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Angiographic Features at Ischemia- or Infarct-Related Sites in Patients With Acute Coronary Syndrome: Morphology Changing in a Relatively Short Time

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Abstract

Objectives. Coronary cineangiography was reviewed in patients with acute coronary syndrome to investigate whether angiographic features at identifiable ischemia- or infarct-related lesions can change in terms of luminal diameter and morphology in a short period after development, and whether the amount of thrombus with/without underlying ruptured plaque is the major determinant.

Methods. The present study included 72 patients with unstable angina, 118 with acute myocardial infarction(＜1 month after onset)and 137 with old myocardial infarction(≥1 month after onset). The coronary angiographic findings were compared with those from patients with stable effort angina. The groups of patients were subdivided into two groups based on whether antiplatelet and/or anticoagulant therapy, or fibrinolytic therapy with subsequent anticoagulant agents(antiplatelet and/or anticoagulant, or fibrinolytic therapy)were administered. The morphologies of the ischemia- or infarct-related lesions were classified as totally occlusive, simple(Type Ⅰ lesion)or complex. Complex lesions were further subdivided into Type Ⅱa lesions indicative of the presence of thrombus accumulation with/without an underlying ruptured atheromatous plaque, *e.g*., narrowing with irregular, poorly defined or hazy borders, sharp leading or trailing edges that overhang or are perpendicular to vessel walls, and globular endoluminal negative images, Type Ⅱb lesions with two or more serial, closely spaced narrowings together with multiple irregularities, and Type Ⅱc lesions indicative of the presence of some parts of ruptured plaque with a smaller amount of thrombus, *e.g*., luminal narrowing with extraluminal contrast pooling, single or paired short thin linear radiolucencies with/without a variable degree of outpouching, and narrowing with definite outpouching with/without radiolucency, and Type Ⅱd lesions showing narrowing with morphology not included in Type Ⅱa−Ⅱc lesions. The coronary angiographic findings were related to the elapsed time before the coronary angiographic study, and whether each patient underwent antiplatelet and/or anticoagulant, or fibrinolytic therapy.

Results. Ischemia- or infarct-related lesions were totally occlusive in 9.7% of patients with unstable angina, 40.7% of those with acute myocardial infarction and 21.9% of those with old myocardial infarction. Total occlusion was significantly more prevalent in patients with acute myocardial infarction than in those with stable effort angina(23.9%, $p < 0.05$), and total occlusion was more frequent in patients without antiplatelet and/or anticoagulant, or fibrinolytic drugs(50.6% vs 17.1%, $p < 0.01$). The presence of total occlusion decreased with time after development, and the decrease was more significant with the use of antiplatelet and/or anticoagulant, or fibrinolytic drugs. Type Ⅱa morphology was significantly prevalent immediately after the initial episode, and Type Ⅱa and Ⅱc morphologies increased in the late period, and were more frequent with the use of antiplatelet and/or anticoagulant, or fibrinolytic drugs.

Conclusions. The severity of the luminal diameter at ischemia- or infarct-related lesion sites can progress or even regress in a relatively short period in patients with acute coronary syndrome, and the

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amount of accumulated thrombus with/without underlying ruptured plaque is a major determinant of luminal diameter narrowing and angiographic morphology.

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Key Words

■ **Angiocardiography** ■ **Coronary artery disease** ■ **Thrombolysis**
■ **Myocardial infarction, pathophysiology** ■ **Unstable angina**

INTRODUCTION

Recently, coronary angiographic data was reviewed from patients with ischemic heart disease, and demonstrated that identifiable ischemia- or infarct-related lesions (IRLs) are accompanied by certain features specific to clinical settings[1], including the presence of accumulated thrombus with/without underlying ruptured plaque, or ruptured plaque with/without overlying thrombus[2−16]. Patency at the culprit sites also increased with time, based on the morphological analyses of coronary angiograms from patients with acute coronary syndrome sharing a common pathogenesis[1]. The coronary angiographic features at the culprit sites in acute coronary syndrome could result mainly from the amount of accumulated thrombus with/without underlying ruptured plaque, and that the grade of luminal stenosis and morphology at the sites could change in a relatively short period.

This study further evaluated the data to obtain information on whether the amount of thrombus with/without underlying ruptured plaque is a major determinant of stenosis grade and morphology at IRL sites in acute coronary syndrome, the effect of elapsed time before the coronary angiographic study, and the use of antiplatelet and/or anticoagulant therapy, or fibrinolytic therapy with subsequent anticoagulant agents (antiplatelet and/or anticoagulant, or fibrinolytic therapy) at or before the coronary angiographic study.

