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ORIGINAL RESEARCH

The Effectiveness and Safety of Cardiac Shock Wave Therapy in the Treatment of Ischemic Heart Disease with Refractory Angina Pectoris

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Background: This study investigated the efficacy and safety profile of cardiac shock wave therapy (CSWT) in the treatment of ischemic heart disease (IHD) with refractory angina pectoris.

Methods: A single-arm, pre-post prospective cohort study was conducted on 65 patients with IHD and refractory angina pectoris who were treated at the Department of Cardiology, 108 Military Central Hospital, between March 2015 and March 2021. The participants were prospectively monitored over a 6-month follow-up period.

Results: Significant improvements were observed in angina symptoms, 6-minute walk test performance, Canadian Cardiovascular Society (CCS) angina classification, and New York Heart Association (NYHA) functional class. N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels decreased from 942.75 pg/mL to 410.45 pg/mL. Echocardiographic parameters demonstrated enhanced cardiac function, with the left ventricular ejection fraction (LVEF) increasing from 43.89 ± 12.27% to 48.48 ± 10.57% ($p < 0.05$). The wall motion septal index (WMSI) decreased from 1.54 ± 0.18 to 1.24 ± 0.12, while global longitudinal strain (GLS) improved from −10.28 ± 2.82 to −12.74 ± 2.42. Myocardial perfusion imaging revealed marked reductions in summed stress score (SSS: 17.45 ± 8.61 vs 12.18 ± 7.89), summed rest score (SRS: 11.09 ± 7.74 vs 9.46 ± 7.23), and summed difference score (SDS: 4.37 ± 2.31 vs 2.57 ± 1.56; all $p < 0.05$). Severe perfusion defects decreased from 46.2% to 12.3% and extensive perfusion defect areas decreased from 60% to 26.2%. No adverse events, including arrhythmias or elevated cardiac enzyme levels, were reported.

Conclusion: Cardiac shock wave therapy is effective in patients with ischemic heart disease and refractory angina, as evidenced by improvements in clinical, functional, and imaging parameters, along with a favourable safety profile.

Keywords: ischemic heart disease, IHD, cardiac shock wave therapy, CSWT, refractory angina pectoris, myocardial perfusion, echocardiography

Introduction

Cardiovascular diseases remain the leading cause of global mortality, accounting for 17.9 million annual deaths and approximately 31% of all deaths worldwide, with coronary artery disease (CAD) and stroke being responsible for 85% of these deaths.[1] Ischemic heart disease (IHD), the predominant manifestation of CAD, is conventionally managed through a triad of therapies including pharmacotherapy, percutaneous coronary intervention (PCI), and coronary artery bypass grafting (CABG). These strategies aim to restore coronary perfusion, alleviate the symptoms, preserve myocardial function, and reduce mortality. However, a significant subset of patients continue to experience refractory angina pectoris despite optimal revascularization. In Europe, 26% of post-PCI patients and 30% of post-CABG patients report persistent angina.[2] Furthermore, revascularization is often precluded in patients with a complex coronary anatomy (eg, diffuse small-vessel disease and severe calcification), advanced age, or prohibitive comorbidities, leaving them reliant on medical therapy with suboptimal symptom control.

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Cardiac shock wave therapy (CSWT) has emerged as a noninvasive therapeutic alternative for this challenging population. First reported for clinical use in 2006, CSWT employs energy acoustic waves to induce localized mechanical and biochemical effects.[3] Mechanistically, shock waves stimulate endothelial nitric oxide (NO) synthase, catalyzing the conversion of L-arginine to NO, stimulating endothelial nitric oxide synthase. This molecule exerts pleiotropic benefits, including vasodilation, inhibition of leukocyte adhesion, smooth muscle proliferation, and attenuation of platelet aggregation. Concurrently, NO enhances the expression of vascular endothelial growth factor (VEGF) and promotes neovascularization.

