

# Clinical Efficacy and Mechanism of Qinjin Tablet in Treating Unstable Angina with Phlegm-Stasis Syndrome Based on the HDL-C/MMP-9 Signaling Axis

Yuan WANG<sup>1</sup>, Jingru GAO<sup>2</sup>, Hongjun ZHU<sup>3\*</sup>

1. Nanjing University of Chinese Medicine, Nanjing 210000, China; 2. Jiangxi Street Community Health Service Center, Xinwu District, Wuxi 214000, China; 3. Wuxi Hospital Affiliated to Nanjing University of Chinese Medicine, Wuxi 214000, China

**Abstract** [Objectives] To explore the clinical efficacy of Qinjin Tablet in treating unstable angina with phlegm-stasis syndrome and preliminarily explore its intervention mechanism. [Methods] Sixty unstable angina patients with phlegm-stasis syndrome were randomly divided into a treatment group and a control group (30 each). The control group received standardized Western medicine treatment, while the treatment group also took Qinjin Tablet. After a 4-week treatment, comparisons were made on improvement of angina symptoms, TCM syndrome scores, blood stasis scores, reduction/cessation of short-acting anti-angina drugs, Seattle Angina Questionnaire (SAQ) scores, blood lipid levels, and matrix metalloproteinase-9 (MMP-9) concentrations between the two groups. [Results] No statistical differences existed in baseline data like age, gender, and disease duration between the two groups ( $P > 0.05$ ). The treatment group showed significantly better efficacy than the control group in improving angina symptoms, TCM syndrome scores, blood stasis scores, and reduction or discontinuation of short-acting anti-anginal drugs ( $P < 0.05$ ). The SAQ score increase was more significant in the treatment group ( $P < 0.05$ ). The control group significantly reduced low-density lipoprotein cholesterol (LDL-C) ( $P < 0.05$ ), while the treatment group markedly increased high-density lipoprotein cholesterol (HDL-C) ( $P < 0.05$ ). Post-treatment, the treatment group had a marked reduction in serum MMP-9 ( $P < 0.05$ ), with no significant change in the control group. [Conclusions] Qinjin Tablet can significantly alleviate clinical symptoms and improve quality of life in UA patients by modulating the HDL-C/MMP-9 signaling pathway.

**Key words** Qinjin Tablet, Unstable angina, Phlegm-stasis interlocking syndrome, High-density lipoprotein cholesterol, Matrix metalloproteinase-9

## 1 Introduction

Every year, approximately 17.9 million people die from cardiovascular diseases globally, with coronary heart disease (CHD) being the leading cause of death<sup>[1]</sup>. As a severe clinical subtype of CHD, the incidence and mortality of unstable angina (UA) have been continuously rising in developing countries in recent years, increasing the socio-economic burden<sup>[2]</sup>. In view of this, effective treatment of UA is crucial to alleviating pressure on the public health system<sup>[3]</sup>. However, although the treatment strategies recommended by current clinical practice guidelines can alleviate the risk of major adverse cardiovascular events, issues such as drug resistance and in-stent restenosis still severely limit their efficacy<sup>[4–5]</sup>. Studies have shown that traditional Chinese medicine (TCM) compound formulas, relying on their overall regulatory advantages of "multi-component, multi-target, multi-pathway", demonstrate unique and significant efficacy and value in the prevention and treatment of UA<sup>[6–7]</sup>. Therefore, based on TCM's Five Elements theory and years of clinical experience, Professor Zhu Hongjun summarized the TCM empirical formula Qinjin Tablet for treating UA, which shows remarkable efficacy. It can not only

quickly relieve the clinical symptoms of UA patients but also reverse atherosclerotic (AS) plaques<sup>[8]</sup> though its intervention mechanism needs further clarification. For this purpose, this study employed a randomized controlled trial (RCT), starting from the perspective of "lipid metabolism-inflammatory regulation", to explore the clinical efficacy and mechanism of Qinjin Tablet in treating UA by regulating the HDL-C/MMP-9 signaling axis.

