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Original Article

Effect of hyaluronic acid and laminin in coronary heart disease patients complicated with myocardial infarction

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Abstract: Objective: To investigate the expression and significance of serum hyaluronic acid (HA) and laminin (LN) in patients with coronary heart disease (CHD) complicated with myocardial infarction (MI). Methods: From July 2016 to July 2019, 280 CHD patients without MI admitted to the department of cardiology in our hospital were enrolled into a CHD group, and another 280 CHD patients complicated with MI into an infarction group. The expressions of serum LN and HA were compared between the two groups to analyze its correlation with blood lipid and blood glucose levels, and the results of serum protein electrophoresis (SPE) as well as the levels of mean platelet volume (MPV), high-sensitivity C-reactive protein (hs-CRP), cardiac troponin I (cTnI) and left ventricular ejection fraction (LVEF) were compared between the two groups. Result: The infarction group showed significantly higher expressions of serum LN and HA than the CHD group ($P < 0.01$), and their expressions increased with the elevation of the number of lesions ($P < 0.05$). Compared with patients with combined anterior and inferior wall infarction, LN and HA showed remarkably lower levels in patients with anterior wall infarction or inferior wall infarction ($P < 0.05$). Additionally, the infarction group obtained higher expressions of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglyceride (TG), and fasting plasma glucose (FPG), but a lower level of high-density lipoprotein cholesterol (HDL-C) than the CHD group (all $P < 0.01$), which demonstrated that LN and HA were positively correlated with TC, TG, hs-CRP, FPG, and LDL-C (all $P < 0.05$), but negatively correlated with HDL-C ($P < 0.05$). Moreover, the infarction group presented a notably higher level of β1-globulin than the CHD group, and the increase in β1-globulin level may have increased the readmission rate and mortality of patients with CHD complicated with MI. MPV was negatively correlated with LVEF ($P < 0.05$), but positively correlated with the serum hs-CRP and cTnI levels (both $P < 0.05$). Conclusion: The expressions of serum HA and LN in patients with CHD complicated with MI witnessed an elevation, which indicated a positive correlation between their expression and the levels of blood lipid and blood glucose. A higher readmission rate and mortality of patients with CHD complicated with MI may be triggered by the elevation of β1-globulin, and MPV can be used as an objective index to evaluate the condition of patients with MI.

Keywords: Serum hyaluronic acid, CHD, laminin, MI

Introduction

CHD is the coronary atherosclerotic lesions caused by various factors, such as vascular stenosis, occlusion, myocardial ischemia, hypoxia, necrosis, inflammation, and myocardial cell apoptosis, in which myocardial ischemia and collagen deposition are contributory factors to MI [1-3]. The extracellular matrix of the myocardium is mainly composed of hyaluronic acid (HA), type III procollagen peptide, and laminin (LN), which are crucial in maintaining the shape of the heart chamber and the arrangement of myocardial cells. Serum HA and LN play a key role in the interaction between vascular tissue and blood components [4]. Studies have shown that the deposition of collagen is accelerated by HA and LN, which facilitates the formation of a collagenous fiber network in vascular smooth muscle [5, 6]. In recent years, mean platelet volume (MPV) has been proven to be an effective indicator for the degree of platelet activation, with close correlation with the development, progression, and prognosis of MI. However, whether it is consistent with LVEF, hs-CRP, and cTnI is still poorly understood.

This study analyzed the expression of HA and LN in patients with CHD complicated with acute myocardial infarction (AMI) and discussed the diagnostic value of serum HA and LN. At the same time, the characteristics of serum protein electrophoresis (SPE) in two groups were analyzed, and the discharged patients were followed up by phone call to evaluate the relationship between SPE and the prognosis of CHD complicated with MI.

