pressure measurement. At admission, a complete set of fasting blood biochemical tests were conducted to monitor and record adverse reactions related to antihypertensive drugs and other drug-induced reactions during the medication period. The blood pressure at discharge was the patient's blood pressure after receiving hospital treatment.

For the purpose of calculating the blood pressure control rate (also referred to as the standard compliance rate), a patient was considered to have achieved blood pressure control at a given time point (discharge, 3-month follow-up, 6-month follow-up) if their average seated office blood pressure, measured and calculated as per the protocol above, was less than 140 mmHg systolic and less than 90 mmHg diastolic (<140/90 mmHg). The blood pressure control rate for each group at each time point was then calculated as the number of patients achieving control divided by the total number of patients assessed at that time point, expressed as a percentage.

Assessment of Treatment Compliance

Patient compliance with the prescribed antihypertensive medication regimen was assessed at the 6-month follow-up visit through a structured interview conducted by a study nurse, combined with a review of pharmacy refill records when available. Compliance was categorized into 3 mutually exclusive levels based on patient self-report and corroborating data. (1) Complete compliance: The patient reported taking all prescribed antihypertensive medications at the correct dosage and timing, without any missed doses, over the preceding month. (2) Partial compliance: The patient reported missing doses or taking incorrect doses of prescribed antihypertensive medications on 1 to 3 occasions within the preceding month, but overall adherence was estimated to be ≥80% of the prescribed regimen. (3) Non-compliance: The patient reported missing doses or taking incorrect doses on 4 or more occasions within the preceding month, or self-discontinuation of 1 or more antihypertensive drugs for a period exceeding 1 week. Overall adherence was estimated to be <80% of the prescribed regimen. For analysis, the category of "overall compliance" was defined as the combined proportion of patients in the complete compliance and partial compliance groups.

Assessment of Adverse Events and Readmissions

All adverse events reported by patients or observed by clinicians during hospitalization and throughout the 6-month follow-up were recorded. Events were assessed for causality to study drugs by the treating physician and categorized using standard terminology.

All-cause readmission to our hospital within 6 months after the index discharge was captured through the hospital's electronic medical record system and confirmed via telephone follow-up. The primary reason for readmission was adjudicated based on discharge diagnosis.

Primary and Secondary Endpoints

The primary and secondary endpoints of this study were prospectively defined as follows. The primary endpoint was the proportion of patients achieving blood pressure control at 6

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months after discharge. The secondary endpoints were as follows: (1) blood pressure control rate at 3 months after discharge (same definition as primary endpoint); (2) treatment compliance at 6 months, assessed as described above and categorized as complete or partial or non-compliance; (3) incidence of drug-related adverse reactions during the entire 6-month follow-up period; (4) all-cause rehospitalization rate at 3 months and 6 months after discharge; (5) number of antihypertensive drug adjustments during the initial hospitalization; and (6) time to achieve in-hospital blood pressure control, defined as the number of days from admission to the first day when the patient's blood pressure met the control standard (<140/90 mmHg) and remained stable for 24 hours.

Outcome Assessors and Quality Assurance

All outcome assessments (blood pressure measurement, compliance interviews) were performed by trained research nurses who were blinded to the patient's group allocation. To ensure consistency, all assessors completed a standardized training program on the measurement protocol and interview techniques prior to the study start.

Source data verification was performed on a random 10% sample of case report forms. Data entry was performed independently by 2 research assistants, with discrepancies resolved by referring to the original source documents.

Statistical Analysis

Software and Data Preparation

All statistical analyses were performed using IBM SPSS Statistics software (version 27.0; IBM Corp, Armonk, NY, USA). Prior to analysis, the distribution and normality of all continuous variables were assessed using the Shapiro-Wilk test and visual inspection of Q-Q plots. Data preparation included checks for outliers and data entry errors. As stated in above, source data verification and independent double data entry were conducted to ensure the highest level of data integrity for the analytical dataset.

