

Significant association between serum resistin and hypersensitive troponin I levels in patients with a first ST-segment elevation myocardial infarction

Yong Zhu

Capital Medical University Affiliated Anzhen Hospital

Chengping Hu

Capital Medical University Affiliated Anzhen Hospital

Yu Du

Capital Medical University Affiliated Anzhen Hospital

Yan Liu

Capital Medical University Affiliated Anzhen Hospital

Jinxing Liu

Capital Medical University Affiliated Anzhen Hospital

Jianwei Zhang

Capital Medical University Affiliated Anzhen Hospital

Guojie Cheng

Beijing Daxing Hospital, Capital Medical University

Hongya Han

Capital Medical University Affiliated Anzhen Hospital

Qi Zhao

Capital Medical University Affiliated Anzhen Hospital

Zichuan Tong

Beijing Daxing Hospital, Capital Medical University

Yingxin Zhao (✉ zyingxinmi@163.com)

Capital Medical University Affiliated Anzhen Hospital <https://orcid.org/0000-0002-4897-2779>

Research article

Keywords: Adipocytokines, Resistin, STEMI, Peak hs-cTnI, Inflammation, Oxidative stress

Posted Date: October 2nd, 2019

DOI: <https://doi.org/10.21203/rs.2.15582/v1>

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Abstract

Background Resistin, a proinflammatory adipocytokine secreted predominately by macrophages in humans, plays an important role in the pathogenesis and development of atherosclerosis. The present research mainly investigated the association between serum resistin level and peak hypersensitive cardiac troponin I (hs-cTnI) in patients with ST-segment elevation myocardial infarction (STEMI). Methods We consecutively enrolled 92 patients with a first STEMI in this cross-sectional and observational study. Resistin concentrations upon admission and 24 h and 72 h after primary percutaneous coronary intervention (PCI) were all measured. The change in resistin (δ Resistin) was defined as (serum resistin concentration at admission)-(serum resistin concentration 24 h after intervention). Results Serum resistin concentration decreased rapidly after primary PCI. Resistin at admission correlated positively with tumour necrosis factor- α ($r = 0.522$, $p < 0.001$) and macrophage migration inhibitory factor ($r = 0.471$, $p < 0.001$). Additionally, resistin at admission correlated negatively with the reactive oxygen species scavengers superoxide dismutase ($r = -0.261$, $p = 0.012$) and glutathione peroxidase ($r = -0.235$, $p = 0.024$). Most importantly, serum resistin concentrations upon admission ($r = 0.381$, $p < 0.001$) and 24 h ($r = 0.372$, $p < 0.001$) and 72 h ($r = 0.347$, $p = 0.001$) after primary PCI all correlated with peak hs-cTnI, while δ Resistin was not associated with peak hs-cTnI. After multiple linear regression analysis, serum resistin ($\beta = 13.593$, 95% CI 5.951 to 21.235, $p < 0.001$) at admission and 24 h ($\beta = 13.972$, 95% CI 5.662 to 22.282, $p = 0.001$) and 72 h ($\beta = 14.455$, 95% CI 5.178 to 23.733, $p = 0.003$) after intervention remained associated with peak hs-cTnI. Conclusions In our present research, serum resistin concentrations at different time points all correlated positively with peak hs-cTnI, which may suggest that serum resistin concentrations during the acute phase of STEMI are useful for forecasting myocardial infarction size and prognosis in patients after primary PCI. Additionally, our research also indicated that resistin may regulate myocardial IRI partly by promoting the inflammatory process and oxidative stress.

Background

Various adipocytokines and proinflammatory cytokines, including resistin, can deteriorate in obesity-related disorders, including insulin resistance, type 2 diabetes, atherosclerosis, and coronary artery disease (CAD)[1, 2]. Resistin is primarily known as a hormone secreted by adipocytes primarily in rodents, while it is mainly expressed by macrophages in humans[3, 4]. Given the correlation between murine resistin and insulin resistance, numerous experimental and clinical studies have found that elevated human resistin may play a critical role in the pathophysiology of insulin resistance via various potential mechanisms[5, 6]. Additionally, growing evidence supports the idea that resistin promotes atherogenic dyslipidaemia and contributes to the development of hypertension[7-10].

Elevated resistin contributes to the formation of atherosclerosis through not only its indirect influences on the exacerbation of risk factors, such as insulin resistance, atherogenic dyslipidaemia, and hypertension, but also its direct effects on key steps of atherosclerosis[5]. Some experimental studies have indicated that resistin regulates the progression of atherosclerosis via inducing endothelial dysfunction, the artery inflammatory response, and the formation of foam cells in human macrophages[1, 2, 5]. Additionally,

clinical research with a large sample size also indicated that serum resistin may act as a risk factor for coronary disease in the general population, especially in women[11]. Furthermore, serum resistin concentration is also associated with the severity of CAD and in-stent restenosis, as some studies have shown[12, 13].

