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Calcified Plaque Component as a Negative Predictive Marker for Coronary Plaque Progression

심혈관 죽상경화반 진행에 대한 음성 예측 인자로서의 석회화 부분

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Abstract

Calcified Plaque Component as a Negative Predictive Marker for Coronary Plaque Progression

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Background and purpose:

Coronary calcification is an important predictor of coronary artery disease and cardiovascular event, although calcified coronary plaque itself cause less plaque rupture or thrombosis, compared with non-calcified or

mixed plaque. Development and regression of atherosclerotic plaque and formation of coronary calcification can be observed with Multi-detector computed tomography.

Methods:

Seventy patients who had undergone coronary CT scan at least 2 times were retrospectively reviewed. Two scan were performed with interval of at least 10 months. All of these patients had more than one intermediate coronary lesion on initial scan. Volume and characteristics of coronary plaque were quantitatively assessed using semi-automated software. The plaques were classified to one of three categories; non-calcified, calcified, and mixed plaques. Calcified plaque was excluded from the study.

Results:

Seventy patients were included in the analysis and no patients experienced acute coronary syndrome or sudden

cardiac death during follow up period. Mean duration between two scan was 10.3 ± 60.6 months. Total plaque volume was not significantly changed during follow-up period; initial volume of plaque was 366.1 ± 145.8 mm^3 and follow up volume of plaque was 366.2 ± 161.2 mm^3 with annualized absolute volume change of -3.81mm^3 and annualized percent change of -0.5%. Initial low calcified plaque volume was associated with acceleration of plaque progression. Statin didn't affect plaque progression or regression in this study.

Conclusion:

We retrospectively observed progression of coronary atherosclerotic plaque with multi-detector coronary CT. Initial smaller volume of calcified plaque component was associated with plaque progression.

Key words:

Coronary atherosclerotic plaque, computed tomography,

CT

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Introduction

Coronary atherosclerosis is a major cause of myocardial infarction and heart failure, and early diagnosis and treatment of atherosclerosis is mandatory for the treatment. Insignificant coronary lesion can be observed without medical treatment or intervention, while treatment with intervention or surgery is recommended on critical lesion (stenosis > 70% of lumen diameter).

Atherosclerotic plaque develops via several steps. Initially, lipoprotein particles accumulate beneath the vascular endothelium. Then leukocytes were recruited to the early lesion, where the inflammatory process begins. After recruitment, mononuclear phagocytes uptake LDL-cholesterol by receptor-mediated endocytosis and mature into lipid-laden foam cells, which composes 'fatty streak' of the lesion.

If inflammation proceeds further, endothelial integrity is damaged, microbleeding and thrombosis occurs on the atherosclerotic plaque. Proliferation of smooth muscle cell is activated by microbleeding which forms 'fibrofatty' lesion.

Finally, calcium accumulation occurred in the plaque via bone-formation pathways such as osteocalcin or osteopontin.

After the process, atherosclerotic plaque is stabilized and 'calcified' lesion is left behind.[1, 2]

Coronary calcification, the end product of atherosclerotic plaque, appealed its clinical importance long time ago. Quantification of coronary arterial calcification (CAC) via non-contrast cardiac CT has been in practice for over 15 years. There were several studies which provide highly significant relationship between CAC and coronary artery disease (CAD).[3, 4] Furthermore, CAC scoring provided additional predictive value over conventional cardiac risk factors for CAD.[5]

On the meanwhile, position and burden of calcified plaque and degree of stenosis were not closely related. Autopsy study by Mautner[6] et al. showed that the quantity of calcium and degree of stenosis had variable relationship. Most studies regarding calcium scores and stenosis of vessel reports high sensitivity but low specificity of CAC score.

Furthermore, highly calcified plaque presents with stable angina more likely, rather than cardiovascular event or mortality.[7] Recent study with coronary angiography showed that most myocardial infarctions were caused by small, mixed

plaque, rather than huge, calcified plaque. Heavily calcified plaque tended to less thrombus formation than non-calcified or mixed plaque.[8]

With recent development of Multi-detector computed tomography (MDCT), not only CAC score but volume and composition of coronary plaque were measurable. Several studies showed that low attenuation plaque volume is associated with plaque instability.[9-11] However, serial monitoring of plaque characteristics by MDCT was not evaluated extensively. Therefore, our longitudinal retrospective study was intended to evaluate the influence of intrinsic composition of coronary atherosclerotic plaques on plaque progression, which was measured by MDCT.