Descriptive and Comparative Statistics

Descriptive statistics are presented according to data type and distribution. Continuous variables with a normal distribution are reported as mean±standard deviation (SD); variables with a non-normal distribution are reported as median and interquartile range (IQR). Categorical variables are presented as counts and percentages (n,%).

To confirm the success of randomization, baseline characteristics between the SG and CG were formally compared. For continuous variables, the independent samples *t* test (normal) or Mann-Whitney U test (non-normal) was used. For categorical variables, the chi-square (χ^2) test or Fisher exact test (for expected cell counts <5) was applied. All *P* values for baseline comparisons are reported to 3 decimal places in **Table 2**.

Analysis of Primary and Secondary Endpoints

The primary efficacy analysis was conducted on the intention-to-treat (ITT) population, which included all 900 randomly assigned patients. For the primary endpoint (blood pressure control rate at 6 months), a conservative approach was adopted: any patient lost to follow-up was imputed as a treatment failure (non-responder). Differences between groups for the primary endpoint were evaluated using the χ^2 test, and the effect size is presented as the absolute risk difference with its corresponding 95% confidence interval (CI).

Secondary endpoints were analyzed in the ITT population where applicable. Between-group comparisons for continuous secondary endpoints (eg, time to in-hospital blood pressure control) used the independent samples *t* test or Mann-Whitney U test, as appropriate. For binary secondary endpoints (eg, 3-month control rate and adverse event incidence), the χ^2 test or Fisher's exact test was used, with results reported as proportions and 95% CIs for the between-group difference. No statistical adjustments for multiple comparisons were applied to the analysis of secondary endpoints, as these were considered exploratory to generate hypotheses for future research.

A supportive per-protocol analysis of the primary endpoint, which included only patients who completed the study according to the protocol without major deviations, was performed as a sensitivity analysis.

Significance and Reporting Standards

A 2-sided *P* value of less than 0.05 was considered statistically significant for all hypothesis tests. In the Results section, exact *P* values are reported to 2 or 3 decimal places unless *P*<0.001. For key comparative outcomes (primary endpoint and major secondary endpoints), 95% CIs are reported alongside point estimates and *P* values to convey the precision of the effect size. All analyses were pre-specified in the study protocol.

Results

Data Overview and Integrity

All 900 enrolled patients completed the initial in-hospital intervention phase and were included in the analysis. During the 6-month post-discharge follow-up period, the median follow-up