PATIENTS AND METHODS

Patients

This study included 327 patients with acute coronary syndrome who underwent a coronary angiographic study for the first time and whose baseline angiographic coronary morphology findings were reviewed recently (**Table 1**)[1]. Seventy-two patients had an association with unstable angina pectoris (UAP)[17], 118 with acute myocardial infarction (AMI, <1 month after onset), and 137 had old myocardial infarction (OMI, ≥ 1 month after onset). The variables from the patients were compared with the control data from 71 patients with stable effort angina pectoris (SAP).

Coronary angiography

Baseline medical therapy was continued in most patients. All coronary angiographic studies were performed using Toshiba equipment (ANGIOREX-C/Ω, Toshiba Co.) at a film speed of 50 frames/sec using either Judkins' or Sone's technique. The effects of the coronary vasomotor tone on coronary luminal diameter size were minimized by nitrate administration. Coronary angiography analyses were performed using cinefilm viewers (ELK CAP-35B V, Nishimoto Sangyo, Co.) by three independent observers.

An identifiable ischemia- or infarct-related coronary artery was defined as the presence of at least two of the following: a perfused area distal to the lesion on a specific coronary artery compatible with the distribution of transient or persistent ischemic ST changes on 12-lead electrocardiography; a transient or persistent asynergic area on two-dimensional echocardiography and/or left ventriculography; or an area accumulated by technetium-99m pyrophosphate or with a transient or persistent perfusion defect detected by thallium-201 scintigraphy[18]. An identifiable IRL was defined as complete occlusion, complex morphology, or the most severe stenosis[10].

Intraluminal diameter stenosis was quantified in orthogonal views with a digital analyzing system (CAM-1000, Nishimoto Sangyo, Co.). Intraluminal stenosis in lesions consisting of two or more closely spaced serial narrowings and accompanied by diffuse luminal irregularities or a ribbon lesion was determined based upon the most severe site.

Angiographic coronary morphology at the sites of identifiable ischemia- or infarct-related lesions

Angiographic morphology at the sites of IRLs was classified as total occlusion or variable degrees and forms of luminal narrowing based upon the

Table 1 Patient characteristics

	SAP group (n=71)	UAP group (n=72)	AMI group (n=118)	OMI group (n=137)
Mean age (yr, range)	62.1±9.1 (33−78)	62.6±10.4 (36−83)	59.8±11.2 (31−73)	59.0±9.7 (31−78)
Male	54 (76.1%)	60 (83.3%)	102 (86.5%)	107 (78.1%)
Antiplatelet and/or anticoagulant,	7 (9.9%)	36 (50.0%)**	20 (16.9%)**	36 (26.3%)**
or fibrinolytic agents	0	4 (5.6%)**	15 (12.7%)**	39 (28.5%)**
Elapsed time (days)	432.6±702.6	35.1±26.0*	8.7±12.1*	84.8±204.6*

Continuous values are mean±SD. *p<0.05, **p<0.001 (vs patients with stable effort angina pectoris).
Elapsed time: Time between the first ischemic episode or development of angina, or myocardial infarction and coronary angiography. Fibrinolytic agent: Fibrinolytic and subsequent anticoagulant agents.
SAP=stable effort angina pectoris; UAP=unstable angina pectoris; AMI=acute myocardial infarction; OMI=old myocardial infarction.
〔Adapted with modifications from J Cardiol 2000; **36**: 91−102〕

agreed interpretations of all observers. The morphologies of the IRLs were classified as totally occlusive, and simple (Type Ⅰ lesions) or complex. Complex lesions were further subdivided into Type Ⅱa lesions showing narrowing with irregular, poorly defined or hazy borders, sharp leading or trailing edges that overhang or are perpendicular to vessel walls, and globular endoluminal negative images, Type Ⅱb lesions with two or more serial, closely spaced narrowings together with multiple irregularities, Type Ⅱc lesions showing luminal narrowing with extraluminal contrast pooling, single or paired short thin linear radiolucencies with/without a variable degree of outpouching, and narrowing with definite outpouching with/without radiolucencies, and Type Ⅱd lesions showing narrowing with morphology not included in Type Ⅱa−Ⅱc lesions (**Fig. 1**). The definition of morphology was presented in detail previously[1].