## 2 Materials and methods

**2.1 Case source** Sixty patients with unstable angina of phlegm-stasis syndrome who visited the Department of Cardiology, Wuxi Hospital Affiliated to Nanjing University of Chinese Medicine from October 2021 to January 2023 were screened and randomly divided into a treatment group and a control group using a random number table, with 30 cases in each group. This study was approved by the Ethics Committee of Wuxi Hospital of Traditional Chinese Medicine [Ethics Review Approval: YJS2022041803].

### 2.2 Diagnostic criteria

**2.2.1 Western Medicine Diagnostic Criteria.** The Western medicine diagnosis met the UA diagnostic criteria in the *Guidelines for the Diagnosis and Treatment of Non-ST-Segment Elevation Acute Coronary Syndromes (2016 Edition)*<sup>[9]</sup>: (i) Clinical manifestations; rest angina; chest pain lasting  $\geq 20$  min, not relieved by rest or sublingual nitroglycerin; new-onset angina; angina severity classification III or IV (can be induced by mild daily activities); worsening angina; significant deterioration in frequency, duration, or triggering threshold compared to previous stable angina. (ii) Electrocardiogram (ECG) manifestations; typical features include

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Yuan WANG, master's degree candidate. \* Corresponding author. Hongjun ZHU, chief physician of Traditional Chinese Medicine, doctoral supervisor.

ST-segment depression, transient ST-segment elevation, or T-wave changes. (iii) Myocardial injury markers: cardiac troponin (cTn) normal or mildly elevated, creatine kinase-MB (CK-MB) usually normal. Meeting any one of the above clinical manifestations, with or without ECG changes, can diagnose unstable angina.

**2.2.2 TCM Syndrome Diagnostic Criteria.** The TCM syndrome diagnosis met the relevant criteria for phlegm-stasis syndrome in *Internal Medicine of Traditional Chinese Medicine*<sup>[10]</sup> and *Guidelines for Clinical Research of New Chinese Medicines*<sup>[11]</sup>: Main symptoms: chest tightness with suffocating pain; or pain radiating to the shoulder and back. Secondary symptoms: obesity; profuse phlegm; loss of appetite and nausea; heavy body and fatigue; sticky mouth. Tongue and pulse: tongue pale or dark, coating white and greasy; pulse wiry and slippery. Syndrome diagnosis: 1–2 main symptoms plus 2–3 secondary symptoms, referring to the tongue and pulse, can confirm the diagnosis.

**2.3 Inclusion criteria** Meeting the above Western and TCM diagnostic criteria; age 18–82 years; good physical condition, stable vital signs; informed consent, voluntarily signing the informed consent form.

**2.4 Exclusion criteria** Not meeting the inclusion criteria; Having arrhythmias, other heart diseases (myocarditis, myocardial infarction, etc.), cerebral infarction, pulmonary insufficiency, or other acute and severe diseases; having severe primary diseases of the liver, kidney, or hematopoietic system; women planning pregnancy, pregnant, lactating, or allergic to known components of the study drug; patients with malignant diseases or unsuitable for participating in this trial; patients with severe psychiatric disorders; those who have participated in or are participating in other clinical trials within the last three months; Patients with low cooperation.

### 2.5 Criteria for exclusion, dropout, and trial termination

**2.5.1** Decision by the researcher to withdraw. Occurrence of allergic reactions or serious adverse events requiring trial cessation; during the trial, the patient develops other complications and special physiological changes, making it inappropriate to continue the trial; poor patient compliance or refusal of treatment.

**2.5.2** Voluntary withdrawal by the subject. For any reason, the patient is unwilling or unable to continue the clinical trial, requests withdrawal from the trial from the supervising physician; the subject, although not explicitly withdrawing, stops taking the medication and is lost to follow-up.

