International Journal of General Medicine

Dovepress
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ORIGINAL RESEARCH

Effects of Trimetazidine Combined with Atorvastatin on Cardiac Function in Patients with Stable Angina Pectoris of Coronary Heart Disease

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Objective: Growing evidence suggests that metabolic modulation combined with lipid-lowering therapy may provide synergistic benefits in coronary heart disease (CHD). This study aimed to evaluate the effects of trimetazidine combined with atorvastatin on cardiac function and inflammatory responses in stable angina pectoris.

Methods: This retrospective study analyzed 100 CHD patients with stable angina (2019 ESC criteria) treated at the Third Affiliated Hospital of Qiqihar Medical College from 2021–2024. Patients were categorized into two groups based on prescription records, control group (n=50), receiving atorvastatin 20 mg/day, and an observation group (n=50), receiving atorvastatin 20 mg/day plus trimetazidine 20 mg TID. Outcomes included angina characteristics, lipid profile, endothelial function (NO/ET-1), specified inflammatory markers (hs-CRP, TNF-α), cardiac function (LVEF, NT-proBNP), and safety.

Results: The observation group had a significantly higher overall response rate (94.0% vs 80.0%, P<0.05). Both groups showed reduced angina frequency and duration post-treatment, with more pronounced improvements in the observation group (P<0.05). Chest pain severity (VAS) also decreased more significantly in the observation group, indicating superior efficacy (P<0.05). Lipid metabolism improved in both groups, with greater reductions in TC, TG, and LDL-C and a more substantial HDL-C increase in the observation group (P<0.05). Endothelial function markers improved, with lower ET-1, TNF-α, and hs-CRP levels and higher NO levels, showing more significant changes in the observation group (P<0.05). Cardiac function parameters improved in both groups, with greater reductions in LVEDD and NT-proBNP and greater increases in LVEF and 6MWD in the observation group (P<0.05). Adverse effects were low and comparable between groups (8.0% vs 6.0%, P>0.05).

Conclusion: The trimetazidine-atorvastatin combination demonstrates synergistic effects in improving angina symptoms, lipid metabolism, and cardiac function in stable CHD, with additional benefits in endothelial protection and measured inflammatory regulation. These findings support its consideration as adjunctive therapy, though further validation of broader inflammatory impacts is warranted.

Keywords: trimetazidine, atorvastatin, stable angina pectoris, coronary heart disease, cardiac function, inflammatory regulation

Introduction

Coronary heart disease (CHD) is a major cardiovascular condition that arises from the narrowing or blockage of coronary arteries due to atherosclerosis or coronary artery spasm, leading to inadequate blood supply to the myocardium. CHD is characterized by its high prevalence and associated mortality, posing a substantial public health burden worldwide.[1] Angina pectoris, a frequent clinical manifestation of CHD, is defined by chest pain or discomfort resulting from reduced myocardial oxygen supply during exertion or stress. This condition not only diminishes the quality of life of affected individuals but also escalates the risk of severe complications, such as myocardial infarction and heart failure, thereby significantly increasing patient morbidity and mortality.[2] Consequently, improving the therapeutic outcomes in angina pectoris, relieving its symptoms, and reducing the likelihood of catastrophic cardiovascular events are essential goals in the management of CHD.

Received: 9 January 2025
Accepted: 7 April 2025
Published: 16 April 2025

Treatment options for CHD include pharmacological therapies, interventional procedures, and surgical interventions. Among these, pharmacological treatment remains the most widely employed and accepted approach, particularly with the use of statins and anti-anginal medications. Atorvastatin, a well-known statin, lowers serum cholesterol levels by inhibiting the enzyme HMG-CoA reductase, thus stabilizing atherosclerotic plaques, slowing the progression of coronary artery disease, and improving patient prognosis.[3] Beyond its lipid-lowering effects, atorvastatin also exerts pleiotropic benefits, including anti-inflammatory actions through inhibition of C-reactive protein (CRP) and interleukin-6 (IL-6), as well as improvement of endothelial function by enhancing nitric oxide bioavailability.[4] Trimetazidine, a metabolic agent, offers an alternative mechanism of action by improving myocardial energy metabolism, specifically through inhibition of mitochondrial long-chain 3-ketoacyl-CoA thiolase (3-KAT), which shifts cardiac energy substrate utilization from fatty acid oxidation to glucose oxidation under ischemic conditions.[5] This metabolic shift reduces intracellular acidosis and calcium overload, preserves ATP production, and mitigates ischemia-reperfusion injury.[6]

Recent clinical studies have explored the synergistic effects of combining trimetazidine with statins in the treatment of CHD. For instance, a randomized trial by Zhao et al (2021) demonstrated that trimetazidine combined with atorvastatin significantly improved exercise tolerance and reduced angina frequency compared to statin monotherapy, attributed to complementary mechanisms of lipid regulation and metabolic protection.[7] Other studies further suggest that this combination may enhance plaque stability by suppressing matrix metalloproteinase-9 (MMP-9) activity and reducing oxidative stress biomarkers such as malondialdehyde (MDA).[8] However, there remains a lack of comprehensive studies specifically investigating the impact of combined trimetazidine and atorvastatin therapy on cardiac function and inflammatory markers in patients with stable angina pectoris.

