Different impacts of C-reactive protein and lipid profile on coronary lesions following a percutaneous coronary intervention

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Objective **In-stent restenosis (ISR) and lesion progression are major obstacles for a percutaneous coronary intervention (PCI). Although previous studies have suggested that inflammation and lipid profile may be involved in those pathophysiological events, it remains controversial to date. The aim of the present study was to investigate the impacts of inflammation and lipid profile on both ISR and lesion progression in patients receiving PCI and scheduled follow-up.**

Methods **A retrospective analysis of 513 patients was performed in patients who underwent PCI and received coronary angiography again at an average of 7 months. The data of lipid profile and C-reactive protein (CRP) at both pre-PCI and follow-up were analyzed in patients with 94 ISR group and 65 lesion progression (progression group) alone, which was compared with 307 patients with neither ISR nor lesion progression (control group).**

Results **CRP levels at pre-PCI in the ISR group were higher than those in the control group ($P<0.05$). The multivariate analysis indicated that the CRP levels at both pre-PCI and follow-up were significantly correlated with ISR [odds ratio (OR)=1.095, 95% confidence interval (CI) 1.005–1.194 for pre-PCI, OR=1.156, 95% CI 1.054–1.267 for follow-up, $P<0.05$, respectively]. When the cut-off of CRP was 2 mg/l, logistic regression analysis suggested an increased risk of ISR in patients with greater than 2 mg/l (OR=1.89, 95% CI 1.031–3.465) at pre-PCI CRP. The levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and non-high-density lipoprotein cholesterol (non-HDL-C) at follow-up in the progression group were higher than those in the control group ($P<0.05$, respectively). Logistic regression showed that the risk for lesion progression was associated with the concentrations of TC, LDL-C, and non-HDL-C ($P<0.05$).**

Conclusion **The levels of pre-PCI CRP were strongly associated with ISR, whereas diabetes, serum levels of TC, LDL-C, and non-HDL-C were significantly correlated with coronary lesion progression. *Coron Artery Dis* 23:181–187**

Coronary Artery Disease 2012, **23**:181–187

Keywords: inflammation, in-stent restenosis, lesion progression, lipid, percutaneous coronary intervention

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Received 25 November 2011 **Revised** 3 January 2012
Accepted 19 January 2012

Introduction

Percutaneous coronary intervention (PCI) is a major therapeutic method for coronary artery disease (CAD) in current cardiovascular practice. Although the recent clinical introduction of drug-eluting stent (DES) has reduced the occurrence of restenosis, in-stent restenosis (ISR) and progression of native coronary atherosclerotic lesions still remain major clinical issues after PCI, which is associated with a series of complex pathophysiological mechanisms involving dyslipidemia and inflammation. Several previous studies have suggested that inflammation and abnormal serum lipid metabolism might play important roles in the occurrence and progression of nontarget coronary lesions and ISR [1–4]. Accordingly, the application of inflammatory or lipid biomarkers that may reliably predict atherosclerotic progression in such a large group of patients is of paramount clinical importance. In addition, the pathogenesis of ISR may differ from that of progression of nontarget lesions, which may present as different clinical contributing factors. Furthermore, whether the effects of inflammation and lipid metabolism abnormality are comparable between post-PCI ISR and lesion progression of atherosclerosis remains controversial to date.

In this retrospective study, we evaluated the impacts of C-reactive protein (CRP) and serum lipid parameters on ISR and progression of nontarget coronary lesions in patients who had received PCI and angiographic follow-up at 7 months; the data were collected at both baseline and follow-up at 8 months.

Methods

Patients

Patients who underwent successful PCI with stent implantation between April 2005 and January 2006 in Fu Wai Hospital and received scheduled follow-up coronary reangiography after 3 months to 1 year were

0954-6928 © 2012 Wolters Kluwer Health | Lippincott Williams & Wilkins DOI: 10.1097/MCA.0b013e3283519f44

retrospectively surveyed. Patients who had complications such as coronary artery dissection or no reflow phenomenon or other serious complications during the PCI procedure were excluded. Patients who received PCI after coronary bypass or ISR, and those who had other medical conditions such as infection and tumor were also excluded. Finally, 513 patients were enrolled in this study, and they were followed up for a mean period of 7.4 ± 2.2 months after PCI. They were divided into the following groups: patients with ISR alone (ISR group, $n = 94$), patients with progression of other nontarget coronary lesions alone (progression group, $n = 65$), patients with overlapped lesions (with both ISR and lesion progression, $n = 30$), patients with coronary restenosis following coronary balloon angioplasty alone ($n = 17$), and patients with neither restenosis nor lesion progression (control group, $n = 307$). The ISR group, the progression group, and the control group were subjected to analyses in the current study.