**2.6 Treatment methods** Standardized Western medicine treatment: Following the *Guidelines for the Diagnosis and Treatment of Non-ST-Segment Elevation Acute Coronary Syndromes (2016 Edition)*<sup>[9]</sup>, both groups received standardized Western medicine treatment, covering anti-myocardial ischemia, antiplatelet aggregation, vasodilation, lipid metabolism regulation, etc. Medications included: Aspirin Enteric-coated Tablets (Bayer Healthcare, Germany) 100 mg QD, Clopidogrel Hydrogen Sulfate Tablets (Lepu Pharmaceutical Co., Ltd.) 75 mg QD, Atorvastatin Calcium Tablets (Zhejiang Lepu Pharmaceutical Co., Ltd.)

10 mg QN, Isosorbide Mononitrate Dispersible Tablets (Shandong Fangming Pharmaceutical Group Co., Ltd.) 20 mg BID, Metoprolol Succinate Sustained-Release Tablets (Nantong United Pharmaceutical Co., Ltd.) 23.75 mg QD, etc. Patients with underlying conditions like hyperlipidemia, hypertension, and diabetes were also given corresponding glucose-lowering, lipid-lowering, and antihypertensive drugs to comprehensively control the condition and reduce cardiovascular event risk.

The treatment group received Qinjin Tablet in addition to standardized Western medicine treatment. Qinjin Tablet formula: Huangqi (Astragalus Root) 30 g; Huanglian (Coptis Root) 3 g; Qingbanxia (Pinellia Tuber) 10 g; Quanguailou (Snakegourd Fruit) 10 g; Zhebeimu (Thunberg Fritillary Bulb) 10 g; Hehuanpi (Silktree Albizia Bark) 30 g; Jiaoshanzha (Charred Hawthorn Fruit) 30 g; Haizao (Seaweed) 10 g; Jinei jin (Gallus Gallus Domesticus) 10 g; Muli (Oyster Shell) 10 g; Guijia (Tortoise Carapace) 10 g; Tubiechong (Ground Beetle) 10 g; Tangshuizhi (Scalded Leech) 3 g; Haijinsha (Climbing Fern Spores) 10 g; Jinqiancao (Longhairy Antenoron Herb) 10 g; and Danshen (Salvia Root) 30 g. The decoction was uniformly prepared by the pharmacy of Wuxi Hospital Affiliated to Nanjing University of Chinese Medicine, concentrated to 200 mL, packaged into 100 mL/bag, taken warm, one bag each morning and evening after meals.

### 2.7 Observation indicators and evaluation criteria

**2.7.1** Efficacy indicators and evaluation criteria. (i) Degree of improvement in angina symptoms: using the Canadian Cardiovascular Society (CCS) Angina Severity Classification. Evaluation criteria: Markedly effective: after treatment, angina symptom grade decreased by two levels; or grade I, II symptoms basically disappeared; effective: after treatment, angina symptom grade decreased by one level; or grade I symptoms basically disappeared; ineffective: after treatment, no significant change in angina symptoms; worsened: after treatment, frequency, severity, and duration of angina attacks increased, or angina symptom grade increased. Total effective rate = (Markedly effective + Effective)/Total cases × 100%

(ii) TCM syndrome score and blood stasis syndrome quantitative score: Referenced the 13<sup>th</sup> Five-Year Plan edition of *Internal Medicine of Traditional Chinese Medicine*<sup>[10]</sup> and *Guidelines for Clinical Research of New Chinese Medicines*<sup>[11]</sup>. Evaluation criteria: markedly effective: clinical symptoms and signs significantly improved,  $N \geq 70\%$ ; effective: clinical symptoms and signs improved,  $30\% \leq N < 70\%$ ; ineffective: no significant improvement in clinical symptoms and signs,  $0\% \leq N < 30\%$ ; worsened: clinical symptoms and signs worsened,  $N < 0$ ; TCM efficacy index ( $N$ ) = (Pre-treatment score – Post-treatment score)/Pre-treatment score × 100%; Total effective rate = (Markedly effective + Effective)/Total cases × 100%.