Materials and methods

Clinical data

A total of 280 CHD patients without MI admitted to the department of cardiology of our hospital from July 2016 to July 2019 were enrolled into a CHD group, including 159 men and 121 women, aged from 40 to 70 years, with an average age of 52.4±4.8 years and a disease course of 1 to 20 days (10.2±4.2 days on average). Coronary artery lesions: There were 120 cases of single-vessel disease, 100 cases of double-vessel disease, and 60 cases of three-vessel disease. Additionally, 280 CHD patients with MI were included and assigned to an infarction group, including 167 men and 113 women, aged from 42 to 71 years, with an average age of 52.5±4.8 years and a disease course of 1 to 20 days (10.1±4.0 days on average). Coronary artery lesions: There were 130 cases of single-vessel disease, 87 cases of double-vessel disease, and 63 cases of three-vessel disease. In terms of the infarct sites, there were 104 cases of anterior wall area (29 cases of anterior wall, 31 cases of anterior wall with high lateral wall, 22 cases of anteroseptal wall, 12 cases of anterolateral wall, 10 cases of high lateral wall), 122 cases of inferior wall area (42 cases of inferior wall, 32 cases of inferior wall with posterior wall and right ventricle, 28 cases of inferior wall with right ventricle, 20 cases of posterior wall), and 54 cases of the combined anterior wall and inferior wall. This study has been supervised and approved by the Medical Science Research Ethics Committee of Shanxi Bethune Hospital, with the approved No. of 2017LC(257)-113. There was no significant difference in gender, age, course of the disease, and the number of coronary artery lesions between the two groups (all $P > 0.05$).

Inclusion and exclusion criteria

Inclusion criteria: (1) Patients diagnosed with CHD by DSA in line with *Nomenclature and Criteria for Diagnosis of Ischemic Heart Disease* issued by the World Health Organization (WHO) [7]; (2) Patients who signed informed consent; (3) Patients with cardiac function of grade I-III. Exclusion criteria: (1) Patients with cardiac function of grade IV; (2) Patients complicated with severe lung, liver, kidney, or connective tissue diseases; (3) Patients with mental diseases or poor treatment compliance; (4) Patients with other diseases affecting HA and LN expression. In addition to the general inclusion criteria, patients in the infarction group also met the diagnostic criteria of AMI, while patients in the CHD group were not comorbid with AMI [8, 9].

HA test

Fasting venous blood (5 ml) was collected from the patients of two groups at 8:00 am on the next day of admission, and the serum was isolated after centrifugation. Then, the enzyme-linked immunosorbent assay (ELISA) was used to determine the serum HA expression strictly in accordance with the instructions of the kit (Shanghai Thermo Fisher Scientific Co., Ltd.).

Serum LN test

Fasting venous blood (5 ml) was collected from the patients of two groups at 8:00 am on the next day of admission, and the serum was isolated after centrifugation. Then, the expression of LN was determined by radioimmunoassay in strict accordance with the instructions of the kit (CHENGUANG Biotech Group Co., Ltd.).

Blood lipid and blood glucose test

Fasting venous blood (5 ml) was collected from the patients of two groups at 8:00 am on the next day of admission, and the serum was isolated after centrifugation. Then, TG, HDLC, TC, and LDL-C were measured by an automatic biochemical analyzer, and FPG was measured by the glucose oxidase method using an automatic biochemical analyzer within 3 hours.

Table 1. Comparison of serum LN and HA expressions between the two groups ($\bar{x} \pm sd$)

Group	cases	LN (μg/L)	HA (μg/L)
Infarction group	280	195.8±35.7	176.2±44.8
CHD group	280	243.5±32.4	201.7±52.4
t		16.56	6.19
P		< 0.001	< 0.001

Determination of SPE index

Fasting blood (3.5 ml) was collected from patients of both groups in the morning after admission. The serum was separated and detected by SPE with capillarys2 automatic capillary electrophoresis instrument (Sebia Company of France). With capillary tube as separation channel and high voltage direct current electric field as a driving force, serum proteins were separated based on the differences of migration speed and distribution of action among protein components in serum, with a form of band. The reagents manufactured by Sebia Company were applied in strict accordance with the instrument instructions and reagent instructions.