The rationale for combining these agents lies in their complementary pathophysiological targets: while atorvastatin primarily addresses atherosclerotic progression and systemic inflammation, trimetazidine directly optimizes myocardial cellular metabolism during ischemic stress. This dual approach may offer novel therapeutic advantages by simultaneously improving coronary macrovascular health (via plaque stabilization) and microvascular/metabolic function (via ischemic preconditioning effects).[9] Therefore, this study aims to compare the therapeutic effects of the combination of trimetazidine and atorvastatin against atorvastatin monotherapy, with a focus on cardiac function, lipid profiles, vascular endothelial markers, and inflammatory factors. To the best of current knowledge, this study is the first to systematically evaluate the combined effects on both ventricular remodeling biomarkers (eg, B-type natriuretic peptide) and pro-inflammatory cytokines (eg, hs-CRP, IL-6, TNF-α) in stable angina patients. The findings of this research will provide valuable insights into optimizing the treatment strategy for stable angina pectoris and offer evidence-based guidance for clinical decision-making.

Study Participants and Methods

Study Population

This retrospective study analyzed clinical records of 100 patients diagnosed with stable angina pectoris due to coronary heart disease (CHD) who were admitted to the cardiology department of the Third Affiliated Hospital of Qiqihar Medical College between January 2021 and January 2024. Diagnostic criteria for stable angina pectoris followed the 2019 ESC Guidelines on Chronic Coronary Syndromes, including: (1) typical exertional chest pain relieved by rest/nitrates; (2) evidence of myocardial ischemia via exercise stress testing or coronary CT angiography (≥50% stenosis in ≥1 major coronary artery).[10]

Inclusion Criteria

Patients were enrolled based on the following conditions: ① Diagnosis confirmed according to the above criteria; ② Age ≥18 years, with no gender restrictions; ③ Classification of cardiac function as NYHA grade II–IV[11] and CCS angina grade I–III;[12] ④ No prior relevant treatment within the last 90 days before enrollment; ⑤ Availability of complete and reliable clinical data. Exclusion criteria: Patients meeting any of the following conditions were excluded: ① Presence of other cardiovascular diseases aside from CHD; ② Severe dysfunction of critical organs (eg, liver, kidney, or lung failure); ③ Significant immune system disorders, neurological abnormalities, coagulation or hematopoietic dysfunctions, or endocrine disorders; ④ History

of malignant tumors; ⑤ Presence of severe infections; ⑥ Known drug allergies or contraindications to the medications or procedures used in this study; ⑦ Cognitive impairment, altered consciousness, or severe psychiatric disorders.

Patients were categorized into two groups based on prescription records: a control group (n=50): Patients prescribed atorvastatin monotherapy during the study period. Observation group (n=50): Patients prescribed atorvastatin combined with trimetazidine. To minimize selection bias, propensity score matching (1:1 ratio) was applied using covariates including age (±5 years), gender, baseline LVEF (±5%), and LDL-C levels (±0.5 mmol/L).

The study protocol was reviewed and approved by the hospital's Medical Ethics Committee of the Third Affiliated Hospital of Qiqihar Medical College (Approval No. 2024LL-31) and complied with the Declaration of Helsinki. Informed consent was obtained from all study participants.

Treatment Methods

Rationale for 8-week duration: The treatment period was determined based on previous studies demonstrating significant improvements in angina symptoms and lipid profiles within 6–8 weeks of trimetazidine-statin combination therapy.[13]

Both groups received standard treatment measures, including the following: Nitrate therapy: Isosorbide mononitrate sustained-release tablets (Lunan Better Pharmaceutical Co., Ltd., National Drug Approval No. H10950235), administered at 40 mg once daily, to alleviate and prevent angina attacks. Antiplatelet therapy: Aspirin enteric-coated tablets (Bayer Healthcare Co., Ltd., National Drug Approval No. J20130080) at 100 mg once daily, in combination with clopidogrel (Sanofi-Aventis (Hangzhou) Pharmaceutical Co., Ltd., National Drug Approval No. H20051408) at 75 mg once daily, to reduce the risk of thrombus formation. Beta-blocker therapy: Bisoprolol tablets (Novartis Beijing Pharmaceutical Co., Ltd., National Drug Approval No. H20067638), given at a dose of 5 mg once daily, to control heart rate and decrease myocardial oxygen demand. Angiotensin-converting enzyme inhibitor (ACEI) therapy: Perindopril tablets (Servier Tianjin Pharmaceutical Co., Ltd., National Drug Approval No. H20056912), prescribed at 4 mg once daily, to lower blood pressure and improve cardiac remodeling. Oxygen therapy: Administered as low-flow oxygen (2–4 L/min) based on blood oxygen saturation (SpO_2), ensuring that SpO_2 remained at or above 95%. Treatment protocols for the study groups: Control group: Patients were given atorvastatin (Beijing Jialin Pharmaceutical Co., Ltd., National Drug Approval No. H19990258) at 20 mg once daily for 8 weeks. Observation group: In addition to the control group treatment, patients received trimetazidine (Beijing Wansheng Pharmaceutical Co., Ltd., National Drug Approval No. H20065167) at 20 mg three times daily for 8 weeks.

Adverse reaction monitoring: Adverse events were classified using CTCAE v5.0 criteria,[14] with severity graded as: Mild (Grade 1): Asymptomatic or mild symptoms (eg, transient nausea). Moderate (Grade 2): Symptomatic but not interfering with daily activities (eg, persistent headache). Severe (Grade 3–4): Life-threatening or requiring hospitalization (eg, rhabdomyolysis).

