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Original research

STUDY OF PERIOSTIN LEVELS IN PATIENTS BEFORE AND AFTER ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

Background: *There is a clear correlation between inflammatory outcome signs and adverse outcomes in patients after acute myocardial infarction. Periostin - an inflammatory biomarker in recent times promises to be an effective and necessary factor in predicting disease progression. This study described characteristics of serum periostin levels in patients with acute MI and some follow up results of this biomaker.*

Methods: *Study design: Analytical cross-sectional study. Non-probability, purposive sampling. The research subjects were divided into 2 groups: the group of patients diagnosed with acute MI (including 153 patients) and the remaining group was the control group (including 153 healthy people) . All patient groups and the control group were hospitalized and treated from September 2019 to March 2023.*

Results: *There was no difference in age, BMI and sex between the patient group and the control group ($p < 0.05$). The serum periostin level in acute MI patients was the highest (149.37ng/ml, IQR: 120.69 - 208.18), then the level at 3 months post-MI (77.69 ng/ml, IQR: 61.63 - 101.05), the control group's periostin level was the lowest (63.04 ng/ml, IQR: 40.96 - 80.98), the difference was statistically significant ($p < 0.05$). Median periostin level in Killip I group (132.28 ng/ml) was lower than in the remaining group (187.84 ng/ml) ($p < 0.05$). Mean serum periostin level in the LVEF < 50% group (208.15 ± 92.33 ng/ml) was significantly greater than that in the LVEF ≥ 50% group (136.92 ± 38.68 ng/ml), ($p < 0.05$).*

Conclusion: *Serum periostin levels increase in acute myocardial infarction and decrease gradually post myocardial infarction. There is a difference in admission periostin levels between preserverd and reduced EF groups.*

Keywords: *Periostin, myocardial infarction.*

I. BACKGROUND

Cardiovascular diseases, of which myocardial infarction (MI) is the main cause of morbidity and mortality in Western countries, are now rapidly becoming more common in developing and underdeveloped countries [1]. Up to 3 million people worldwide suffer from MI and 1 million people die from this disease every year [2]. According to the World Health Organization in 2016, it is estimated that 31% of deaths in Vietnam are due to cardiovascular disease, more than half of which are coronary artery disease [3]. Research on biomarkers in patients with MI has made great progress in recent years and a solution to this problem is expected to be found in the near future, possibly in the form of multi-marker assessment [4]. There are many biomarkers studied in patients with myocardial infarction such as troponin, myoglobin, CK-MB, h-FAPB, IMA, etc. However, in general, their role is still limited because they are mainly studied in the acute phase. There is a clear correlation between inflammatory outcome signs and adverse outcomes in patients after acute myocardial infarction. In that general trend, periostin - an inflammatory biomarker in recent times promises to be an effective and necessary factor in accurately predicting disease progression, helping to choose treatment and is a target in preventive care of coronary artery disease

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[5]. In the world, the role of periostin on the heart is increasingly studied in developed countries [6, 7]. In Vietnam, there is currently no research on the role of periostin in acute MI. Therefore, we conducted a study on the characteristics of serum periostin levels in patients with acute MI.

II. MATERIALS AND METHODS

2.1. Materials

The research subjects were divided into 2 groups: the group of patients diagnosed with acute MI and the remaining group was the control group. All patient groups and the control group were hospitalized and treated from September 2019 to March 2023.

Disease group: Including 153 patients diagnosed with acute MI treated at Hue Central Hospital and Vinh Long Hospital, including 55 patients with STEMI and 98 patients with NSTEMI.

Exclusion criteria for the disease group: moderate to severe valvular heart disease, concurrent inflammation, malignancy, hypertrophic cardiomyopathy, dilated cardiomyopathy, blood creatinine ≥ 4 mg/dL (353.6 µmol/L), disagreeing to participate in the study.

Control group: Including 153 healthy subjects who did not have acute MI (clinically no chest pain, hs-Troponin T within normal limits), who had regular health check-ups at Vinh Long hospital.

2.2. Methods

Study design: Analytical cross-sectional study. Non-probability, purposive sampling.