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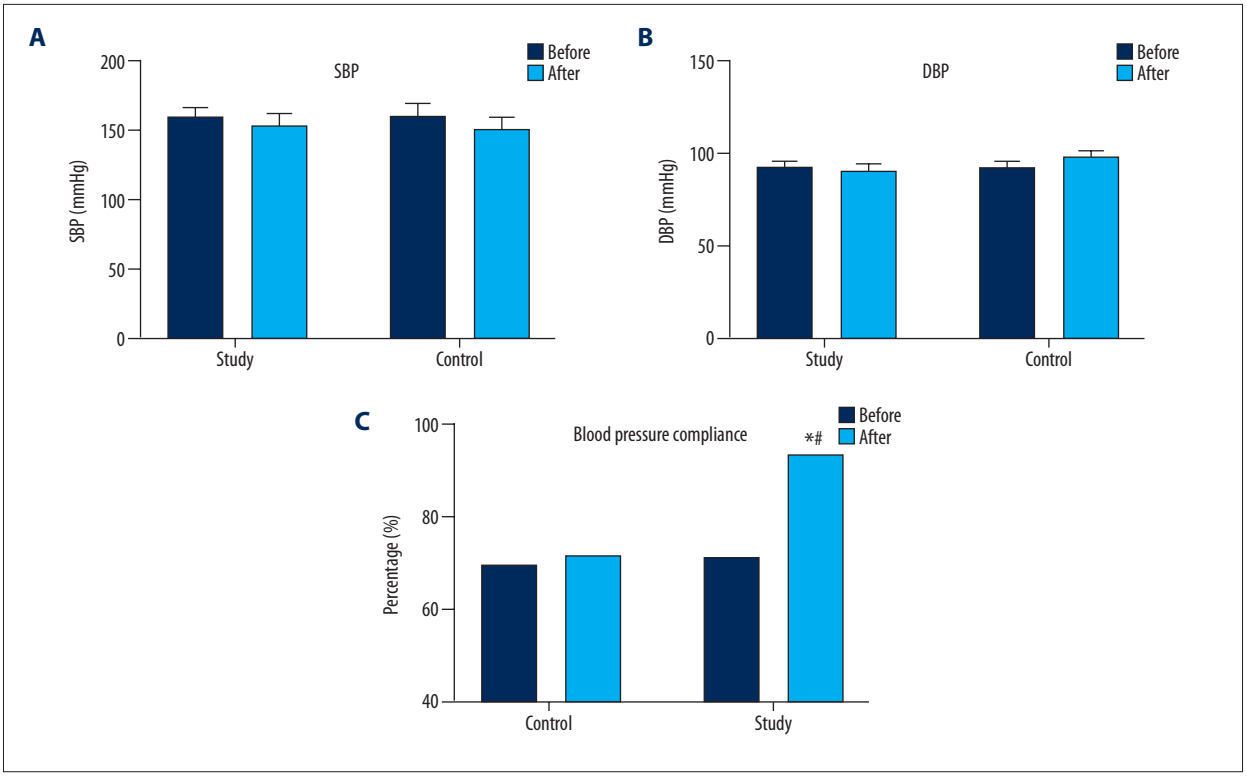

Figure 1. Blood pressure outcomes during the study. (**A**) Mean (±SD) systolic blood pressure (SBP) at baseline, discharge, 3-month, and 6-month follow-up. (**B**) Mean (±SD) diastolic blood pressure at the same time points. (**C**) Proportion of patients achieving blood pressure control (<140/90 mmHg) at discharge, 3-month, and 6-month follow-up. Data are from the intention-to-treat population (n=450 per group). Between-group comparisons at each post-baseline time point were performed using the independent samples *t* test (**A, B**) or chi-square test (**C**). * *P*<0.001 vs control group at the same time point; # *P*<0.05 vs baseline within the same group. Error bars in C represent 95% CIs.

Table 4. Treatment compliance at 6-month follow-up in the study and control groups.

Group	n	Complete compliance |n (%)	Partial compliance n (%)	Non-compliance n (%)	Overall compliance n (%)	Between-group difference in overall compliance% (95% CI)
Control group	450	198 (44.0)	135 (30.0)	117 (26.0)	**333 (74.0)**	**Reference**
Study group	450	297 (66.0)	144 (32.0)	9 (2.0)	**441 (98.0)**	**+24.0 (19.8-28.2)**
***P* value**		**<0.001**	**0.502**	**<0.001**	**<0.001**	

Overall compliance was defined as the sum of complete and partial compliance. Percentages may not sum to 100% due to rounding. Between-group differences and 95% CIs for proportions were calculated using the chi-square test. *P* values are for the comparison between the study group and control group for each compliance category and for overall compliance.

time was 185 days (range: 180 to 210 days) for both groups. No patients were lost to follow-up in either group. The distribution of all continuous outcome variables was assessed and confirmed to meet the assumptions of the applied parametric tests (or non-parametric alternatives where needed). The analytical dataset was locked after final quality checks.

Standard Blood Pressure Value

Before treatment, no significant difference was observed in systolic blood pressure (SG: 152.3±10.4 mmHg vs CG: 151.8±10.1 mmHg; *P*=0.480) or diastolic blood pressure (SG: 94.2±6.8 mmHg vs CG: 93.9±6.6 mmHg; *P*=0.531) between groups. After intervention, no significant difference was observed in the rate of blood pressure meeting the standard at discharge between

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Table 5. Prescription patterns of major antihypertensive drug classes during the study period.