Observation Indicators

Sample size justification: The sample size of 50 per group was calculated using G*Power 3.1 with α=0.05, β=0.20, and effect size d=0.65 (based on prior LDL-C reduction data[15]), yielding a required total sample of 94 patients.

Clinical Efficacy

Evaluated using 2018 ACC/AHA Stable Ischemic Heart Disease Guidelines.[16] The response to treatment was classified into three categories: Significantly effective: Angina symptoms completely disappeared, or attack frequency decreased by more than 80% compared to pre-treatment levels, with electrocardiogram (ECG) results approaching normal. Effective: Angina attack frequency reduced by 50%–80%, ECG findings showed incomplete normalization, with ST-segment elevation of 0.05 mV, T-wave inversion shallowing by more than 25%, or T-wave becoming upright. Ineffective: Angina attack frequency reduction was less than 50%, with no significant ECG improvements, or even worsening trends such as ST-segment depression or deepened T-wave inversion.

Angina Attack Characteristics

Data on the frequency and duration of angina episodes were recorded both before and after treatment, with patient diaries and ECG Holter validation used for accuracy. The records were standardized and maintained by the medical staff for

consistency and reliability. Chest pain severity was assessed using the Visual Analog Scale (VAS). The VAS scale ranges from 0 to 10, with higher scores indicating more severe chest pain. Changes in VAS scores before and after treatment were used to evaluate therapeutic efficacy.

Blood Lipid Profile

Fasting venous blood (10 mL) collected in serum separation tubes; centrifuged at 3000 rpm for 15 min; serum supernatant analyzed for Total cholesterol (TC), Triglycerides (TG), High-density lipoprotein cholesterol (HDL-C), Low-density lipoprotein cholesterol (LDL-C).

Vascular Endothelial Function

Serum nitric oxide (NO) concentration was assessed using the nitrate reductase method from serum, while endothelin-1 (ET-1) levels were quantified via radioimmunoassay in plasma to evaluate endothelial function status before and after treatment.

Inflammatory Biomarkers

Inflammatory response levels were monitored using: Tumor necrosis factor-α (TNF-α): Measured from serum by enzyme-linked immunosorbent assay (ELISA). High-sensitivity C-reactive protein (hs-CRP): Assessed from serum via immunoturbidimetric analysis.

Cardiac Function Assessment

Key cardiac function indicators were measured both before and after treatment: N-terminal pro-brain natriuretic peptide (NT-proBNP) levels were obtained from serum samples. Echocardiographic assessment by two blinded cardiologists was performed using color Doppler ultrasound to determine: Left ventricular ejection fraction (LVEF), Left ventricular end-diastolic diameter (LVEDD), Functional capacity assessment was conducted via the 6-minute walk test (6MWT) in a standardized 30-m hospital corridor to evaluate exercise tolerance.

Adverse Reactions

All adverse events were systematically documented to assess treatment safety.

Statistical Analysis

Data processing and statistical analyses were conducted using SPSS 22.0 software, while visual representations of the results were generated using GraphPad Prism 8. Normality of data was confirmed using the Shapiro–Wilk test. Categorical variables were presented as percentages (%), and comparisons between groups were evaluated using the chi-square (χ^2) test. Continuous variables were expressed as ($\bar{x} \pm s$). For comparisons between two independent groups, an independent sample *t*-test was applied, whereas paired t-tests were utilized to assess pre- and post-treatment differences within the same group. For skewed variables, the non-parametric Mann–Whitney *U*-test was applied. A significance threshold of $P < 0.05$ was adopted to determine statistically significant differences.

Results

Comparison of Baseline Data

Baseline characteristics of both groups were statistically comparable ($P > 0.05$), as shown in Table 1. Propensity score matching successfully balanced key covariates between groups (standardized mean differences <0.1 for all variables).

Comparison of Clinical Efficacy

The total effective treatment rate of the observation group (94.0%) was higher than that of the control group (80.0%) ($P<0.05$), as shown in Table 2.

Comparison of Angina Pectoris Attacks

The frequency and duration of angina pectoris attacks decreased in both groups after treatment compared to before treatment, with a greater reduction observed in the observation group ($P<0.05$), Chest pain severity (Visual Analog

Table 1 Comparison of Baseline Data ($\bar{x} \pm s$, n [%])

	Control (n=50)	Observation (n=50)	t/x^2	P
Gender	–	–	0.162	0.687
Male	27 (54.0)	29 (58.0)	–	–
Female	23 (46.0)	21 (42.0)	–	–
Age (years)	61.79±5.43	62.16±5.64	0.334	0.739
Duration of disease (years)	6.57±1.59	6.83±1.46	0.851	0.396
BMI (kg/m²)	23.26±1.54	23.09±1.61	0.539	0.590
HR (beats/min)	75.84±6.32	74.92±6.58	0.713	0.477
SBP (mmHg)	131.82±8.67	130.54±9.12	0.719	0.473
DBP (mmHg)	79.45±5.21	80.16±5.43	0.667	0.506
NYHA functional classification	–	–	0.160	0.688
II	25 (50.0)	23 (46.0)	–	–
III	18 (36.0)	19 (38.0)	–	–
IV	7 (14.0)	8 (16.0)	–	–
CCS angina classification	–	–	0.164	0.685
I	20 (40.0)	22 (44.0)	–	–
II	22 (44.0)	19 (38.0)	–	–
III	8 (16.0)	9 (18.0)	–	–

Table 2 Comparison of Clinical Efficacy [n (%)]

Group (n)	Markedly Effective	Improved	Ineffective	Total Effective Rate
Control (n=50)	14 (28.0)	26 (52.0)	10 (20.0)	40 (80.0)
Observation (n=50)	22 (44.0)	25 (50.0)	3 (6.0)	47 (94.0)
x^2	–	–	–	4.332
P	–	–	–	0.037

Scale): Decreased from 6.23±1.14 to 2.16±0.81 in the observation group vs 6.03±1.30 to 3.85±1.25 in controls (between-group P=0.016). as shown in Figure 1.

