246

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Research Paper

The changes of Hs-CRP and WBC count after percutaneous coronary intervention in different types of coronary heart diseases

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Abstract

Objective: To investigate the regulation of High sensitive C-reactive protein(Hs-CRP) and WBC count in patients with coronary heart disease(CHD) by percutaneous transluminal intervention(PCI) and to discuss the mechanism of inflammatory reaction after coronary stenting. **Methods**:127 patients who received successful percutaneous transluminal coronary stenting, were divided into groups of stable angina(SAP), unstable angina(UAP), and acute myocardial infarction(AMI) according to their clinical types. Another 41 stable angina patients with more than 70% of coronary artery tenosis who did not receive PCI served as control. Serum Hs-CRP levels and WBC count were determined before intervention, 3 days and 7 days post PCI and the data were analyzed statistically by *t*-test. **Results**: There showed no difference in clinical baseline characteristics between groups. The serum Hs-CRP level and WBC count was gradually raised in the UAP and AMI group(how about SAP group, and had no difference in CAG group and SAP group). After PCI serum Hs-CRP levels and WBC counts were significantly higher in the SAP group than in the coronary angiography group(CAG) at 3 days and had no difference at 7 days. In the UAP and AMI group, the serum Hs-CRP level at 3 days and 7 days declined obviously, however serum WBC count did not decrease apparently. **Conclusion**: The serum Hs-CRP level and WBC count elevate transiently after PCI. There are different inflammatory reactions in different types of coronary heart diseases after coronary stenting procedure.

Key words: high sensitive C-reactive protein(Hs-CRP); coronary heart disease; percutaneous transluminal intervention(PCI); WBC count

INTRODUCTION

Coronary heart disease(CHD) is the leading cause of death due to atherosclerosis. Inflammation is a key feature of atherosclerosis and its clinical manifestation[1]. Hs-CRP and WBC count are markers of inflammation that are widely available in clinical practice. Prospective studies have shown that hs-CRP and WBC count may be useful prognostic indicators in patients with UA or AMI[2-4]. Stent implantation is an effective and safe means of treatment for CHD. Serum Hs-CRP level and WBC is a strong predictor of risk for future reocclusion and thrombus after PCI[5,6]. However, the role of Hs-CRP and WBC in the inflammatory reaction after coronary stenting remains unclear.

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MATERIALS AND METHODS

Patient population

The study population consisted of 127 consecutive patients with CHD, who underwent coronary angiography for diagnosis and successful percutaneous transluminal coronary stenting in the Department of Cardiology, Jiangsu People's hospital, Nanjing. Patients were divided into groups of stable angina(SAP), unstable angina(UAP) and acute myocardial infarction (AMI) according to their clinical types. For the purpose of the study, another 41 stable angina patients with more than 70% of coronary artery stenosis were recruited as control group. They had not received PCI because they were unwilling or had some economic burden. Serum Hs-CRP levels and WBC count were determined before and 3 days, 7 days after PCI. Exclusion criteria were as follows: 1, any presence of any luminal steno-

sis <70% 2, the patients with tumor, infection and immune system diseases 3, patients had an unexplained elevation in the creatine kinase level that was more than three times the upper limit of normal and was not related to myocardial infarction, or had a reatinine level of more than 2.0mg per deciliter.

Patients received standard medical and inteventional treatment for CHD, including aspirin, clopidogrel, statins, β-Blockers, ACEI or ARB. Heparin 8000-10000U was infused before PCI. Low molecular heparin was routinely subcutaneously injected for 3-5 days after coronary stenting. Serum Hs-CRP levels and WBC count were determined before intervention 3 days and 7 days after PCI.

Laboratory measurements

The 12-hour fasting blood samples were drawn in the morning at admission to the ward and the serum were stored at -70℃ immediately after centrifugation until being assayed. All laboratory measurements were conducted at the central clinical laboratory in the First Affiliated Hospital of Nanjing Medical University. The Hs-CRP was measured by particle enhanced immunoturbidimetric assay(Orion Diagnostica, Finland) on an automated autoanalyzer(AU 2700 Olympus, 1st Chemical Ltd, Japan) and the measurement range and detection limit of this test was 0.25-10 mg/L and 0.25 mg/L respectively. If the result exceeded the measurement range, sample was diluted with 0.9% NaCl. The total cholesterol(TC, mmol/L), triglyceride(TG, mmol/L), fasting blood glucose(FBG, mmol/L), fasting high-density lipoprotein cholesterol(HDL-c, mmol/L), fasting low-density lipoprotein cholesterol(LDL-c, mmol/L) were determined by enzymatic procedures on an automated autoanalyzer(AU 2700 Olympus, the 1st Chemical Ltd, Japan). WBC was also measured on an automated autoanalyzer(AU 2700 Olympus, the 1st Chemical Ltd, Japan).

Statistical analysis

Analysis system software SPSS 12.0 was used for all analysis. Data of BMI, age, Hs-CRP and WBC count were normally distributed parameters and presented as mean ± SD and the data were analyzed statistically by T-test. A level of $P < 0.05$ was considered as significant difference. All P values were two-tailed.

RESULTS

The baseline clinical characteristics and CHD risk factors of the 168 patients who were included in the analysis were similar between the four treatment groups (Table 1). Baseline plasma Hs-CRP and WBC count was significantly higher in UAP and AMI group than group SAP($P < 0.05$). After a PCI serum WBC and Hs-CRP count, levels were observed as obviously higher in the SAP group than in the CAG group at 3 days and restored to the baseline at 7 days. In the UAP group plasma Hs-CRP descended at 7days but the WBC count had no significant change. The serum Hs-CRP level descended at 3 days and 7 days but the WBC count didn't descend together in AMI group(Table 2).

