

## 안정형 및 불안정형 협심증 환자에서 관동맥조영술상 관동맥 병변의 형태학적 차이 분석

전정현 · 정익모 · 신길자

### Angiographic Differences Analysis of Coronary Artery Lesions in Patients with Stable and Unstable Angina Pectoris

Chung Hyun Chun, MD, Ick-Mo Chung, MD and Gil Ja Shin, MD

Department of Internal Medicine, College of Medicine, Ewha Womans University, Seoul, Korea

#### ABSTRACT

**Background and Objectives :** As previously reported, unstable angina is usually related to characteristic coronary artery lesion's morphology analyzed by coronary angiogram. This takes the form of an eccentrically placed convex stenosis with a narrow neck due to one or more overhanging edges or irregular, scalloped borders, or both. Although most studies were done for lesions with high degree stenosis (>50%), recent studies emphasized the role of vulnerability of plaque in acute coronary syndrome and even mild degree stenotic lesions may progress rapidly to evoke acute coronary syndrome. Therefore in this study, we analyzed the morphological characteristics of coronary artery lesions with mild degree stenosis as well as severe stenosis. **Materials and Methods :** We conducted a retrospective study of 96 patients with angina pectoris (42 of stable patients and 54 of unstable patients) who underwent coronary angiography. Each lesions with 25% or greater diameter stenosis were categorized into simple and complex lesion (convex intraluminal obstruction with a narrow neck or irregular borders, diffuse irregularities, ulceration, thrombus). Calcification of coronary artery, extents of lesions were analyzed and stenosis grade and location were categorized by AHA classification. **Results :** There were no significant differences between the stable angina and unstable angina in risk factors and vessel involvement, numbers of lesions, calcification and total obstruction. In morphologic analysis, complex lesions were more frequent in unstable angina than stable angina (49% vs 33%,  $p < 0.05$ ). The mean of percent diameter stenosis was not significantly different between two groups, but severe stenotic lesions with 90% or more stenosis were more frequent in unstable angina (34% vs 22%,  $p < 0.05$ ). Locations of involved vessels were similar between the angina groups. Complex lesions were distributed more frequent in RCA and simple lesions were more in LAD and LCX ( $p < 0.05$ ). **Conclusions :** The lesions with both complex morphology and severe degree stenosis are closely implicated in unstable angina. (**Korean Circulation J 2000;30(9):1099-1106**)

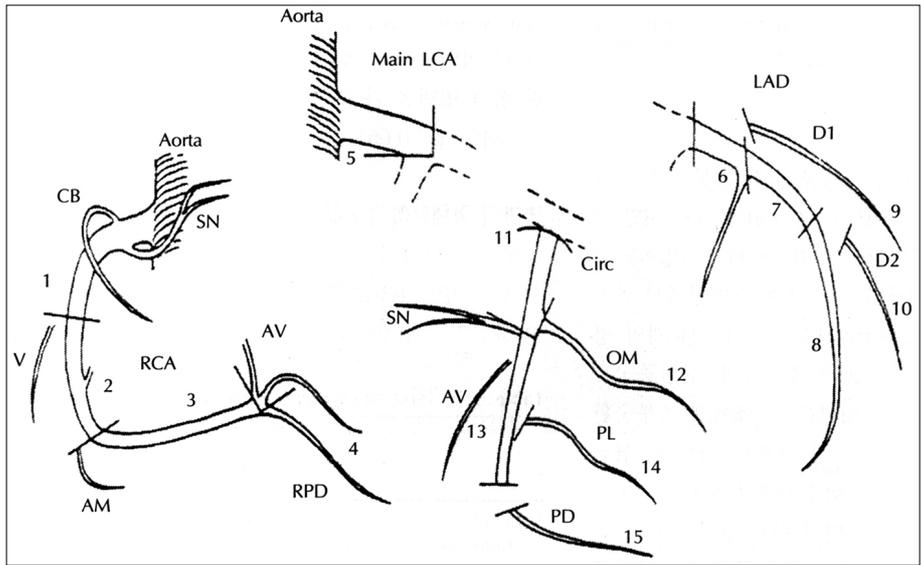
**KEY WORDS :** Unstable angina · Coronary angiography · Complex lesion · Severe stenosis.