The baseline characteristics of the ISR group, the progression group, and the control group were well matched, except for the stent type used and the percentage of patients with diabetes mellitus (Table 1).

Coronary angiography and percutaneous coronary intervention

Selective coronary angiography was performed using the Judkins technique and multiangle radiography. The image analysis was programmed and evaluated by two independent experienced interventional cardiologists. The criteria of successful coronary stent implantation included residual narrowing less than 30%, thrombolysis in MI flow grade III, and no complications. A nontarget coronary lesion was defined as vessels that did not receive balloon angioplasty and/or stenting. Two coronary segments (in-stent and in-segment) were assessed for ISR analysis; the in-stent analysis comprised only the segment of the lesion encompassing the stent and the in-segment was defined as the in-stent segment plus 5 mm proximal and distal of segments to the edge of the stent. The minimal lumen diameter and the percentage of diameter stenosis were measured for each segment. Images of the coronary tree were obtained by routine standardized projections using the Digital Integris CV System (Philips Electronics, Amsterdam, the Netherlands). For each segment, the measurements were carried out on end-diastolic frames that indicated the maximal stenosis. ISR was defined as at least 50% of the diameter of stenosis at the stented target lesion site (in-stent and/or in-segment) by the follow-up angiography. Progression of atherosclerosis was defined as an increase by at least 25% in luminal diameter stenosis of any nontarget native coronary lesion or new-onset lesion, resulting in an angiographically significant lesion ($\geq$ 50% luminal diameter stenosis) on a previously no-lesion segment at follow-up reangiography [5,6].

Table 1 Baseline characteristics of the patients

Variables	ISR group ($N=94$)	Progression group ($N=65$)	Control group ($N=307$)	P-value
Male [n (%)]	80 (85%)	57 (88%)	256 (83%)	0.66
Age (years)	58.57 ± 10.33	57.46 ± 10.84	56.85 ± 10.52	0.38
Hypertension [n (%)]	61 (65%)	40 (62%)	181 (60%)	0.58
Diabetes [n (%)]	28 (30%)	24 (37%)	70 (23%)	0.05
History of smoking [n (%)]	62 (66%)	48 (74%)	204 (66%)	0.47
Number of lesion vessels [n (%)]				0.83
1 vessel	25 (27%)	20 (31%)	100 (33%)	
2 vessels	32 (34%)	23 (35%)	100 (33%)	
3 vessels	37 (39%)	22 (34%)	107 (34%)	
Stent types [n (%)]				0.00
DES	34 (36%)		225 (73%)	
BMS	33 (35%)		35 (11%)	
Both	27 (29%)		47 (15%)	
Medications [n(%)]				
Aspirin	94 (100%)	65 (100%)	307 (100%)	1.00
Statins	83 (88%)	53 (82%)	271 (88%)	0.35
β-Blocker	70 (74%)	49 (75%)	223 (73%)	0.87
ACEI/ARB	39 (41%)	26 (40%)	116 (38%)	0.80

Values are mean ± SD or numbers (percentages).
ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMS, bare-mental stent; DES, drug-eluted stent; ISR, in-stent restenosis.

C-reactive protein and lipid measurements

EDTA-anticoagulated peripheral blood samples were taken after a 12 h overnight fast at day 1 during the initial hospitalization before the PCI procedure and at the time of follow-up before quantitative coronary angiography (CAG) in all study patients and plasma was obtained after centrifugation of 3000 rpm at 4°C for 15 min. Serum total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were determined by enzyme assays using a fully automatic biochemical analyzer (Beckmann DXC800, Bera, California, USA). The level of non-HDL-C was derived from TC after subtracting HDL-C. Serum CRP levels were measured using an immunoturbidometric method (Beckmann Assay 360, Bera, California, USA). The interassay coefficients of variation were 4.4 and 4.8%, respectively, and the intraassay coefficients were 3.5 and 5.1%, respectively.