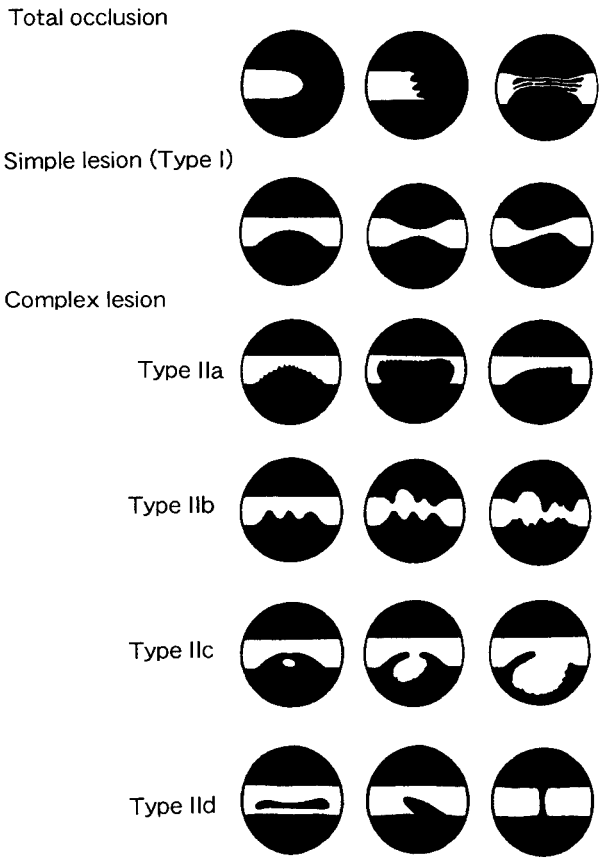


Fig. 1 Schematic coronary morphology at the diseased sites
Morphology was classified as total occlusion, and simple (Type Ⅰ) or complex (Type Ⅱ) lesions. Complex lesions were subdivided into Types Ⅱa−Ⅱd lesions. See text for details.
〔From J Cardiol 2000; **36**: 91−102 with permission〕[1].

Data analyses

Variables derived from coronary angiography analyses were determined using either the unpaired *t*-test or chi squared test. ANOVA was used to compare data between three or more groups. Significance was defined as a p value below 0.05. Values are expressed as mean ± SD.

RESULTS

The clinical profiles of the groups are summarized in **Table 1**. Patients were aged from 31 to 83 years, but there was no significant difference in either age or sex between any of the groups. The elapsed time between the first ischemic anginal

episode, or development of coronary events, and the coronary angiographic study was significantly different between all groups.

Seven patients of the 71 with SAP(9.9%)were receiving antiplatelet therapy at the coronary angiographic study; 36(50.0%)and 4 patients(5.6%)of the 72 with UAP had received antiplatelet and/or anticoagulant or fibrinolytic therapy at or before the coronary angiographic study, respectively; 20 (16.9%)and 15 patients(12.7%)of the 118 with AMI had received anticoagulant and/or antiplatelet therapy or fibrinolytic therapy, respectively; and 36 (26.3%)and 39 patients(28.5%)of the 137 with OMI had received anticoagulant and/or antiplatelet therapy or fibrinolytic therapy, respectively(**Table 1, Figs. 1, 2**). Antiplatelet and/or anticoagulant therapy was administered to more patients with UAP, AMI, and OMI than those with SAP($p < 0.01$). Similarly, more patients with UAP, AMI, and OMI had received fibrinolytic therapy than those with SAP($p < 0.001$).

Antiplatelet therapy in patients with SAP included aspirin or ticlopidine. Anticoagulation therapy used bolus or continuous heparin administered subcutaneously or intravenously with/without warfarin, or oral warfarin with/without antiplatelet agents. Fibrinolytic therapy was performed using continuous infusion of urokinase or alteplase with/without following heparin or warfarin. The antiplatelet and/or anticoagulant, or fibrinolytic therapy was transient, and the therapy appeared insufficient in some patients. Based on whether the patients had undergone antiplatelet and/or anticoagulant, or fibrinolytic therapy at or before the coronary angiographic study, the groups were subdivided into 7 patients with SAP(+)and 64 with SAP(−), 40 with UAP(+)and 32 with UAP(−), 35 with AMI(+)and 83 with AMI(−), and 75 with OMI (+)and 62 with OMI(−)(**Table 2**).