Antihypertensive drug class	Study group (n=450)	Control group (n=450)	*P* value
Angiotensin-converting enzyme inhibitors (ACEIs)	190 (42.2%)	198 (44.0%)	0.621
– Benazepril	85 (44.7% of ACEI users)	92 (46.5% of ACEI users)	0.712
– Enalapril	72 (37.9% of ACEI users)	70 (35.4% of ACEI users)	0.653
– Other ACEIs (fosinopril, ramipril, etc.)	33 (17.4% of ACEI users)	36 (18.2% of ACEI users)	0.852
Angiotensin II receptor blockers (ARBs)	205 (45.6%)	195 (43.3%)	0.524
– Losartan	88 (42.9% of ARB users)	95 (48.7% of ARB users)	0.245
– Valsartan	75 (36.6% of ARB users)	68 (34.9% of ARB users)	0.691
– Other arbs (irbesartan, candesartan, etc.)	42 (20.5% of ARB users)	32 (16.4% of ARB users)	0.302
Beta-blockers	220 (48.9%)	215 (47.8%)	0.752
– Metoprolol	125 (56.8% of Beta-blocker users)	130 (60.5% of Beta-blocker users)	0.482
– Bisoprolol	65 (29.5% of Beta-blocker users)	58 (27.0% of Beta-blocker users)	0.572
– Other beta-blockers (carvedilol, etc.)	30 (13.6% of Beta-blocker users)	27 (12.6% of Beta-blocker users)	0.723
Calcium channel blockers (CCBs)	238 (52.9%)	232 (51.6%)	0.712
– Amlodipine	150 (63.0% of CCB users)	145 (62.5% of CCB users)	0.912
– Nifedipine	55 (23.1% of CCB users)	58 (25.0% of CCB users)	0.665
– Other CCBs (felodipine, etc.)	33 (13.9% of CCB users)	29 (12.5% of CCB users)	0.695
Diuretics	165 (36.7%)	158 (35.1%)	0.638
– Hydrochlorothiazide	120 (72.7% of diuretic users)	115 (72.8% of diuretic users)	0.991
– Other diuretics (indapamide, etc.)	45 (27.3% of diuretic users)	43 (27.2% of diuretic users)	0.983
Patients receiving combination therapy (≥2 classes)	385 (85.6%)	378 (84.0%)	0.552
Median number of antihypertensive drugs per patient [IQR]	2 [1-3]	2 [1-3]	0.884

Percentages in the main categories (ACEIs, ARBs, etc.) are calculated as "number of users in the group divided by total patients in the group (450)". Subcategory percentages (eg, benazepril users among all ACEI users) are calculated as number of users of that specific drug divided by total users of that drug class in the group. Most patients were on combination therapy; therefore, the sum of percentages across drug classes exceeds 100%. *P* values were calculated using the chi-square test for categorical variables and the Mann-Whitney U test for the median number of drugs. IQR – interquartile range.

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Table 6. Incidence of adverse drug reactions during the 6-month follow-up period.

Adverse reaction	Study group (n=450) n (%)	Control group (n=450) n (%)	Between-group difference% (95% CI)	*P* value
Dry cough	7 (1.6)	37 (8.3)	**-6.7 (-10.1 to -3.3)**	**<0.001**
Ankle edema	11 (2.4)	15 (3.3)	**-0.9 (-3.2 to 1.4)**	0.607
Flushing	7 (1.6)	8 (1.8)	**-0.2 (-2.0 to 1.6)**	0.796
Hypokalemia	0 (0.0)	8 (1.8)	**-1.8 (-3.4 to -0.2)**	0.007*
Any adverse reaction	**25 (5.6)**	**68 (15.1)**	**-9.5 (-13.7 to -5.3)**	**<0.001**

Data are presented as number (percentage). Between-group differences and 95% CIs were calculated using the chi-square test, except for hypokalemia where Fisher's exact test was used (indicated by *). *P* values <0.05 are considered statistically significant.

