



# Effect of PCI on inflammatory factors, cTnI, MMP-9 and NT-pro BNP in patients with unstable angina pectoris

Ke-Tong Liu\*, Xin Wang, Di Zhao

Third Department of Cardiology, The Third Affiliated Hospital of Qiqihar Medical University, Heilongjiang, Qiqihar 161000, China

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## ABSTRACT

**Objective:** To investigate the effect of PCI on inflammatory factors, cTnI, MMP-9 and NT-pro BNP in patients with unstable angina pectoris. **Methods:** A total of 80 unstable angina pectoris patients were divided into observation group (40 cases) and control group (40 cases). The observation group was given the therapy of PCI, and the control group was given coronary angiography. To observe the of inflammatory factors, cTnI, MMP-9 and NT-pro BNP were tested and compared before and after operation. **Results:** At 24 h after operation, CRP and IL-18 levels were increased significantly after treatment in operation groups, there was no difference on inflammatory factors in control group, and had significant difference on inflammatory factors in two groups; At 24 h after operation, cTnI, MMP-9 and NT-pro BNP levels were increased significantly after treatment in operation groups, there was no difference on inflammatory factors in control group, and had significant difference on inflammatory factors in two groups. **Conclusion:** PCI therapy can induce inflammation and myocardial injury in patients with unstable angina pectoris.

## 1. Introduction

Unstable angina pectoris, which is the intermediate clinical syndrome between chronic stable angina and acute myocardial infarction, is a common acute cardiac event of coronary artery atherosclerosis[1–3]. Percutaneous coronary intervention (PCI) is a very effective treatment method for coronary syndrome. It can stabilize the coronary flow, reduce the cardiac damage and improve the prognosis[4–6]. However, there are still exist the case of inflammatory response and myocardial injury in PCI treatment. In our study, 40 patients with unstable angina pectoris were treated with PCI and we aims to explore the effect of PCI on inflammatory factors, cardiac troponin I (cTnI), matrix metalloproteinase-9 (MMP-9) and N-terminal pro brain natriuretic peptide precursor (NT-pro BNP) in patients with unstable angina pectoris.

## 2. Materials and methods

### 2.1. General information

Selected 80 cases patients with unstable angina pectoris for diagnosis and treatment of coronary angiography (CAG) in the Third Affiliated Hospital of Qiqihar Medical College from October 2014 to September 2015 as the research object. The patients were randomly divided into the observation group (40 cases) and the control group (40 cases). The observation group with male 25 cases, female 15 cases, age from 55 to 75 years old with an average ( $63.41 \pm 12.11$ ) years. The control group with male 26 cases, female 14 cases, age from 54 to 77 years old with an average ( $64.67 \pm 11.35$ ) years. Inclusion criteria: all patients were CAG clear with the main branch was not less than 70% or the left main trunk was not less than 50%; excluded patients with liver, lung and renal failure; excluded patients with cardiac shock and cardiac arrhythmias; excluded patients with hematologic diseases and malignant tumor; Excluded pregnancy, lactation patients; All patients were voluntarily joined and signed the informed consent. Two groups patients were comparable in age, gender and other general information.

\*Corresponding author: Ke-Tong Liu, Third Department of Cardiology, The Third Affiliated Hospital of Qiqihar Medical University, Heilongjiang, Qiqihar 161000, China.

Tel: 18714323683

Email: liuketong838@163.com

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## 2.2. Method

All patients underwent CAG and were treated with conventional drug therapy: aspirin 100 mg/d, clopidogrel 75 mg/d. The observation group was treated with PCI and the control group was treated with CAG.

## 2.3. Observation indexes

Before and 24 h after surgery, 6 mL fasting venous blood were taken from patients, injected into anticoagulant tubes, separated the plasma by centrifugal (4 °C, 3 000 r/min, 10 min), and the plasma were stored at the refrigerator(-20 °C). Plasma levels of inflammatory cytokines CRP, IL-18 and cTnI, MMP-9 and NT-pro BNP were measured by enzyme-linked immunosorbent assay (ELISA).

## 2.4. Statistical treatment

Using SPSS 18 software for statistical analysis, The measurement data were expressed by (Mean ± SD) and using the chi-square test, using *t* test to analyze the differences between groups and paired *t* test to analyze the differences within group, *P*<0.05 was considered the difference to be statistically significant.

