Serum Lipoprotein (a) Level and Restenosis After Percutaneous Coronary Intervention

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Abstract

Restenosis is regarded as the result of a combination of various pathological events. The mechanisms are complex and not completely understood. In this study, the authors focused on the lipoprotein (a) (Lp (a)). It is one of the novel risk factors in atherosclerotic vascular disease. Numerous clinical studies suggest that individuals with elevated blood levels of Lp (a) have been shown to be associated with atherosclerotic vascular disease. However, whether a high serum concentration of Lp (a) affects restenosis after PCI remains controversial. In this study, the relationship between serum Lp (a) levels and restenosis after PCI was examined to investigate whether serum Lp (a) levels may be a predictor of restenosis after PCI. Of the 100 patients studied, 31 patients (31%) were classified as the restenosis group and 69 patients (69%) the non-restenosis group. Both groups did not significantly differ in serum concentration of total cholesterol, triglyceride, HDL-C, and LDL-C. The mean serum Lp (a) concentration in patients with restenosis was 41.50±34.99 mg/dL compared with a mean serum Lp (a) concentration of 29.87±25.47 mg/dL in those without restenosis. There was no statistical significance of Lp (a) level between the restenosis and non-restenosis groups (p=0.06). In healthy subjects, the normal reference range of serum Lp (a) concentration is below 30 mg/dL. From this reference, if a cut off point of serum Lp (a) concentration equal to 30 mg/dL or above to identify high Lp (a) level group was used. High serum Lp (a) level was established in 15 patients with restenosis *versus* 21 patients without restenosis. From this cut off point of serum Lp (a) level, the authors did not find a correlation between serum Lp (a) level and the restenosis group. (p=0.08).

Key word : Serum Lipoprotein (a) Level, Restenosis, Percutaneous Coronary Intervention

**LOLEKHA P, MAHANONDA N,
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J Med Assoc Thai 2001; 84 (Suppl 3): S628-S635**

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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 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 remain 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, identification of the novel risk factors would enable us to setup a more effective therapeutic strategy to ameliorate the outcome of PCI.

In this study, the authors focused on the lipoprotein (a) (Lp (a)). It is one of the novel risk factors in atherosclerotic vascular disease. Lp (a) is a LDL-like particle with a characteristic polymorphic glycoprotein known as apolipoprotein (a) (apo (a)) that is disulfide linked to the apolipoprotein B-100 moiety of LDL[7]. Apo (a) has 80-90 per cent homology with plasminogen[8,9]. It has been suggested that Lp (a) competitively inhibits the binding of plasminogen to the surfaces of endothelial cells, thereby modulating fibrinolysis system[10,11]. From this pathogenesis, numerous clinical studies suggest that individuals with elevated blood levels of Lp (a) have been shown to be associated with atherosclerotic vascular disease, including an increased prevalence and severity of coronary artery disease, carotid artery disease and peripheral vascular disease[12,13]. However, whether a high serum concentration of Lp (a) affects restenosis after PCI remains controversial [14-21]. In this study, the authors examined the relationship between serum Lp (a) levels and restenosis after PCI to investigate whether serum Lp (a) 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 the research laboratory was available to measure some factors in his/her specimens. Exclusion criteria were patients who would not permit blood samples to be drawn and for emergency or unplanned PCI. From that period, 100 consecutive patients who underwent successful PCI (residual stenosis immediately after PCI of < 50 per cent were enrolled 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

The authors used angiographic criteria and/or clinical criteria to define the restenosis within 6 months after PCI[22,23]. 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 the pain before being treated with PCI, death, acute myocardial infarction or abnormal noninvasive exercise or nuclear stress test[24-29]. 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 into one of two restenosis criteria were enrolled in the restenosis group and those who did not fit into 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 the measurement of serum Lp (a) levels were performed within 1 year of blood drawing. Routine lipid profiles (total cholesterol, triglyceride, HDL-C and LDL-C) were analyzed on the day of venipuncture.