Although the serum resistin level increased markedly in patients with CAD, especially ST-segment elevation myocardial infarction (STEMI) patients[12, 14-16], there are no studies investigating the kinetics of serum resistin during the acute phase of STEMI and the correlation between serum resistin level and myocardial injury in STEMI patients. Therefore, the present research demonstrated the serial change in serum resistin concentration and the association between serum resistin and peak hypersensitive cardiac troponin I (hs-cTnI) level in STEMI patients who underwent primary percutaneous coronary intervention (PCI).

Methods

Study population

We consecutively enrolled 92 first STEMI patients from Beijing Anzhen Hospital from August 2017 to July 2018. To be recruited in the present study, each patient had to meet the following inclusion criteria: age range from 18 to 80 years, admitted to the emergency department for typical symptoms and subsequently confirmed STEMI according to the fourth universal definition of myocardial infarction, primary PCI performed successfully within 12 h from the onset of symptoms. All participants who had acute and chronic inflammation diseases, hepatic and renal dysfunction, active infection, a previous history of acute myocardial infarction (AMI) or a previous history of PCI and coronary artery bypass grafting (CABG) were excluded from enrolment.

The present research had ethics approval from the local Ethics Committee and was also performed according to the Declaration of Helsinki. Additionally, we secured written informed consent from the patients included in this study.

Primary PCI and medication

All participants who met the inclusion criteria were treated with a loading dose of 600 mg clopidogrel and 300 mg aspirin. Additionally, routine use of unfractionated heparin (70-100 U/kg) was recommended for all the patients recruited during the periprocedure. Then, the primary PCI was conducted in compliance with the newest guidelines on myocardial revascularization by the interventional cardiologist. The selection of new-generation drug-eluting stents, thrombus aspiration and intracoronary injection with glycoprotein IIb/IIIa inhibitors and vasodilators were all at the discretion of the operator. In addition to lifestyle intervention and risk factor control, dual antiplatelet therapy, beta-blockers, angiotensin-converting enzyme inhibitors (ACEI)/angiotensin receptor blocker (ARB) and statins were also recommended to the patients after primary PCI.

Baseline clinical data collection

Baseline data such as age, sex, body mass index (BMI), history of disease, and medication use at discharge were all gathered through the medical records. We also gathered data concerning primary PCI, including total ischaemic time, the culprit vessel, the number of diseased vessels, thrombolysis in myocardial infarction (TIMI) flow grade pre-PCI or post-PCI, and the number of stents implanted. Additionally, medication use at discharge was recorded.

Sample collection and assays

We obtained 5 ml fasting venous blood from the participants upon admission and 24 h and at 72 h after intervention. Samples upon admission and at 24 h and 72 h after intervention were centrifuged at 2500 rpm for 15 min. Then, we collected the serum and kept it at -80°C. Enzyme linked immunosorbent assay (ELISA) kits were applied to measure resistin levels upon admission and 24 h and 72 h after primary PCI. Tumour necrosis factor- α (TNF- α) and macrophage migration inhibitory factor (MIF) levels upon admission were also determined by ELISA (Shanghai BlueGene Biotech Co., Ltd., Shanghai, China). Additionally, we measured serum superoxide dismutase (SOD) and glutathione peroxidase (GSH-PX) levels at admission by a colorimetric method (Nanjing JianCheng Biotech Co., Ltd., Nanjing, China). δ Resistin was defined as (Serum resistin concentration at admission)-(serum resistin concentration 24 h after intervention).

Both the intra-assay and inter-assay coefficient of variation were < 5%. The samples included in the present research were all measured in duplicate.

We determined the serum Hs-cTnI level on admission and every 12 h for the following 3 days at the clinical biochemistry laboratory of Beijing Anzhen Hospital to identify peak Hs-cTnI. Additionally, the other routine biochemical parameters were also measured at the clinical biochemistry laboratory of Beijing Anzhen Hospital.

Statistical analysis

Normally distributed continuous parameters were summarized as the mean \pm SD, and continuous parameters with skewed distributions were summarized as medians (25th, 75th percentiles). Additionally, categorical variables were summarized as percentages. The association between resistin and other parameters was evaluated by Pearson correlation analysis. Furthermore, we applied univariate and multivariate linear models to assess the correlation between resistin and peak hs-cTnI after adjustment for other variables. SPSS software (version 20.0; SPSS, Inc., Chicago, IL) was used to conduct the statistical analysis. Additionally, p-values < 0.05 were considered significant.

Results

Baseline characteristics

The baseline clinical characteristics are indicated in table 1. In the present study, 92 patients (25.0% female) aged 55.82 ± 12.04 years were recruited. Atherosclerosis risk factors, hypertension, diabetes mellitus, and current smoking status were documented in 47 (51.1%), 17 (18.5%), and 63 (68.5%) patients, respectively. Regarding angiographic data, the culprit vessel was the left anterior descending artery in 57.6% of the patients, and TIMI flow grade 0 was seen in 84.8% of the patients. The patients enrolled in the present research all received timely reperfusion therapy with a median time of 286.50 mins, ranging from 197.00 mins to 412.75 mins. After the primary PCI, TIMI flow grade 3 was observed in 76.1% of the participants. Additionally, the percentages of patients receiving dual antiplatelet therapy, statin, ACEI/ARB, and beta-blockers were 100.0%, 100.0%, 69.6%, and 88.0%, respectively, at discharge.

