Homocysteine and Restenosis After Percutaneous Coronary Intervention

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Abstract

Numerous clinical studies in Western and Asian countries suggest that individuals with elevated blood levels of homocysteine have an increased risk of atherosclerosis, myocardial infarction, cerebral infarction, and deep vein thrombosis. Homocysteine is also known to induce both atherogenic and thrombogenic mediators in cultured vascular cells so that homocysteine may influence the damage of endothelial cells, promote smooth muscle cell growth, induce atherogenic mediators and thrombus formation after coronary angioplasty. The association between homocysteine and restenosis after percutaneous coronary intervention (PCI) has been discussed. In this study, the relationship between plasma homocysteine levels and restenosis after PCI to investigate whether plasma homocysteine levels may be a predictor of restenosis after PCI was examined. One hundred consecutive patients who underwent successful PCI were enrolled and plasma homocysteine level was measured in all patients prior to PCI. Plasma for homocysteine level was obtained in 99 of 100 patients who had angioplasty. The mean plasma homocysteine concentration in the enrolled patients was 13.61±6.04 μmol/L. The minimum and maximum of plasma homocysteine were 4.40 μmol/L and 50.00 μmol/L, respectively. In healthy subjects, the normal reference range of homocysteine level is 5-15 μmol/L However, recent data suggest that some patients may be at increased cardiovascular and cerebrovascular risk at levels as low as 12 μmol/L. For this reason, both cut off points of homocysteine level ≥ 15 μmol/L or ≥ 12 μmol/L to identify the high homocysteine level group were used. Of 99 patients, high homocysteine level (≥ 15 μmol/L) was established in 9 patients with restenosis *versus* 20 patients without restenosis. If the cut off point of homocysteine level ≥ 12 μmol/L was used, high homocysteine level was established in 14 patients with restenosis *versus*

39 patients without restenosis. From both cut off points of homocysteine level, there was no correlation between plasma homocysteine level and the restenosis group. ($p>0.05$).

Key word : Plasma Homocysteine, Restenosis, Percutaneous Coronary Intervention

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Percutaneous coronary intervention (PCI) is an established and effective technique for treating coronary artery disease. Despite multiple advances in the field of interventional cardiology and new pharmacological agents to prevent restenosis, approximately one-third of the patients have this problem within 6 months after PCI[1,2]. Several factors such as diabetes[3-5], unstable angina[6], some lesion related factors and procedural related factors[2] clearly impact the likelihood of restenosis but some factors are controversial.

Restenosis is regarded as the result of a combination of various pathological events including neointimal formation and arterial remodeling. The mechanisms are complex and not completely understood. Thus, the identification of novel risk factors would enable us to setup a more effective therapeutic strategy to ameliorate the outcome of PCI.

Numerous clinical studies in Western and Asian countries suggest that individuals with elevated blood levels of homocysteine have an increased risk of atherosclerosis, myocardial infarction, cerebral infarction, and deep vein thrombosis[7-19]. Homocysteine is also known to induce both atherogenic and thrombogenic mediators in cultured vascular cells so that homocysteine may influence the damage of endothelial cells[20], promote smooth muscle cell growth(21), induce atherogenic mediators and thrombus formation after coronary angioplasty. A few studies have discussed the association between homocysteine and restenosis after PCI[22,23]. The present study examined the relationship between plasma homocysteine levels and restenosis after PCI to investigate whether plasma homocysteine levels may be a predictor of restenosis after PCI.

METHOD

Study Population

This study was performed in Her Majesty Cardiac Center, Siriraj Hospital. Patients who were treated with PCI successfully between October 14, 1999 and July 31, 2000 were included in the study. The hospital ethics commitee approved this study protocol and all patients gave informed consent to obtain blood samples for further studies if research laboratory available for measure some factors in his/her specimens. Exclusion criteria were patients who would not permit blood samples to be drawn and emergency or unplanned PCI. From that period, 100 consecutive patients who underwent successful PCI (residual stenosis immediately after PCI of < 50 per cent and had no major complications: death, acute myocardial infarction or emergency coronary artery bypass surgery) of one or more native coronary arteries.

