SCIENTIFIC LETTER

Positive correlation between coronary arterial remodelling and prodromal angina in acute myocardial infarction

Y Shimada, M Yoshiyama, Y Kobayashi, H Tanaka, S Jissho, H Iida, Y Nakamura, S Ehara, K Shimada, K Takeuchi, J Yoshikawa

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Recently, the relation between coronary arterial remodelling and ischaemic coronary disease has become a focus of investigation.[1] Positive coronary arterial remodelling has been shown to be more frequent in acute coronary syndromes, including acute myocardial infarction (AMI), whereas negative remodelling has been shown to be more frequent in stable angina.[2] However, in the case of AMI, preceding clinical presentations of the onset vary among patients and it is well known that patients often undergo episodes of unstable or stable angina.[3] Furthermore, no studies have correlated remodelling and the presence of prodromal symptoms. In this study intravascular ultrasound (IVUS) was performed before coronary intervention to evaluate the possible correlation between the arterial remodelling at the culprit lesion site of AMI and the prodromal symptoms.

METHODS

Ninety four consecutive patients presenting with their first AMI were considered for entry into this study. AMI was diagnosed on the basis of coronary angiography, ECG, prolonged chest pain (> 30 minutes), and subsequent elevation of creatine kinase. Twenty two of these patients were excluded because of either an inability to make the IVUS catheter pass across the lesion before angioplasty ($n = 5$), no proximal reference site defined due to a large side branch ($n = 4$), or poor ultrasound image quality ($n = 13$). Finally, 72 patients (60 men, 12 women) with a mean age of 62 (range 43–81) years met our criteria. The detailed status of prodromal symptoms[3] was obtained by a physician from all of the patients. Informed written consent was obtained from either the patient or their family before catheterisation.

An IVUS catheter (30 MHz, 3.2 French, Ultracross or 40 MHz, 3.0 French, Atlantis, Boston Scientific) was advanced distal to the target lesion before any intervention, and then pulled back automatically (0.5 mm/s). The images were recorded on 0.5 inch S-VHS videotape for off line quantitative and qualitative analysis.[1][2] The remodelling ratio (RR) was defined as the ratio of the external elastic membrane (EEM) area at the lesion site to that at the proximal reference site. Positive remodelling was defined as an RR > 1.1 and negative remodelling as an RR < 0.9.

All data are presented as mean (SD). Continuous variables were compared with an unpaired t test and categorical variables were compared with the χ^2 test. For all analysis, $p < 0.05$ was considered significant.

RESULTS

Overall, 36 patients (50%) had an episode of sudden onset, and 36 (50%) had prodromal angina (19 worsening angina, 12 new onset of rest angina, 4 stable angina). There was no significant difference in sex, frequency of risk factors, or lesion location. However, patients without prodromal angina were significantly older than those with prodromal angina (64.1 (8.1) years *v* 59.8 (9.1) years, $p < 0.05$). There was no significant difference in EEM area (19.1 (7.6) mm^2 *v* 16.3 (5.9) mm^2), lumen area (2.2 (1.1) mm^2 *v* 2.2 (1.0) mm^2), or plaque area (17.0 (7.2) mm^2 *v* 14.1 (5.9) mm^2) at the culprit lesion site. However, RR was significantly greater in patients without prodromal angina (1.1 (0.3) *v* 0.8 (0.2), $p < 0.001$) The frequency of fibrofatty, fibrous, calcified, and mixed plaques was 25 (69%), 4 (11%), 6 (17%), and 2 (3%), respectively, in patients without prodromal angina, and 20 (56%), 4 (11%), 8 (22%), and 4 (11%), respectively, in patients with prodromal angina.

In the whole group of patients, 22 of the 72 patients (31%) showed positive remodelling, whereas 27 patients (38%) showed negative remodelling. The frequency of the preceding clinical presentations was significantly different between the positive and negative remodelling groups ($p < 0.01$) (fig 1). An episode of sudden onset was more common in the positive remodelling group (77% *v* 30%); conversely an episode of prodromal angina was more common in the negative remodelling group (23% *v* 70%).

DISCUSSION

To our knowledge, this is the first study that suggests a positive correlation between coronary arterial remodelling of the culprit lesion site and preceding clinical presentation in AMI. Several reports have demonstrated that, in unstable angina, positive remodelling is observed in 30–50% of culprit lesions, whereas negative remodelling is seen in 20–40%.[1][2] The result of our study is consistent with these findings. However, in our study, we included only patients with AMI, because of a possibility that remodelling of the coronary arteries related to AMI is different from that in other ischaemic diseases, just as the mechanisms of lesion progression are considered to be different as well.