Comparison of Lipid Levels

After treatment, the levels of TC, TG, and LDL-C decreased, while the levels of HDL-C increased in both groups compared to before treatment. The changes were more pronounced in the observation group (P<0.05), as shown in Figure 2.

Comparison of Vascular Endothelial Function and Inflammatory Factor Levels

After treatment, the levels of ET-1, TNF-α, and hs-CRP decreased, while the levels of NO increased in both groups compared to before treatment. The changes were more significant in the observation group (P<0.05), as shown in Figure 3.

Comparison of Cardiac Function Indicators

After treatment, the levels of LVEDD and NT-proBNP decreased, while the levels of LVEF and 6MWD increased in both groups compared to before treatment. The changes were more significant in the observation group (P<0.05), as shown in Figure 4.

Comparison of Adverse Reactions

The incidence of adverse reactions in the observation group (8.0%) compared to the control group (6.0%) showed no significant difference (P>0.05), as shown in Table 3. All adverse events were Grade 1–2 according to the Common

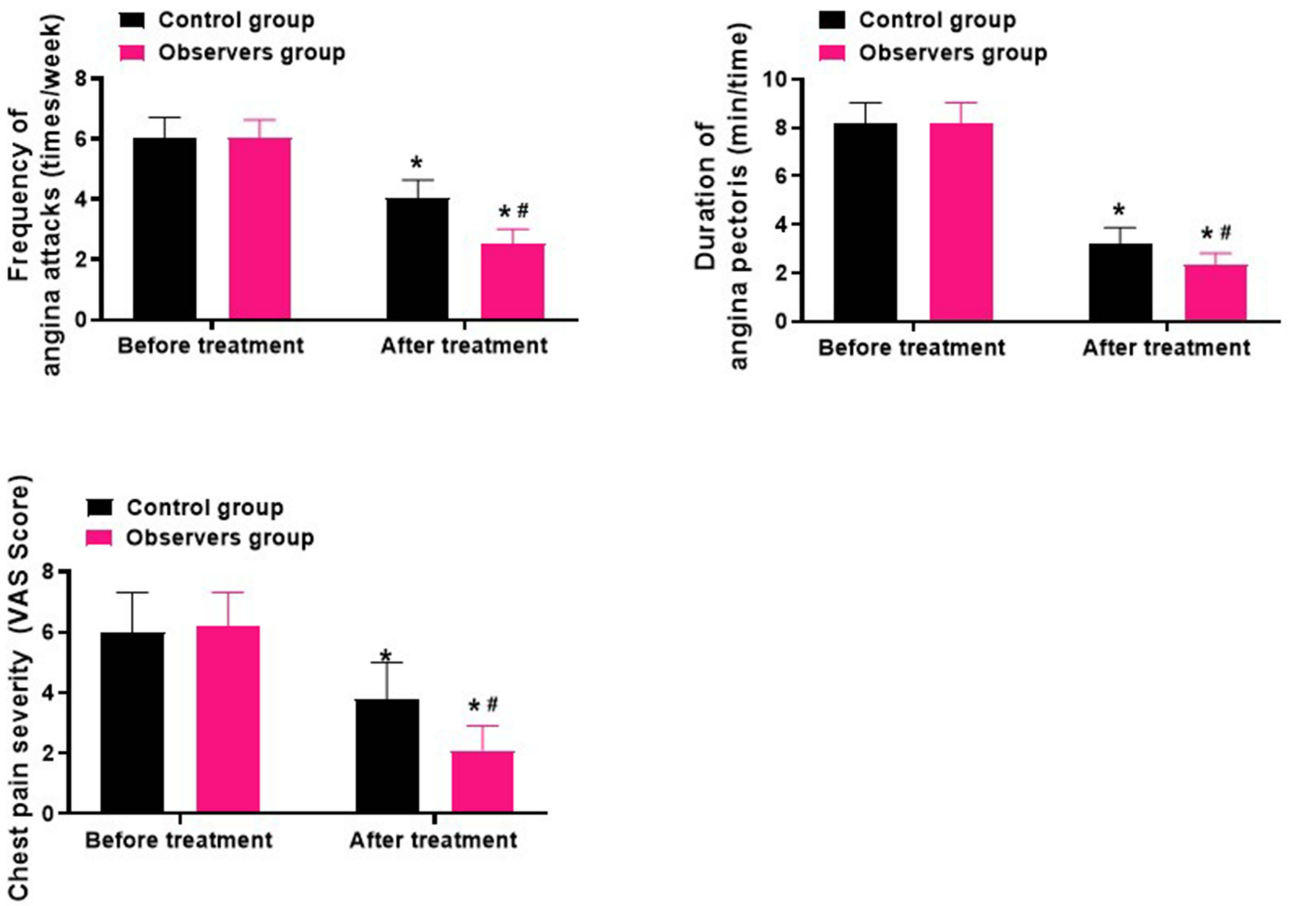

Figure 1 Comparison of Angina Pectoris Attacks ($\bar{x} \pm s$).
Note: Compared with before treatment, *P<0.05; compared between groups, #P<0.05.