In all the enrolled patients, lipid profiles and serum Lp (a) level were measured before undergoing PCI or coronary angiographic examination. Total cholesterol and triglyceride were measured with enzymatic assays and HDL- cholesterol (HDL-C) was measured by homogeneous immunoassay (Roche Diagnostics, Switzerland) on a Hitachi autoanalyzer. LDL-cholesterol was calculated with the Friedewald formula. The test for serum Lp (a) levels was done by the immunoturbidimetric method from

Roche Diagnostics Switzerland. The coefficient of variation in all assays was lower than 5 per cent.

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 serum Lp (a) level is lower than 30 mg/dL. From this normal reference, a cut off point of Lp (a) level equal to 30 mg/dL or above was used to identify the high Lp (a) level group. The high Lp (a) level group and the normal Lp (a) 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 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 both the angiographic and clinical restenosis criteria.

Clinical and angiographic characteristics of restenosis and non-restenosis groups

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, symptom and clinical diagnosis ($p>0.05$).

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.

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

Table 4. Lipid profiles and serum concentration of Lp (a) between restenosis and non-restenosis groups.

	Restenosis n=31	Non-restenosis n=69	P value
Lipid profiles			
Total cholesterol	196.23 ± 43.67	194.46 ± 37.59	0.84
Triglyceride	138.65 ± 79.14	133.06 ± 75.02	0.74
HDL-C	41.71 ± 15.59	41.60 ± 10.84	0.97
LDL-C	124.81 ± 42.60	124.44 ± 33.85	0.96
Lipoprotein (a)	41.50 ± 34.99	29.87 ± 25.47	0.06

Table 2 and 3 compare the angiographic characteristics of the restenosis group and non-restenosis groups. No angiographic data were different between both groups ($p>0.05$) except lesion type A which was more common in the non-restenosis group than the restenosis group ($p=0.02$).

Lipid profiles and Lp (a) concentration

Table 4 shows the serum lipid concentration in the restenosis and non-restenosis groups. Both groups did not significantly differ in the serum concentration of total cholesterol, trigleceride, HDL-C, and LDL-C. The mean serum Lp (a) concentration