All patients will be clinically examined and assigned routine investigations during the patient's hospital stay, except for serum periostin levels which is performed 5-7 days after MI (levels at first time). Patients will be periodically re-examined after discharge. After 3 months of MI, an echocardiogram (assessing LVEF parameters) and a second periostin blood test will be performed (levels at second time). Serum periostin concentration testing is performed by enzyme-linked immunosorbent assay, which is ELISA (Enzyme-Linked ImmunoSorbent Assay) and serum periostin concentration quantification is performed at the Department of Immunology, University of Medicine and Pharmacy, Hue University with the Human Periostin Kit from MyBioSource.com.

Data processing: Data were processed using SPSS version 26 (IBM SPSS Statistics for Windows, Version 26.0), Medcalc version 22.017 and Excel 2013 software.

III. RESULTS

The baseline characteristics are listed in Table 1. There was no difference in age, BMI and sex between the patient group and the control group, $p > 0.05$. The serum periostin level in acute MI patients was the highest, the control group's periostin level was the lowest, $p < 0.001$ (Table 2). Periostin level in Killip I group was lower than in the remaining group, the difference was statistically significant with $p < 0.001$ (Table 3). Mean serum periostin level in the LVEF < 50% group was significantly greater than that in the LVEF ≥ 50% group, $p < 0.05$ (Figure 1).

Table 1: Common characteristics between the disease group and the control group

Characteristics		Disease group		Control group		p
		n	X ± SD (min-max)	n	X ± SD (min-max)	
BMI (kg/m^2)	Male	92	23.95 ± 3.44 (17,10 - 35,38)	92	23.60 ± 2.16 (18,49 - 28,76)	0.642
	Female	61	24.99 ± 4.18 (16.89 - 36.21)	61	25.43 ± 3.03 (15.77 - 37.46)	0.327
	Total	153	24.36 ± 3.77 (16.89 - 36.21)	153	24.33 ± 2.70 (18.49 - 30.82)	0.848

Characteristics		Disease group		Control group		p
		n	X ± SD (min-max)	n	X ± SD (min-max)	
Age (years)	Male	92	67.70 ± 12.70 (38.00 - 96.00)	92	69.10 ± 11.94 (47.00 - 88.00)	0.417
	Female	61	74.18 ± 10.67 (44.00 - 96.00)	61	72.13 ± 12.10 (50.00 - 90.00)	0.330
	Total	153	70.29 ± 12.32 (38.00 - 96.00)	153	70.31 ± 12.08 (47.00 - 90.00)	0.986
	Age groups	**n**	**%**	**n**	**%**	0.178
	< 60	29	18.95	37	24.18	
	60 - 69	52	33.99	35	22.88	
	70 - 79	32	20.92	34	22.22	
	≥ 80	40	26.14	47	30.72	
Gender (%)	Male	92	60.13	92	60.13	1.000
	Female	61	39.87	61	39.87	

Table 2: Periostin level between groups

Periostin (ng/mL)		n	Median	IQR	p
Disease group at 1st time	Male	92	142.41	117.17 - 203.56	0.236*
	Female	61	156.00	124.04 - 218.01	
Disease group at 2nd time	Male	92	73.61	59.07 - 97.20	0.158
	Female	61	78.61	66.74 - 112.63	
Comtrol group	Male	92	65.76	36.14 - 80.71	0.579
	Female	61	60.66	42.33 - 81.09	
Comparing	1st time		149.37	120.69 - 208.18	$p^{(a-b)} < 0.001$
	2nd time		77.69	61.63 - 101.05	
	Control		63.04	40.96 - 80.98	$p^{(a-c)} < 0.001$ $p^{(b-c)} < 0.001$

a: periostin concentration in the first patient group, b: periostin concentration in the second patient group, c: periostin concentration in the control group

Table 3: Periostin levels at admission and general characteristics

General characteristics		n	Periostin (ng/mL)		p
			Median	IQR	
Age	< 60	29	151.20	116.30 - 210.89	0.082
	60 - 69	52	132.28	113.93 - 179.29	
	70 - 79	32	175.49	124.82 - 223.07	
	≥ 80	40	159.84	126.06 - 205.64	
BMI (kg/m^2)	< 23	61	138.61	116.94 - 201.40	0.369
	23 - 24.9	27	179.29	127.22 - 234.66	
	≥ 25	65	147.78	120.93 - 207.52	
Killip class	I	93	132.28	114.56 - 180.94	< 0.001
	II - IV	60	187.84	132.85 - 235.16	
Total		153	149.37	120.69 - 208.18	