Terminology Criteria for Adverse Events (CTCAE), and no treatment discontinuations occurred. The similar safety profiles between groups (P=0.72) remained consistent after adjusting for concomitant medications via Poisson regression.

Discussion

Stable angina pectoris, a common manifestation of coronary heart disease (CHD), arises from inadequate coronary perfusion, leading to episodic chest pain that significantly diminishes patients' quality of life while elevating their cardiovascular risk.[17] Pharmacological intervention remains the cornerstone of managing this condition, with statins playing a pivotal role due to their capacity to stabilize atherosclerotic plaques and lower the incidence of adverse cardiovascular events.[18] Among them, atorvastatin, a widely prescribed synthetic statin, functions by inhibiting hepatic hydroxymethylglutaryl-CoA reductase, thereby disrupting the cholesterol biosynthesis pathway. This mechanism contributes to the effective regulation of blood lipid levels, leading to reductions in total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) concentrations.[19] Compared to other statins, such as rosuvastatin, atorvastatin possesses distinct advantages. Firstly, its prolonged pharmacological activity and superior patient tolerance contribute to more stable lipid-lowering effects. Secondly, atorvastatin exhibits enhanced efficacy in suppressing cholesterol oxidation and facilitating reverse cholesterol transport, thereby improving vascular endothelial integrity and reducing the likelihood of atherosclerotic plaque rupture.[20] Considering its broad therapeutic spectrum, robust efficacy, and favorable safety profile, this study opted for atorvastatin as the principal pharmacological agent. Trimetazidine, a novel metabolic modulator derived from the piperazine class, exerts cardioprotective effects through its impact on myocardial energy metabolism.[21] By enhancing mitochondrial efficiency, trimetazidine facilitates fatty acid oxidation, optimizes coronary and peripheral circulation, and strengthens myocardial contractility. Additionally, this agent contributes to reducing blood viscosity and

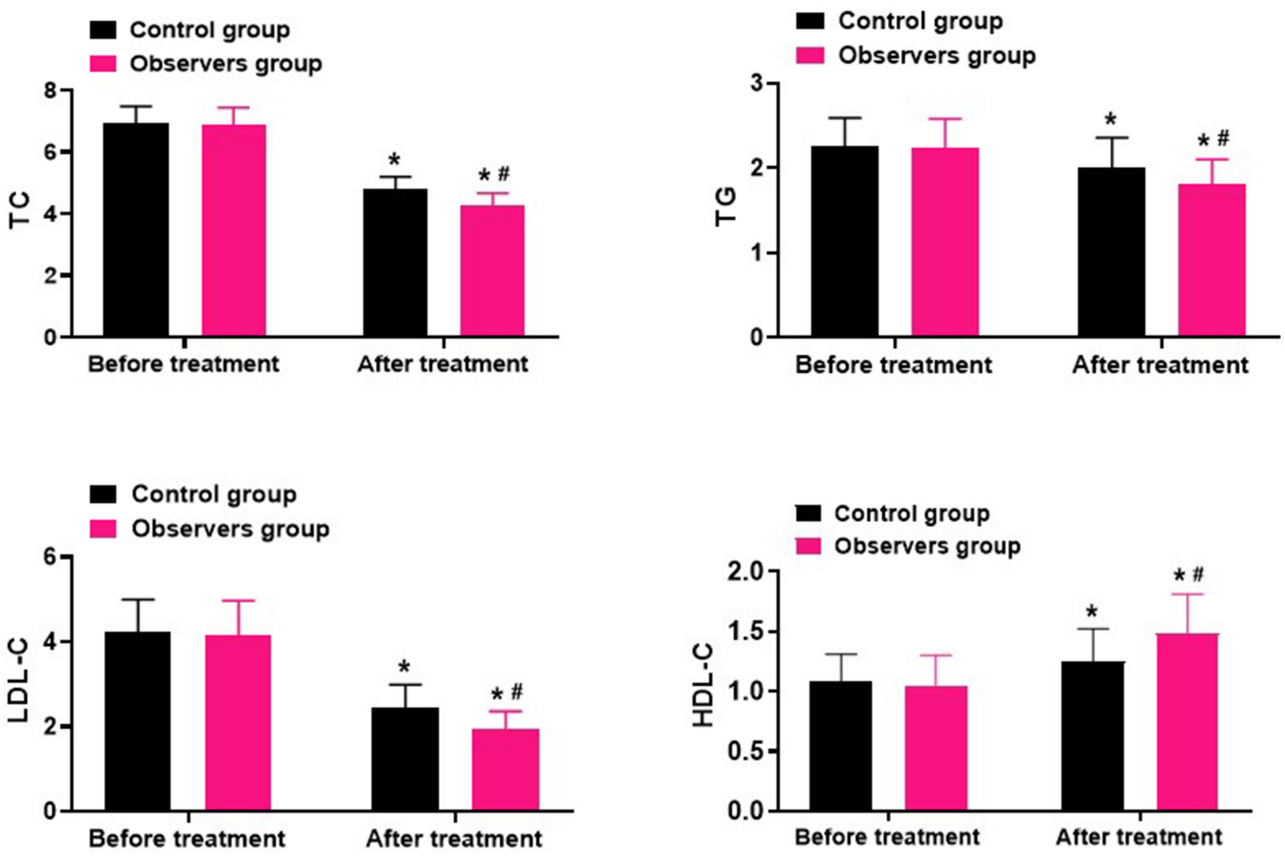

Figure 2 Comparison of Lipid Levels ($\bar{x} \pm s$, mmol/L).
Note: Compared with before treatment, *P<0.05; compared between groups, #P<0.05.