: 2000 3 6  
: 2000 9 25  
: , 110 - 126 674 70  
: (02) 760 - 5076 · : (02) 762 - 7756 E - mail : ickmo@hitel.net

서 론

대상 및 방법

(vulnerability) , 대 상  
 (erosion), (ulceration), 1994 8 1999 6  
 1-3) 42  
 54  
 4) 1  
 가  
 가  
 (vulnerability) 가 , 가  
 가 1  
 4)5)  
 방 법  
 (complex lesion) , , , , ,  
 1)6)7) 140 mmHg 90 mmHg  
 140 mg/dL  
 22)24) 220 mg/dL  
 35 mg/dL  
 Vanguard XR (Vanguard, USA) Tagano  
 (Horsens, Denmark)  
 1)13)20)22)24) 50% 75%  
 가  
 가 25% 240  
 25% 50% , 50% 90%  
 (vulnerability) 1)8)9) , 90%  
 American Heart Assoc-  
 iation 15 9) (Fig. 1) 25%  
 가 25% 50% 가



**Fig. 1.** Segments of coronary artery bed according to the American Heart Association classification.

(concentric lesion)  
(smooth surface) 가  
surface) 가

(irregular

통계분석

SPSS 9.0

Student's t-test  
p<0.05

Chi square

(%)

결 과

위험인자 분석(Table 1)

96  
26/16 , 63 ± 10 )  
54 ( / 27/27 , 62 ± 9 )

**Table 1.** Clinical characteristics of study patients

	Stable Angina N = 42 (%)	Unstable Angina N = 54 (%)	p
Age (yr)	64 ± 10	62 ± 9	NS*
Male	26 (62)	27 (50)	NS
Hypertension	17 (40)	30 (56)	NS
Diabetes mellitus	10 (24)	10 (19)	NS
Smoking	15 (36)	22 (41)	NS
Total cholesterol (mg/dl)	189.7 ± 42.8	193.7 ± 37.3	NS
HDL-cholesterol (mg/dl)	41.5 ± 8.3	44.5 ± 18.7	NS
High cholesterol <sup>†</sup>	5 (12)	12 (22)	NS
Low HDL-cholesterol <sup>‡</sup>	9 (21)	16 (30)	NS

\*NS ; non-significant

<sup>†</sup> : High cholesterol ; total cholesterol 220 mg/dl

<sup>‡</sup> : Low HDL-cholesterol ; HDL-cholesterol 35 mg/dl

189.7 ± 42.8 mg/dl 193.7 ± 37.3 mg/dl  
41.5 ± 8.3 mg/dl, 44.5 ± 18.7 mg/dl

(220 mg/dl)  
(35 mg/dl)

병변의 침범범위 분석(Table 2)

26%

41%, 74% 59%  
 . 25% American Heart  
 Association  
 $2.7 \pm 1.6$ , 2.9  
 $\pm 1.7$   
 병변의 협착정도 분석 (Table 3)  
 96  
 25% 가 259 . 96  
 50% 가 11  
 18 . 259  
 19 240  
 100 ,  
 140 .  
 grade 2 3  
 11 , 8  
 $58.9 \pm 23.4\%$ ,

**Table 2.** Extent of coronary artery disease

	Stable Angina N = 42 (%)	Unstable Angina N = 54 (%)	p
One vessel	11 (26)	22 (41)	NS
Two vessel	15 (36)	12 (22)	NS
Three vessel	16 (38)	20 (37)	NS
Multi vessel	31 (74)	32 (59)	NS
Lesion number*	$2.7 \pm 1.6$	$2.9 \pm 1.9$	NS