Definition of restenosis

Angiographic criteria and/or clinical criteria were used to define the restenosis within 6 months after PCI[24,25]. The angiographic criteria was defined by repeated angiography at 6 months or earlier

associated with ≥ 50 per cent stenosis at the site of angioplasty. The clinical criteria was defined at 6 months or earlier by recurrent angina pain; pain characteristic was the same as pain before being treated with PCI, death, acute myocardial infarction or abnormal noninvasive exercise or nuclear stress test (26-31). After the 6-month observation period, each patient was classified by a cardiologist who was unaware of the outcome of the laboratory tests to define the clinical outcome of the study. The patients who fitted one of two restenosis criteria were enrolled in the restenosis group and those who did not fit in the restenosis group were enrolled in the non-restenosis group.

Laboratory examinations

This study was part of an overall effort at this institution to identify new risk factors for restenosis. Fasting blood samples were drawn before PCI. Serum samples were frozen at -70°C and measurement of homocysteine levels was performed within 1 year of blood drawing.

In all the enrolled patients, plasma homocysteine level was measured before undergoing PCI. Test for homocysteine levels was done by the Fluorescence Polarization Immunoassay (FPIA) method with a commercially available kit (IMx Homocysteine Assay, Abbott, USA).

Statistical analysis

Statistical analysis was performed on a personal computer using the Microsoft Excel version 2000 and SPSS software package version 10.0.7). In healthy subjects, the normal reference range of homocysteine level is 5-15 μmol/L. However, recent data suggest that some patients may be at increased cardiovascular and cerebrovascular risk at levels as low as 12 μmol/L. For this reason, both cut off points of homocysteine level equal to 15 μmol/L or above or ≥ 12 μmol/L to identify high homocysteine level group were used(32). The high homocysteine level group and normal homocysteine level group were statistically tested using chi-square test. Data were expressed as mean±SD or nominal number. Patients with and without restenosis were compared with unpaired student's *t*-test for continuous variables or with chi-square test for categorical data. Differences were considered significant when the $p < 0.05$ (two-tailed).

RESULTS

Classification of restenosis

Of the 100 patients studied, clinical follow-up was achieved in 100 per cent of the patients and angiographic studies were performed within 6 months in 34 (34%) of the 100 patients. From the inclusion criteria, 31 patients (31%) were classified as the restenosis group and 69 patients (69%) the non-restenosis group.

The restenosis group was established as follows: 20 of 31 patients (64.5%) underwent repeated coronary angiography within 6 months and fitted the angiographic criteria for restenosis, 11 of 31 patients (35.5%) fitted the clinical criteria by recurrent angina, death, acute myocardial infarction or abnormal noninvasive test. 11 of 31 patients (35.5%) fitted both the angiographic and clinical restenosis criteria.

The non-restenosis group was established as follows: 14 of 69 patients (20.3%) underwent repeated coronary angiography within 6 months and did not fit the angiographic criteria, 55 of 69 patients (79.7%) did not have recurrent angina, death, acute myocardial infarction and abnormal noninvasive test. 14 of 69 patients (20.3%) did not fit either the angiographic or clinical restenosis criteria.

Associations with restenosis

Table 1 shows the baseline clinical characteristics of the restenosis and non-restenosis group. The two groups did not significantly differ with respect to age, gender, coronary risk factors, symptoms and clinical diagnosis ($p > 0.05$).

Table 2 and 3 compare the angiographic characteristic of the restenosis group and non-restenosis group. No angiographic data were different between both groups ($p > 0.05$) except that lesion type A was found more often in the non-restenosis group than the restenosis group.

Plasma for homocysteine level was obtained in 99 of 100 patients who had angioplasty. The minimum and maximum of plasma homocysteine were 4.40 μmol/L and 50.00 μmol/L, respectively. The mean plasma homocysteine concentration for this group was 13.61±6.04 μmol/L (Fig. 1). The mean plasma homocysteine concentration in patients with restenosis was 14.46±8.41 μmol/L compared with a mean plasma homocysteine concentration of 13.25± 4.69 μmol/L in those without restenosis. There was no find statistical significance of homocysteine level between the restenosis and non-restenosis groups.