Statistical analysis

The data for continuous variables with a normal distribution were expressed as mean ± SD of the mean. CRP was checked as a non-normal distribution and expressed as median (interquartile intervals) [$M(Q_R)$]. Categorical data were expressed as percentages. Normality of the continuous variables was analyzed using the Kolmogorov–Smirnov test. Differences in continuous variables among the groups were assessed by an independent t-test and the Mann–Whitney U or the Wilcoxon test. TG was analyzed following the logarithmic transformation. Categorical data and proportions were analyzed by the χ^2-test or Fisher's exact test. Then the correlations of significant markers with ISR or lesion progression were further examined by logistic multivariate regression analysis. Odds ratios and

95% confidence intervals were calculated. A *P*-value (two tailed) less than or equal to 0.05 was considered statistically significant; all probability values were two tailed. The SPSS11.5 statistical software package (SPSS Inc., Chicago, Illinois, USA) was used for all data management and statistics.

Results

Baseline characteristics

The baseline characteristics of the ISR group, the progression group, and the control group are shown in Table 1. There were no significant differences in the baseline characteristics among the three groups, except for the stent type used and the percentage of patients with diabetes mellitus. As shown in Table 1, stent type varied significantly between the ISR group and the control group ($\chi^2 = 46.22$, $P < 0.001$), whereas the rate of concurrent diabetes mellitus differed significantly between the progression group and the control group ($\chi^2 = 5.67$, $P < 0.05$). The number of patients with a history of smoking seemed higher in the progression group but no statistical significance was found.

Changes in serum lipid and inflammation markers

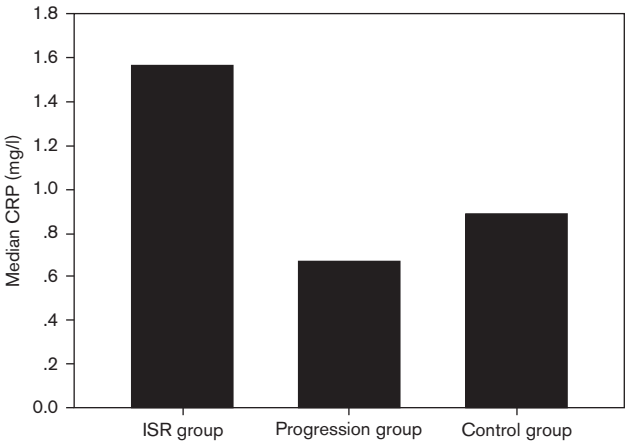
The data of the serum lipid profile and inflammation markers at pre-PCI and at the follow-up visit from the three groups are shown in Table 2. Compared with the control group, the CRP levels of pre-PCI were significantly higher in the patients of the ISR group ($P < 0.05$, Fig. 1). Serum lipid parameters of the ISR group, the progression group, and the control group obtained at the follow-up visit changed significantly compared with the data obtained at baseline. However, the levels of TC, LDL-C, and non-HDL-C at the follow-up visit were significantly higher in patients of the progression group than those in the control group ($P \leq 0.05$, Fig. 2). In addition, the levels of TC were higher in patients in the progression group than those in the ISR group at the time of the follow-up visit ($P < 0.05$).

Table 2 Comparisons of serum lipid profile and C-reactive protein levels among the patients