MPV, hs-CRP, cTnI, and LVEF test

The levels of MPV, hs-CRP, and cTnI in patients were detected after their emergency admission before antiplatelet and anticoagulant treatment. MPV was determined by the automatic blood analysis instrument LH750 (Beckman Coulter, Inc., U.S), hs-CRP by ELISA (the kit was produced by Orion Company, France), and cTnI by chemiluminescence immunoassay using Abbott luminescence instrument (American ARCHITECT i2000SR). Then, the echocardiography was performed in patients with relative stable heart function by the cardiac ultrasonic diagnostic instrument manufactured by GE company of the United States (model: Voluson 730 Expea) to record their LVEF.

Statistical methods

SPSS22.0 statistical software was used for data analyses, with measurement data expressed by $\bar{x} \pm sd$ and compared between groups using one-way ANOVA or t-test. χ^2 test was used to test the count data, and Pearson method was used to analyze the correlation between each index and serum LN and HA. A *P*-value less than 0.05 was considered statistically significant.

Results

LN and HA level in patients of the two groups

In comparison with the CHD group, the expression of serum LN and HA in patients of the infarction group presented a significantly higher level ($P < 0.01$), as shown in **Table 1**.

Testing results of LN and HA in patients with different coronary artery lesions

In the two groups, serum LN and HA were at the highest level in patients with three-vessel disease and the lowest level in patients with single-vessel disease (both $P < 0.05$), and significantly higher expression levels of LN and HA were obtained in the infarction group than those in the CHD group ($P < 0.05$), as shown in **Table 2**.

Testing results of LN and HA in patients with different infarction locations

Compared with the other two groups, the expression levels of LN and HA in patients with combined anterior and inferior wall infarction were remarkably higher ($P < 0.05$), and no significant difference was found in the expression levels of LN and HA in patients with either anterior wall infarction or inferior wall infarction ($P > 0.05$), as shown in **Table 3**.

Comparison of the expression of TC, LDL-C, HDLC, TG, and FPG between the two groups

In comparison with the CHD group, the infarction group showed higher expressions of TC, LDL-C, and TG and a lower expression of HDL-C (all $P < 0.05$), as shown in **Table 4**.

Correlation analysis of serum LN and HA with the expression of each test value

LN and HA were positively correlated with the expressions of TC, TG, hs-CRP, FPG, and LDL-C (all $P < 0.05$), and negatively correlated with HDL-C ($P < 0.05$), as shown in **Table 5**.

SPE test results

The two groups showed no significant differences in albumin, α1-globulin, α2-globulin, β2-

Table 2. Comparison of serum LN and HA expressions in patients with different numbers of coronary artery lesions ($\bar{x}$ ± sd)

Group		cases	LN (μg/L)	HA (μg/L)
CHD group	Single-vessel disease	120	176.8±22.84	150.7±19.1
	Double-vessel disease	100	186.9±24.79*	174.5±21.4*
	Three-vessel disease	60	202.8±33.57*,#	193.5±58.6*,#
	F		19.87	37.61
	P		< 0.001	< 0.001
Infarction group	Single-vessel disease	130	182.5±31.7Δ	165.3±42.1Δ
	Double-vessel disease	87	206.8±36.2*,Δ	185.3±50.7*,Δ
	Three-vessel disease	63	257.2±50.7*,#,Δ	237.2±64.3*,#,Δ
	F		81.64	43.17
	P value		< 0.001	< 0.001

Note: *P < 0.05 *vs.* single-vessel disease; ΔP < 0.05 *vs.* CHD group; #P < 0.05 *vs.* double-vessel disease.

Table 3. Comparison of LN and HA expressions in patients with different infarction locations ($\bar{x}$ ± sd)

Group	cases	LN (μg/L)	HA (μg/L)
Anterior wall area	104	232.4±30.4	191.8±48.6
Inferior wall area	122	236.4±31.4	192.6±42.9
anterior plus inferior wall	54	268.9±52.4*,#	239.7±68.2*,#
F		20.12	19.10
P-value		< 0.001	< 0.001

Note: *P < 0.05 *vs.* anterior wall infarction; #P < 0.05 *vs.* inferior wall infarction.

globulin, γ-globulin, and white/globulin ratio (P > 0.05), but the β1-globulin level in the infarction group was remarkably higher than that in the CHD group (P < 0.05), as shown in **Table 6**.

