Research Article

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# Serum miR-142-5p serves as a biomarker to predict onset and short-term prognosis in acute coronary syndrome patients

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**Abstract**

**Objectives:** The target of this study was to explore the value of miR-142-5p for diagnosis and prognosis in patients with acute coronary syndrome (ACS).

**Methods:** RT-qPCR was utilized to investigate serum miR-142-5p in 77 ACS patients, 65 stable angina pectoris (SAP) patients, and 70 healthy volunteers. Receiver Operating Characteristic (ROC) curves were developed to estimate the clinical diagnostic importance of miR-142-5p for ACS. Kaplan-Meier and Cox regression were utilized to estimate the prognostic value of miR-142-5p for the incidence of major adverse cardiovascular events (MACE) during the 6-month post-treatment surveillance period after percutaneous coronary intervention (PCI) in patients with ACS.

**Results:** Serum miR-142-5p was significantly higher in ACS patients than in healthy controls (HC) and SAP groups. ROC curve indicated that serum miR-142-5p was effective in distinguishing ACS patients from HC and also distinguishing ACS patients from SAP patients. Pearson correlation coefficients confirmed that miR-142-5p was positively correlated with cardiac troponin I (cTnI, r=0.690) and Gensini score (GS, r=0.734). Furthermore, patients with high miR-142-5p expression had a greater probability of developing MACE after PCI treatment (Log Rank=0.016), which is an independent prognostic biomarker for ACS.

**Conclusions:** MiR-142-5p expression was significantly elevated in the serum of ACS patients. It not only demonstrates a strong correlation with an elevated risk of MACE following PCI, but also exhibits diagnostic utility and serves as an independent prognostic biomarker. It is a promising biomarker for the diagnosis and prognosis of ACS.

**Keywords:** MiR-142-5p; acute coronary syndrome; prognosis; biomarker

## Introduction

Acute coronary syndrome (ACS) is coronary heart disease resulting from the rupture of coronary atherosclerotic plaques, which gives rise to myocardial ischemia and thrombus formation. It is mainly shown as acute myocardial infarction (MI) and unstable angina pectoris [1, 2]. ACS is a group of diseases related to factors such as age, gender, hypertension, or diabetes mellitus, and is marked by a sudden onset and high fatality rate [3, 4]. At present, diagnosing ACS in a clinical setting is mainly ground on clinical manifestations, electrocardiogram, color ultrasound, etc. Percutaneous coronary intervention (PCI) is an effective tool for treating patients with ACS [5]. Even though considerable advances have been achieved regarding the diagnosis and treatment of ACS with advances in medical technology, its prognosis is not favorable and cardiovascular disease remains a principal cause of high mortality across the world [6]. Thus, the research of new biomarkers and molecular that aims to offer improved therapeutic alternatives for patients with ACS is very necessary and is something we urgently have to address now.

MicroRNAs (miRNAs), which are comprised of non-coding and small RNAs, possess a length ranging from approximately 19 to 25 nucleotides. The transcription-translation process is carried out by targeting and attaching to the 3′UTR region [7].