CSWT has demonstrated safety in clinical studies, with proposed mechanisms of action involving tissue regeneration pathways, including attenuation of adverse cardiac remodelling and modulation of heart failure progression. Further investigations are warranted to elucidate the optimal therapeutic timing and dosage to maximize clinical efficacy.[4] Within the clinical domain, emerging evidence supports CSWT as a noninvasive, well-tolerated intervention for refractory angina, offering significant symptomatic improvement without procedural risks.[5] A case-control study corroborates these findings, highlighting the therapeutic benefits of CSWT and reinforcing its potential role as a viable noninvasive alternative for patients ineligible for conventional revascularization strategies.[6] These outcomes are further validated by multicenter trial data, which underscores the utility and safety of CSWT in managing refractory angina pectoris in patients with contraindications to PCI or CABG.[7]

Despite its theoretical promise, robust clinical data regarding CSWT's long-term efficacy and safety of CSWT for refractory angina remain limited. This study evaluated the 6-month outcomes of CSWT in patients with IHD and refractory angina, focusing on both clinical and subclinical parameters, to inform evidence-based integration of this therapy into contemporary cardiological practice.

Methods

This is a single-arm, pre-post, prospective cohort study that enrolled 65 patients diagnosed with ischemic heart disease and refractory angina pectoris at the Department of Cardiology, 108 Military Central Hospital, between March 2015 and January 2021. The participants underwent a 6-month follow-up to evaluate the effectiveness and safety of cardiac shock wave therapy.

The inclusion criteria were patients with IHD deemed unsuitable for PCI or CABG because of anatomical complexity (eg, diffuse small-vessel disease, severe calcification) or comorbidities, despite optimal medical therapy (OMT) and persistent angina, refractory angina following PCI/CABG despite OMT; Objective evidence of moderate-to-severe myocardial ischemia on single-photon emission computed tomography (SPECT). The exclusion criteria included intracardiac thrombus, active coronary arteritis, acute myocardial infarction within 30 days, malignancy in the thoracic region, suboptimal echocardiographic imaging windows, or patients who did not consent to participate in the study.

Procedure

Ischemic Myocardial Localization

Myocardial ischemia was identified via 12-lead electrocardiography (ECG), transthoracic echocardiography, and SPECT. Regions of interest were mapped using the Medispect system (Protocon GmbH), which integrates imaging data to guide the therapy.

CSWT Administration

Low-energy extracorporeal shockwaves (0.09 mJ/mm^2 energy flux density) were delivered to ischemic zones at 100 shocks per spot, targeting 3–6 spots per session. Treatments were administered three times weekly during the first week of each month for three consecutive months, according to established protocols. Specified that optimal medical therapy (OMT) was based on guideline-directed medical therapy, including maximally tolerated antianginal and heart failure medications per ESC/ACC guidelines at study entry. We clarified that medication regimens were kept stable during the intervention period.

Outcome Measures

Primary endpoints at 6 months included Clinical Parameters: Angina symptoms (assessed by the Canadian Cardiovascular Society [CCS] class), exercise tolerance (6-minute walk test), and dyspnea severity (New York Heart

Association [NYHA] functional class); echocardiography metrics: left ventricular ejection fraction (LVEF, Simpson's method), wall motion septal index (WMSI), and global longitudinal strain (GLS); Myocardial Perfusion Imaging: Summed stress score (SSS), summed rest score (SRS), summed difference score (SDS); and Safety: Serial cardiac enzyme assessments (troponin, CK-MB), and continuous ECG monitoring for arrhythmias. Blinded observers performed CCS angina classification and echocardiographic assessments. Another cardiologist performed shock wave therapy based on the data. Another doctor of the Department of Nuclear Medicine did the SPECT results.

SPECT Technical Details

Myocardial perfusion was evaluated using gated SPECT with technetium-99m sestamibi (Tc-99m MIBI). A two-day protocol included stress and rest imaging. Stress testing was performed either by exercise or pharmacologic agents (dipyridamole or dobutamine) when exercise was not feasible. Imaging used dedicated cardiac cameras (Ventri, GE Healthcare, USA; Nucline Spirit, Mediso, Hungary) with low-energy high-resolution collimators. The 17-segment model was applied for semiquantitative assessment, deriving Summed Stress Score (SSS), Summed Rest Score (SRS), and Summed Difference Score (SDS = SSS − SRS). All procedures followed standardized SPECT protocols for reproducibility. In SPECT myocardial perfusion imaging, lesion severity is typically graded using the summed stress score (SSS) (Normal: SSS ≤ 3; Mild defect: SSS = 4–8; Moderate defect: SSS = 9–12; Severe defect: SSS > 12).