\*Lesion number ; numbers of lesions (>25% stenosis) at AHA segments

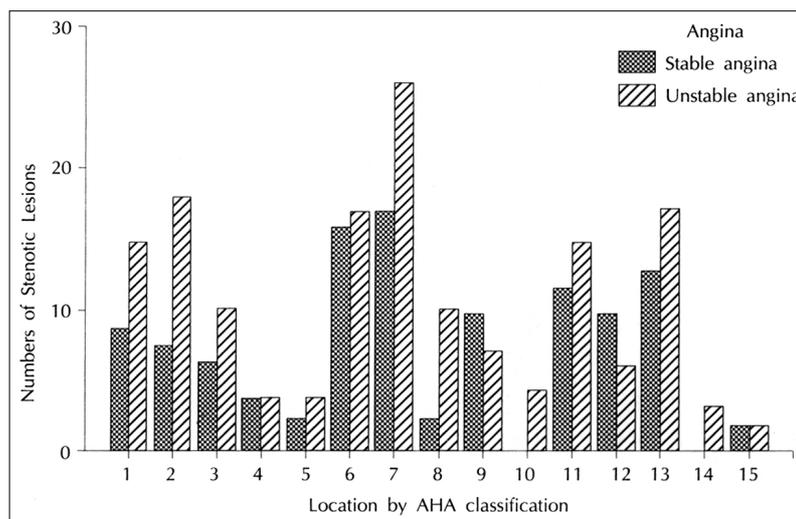
$69.2 \pm 22.1\%$   
 25% 50% , 50% 90%  
 , 90%  
 50%  
 가 (28% vs. 15%,  $p < 0.05$ ) 90%  
 가 가 (22%  
 vs. 34%,  $p < 0.05$ ).

**Table 3.** Severity of coronary artery stenosis

	Stable Angina N = 100 (%)	Unstable Angina N = 140 (%)	p
Total obstruction with grade II-III collaterals	11	8	NS
Mean % stenosis ( $\pm$ SD)	$58.9 \pm 23.4$	$69.3 \pm 22.2$	NS
Numbers of vessels with <50% obstruction	28 (28)	21 (15)	<0.05
Numbers of vessels with 50% < 90% obstruction	50 (50)	72 (51)	NS
Numbers of vessels with 90% obstruction	22 (22)	47 (34)	<0.05

**Table 4.** Morphological analysis

	Stable Angina N = 100 (%)	Unstable Angina N = 140 (%)	p
Simple lesion	67 (67)	72 (51)	<0.05
Complex lesion	33 (33)	68 (49)	<0.05
Calcification	9 (9)	10 (7)	NS



**Fig. 2.** Bar diagram presents correlation between the location of stenosis at AHA segments and the type of angina.

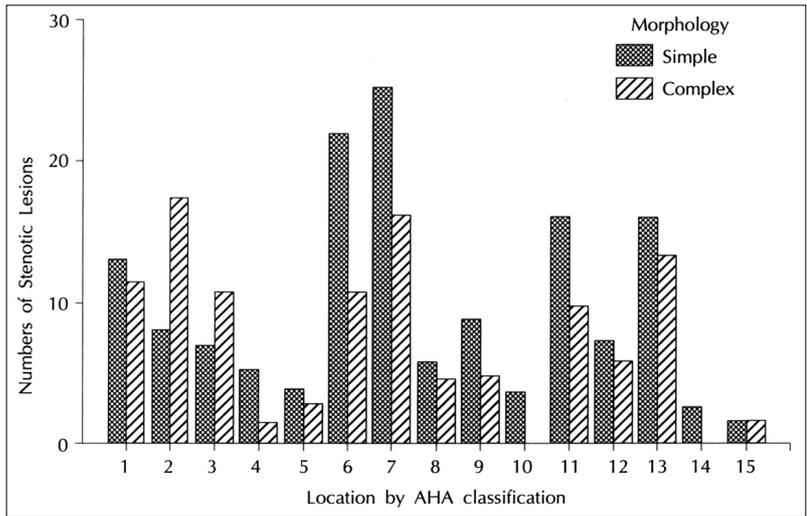


Fig. 3. Bar diagram presents correlation between the location of stenosis at AHA segments and the type of morphology.