Variables	ISR group ($N=94$)	Progression group ($N=65$)	Control group ($N=307$)	*P*-value
Pre-PCI				
TC (mmol/l)	4.66 ± 1.03	4.83 ± 1.20	4.62 ± 1.06	0.36
TG (mmol/l)	1.52 ± 0.89	1.69 ± 1.00	1.71 ± 1.21	0.27
HDL-C (mmol/l)	1.05 ± 0.28	1.03 ± 0.26	1.04 ± 0.29	0.91
LDL-C (mmol/l)	2.58 ± 0.79	2.69 ± 1.00	2.51 ± 0.81	0.27
Non-HDL-C (mmol/l)	3.61 ± 1.02	3.80 ± 1.19	3.58 ± 1.02	0.30
CRP (mg/l)	1.56 (3.81)[a]	0.67 (4.25)	0.89 (2.37)	0.10 ([a]0.04)
At follow-up				
TC (mmol/l)	4.25 ± 0.89	4.62 ± 1.14[a]	4.26 ± 1.01	0.03 ([a]0.01)
TG (mmol/l)	1.59 ± 0.87	1.77 ± 0.98	1.58 ± 0.90	0.18
HDL-C (mmol/l)	1.05 ± 0.27	1.10 ± 0.32	1.06 ± 0.25	0.47
LDL-C (mmol/l)	2.27 ± 0.66	2.51 ± 0.93[a]	2.25 ± 0.75	0.04 ([a]0.02)
Non-HDL-C (mmol/l)	3.21 ± 0.84	3.52 ± 1.12[a]	3.20 ± 0.98	0.05 ([a]0.02)
CRP (mg/l)	0.99 (2.11)	0.95 (1.95)	0.93 (1.24)	0.30

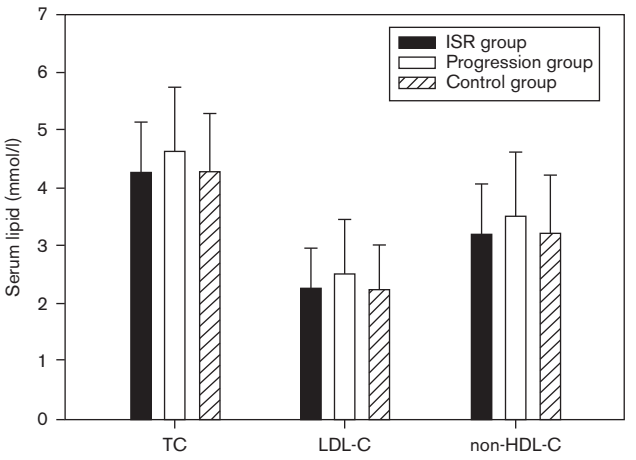
Values are mean ± SD or median (interquartile interval, calculated by the 75th percentage minus the 25th percentage).
CRP, C-reactive protein; HDL-C, high-density lipoprotein-cholesterol; ISR, in-stent restenosis; LDL-C, low-density lipoprotein-cholesterol; PCI, percutaneous coronary intervention; TC, total cholesterol; TG, triglyceride.
[a]Compared with that in the control group.

Fig. 1



Comparison of C-reactive protein (CRP) levels at pre-percutaneous coronary intervention (PCI) in the in-stent restenosis (ISR) group, the progression group, and the control group.

Fig. 2



Comparison of serum total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-C), and non-high-density lipoprotein-cholesterol (non-HDL-C) levels at follow-up of the in-stent restenosis (ISR) group, the progression group, and the control group.

Multivariate logistic regression of in-stent restenosis

The correlation of the above significant markers to the risk of ISR was further analyzed using a multivariate logistic regression model. The data indicated that the CRP levels of pre-PCI and the follow-up visit were significantly correlated with ISR, and were an independent risk factor for ISR after adjusting for sex, age, hypertension, diabetes mellitus, history of smoking, vessel number of coronary lesion, and stent type ($P < 0.05$, Table 3).

The patients were classified into the two categories according to the pre-PCI CRP level, namely, greater than 2 mg/l group and less than 2 mg/l group, with the cut-off of CRP being 2 mg/l. The rate of ISR was significantly higher in patients in the CRP greater than 2 mg/l group than those in the less than or equal to 2 mg/l group (25.8 vs. 15.5%, respectively, $\chi^2 = 4.32$, $P < 0.05$, Fig. 3). Logistic regression analysis suggested that patients with higher levels of CRP had a greater risk of ISR compared with patients with lower levels of CRP [odds ratio = 1.89, 95% confidence interval (1.031–3.465), $P < 0.05$].