Extracorporeal Shock Wave Therapy Protocol

Low-intensity cardiac shock wave treatment was performed using the Cardiospect device (Medispect Inc., USA; manufactured 2012) equipped with an electromagnetic shock wave generator. Real-time ultrasound guidance was provided by a Vivid 7 system (GE Healthcare, USA; 2006) with a 2.5–3.5 MHz cardiac probe. Target regions were predefined based on perfusion or wall-motion abnormalities, following a standardized segmentation scheme. Each treatment consisted of 100 focused shock waves per target point, with 3–6 target points per session (total 300–600 shots), delivered at an energy flux density (EFD) of 0.09 mJ/mm^2. When multiple targets were not aligned within the same echocardiographic plane, probe orientation was adjusted according to the treatment plan to localize subsequent sites. Treatment was repeated over nine sessions during three consecutive weeks. The device calibration was routinely verified before each session to ensure consistent EFD output. Continuous ECG monitoring was applied throughout the procedure, and shock delivery was synchronized to the R-wave to prevent arrhythmic events. Patient motion was minimized to avoid signal loss or mistimed pulse emission.

Statistical Analysis

Data were analyzed using SPSS software (version 20.0), paired t-tests for continuous variables, and chi-square tests for categorical variables, with significance set at p-value < 0.05. The results are presented as mean ± standard deviation and percentages, as appropriate.

Results

Baseline Patient Characteristics

The study included 65 patients (mean age 72.2 ± 10.1 years; 78.5% male). The baseline demographic and clinical characteristics of the patients are summarized in Table 1. Most patients (72.3%) had undergone prior revascularization. The most prevalent comorbidities included hypertension (95.4%), dyslipidemia (93.8%), advanced age (≥65 years: 84.6%), and diabetes mellitus (36.9%). Coronary angiography (digital subtraction angiography, DSA) revealed double-vessel disease in 33.8% and triple-vessel disease in 21.5% of patients (Table 2).

Clinical Outcomes

Significant symptom improvement was observed following cardiac shock wave therapy (Table 3 and Figures 1, 2). Post-treatment reductions in angina frequency, sublingual nitrate consumption, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels (942.75 pg/mL vs 410.45 pg/mL; p < 0.05) were noted. Exercise tolerance, assessed using the

Table 1 Baseline Patient Characteristics

Characteristics			Percentage (n%)
Gender	Male		51(78.5%)
	Female		14 (21.5%)
Average age (year)			72.2 ± 10.1
Revascularization	No		18 (27.7%)
	Yes	PCI	40 (61.5%)
		CABG	5 (7.7%)
		PCI + CABG	2 (3.1%)
BMI (kg/m^2) ≥ 25			27 (41.5%)
Age ≥ 65			55 (84.6%)
Diabetes			24 (36.9%)
Hypertension			62 (95.4%)
Smokers			44 (67.7%)
Dyslipidemia			61 (93.8%)

Table 2 Coronary Artery Lesions on DSA

Artery Numbers	Number of Patients (n = 65)	Ratio (%)
Single lesion	29	44.62
Double lesions	22	33.84
Three lesions	14	21.54

Table 3 Changes in Clinical and Subclinical Symptoms

Symptom	Before Treatment (X ± SD)	After Treatment (X ± SD)	p Value
Angina pectoris/week (times)	6.26 ± 2.59	0.58 ± 0.56	< 0.001
Nitrate used/week (tablets)	6.34 ± 2.62	0.60 ± 0.55	< 0.001
6-minute walk test (m)	280.8 ±71.1	388.6 ± 55.4	< 0.001
NT-proBNP (pg/mL)	942.75 ± 1618.37	410.45 ± 461.54	< 0.05

6-minute walk test, increased markedly ($p < 0.05$). Additionally, heart failure exacerbations (NYHA class III/IV) declined from 15.39% to 1.94% (p-value < 0.05; Figure 2).