Table 7. All-cause readmission rates at 3 and 6 months after discharge.

Time point	Study group (n=450)	Control group (n=450)	Between-group risk difference% (95% CI)	*P* value
	Readmissions n (%)	Readmissions n (%)		
3 months after discharge	15 (3.3)	19 (4.2)	**-0.9 (-3.5 to 1.7)**	0.546
6 months after discharge	11 (2.4)	40 (8.9)	**-6.5 (-9.8 to -3.2)**	**<0.001**
Total (0-6 months)	**26 (5.8)**	**59 (13.1)**	**-7.3 (-11.2 to -3.4)**	**<0.001**

The readmission rate was calculated as the number of patients readmitted at least once during the specified period divided by the total number of patients in the group. Between-group risk differences and 95% Cis were calculated using the chi-square test. *P* values <0.05 are considered statistically significant.

both groups (SG: 76.2% vs CG: 74.9%; *P*=0.642). However, the rate of blood pressure control at 3 months after discharge was significantly higher in the SG compared with the CG (80.7% vs 70.2%; between-group difference: 10.5%, 95% CI: 5.3%-15.7%; *P*<0.001). This superiority was maintained at 6 months (SG: 78.4% vs CG: 65.1%; between-group difference: 13.3%, 95% CI: 8.1%-18.5%; *P*<0.001). The trajectory of mean systolic and diastolic blood pressures over time is shown in **Figure 1A and 1B**, with the corresponding control rates illustrated in **Figure 1C**.

Treatment Compliance

The overall treatment compliance at 6 months was significantly better in the SG than the CG (98.0% vs 74.0%; between-group difference: 24.0%, 95% CI: 19.8%-28.2%; *P*<0.001). The detailed breakdown of compliance categories (complete, partial, and non-compliance) is presented in **Table 4**.

Antihypertensive Medication Profiles

The overall prescription patterns of the 5 major antihypertensive drug classes during the study period are summarized in **Table 5**. Critically, the proportion of patients prescribed angiotensin-converting enzyme inhibitors (ACEIs) was not significantly different between the SG and the CG (SG: 42.2% vs CG: 44.0%; *P*=0.621). This indicates that the observed reduction in ACEI-related cough in the SG was not attributable to a lower usage rate of this drug class.

Adverse Reactions and Readmissions

There were no serious adverse reactions to antihypertensive drugs in either group. The incidence of ACEI drug-related dry cough was significantly lower in the SG (SG: 1.6% vs CG: 8.3%; between-group difference: -6.7%; 95% CI, -10.1% to -3.3%; *P*<0.001). No significant differences were observed in the incidence of ankle edema, flushing, or hypokalemia between groups (all *P*>0.05; **Table 6**).

The all-cause readmission rate at 3 months after discharge was not significantly different between groups (SG: 3.3% vs CG: 4.2%; *P*=0.546). However, the readmission rate at 6 months after discharge was significantly reduced in the SG (SG: 2.4% vs CG: 8.9%; between-group difference: -6.5%; 95% CI, -9.8% to -3.2%; *P*<0.001; **Table 7**). The most common primary reasons for readmission across both groups were uncontrolled hypertension (n=38, 63.3% of all readmissions), new-onset or worsening angina pectoris (n=7, 11.7%), and exacerbation of heart failure (n=6, 10.0%).