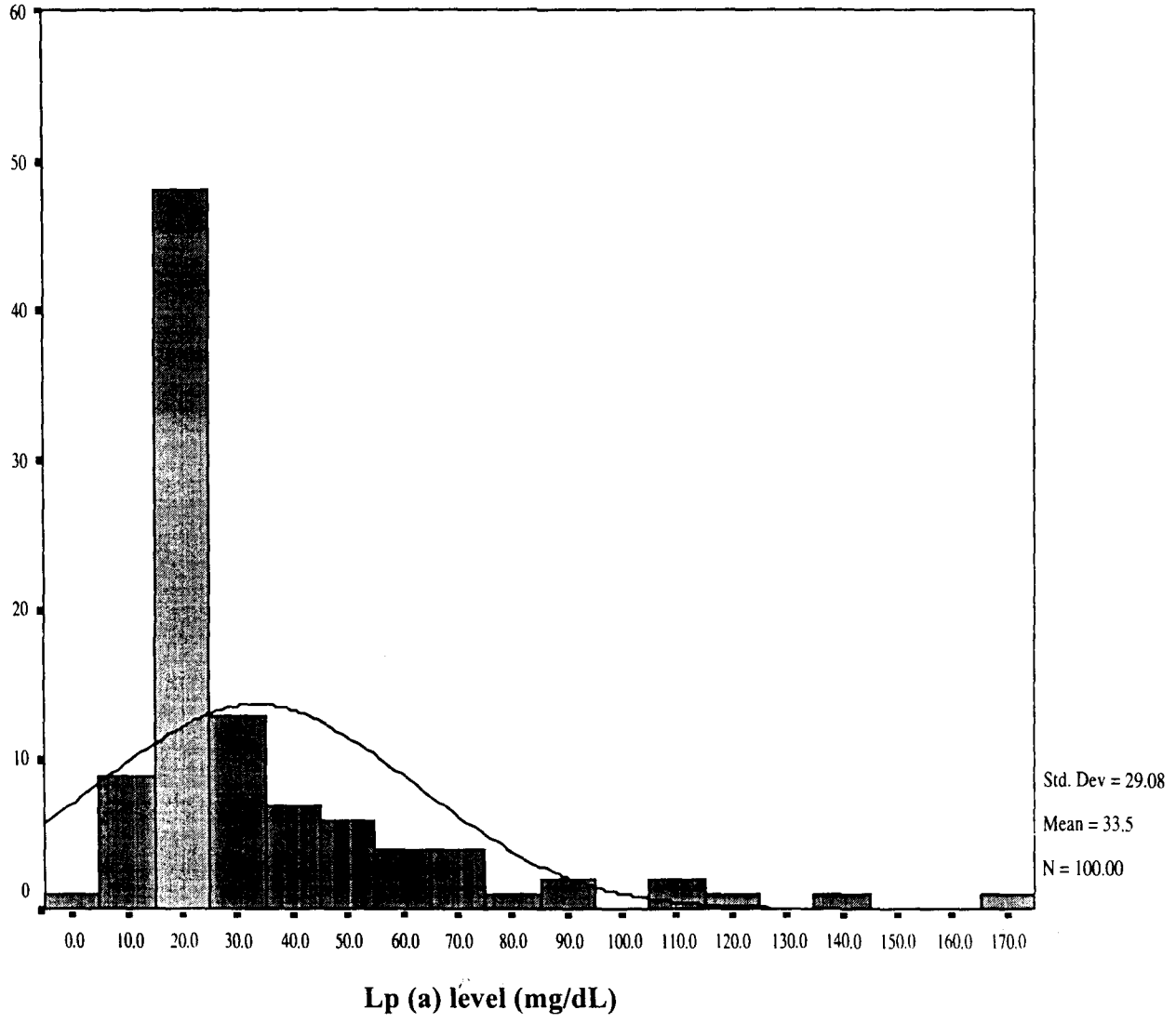

Fig. 1. The distribution of values for serum Lp (a) level.

in the 100 patients was 33.5±29.06 mg/dL (Fig. 1). The minimum and maximum of serum Lp (a) were 0 mg/dL and 165.6 mg/dL, respectively. The mean serum Lp (a) concentration in patients with restenosis was 41.50±34.99 mg/dL compared with a mean serum Lp (a) concentration of 29.87±25.47 mg/dL in those without restenosis. There was no statistical significance of Lp (a) level between the restenosis and non-restenosis groups (p=0.06).

In healthy subjects, the normal reference range of serum Lp (a) concentration is below 30 mg/dL. From this reference, if the authors used a cut off point of serum Lp (a) concentration equal to 30 mg/dL or above to identify high Lp (a) level group. Of 100 patients, a high serum Lp (a) level was established in 36 patients and low serum Lp (a) level in 64 patients. A high serum Lp (a) level was established in 15 patients with restenosis *versus* 21

Table 5. Comparison of serum Lp (a) level between the restenosis group (n=31) and non-restenosis group (n=69).

Lp (a) level	Restenosis	Non-restenosis	Total	P value
Low (< 30 mg/dL)	16	48	64	
High (≥ 30mg/dL)	15	21	36	
Total	31	69	100	0.08

patients without restenosis (Table 5). From this cut off point of serum Lp (a) level, there was no correlation between serum Lp (a) level and the restenosis group. (p=0.08).

DISCUSSION

Lipoprotein (a) has been implicated as an independent risk factor for coronary artery disease in many prospective studies, although this finding has not been universal[12]. The postulated mechanisms of this association included impaired fibrinolysis, increased smooth muscle cell migration and smooth muscle cell proliferation[10,11]. Because these mechanisms may be important in the restenosis process, several studies have suggested that Lp (a) is a risk factor for restenosis after PCI but some studies failed to find a correlation between Lp (a) concentrations and restenosis[14-21]. From this study, the authors assessed whether Lp (a) was a risk factor for restenosis after PCI in Thai patients with coronary artery disease. The serum Lp (a) concentration in the restenosis group was not significantly higher than the non-restenosis group (p=0.06). When a cut off point equal to 30 mg/dL or above was used to identify the high Lp (a) level group, the authors also did not find the correlation of the restenosis rate and the patients who had high serum Lp (a) level (p= 0.08). The incidence of other risk factors did not significantly differ between the two groups except the types of lesion before PCI. Because lesion type A is easy to do and has a higher success rate of PCI than other lesion types, so lesion type A has a lower rate of restenosis than other lesion types. This study, had some limitations because both clinical restenosis and angiographic restenosis criteria were used to divide the patients into the restenosis and non-restenosis groups. However, many studies[24-28] used these criteria because Weintraub, et al[29] reported the correlation between the clinical and angiographic criteria but clinical restenosis was not exactly the same as correlates of angiographic restenosis. This point may be a problem for interpretation of defining 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. Prior studies have indicated that 15 to 20 per cent of asymptomatic patients have angiographic evidence of restenosis and about 30 per cent of patients with symptoms have no angiopraphic evidence of restenosis at the time of follow-up[23].