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Former studies have shown that over 200 miRNAs have significant functions in cardiac development and pathological procedures like MI, arrhythmias, and atherosclerosis (AS) [8]. Inflammatory response is an important pathological basis for the occurrence and development of ACS in cardiovascular diseases. The release of inflammatory cytokines and inflammatory cell infiltration and other processes promote the instability and rupture of coronary atherosclerotic plaques, thus triggering ACS. Changes in blood eosinophil counts, platelet activation, and changes in erythrocyte morphology can reflect the important role of inflammation in the development of cardiovascular disease [9–11]. It has also been revealed that irregular lipid levels are intimately associated with inflammation and can play a part in the formation and progression of coronary atherosclerotic plaques [12, 13]. Bilgin S et al. discovered that attenuating the inflammatory response can have a protective effect on the cardiovascular system [14]. MiRNAs also exert a crucial function in the modulation of inflammation. They are capable of affecting the magnitude and progression of the inflammatory reaction by controlling the expression of genes associated with inflammation. MiR-142-5p is among the miR-142 miRNA family and has a role in biological processes like tumor development, angiogenesis, and immune-related diseases [15, 16]. The excessive expression of miR-142-5p suppresses the serum concentrations of pro-inflammatory factors and elevates the levels of anti-inflammatory factors in individuals suffering from nonalcoholic steatohepatitis [17]. In Kayvanpour et al.'s study using a neural network model to screen for miRNAs related to ACS, miR-142-5p exhibited aberrant expression, which drew our attention [18]. In addition, studies have demonstrated that miR-142-5p increased in mouse atherosclerotic plaques and in ox-LDL-induced macrophages, potentially playing a key role in AS and arterial coronary disease progression [16, 19]. Wu et al. observed that serum miR-142-5p was elevated in AS patients, and its inhibition could suppress SPCs' proliferation and migration, suggesting it as a new treatment target [20]. Furthermore, miR-142-5p was also elevated in patients suffering from PCI restenosis [21]. Nevertheless, the mechanism by which miR-142-5p exerts its effect in ACS has not been comprehensively elucidated, and its potential as a diagnostic and prognostic biomarker for ACS still demands further thorough investigation.

This research evaluated the diagnostic and prognostic significance of miR-142-5p in ACS. The linkage between serum miR-142-5p and major adverse cardiovascular events (MACE) was then evaluated. This is expected to offer new ideas and methods for ACS clinical diagnosis and treatment, and lay a foundation for further study on miR-142-5p's role in ACS.

# Materials and methods

## Sample selection

The study obtained approval from the Ethics Committee of The First People's Hospital of Xiaoshan District and all subjects gave full informed consent. Altogether 142 patients who suffered from coronary artery disease and were treated in The First People's Hospital of Xiaoshan District were recruited into this study and further classified into ACS group (n=77) and stable angina pectoris (SAP) group (n=65) in light of electrocardiogram, color Doppler ultrasonography, and markers of myocardial injury, and 70 age-matched healthy volunteers were chosen. The results of the post hoc test indicated that, with an α value set at 0.05, the statistical power of the sample incorporated in the study was 0.91. Inclusion criteria for patients with ACS: (1) all patients met the diagnostic criteria for ACS, and the ACS diagnosis was verified by coronary angiography; (2) all of them experienced PCI for the first time and had not received any substance abuse before the procedure; and (3) the clinical data and follow-up data were complete. The clinical data gathered from ACS patients, SAP patients and healthy volunteers is presented in a summarized form in Table 1, including age, gender, body mass index (BMI), hypertension (HTN), fasting blood glucose (FBG), TG (triglyceride), HDL-C (high-density lipoprotein cholesterol), LDL-C (low-density lipoprotein cholesterol) as well as cardiac troponin I (cTnI). Measurements >140/90 mmHg were defined as hypertension [22]. Coronary angiographic findings were inspected by two experts and the extent of coronary atherosclerosis among ACS patients was evaluated through the Gensini score (GS) [23]. All subjects fasted for 12 h before treatment, and upper extremity venous blood was collected on the next day while they were still fasting. Let the blood sample stand at room temperature for 30 min. Then centrifuge it at a rotational speed of 1,000 g for 15 min. Take the supernatant serum and transfer it into EP tubes, and store at −80 °C pending the next step of the experiment.

## Real-time quantitative PCR(RT-qPCR)

According to the product instructions given by the manufacturer, total RNA isolation from serum samples was carried out by means of TRIzol reagent (Thermo Fisher Scientific, USA). The purity and quality of the total RNA that was extracted were gauged by a NanoDrop ND-1000 spectrophotometer. RNA was converted to cDNA through reverse transcription with the application of PrimeScript