inhibiting platelet aggregation, thereby improving coronary perfusion. These effects collectively mitigate myocardial ischemia-induced anaerobic glycolysis, restoring the equilibrium between myocardial oxygen supply and demand. Prior investigations indicate that co-administration of trimetazidine with statins produces synergistic effects, further amplifying anti-anginal benefits. Recent studies have further confirmed that this combination not only enhances myocardial oxygen utilization but also exhibits additional anti-inflammatory and antioxidative properties, potentially improving endothelial function and mitigating atherosclerosis progression.[22] As a result, the combination of trimetazidine and atorvastatin holds considerable promise in the management of stable angina pectoris in CHD and may emerge as a preferred therapeutic strategy in the future.

The findings of this study demonstrated that the combination therapy of trimetazidine and atorvastatin yielded significantly better outcomes in managing stable angina pectoris in CHD compared to atorvastatin monotherapy. The observation group exhibited a higher total effective rate than the control group (P<0.05), and post-treatment assessments indicated a greater reduction in both the frequency and duration of angina episodes in the observation group (P<0.05). The Visual Analog Scale (VAS) for chest pain severity showed a significant reduction after treatment, with a greater improvement in the observation group compared to the control group, indicating a superior therapeutic effect of the combined treatment (P<0.05). These results highlight the enhanced therapeutic efficacy of the combined regimen in relieving angina symptoms and improving overall patient outcomes. From a lipid metabolism perspective, patients in the observation group experienced more pronounced reductions in TC, TG, and LDL-C levels, alongside an elevation in HDL-C levels compared to the control group (P<0.05). The significant reduction in cholesterol levels in the observation group may be attributed to the synergistic effects of trimetazidine and atorvastatin. While atorvastatin primarily functions by inhibiting cholesterol synthesis, trimetazidine enhances cellular energy metabolism and improves mitochondrial efficiency, thereby reducing oxidative stress and inflammation, which are key contributors to lipid metabolism