Change in serum resistin concentration

The mean levels of serum resistin on admission and 24 h and 72 h after intervention were 1.85 ± 0.42 ng/ml, 1.62 ± 0.40 ng/ml, and 1.64 ± 0.36 ng/ml, respectively ($p < 0.001$). As indicated in Figure 1, admission resistin was highest. Then, serum resistin declined markedly following the first 72 h after primary PCI and reached its lowest level 24 h after intervention. Serum resistin increased from 1.62 ± 0.40 ng/ml (24 h) to 1.64 ± 0.36 ng/ml (72 h) gradually over time ($p = 0.661$), although without significance. Additionally, there was no significant difference in the mean admission resistin concentration between men and women (1.84 ± 0.44 ng/ml vs 1.87 ± 0.39 ng/ml, $p = 0.621$). Furthermore, admission resistin has no association with BMI ($r = -0.099$, $p = 0.350$), fasting blood glucose ($r = 0.050$, $p = 0.638$), triglycerides ($r = 0.164$, $p = 0.118$), low density lipoprotein ($r = -0.036$, $p = 0.733$), high density lipoprotein ($r = -0.068$, $p = 0.517$), or creatinine ($r = 0.058$, $p = 0.585$).

Serum resistin was correlated with the inflammatory response and oxidative stress

We first examined the correlation between serum resistin concentration and inflammation. As indicated in Figure 2A, serum resistin concentration at admission correlated positively with admission TNF- α ($r = 0.522$, $p < 0.001$). Additionally, serum resistin concentration at admission had a positive association with admission MIF levels ($r = 0.471$, $p < 0.001$), as shown in Figure 2B. Furthermore, as demonstrated in Figure 2C and Figure 2D, admission serum resistin concentration correlated negatively with serum SOD ($r = -0.261$, $p = 0.012$) and GSH-PX ($r = -0.235$, $p = 0.024$) concentrations on admission.

Serum resistin was associated with myocardial injury

We next analysed the association between serum resistin concentration at each time point with peak hs-cTnl. As revealed in Figure 3, serum resistin level at admission ($r = 0.381$, $p < 0.001$; Figure 3A), serum resistin concentration at 24 h ($r = 0.372$, $p < 0.001$; Figure 3B) and serum resistin concentration at 72 h ($r = 0.347$, $p = 0.001$; Figure 3C) after intervention were all correlated positively with peak hs-cTnl. However, as shown in Figure 3D, serum peak hs-cTnl had no association with δ Resistin ($r = 0.046$, $p = 0.667$).

Furthermore, as shown in table 2, table 3, and table 4, serum resistin at admission (beta = 13.593, 95% CI 5.951 to 21.235, $p < 0.001$; table 2), serum resistin at 24 h (beta = 13.972, 95% CI 5.662 to 22.282, $p =$

0.001; table 3) and serum resistin at 72 h (beta = 14.455, 95% CI 5.178 to 23.733, p = 0.003; table 4) after primary PCI remained associated with peak hs-TnI after multiple linear regression analysis, which adjusted for variables including age, sex, culprit vessel, total ischaemic time, triglycerides, and fasting blood glucose.

Discussion

This is the first prospective study investigating the kinetics of serum resistin in patients with a first STEMI after primary PCI, and several relationships were observed. First, our present research revealed that serum resistin concentration was highest at admission in patients with STEMI and declined after intervention, reaching the lowest level 24 h after primary PCI. Second, we revealed that admission serum resistin has an association with inflammatory parameters (TNF- α and MIF) and markers of oxidative stress (SOD and GSH-PX). Last, our present study indicated that serum resistin at admission and serum resistin 24 h and 72 h after primary PCI were all predictive of later changes in the necrotic marker peak hs-cTnI after multiple linear regression analysis.

Resistin, a 12.5 kDa peptide, is secreted predominately by white adipose tissue in mice[5, 6]. However, human resistin is expressed mainly by peripheral blood mononuclear cells (PBMCs), macrophages, and bone marrow cells, which suggests that resistin may be linked to inflammation[5, 6]. There is much evidence indicating that serum resistin concentration is markedly increased in CAD patients, especially in patients with acute coronary syndrome (ACS)[15-17]. Additionally, both resistin protein and resistin mRNA increase significantly in epicardial adipose tissue in patients with ACS[18]. Our present paper extends these prior investigations by showing that the increased serum resistin admission decreased significantly 72 h after intervention. Furthermore, clinical research with 6636 adults from the general population indicated that the mean resistin level in men was lower than that in women[11]. In contrast, our present research indicates that there are no significant differences in resistin concentration at admission between women and men in STEMI patients. We speculated that the dramatically elevated serum resistin level at the onset of STEMI may contribute partly to this controversy. Additionally, there were no significant associations between resistin at admission and BMI, fasting blood glucose, triglycerides, low density lipoprotein, high density lipoprotein, and creatinine in our present research, which is in accordance with the studies by Tarek E Korah et al and Qiao et al[15, 16].