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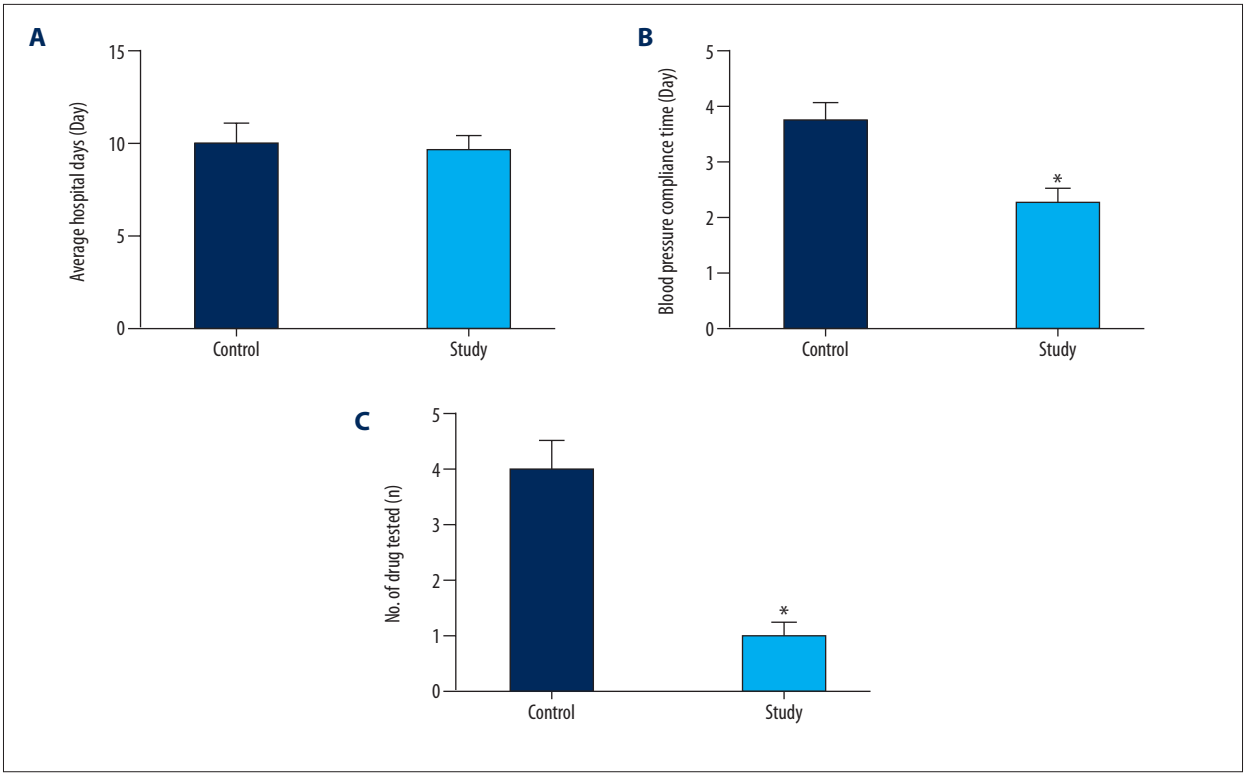

Figure 2. Metrics of in-hospital management efficiency and treatment complexity. (**A**) Average length of hospital stay (mean±SD). (**B**) Time to achieve in-hospital blood pressure control (median with interquartile range). (**C**) Number of distinct antihypertensive drug varieties tried during hospitalization (mean±SD). Data are presented for the study group (SG, n=450) and control group (CG, n=450). Between-group comparisons were performed using the independent samples *t* test (**A, C**) and Mann-Whitney U test (**B**). * *P*<0.001; NS, not significant (*P*=0.451). Error bars represent SD (**A, C**) or IQR (B).

In-Hospital Process Efficiency and Treatment Complexity

The efficiency of in-hospital management and the complexity of initial treatment regimens are shown in **Figure 2**. The average length of hospital stay was similar between the 2 groups (SG: 10.2±2.1 days vs CG: 10.1±2.3 days; mean difference: 0.1 days; 95% CI, -0.18 to 0.38; *P*=0.451; **Figure 2A**). However, the time to achieve in-hospital blood pressure control (<140/90 mmHg) was significantly shorter in the SG compared with the CG (median: 4 days, IQR: 2-6 vs median: 6 days; IQR: 4-9; *P*<0.001; **Figure 2B**). Correspondingly, the number of distinct antihypertensive drug varieties tried during the initial hospitalization was lower in the SG (mean: 1.8±0.7 vs 2.3±0.9; mean difference: -0.5; 95% CI: -0.65 to -0.35; *P*<0.001; **Figure 2C**).