https://doi.org/10.2147/IJGM.S515436

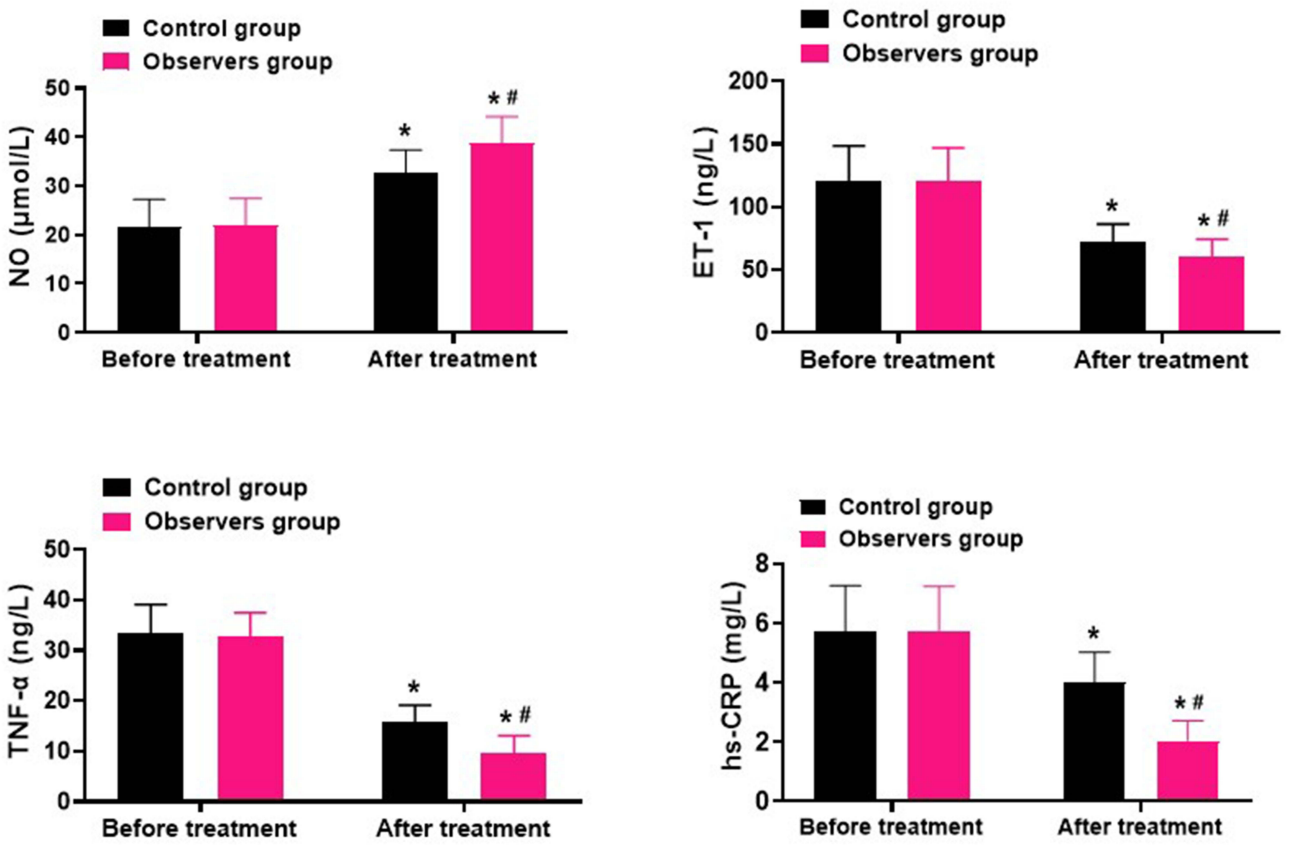

Figure 3 Comparison of Vascular Endothelial Function and Inflammatory Factor Levels ($\bar{x} \pm s$).
Note: Compared with before treatment, *P<0.05; compared between groups, #P<0.05.

disorders.[23] Additionally, trimetazidine has been shown to attenuate lipid peroxidation and enhance endothelial nitric oxide bioavailability, which may contribute to improved lipid homeostasis and atheroprotection.[24] The underlying mechanism may involve atorvastatin's ability to mitigate oxidative stress and protect vascular endothelial function by inhibiting malondialdehyde oxidase activity, thereby promoting plaque stabilization. Concurrently, trimetazidine enhances myocardial energy metabolism, alleviates cardiac workload, and minimizes myocardial cell injury. Through their complementary mechanisms of action, these two drugs exert a synergistic effect, leading to significant improvements in lipid homeostasis and angina relief.

The development of angina is closely linked to the rupture of coronary atherosclerotic plaques and the subsequent inflammatory response triggered by endothelial damage.[25] High-sensitivity C-reactive protein (hs-CRP) serves as a crucial biomarker of systemic inflammation, with elevated levels often reflecting heightened inflammatory activity.[26] Additionally, tumor necrosis factor-alpha (TNF-α) plays a pivotal role in promoting inflammatory responses and cellular necrosis, contributing to vascular endothelial dysfunction and accelerating CHD progression.[27] Vascular endothelial function is also regulated by nitric oxide (NO) and endothelin-1 (ET-1), two key molecules secreted by endothelial cells. NO facilitates vasodilation and inhibits monocyte and platelet adhesion, thereby protecting endothelial integrity,[28] while ET-1 acts as a potent vasoconstrictor, with increased levels often indicating impaired endothelial function.[29] This study demonstrated that post-treatment levels of ET-1, TNF-α, and hs-CRP were significantly lower in the observation group than in the control group, whereas NO levels were higher (P<0.05). These findings suggest that the combined use of trimetazidine and atorvastatin offers superior benefits in reducing vascular inflammation and improving endothelial function in patients with stable angina pectoris. Recent research has also emphasized the potential anti-inflammatory role of trimetazidine, as it has been shown to inhibit NLRP3 inflammasome activation and modulate endothelial cell apoptosis, which may further contribute to its protective effects on vascular function.[30]