Multivariate logistic regression of lesion progression

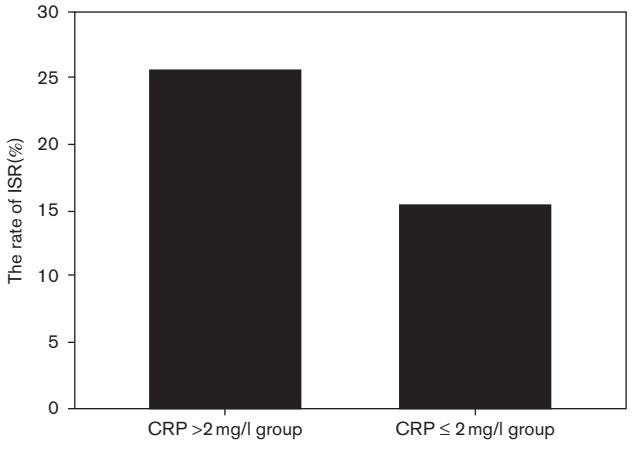
The correlation of the above significant markers to lesion progression of atherosclerosis was also analyzed by a logistic regression model. After adjusting for sex, age, hypertension, diabetes mellitus, history of smoking, and vessel number of coronary disease, the data showed that TC, LDL-C, and non-HDL-C levels at the follow-up visit were significantly correlated with lesion progression ($P < 0.05$, Table 3).

Table 3 Logistic regression analysis of risk factors for in-stent restenosis and lesion progression

Variables	OR	95% CI	*P*-value
ISR			
Pre-PCI CRP	1.095	1.005–1.194	0.037
Follow-up CRP	1.156	1.054–1.267	0.002
Lesion progression			
Follow-up TC	1.376	1.072–1.767	0.012
Follow-up LDL-C	1.505	1.090–2.079	0.013
Follow-up non-HDL-C	1.345	1.036–1.746	0.026

Risk factors adjusted: sex, age, hypertension, diabetes mellitus, history of smoking, vessel number of coronary lesion, and stent type.
CI, confidence interval; CRP, C-reactive protein; HDL-C, high-density lipoprotein-cholesterol; ISR, in-stent restenosis; LDL-C, low-density lipoprotein-cholesterol; OR, odds ratio; PCI, percutaneous coronary intervention; TC, total cholesterol.

Fig. 3



Comparison of the rate of in-stent restenosis (ISR) in patients with pre-PCI C-reactive protein (CRP) levels greater than 2 mg/l and CRP less than or equal to 2 mg/l. PCI, percutaneous coronary intervention.

Discussion

In this study, data showed that the CRP levels of pre-PCI were strongly associated with ISR, whereas diabetes, serum levels of TC, LDL-C, and non-HDL-C were significantly correlated with nontarget coronary lesion progression. Obviously, the present study confirmed and extended the results obtained in previous investigations on the relationship between the pathogenesis of ISR or lesion progression and inflammation markers or lipid profile.

PCI is currently thought to be one of the primary and important therapeutic methods of CAD. However, either ISR or progression of nontarget coronary lesions that require vascular reconstruction will undoubtedly affect patients physically and psychologically and place a huge financial burden on the healthcare system. Therefore, long-term secondary prevention following PCI to decrease the readmission rate is a major concern of cardiovascular medicine. It has been shown that inflammation and abnormal lipid metabolism play critical roles in the occurrence and progression of CAD [2,4]. However, it remains unclear whether their effects are comparable between ISR and progression of nontarget coronary lesions.

In-stent restenosis after percutaneous coronary intervention

Our study retrospectively reviewed patients who were followed up for coronary angiography within 3 months to 1 year following a successful PCI with stenting. First, our findings showed that the type of stent in use varied significantly between the ISR group and the control group. Bare-metal stents were more frequently used in the ISR group compared with the control group. This finding was in accordance with a previously reported study [7].

In fact, the mechanism of ISR is a very complex pathophysiological process. It has been demonstrated that inflammation was associated not only with the occurrence of coronary atherosclerosis and cardiovascular events but also with ISR. CRP, a marker of systemic inflammation, has been suggested to predict clinical and angiographic outcome after PCI [8,9]. However, there are still conflicting data regarding the prognostic value of CRP. In analyzing the risk factors of ISR, our study showed that serum levels of pre-PCI CRP in the ISR group were significantly higher than those in the control group in a level-dependent manner. When the cut-off was set at 2 mg/l of pre-PCI CRP, the rate of ISR was significantly higher in patients with greater than 2 mg/l pre-PCI CRP levels than that in patients with less than or equal to 2 mg/l levels, suggesting that patients who exhibited a higher level of pre-PCI CRP were at a greater risk of ISR than those with a lower CRP. Moreover, multivariate logistic regression indicated that CRP levels at the follow-up visit after PCI were also significantly correlated with ISR, which was an independent risk factor

for ISR. It might be concluded from our study that persistent pre-PCI and post-PCI inflammatory status was a major risk factor of ISR, and CRP might be of important predictable value for ISR.