**Table 1:** The baseline information comparison among the ACS, SAP, and HC groups.

| Indicators | | HC group (n=70) | SAP group (n=65) | ACS group (n=77) | $P_1$ | $P_2$ | $P_3$ |
|---|---|---|---|---|---|---|---|
| Age | | 48.43 ± 8.37 | 50.35 ± 8.83 | 49.65 ± 11.10 | 0.198 | 0.456 | 0.681 |
| Gender | Male | 46 | 43 | 48 | 0.957 | 0.670 | 0.637 |
| | Female | 24 | 22 | 29 | | | |
| BMI | | 22.90 ± 1.99 | 23.36 ± 1.74 | 23.61 ± 1.84 | 0.398 | 0.098 | 0.398 |
| HTN | HTN | 20 | 24 | 32 | 0.301 | 0.100 | 0.573 |
| | N-HTN | 50 | 41 | 45 | | | |
| FBG | | 4.88 ± 0.70 | 4.83 ± 0.55 | 4.91 ± 0.69 | 0.701 | 0.778 | 0.487 |
| TG | | 1.25 ± 0.25 | 1.21 ± 0.19 | 1.37 ± 0.35 | 0.310 | 0.021 | 0.002 |
| HDL-C | | 1.32 ± 0.15 | 1.29 ± 0.18 | 1.28 ± 0.12 | 0.260 | 0.072 | 0.744 |
| LDL-C | | 3.34 ± 0.40 | 3.44 ± 0.54 | 3.90 ± 0.43 | 0.253 | <0.0001 | <0.0001 |
| cTnI | | – | – | 1.70 ± 0.16 | – | – | – |
| GS | | – | 36.71 ± 5.61 | 55.66 ± 11.83 | – | – | <0.0001 |

HC, healthy control; SAP, stable angina pectoris; ACS, acute coronary syndrome; HR, hazard ratio; CI, confidence interval; cTnI, cardiac troponin I; GS, Gensini score; BMI, body mass index; HTN, hypertension; N-HTN, non-hypertension; FBG, fasting blood glucose; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. $P_1$, HC vs. SAP; $P_2$, HC vs. ACS; $P_3$, SAP vs. ACS.

RT kit (TaKaRa, Japan). The synthesized cDNA underwent RT-qPCR utilizing SYBR Green Real-time PCR Master Mix (Toyobo, Japan). The reaction conditions were 95 °C predenaturation for 5 min; 95 °C, 15 s, 60 °C, 30 s, 72 °C, 30 s, 30 cycles, and dissolution curve analysis at the end of the reaction. U6 was selected as an internal reference and the relative expression of miR-142-5p was analyzed using Equation $2^{-\Delta\Delta Ct}$. The primer sequences employed in the experiments are presented in Table S1.

### Follow-up

All ACS subjects underwent a 6-month period of follow-up study after PCI to document the occurrence of MACE. The follow-up event endpoints were cardiac death, revascularization, fresh heart failure, and recurrent MI as event endpoints. Patients with ACS had their information recorded through the outpatient chart each month, and no patients lost the opportunity for follow-up. All endpoints and adverse events were evaluated by specialized physician staff.

### Statistical analysis

Data were analyzed in this study using GraphPad Prism 9.0 (GraphPad Software, Inc., USA) and SPSS 23.0 (SPSS, Inc., Chicago, USA) software. Data are presented as mean ± standard deviation (SD). *T*-test was applied to contrast the results between the two groups. ANOVA was utilized for multi-group comparisons. The prognostic diagnostic significance of miR-142-5p for ACS was determined by receiver operating characteristic (ROC) curve. Pearson correlation coefficient was applied to examine the connection of serum miR-142-5p with ACS severity in ACS patients. The Kaplan-Meier method was utilized to plot survival curves, and the comparison of survival rates among patients was carried out by means of Log Rank. Cox regression was employed for analyzing the hazard elements that impact the prognosis of ACS patients. $p<0.05$ was regarded as a statistically significant difference.