Coronary angiographic findings

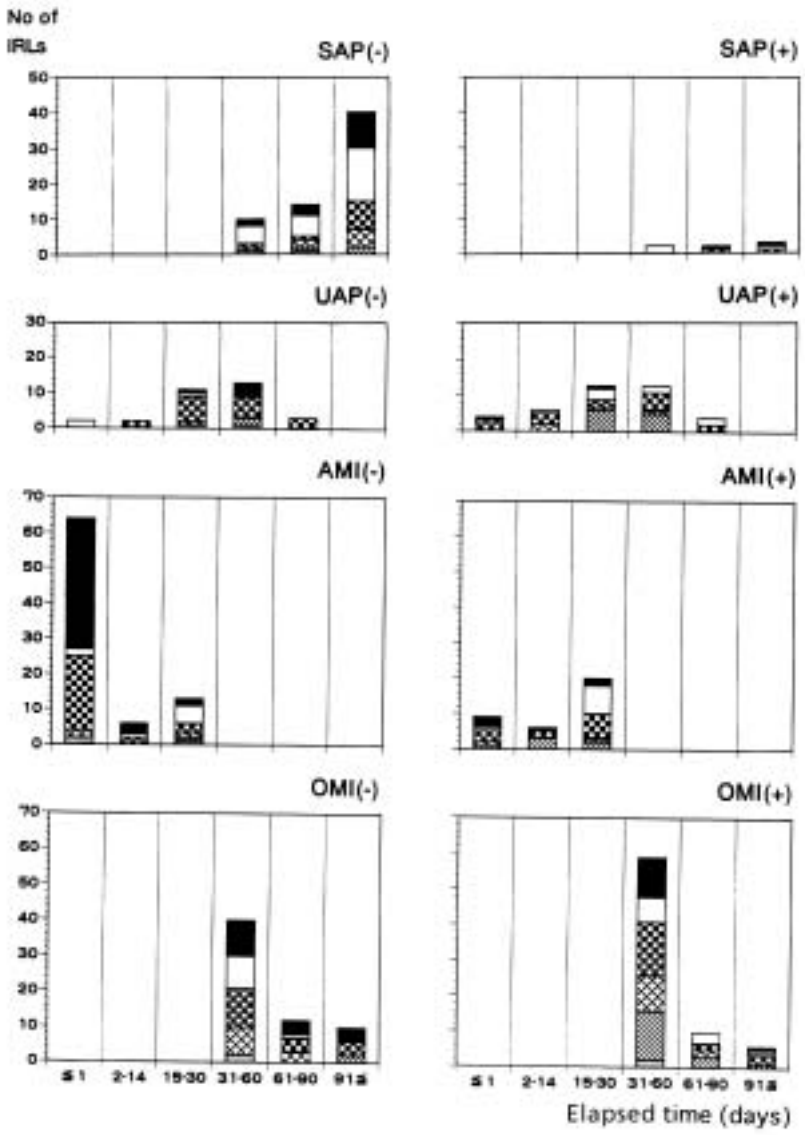
The coronary angiographic findings are summarized in **Tables 2, 3**, and **Fig. 2**. In the patient groups with SAP, UAP, AMI and OMI, 17, 7, 48 and 30 IRLs, respectively, were totally occulusive. The incidence of total occlusion was 48 of 118 IRLs(40.7%)in patients with AMI, and significantly higher than 17 of 71 IRLs(23.9%)in those with SAP($p < 0.05$)[1]. In addition, total occlusion was also more frequent in patients with AMI(−)than in those with AMI(+)(42 of 83 IRLs, 50.6% vs 6 of 35 IRLs, 17.1%, $p < 0.01$).

Mean luminal diameter narrowing in each subdivided group at the sites of IRLs is shown in **Table 2**. The severity of the narrowing was not affected by antiplatelet and/or anticoagulant, or fibrinolytic therapy in the patients with SAP, UAP and OMI. However, the narrowing was significantly less in patients with AMI(+)87.3% than in patients with AMI(−) (95.6%; $p < 0.05$, **Table 2**).

Morphological analyses showed Type IIa (47.2%)and IIc(18.1%)lesions were characteristic in patients with UAP[1], whereas Type IIa morphology was more frequently shown in patients with UAP(−)than in those with UAP(+)(19 of 32 IRLs, 37.5%, $p < 0.05$), and Type IIc morphology was more frequent in patients with UAP(+)than in those with UAP(−)(11 of 40 IRLs, 27.5% vs 2 of 32 IRLs, 6.3%, $p < 0.05$). Type IIa morphology (32.2%)was noted in addition to total occlusion (40.7%)in patients with AMI[1]. However, no significant difference was revealed between the presence in patients with AMI(+)and in those with AMI(−)(12 of 35 IRLs, 34.3% vs 26 of 83 IRLs, 31.3%). Conversely, Type IIc morphology frequency was similar in all patients with SAP and AMI(3 of 71 IRLs, 4.2% vs 7 of 118 IRLs, 5.9 %)[1], but developed more frequently in patients with AMI(+)than in ones with AMI(−)(6 of 35 IRLs, 17.1% vs 1 of 83 IRLs, 1.2%, $p < 0.01$).