Materials and Methods

Patients and methods

A retrospective observational study was performed on seventy patients who had undergone more than two coronary angiography scans with MDCT. All tests were performed with clinical indications. Last MDCT scans were at least 10 months

apart from first scan (10.3 months to 60.6 months, median 27.2 months). All of these patients had more than one intermediate coronary lesion (coronary stenosis between 30% and 70% of diameter) on initial scan. Patients with insignificant coronary lesion or stenosis over 70% of diameter were excluded from the study. Patients who had undergone percutaneous coronary intervention at target lesion or coronary bypass graft surgery at target vessel were also excluded. Characteristics and volume of plaque on initial and last coronary CT scan were assessed and progression and development of plaque were analyzed. The analysis was approved by the institutional review board; written informed consent for the analysis was waived by the board. Sex, age, underlying disease, medication, laboratory finding and echocardiographic parameters were retrospectively reviewed with medical records.

Plaque Analysis of coronary CT image

The plaques were classified to one of three categories by the radiologist; non-calcified, calcified, and mixed plaques. Plaques in which 50% of the plaque area was occupied by calcified tissue were classified as calcified, and lesions with 50%

calcium as mixed and lesions without any calcium were classified as non-calcified lesions. Calcified plaque was excluded from the study.

When there are multiple plaques on a patient, only the largest plaque was included in the study.

MDCT images were analyzed with semi-automatic software which was developed to evaluate coronary plaque volume and characteristics. Target lesion boundaries matching for the two studies were carried out manually with identifiable side branches. Plaque characteristics were divided into fibrous, fatty and calcified components. Plaque voxels from -100 to 49 Hounsfield units were classified as fatty components, plaque voxels from 50 to 149 Hounsfield units were classified as fibrous components, and plaque voxels over 150 Hounsfield units were classified as calcified components. Fibrofatty components were sum of fibrous and fatty components. Unevaluable images due to poor image quality were excluded from the analysis. (7 patients)

Statistics

Statistical analysis was performed using SPSS V.18 for

Windows (SPSS Inc., Chicago, Illinois) statistical software. Annualized absolute change of plaque volume was calculated as: (plaque volume of last scan) − (plaque volume of initial scan) / (time interval between two scans (year)). And annualized percent change of plaque volume was calculated as: (plaque volume of last scan) − (plaque volume of initial scan) / (plaque volume of initial scan) * (time interval between two scans (year)). Patients were divided into two groups according to plaque volume change, and differences of baseline factors between two groups were compared.

Continuous variables were analyzed with Student's T-test or paired T-test, while non-continuous variables were analyzed with Pearson's chi-square test or Fisher's exact test.

Results

Patient characteristics

Seventy patients were included in the analysis. Mean age of patients were 60.6 ± 9.7 years and 63 patients (90%) were men. Majority (60%) of patients had hypertension and about half of them had dyslipidemia. Indications of initial CT

scan were varied, but most common reason was patients' intent (81.4%). (Table 1) Patients were followed up for 30.2 ± 12.5 months. No patients experienced acute coronary syndrome or sudden cardiac death during follow up period. Initial total cholesterol was 192.1 ± 26.7 mg/dL and HDL and LDL cholesterol were 51.6 ± 11.6 mg/dL and 111.2 ± 28.4 mg/dL. Total cholesterol, triglyceride and LDL cholesterol were significantly reduced during follow up period while HDL cholesterol showed a little trend toward increase. Serum creatinine also slightly reduced during follow up period (Table 2).