Some previous studies have demonstrated that inflammation markers such as plasma CRP and IL-6 levels at several hours and days after stenting significantly increased compared with the baseline, suggesting that PCI could trigger a local and systemic inflammatory response, whose magnitude was associated with PCI complications [10]. Chyrchel *et al.* [11] reported on the acute inflammatory response induced by stent implantation, manifesting as an increase in CRP levels following PCI. They found that the amplitude of the increase in CRP following DES implantation was lower than that following bare-metal stent implantation, suggesting that the anti-inflammatory activity of the DES partly contributed to the reduction in ISR following PCI.

Statins have been reported to have a potent anti-inflammatory activity besides modulating the lipid profile [11]. The ARNMYDA-RECAPTURE study showed that statin at a loading dose before PCI could significantly reduce the frequency of cardiovascular events after PCI [12]. Regardless of the serum lipid level, intensive use of statin should be continuously given to patients after PCI according to recent guideline, the benefits of which can be obtained from their pleiotropic effects including anti-inflammation. Although statins were used in over 80% of the patients, a substantial number of patients suffered from ISR, suggesting that ISR was not only a complex pathophysiological process but also a subject for investigation. Accordingly, more possible therapeutic approaches to reduce inflammatory burden with the aim of improving clinical and angiographic outcomes after PCI should be further explored. Interestingly, Pesarini *et al.* [13] reported that a high dose of oral prednisone could reduce proinflammatory cytokine release in circulating activated monocytes of patients treated with coronary stenting, and tumor necrosis factor-alpha release reduction was correlated with decreased late luminal loss. A randomized-controlled trial evaluating the relationship with endothelial function and inflammation also indicated that high-intensity interval training could reduce ISR following PCI with stent implantation [14].

Dyslipidemia is a well-defined risk factor of atherosclerosis. However, the current study did not show an association between lipid profile including serum levels of TC, LDL-C, HDL-C, TG, non-HDL-C, and ISR. Zairis *et al.* [4] have previously reported that higher levels of serum HDL-C were significantly associated with a lower incidence of ISR as well as major adverse cardiac events. In Kim *et al.*'s [15] study, the LDL subfraction and lipid profiles were measured in 274 patients at both baseline and follow-up CAG. They found that the incidence of ISR was lower in patients with increased LDL particle size than in those with no change or decrease in LDL particle size. Logistic multivariate analysis revealed that a decrease in HDL-C or an increase in LDL particle size was an independent predictor for ISR. An increase in the LDL particle size between baseline and follow-up CAG was associated with a reduced incidence of ISR. They concluded that the modification of LDL particle size had a beneficial effect on the risk of ISR. Kamitani *et al.* [16] showed that plasma Lp (a) concentrations in patients with ISR were higher than those in patients without ISR, whereas other lipid parameters were similar in both groups. Subanalysis of the plasma Lp (a) concentrations, it was found that the rate of ISR in the highest Lp (a) group (> 40 mg/dl) was greater than that in the intermediate Lp (a) group (10–40 mg/dl) and the low Lp (a) group (< 10 mg/dl), indicating that plasma Lp (a) concentration might be an independent predictor of ISR.

Progression of nontarget coronary lesions

It has been shown that ISR differed from progression of atherosclerotic lesions in pathophysiology and mechanisms. On the basis of this, the present study assessed the potential factors in patients with CAD following stenting, which may influence the process of either ISR or progression of nontarget coronary atherosclerotic lesions. First, our study showed that patients with progression of atherosclerotic lesions were more likely to have complications of diabetes mellitus, indicating that diabetes mellitus was a risk factor of progression of atherosclerotic lesions.