Type IIa(28.5%)and IIc morphologies(14.6%) were characteristic features in patients with OMI[1]. However, no significant difference was shown in the development of Type IIa morphology between patients with OMI(+)and those with OMI(−) (20 of 75 IRLs, 26.7% vs 19 of 62 IRLs, 30.6%). Type IIc morphology was significantly less frequent in all patients with SAP compared to those with OMI(3 of 71 IRLs, 4.2% vs 20 of 137 IRLs, 14.6%, $p < 0.05$), and developed more frequently in patients with OMI(+)than in those with OMI (−)(18 of 75 IRLs, 24.0% vs 2 of 62 IRLs, 3.3%, $p < 0.01$).

The coronary morphology of each patient is demonstrated in **Table 3** in the elapsed time between the development of SAP, UAP, AMI and OMI, and the coronary angiographic study. All studies were performed on the day of AMI in patients fulfilling our criteria for urgent revascularization procedure within 6 hr after development[8]. In many patients with UAP, the coronary angiographic studies were performed after stabilization



■ = Total occlusion; □ = Type Ⅰ morphology; ▩ = Type Ⅱa morphology; ▩ = Type Ⅱb morphology; ▩ = Type Ⅱc morphology; ▩ = Type Ⅱd morphology. Type Ⅰ and Ⅱa−Ⅱd morphology, see text for details.

Fig. 2 Numbers of patients in each group who underwent coronary angiographic study on the first day, between the second−14th, 15−30th, 31−60th and 61−90th days, and after the 91st day from the first ischemic episode or development of unstable angina, or myocardial infarction

Elasped time as in Table 1.

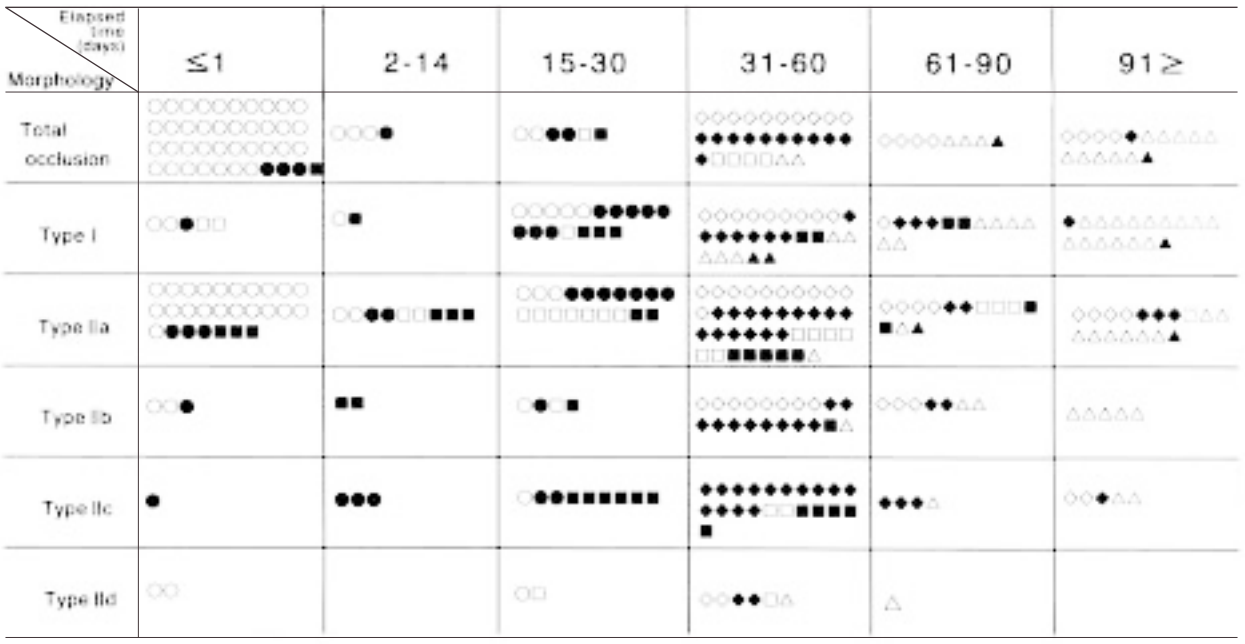
No of IRLs = number of identifiable ischemia- or infarct-related lesions; SAP(+) or SAP(−) = stable effort angina pectoris with/without anticoagulant or fibrinolytic and subsequent anticoagulant therapy(anticoagulant or fibrinolytic therapy); UAP(+) or UAP(−) = unstable angina pectoris with/without anticoagulant or fibrinolytic therapy; AMI(+) or AMI(−) = old myocardial infarction with/without anticoagulant or fibrinolytic therapy; OMI(+) or OMI(−) = old myocardial infarction with/without anticoagulant or fibrinolytic therapy. Other abbreviations as in Table 1.