Relationship between post-discharge event rate and β1-globulin level in patients with CHD

The relationship between the elevation of β1-globulin and the prognosis of the patients was further assessed given the significant difference of β1-globulin between the two groups. In this study, patients with CHD were divided into a group of β1-globulin ≥ 6% and a group of β1-globulin < 6% for follow-up observation with an upper limit of a normal reference value of 6% β1-globulin. Due to the limited number of death cases in this study, the mortality and readmission rates were collectively considered as the event rates in order to avoid statistical bias. The follow-up results showed no significant difference in the event rates between the two groups 7 months after discharge (P > 0.05), and the event rate of the group of β1-globulin ≥ 6% increased with time. Additionally, 24 months after discharge, among the 30 patients with a β1-globulin level < 6%, 6 patients were readmitted or died, while among 70 patients with β1-globulin ≥ 6%, 20 were readmitted or died, as shown in **Figure 1**.

Correlation between MPV and LVEF, serum hs-CRP and cTnI in the infarction group

LVEF in the infarction group decreased with the increase of MPV level, which verified a negative linear correlation between MPV and LVEF (r_{LVEF} = -0.362, P < 0.05). Meanwhile, serum hs-CRP and cTnI levels rose with the increase of MPV level, which confirmed the positive correlation between MPV and hs-CRP, cTnI levels in the linear correlation analysis ($r_{hs\text{-}CRP}$ = 0.448, r_{CTnI} = 0.512, both P < 0.05), as shown in **Figures 2-4**.

Discussion

LN is a major protein of the basement membrane and a special extracellular matrix (ECM) of the arterial wall, whose modification may lead to endothelial dysfunction and changes in ECM structure and assembly, thus facilitating the occurrence of atherosclerosis [10, 11]. The high metabolism of HA may result in its instability, as HA increases in vascular plaque 2; accordingly, serum HA can be used to assess

Table 4. Comparison of the expressions of TC, LDL-C, HDL-C, TG and FPG in each group ($\bar{x}$ ± sd, mmol/L)

Group	cases	TC	TG	LDL-C	HDL-C	FPG
CHD group	280	5.09±1.49	2.46±1.12	3.79±0.89	1.59±0.34	5.35±1.55
Infarction group	280	6.25±1.85*	3.69±1.62*	4.53±1.13*	1.17±0.30*	6.41±2.24
t		8.171	10.45	8.609	15.50	6.511
P		< 0.001	< 0.001	< 0.001	< 0.001	< 0.001

Note: *P < 0.05 *vs.* CHD group.

Table 5. Correlation analysis between expression of LN and HA and test values

Group		TC	LDL-C	TG	FPG	HDL C
LN	r	0.373	0.375	0.314	0.295	-0.281
	P	0.026	0.025	0.028	0.032	0.034
HA	r	0.512	0.452	0.421	0.291	-0.298
	P	0.0073	0.013	0.018	0.045	0.035

Table 6. Comparison of the SPE results between the two groups

Project	Infarction group (n = 280)	CHD group (n = 280)	T	P
Albumin (%)	55.22±4.91	56.45±5.01	0.654	0.963
α1-globulin (%)	5.36±1.36	5.29±1.40	0.364	0.223
α2-globulin (%)	10.77±3.01	11.01±2.98	1.324	0.365
β1-globulin (%)	6.05±1.36	5.11±0.76	3.654	0.001
β2-globulin (%)	5.36±1.26	5.32±1.22	5.364	0.964
Gamma globulin (%)	17.36±3.40	17.11±3.26	0.587	0.256
White/ball ratio	1.42±0.26	1.39±0.25	1.364	0.541