(iii) Reduction or discontinuation of short-acting anti-anginal drugs: markedly effective: drug usage frequency significantly reduced, dosage significantly reduced, or able to stop medication;

effective: drug usage frequency reduced, dosage reduced; ineffective: drug usage frequency and dosage unchanged; worsened: drug usage frequency increased compared to before, dosage increased, or other anti-angina drugs added; Total effective rate = (Markedly effective + Effective)/Total cases  $\times$  100%.

**2.7.2 Quality of life assessment.** Evaluated using the Seattle Angina Questionnaire (SAQ) score across multiple dimensions, including Physical Limitation (PL), Angina Stability (AS), Angina Frequency (AF), Treatment Satisfaction (TS), and Disease Perception (DP). Each item was scored 1, 2, 3, 4, 5, or 6 based on severity. The score changes in the five dimensions before and after treatment were compared between the two groups. Standard score = (Actual score - Dimension minimum score)/(Dimension maximum score - Dimension minimum score)  $\times$  100%. Higher scores indicate better quality of life and physical function.

**2.7.3 Clinical indicators.** Blood lipid levels: serum triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) concentrations, tested by the laboratory department of Wuxi Hospital Affiliated to Nanjing University of Chinese Medicine. Mechanism indicator: Serum matrix metalloproteinase-9 (MMP-9) concentration. Fasting morning blood (3 mL) was collected from enrolled patients before and after treatment, centrifuged at 3 000 r/min for 10 min to separate serum. Serum MMP-9 concentration was detected using the ELISA method according to the kit instructions (Wuhan Elabscience Biotechnology Co., Ltd.).

**2.8 Safety indicators** Included biochemical indicators such as liver and kidney function, electrolytes, and complete blood count, as well as possible adverse reactions such as gastrointestinal reactions, cardiovascular adverse reactions, liver toxicity, allergic reactions, and general adverse reactions.

**2.9 Statistical processing** SPSS 20.0 statistical software was used. Measurement data were expressed as ( $\bar{X} \pm S$ ). When data followed a normal distribution, paired sample *t*-test was used within groups, and independent sample *t*-test or corrected *t*-test was used between groups. If data were skewed, they were expressed as M (P25, P75), using the independent sample rank-sum test between groups and the paired sample rank-sum test within groups. Count data and categorical data were expressed as "n, %". Ranked data were analyzed using the chi-square test or rank-sum test. *P* < 0.05 was considered statistically significant.

### 3 Results and analysis

**3.1 Comparison of baseline data and case completion** This study included 60 valid cases, randomly divided into a treatment group and a control group. The treatment group had 30 cases, 17 males and 13 females, aged 49 - 82 years, with an average age of (69.9  $\pm$  10.78) years. The control group had 30 cases, 20 males and 10 females, aged 43 - 81 years, with an average age of (65.0  $\pm$  7.75) years. There were no statistically significant differences in baseline data such as gender composition, age, disease duration distribution, smoking history, drinking history, and angina severity classification between the two groups (*P* > 0.05), indicating comparability. During the treatment period, indicators such as blood routine, liver and kidney function were normal in both groups without abnormal changes, and there were no drug-related adverse events or dropouts.

**3.2 Clinical efficacy analysis** The total effective rates for improvement of angina symptoms, TCM syndrome scores, blood stasis scores, and reduction or discontinuation of short-acting anti-anginal drugs were all better in the treatment group than in the control group (Table 1).