It is well known that inflammation plays a critical role in the process of myocardial ischaemia-reperfusion injury (IRI)[19-21]. Prior clinical and experimental studies have shown that resistin may promote the inflammatory process via upregulating the expression of proinflammatory cytokines, including TNF- α , interleukin-6, and monocyte chemoattractant protein-1[5, 22]. TNF- α and MIF levels upon admission all participate in the process of IRI [19, 23, 24], and our present research indicated for the first time that resistin at admission correlated positively with them in STEMI patients. In addition to inflammation, previous studies revealed that oxidative stress also contributed to IRI via reactive oxygen species (ROS) [20, 21, 25]. SOD and GSH-PX may produce cardioprotective effects by eliminating ROS[26]. Our present study revealed that serum resistin has a negative association with SOD and GSH-PX in STEMI patients.

Additionally, previous research by D.V. Godin also suggested that nitric oxide (NO) can prevent myocardium damage due to ischaemic/reperfusion by balancing the homeostasis of mitochondrial Ca^{2+} [26, 27]. However, resistin may reduce the NO concentration significantly in human endothelial cells, as previous studies indicated [28]. Furthermore, research by Laurikka and colleagues revealed that resistin levels also correlate with the oxidative stress marker myeloperoxidase [29].

Subsequently, we investigated the association between serum resistin and myocardial injury. Previous studies revealed that serum resistin levels correlated positively with peak creatine kinase (CK), peak MB isoenzyme of CK (CK-MB), and peak cardiac troponin I (cTnI) [15-17]. Our present research extended previous studies by finding that resistin at admission and resistin 24 h and 72 h after primary PCI all had a positive correlation with peak hs-cTnI. Additionally, Sarah Rothwell et al demonstrated that human resistin can exacerbate myocardial IRI through the TNF- α inducible NF- κ B signal pathway in vitro [30]. Resistin can also induce sterile inflammation partly through an important receptor and mediator, Toll-like receptor-4 (TLR4) [31]. Whether the resistin-TLR4 signalling pathway participates in regulating the process of IRI should be elaborated in future research. Furthermore, Laurikka and colleagues showed that the maximal change in resistin concentration has a positive association with myocardial injury in patients who underwent cardiac surgery [29]. However, the change in resistin level (δ Resistin) has no association with myocardial injury in STEMI patients, as indicated by our data. Various factors, such as the type of patient recruited and the time points of sample extraction, may all contribute to discrepancies. From the evidence above, we speculated that resistin may promote myocardium IRI, and promoting the inflammatory process and oxidative stress may be part of the potential mechanisms.

Several limitations in the present research should be noted. First, this is a cross-sectional and observational study with a small sample size. Second, the patients recruited in the present study were all the first STEMI patients who underwent primary PCI successfully, which may affect the results and make the results of our study unsuitable for the general population. Third, we only used peak hs-cTnI as a marker of myocardial necrosis. Future studies should use cardiac magnetic resonance imaging or single-photon emission computed tomography imaging to quantify myocardial infarction size. Finally, our present research mainly focuses on the association between resistin and peak hs-cTnI. Future studies are required to evaluate the mechanism and causality between resistin and myocardial IRI.

Conclusions

The present study established that serum resistin concentration was associated with peak hs-cTnI in STEMI patients. Additionally, from our research, we speculated that resistin may regulate myocardial IRI partly through promoting inflammatory processes and oxidative stress. Future studies are warranted to elucidate the causality and underlying mechanisms between serum resistin expression and myocardial IRI.

Abbreviations

CAD	coronary artery disease
STEMI	ST-segment elevation myocardial infarction
Hs-cTnl	hypersensitive cardiac troponin I
PCI	percutaneous coronary intervention
AMI	acute myocardial infarction
CABG	coronary artery bypass grafting
ACEI/ARB	angiotensin-converting enzyme inhibitors /angiotensin receptor blocker
BMI	body mass index
TIMI	thrombolysis in myocardial infarction
TNF- α	tumour necrosis factor- α
MIF	macrophage migration inhibitory factor
SOD	superoxide dismutase
GSH-PX	glutathione peroxidase
PBMCs	peripheral blood mononuclear cells
ACS	acute coronary syndrome
IRI	ischaemia-reperfusion injury
ROS	reactive oxygen species
TLR4	Toll-like receptor-4

Declarations

Authors' contributions

YZ and CPH interpreted the data and wrote the manuscript. YXZ and ZCT designed the study protocol and supervised the project. JXL and QZ determined serum MCP-1 levels through ELISA kits. YD and YL enrolled the participants. HYH, JWZ, and GJC managed the participants. All authors read and approved the final manuscript.

Acknowledgment

We thank Weiwei Fu for her help in writing this article.