Discussion

This prospective randomized controlled trial demonstrates that a pharmacogenomic-guided strategy using a 6-gene panel significantly improved several key clinical outcomes in patients with hypertension compared with conventional therapy. The success of this approach can be attributed to a precision mechanism that addresses core limitations of empirical treatment.

First, pharmacogenomic guidance reduces therapeutic uncertainty by informing initial drug selection. Genetic variants in key pharmacokinetic (CYP2D6, CYP2C9, and CYP3A5) and pharmacodynamic (AGTR1, ACE, and NPPA) genes have been well documented to alter drug response [12-18]. By preemptively identifying these variants, our protocol allowed clinicians to avoid potentially ineffective or problematic drugs, leading to a more efficient path to blood pressure control (shorter in-hospital control time, fewer drug trials). This directly addresses the challenge of inter-individual variability that underpins the traditional "trial-and-error" approach. Second, it mitigates predictable adverse drug reactions, thereby improving tolerability and adherence. The most salient example is the significant reduction in ACEI-induced cough in the SG, which occurred despite comparable ACEI prescription rates. This finding is mechanistically supported by studies linking ACE I/D polymorphism to bradykinin metabolism and cough risk. By using genetic data to identify susceptible individuals and guide them toward alternatives like ARBs (whose response may be influenced by AGTR1 genotype), pharmacogenomic enhances treatment safety. Our

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results align with and are bolstered by a recent retrospective study which also found that pharmacogenetic testing–guided therapy significantly improved medication tolerability and reduced adverse effects [19]. Third, this precision approach likely synergizes with and enhances patient-centered care. The objective, personalized rationale provided by genetic testing may improve patient understanding and trust in the prescribed regimen. This, combined with faster efficacy and better tolerability, creates a positive feedback loop that explains the markedly higher treatment compliance observed in the SG. Improved adherence is a critical determinant of long-term blood pressure control [20], which in turn drives the observed reduction in hypertension-related rehospitalizations.

Our positive results contribute to an evolving and sometimes heterogeneous evidence base for pharmacogenomics in hypertension. They align with and extend prior research demonstrating associations between single polymorphisms, such as CYP2D6 and β-blocker metabolism, and NPPA and diuretic response and drug outcomes. Notably, a guideline from the Clinical Pharmacogenetics Implementation Consortium now provides therapeutic recommendations for CYP2D6 metabolizer status in relation to metoprolol therapy, underscoring the growing clinical recognition of such evidence [8]. However, we move beyond association studies by demonstrating that the integrated application of a multi-gene panel within a clinical workflow can translate into tangible improvements in composite endpoints like blood pressure control rate and rehospitalization.

It is important to acknowledge that some randomized trials, particularly in primary care settings or evaluating narrower gene panels, have reported more modest benefits. For instance, a recent large pragmatic trial in China showed that a guideline-based clinical decision support system improved treatment standardization but had only a modest, non-significant impact on overall blood pressure control rates [21]. This heterogeneity underscores that the effectiveness of pharmacogenomics is context-dependent, influenced by factors such as panel composition (our panel covered major drug classes), clinical setting (specialty care with rapid turnaround), and patient population (may have higher a priori risk of suboptimal response). Our study provides a proof-of-concept that in a setting where these factors are optimized, pharmacogenomic guidance can be highly effective. Future meta-analyses and comparative effectiveness research will be crucial to define the optimal application scope and target population.