**Table 1 Comparison of angina symptom improvement, TCM syndrome scores, blood stasis scores, and short-acting anti-angina drug reduction/cessation between the two groups (n = 30)**

Category	Group	Markedly effective n (%)	Effective n (%)	Ineffective n (%)	Worsened n (%)	Total effective rate//%
Angina symptom improvement	Treatment	11 (36.7)	17 (56.6)	2 (6.7)	0 (0)	93.3 *
	Control	5 (16.7)	20 (66.6)	5 (16.7)	0 (0)	83.3
TCM syndrome scores	Treatment	24 (80)	6 (20)	0 (0)	0 (0)	100 *
	Control	4 (13.3)	14 (46.7)	12 (40)	0 (0)	60
Blood stasis scores	Treatment	26 (86.7)	4 (13.3)	0 (0)	0 (0)	100 *
	Control	1 (3.3)	18 (60)	11 (36.7)	0 (0)	63.3
Short-acting drug reduction	Treatment	15 (50)	14 (46.7)	1 (3.3)	0 (0)	96.7 *
	Control	5 (16.7)	22 (73.3)	3 (10)	0 (0)	90

**NOTE** Compared with the control group, \* *P* < 0.05.

**3.3 SAQ score comparison** Inter-group comparison: before treatment, there were no statistically significant differences in various scores between the two groups (*P* > 0.05), indicating comparability; after treatment, the differences in various scores between the two groups were statistically significant (*P* < 0.05). Intra-group comparison: After treatment, scores in both groups increased, with the treatment group showing more significant improvement (*P* < 0.05), as shown in Table 2.

### 3.4 Clinical indicator comparison

**3.4.1 Blood lipid level comparison.** Before treatment, there were no statistically significant differences in various indicators between the groups (*P* > 0.05), indicating comparability. After treatment, no significant changes were observed in serum TG and TC levels in either group (*P* > 0.05); the control group showed a decrease in serum LDL-C levels, and the treatment group showed an increase in serum LDL-C levels, both differences being statisti-

**Table 2 Comparison of SAQ dimension scores before and after treatment between the two groups** [ $n=30$ , score, M (P25, P75)]

Category	Group	Before treatment	After treatment
PL	Treatment	22.2 (20.0, 24.4)	51.1 (48.9, 53.3) * $\Delta$
	Control	22.2 (22.2, 25.0)	46.7 (44.4, 48.9) $\Delta$
AS	Treatment	20.0 (0, 20.0)	80.0 (80.0, 80.0) * $\Delta$
	Control	20.0 (0, 20.0)	60.0 (60.0, 60.0) $\Delta$
AF	Treatment	40.0 (40.0, 40.0)	60.0 (60.0, 90.0) * $\Delta$
	Control	40.0 (40.0, 40.0)	60.0 (60.0, 80.0) $\Delta$
TS	Treatment	17.5 (15, 25)	60.0 (60.0, 65.0) * $\Delta$
	Control	20.0 (15, 25)	55.0 (50.0, 60.0) $\Delta$
DP	Treatment	20.0 (13.3, 20)	46.7 (46.7, 48.35) * $\Delta$
	Control	20.0 (20, 20)	40.0 (33.3, 46.7) $\Delta$

**NOTE** Compared with the control group, \* $P < 0.05$ ; compared with before treatment within the group,  $\Delta P < 0.05$ .

cally significant ( $P < 0.05$ ), as indicated in Table 3.

**Table 3 Comparison of blood lipid levels before and after treatment between the two groups** ( $n=30$ , mmol/L,  $\bar{X} \pm S$ )

Category	Group	Before treatment	After treatment
TG	Treatment	1.50 $\pm$ 0.69	1.58 $\pm$ 0.69
	Control	1.53 $\pm$ 0.79	1.56 $\pm$ 0.86
TC	Treatment	4.30 $\pm$ 1.18	4.33 $\pm$ 0.90
	Control	4.15 $\pm$ 1.19	3.96 $\pm$ 0.84
LDL-C	Treatment	2.89 $\pm$ 0.98	2.77 $\pm$ 0.78
	Control	2.80 $\pm$ 0.69	2.52 $\pm$ 0.71 *
HDL-C	Treatment	1.07 $\pm$ 0.31	1.16 $\pm$ 0.30 *
	Control	1.04 $\pm$ 0.37	1.00 $\pm$ 0.39