Ethics approval and consent to participate

The present research had ethics approval from Ethics Committee of Beijing Anzhen Hospital, Capital Medical University and was also performed according to the Declaration of Helsinki. Additionally, we secured written informed consent from the patients included in this study.

Consent for publication

Not applicable.

Availability of data and materials

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

Competing interests

The authors declare that they have no competing interests.

Funding

This research was supported by a grant from the Beijing Municipal Science and Technology Commission (Z171100000417042), National Key Research and Development Program of China (2017YFC0908800), the “Beijing Municipal Administration of Hospitals” Ascent Plan (DFL20150601) and Mission plan(SML20180601),and Beijing Municipal Health Commission “Project of Science and Technology Innovation Center”(PXM2019-026272-000006) (PXM2019-026272-000005). There was no role of the funding body in the design of the study and collection, analysis, and interpretation of data and in writing the manuscript.

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Tables

Table 1 Baseline characteristics

Anthropometric variables	
Age (years)	55.82 ± 12.04
Female sex (%)	23 (25.0%)
Body mass index (kg/m ²)	26.06 ± 3.42
Baseline LVEF (%)	59.0% (50.3%,62.0%)
Killip class II on admission (%)	41 (44.6%)
Risk factors for atherosclerosis	
Hypertension (%)	47 (51.1%)
Diabetes mellitus (%)	17 (18.5%)
Current smoking (%)	63 (68.5%)
Laboratory findings	
White blood cell (10 ⁹ /L)	11.14 ± 3.17
C-reactive protein(mg/L)	3.00 (1.00,9.00)
Haemoglobin (g/L)	138.19 ± 23.64
Fasting blood glucose (mmol/L)	6.40 (5.45,7.84)
Triglycerides (mmol/L)	1.45 (1.00,2.04)
LDL-C (mmol/L)	2.77 ± 0.77
HDL-C (mmol/L)	0.99 ± 0.20
Uric acid (mg/dl)	315.42 ± 89.21
Serum creatinine (µmol/L)	60.52 ± 16.93
Peak hs-TnI (pg/ml)	48.58 ± 16.54
Angiographic data	
Culprit vessel (%)	
LAD	53 (57.6%)
Others	39 (42.4%)
Number of diseased vessels (%)	
1-vessel disease	46 (50.0%)
2-vessel disease	28 (30.4%)
3-vessel disease	18 (19.6%)
Gensini score	48.00 (36.00,59.50)
TIMI = 0, Before primary PCI (%)	78 (84.8%)
Primary PCI	
Total ischaemic time (min)	286.50 (197.00,412.75)
Number of stents implanted	1.00 ± 0.33
Thrombus aspiration (%)	11 (12.0%)
Glycoprotein IIb/IIIa inhibitors (%)	60 (65.2%)
TIMI = 3, After primary PCI (%)	70 (76.1%)
Medication use at discharge (%)	
Aspirin	92 (100.0%)
Clopidogrel	92 (100.0%)
Statin	92 (100.0%)
ACEI/ARB	64 (69.6%)
Beta-blocker	81 (88.0%)

LVEF, left ventricular ejection fraction; LDL-C, low-density lipoprotein cholesterol; HDL-C high-density lipoprotein cholesterol; hs-cTnI, hypersensitive cardiac troponin I; LAD, left anterior descending coronary artery; TIMI, thrombolysis in myocardial infarction; PCI, percutaneous coronary intervention; Total ischaemic time, from symptom onset to recanalization of culprit vessel; ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker.

Table 2 Correlation between peak hs-TnI and other parameters in univariate and multivariate analyses (Mode A)

Variables	Univariate analysis			Multivariate analysis		
	Beta	95% CI	P-value	Beta	95% CI	P-value
Age	0.053	-0.234 to 0.341	0.715	0.090	-0.196 to 0.376	0.533
Sex	6.848	-0.979 to 14.675	0.086	6.108	-1.565 to 13.781	0.117
Culprit vessel	-0.463	-7.435 to 6.509	0.895	-2.523	-9.057 to 4.011	0.445
Total ischaemic time	-0.008	-0.025 to 0.009	0.364	-0.006	-0.022 to 0.010	0.439
Triglycerides	2.628	0.116 to 5.140	0.041	2.145	-0.433 to 4.724	0.102
Fasting blood glucose	0.489	-0.475 to 1.452	0.316	0.134	-0.808 to 1.075	0.779
Admission resistin	14.877	7.320 to 22.434	<0.001	13.593	5.951 to 21.235	0.001

Total ischaemic time, from symptom onset to recanalization of culprit vessel. Age, gender, culprit vessel, total ischaemic time, fasting blood glucose and other variables with $p < 0.05$ in the univariable analysis were all included in the multivariable linear regression analysis mode.