Table 1 Baseline characteristics

Characteristics	Number (%)	Mean (SD)	Missing value
Age (year)		60.6 (9.7)	0
Male	63 (90)		0
BMI (kg/m^2)		25.2 (2.9)	1
Underlying disease			
Hypertension	32 (60)		0
Diabetes mellitus	22 (31.4)		0
Dyslipidemia	34 (48.6)		0
Indication of initial CT scan			
Patients' intent	57 (81.4)		0
Typical chest pain	2 (2.9)		0
Atypical chest pain	7 (10.0)		0
Dyspnea	2 (2.9)		0
Follow up after thrombolysis	1 (1.4)		0
Follow up after CABG	1 (1.4)		0
Family history of CAD	6 (10.9)		15
Current smoker	20 (35.1)		13
medication			
ACEi or ARB	11 (15.7)		13
Beta blocker	6 (10.5)		13
Statin	15 (23.4)		6

* SD : standard deviation, CABG : coronary artery bypass graft, CAD : coronary artery disease, ACEi : angiotensin-converting enzyme inhibitors, ARB : angiotensin receptor blockers

Table 2 Laboratory findings at initial and follow up scan

Characteristics	Initial	Follow up	*p* value	Missing value
Hb A1c (%)	6.26 ± 1.01	6.37 ± 0.82	0.196	11
Total cholesterol (mg/dL)	192.1 ± 26.69	167.24 ± 38.57	< 0.001	8
Triglyceride (mg/dL)	149.72 ± 100.12	112.97 ± 50.62	0.002	10
HDL cholesterol (mg/dL)	51.58 ± 11.64	53.00 ± 10.65	0.181	10
LDL cholesterol (mg/dL)	111.20 ± 28.40	95.14 ± 38.25	0.001	10
Serum Creatinine (mg/dL)	1.11 ± 0.16	1.04 ± 0.22	0.002	8
high-sensitive CRP (mg/dL)	0.14 ± 0.15	0.32 ± 0.83	0.127	24

Changes of plaque volume and composition

Table 3 represents plaque volume and characteristics on initial and second CT scan. Mean duration between two scan was 30.2 ± 12.5 months. Total plaque volume was not significantly changed during follow-up period; initial volume of plaque was 366.1 ± 145.8 mm^3 and follow up volume of plaque was 366.2 ± 161.2 mm^3 with annualized absolute volume change of −3.81mm^3 and annualized percent change of –0.5%. Initial fibrofatty component was 81.8% of total plaque volume, while calcified component occupy 18.2% of total volume. Fibrous component and fatty component of plaque were slightly reduced during follow-up period with annual volume change of 10.9 mm^3 and 4.4 mm^3 or annualized percent change of −5.7% and −3.3% each. Calcified component showed substantial progression between two scans. Annualized volume change was 12.0 mm^3 with annual percent change of 93.6%. (Figure 1, 2)

Table 3 Plaque characteristics at initial and follow up scan

Characteristics	Initial scan			Follow up scan			Annualized change (mm^3)		Annualized change (%)		*p* value*	*p* value†	*p* value‡
	Mean	SD	%	Mean	SD	%	Mean	SD	mean	SD			
Total volume	366.1	145.8	100	366.2	161.2	100	-3.81	41.7	-0.5	10.1	0.654		-
Fibrous component vol.	178.6	76.0	49.3	154.2	65.5	42.1	-10.9	16.0	-5.7	7.9	< 0.001	< 0.001	0.127
Fatty component vol.	115.6	44.2	32.5	105.4	41.1	28.8	-4.4	10.8	-3.3	9.8	< 0.001	0.033	0.904
Fibrofatty component vol.	294.2	117.1	81.8	259.7	103.9	70.9	-15.3	22.9	-5.0	7.4	< 0.001	< 0.001	0.011
Calcified component vol.	72.4	64.4	18.2	106.5	80.7	29.1	12.0	36.7	93.6	220.9	< 0.001	0.001	< 0.001