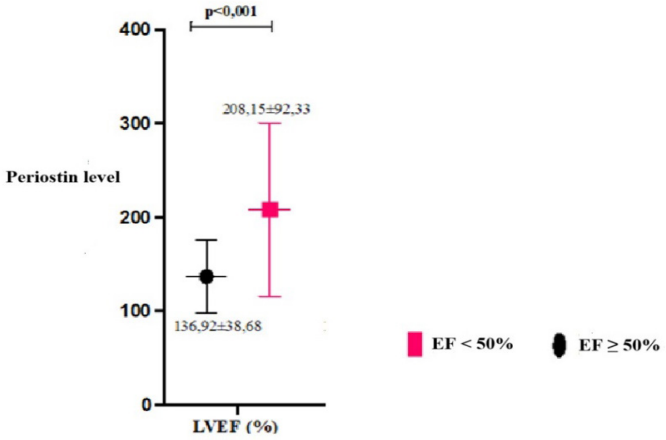

Figure 1: Mean periostin level at admission between EF groups on echocardiography 3 months after MI

IV. DISCUSSION

Regarding the general characteristics of the study subjects, in our study there was no difference in the ratio of gender, BMI and age between the two groups of patients and the control group (Table 1), which is similar to the subjects of studies related to periostin after acute MI in the world [8]. There is a change in serum periostin concentration over time after acute MI, from 2016 to present, it has been shown that although periostin concentration has increased from the first day of injury, it only peaks after 5 - 7 days of illness and then gradually decreases [9]. That is the basis for us to choose the time for the first serum periostin test on the 5 - 7th day of MI, when the periostin concentration reaches its maximum value. In our study, the serum periostin concentration in patients with acute MI at the initial test was the highest, with a median of 149,37 ng/mL (Table 2). The second test 3 months after acute MI, the median serum periostin concentration decreased to 77.69 ng/mL, while the periostin concentration in the control group was the lowest, with a median of 63.04 ng/mL, compared with the reference value of normal serum periostin concentration of < 100 ng/mL according to Mybiosource.com - the manufacturer of the serum periostin test kit. The median periostin

concentration in our control group was 63.04 ng/mL, not much different from the results of study of Evan Tan examining periostin levelsin Chinese adults with a median value of 57.0 ng/mL [10]. The study results were consistent with most previous literature [9], except Chi-Wen Cheng showed that periostin concentration decreased early after AMI, which is not similar to the natural progression of the disease and was not expected by the author himself [11]. However, the sample size in Chi-Wen Cheng's study was small (45 MI patients), so it is only for reference. Under normal conditions, serum periostin levels is very low, but when there is damage such as acute MI, periostin levels will increase rapidly. Research of Lin Ling et al(2014) recorded the median periostin concentration in acute MI patients as 188 ng/mL [12]. Periostin concentration peaked on days 5 to 7 after a acute MI and then gradually decreased over time [13]. Xinwei He et al studied patients with large-vessel atherosclerotic stroke, tested blood periostin levelsat 3 different times on days 1, 6, and 28 of stroke and found that periostin levelsbegan to increase from the first day of the disease, reached the highest concentration on day 6, and remained higher than normal after stroke for at least 28 days [14].

According to Caswell-Smith, periostin levels did not differ by age or common comorbidities other than respiratory diseases [15]. We noted no difference in serum periostin levels by age group (Table 3). In addition, there was no difference in periostin levels by BMI classification. This suggests that serum periostin levels are independent of age and body mass index. Meanwhile, periostin levels in the Killip I classification group were lower than in the Killip II - IV classification group, suggesting that serum periostin levels are more likely to correlate with disease severity. Regarding the difference of serum periostin in echocardiographic parameters after 3 months of MI: after the patient has an acute MI, periostin stimulates excessive myocardial remodeling, leading to a state of fibrotic scar tissue that does not function to replace the damaged myocardial area, which will reduce heart function, reduce LVEF, reduce cardiac contractility and lead to expansion of the size of the heart chamber, shown by an increase in the diameter of the heart chamber on echocardiography. The more periostin is produced, the more obvious the remodeling process becomes. The study results showed that the serum periostin concentration in the LVEF $\geq$ 50% group was much lower than that in the remaining group (Figure 1). This contributes to the demonstration that periostin is associated with cardiac remodeling after acute MI.