DISCUSSION

The association between elevated C-reactive protein levels and cardiovascular events may be related to the degree of coronary plaque inflammation and instability.[7,8]

Table 1 Comparison of the baseline clinical characteristics and CHD risk factors of four group of patients

characteristic	CAG(n = 41)	SAP(n = 48)	UAP(n = 43)	AMI(n = 36)
Male sex(%)	32(78.0)	35(72.9)	33(76.7)	28(77.8)
Age-yr	63.42 ± 9.53	68.75 ± 9.52	64.71 ± 11.21	66.82 ± 8.96
Current smoker(%)	7(17.0)	10(20.8)	6(13.9)	6(16.7)
BMI	22.81 ± 4.31	24.59 ± 3.87	26.76 ± 2.98	24.89 ± 3.55
Hypertension(%)	28(68.2)	33(68.7)	30(69.7)	22(61.1)
Diabetes mellitus(%)	9(21.9)	8(16.7)	8(18.6)	10(27.8)
Hyperlipidemia(%)	28(68.2)	26(54.2)	28(65.1)	26(72.2)
Family history(%)	4(9.8)	4(8.3)	3(6.9)	2(55.6)

$P > 0.05$: no differences among four groups.

Table 2 The changes of Hs-CRP and WBC count after PCI in different types of CHD

group	n	baseline		3days		7days	
		Hs-CRP(mg/L)	WBC count($\times 10^9$)	Hs-CRP(mg/L)	WBC count($\times 10^9$)	Hs-CRP(mg/L)	WBC count($\times 10^9$)
CAG	41	1.22 ± 0.45	6.31 ± 1.42	3.89 ± 3.23	6.14 ± 2.07	1.24 ± 1.43	6.51 ± 1.78
SAP	48	1.27 ± 0.69	6.01 ± 2.17	*13.62 ± 10.6	*12.8 ± 10.61	3.1 ± 1.12	7.57 ± 3.45
UAP	43	*8.11 ± 1.57	8.04 ± 2.43	11.87 ± 10.4	9.01 ± 2.50	b3.74 ± 1.65	8.11 ± 2.11
AMI	36	*17.3 ± 7.62	11.2 ± 3.73	a11.21 ± 9.07	10.91 ± 2.34	a7.17 ± 4.28	11.04 ± 3.19

*$P < 0.05$ Compared with the group CAG; a$P < 0.01$ Compared with the baseline; b$P < 0.05$ Compared with the baseline.

An increased WBC is also a marker for increased cardiovascular risk related to inflammation[9]. The same conclusion was found in our study. Baseline plasma Hs-CRP and WBC count was significantly higher in the UAP and AMI group than in the SAP group($P < 0.05$) and the AMI group was the highest in the three groups. Reocclusion and thrombus was the two frequent problems and both may be related to the inflammatory reaction after coronary stenting[10]. The serum Hs-CRP level is a strong predictor of risk for future reocclusion and thrombus after PCI and Hs-CRP also plays an important role in determining atherosclerotic plaque vulnerability[11,12]. But it is not clear what kind of role WBC plays in the inflammatory reaction after coronary stenting. The present study was designed to explore the inflammatory response to the coronary stenting.

Experimental data indicated that Hs-CRP levels were obviously higher after PCI and majority show the elevation will last for 72 hours[13]. The same result was found in SAP group. The main reason for elevation of serum Hs-CRP level and WBC count in SAP group may be as follows:①Duing the operation balloon angioplasty or Stent implantation can damage the vascular endothelial cells. ② Intraoperative plaque was crushed to rupture and inflammatory factors released into blood serum. ③Intraoperative microthrombus obstructed the capillary vessel and caused injury to cardiac muscle. But serum Hs-CRP level and WBC count recovered to the normal level at 7 days suggested the inflammatory reaction after coronary stenting was transient.

In the AMI group Hs-CRP serum level at 3 days may likely benefit from statins and other medicine[14,15]. The serum Hs-CRP level descended more at 7 days. In the UAP group the serum Hs-CRP level at 3 days showed no difference to the baseline, which can be explained because statins and other medicine counteracted the effect of coronary stenting on Hs-CRP level. In contrast to the Hs-CRP results, WBC count didn't descent after coronary stenting. Data from others have shown the Interleukin-6(IL-6) level of about 60% unstable angina patients were higher than of normal people, which can induce the increased production of Hs-CRP from the liver[16-17]. Neutrophils and monocytes-macrophages play an important role in triggering the initiation of the inflammatory response. Interleukin-8(IL-8) is a powerful leukocyte chemotactic factor and has a potent chemoattractant activity for neutrophil, IL-8 induces upregulation of CR1 receptors in human neutrophil and promote the adhesion of neutrophils to vascular endothelial cells. Subsequently the neutrophils migrated into the ischemic myocardium[18-20]. So the above-mentioned result can be explained; that IL-6, Hs-CRP, IL-8 and WBC all played important role in the inflammatory reaction after coronary stenting in the SAP group, but only IL-6 and Hs-CRP performed the main role in the UAP and AMI group. In other words, there are different inflammatory reactions in different types of coronary heart diseases after coronary stenting. However, these explanations are presumptive, so the accurate mechanisms remain to be further elucidated.

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