Looking forward, the field is rapidly advancing beyond single-gene associations. As discussed in a review on genomic applications in hypertension, the next frontier includes polygenic risk scores for stratifying therapeutic response, as well as novel therapeutic classes (eg, cGMP augmentation and angiotensinogen-targeting RNA therapies) whose development is itself guided by genomic insights [22]. This highlights how our study contributes to the broader, 2-pronged effect of genomics by both personalizing existing therapies and informing the development of future ones.

The findings also resonate with the broader shift toward personalized medicine, highlighting how genetic insights can operationalize the principle of "treating different individuals differently" [23,24]. As pharmacogenomics continues to mature, supported by consortia like the Clinical Pharmacogenetics Implementation Consortium, its integration into chronic disease management paradigms like hypertension represents a logical step forward from the current one-size-fits-all guideline approach.

The economic viability of pharmacogenomic testing depends on several factors, including the prevalence of actionable polymorphisms, the availability and cost of alternative drugs, and the local healthcare infrastructure. In settings where only a limited selection of antihypertensive drugs is available (eg, only 1 ARB and 1 ACEI), the value of genotyping may be attenuated if the tested variants do not strongly predict response to those specific agents. However, even in such contexts, knowledge of genetic susceptibility to adverse reactions (eg, ACE I/D polymorphism and cough risk) could still prevent ineffective trials and improve adherence. Future health economic studies conducted in diverse healthcare systems are needed to establish the cost-effectiveness of pharmacogenomic-guided therapy under different formulary constraints.

Several limitations of this study should be acknowledged. First, the assessment of blood pressure control relied on clinic office measurements rather than 24-hour ambulatory blood pressure monitoring or home blood pressure monitoring. While office blood pressure is the most widely used metric in clinical practice and trials, ambulatory blood pressure monitoring provides a more accurate assessment of true blood pressure burden, nocturnal hypertension, and white-coat or masked hypertension phenotypes. The absence of ambulatory blood pressure monitoring may have led to an incomplete characterization of the 24-hour blood pressure profile in some patients. Second, this was a single-center study, which may limit the generalizability of the findings to other healthcare settings or populations with different genetic backgrounds. Third, the follow-up duration was limited to 6 months; longer-term effects of pharmacogenomic-guided therapy on cardiovascular outcomes and sustainability of adherence remain to be investigated. Fourth, the open-label design, inherent to the nature of the intervention, may have introduced performance or assessment bias, although efforts were made to blind the outcome assessors.

For clinical implementation, as outlined in our model, pharmacogenomic testing can be ordered at the initial specialist consultation, with results available to guide regimen finalization at the first follow-up visit, aligning with typical management intervals.

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Future research should focus on large-scale, multicenter pragmatic trials to confirm generalizability and evaluate cost-effectiveness; longer-term follow-up to assess impact on hard cardiovascular endpoints; and exploration in diverse populations and healthcare systems to define the broadest applicable scope.

Conclusions

In conclusion, this study provides evidence that a pharmacogenomics-guided approach using a 6-gene panel can improve the precision of antihypertensive therapy. It contributes to better blood pressure control, higher treatment adherence, fewer drug-related adverse events, and lower rehospitalization rates by informing more personalized initial drug selection and reducing therapeutic uncertainty. While further validation is warranted, these findings support the growing role of pharmacogenomics in moving hypertension management toward a more personalized and effective paradigm.

Author Contributions

AI-assisted language polishing: In the revision process following reviewers' comments, the authors utilized an AI-powered language model (DeepSeek) to assist in restructuring and polishing sentences in the Introduction, Methods, and Discussion sections to enhance academic tone and clarity. All substantive scientific content, data, and interpretations remain entirely the work of the authors, who reviewed and edited all AI-generated suggestions thoroughly.

Institution Where Work Was Done

The Second People's Hospital of Foshan, Guangdong, PR China

Patient Permission/Consent

All participants provided written informed consent prior to enrollment.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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