Echocardiographic Parameters

Echocardiographic metrics demonstrated selective improvement (Table 4). While the left ventricular end-diastolic diameter (LVEDD) and baseline ejection fraction (EF) remained unchanged, the EF quantified by Simpson's method increased significantly (43.89 ± 12.27% vs 48.48 ± 10.57%; $p < 0.05$). Simpson's biplane method was used for primary

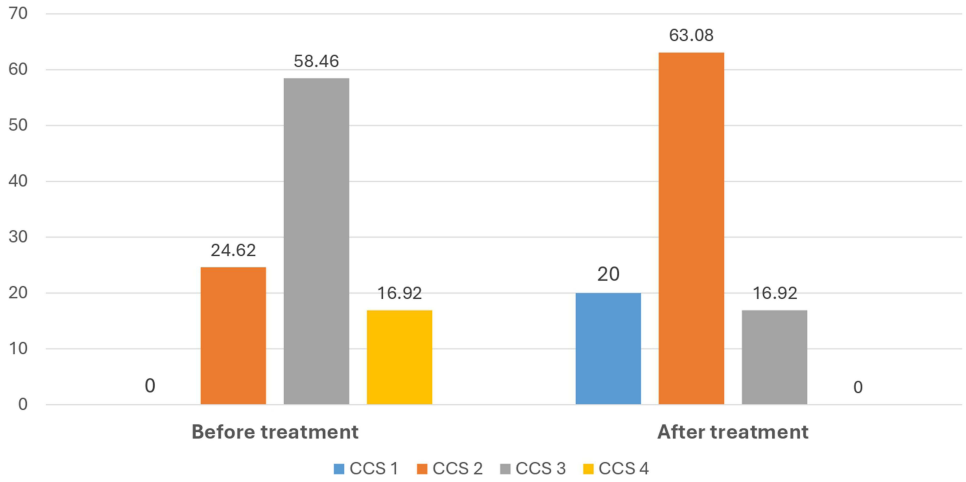

Figure 1 CCS before and after treatment.

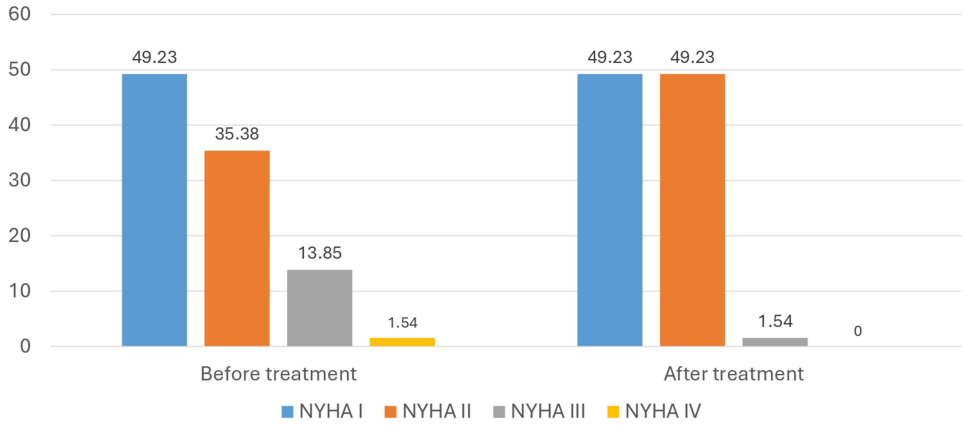

Figure 2 Heart failure rates before and after treatment.

EF results; reported M-mode for completeness. Regional wall motion abnormalities improved, as evidenced by reduced wall motion score index (WMSI: 1.54 ± 0.18 vs 1.24 ± 0.12; p-value < 0.05). Global longitudinal strain (GLS) also improved, shifting from −10.28 ± 2.82 to −12.74 ± 2.42 (p-value < 0.05).