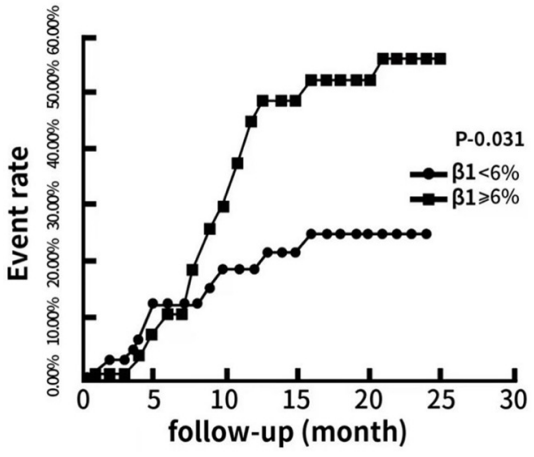

Figure 1. The curve of event rate within 24 months after discharge in the two groups.

the severity of myocardial fibrosis [12]. The role of HA in the formation of atherosclerosis has been proven to play an essential role in the early development of atherosclerosis. Preliminary studies have shown that the concentration of HA in the human aortic wall decreases with the development of atherosclerosis. Diffuse intimal thickening is related to the strong expression of HA around the fibrous plaques. HA levels elevate under inflammatory conditions, such as diabetes and vascular remodeling, which regulates the secretion of chemokines and cytokines by white blood cells. Moreover, it has also been confirmed that high expressions of HA and LN can promote smooth muscle fibrosis of organs, which demonstrates a correlation between the expression of HA and LN and myocardial fibrosis or even MI [13].

This study demonstrated higher expressions of serum LN and HA in patients with both MI and CHD than those in patients with only CHD (P < 0.01), indicating that MI patients are accompanied by higher expressions of LN and HA, which may stem from the development of coronary atherosclerosis into AMI after myocardial fibrosis under the high expressions of LN and HA. This study also found that the levels of serum LN and HA in the two groups were at the highest level in patients with three-vessel disease, and higher expressions of LN and HA were obtained in the infarction group than those in the CHD group, which pointed out the close relation of LN and HA with MI. LN exists in the basement membrane of the coronary vessels of the myocardium. Endothelial cells in the myocardium secrete a large amount of endothelin under the action of angio-

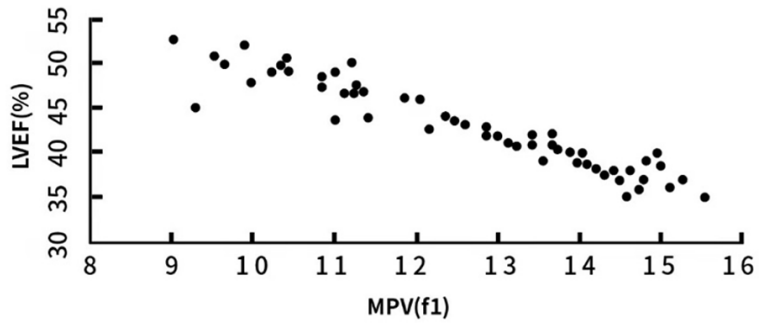

Figure 2. Correlation between MPV and LVEF.

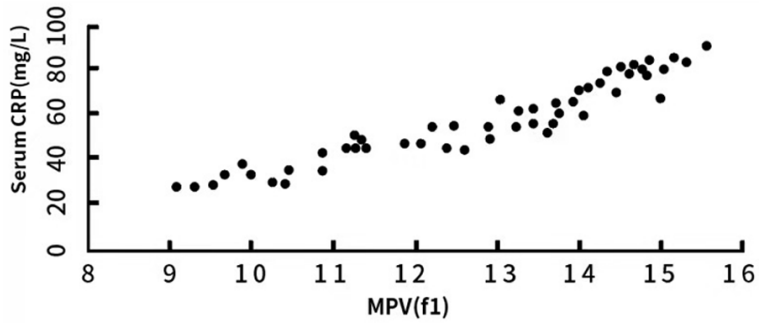

Figure 3. Correlation between MPV and hs-CRP.