**NOTE** Compared with before treatment within the group, \* $P < 0.05$ .

**3.4.2 Serum MMP-9 concentration level comparison.** Before treatment, there was no statistically significant difference between the groups ( $P > 0.05$ ), indicating comparability. After treatment, the serum MMP-9 concentration level in the treatment group decreased significantly, and the difference was statistically significant ( $P < 0.05$ ); the decrease in the control group was not significant ( $P > 0.05$ ), as indicated in Table 4.

**Table 4 Comparison of serum MMP-9 concentration levels before and after treatment between the two groups** ( $n=30$ , ng/mL,  $\bar{X} \pm S$ )

Category	Group	Before treatment	After treatment
MMP-9	Treatment	438.74 $\pm$ 196.95	353.81 $\pm$ 163.60 *
	Control	426.68 $\pm$ 153.35	380.47 $\pm$ 224.55

**NOTE** Compared with before treatment within the group, \* $P < 0.05$ .

## 4 Discussion

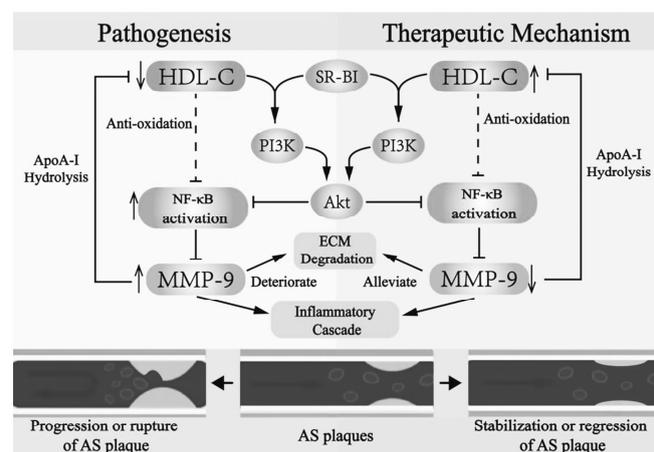
UA, as a high-risk clinical subtype of CHD, has received significant clinical attention in recent years due to its markedly increased risk of cardiovascular events and poor prognosis<sup>[12]</sup>. Currently, although lipid-lowering therapy dominated by statins combined with dual antiplatelet therapy has made considerable progress, clinical practice still faces many challenges such as residual cardiovascular risk and adverse drug reactions<sup>[13]</sup>, most prominent in cases with multiple risk factors or complex coronary artery disease. The underlying mechanism of this treatment dilemma may be closely relat-

ed to the lipid-inflammatory interaction during the evolution of AS plaques, particularly the vicious cycle mechanism mediated by HDL-C dysfunction leading to MMP-9 activation<sup>[14]</sup> which has become a key breakthrough in current research. Literature indicates that the protective mechanisms of HDL-C include promoting reverse cholesterol transport, inhibiting inflammatory responses, and reducing oxidative stress, among multiple pathways<sup>[15-17]</sup>. Specifically, HDL-C can effectively inhibit NF- $\kappa$ B activation and down-regulate MMP-9 transcriptional activity through dual mechanisms involving the SR-BI/PI3K/Akt axis and antioxidant pathways<sup>[18-20]</sup> thereby exerting anti-AS effects. Notably, MMP-9, as a key mediator of AS plaque instability, not only promotes AS plaque progression and increases its risk of rupture through multiple pathways<sup>[21-26]</sup> but can also disrupt HDL-C structural integrity by hydrolyzing apolipoprotein A-I<sup>[27]</sup>, forming a vicious cycle mechanism of "HDL-C reduction—MMP-9 activation", promoting the occurrence and development of AS plaques.