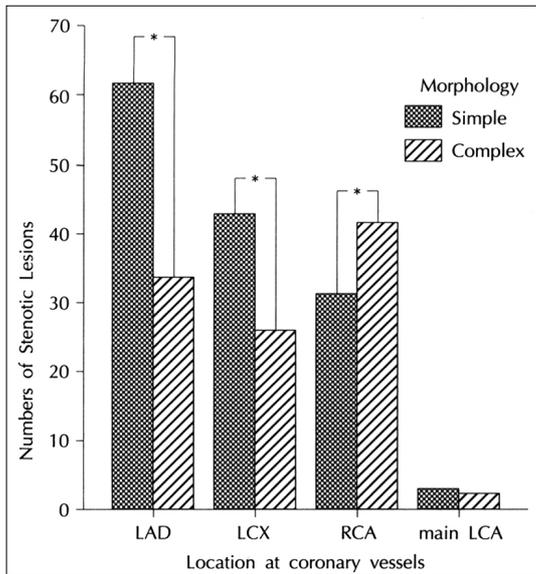


Fig. 4. Bar diagram presents correlation between the location of stenosis at three coronary vessels and the type of morphology (\* ; p<0.05).

63(63%) , 37(37%)  
 68(49%) , 72(51%)

(p<0.05).

10

50%

가

11

가 5 , 가 6 ,  
 7 , 12  
 6 , 5

병변의 위치분석

American Heart Association

1 15

(Fig. 2).

erican Heart Association

15

Am -

(p<0.05)(Figs. 3 and 4).

고 찰

25%

가 .

가 Halt <sup>15)</sup>

가 50%

Ambrose <sup>11)</sup> 75%

가 I Little <sup>9)</sup>

가 II

3 50% 가 66%, 70%

97% 50%

II 가

Dangas <sup>7)</sup> Levin <sup>16)</sup>

가

가

가

가

가 thromboxane A2

가 Falk <sup>4)</sup> <sup>17)18)</sup> Kaski<sup>19)</sup> Sakai <sup>28)</sup>

가

가 Badimon <sup>14)</sup>

가

가

가

90%

50% Kragel <sup>13)</sup> 95%

가

가 Koichi <sup>20)</sup>

50% Ambrose II

가 11 가 50%

가 5 가

25%

50%

대상 및 방법 :

1994 8 1999 6 96

가

American Heart Association

15

AHA

결 과 :

1) 가

23) 59 80 Fawal 가

2) 가

3) 11 ,

가 8

Ryu 24) grade 2 3

4) 가 <50%, 50% , <90%, 90%

가

90%

5) 가 (22% vs. 34%, p<0.05).

가 가 (33% vs. 49%, p<0.05).

6) AHA

(p<0.05).

결 론 :

90%

요 약

가

연구목적 :

50%

중심 단어 :