Table 2 Angiographic findings at sites of identifiable ischemia- or infarct-related lesions in the patients with stable effort angina, unstable angina pectoris, acute myocardial infarction and old myocardial infarction

	SAP group		UAP group		AMI group		OMI group	
	(+) $n=7$	(−) $n=64$	(+) $n=40$	(−) $n=32$	(+) $n=35$	(−) $n=83$	(+) $n=75$	(−) $n=62$
Mean stenosis (%)	85.0±10.6	85.0±11.0	89.6±14.3	94.7±7.0	87.3±13.1	95.6±9.2*	85.4±15.4	89.7±12.7
Total occlusion	2(28.6)	15(23.5)	2(5.0)	5(15.6)	6(17.1)	42(50.6)**	12(16.0)	18(29.0)
Type Ⅰ	3(42.9)	26(40.6)	8(20.0)	3(9.3)	9(25.7)	8(9.6)*	11(14.6)	10(16.1)
Type Ⅱa	2(28.5)	10(15.6)	15(37.5)	19(59.4)*	12(34.3)	26(31.3)	20(26.7)	19(30.6)
Type Ⅱb	0	8(12.5)	4(10.0)	1(3.1)	2(5.7)	3(3.6)	12(16.0)	11(17.7)
Type Ⅱc	0	3(4.7)	11(27.5)	2(6.3)*	6(17.1)	1(1.2)**	18(24.0)	2(3.3)**
Type Ⅱd	0	2(3.1)	0	2(6.3)	0	3(3.6)	2(2.7)	2(3.3)

( ): %.*$p<0.05$, **$p<0.01$(vs patients in each group with antiplatelet and/or anticoagulant, or fibrinolytic therapy).
(+): Use of antiplatelet and/or anticoagulant therapy, or fibrinolytic therapy with subsequent anticoagulant agents(antiplatelet and/or anticoagulant, or fibrinolytic therapy). (−) : No use of antiplatelet and/or anticoagulant, or fibrinolytic therapy. Type Ⅰ and Ⅱa−Ⅱd, see text for details.
Abbreviations as in Table 1.

Table 3 Angiographic coronary morphology at the sites of ischemia- or infarct-related lesions and elapsed time after the first ischemic episode or development of angina or myocardial infarction(days)



● or ○: Acute myocardial infarction with/without antiplatelet and/or anticoagulant therapy, or fibrinolytic therapy with subsequent anticoagulant agents(antiplatelet and/or anticoagulant, or fibrinolytic therapy). ■ or □: Unstable angina pectoris with/without anticoagulant, or fibrinolytic therapy. ◆ or ◇: Old myocardial infarction with/without anticoagulant, or fibrinolytic therapy. ▲ or △: Stable effort angina pectoris with/without antiplatelet and/or anticoagulant therapy. Elaspsed time as in Table 1. Types Ⅰ and Ⅱa−Ⅱd morphology, see text for details.

of disease activity by medical treatment including heparinization[19]. Comparison of the coronary morphology at the sites of the IRLs in patients with AMI who underwent the study on the day of development with that in patients with OMI who underwent the coronary angiographic study between 31 and 60 days after development showed that total occlusion was less frequent in the patients with OMI than in patients with AMI(21 IRLs of 99, 21.2% vs 40 IRLs of 73, 54.8%, $p < 0.01$), with an increasing occurrence of Type Ⅰ(16 IRLs of 99, 16.2% vs 3 IRLs of 73, 4.1%, $p < 0.10$), Type Ⅱb (18 IRLs, 18.2% vs 3 IRLs, 4.1%, $p < 0.05$)and Type Ⅱc morphologies (14 IRLs, 14.1% vs 1 IRL, 1.4%, $p < 0.05$), especially in patients who had received antiplatelet and/or anticoagulant, or fibrinolytic therapy(**Fig. 2, Table 3**). Therefore, shorter elapsed time after the development of acute coronary syndrome was associated with higher prevalence of total occlusion, and longer elapsed time after development was associated with higher prevalence of patency. Furthermore, frequency of total occlusion decreased, and development of Types Ⅰ, Ⅱb and Ⅱc morphologies increased in patients who underwent anticoagulant or fibrinolytic therapy(**Tables 2, 3, Fig. 2**).