* *p* value : compared between initial and follow up scan

† *p* value : compared with annualized percent change of total plaque volume

‡ *p* value : compared with annualized change of total plaque volume

Figure 1 Absolute plaque volume changes during follow-up period

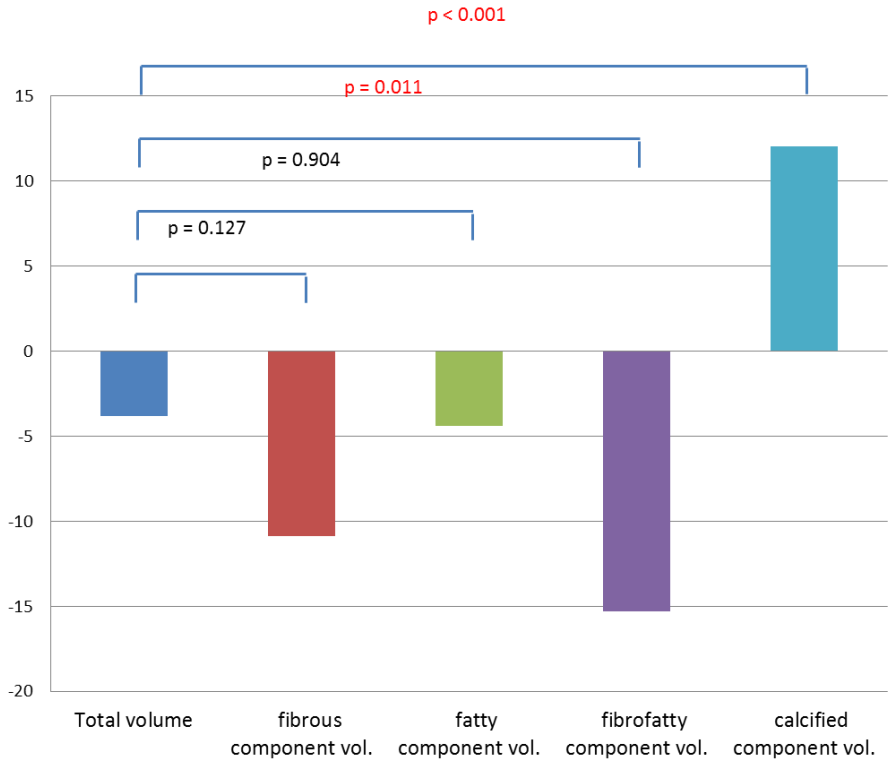
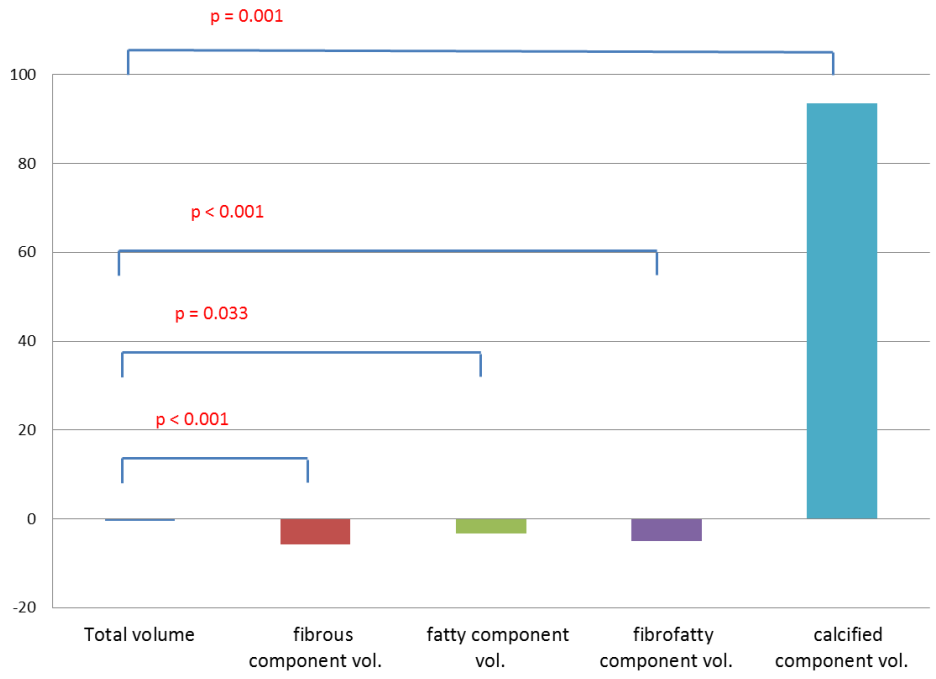