Table 4 Comparison of Echocardiography Parameters Before and After Treatment

Parameters	Before Treatment	After Treatment	p Value
Dd (mm)	50.26 ± 8.60	48.71 ± 6.39	> 0.05
Ds (mm)	35.32 ± 10.24	33.03 ± 8.08	> 0.05
FS (%)	30.34 ± 10.67	32.48 ± 8.75	> 0.05
EF (%)	55.23 ± 15.75	58.78 ± 12.85	> 0.05
EF sympson's (%)	43.89 ± 12.27	48.48 ± 10.57	< 0.05
WMSI	1.54 ± 0.18	1.24 ± 0.12	< 0.001
GLS (-%)	−10.28 ± 2.82	−12.74 ± 2.42	< 0.001

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Table 5 Imaging Characteristics of Myocardial Perfusion

Characteristics of SPECT		Before Treatment (n, %)	After Treatment (n, %)	p Value
Severity of perfusion defect	Mild	3 (4.6)	26 (40.0)	p < 0.001
	Moderate	32 (49.2)	31 (47.7)	p < 0.001
	Severe	30 (46.2)	8 (12.3)	p < 0.001
Extent of perfusion defect	Narrow	2 (3.1)	23 (35.4)	p < 0.001
	Moderate	24 (36.9)	25 (38.5)	p < 0.001
	Large	39 (60.0)	17 (26.2)	p < 0.001

Myocardial Perfusion Imaging

SPECT imaging revealed substantial perfusion enhancement post-CSWT (Table 5). The proportion of patients with severe perfusion defects decreased from 46.2% to 12.3%, whereas extensive defect areas decreased from 60.0% to 26.2% (both $p < 0.05$). Figure 3 shows a case of SPECT before and after treatment with increased perfusion in both phases of SPECT. Quantitative perfusion analysis (Table 6) showed reductions in summed stress score (SSS: 17.45 ± 8.61 vs 12.18 ± 7.89), summed rest score (SRS: 11.09 ± 7.74 vs 9.46 ± 7.23), and summed difference score (SDS: 4.37 ± 2.31 vs 2.57 ± 1.56; all p-values < 0.05).

Safety Profile

No procedural complications, arrhythmias, or elevations in cardiac enzymes (troponin or CK-MB) were observed during the study period.

Discussion

Cardiovascular risk factors exhibit robust correlations with cardiovascular morbidity and mortality, necessitating systematic evaluation and mitigation to optimize risk stratification, therapeutic management, and preventive strategies in ischemic heart disease (IHD). In our cohort, the severity of myocardial perfusion defects, characterized by diffuse multivessel atherosclerosis and prior myocardial infarction (MI) in 46% of the patients, reflected an advanced ischemic burden. Following cardiac shock wave therapy (CSWT), angina frequency and duration decreased progressively, with

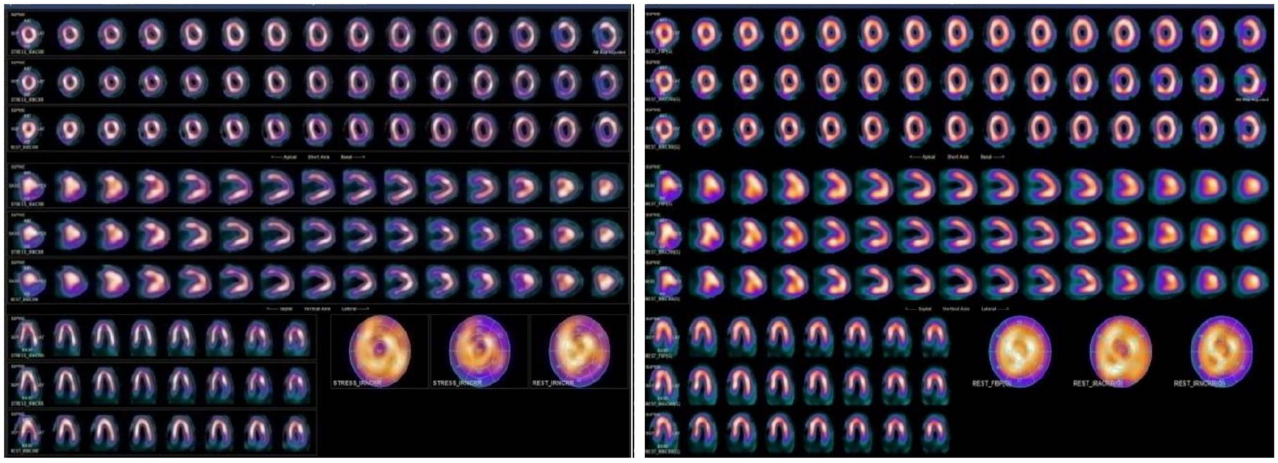

Figure 3 SPECT image before and after treatment.