Table 3 Correlation between peak hs-TnI and other parameters in univariate and multivariate analyses (Mode B)

Variables	Univariate analysis			Multivariate analysis		
	Beta	95% CI	P-value	Beta	95% CI	P-value
Age	0.053	-0.234 to 0.341	0.715	0.049	-0.240 to 0.339	0.736
Sex	6.848	-0.979 to 14.675	0.086	4.273	-3.554 to 12.101	0.281
Culprit vessel	-0.463	-7.435 to 6.509	0.895	-2.763	-9.352 to 3.826	0.407
Total ischaemic time	-0.008	-0.025 to 0.009	0.364	-0.007	-0.023 to 0.010	0.425
Triglycerides	2.628	0.116 to 5.140	0.041	2.458	-0.118 to 5.033	0.061
Fasting blood glucose	0.489	-0.475 to 1.452	0.316	0.156	-0.792 to 1.104	0.744
Serum resistin (24 h)	15.441	7.373 to 23.509	< 0.001	13.972	5.662 to 22.282	0.001

Total ischaemic time, from symptom onset to recanalization of culprit vessel. Age, gender, culprit vessel, total ischaemic time, fasting blood glucose and other variables with $p < 0.05$ in the univariable analysis were all included in the multivariable linear regression analysis mode.

Table 4 Correlation between peak hs-TnI and other parameters in univariate and multivariate analyses (Mode C)

Variables	Univariate analysis			Multivariate analysis		
	Beta	95% CI	P-value	Beta	95% CI	P-value
Age	0.053	-0.234 to 0.341	0.715	0.027	-0.267 to 0.322	0.853
Sex	6.848	-0.979 to 14.675	0.086	4.998	-2.844 to 12.839	0.209
Culprit vessel	-0.463	-7.435 to 6.509	0.895	-2.470	-9.105 to 4.165	0.461
Total ischaemic time	-0.008	-0.025 to 0.009	0.364	-0.009	-0.026 to 0.007	0.265
Triglycerides	2.628	0.116 to 5.140	0.041	2.292	-0.319 to 4.904	0.085
Fasting blood glucose	0.489	-0.475 to 1.452	0.316	0.091	-0.866 to 1.048	0.850
Serum resistin (72 h)	15.828	6.883 to 24.773	0.001	14.455	5.178 to 23.733	0.003

Total ischaemic time, from symptom onset to recanalization of culprit vessel. Age, gender, culprit vessel, total ischaemic time, fasting blood glucose and other variables with $p < 0.05$ in univariable analysis were all included in multivariable linear regression analysis mode.