## Results

### Basic information of all subjects

Table 1 shows the baseline data comparison among ACS patients, SAP patients and healthy controls (HC). There were 48 males with a mean age of 49.65 ± 11.10 years in ACS patients,43 males with 50.35 ± 8.83 years in SAP patients and 46 males with 48.43 ± 8.37 years in HC. BMI was 23.61 ± 1.84 in ACS patients, 23.36 ± 1.74 in SAP patients and 22.90 ± 1.99 in HC. 32 patients with HTN were present in the ACS group, 24 patients with HTN in the SAP group, and 20 patients with HTN in the HC group. In ACS patients, FBG was 4.91 ± 0.69 mmol/L and TG was 1.37 ± 0.35 mmol/L, in SAP patients, FBG was 4.83 ± 0.55 mmol/L and TG was 1.21 ± 0.19 mmol/L, and in HC, FBG was 4.88 ± 0.70 mmol/L and TG was 1.25 ± 0.25 mmol/L. HDL-C and LDL-C were 1.28 ± 0.12 and 3.90 ± 0.43 mmol/L in ACS patients, HDL-C and LDL-C were 1.29 ± 0.18 and 3.44 ± 0.54 mmol/L in SAP patients, and HDL-C and LDL-C were 1.32 ± 0.15 and 3.34 ± 0.40 mmol/L in HC. Statistical analysis revealed that the differences in age, gender, BMI, HTN, FBG, and HDL-C indicators did not show statistical significance for all subjects ($p>0.05$). However, in the TG and LDL-C indicators, there were significant differences among the comparisons

of ACS with HC and ACS with SAP groups ($p<0.05$). The cTnI content in ACS patients was 1.70 ± 0.16 ng/mL, and GS was 55.66 ± 11.83, while in the SAP group GS was 36.71 ± 5.61. A statistically significant difference in GS indicators was observed between the two groups ($p<0.05$).

## Serum level of miR-142-5p and ROC curve analysis

To estimate the function of miR-142-5p in ACS patients, we detected serum miR-142-5p of all subjects by RT-qPCR and plotted ROC curves. The study demonstrated that serum miR-142-5p was markedly raised in ACS patients as opposed to that in the HC and SAP groups ($p<0.001$, Figure 1A). Furthermore, the ROC curve indicated that serum miR-142-5p was capable of differentiating ACS patients from HC, with area under curve (AUC) was 0.886, and the sensitivity and specificity were 81.82 and 87.14 %. Serum miR-142-5p could also distinguish SAP patients with an AUC=0.841 and a sensitivity and specificity of 76.62 and 80.00 % (Figure 1B and C). This revealed that serum miR-142-5p can probably offer a reference for clinical diagnosis.

## Correlation analysis between miR-142-5p expression and ACS severity in ACS patients

By analyzing the relationship of serum miR-142-5p with ACS severity in ACS patients, we discovered that miR-142-5p expression was positively correlated with cTnI ($r=0.690$, $p<0.001$, Figure 2A) and GS ($r=0.734$, $p<0.001$, Figure 2B).

## Impact of miR-142-5p on the prognosis of ACS patients

To examine the prognostic significance of serum miR-142-5p in ACS patients regarding the incidence of MACE after PCI, a follow-up was conducted. The occurrence of MACE was documented at 6 months of follow-up. Grounded on the mean value of miR-142-5p expression, ACS subjects were grouped into miR-142-5p high and low expression groups, and Kaplan-Meier curves were plotted to assess the correlation between miR-142-5p expression and MACE occurrence. As illustrated in Figure 3, the incidence of MACE after PCI was higher in patients with high miR-142-5p expression (Log Rank, $p=0.016$). In addition, multivariate Cox regression models were adopted to explore the factors which have an impact on the prognosis of ACS patients. The results illustrated that serum miR-142-5p (hazard ratio (HR) =6.215, 95 % confidence interval (CI) =1.356–28.491, $p=0.019$), cTnI (HR=5.925, 95 % CI=1.269–27.655, $p=0.024$), GS (HR=11.893, 95 % CI=1.427–99.138, $p=0.022$), TG (HR=4.309, 95 % CI=1.005–18.476, $p=0.049$) and LDL-C (HR=3.186, 95 % CI=1.013–10.023, $p=0.048$) were independent predictors (Table 2).