## 3. Results

### 3.1. The changes of inflammatory factors in two groups patients before and after operation

Before operation, the level of inflammatory factors CRP and IL-18 in the observation group was (6.53±2.57) mg/L and (32.31±5.81) µg/L respectively while the CRP and IL-18 level in the control group was (6.49±2.55) mg/L and (33.09±7.47) µg/L; Postoperative 24 h, the CRP and IL-18 level in the observation group was (7.41±2.49) mg/L and (39.21±14.57) µg/L respectively while the CRP and IL-18 level in the control group was (6.14±2.64) mg/L and (30.24±8.87) µg/L. Before operation, there were no significant differences between the plasma inflammatory factors levels in the two groups (*P*>0.05); Postoperative 24 h, the serum inflammatory factors CRP and IL-18 levels were significantly higher in the observation group (*P*<0.05), while had no significant changes in the control group (*P*>0.05), and there were significant differences between the two groups (*P*<0.05). See table 1.

**Table 2**

The changes of cTnI, MMP-9 and NT-pro BNP in two groups patients before and after operation.

Group	<i>n</i>	Time	cTnI (µg/L)	MMP-9 (µg/L)	NT-pro BNP (ng/L)
Observation	40	Before Surgery	23.61±6.53	43.83±11.85	330.21 ± 178.22
		Postoperative 24 h	30.62±7.86 <sup>ab</sup>	52.93±18.04 <sup>ab</sup>	558.61 ± 303.24 <sup>ab</sup>
Control	40	Before Surgery	23.21±6.82	45.64±13.77	328.65 ± 176.81
		Postoperative 24 h	22.32±6.97	44.04±14.79	319.63 ± 172.74

Ps: Compared with before surgery, <sup>a</sup>*P*<0.05; Compared with the control group, <sup>b</sup>*P*<0.05.

**Table 1**

The changes of inflammatory factors in two groups patients before and after operation.

Group	<i>n</i>	Time	CRP (mg/L)	IL-18 (µg/L)
Observation	40	Before Surgery	6.53±2.57	32.31±5.81
		Postoperative 24 h	7.41±2.49 <sup>ab</sup>	39.21±14.57 <sup>ab</sup>
Control	40	Before Surgery	6.49±2.55	33.09±7.47
		Postoperative 24 h	6.14±2.64	30.24±8.87

Ps: Compared with before surgery, <sup>a</sup>*P*<0.05; Compared with the control group, <sup>b</sup>*P*<0.05.

### 3.2. The changes of cTnI, MMP-9 and NT-pro BNP in two groups patients before and after operation

Before operation, the plasma cTnI, MMP-9 and NT-pro BNP level in the observation group was (23.61±6.53) µg/L, (43.83±11.85) µg/L and (330.21±178.22) ng/L respectively while the cTnI, MMP-9 and NT-pro BNP level in the control group was (23.21±6.82) µg/L, (45.64±13.77) µg/L and (328.65±176.81) ng/L respectively; Postoperative 24 h, the plasma cTnI, MMP-9 and NT-pro BNP level in the observation group was (30.62±7.86) µg/L, (52.93±18.04) µg/L and (558.61±303.24) ng/L respectively while the cTnI, MMP-9 and NT-pro BNP level in the control group was (22.32±6.97) µg/L, (44.04±14.79) µg/L and (319.63±172.74) ng/L respectively. Before operation, there were no significant differences between the plasma cTnI, MMP-9 and NT-pro BNP levels in the two groups (*P*>0.05); Postoperative 24 h, the plasma cTnI, MMP-9 and NT-pro BNP levels were significantly higher in the observation group (*P*<0.05), while had no significant changes in the control group (*P*>0.05), and there were significant differences between the two groups (*P*<0.05). See table 2.

## 4. Discussions

Unstable angina pectoris, which is the intermediate clinical syndrome between chronic stable angina and acute myocardial infarction, is an acute cardiac event of coronary heart disease. It is characterized by an increase angina pectoris symptoms, a new onset of rest or nocturnal angina or a prolonged duration of angina pectoris[7-9]. Patients are likely develop to acute myocardial infarction if with no timely and appropriate treatment. PCI[10,11] is an important and effective treatment for coronary syndrome, it only needs to make a small incision in the skin, the catheter with a balloon dilator inserted into the foot or hand artery, send to the narrow

coronary artery for expansion. Patients with unstable angina pectoris through PCI to restore stability of coronary blood flow, reduce heart damage, improve the prognosis and reduce the mortality rate.