Table 1. Baseline clinical characteristics between restenosis and non-restenosis groups.

Clinical characteristics	Restenosis n=31	%	Non-restenosis n=69	%	P value
Age (years)	60.97 ± 11.99		63.23 ± 10.59		0.35
Sex (male)	21	67.74	47	68.11	0.97
Body weight (kg)	67.58 ± 16.42		64.99 ± 8.97		0.42
Height (cm)	160.71 ± 8.97		160.35 ± 7.59		0.84
Coronary risk factors					
Aging	26	83.87	62	89.86	0.60
Diabetes	11	35.48	20	28.99	0.52
Hypertension	21	67.74	47	68.12	0.97
Dyslipidemia	21	67.74	49	71.01	0.74
Smoking	9	29.03	23	33.33	0.67
Family history	7	22.58	15	21.74	0.93
Symptoms and signs					
Angina pain	30	96.77	63	91.30	0.57
Congestive heart failure	2	6.45	4	5.80	1.0
Dyspnea on exertion	2	6.45	7	10.14	0.83
Diagnosis					
Chronic stable angina	16	51.61	43	62.32	0.31
Unstable angina	10	32.26	19	27.54	0.63
Non-Q wave MI	2	6.45	1	1.45	0.47
Old myocardial infarction	9	29.03	19	27.54	0.88

Table 2. Angiographic findings before PCI was done.

	Restenosis n=31	%	Non-restenosis n=69	%	P value
Angiographic data					
LMT	1	0.03	1	0.01	0.56
LAD	28	90.32	51	73.91	0.06
LCX	16	51.61	27	39.03	0.11
Intermediate	1	0.03	1	0.01	0.56
RCA	16	51.61	39	56.52	0.73
Vessel disease					
Single	8	25.80	30	43.48	0.09
Double	14	45.16	27	39.13	0.57
Triple	9	29.03	12	17.39	0.19

LMT = left main trunk, LAD = left anterior descending artery, LCX = left circumflex artery, Intermediate = intermediated branch, RCA = right coronary artery.

In healthy subjects, the normal reference range of homocysteine level is 5-15 μmol/L. From this reference, a cut off point of homocysteine level equal to 15 μmol/L or above was used to identify the high homocysteine level group. Of 99 patients, high homocysteine level was established in 29 patients and low homocysteine level in 70 patients. High homocysteine level was established in 9 patients with restenosis *versus* 20 patients without restenosis (Table 4). If we used the cut off point of homocysteine level equal to 12 μmol/L or above to identify the high homocysteine level group, high homocysteine level was established in 53 patients and low homocysteine level in 46 patients. High homocysteine level was established in 14 patients with restenosis *versus* 39 patients without restenosis (Table 5). From both cut off points of the homocysteine level, there was no correlation between plasma homocysteine level and the restenosis group. ($p>0.05$).

DISCUSSION

Homocysteine has been implicated as a risk factor for the development of coronary artery disease and as a risk factor for mortality in patients who had

Table 3. Lesion type and intervention techniques.

	Restenosis n=58	%	Non-restenosis n=110	%	P value
Lesion type* (lesions)					
Type A	7	12.07	31	28.18	0.02
Type B	41	70.69	70	63.64	0.36
Type C	10	17.24	9	8.18	0.08
Procedural (lesions)					
Balloon angioplasty	29	50	41	37.27	0.11
Balloon with coronary stents	28	48.28	65	59.09	0.18
Rotablation	1	1.72	4	3.64	0.49

LMT = left main trunk, LAD = left anterior descending artery, LCX = left circumflex artery, Intermediate = intermediated branch, RCA = right coronary artery.
* American Heart Association/American College of Cardiology task force classification