# Discussion

ACS is brought about by the rupture of coronary atherosclerotic plaques [24]. AS is a chronic inflammatory ailment in which lesions emerge on the walls of medium-sized and large-sized arteries. It represents the principal reason for mortality in developed countries [25]. The precise cause of AS remains unclear; however, relevant risk factors affecting

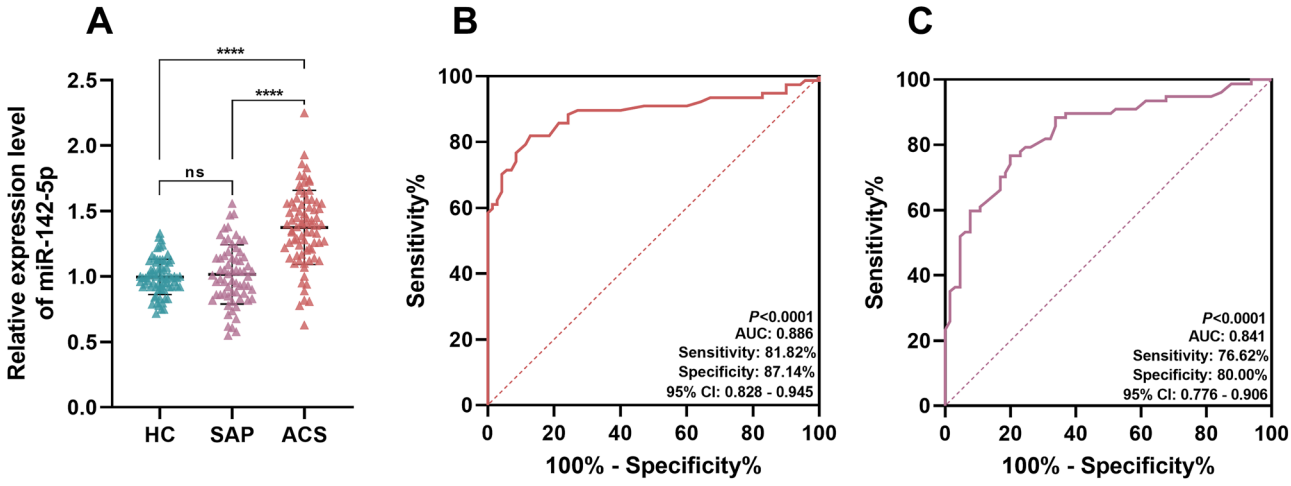


**Figure 1:** The diagnostic values of serum miR-142-5p. (A). Serum miR-142-5p in the three groups of subjects (***p<0.001). (B) ROC curve of serum miR-142-5p in the diagnosis of ACS and HC patients. (C) ROC curve of serum miR-142-5p in the diagnosis of ACS and SAP patients.

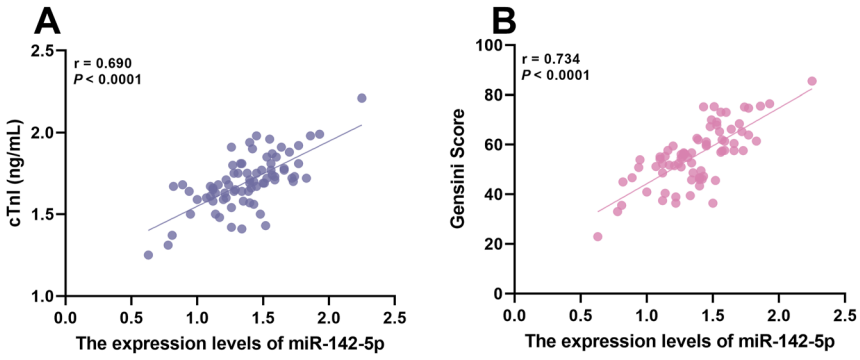


**Figure 2:** Correlation between serum miR-142-5p and cTnI (A) and gensini score (B) was evaluated by pearson correlation coefficient in ACS patients.