Conclusion, high serum concentration of Lp (a) may be a risk factor of restenosis (p=0.06) but it was not statistically significant. Further large-scale prospective studies should be designed to find the relation of serum Lp (a) levels and restenosis if there is evidence of basic science support for this idea.

(Received for publication on September 21, 2001)

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ไลโปโปรตีน ชนิด เอ กับภาวะหลอดเลือดตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์

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ปัจจุบันสาเหตุของภาวะหลอดเลือดตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์ ยังไม่ทราบแน่ชัด พบว่ามีหลายสาเหตุและหลายพยาธิสภาพที่สามารถทำให้เกิดภาวะดังกล่าวนี้ได้

ในการศึกษานี้เน้นไปที่ การหาความสัมพันธ์ของไลโปโปรตีนชนิด เอ ซึ่งเป็นปัจจัยเสี่ยงใหม่ที่อาจก่อให้เกิดโรคหลอดเลือดแข็งตัว กับภาวะหลอดเลือดตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์ ว่ามีความสัมพันธ์กันหรือไม่

การศึกษานี้ทำในผู้ป่วย 100 ราย ที่ได้รับการตกแต่งหลอดเลือดโคโรนารีย์ พบว่ามีผู้ป่วย 31 ราย ที่มีภาวะหลอดเลือดตันซ้ำ หลังการตกแต่งหลอดเลือดโคโรนารีย์ และอีก 69 ราย ที่ไม่เกิดภาวะนี้ ระดับของไขมันทั้ง คลอเลสเตอรอล, ไตรกรีเซอไร, เอ็ชดีแอล, แอลดีแอล ไม่แตกต่างกันในสองกลุ่มดังกล่าว ระดับของไลโปโปรตีนชนิด เอ ในกลุ่มที่มีภาวะตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์ เท่ากับ 41.50 ± 34.99 mg/dl กลุ่มที่ไม่ตีบตัน เท่ากับ 29.87 ± 25.47 mg/dl พบว่าทั้งสองกลุ่มมีแนวโน้มที่ระดับของไลโปโปรตีน ชนิด เอ จะแตกต่างกัน แต่เมื่อทดสอบทางสถิติไม่พบความแตกต่างกันในสองกลุ่มดังกล่าว ($p = 0.06$) ระดับของไลโปโปรตีน ชนิด เอ ในคนปกติจะมีค่าน้อยกว่า 30 mg/dl เมื่อใช้ค่านี้เป็นค่ามาตรฐาน ในการแบ่งผู้ป่วยเป็นกลุ่ม ที่มีระดับไลโปโปรตีน ชนิด เอ สูง ซึ่งมีค่ามากกว่าหรือเท่ากับ 30 mg/dl พบว่าในกลุ่มที่มีภาวะหลอดเลือดตีบตันซ้ำ หลังการตกแต่งหลอดเลือดโคโรนารีย์ มีระดับไลโปโปรตีน ชนิดเอ สูง 15 ราย และในกลุ่มที่ไม่มีภาวะตีบตัน มี 21 ราย โดยไม่พบความสัมพันธ์ของระดับไลโปโปรตีน ชนิด เอ ที่สูง กับภาวะหลอดเลือดตีบตัน ($p=0.08$)

คำสำคัญ : ไลโปโปรตีน ชนิด เอ, การตีบตันซ้ำหลังการตกแต่งหลอดเลือดโคโรนารีย์, หัตถการตกแต่งหลอดเลือดโคโรนารีย์

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จดหมายเหตุทางแพทย์ ฯ 2544; 84 (ฉบับพิเศษ 3): S628–S635

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