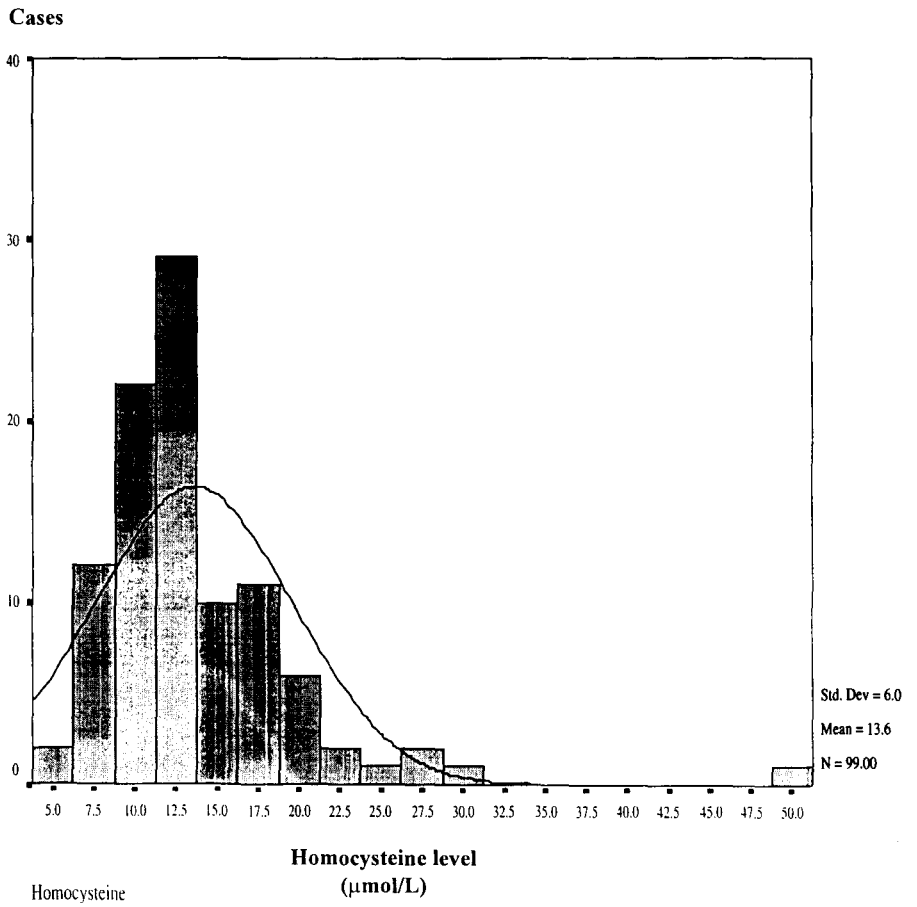

Fig. 1. The distribution of values for plasma homocysteine level.

coronary artery disease. In contrast to cross-sectional and case-control studies, the results of prospective studies indicated less or no predictive ability for plasma homocysteine in cardiovascular disease(33). Instead, elevated homocysteine level may be an acute-phase reactant that is predominantly a marker of atherogenesis, or a consequence of other factors more closely linked to risks of cardiovascular disease

Table 4. Comparison of plasma homocysteine level between the restenosis group (n=30) and non-restenosis group (n=69).

Homocysteine level	Restenosis	Non-restenosis	Total	P value
Low (< 15 μmol/L)	21	49	70	
High (≥ 15 μmol/L)	9	20	29	
Total	30	69	99	0.92

Table 5. Comparison of plasma homocysteine level between the restenosis group (n=30) and non-restenosis group (n=69).

Homocysteine level	Restenosis	Non-restenosis	Total	P value
Low (< 12 μmol/L)	16	30	46	
High (≥ 12 μmol/L)	14	39	53	
Total	30	69	99	0.37