Figures

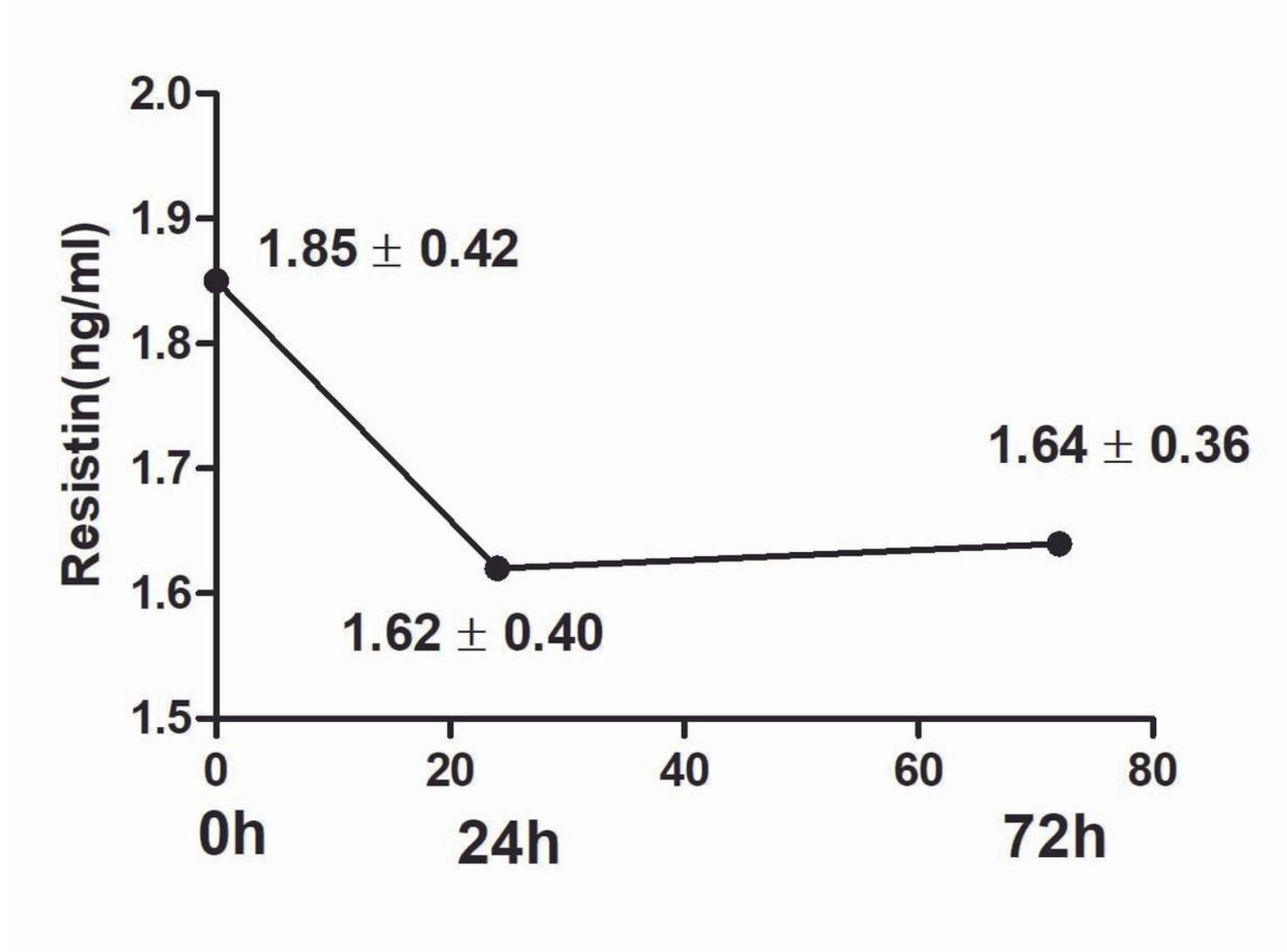


Figure 1

Change in serum resistin concentration in patients with STEMI after primary PCI. STEMI, ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention.

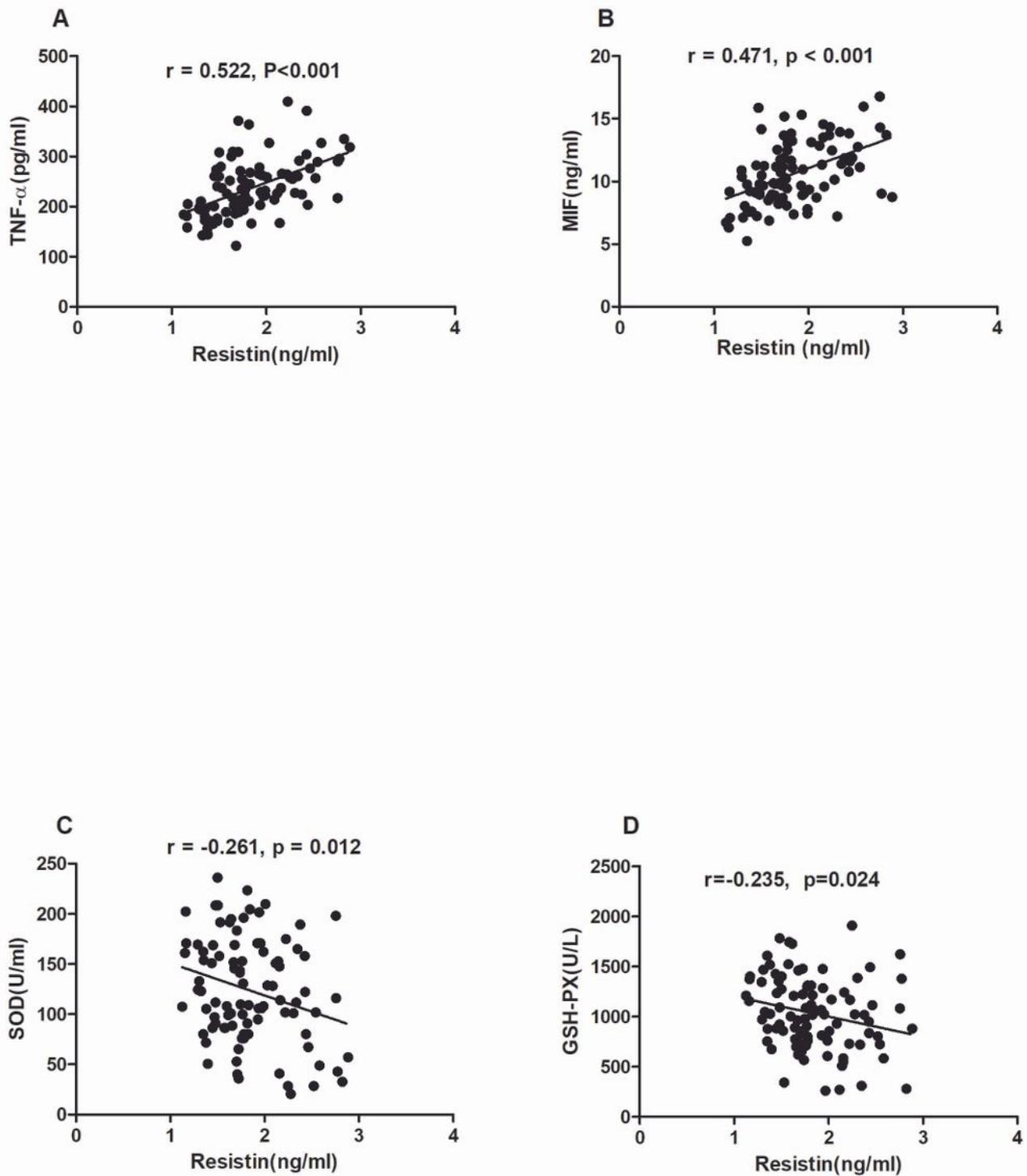


Figure 2

Correlation of resistin at admission with TNF- α (A) and MIF(B). Correlation of resistin at admission with SOD(C) and GSH-PX(D). TNF- α , tumour necrosis factor- α ; MIF, macrophage migration inhibitory factor; SOD, serum superoxide dismutase; GSH-PX, glutathione peroxidase.