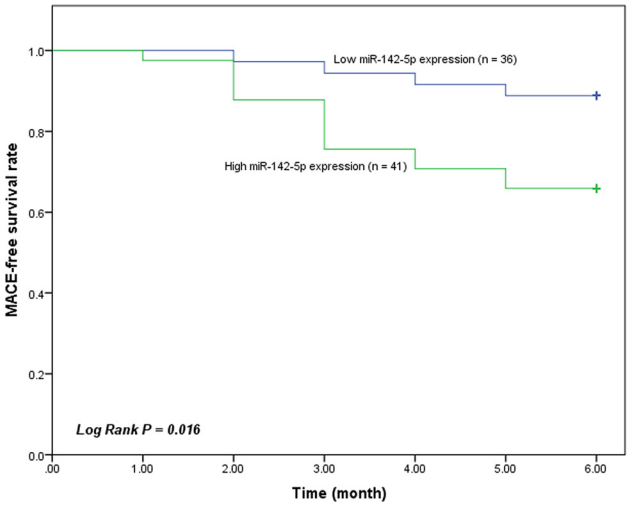


**Figure 3:** Kaplan-Meier survival analysis for serum miR-142-5p in ACS patients (log rank test p=0.016).

**Table 2:** The multivariate COX regression analysis of independent prognostic factors for ACS.

| Indicators | HR value | 95 % CI | | p-Value |
|---|---|---|---|---|
| | | Lower limit | Upper limit | |
| miR-142-5p | **6.215** | **1.356** | **28.491** | **0.019** |
| cTnI | **5.925** | **1.269** | **27.655** | **0.024** |
| GS | **11.893** | **1.427** | **99.138** | **0.022** |
| Age | 3.270 | 0.884 | 12.101 | 0.076 |
| Gender | 1.172 | 0.372 | 3.688 | 0.786 |
| BMI | 1.005 | 0.304 | 3.325 | 0.994 |
| HTN | 3.585 | 0.992 | 12.960 | 0.051 |
| FBG | 1.913 | 0.632 | 5.790 | 0.251 |
| TG | **4.309** | **1.005** | **18.476** | **0.049** |
| HDL-C | 0.556 | 0.133 | 2.327 | 0.421 |
| LDL-C | **3.186** | **1.013** | **10.023** | **0.048** |

ACS, acute coronary syndrome; HR, hazard ratio; CI, confidence interval; cTnI, cardiac troponin I; GS, gensini score; BMI, body mass index; HTN, hypertension; FBG, fasting blood glucose; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. The significant difference was marked in bold font and p<0.05.

the disease including hypertension, diabetes mellitus, smoking, high LDL levels and low HDL levels have been studied [26, 27]. The detection limit of the highly sensitive cTn is 10–100 times lower than of conventional methods. It can recognize myocardial injury earlier and improve the detection rate of MI [28]. Therefore, the search for new biomarkers is important for in-depth study of the molecular mechanisms of ACS.

In cardiovascular health, especially in ACS, miRNAs play an essential function [29]. Recent studies have demonstrated that a variety of miRNAs are involved in significant functions related to ACS and MACE. Jiang et al. discovered that dysregulation of the plasma miR-497/FGF23 axis was associated with clinical features and MACE in female patients with early-onset ACS [30]. Zhang et al. reported that miR-223-5p diagnosed ACS and predicted post-PCI outcomes by mechanisms that may involve influencing cardiomyocyte apoptosis, autophagy, and inflammatory cell infiltration [31]. In addition, serum miR-483-5p was upregulated in ACS patients and positively correlated with SYNTAX score and GS. It can be employed as a biomarker for the diagnosis of ACS and predictive of the incidence of MACE following PCI [32]. The mechanism of miR-142-5p in ACS is not clearly defined, but it may be similar to other miRNAs and thus worth investigating. This study focused on miR-142-5p with the aim of exploring its value in the diagnosis and prognostic assessment of ACS. The findings indicated that serum miR-142-5p was elevated, and it exhibited excellent diagnostic effectiveness. This implies its potential in the early detection of ACS, which could contribute to enhancing the prognosis of patients.