It is well known that approximately half of patients with AMI have experienced angina.[3] Many angiographic studies demonstrate that most lesions responsible for AMI are minimally occlusive before the onset; however, the presence of prodromal angina suggests the existence of severe stenosis. Recent angiographic studies demonstrate that there are some cases that reveal a rapid progression of coronary artery lesions before the onset of AMI.[4] As an episode of sudden onset is suggestive of a sudden occlusion of a coronary artery, the presence of prodromal angina may stand for the rapid progression of coronary stenosis. In this study, arterial remodelling was associated with the presence of prodromal symptoms. Therefore, our results suggest that there may be a relation between arterial remodelling and plaque progression or disruption in AMI.

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Abbreviations: AMI, acute myocardial infarction; EEM, external elastic membrane; IVUS, intravascular ultrasound; RR, remodelling ratio
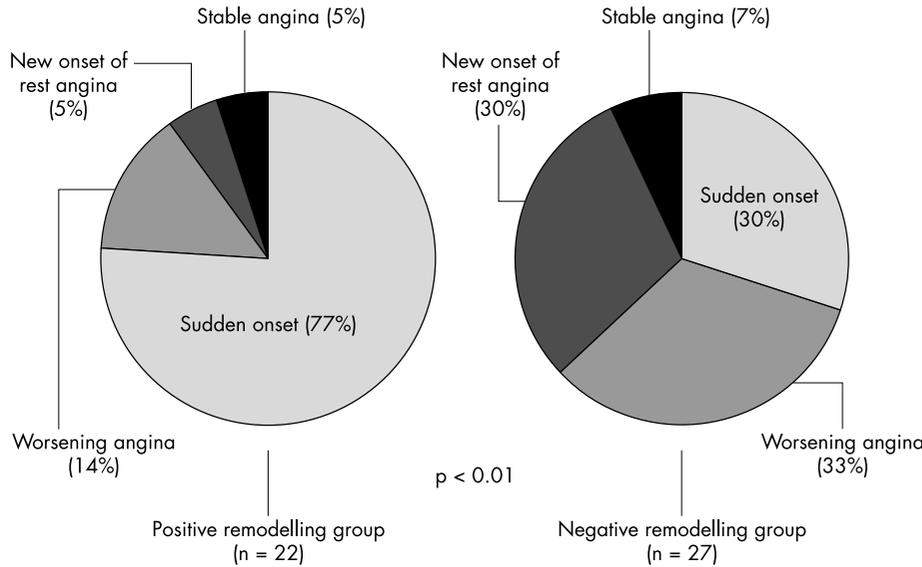
Figure 1 Pie charts showing the preceding clinical presentation of patients with positive and negative remodelling. The patients with negative remodelling had a higher incidence of prodromal angina than those with positive remodelling (p < 0.01).

Histological findings have confirmed that plaque rupture and plaque erosion cause AMI. It is said that lipid rich plaques with a thin fibrous cap are prone to rupture and that plaque rupture leads to subsequent thrombosis and a sudden occlusion of the artery. In this study, patients without prodromal angina showed positive remodelling. This result suggests that considerable compensatory enlargement had prevented angiographic stenosis; however, coronary atherosclerosis with positive remodelling and large plaque burden might be vulnerable to rupture and lead to sudden occlusion. On the other hand, eroded plaques do not always contain much plaque burden. A recent study reported the possibility that eroded plaques may grow rapidly compared to ruptured plaques.[5] This finding may relate to prior angina just before the onset of AMI.

It is well known that myocardial ischaemia is not always symptomatic. As this study was based on a detailed history of symptoms of cardiac origin from each patient, the significance of this silent ischaemia could not be evaluated and could be a limitation of this study.

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Authors' affiliations
Y Shimada, H Tanaka, S Jissho, H Iida, Cardiology Department, Tsukazaki Memorial Hospital, Hyogo, Japan

M Yoshiyama, Y Kobayashi, Y Nakamura, S Ehara, K Shimada, K Takeuchi, J Yoshikawa, Department of Internal Medicine and Cardiology, Postgraduate School of Medicine, Osaka City University, Osaka, Japan

Correspondence to: M Yoshiyama MD, Department of Internal Medicine and Cardiology, Postgraduate School of Medicine, Osaka City University, 1-4-3 Asahimachi, Abeno-ku, Osaka 545-8585, Japan; yoshiyama@med.osaka-cu.ac.jp

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