(33). The mechanism by which homocysteine might promote atherogenesis is controversial, but postulated mechanisms include increased oxidative stress, excessive thrombogenesis, the development of endothelial dysfunction(20), and its ability to act as a mitogen of smooth muscle cells(21). If homocysteine induces atherosclerosis through any of these pathways, it would be reasonable to suggest that it might also affect the restenosis process. The Morita H, et al (22) study suggests that plasma homocysteine is a potential risk factor of restenosis after PCI, and therapeutic strategy targeted against hyperhomocysteinemia may be beneficial for preventing restenosis. But a prospective study from Stevens ES. Miner, et al(23) did not support that finding. From our study, we did not find the correlation of high level of homocysteine and restenosis rate. We suggest that plasma homocysteine levels were not significantly associated with restenosis after PCI independently of the other clinical parameters. From this present study, there were some limitations because the authors used both clinical restenosis and angiographic restenosis criteria to divide the patients into restenosis and non-restenosis groups. However, many studies(26-30) used these criteria because Weintraub et al(31) reported a correlation between the clinical and angio-graphic criteria but clinical restenosis was not exactly the same as correlates of angiographic restenosis. This point may be a problem for interpretation to define the restenosis and non-restenosis groups. A certain limitation of the present study may be the fact that not all angioplasty patients underwent routine follow-up cardiac catheterization. Clinical assessment of cardiac events is known to have some inaccuracy in predicting restenosis(25). Prior studies have indi-cated that 15 to 20 per cent of asymptomatic patients have angiographic evidence of restenosis and that about 30 per cent of patients with symptoms have no angiopraphic evidence of restenosis at the time of follow-up(25).

Further large-scale prospective studies should be designed to find the relation of homocysteine levels and restenosis if there is evidence of basic science to support this idea.

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โฮโมซีสตีอีน กับภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์

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จากการศึกษาทั้งในประเทศแถบตะวันตกและเอเชียพบว่าระดับโฮโมซีสตีอีนที่สูงกว่าปกติเป็นปัจจัยเสี่ยงที่ทำให้เกิดโรคหลอดเลือดแดงโคโรนารีย์, กล้ามเนื้อหัวใจตาย, โรคหลอดเลือดสมอง และเส้นเลือดดำอุดตัน โฮโมซีสตีอีน สามารถทำให้เกิดการอุดตันของหลอดเลือดโดยการทำลายเซลล์เอนโดทีเลียว เพิ่มการแบ่งตัวของเซลล์กล้ามเนื้อเรียบ และกระตุ้นสารที่ทำให้เกิดการอุดตันของหลอดเลือดมากขึ้น

ในปัจจุบันมีความรู้เกี่ยวกับโฮโมซีสตีอีน กับภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์มีน้อยมาก จึงได้มีการศึกษาในครั้งนี้เพื่อหาความสัมพันธ์ของโฮโมซีสตีอีน กับภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์ว่ามีความสัมพันธ์กันหรือไม่

การศึกษานี้ทำในผู้ป่วย 100 ราย ที่ได้รับการตกแต่งหลอดเลือดโคโรนารีย์ โดยสามารถเก็บตัวอย่างเลือดผู้ป่วยมาวิเคราะห์หาระดับโฮโมซีสตีอีนได้ 99 ราย พบว่า ระดับโฮโมซีสตีอีน โดยเฉลี่ยอยู่ที่ 13.61±6.04 μmol/L โดยค่าปกติของโฮโมซีสตีอีน อยู่ระหว่าง 5-15 μmol/L แต่ปัจจุบันพบว่าระดับโฮโมซีสตีอีน ที่มากกว่าหรือเท่ากับ 12 μmol/L ก็สามารถทำให้เกิดโรคหลอดเลือดแดงโคโรนารีย์และโรคหลอดเลือดสมองได้ ดังนั้น ในการศึกษานี้จึงมีการทดสอบหาความสัมพันธ์ของระดับโฮโมซีสตีอีน ที่มากกว่าหรือเท่ากับ 15 หรือมากกว่าหรือเท่ากับ 12 μmol/L ว่ามีความสัมพันธ์กันหรือไม่กับภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์

จากการศึกษาพบว่ากลุ่มที่มีระดับโฮโมซีสตีอีนที่สูงกว่าหรือเท่ากับ 15 μmol/L มีการเกิดภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์ 9 รายใน 29 รายหรือ ถ้าใช้จุดตัดที่มากกว่าหรือเท่ากับ 12 μmol/L พบว่ามีการเกิดภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์ 14 รายใน 53 ราย โดยไม่พบความสัมพันธ์ระหว่างระดับโฮโมซีสตีอีนที่สูง กับภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์แต่อย่างใด ($p>0.05$)

คำสำคัญ : โฮโมซีสตีอีน, การตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์, หัตถการตกแต่งหลอดเลือดโคโรนารีย์

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