Symposium on Acute Coronary Syndrome: Pathogenesis and Treatment*

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1. Development of Acute Coronary Syndrome and Progression of Coronary Artery Disease: A Serial Clinical-angiographic Analysis

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Key words: atherosclerotic lesion, progression, collateral circulation, coronary angiography

Coronary angiography has been widely used in daily clinical practice for evaluating the patients with suspected and known coronary artery disease. Although we can not directly observe the atherosclerotic vessel walls of the coronary artery by coronary angiography, angiographically apparent stenoses are well correlated to clinical manifestations.

Coronary atherosclerosis is a progressive process, angiographic pattern of progression and development of acute coronary syndromes is not well defined. In this review, we will summarize the evidence of the relationships between the angiographic progression and development of acute coronary syndrome.

Coronary angiographic findings in acute myocardial infarction

Angiographic studies performed in the earliest hours of acute myocardial infarction in patients presenting with ST-segment elevation have revealed approximately a 90% incidence of total occlusion of the infarct-related vessel (1). Recanalization from spontaneous thrombolysis as well as attribution due to some mortality among those patients with total occlusion results in a diminishing incidence of angiographically totally occluded vessels in the period following myocardial infarction. In contrast to patients with a Q-wave infarction, those patients who sustain a non-Q-wave infarction have a much lower incidence (30%) of complete occlusion of the infarct-related coronary artery.

Intracoronary infusion of streptokinase was reported as successful therapy for acute myocardial infarction (2). Soon after that, the common morphologic feature, probably representing a disrupted atherosclerotic plaque, on angiogram in acute myocardial infarction after coronary thrombolysis was found. In many series of patients studied in the early and subacute phase by serial coronary angiograms, 5–10% of patients with acute myocardial infarction are found to have mild stenotic vessels in the subacute phase. In these patients, thrombus that has lysed and the transient occlusive platelet aggregate may have been responsible for occlusion. These clinical data suggest that angiographically complex lesions with thrombosis are frequent in the pathogenesis of myocardial infarction.

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Coronary angiographic morphology in myocardial infarction and unstable angina

The angiographically common features of an eccentric stenosis with a narrow neck or irregular borders, or both were found in patients with unstable angina and with myocardial infarction who underwent intracoronary streptokinase infusion (3). These results suggested unstable angina and myocardial infarction might form a continuous spectrum with clinical syndrome dependent on the subsequent change in coronary supply relative to myocardial demand.

Development of acute coronary syndrome and the progression of coronary atherosclerosis

Progression pattern of coronary atherosclerosis

The relationship between angiographic progression of atherosclerosis and clinical outcomes such development of acute coronary syndrome has been confirmed by the serial clinical-angiographic analysis (4). But serial angiographic studies have shown that progression of coronary atherosclerosis is a highly unpredictable process that follows a nonlinear course and that information about the morphology and stenotic severity can not predict future progression (5).

Development of acute coronary syndrome

Coronary occlusion and myocardial infarction most frequently evolve from mild to moderate stenosis (Fig. 1), (6–7). Studies of patients who develop acute myocardial infarction after having undergone coronary arteriography at some time before its occurrence have been helpful in clarifying coronary anatomy before infarction. The majority of occlusions actually occur in vessels with a previously identified stenosis less than 70% on angiogram performed months to years earlier. Less stenotic plaques are vulnerable to rupture than the larger plaques, and the smaller plaques could be most dangerous just because of their number. These findings support the concept that acute myocardial infarction occurs as a result of sudden thrombotic occlusion at the site of rupture of a previously nonobstructive but lipid-rich plaque. These angiographic findings have been prospectively confirmed in FATS (8).

The Coronary Artery Surgery Study (CASS) prospectively evaluated 2,938 non bypass coronary segments in 298 patients. Although individual severe stenosis became occluded more frequently than did an individual less severe stenosis (<75% stenosis at baseline), the less stenotic lesions gave rise to more occlusions than did severely obstructive lesions because of their greater number. It appear to be impossible to predict the progression to total obstruction in the mildly to moderately stenotic lesions because of the very low progression rate.