DISCUSSION

Patients with UAP had characteristic Types Ⅱa and Ⅱc morphologies at the sites of IRLs, but Type Ⅱa morphologies were more frequently shown in patients with UAP(−), and more Type Ⅱc morphologies were found in patients with UAP(+) (**Tables 2, 3, Fig. 2**). Patients immediately after developing myocardial infarction showed similar incidences of total occlusion and Type Ⅱa morphology despite antiplatelet and/or anticoagulant therapy(**Tables 2, 3, Fig. 2**). In contrast, Type Ⅱc morphology appeared at similar incidences in patients with SAP and with AMI, but developed more frequently in patients with AMI(+) than in ones with AMI(−). The frequency of total occlusion and Type Ⅱa morphology decreased in patients more than one month after myocardial infarction, with no significant difference between patients with OMI (+) and those with OMI(−). Type Ⅱc morphology was more frequent in patients with OMI than in those with AMI[1], with a higher incidence in OMI (+) than in AMI(+)(**Tables 2, 3, Fig. 2**).

Therefore, the morphology at the sites of IRLs in acute coronary syndrome can change in a relatively short time(**Tables 2, 3, Fig. 2**), with coronary morphology and narrowing resulting mainly from the amount of thrombus accumulated with/without underlying ruptured atheromatous plaque or ruptured plaque with/without an overlying thrombus. Subsequently, the patency and morphology at the sites of IRLs are affected by the elapsed time after the first development of symptoms, and whether antiplatelet and/or anticoagulant, or fibrinolytic therapy was administered.

Study limitations

There were some limitations to the present study. First, the coronary angiographic study has methodological limitations specific to the approach, even if recorded in enough projections for analysis[1]. Second, the coronary angiography is a shadowgram and provides only indirect estimates of the coronary arteries, and the morphological features and severity of the diseased sites are never definite. Third, we cannot correlate the coronary angiographic features and histological findings[1]. Type Ⅱa complex lesions are probably indicative of thrombus accumulation with/without an underlying ruptured atheromatous plaque[5,6,20,21], although this has not yet been clearly correlated with histological findings. We do not have enough information about what each morphology represents, except for the histological findings from two patients[8,9], and sequential coronary angiographic findings in patients with AMI who underwent intracoronary urokinase therapy. The development of Type Ⅱc morphology was demonstrated at severely or totally occlusive sites at baseline on the sequential coronary angiography during and after progressive removal of the overlying thrombus and plaque content by intracoronary urokinase. A further reduction in the grade of stenosis at the culprit sites was shown 1 month after aggressive anticoagulation therapy with heparin followed by warfarin[8]. The severity of stenoses at culprit sites, with significantly narrow and complex morphology but no myocardial ischemia despite severe stress tests, was reduced after 3−6 months of anticoagulation by warfarin, and was not associated with symptoms of myocardial ischemia[22]. Thus, some Types Ⅱa and Ⅱc lesions may be indicative of thrombus accumulation with/without underlying ruptured atheromatous plaque and of certain parts of ruptured plaque[22,23]. Types Ⅱb and Ⅱc lesions may also be more frequently associated with a smaller amount

of thrombus than Type IIa lesions, and Type IIc lesions may be more often accompanied by ruptured atheromatous plaque than Types IIa and IIb lesions[8,10].

Another major limitation is that the antiplatelet and/or anticoagulant, or fibrinolytic therapy was transient and may have been insufficient in some patients. The coronary angiographic findings could be somewhat different from those in the presence of adequate therapy(**Tables 2, 3, Fig. 2**). We do not know the composition of accumulated thrombi. The location of fissuring on the atheromatous plaque, proximal or distal, determines whether occlusive thrombi easily accumulate, resulting in the development of different clinical manifestations, unstable angina or AMI[24]. This study shows that later *in vivo* observations of patients with acute coronary syndrome under antiplatelet and/or anticoagulant, or fibrinolytic drugs may provide important information on the composition of the accumulated thrombus, shedding further light on the *in vivo* sequence of the pathophysiology.

The elapsed time between the development of UAP or myocardial infarction and coronary angiographic study was widely distributed in each group of patients(**Tables 1, 3, Fig. 2**). All patients with AMI within 6 hr of development and fulfilling our criteria for an urgent revascularization procedure[8] underwent immediate coronary angiographic studies, whereas the other patients not fulfilling the criteria were usually studied 2 to 4 weeks after development. Similarly, the coronary angiographic studies were performed in many patients with UAP after stabilization by medical treatment including heparinization[19]. Subsequently, Types IIa and IIc lesions were characteristic of the patients with UAP, but if the patients had undergone coronary angiography immediately after the initial episodes, Type IIa lesions could have been more frequently found as previously observed[5,6]. Thus, the wide time distribution is another limitation, influencing the results in the present analyses.