Inflammation plays an important role in the occurrence and development of coronary heart disease. The active inflammatory reaction that in the vulnerable plaque of coronary artery causes plaque rupture and thrombosis, then exacerbate coronary artery stenosis and thereby increase the incidence of acute coronary events such as unstable angina or acute myocardial infarction[12,13]. Many studies have indicated that inflammatory factors CRP and IL-18 were closely related to the acute coronary syndrome such as unstable coronary atherosclerotic plaque and unstable angina pectoris[14]. CRP[15] is an acute phase protein with very low concentration in normal people, it is closely related to the degree of trauma, sooner than the changes of body temperature and peripheral blood white cell counts, its an independent indicator of acute phase response after trauma and can reflect the inflammation of coronary artery diseases after the exclusion of other factors that causes CRP increases. IL-18[16] is an intermediate molecule that induces interferon synthesis. It can stimulate the proliferation of T cells and enhance natural killer cell activity, it participate in the cytokines production and has the synergistic effect with interleukin-12. Our study shows that before operation, there was no significant difference in plasma inflammatory factor CRP and IL-18 levels between the two groups ( $P>0.05$ ); postoperative 24 h, the plasma inflammatory factor CRP and IL-18 levels were significantly increased in the observation group (patients with PCI) ( $P<0.05$ ) while had no significant changes ( $P>0.05$ ) in the control group (patients with CAG), and the difference between the two groups was significant ( $P<0.05$ ). which indicates that PCI may cause or aggravate the patient's own inflammatory reaction, and cause adverse reactions to the myocardium and coronary vessels, detection of inflammatory cytokines may be an important indicator of cardiovascular events after PCI.

cTnI[17] is a structural protein in the myocardium and has become an important index to evaluate the myocardial injury. MMP-9 [18] is a kind of gelatinase, it regulates the activity of other proteases and cytokines and plays a crucial role in the occurrence and development of atherosclerosis, stroke and other vascular diseases. MMP-9 can hydrolysis extracellular matrix, so it plays an important role in the remodeling of extracellular matrix collagen, MMP and Ca ions jointly promote ventricular remodeling. Both NT-pro BNP[19] and BNP are sensitive indicators of cardiac function, and the nature of NT-pro BNP is more stable and its plasma concentration is higher, it has reported that NT-pro BNP had a close relationship with myocardial ischemia[20]. Our study shows that before operation, there was no significant difference in plasma cTnI, MMP-9 and NT-pro BNP levels between the two groups ( $P>0.05$ ); postoperative 24 h, the plasma cTnI, MMP-9 and NT-pro BNP levels were significantly increased in the observation group ( $P<0.05$ ) while had no significant

changes in the control group ( $P>0.05$ ), and the difference between the two groups was significant ( $P<0.05$ ). The possible mechanism may be that the rapid rupture of the plaque, occlusion of the side branch vessel and thrombosis of the coronary artery cause the minor myocardial injury or transient myocardial ischemia.

In summary, PCI as an effective treatment method for coronary heart disease will also cause or aggravate the inflammatory reaction, and cause the abnormal level of cTnI, MMP-9 and NT-pro BNP. Timely monitoring the various indicators of patients will help to improve the level of myocardial injury, reduce the inflammatory response, and judge the patient's prognosis, so we need to take active measures to prevent the adverse reactions after PCI.