Moderate lesions and collateral development

The mild to moderate rather than severe stenotic lesions are more likely to lead to acute coronary syndromes in case of abrupt occlusion because they are less frequently associated with protective collateral circulation (9). Collateral blood flow and other factors – the presence and location of stenoses in other coronary arteries, the rate of development of the obstruction and the quantity of myocardium supplied by the obstructed vessel – all influence acute clinical events and the viability of myocardial cells distal to the occlusion.

Prevention of acute coronary syndrome

Previous studies have shown that revascularization does not prevent myocardial infarction in patients with stable coronary artery disease (10, 11). The pathophysiology of development of acute myocardial infarction provides the basis for understanding why revascularization does not prevent myocardial infarction. Both coronary artery bypass graft surgery and percutaneous transluminal angioplasty (PTCA) are directed at more severe coronary stenoses. At present we do not have a useful clinical method to identify the dangerous lesions that will lead to myocardial infarction in the future. In contrast to revascularization, lipid lowering therapy reduces the rate of myocardial infarction by 30% over a period of 5 years (12). Many angiographic and clinical trials indicate that human plaques may be stabilized against rupture or disruption even when regression does not occur. For the prevention of acute coronary syndrome, a systemic therapy to the entire coronary artery is clinically important.

References

Role of Plaque Rupture

1985.


2. The Role of Plaque Rupture in the Development of Acute Coronary Syndrome Evaluated by the Coronary Angioscope

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Key words: atherosclerosis, vulnerable plaque, hyperlipidemia, yellow plaque, thrombus

Rupture of an atherosclerotic plaque in the coronary arteries is observed in patients who died suddenly or shortly after an episode of unstable angina or myocardial infarction. This pathological finding was first reported in 1844, but its importance was focused upon nearly 150 years later with the findings of the coexistence of plaque rupture and thrombosis. These pathological findings have been studied in the clinical setting following coronary angiography performed in the acute stage of patients with acute myocardial infarction or unstable angina. The irregularity of the arterial wall was suggested to be the plaque disruption and the contribution of the thrombus to the onset was clarified by reperfusion of the occluded artery using thrombolytic agents. Now, It is generally accepted that the sudden rupture of the plaque surface and the subsequent thrombus formation underlies a great majority of acute coronary syndromes (1).

Coronary angioscope would be useful to observe the color of the plaque surface and the characteristics of the thrombus. The coronary angiographic catheter (Nihon Koden, Japan) used in this study had an outer diameter of 0.73 mm and a fiber containing 6,000 pixels, which might be useful to observe the lesion characteristics in patients with acute coronary syndrome (2, 3). The glittering yellow plaques have been observed in unstable coronary lesions by angioscope, and are thought to be vulnerable plaques (4).

In this article, we focused on the yellow plaque, and demonstrated the progression of the yellow plaque frequently observed in the angiographically normal coronary artery and its rupture as a cause of acute coronary syndrome based on our findings by a coronary angioscope according to the atherosclerotic process (5).

Plaque formation

The first atherosclerotic lesion consisting of fatty dots or streaks barely raised above the intimal surface, is formed by lipid-laden form cells transformed from macrophages which take modified LDL inside through the scavenging receptors. The fatty streak is first recognized as a thin yellow plaque by angioscope in angiographically normal artery. We studied the incidence of the yellow plaques in the angiographically normal coronary arteries and found that yellow plaques were observed in 65 coronary arteries (72%) among 90 angiographically normal coronary arteries. The mean number of the isolated yellow plaques was 3 in the coronary arteries with yellow plaques. This high incidence of yellow plaques was due to the selection bias because the subjects studied were suspected to have coronary heart disease.

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