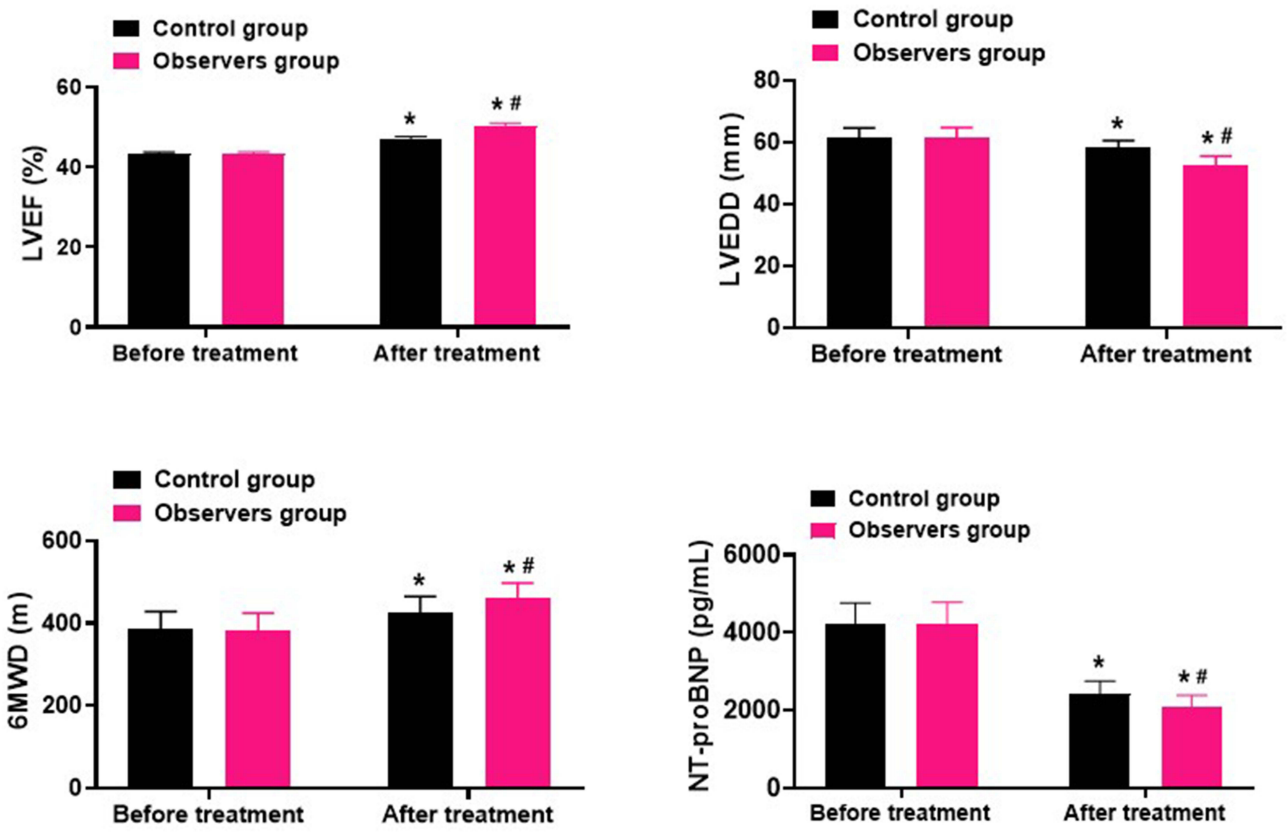

Figure 4 Comparison of Cardiac Function Indicators ($\bar{x} \pm s$).
Note: Compared with before treatment, *P<0.05; compared between groups, #P<0.05.

This study comprehensively assessed cardiac function using multiple parameters. Left ventricular ejection fraction (LVEF) is a critical measure of myocardial contractility, indicating the proportion of blood ejected from the left ventricle per contraction. A decline in LVEF typically reflects impaired ventricular function or weakened myocardial contractility.[31] Left ventricular end-diastolic diameter (LVEDD) is another essential parameter, with an abnormal increase suggesting ventricular remodeling and diminished cardiac output.[32] The six-minute walk distance (6MWD) objectively evaluates a patient's exercise capacity, while N-terminal pro-brain natriuretic peptide (NT-proBNP), a biomarker secreted in response to increased myocardial stress, is widely recognized for its role in diagnosing heart failure and other cardiac dysfunctions.[33] This study found that post-treatment LVEDD and NT-proBNP levels in the observation group were significantly lower than those in the control group, whereas LVEF and 6MWD values were notably higher (P<0.05). These results indicate that the combined use of trimetazidine and atorvastatin leads to a more pronounced improvement in cardiac function among patients with stable angina pectoris. The therapeutic benefits may be attributed to atorvastatin's lipid-lowering effects, which help reduce atherosclerotic plaque formation and enhance myocardial perfusion, thus

Table 3 Comparison of Adverse Reactions [n (%)]

Adverse Reactions	Control (n=50)	Observation (n=50)	x^2	P
Gastrointestinal Reactions	1 (2.0)	2 (4.0)	–	–
Skin Reactions	1 (2.0)	0 (0.0)	–	–
Dizziness and Headache	0 (0.0)	1 (2.0)	–	–
Nausea and Vomiting	0 (0.0)	1 (2.0)	–	–
Sleep Disorders	1 (2.0)	0 (0.0)	–	–
Total Incidence	3 (6.0)	4 (8.0)	0.000	1.000

https://doi.org/10.2147/IJGM.S515436

improving overall heart function. Meanwhile, trimetazidine contributes by modulating intracellular calcium influx, thereby reducing myocardial oxygen demand and alleviating cardiac workload, further strengthening myocardial function. Regarding treatment safety, no significant difference in adverse event incidence was observed between the two groups (P>0.05), confirming the favorable safety profile of the combination therapy. Both drugs exhibit distinct pharmacokinetic advantages, including a short systemic retention time, which minimizes drug accumulation and potential toxicity. Additionally, as they are predominantly metabolized by the liver, their impact on renal function remains minimal, ensuring their suitability for long-term clinical use. Given these advantages, the combined administration of trimetazidine and atorvastatin not only demonstrates superior efficacy over monotherapy but also maintains an excellent safety profile.

Despite these promising outcomes, this study has several limitations that should be acknowledged. Firstly, the relatively small sample size may limit the robustness and generalizability of the findings. Secondly, as a retrospective analysis, inherent biases such as information bias and treatment selection bias cannot be entirely ruled out. Additionally, given that data were sourced from a single medical institution, the external validity of the results may be constrained. Another limitation is the insufficient consideration of individual patient variations, including lifestyle factors and baseline health status, which could potentially influence therapeutic outcomes. To strengthen future research, larger-scale studies with a more rigorous design are necessary. Expanding the sample size, incorporating multi-center data, and adjusting for confounding variables will enhance the reliability and applicability of findings. Addressing these aspects will contribute to refining the study's conclusions, thereby increasing its scientific credibility and clinical relevance.

Conclusion

In patients with stable angina pectoris due to coronary heart disease (CHD), incorporating trimetazidine into atorvastatin therapy yields notable improvements in clinical efficacy. This combination effectively reduces angina symptoms, enhances cardiac performance, optimizes endothelial function, modulates lipid metabolism, and lowers inflammatory markers, all while maintaining a favorable safety profile without increasing adverse reactions.

Data Sharing Statement

All data generated or analysed during this study are included in this published article.

Funding

Research project: Study on the effects of trimetazidine combined with atorvastatin calcium on cardiac function and inflammatory factors in patients with angina pectoris. Contract number:LSFGG-2024035.

Disclosure

The authors declare that they have no competing interests in this work.

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