Table 6 Comparison of the Results of SPECT Before and After Treatment

Parameters	Before Treatment (X± SD)	After Treatment (X ± SD)	p Value
Summed stress score of perfusion (SSS)	17.45 ± 8.61	12.18 ± 7.89	< 0.05
Summed rest score of perfusion (SRS)	11.09 ± 7.74	9.46 ± 7.23	< 0.05
Summed difference score between two phases (SDS)	4.37 ± 2.31	2.57 ± 1.56	< 0.05

clinically meaningful reductions evident within one month ($p < 0.05$) and near resolution in the majority of patients by six months. Notably, individuals with a higher baseline angina burden experienced proportionally greater reductions, underscoring CSWT's efficacy of CSWT for severe myocardial ischemia. Mechanistically, CSWT induces nitric oxide (NO)-mediated vasodilation via endothelial NO synthase activation, enhancing coronary microvascular perfusion and ameliorating the supply-demand mismatch. These findings were consistent with those reported by Vainer et al, who reported a 60% reduction in weekly angina episodes post-CSWT,[8] and Faber et al demonstrated angina resolution in 88% of patients at three months.[9]

Exercise tolerance, quantified by the 6-minute walk distance, improved significantly post-CSWT ($p < 0.05$), which was attributable to enhanced myocardial perfusion and collateralization. While patients with moderate ischemia achieved functional gains within three months, those with extensive multivessel disease required six months for maximal benefit, suggesting time-dependent angiogenesis mediated by vascular endothelial growth factor upregulation. This aligns with the findings of Wang et al, who observed sustained improvements in exercise capacity correlated with perfusion recovery.[10] Serial assessment of NT-proBNP revealed significant post-treatment reductions (942.75 ± 1618.37 pg/mL vs 410.45 ± 461.54 pg/mL; p-value < 0.05), consistent with improved hemodynamic profiles. Takakuwa et al corroborated these findings, demonstrating that NT-proBNP levels decline in non-revascularizable patients post-CSWT,[2] further supporting its role in mitigating ventricular strain. Collectively, these data underscore CSWT as a viable therapeutic modality for refractory angina, addressing both the symptomatic burden and the pathophysiological underpinnings of myocardial ischemia.

Echocardiographic parameters showed mixed trends. The left ventricular end-diastolic dimension (Dd) and ejection fraction (EF) by conventional methods showed nonsignificant changes; EF quantified via Simpson's biplane method increased from 43.89 ± 12.27% to 48.48 ± 10.57% post-intervention ($p < 0.05$). This aligns with Wang et al, who noted EF improvement from 45.02 ± 6.37% to 48.7 ± 10.53% ($p < 0.05$),[10] and Celutkiene et al, who observed a rise in Simpson's EF from 46.5 ± 10.6% to 49.8 ± 8.6% ($p < 0.05$).[11] The limited sample size and follow-up duration in the present study may account for nonsignificant ventricular remodelling trends, although Simpson's method proved superior to Teicholz's for EF assessment, reinforcing its utility in the clinical evaluation of IHD patients. The therapeutic benefits of advanced echocardiographic techniques have been further elucidated. Post-CSWT, the wall motion score index (WMSI) decreased from 1.54 ± 0.18 to 1.24 ± 0.12 ($p < 0.001$), while global longitudinal strain (GLS) improved from −10.28 ± 2.82 to −12.74 ± 2.42 ($p < 0.001$), suggesting enhanced myocardial contractility and perfusion. These findings mirror those of Faber et al, who documented GLS amelioration from −14 ± 5 to −17 ± 6 ($p = 0.04$) in patients with refractory angina.[9]