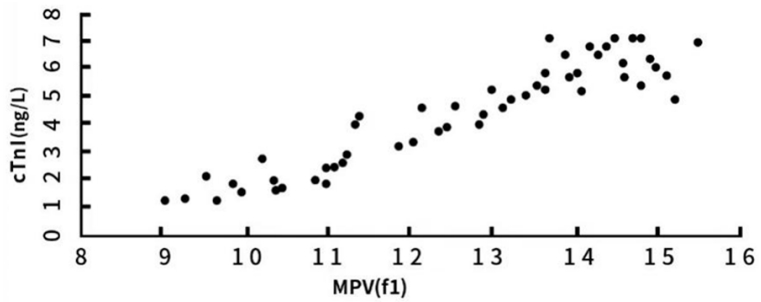

Figure 4. Correlation between MPV and cTnI.

tensin II during the process of myocardial fibrosis after MI, which degrades the type III collagen fibers on the LN, triggering the release of LN from the basement membrane into the blood. In addition, plasma enzymes, LN, etc. enter the interstitium through the blood vessel wall due to increased permeability of the blood vessel wall after MI and activate various growth factors to promote proliferation of fibroblasts in large numbers, which consequently produce a mass of HA, eventually giving rise to interstitial edema. Furthermore, the accumulation of HA, LN and other substances outside of myocardial cells and around the coronary arteries can increase the oxygen diffusion distance and impede myocardial blood supply. The results suggest a positive correlation between the number of coronary artery lesions and the levels of serum LN and HA. Additionally, the expressions of LN and HA in patients with combined anterior wall and inferior wall infarction were significantly higher than those in the other two groups (both $P < 0.05$), suggesting that the larger the infarct size, the higher the expressions of LN and HA, which is consistent with the conclusion of Patra et al. [14].

It has been pointed out that the abnormal expressions of blood lipid and blood glucose are considered risk factors for CHD and MI [15]. This study found higher expressions of TC, LDL-C, TG, and FPG and a lower expression of HDL-C in the infarction group than those in the control group (all $P < 0.05$), which further indicates that the aberrant expressions of blood lipid and blood glucose are interwoven with CHD comorbid with AMI, which is consistent with the results of prior studies [16]. LN and HA were positively correlated with the expression of TC, TG, hs-CRP, FPG, and LDL-C (all $P < 0.05$), and negatively correlated with HDL-C ($P < 0.05$). Relevant studies have revealed that the increase in LN and HA levels is positively correlated with arterial stiffness, which leads to vascular endothelial dysfunction and promotes myocardial fibrosis. In this study, reasons behind the relationship between LN and HA and the metabolism of blood lipids and blood glucose can be possibly concluded as follows: First, elevated serum HA and LN expressions aggravate the blood lipids and blood sugar levels of patients with CHD, and indirectly induce

MI by changing the metabolism of blood lipids and blood sugar. Second, elevated serum HA and LN expressions and abnormal blood lipids and blood glucose metabolism are positively correlated with MI, which is consistent with the results of this study [17, 18]. Moreover, a higher level of β1-globulin, which increases the readmission rate and mortality of patients with CHD complicated with MI, was detected in SPE results of patients with MI than that of patients with normal cardiac function, and MPV, being involved in the development and progression of MI, had a significant correlation with LVEF, hs-CRP and cTnI, suggesting that MPV can be used to evaluate the severity and prognosis of MI [19, 20]. However, some cases with normal MPV levels observed in this study may be attributed to the basic state of MPV or the platelet activation rate, which finding requires confirmation by further clinical trials.

Conclusion

In conclusion, the expressions of serum HA and LN in CHD patients complicated with MI increases along with the elevation of blood lipid and blood glucose levels, and the increase of β1-globulin level may result in a rise in the readmission rate and mortality of patients with both CHD and MI. Additionally, the elevated level of MPV is closely related to the levels of LVEF, hs-CRP, and cTnI, and MPV can be used as an objective indicator for the assessment of MI patients with crucial prognostic value.

Disclosure of conflict of interest

None.

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