## References

- [1] Magalhaes MA, Minha S, Chen F. Response to letter regarding article, Clinical presentation and outcomes of coronary in-stent restenosis across 3-stent generations. *Circ Cardiovas Interv* 2015; **8**(4): 210-214.
- [2] Li Yan. Observe the NT-proBNP level changes before and after percutaneous coronary intervention for patients with unstable angina pectoris. *Chin Med Care Aged* 2015; **24**(1): 46-46, 47.
- [3] Lee K, Yoo SY, Suh J. Efficacy of cilostazol on inhibition of platelet aggregation, inflammation and myonecrosis in acute coronary syndrome patients undergoing percutaneous coronary intervention: the accel-loading-acs (accelerated inhibition of platelet aggregation, inflammation and myonecrosis by adjunctive cilostazol loading in patients with acute coronary syndrome) study. *Int J Cardiol* 2015; **190**(2): 370-375.
- [4] Kluz K, Parenica J, Kubkova L. Unstable angina pectoris prior to ST elevation myocardial infarction in patients treated with primary percutaneous coronary intervention has no influence on prognosis. *Biomed Papers Med Faculty Univ Palacky Olomouc Czechoslovakia* 2015; **159**(2): 251-258.
- [5] Kizilirmak F, Gunes HM, Demir GG. Impact of intracoronary adenosine on myonecrosis in patients with unstable angina pectoris undergoing percutaneous coronary intervention. *Cardiovasc Drugs Ther* 2015; **29**(6): 519-526.
- [6] Wang Linqing. Study on the relationship between C reactive protein and prognosis in patients with unstable angina after interventional therapy. *Chin J Modern Med* 2015; **32**(2): 40-41.
- [7] Giustino G, Baber U, Stefanini GG. Impact of clinical presentation (stable angina pectoris vs unstable angina pectoris or non-st-elevation myocardial infarction vs st-elevation myocardial infarction) on long-term outcomes in women undergoing percutaneous coronary intervention with drug-eluting stents. *Am J Cardiol* 2015; **116**(6): 845-852.
- [8] Zhao Junwen. Effect of coronary intervention on blood flow and inflammatory factors in patients with coronary heart disease. *Pract J Clin Med* 2015; **19**(5): 1-4.
- [9] Zhang XL, Chi YH, Wang le F. Systemic inflammation in patients with

- chronic obstructive pulmonary disease undergoing percutaneous coronary intervention. *Respirology* 2014; **19**(5): 723-729.
- [10]Sun F, Yin Z, Shi Q. Effect of short-term high-dose atorvastatin on systemic inflammatory response and myocardial ischemic injury in patients with unstable angina pectoris undergoing percutaneous coronary intervention. *Chin Medical J* 2014; **127**(21): 3732-3737.
- [11]Loh JP, Pendyala LK. Comparison of outcomes after percutaneous coronary intervention among different coronary subsets (stable and unstable angina pectoris and ST-segment and non-ST-segment myocardial infarction). *Am J Cardiol* 2014; **113**(11): 1794-1801.
- [12]Chen Lei, Liu Xianxia, Zhang Yuansheng. Analysis of the related factors of myocardial injury in patients with coronary heart disease during perioperative period of PCI. *J Hainan Med* 2015; **13**(15): 2218-2220.
- [13]Li Fangjiang, Wang Xiaoyuan, Du Meiling. The Changes and significance of serum inflammatory markers in patients with acute coronary syndrome after percutaneous coronary intervention. *J Clin Department Internal Med* 2015; **16**(3): 178-180.
- [14]Liu HL, Yang Y, Yang SL. Administration of a loading dose of atorvastatin before percutaneous coronary intervention prevents inflammation and reduces myocardial injury in STEMI patients: a randomized clinical study. *Clin Ther* 2013; **35**(3): 261-272.
- [15]Li Q, Deng SB, Xia S. Impact of intensive statin use on the level of inflammation and platelet activation in stable angina after percutaneous coronary intervention: a clinical study. *Med Clin* 2013; **140**(12): 532-536.
- [16]Yang Huanyu, Liu Fangcao, Cao Xiaoyu. The merits and demerits of PCI operation in the treatment of coronary heart disease. *Capital Food Med* 2015; **27**(5): 60.
- [17]Jeong YH, Tantry US, Min JH. Influence of platelet reactivity and inflammation on peri-procedural myonecrosis in East Asian patients undergoing elective percutaneous coronary intervention. *Int J Cardiol* 2013; **168**(1): 427-435.
- [18]Liu Yongsheng, Jiang Hua, Liu Wenwei. Effect of percutaneous coronary intervention on serum interleukin-18 and matrix metalloproteinase -9 levels for patients with unstable angina pectoris. *J Microcirculation* 2013; **23**(4): 34-36.
- [19]Pelliccia F, Del Prete G, Del Prete A. Effects of percutaneous coronary intervention and stenting with different drug-eluting coatings and platforms on endothelial damage and inflammation. *Int J Cardiol* 2012; **156**(2): 242-243.
- [20]Hu Zhangle, Wang Xiaochen, Xu Banglong. Effect and significance of percutaneous coronary intervention on plasma MMP-9, CRP, IL-18 and CTnI in patients with unstable angina pectoris. *J Clin Pathol* 2015; **44**(7): 1351-1355.