The present study demonstrated a statistically significant reduction in post-treatment summed stress score (SSS) compared to baseline measurements (17.45 ± 8.61 vs 12.18 ± 7.89; p-value < 0.05). Similarly, the summed rest score (SRS) declined following extracorporeal cardiac shock wave therapy (CSWT), decreasing from 11.09 ± 7.74 to 9.46 ± 7.23. A marked improvement was observed in the summed difference score (SDS), which decreased from 4.37 ± 2.31 to 2.57 ± 1.56 (p-value < 0.05). These findings correlate with enhanced myocardial perfusion on single-photon emission computed tomography (SPECT), indicating mitigation of perfusion defects.

These results align with those of prior investigations, including a study by Prasad et al, involving 111 patients with refractory angina secondary to ischemic heart disease (IHD). Post-intervention analysis revealed reductions in SSS and SDS, with 60% of the participants demonstrating improvements in both the SSS and SDS indices.[12] Notably, SPECT imaging in patients with IHD delineates reversible perfusion defects, the severity of which correlates with the magnitude

of coronary stenosis and the extent of multivessel involvement. Although fixed defects (irreversible scar tissue secondary to myocardial infarction) are characterized by persistent hypoperfusion and akinetic or dyskinetic wall motion, peri-infarct ischemic regions may exhibit partial reversibility. CSWT application to these ischemic zones appears to augment perfusion, potentially via mechanotransductive modulation of angiogenesis.

The safety outcomes in our study revealed no instances of procedural or postprocedural arrhythmias, pericardial effusion, acute tamponade, myocardial infarction, intracardiac thrombosis, or mortality. Vital parameters, including the heart rate and blood pressure, remained stable Furthermore, no patients required rehospitalization for heart failure, acute coronary syndrome, or repeat revascularization (PCI/CABG). These observations are consistent with those of Takakuwa et al, who reported no significant alterations in troponin T-I (0.023 vs 0.029 ng/mL; $p = 0.32$) or pro-BNP levels (546 vs 897 pg/mL; $p = 0.64$) following CSWT in a cohort of 10 Japanese patients with persistent angina, including those with prior revascularization.[2]

A meta-analysis in 2022 reviewed data from randomized controlled trials and found that CSWT significantly improved summed stress scores (WMD = −3.76, 95% CI −6.15 to −1.37; $p = 0.002$), supporting its effect on myocardial perfusion, albeit with moderate heterogeneity. This analysis concluded that CSWT provided moderate improvement in myocardial perfusion and cardiac function, especially in chronic stable or refractory angina.[13] A recent trial by Zhang et al confirmed that CSWT significantly improved myocardial perfusion and exercise tolerance in patients with coronary heart disease when combined with standard rehabilitation therapy, without significant adverse effects.[14] The mechanism includes microvascular angiogenesis, improved endothelial function, and upregulation of cytokines promoting neovascularization in ischemic myocardium. The 2024 ESC Guidelines for chronic coronary syndromes recognize shock wave therapy as an emerging noninvasive strategy for symptom relief in refractory angina not suitable for revascularization, with a Class IIb, Level of Evidence B recommendation, underscoring its potential while emphasizing the need for larger controlled studies to validate long-term clinical efficacy.[15]

Limitations

Limitations include a single-center design and a modest sample size. This is a single-arm, pre-post prospective cohort that that have lacks of a parallel control group and short follow-up may introduce biases. The future multicenter randomized trials with extended follow-up are warranted to validate the long-term outcomes.

Conclusion

Cardiac shock wave therapy is effective in patients with ischemic heart disease and refractory angina, as evidenced by improvements in clinical, functional, and imaging parameters, along with a favourable safety profile.

Data Sharing Statement

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The study was approved by the Institutional Review Board of 108 Military Central Hospital (No. 424/QĐ-VNC dated Nov 8, 2018). This study adhered to the principles of the Declaration of Helsinki for Biomedical Research.

Consent for Publication

All the authors have read and agreed to the published version of the manuscript.

Author Contributions

All authors made a significant contribution to the work reported, whether in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas. All authors took part in drafting, revising, and approving the manuscript and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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