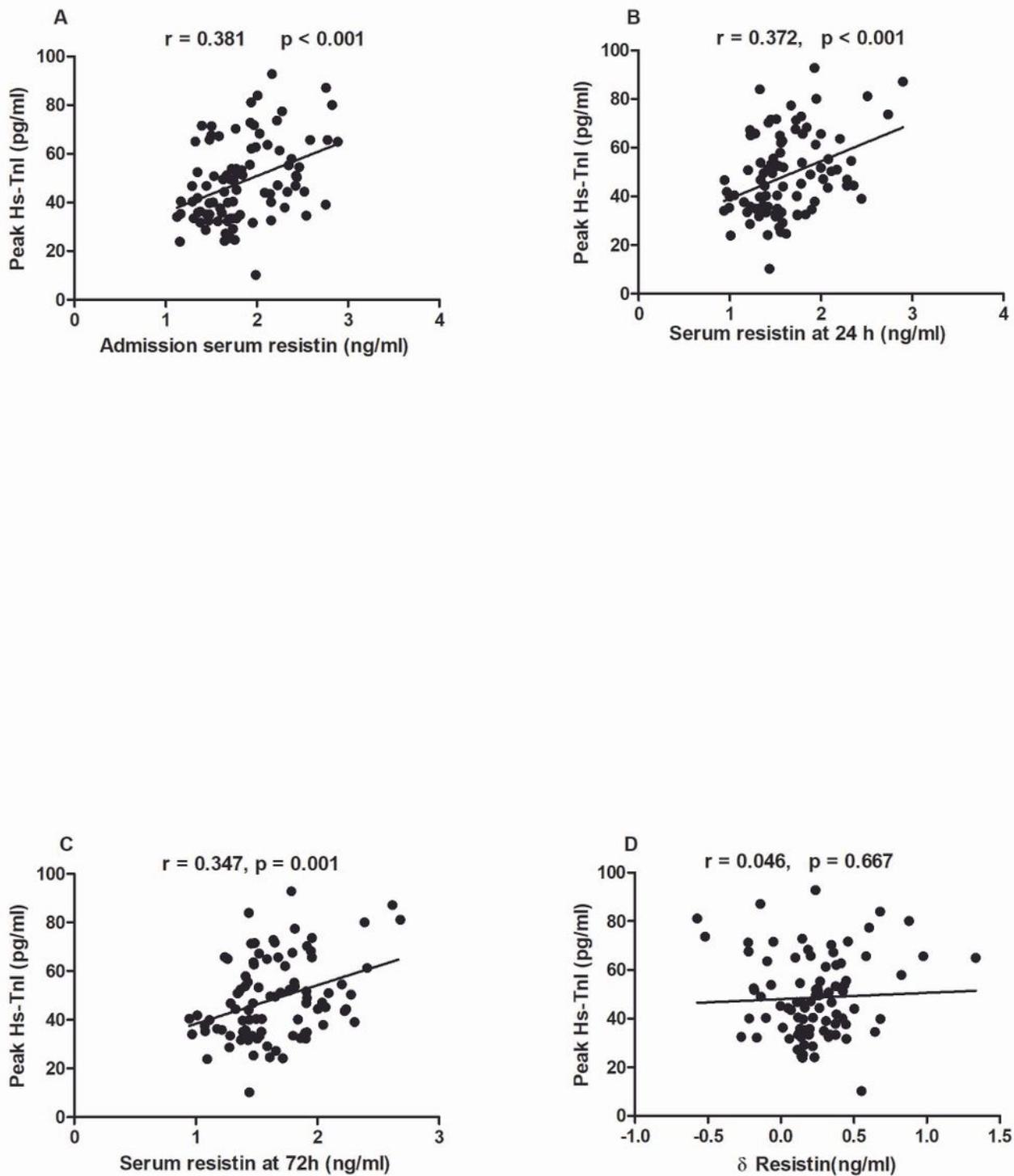


Figure 3

Association between resistin at admission (A) and resistin 24 h (B) and 72 h (C) after primary PCI and peak hs-cTnI. Association of Δ Resistin with peak hs-cTnI (D). PCI, percutaneous coronary intervention; hs-cTnI, hypersensitive cardiac troponin I; Δ Resistin, Δ Resistin was defined as (serum resistin concentration at admission)-(serum resistin concentration 24 h after intervention).