Figure 1 Relative plaque volume changes during follow-up period



Comparison between progressors and non-progressors

We divided patients into two groups, 'progressor' and 'non-progressor'. Progressor group was defined as any increase in total plaque volume per year and contained 33 patients. When baseline clinical and laboratory characteristics were compared, no parameter was associated with acceleration of plaque progression. But progressor group had initial small volume of total plaque, high percentage of fibrofatty component, and low percentage of calcified component. (Table 4) After follow up period, total volume of plaque was decreased in non-progressor group, but increased in progressor group, (-28.7 ± 34.7 vs. 24.1 ± 29.4 mm^3, $p < 0.001$) and this effect was mainly due to change of calcified component volume. (-5.6 ± 30.5 vs. 31.8 ± 33.3 mm^3, $p < 0.001$) (Table 5)

To assess influence of large volume of calcium component to the progression of plaque more accurately, patients were divided into two groups, which are 'high initial calcium group' and 'low initial calcium group'. Cut-off value dividing patients was 50 mm^3 of initial calcified plaque volume which was median value of calcified plaque volume.

High initial calcium group showed regression of total plaque volume, which of the other group was increased during follow up period. (7.3 ± 28.1 vs. −13.2 ± 48.8 mm^3, p = 0.040) Calcified plaque volume of higher initial calcium group showed a little tendency of less progression, but it did not reached statistical significance. (19.5 ± 18.2 vs. 5.8 ± 46.4 mm^3, p = 0.101) Initial calcified plaque volume and absolute or relative annualized change of total plaque volume had inverse correlation. (Figure 3)

Table 4 Clinical characteristics of progressor group and non-progressor group

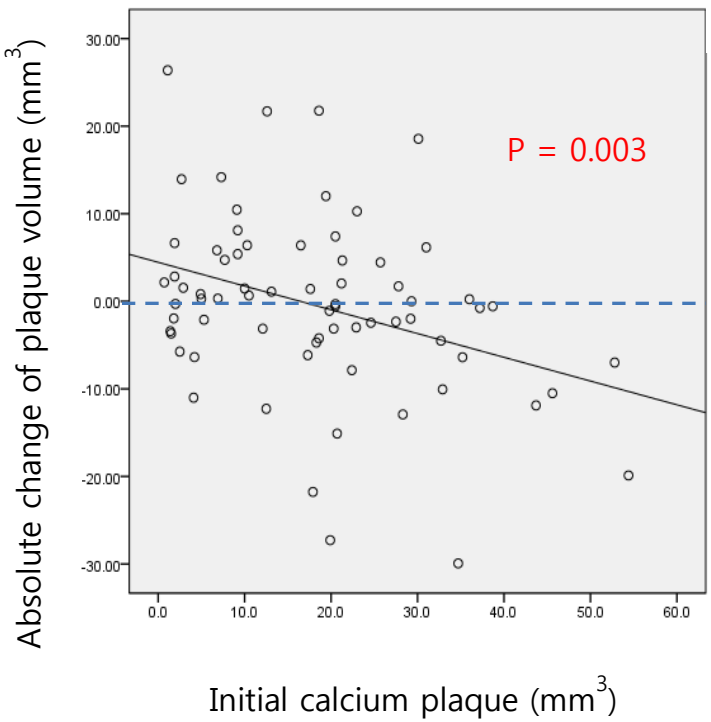
Characteristics	Non-progressor (n = 37)	Progressor (n = 33)	p value
Age (year)	62.1 (7.7)	59.0 (7.5)	0.09
Male	35 (94.5)	28 (84.8)	0.24
BMI (kg/m2)	24.8 (2.5)	25.7 (3.3)	0.17
Underlying disease			
Hypertension	21 (56.8)	21 (63.6)	0.56
Diabetes mellitus	11 (29.7)	11 (33.3)	0.80
Dyslipidemia	17 (45.9)	17 (51.5)	0.64
Indication of initial CT scan			0.50
Patients' intent	33 (89.2)	24 (72.7)	
Typical chest pain	2 (5.4)	0 (0)	
Atypical chest pain	0 (0)	7 (21.2)	
Dyspnea	1 (2.7)	1 (3.0)	
Follow up after thrombolysis	0 (0)	1 (3.0)	
Follow up after CABG	1 (2.7)	0 (0)	
Family history of CAD	2 (6.7)	4 (16.0)	0.27
Current smoker	8 (25.8)	12 (46.2)	0.11
Statin medication	22 (62.9)	17 (58.6)	0.80
Total cholesterol (mg/dL)	196.1 (33.3)	187.5 (26.3)	0.26
HDL cholesterol (mg/dL)	51.7 (11.6)	49.4 (12.1)	0.44
LDL cholesterol (mg/dL)	112.9 (37.0)	111.8 (27.2)	0.89
Total volume (mm^3)	402.4 (138.5)	325.3 (144.9)	0.03
Fibrous component percent (%)	46.7 (9.1)	52.1 (6.2)	0.005
Fatty component percent (%)	30.7 (6.9)	34.5 (7.4)	0.030
Fibrofatty component percent (%)	77.4 (14.4)	86.6 (9.6)	0.003
Calcified component percent (%)	22.5 (14.4)	13.4 (9.7)	0.003