Clinical implications

The present analyses of coronary angiograms demonstrated that the morphology and severity at the sites of IRLs in acute coronary syndrome change mainly by thrombus accumulation and removal, resulting in stenosis progression, development of total occlusion, and recanalization and/or reduction in stenosis. The coronary angiographic findings in acute coronary syndrome are occasionally dynamic in nature and simply reflect the coronary angiographic profiles of individuals at the time point of each study, with the severity of the luminal diameter progressing or regressing in a relatively short period. Also, the morphological change in the early phase mainly depends on whether the patients were receiving antiplatelet and/or anticoagulant, or fibrinolytic drugs, and on the time interval between the onset of symptoms and the coronary angiographic study in the late phase. Other factors may also be critical to the morphological features, including the duration of antiplatelet and/or anticoagulant therapy, whether the patient is receiving antiplatelet and/or anticoagulant agents at the time of the coronary angiographic study, and the elapsed time after the last ischemic episode at rest.

Diseased sites with a complex morphology are prone to progress[2,8,25–31], although there are exceptions[18,19,32]. It is more important to recognize that the severity of the luminal diameter in relatively substantial number of diseased sites could even regress over a short period. Therefore, antiplatelet and/or anticoagulant, or fibrinolytic therapy may be beneficial in certain patients with acute coronary syndrome, especially in those with recent onset and spontaneous and/or prolonged anginal pain[19,31,33], who cannot undergo invasive procedures including coronary angiographic study, although it has been reported that fibrinolytic agents are not so effective in patients with UAP.

At present, we can easily and widely perform coronary angiographic studies, which provide *in vivo* pathophysiological information on the coronary circulation in individual patients. When we establish what each morphology actually represents by histological studies, how the morphology changes over time, and how these changes are modified by pharmacological interventions, coronary angiographic studies will improve our understanding of the pathophysiology for individual patients, and the natural history of "coronary artery disease", providing information for optimal therapeutic strategies[19,34–38].

要　　約

急性冠症候群における冠動脈造影上の責任病変の形態は短期間に変化する

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目　的: 急性冠症候群における責任病変の冠動脈造影上の狭窄度や形態は短期間内に変化しうるか，その変化は付着する血栓やその下にあるとされる破裂粥腫により影響されるかを検討する．

方　法: 対象は不安定狭心症群(UAP)の72例，急性心筋梗塞群(発症1ヵ月以内，AMI)の118例，および陳旧性心筋梗塞群(発症1ヵ月以上，OMI)の137例で，安定労作狭心症(SAP)の71例を対照群とした．これらの症例群を冠動脈造影施行前に抗血小板薬，抗凝固薬の双方，またはその一方，あるいは線溶療法(抗血小板・抗凝固・線溶療法）を受けたかにより，各群をSAP(＋)と(－)，UAP(＋)と(－)，AMI(＋)と(－)，およびOMI(＋)と(－)に分けた．病変形態は完全閉塞，単純病変(TypeⅠ）および複雑病変(TypeⅡ)に分け，TypeⅡ病変をさらにhazinessやoverhangを伴い血栓が付着しているとされるもの(TypeⅡa)，multiple irregularityを伴うもの(TypeⅡb)，円形の管腔外造影剤貯留やoutpouchingを伴う破裂粥腫の一部が描出されていると思われるもの(TypeⅡc)，その他(TypeⅡd)に分類した．ついで，病変の形態と初回症状発生から冠動脈造影施行までの経過日数との関係を検討した．

結　果: 責任病変はSAPでは23.9%が，UAP，AMI，OMIでは9.7%，40.7%，21.9% が完全閉塞を示した．完全閉塞はAMI，とくにAMI(－)に多く{50.6% vs 17.1%[対 SAP(－)]，$p<0.01$}，時間の経過とともに減少した．開通例は抗凝固・線溶療法施行例に多くみられた．開通例の病変形態は発症後早期にはTypeⅡaが多く，時間経過に伴う完全閉塞の減少とともに，TypeⅡb，TypeⅡcの増加が観察された．この傾向は抗血小板・抗凝固・線溶療法施行例でより著明であった．

結　論: 急性冠症候群における責任病変の狭窄度や形態は短期間内に進行したり，軽減しうる．変化の決定要因は，付着する血栓量やその下にあるとされる破裂した粥腫，および症状発生後の経過時間と考えられる．これらを認識して解析することにより，冠動脈造影のみならず，当該症例の病態の解釈がより容易になると思われる．

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