Compared with the limitations of Western medicine treatment models, TCM compound formulas demonstrate unique advantages in UA treatment due to their overall regulatory characteristics of "multi-component, multi-target, multi-pathway"<sup>[28]</sup>. TCM theory holds that UA belongs to the category of "chest impediment and heart pain", with a complex pathogenesis involving the interaction of multiple factors such as external pathogen invasion, dietary irregularities, emotional disorders, and visceral deficiency, leading to functional disorders of Qi, water, vessels, grain (spleen and stomach), and San Jiao<sup>[8]</sup>. Based on this, Professor Zhu Hongjun innovatively integrated TCM's Five Elements academic thought<sup>[29-30]</sup>, proposed the prevention and treatment concept of "simultaneously regulating the five pathways", and summarized the TCM empirical formula Qinjin Tablet based on years of clinical experience. Preliminary clinical observations show that Qinjin Tablet can not only quickly relieve the clinical symptoms of UA patients but also has significant advantages in regulating lipid metabolism disorders and stabilizing and reversing AS plaques<sup>[8]</sup>. Therefore, we hypothesized that Qinjin Tablet might improve the clinical efficacy of UA patients by regulating the HDL-C/MMP-9 signaling axis.

Hence, this study systematically evaluated the clinical efficacy of Qinjin Tablet through an RCT and deeply analyzed its potential intervention mechanism. The results show that Qinjin Tablet has multiple advantages in improving patient clinical efficacy. Qinjin Tablet significantly reduced the frequency and duration of angina attacks and increased the reduction or discontinuation rate of short-acting anti-angina drugs ( $P < 0.05$ ); moreover, the Qinjin Tablet treatment group was significantly better than the control group in improving total TCM syndrome scores and blood stasis scores ( $P < 0.05$ ). Consequently, it significantly improved the quality of life of UA patients; the improvement in SAQ scores across all dimensions was significantly greater in the treatment group than in the control group ( $P < 0.05$ ). Further analysis of blood lipid levels revealed that the control group primarily intervened in UA by lowering LDL-C levels, whereas the treatment group mainly improved patients' clinical symptoms and quality of life by increasing HDL-C levels, highlighting Qinjin Tablet's pro-

density for HDL-C-related protective mechanisms. The results of MMP-9 level testing further confirmed the clue of Qinjin Tablet's regulation of the HDL-C/MMP-9 signaling axis. The study showed that the serum MMP-9 concentration level decreased significantly in the Qinjin Tablet treatment group ( $P < 0.05$ ), while the reduction in MMP-9 levels before and after treatment in the control group was not significant. This validates our hypothesis regarding the mechanism by which Qinjin Tablet improves UA patient symptoms and quality of life by regulating the HDL-C/MMP-9 signaling axis (Fig. 1).



**Fig. 1** Molecular mechanism diagram for Qinjin Tablet regulating the HDL-C/MMP-9 signaling axis to stabilize AS plaques

In summary, Qinjin Tablet improves the clinical efficacy and quality of life of UA patients with phlegm-stasis syndrome by increasing HDL-C and decreasing MMP-9 levels, thereby regulating the HDL-C/MMP-9 signaling axis. However, the specific intervention mechanisms of Qinjin Tablet on targets such as SR-BI, PI3K, and Akt in the HDL-C/MMP-9 signaling pathway still require further exploration through animal experiments, cell experiments, *etc.*

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ics<sup>[10]</sup>. The vertebral artery runs in the extracranial segment, with short muscles between the transverse process and spinous process in the deep layer, which is accompanied by the distribution of arterial and venous plexuses. When stimulating the vertebral artery, it can improve hemodynamic balance<sup>[11]</sup>. Therefore, the patient's cervical  $V_s$  and  $V_m$  both increased after moxibustion treatment, while  $PI$  and  $RI$  both decreased. In summary, the improvement of CSA in the elderly by moxibustion has achieved significant therapeutic effects and is worthy of clinical promotion.

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