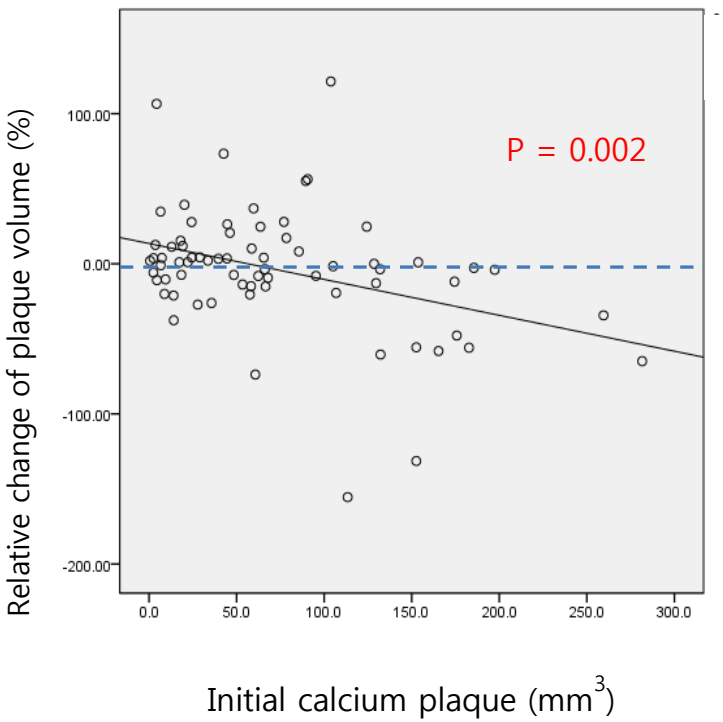
* CAD : coronary artery disease

Table 5 Plaque characteristics of progressor group and non-progressor group

Characteristics	Annualized absolute change of volume (mm^3)		p value
	Non-progressor (n=37)	Progressor (n=33)	
Total volume	-28.7 (34.7)	24.1 (29.4)	< 0.001
fibrous component volume	-15.1 (12.2)	-6.2 (18.4)	0.02
fatty component volume	-7.5 (12.9)	-0.8 (6.3)	0.007
fibrofatty component volume	-22.6 (23.2)	-7.1 (19.9)	0.004
calcified component volume	-5.6 (30.5)	31.8 (33.3)	< 0.001

Figure 3 initial calcified plaque and change of total plaque volume





Discussion

In this article, we retrospectively observed progression of coronary atherosclerotic plaque with MDCT. Among various clinical, laboratory and intrinsic plaque factors, initial smaller volume of calcified component was associated with plaque progression.