Presently, PCI is an effective means of treating patients with ACS, which is simple to perform, has a more pronounced therapeutic effect, and can reduce disease lethality by improving myocardial perfusion, but patients who undergo PCI are prone to MACE in the postoperative period [33]. GS is a scoring system for complex coronary artery lesions that has been proposed since the early days. Coronary artery disease can be rapidly evaluated, and high-

risk recognized and treated promptly [34]. Our research verified a relevant relation between serum miR-142-5p and GS, suggesting that the elevated expression of miR-142-5p is related to disease progression in ACS. Further, we focused on the effect of miR-142-5p on MACE after PCI. It was discovered that the occurrence rate of MACE after PCI was greater in patients with high miR-142-5p. Multivariate Cox regression revealed that miR-142-5p served as an independent prognostic marker regarding the occurrence of MACE in ACS patients. This research effectively uncovered the alterations in the serum miR-142-5p within ACS patients, along with its connection to the severity of the disease and the prognosis.

Finally, Intensive studies of the pathophysiology of ACS have shown that atherosclerotic plaque formation is slow. It consists mainly of the accumulation of lipids and cholesterol under the lining of the blood vessel walls [35]. During this process vulnerable plaques gradually form and their rupture can lead to adverse cardiovascular events. Plaque formation is closely related to abnormalities in lipid metabolism, especially oxidized LDL, which exacerbates the inflammatory response, activates immune mechanisms, and accelerates atherosclerosis [36, 37]. In addition, inflammation is present throughout the process of plaque formation. Macrophages and T cells interact to release enzymes, such as matrix metalloproteinases, which degrade the extracellular matrix and remodel the vessel wall, thereby impairing plaque stability [38, 39]. Finally, while neovascularization facilitates plaque growth to a certain extent, it also brings instability and adversely affects the structure and stability of the plaque [40].

This study has some limitations. Firstly, the small sample size may cause biased results. Secondly, a 6-month follow-up makes it hard to fully evaluate miR-142-5p's predictive ability for long-term prognosis. Thirdly, relying mainly on clinical studies, it lacks animal experiment support, and the mechanism of miR-142-5p in ACS and MACE risk is unclear. In the future, a large-scale multicenter study is planned to include more diverse and representative study subjects and carry out long-term follow-up, so as to more precisely clarify the role of miR-142-5p in the long-term prognostic assessment of ACS patients, and provide a basis for clinical decision-making. Secondly, we will construct an animal model of ACS and carry out cellular experiments, so as to deeply analyze the mechanism of miR-142-5p and reveal the pathogenesis of ACS in an all-round way. Finally, information on patients' medication use, comorbidities, and lifestyles will be comprehensively collected, and complex statistical models such as multifactor regression analysis will be used to explore in depth the relationship between these factors and miR-142-5p levels, as well as their impact on the study results.

In summary, this study determined the diagnostic ability of miR-142-5p in ACS patients and its predictive accuracy for MACE after PCI. The current findings offer novel viewpoints for the diagnosis and prognostic evaluation of ACS, and are anticipated to contribute to the retardation of disease progression.

**Research ethics:** The study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of The First People's Hospital of Xiaoshan District before the study began (No.20221013).

**Informed consent:** The informed consent has been signed and obtained from the participants involved.

**Author contributions:** Conceptualization, H.Y., J.Z., R.F. and H.L., P.W.; Data curation, H.Y., J.Z., R.F. and Y.Z., P.W.; Formal analysis, R.F. and Y.Z.; Funding acquisition, H.L.; Investigation, R.F. and Y.Z.; Methodology, H.Y., J.Z., R.F., H.L. and Y.Z., P.W.; Project administration, P.W., H.L.; Resources, R.F. and Y.Z.; Software, R.F. and Y.Z.; Supervision, P.W., H.L.; Validation, R.F. and Y.Z.; Visualization, R.F.; Roles/Writing – original draft, R.F.; Writing – review & editing, H.Y., J.Z., H.L., P.W.

**Use of Large Language Models, AI and Machine Learning Tools:** no.

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**Data availability:** The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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