### REFERENCES

- 1) Ambrose JA, Winters SL, Arora RR. *Angiographic evaluation of coronary artery morphology in unstable angina. J Am Cardiol* 1986;7:472-8.
- 2) Badimon JJ, Fuster V, Chesebro JH, and Badimon L. *Coronary atherosclerosis A multifactorial disease. Circulation* 1993;87 (supp II):II-3.
- 3) Fuster V, Badimon JJ, Badimon L. *Clinical pathological correlations of coronary disease progression and regression. Circulation* 1992;86 (supp III):III-1.
- 4) Erling Falk. *Why do plaques rupture? Circulation* 1992;86 [suppl III]:III30-42.
- 5) Braunwald E. *Heart disease: a textbook of cardiovascular medicine 5th ed.*:2:1289.
- 6) Fuster V, Stem B, Ambrose JA. *Atherosclerosis plaque rupture and thrombosis. Evolving concepts Circulation* 1990;82 (supplII):47.
- 7) Dargas G, Mehran R, Wallenstein S, Nikolaos A, Courcoutsakis, Kakaraia V, et al. *Correlation of Angiographic Morphology and Clinical Presentation in Unstable Angina. J Am Coll Caiol* 1997;29:519-25.
- 8) Ambrose JA, Tannenbaum MA, Alexopoulos D, Mansen CS, Weiss M, Borriw S, et al. *Angiographic progesion of coronary artery disease and the development of myocardial infarction. J Am Coll Cardiol* 1988;12:56-62.
- 9) Little WC, Constantinescu M, Applegate RJ, Kutcher MA, Burrows MT, Kahl FR, et al. *Can coronary angiography predict the site of subsequent myocardial infarction in patients with mild to moderate coronary artery disease? Circulation* 1988;78:1157-66.
- 10) Austen WG, Edward JE, Frye RL, Gensini GG, Gott VL, Griffith LS, et al. *A Reporting System on Patients Evaluated for Coronary Artery Disease. AHA Committee Report*:7-40
- 11) Ambrose JA, Stephen L. Winters, Andry Stern, Angieng BS, Luis E. et al. *Angiographic Morphology and the Pathogenesis of Unstable Angina pectoris. JACC* 1985;3:609-16.
- 12) Cools FJ, Vrints CJ, Snoeck JP. *Angiographic coronary artery lesion morphology and pathogenetic mechanism of myocardial ischemia in stable and unstable coronary artery disease syndromes. Acta Cardiol* 1996;47:13-30.
- 13) Amy H. Kragel, S. David Gertz, William C. Roberts. *Morphologic comparison of frequency and types of acute lesions in the major epicardial coronary arteries in unstable angina pectoris, sudden coronary death and acute myocardial infarction. JACC* 1991;18:801-8.
- 14) Badimon L, Chesebro JH, Badimon JJ. *Thrombus formation in ruptured atherosclerosis plaques and Rethrombosis on evolving thrombi. Circulation* 1992;86[supplIII]: III74-85.
- 15) Jacob I Halt, Bruce J Halk, Jonathan E Goldstein. *Development of significant coronary artery lesions in areas of minimal disease. Chest* 1988;94:731-6.
- 16) David C. Levin, John T. Fallon. *Significance of the angiographic morphology of localized coronary stenoses. Histopathologic correlations. Circulation* 1982;66:316-20.
- 17) Haberg M, Svensson J, Samuelsson B. *Current concepts of thrombogenesis. Role of platelets. Mayo Clin Proc* 1971; 56:102-12.
- 18) Hirsh PD, Hillis LD, Campbell WB, Firth BG, Willerson JT. *Release of prostaglandins and thromboxane into the coronary circulation in patients with ischemic heart disease. N Engl J Med* 1981;304:685-91.
- 19) Kaski JC, Chester MR, Chen L, Katritsis D. *Rapid angiographic progression of coronary artery disease in patients with angina pectoris. The role of complex stenosis morphology. Circulation* 1995;92:2058-65.
- 20) Koichi Y, Hisato T, Takahiko S, Hiroaki H, Shinsuke O, Tetsuo M, et al. *Process of progression of coronary artery lesions from mild to moderate stenosis to moderate or severe stenosis. Circulation* 1999;100:903-9.
- 21) Sakai Y, Tomobuchi Y, Hashizurme T, Imanishi T, Tomimoto S, Toyoda Y, et al. *Lesionrelated factors associated with restenoses after percutaneous transluminal coronary angioplasty with the absence of patientrelated factors. J Cardiol* 1997;29:1-6.
- 22) Lee RY, Han YC, Jee JH, Cho BD, Chae GS, Jang MK, et al. *Angiographic Coronary Artery Lesion Morphology and Intracoronary Thrombus in the Patients with Stable and Unstable Angina Pectoris. Korean J. of Medicine* 1996; 51:774-80.
- 23) El Fawal MA, Berg GA, Wheatley DJ, Harland WA. *Sudden coronary death in Glasgow: Nature and frequency of acute coronary lesions. Br Heart J* 1987;57:329-35.
- 24) Ryu KH, Lee Y. *Relatioship between Clinical Manifestation and Coronary Angiographic Morphology in Patients with Unstable Angina Pectoris. Korean Circ* 1993;23:3-13.