Second, there was no difference in either the pre-PCI or the post-PCI levels of CRP between the progression group and the control group, which was clearly different from that of the ISR group. However, patients with lesion progression had significantly higher levels of TC, LDL-C, and non-HDL-C than patients in the control group at the time of angiographic follow-up. This finding suggested that lipid profiles such as serum levels of TC, LDL-C, and non-HDL-C levels were the principal risk factors of nontarget coronary atherosclerotic aggravation. In other words, long-term intensive management of serum lipid profile may be required in CAD patients, especially after PCI, to slow down atherosclerotic progression. Although more than 80% of our patients were treated with statins, the status of serum lipid varied considerably. At the follow-up visit, the mean LDL-C level was 2.52 mmol/l in the progression group and 2.25 mmol/l in the control group. Moreover, the mean non-HDL-C level in the progression group was 3.51 mmol/l and 3.20 mmol/l in the control group. Therefore, a further reduction in LDL-C should be attempted, and the management of non-HDL-C should not be overlooked.

LDL-C is one of the well-established and most important risk factors for patients with CAD, and it has been demonstrated that LDL-C-lowering therapy could significantly decrease the rate of cardiac events in patients with CAD, even in those who have undergone revascularization. Moreover, recent studies have suggested that the

predictive value of non-HDL-C for cardiovascular risk and mortality is better than that of LDL-C [17–19]. A meta-analysis performed by Robinson *et al*. [20] showed that each 1% decrease in non-HDL-C resulted in an estimated 4.5-year CHD relative risk of 0.99 for statins, indicating that non-HDL-C was an important target of therapy for CHD prevention. Similarly, the National Cholesterol Education Program Adult Treatment Panel III guidelines have identified non-HDL-C as a secondary target of therapy after the attainment of LDL-C target goals [21]. In patients with CAD or CAD risk equivalents, an optional non-HDL-C goal is less than 100 mg/dl. A study by Orakzai *et al*. [22] showed that non-HDL-C was more strongly associated with subclinical atherosclerosis than other conventional lipid parameters. They concluded that non-HDL-C might be an important treatment target in primary prevention. Park *et al*. [23] investigated the independent predictors of patients with clinically driven PCI due to the progression of pre-existing non-culprit coronary lesions, and found a larger number of significant coronary lesions, low HDL-C, hypercholesterolemia, a history of PCI, and increased TG levels at the time of baseline PCI to be important. The data from Uchida *et al*. [24] showed that the serum Lp (a) level was higher in patients with progression of lesions compared with patients with regression of lesions. Also, remnant-like lipoprotein particle-cholesterol and apolipoprotein-B levels were higher in the progression group. Multiple regression analysis of the lesion progression showed that the progression–regression score was independently correlated with Lp (a). This finding indicated that Lp (a) was an independent predictor of coronary atherosclerotic lesion progression. Accordingly, long-term intensive management of serum lipid profile including TC, LDL-C, non-HDL-C, and Lp (a) might be critical to inhibit the progression of coronary atherosclerosis and improve the clinical outcomes in patients with CAD following PCI.

The major limitations of the current study are its retrospective nature, which could result in several issues including confounding factors and bias from a retrospective study, selection bias in scheduled follow-up patients, failure to characterize coronary lesions as well as plaques before PCI, and analysis of the PCI process and stents, which require further study in a prospective manner.

Conclusion

In the present study, the data showed that increased levels of CRP following PCI were an important risk factor of ISR, whereas concurrent diabetes mellitus and serum levels of TC, LDL-C, and non-HDL-C were associated with the progression of nontarget coronary lesions. These findings suggest that appropriate control of serum lipid parameters as well as inflammatory markers may be a useful target for long-term secondary prevention in patients who have received PCI treatment.

Acknowledgements

This article was partly supported by the National Natural Scientific Foundation (30670861, 30871055, 81070171), the Beijing Natural Scientific Foundation (7082081), and the Specialized Research Fund for the Doctoral Program of Higher Education of China (20060023044, 20070023047) awarded by Dr Jian-Jun Li, MD, PhD. The authors would like to thank Dr Gaoqiang Xie and Dr Yueran Tang at the Center of Biostatistics of Fu Wai Hospital for their data management and statistical assistance.

Conflicts of interest

There are no conflicts of interest.

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