V. CONCLUSION

Serum periostin levels increase in acute myocardial infarction and decrease gradually post myocardial infarction. There is a difference in admission periostin levels between preserverd and reduced EF groups.

Ethics approval

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Hue Central hospital.

Competing interests

The authors declare that they have no competing interests.

REFERENCE

1. Weil BR, Neelamegham S. Selectins and Immune Cells in Acute Myocardial Infarction and Post-infarction Ventricular Remodeling: Pathophysiology and Novel Treatments. Front Immunol. 2019; 10: 300.
2. Mechanic OJ, Gavin M, Grossman SA. Acute Myocardial Infarction. StatPearls 2023; Available from: https://www.ncbi.nlm.nih.gov/books/NBK459269/
3. Pham Manh Hung, Clinical cardiology. 2019, Hanoi, Vietnam: Medical Publishing House.
4. Tilea I, Varga A, Serban RC. Past, Present, and Future of Blood Biomarkers for the Diagnosis of Acute Myocardial Infarction-Promises and Challenges. Diagnostics (Basel). 2021; 11(5): 881.
5. Seropian IM, Sonnino C, Van Tassell BW, Biasucci LM, Abbate A. Inflammatory markers in ST-elevation acute myocardial infarction. Eur Heart J Acute Cardiovasc Care. 2016; 5(4): 382-395.
6. Iekushi K, Taniyama Y, Azuma J, Katsuragi N, Dosaka N, Sanada F. Novel mechanisms of valsartan on the treatment of acute myocardial infarction through inhibition of the antiadhesion molecule periostin. Hypertension. 2007; 49(6): 1409-1414.

7. Pickering ME, Oris C, Chapurlat R. Periostin in Osteoporosis and Cardiovascular Disease. J Endocr Soc. 2023; 7(7): bvad081.
8. Liu X, Zhang Z, Yekeben H, Yumaierjiang B. Periostin as a Potential Biomarker of Acute Myocardial Infarction. J Biol Regul Homeost Agents. 2023; 37(3): 1561–1571.
9. Walker JT, McLeod K, Kim S, Conway SJ, Hamilton DW. Periostin as a multifunctional modulator of the wound healing response. Cell Tissue Res. 2016; 365(3): 453-65.
10. Tan E, Varughese R, Semprini R, Montgomery HC, Olsson J, et al. Serum periostin levels in adults of Chinese descent: an observational study. Allergy Asthma Clin Immunol. 2018; 14: 87.
11. Cheng CW, Wang CH, Lee JF, Kuo LT CW. Levels of blood periostin decrease after acute myocardial infarction and are negatively associated with ventricular function after 3 months. J Investig Med. 2012; 60(2): 523-8.
12. Ling L, Cheng Y, Ding L, Yang X. Association of serum periostin with cardiac function and short-term prognosis in acute myocardial infarction patients. PLoS One. 2014; 9(2): e88755.
13. Balbi C, Milano G, Fertig TE, Lazzarini BS, Taniyama Y, Sanada F, et al. An exosomal-carried short periostin isoform induces cardiomyocyte proliferation. Theranostics. 2021; 11(12): 5634-5649.
14. He X, Bao Y, Shen Y, Wang E HW, Ke S, Jin X. Longitudinal evaluation of serum periostin levels in patients after large-artery atherosclerotic stroke: A prospective observational study. Sci Rep. 8(1): 11729.
15. Caswell-Smith R, Hosking A, Cripps T, Holweg MJ, Holliday M, Maillot C, et al. Reference ranges for serum periostin in a population without asthma or chronic obstructive pulmonary disease. Clin Exp Allergy. 2016; 46(10): 1303-14.