In early stage of atherosclerosis, LDL cholesterol accumulates within the vascular wall where the macrophages differentiate into foam cells and inflammation begins. As inflammatory process aggravates, smooth muscle cells are activated and produce collagens and other extracellular matrix, which form fibrosis. In case of unstable plaques, rupture and thrombus formation of plaque often occurs and finally calcium accumulates within vessel wall.[1, 12] These processes were represented by fatty, fibrous, and calcified component of plaque on CT coronary angiography. [13]

Initial small calcified component volume may represent early atherosclerotic plaque, so there would be more chance to progress. Calcified plaque was excluded from the study because we assumed that it represented end-stage of atherosclerosis. And there were inverse relationship between calcified plaque

volume and progression of plaque, although calcified plaque at initial scan was excluded from the study,

Unlike previous studies regarding statin and plaque regression,[14, 15] statin didn't affect plaque progression or regression in this study. There could be several reasons. First, patients included in this study were relatively at low risk, with low progression of plaque volume. In fact, total plaque volume was slightly reduced rather than increased in this study population. So, it could be possible that reductive ability of statin to regress the plaque wasn't expressed in the study. Second, study population or follow up period may not be sufficient to prove reductive capability of statin. This retrospective study was conducted during mean duration of 30 months, in seventy patients. And drug history of 6 patients could not be obtained. Results might be influenced by relatively small size of patient population and follow-up period. Third, dose of statin was not enough to induce plaque regression. In previous study, plaque regression was induced at a high dose of statin.[16, 17] Because drug and dose of statin prescribed to the patients were various, we couldn't estimate the relationship between dose of statin and plaque regression. But because this

study is retrospective observation study, dose of statin may not be high.

Our study has several limitations. First of all, there was no consideration about inter-observer variability. Because we used semi-automated analysis program, we didn't evaluated inter-observer variability, but manual adjustment of plaque borderline and volume can cause possible inter-observer variability in assessing plaque volume and characteristics.

And this study was conducted in single center, with observational design. There was no bias controlled design, so it cannot fully exclude biased results. Furthermore, study was conducted in retrospective methods, there was much missing data and reliability of data is not highly accurate.

Finally, evaluation of plaque progression with MDCT isn't well validated, with limited available data. So, further research in prospective design is needed.

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국문 초록

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임상 의과학과

서론 : 비록 석회화 죽상경화반 자체는 비석회화성 또는 혼합성 죽상경화반에 비하여 파열이나 혈전 형성을 덜하는 경향이 있지만, 관상 동맥 석회화는 심혈관 질환과 심혈관 사건의 중요한 예측인자이다. 죽상경화반의 발달과 퇴행, 관상동맥 석회화의 형성은 다검출기 전산화 단층 촬영을 이용하여 관찰될 수 있다.

방법 : 최소한 2번 이상의 관상동맥 전산화 단층 촬영을 시행한 70명의 환자가 연구에 포함되었다. 전산화 단층 촬영은 최소한 10개월 이상의 간격을 두고 시행되었으며, 모든 환자는 첫번째 촬영에서 중간정도의 병변을 가지고 있었다. 관상동맥

죽상경화반의 부피와 특성이 반자동화된 프로그램을 통해 정량적으로 분석되었다. 죽상경화반은 비석회화, 석회화, 혼합형의 세가지 형태로 분류되었고, 그 중 석회화반은 연구에서 제외되었다.

결과 : 연구에 포함된 70명의 환자 중 연구기간 동안 급성 관상동맥 증후군이나 급사를 겪은 경우는 없었다. 전산화 단층 촬영 간 시간은 10.3 ± 60.6 개월이었다. 전체 죽상경화반 부피는 경과 관찰 기간 동안 유의한 변화를 보이지 않았으며, 첫 검사 당시의 부피는 366.1 ± 145.8 mm^3 이었고, 나중 검사 당시의 부피는 366.2 ± 161.2 mm^3 이었으며, 연간 절대 부피 변화량은 −3.81mm^3, 연간 상대 부피 변화량은 –0.5%로 측정되었다. 초기 석회화반 크기가 작을수록 전체 죽상경화반의 진행이 빠른 것으로 나타났다. 스타틴 제제는 이번 연구에서 죽상경화반의 진행이나 억제에 별다른 영향을 주지 못했다.

결론 : 다검출기 전산화 단층 촬영을 이용하여 관상동맥 죽상경화반에 대하여 관찰한 결과, 초기의 석회화반 크기와 죽상경화반의 부피 증가 사이에는 역의 상관관계가 존재함을 알 수

있었다.

주요어 : 관상동맥 죽상경화반, 다검출기 전산화 단층 촬영

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