# Clinical and angiographic characteristics according to the culprit lesion in patients with acute myocardial infarction

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**Background:** Proportion of blood supply into left ventricle (LV) depends on which coronary artery contributes and association between different arrhythmias and various distributions of myocardial infarctions (MI) is known. The purpose is to evaluate the difference of clinical and angiographic characteristics according to the culprit lesions in patients with acute MI. **Methods:** We enrolled 1,372 acute MI patients (49.3% patients with non-left anterior descending (LAD) culprit lesion vs. 50.7% patients with LAD culprit lesion) underwent intervention from 2003 to 2010 after excluding the left main culprit lesion. We compared clinical and angiographic characteristics. We defined arrhythmic event as 2nd or 3rd degree atrioventricular block which needed pacemaker, ventricular tachycardia or ventricular fibrillation. Major adverse cardiac event (MACE) was defined as a composite of any cause of death, revascularization and recurrent MI. **Results:** Baseline characteristics were similar except frequent previous MI (8.3% vs. 5.0%, $p$=0.02) and coronary intervention (9.9% vs. 5.7%, $p$<0.01) in non-LAD culprit group. Patients with non-LAD culprit lesion showed higher incidence of cardiogenic shock (14.1% vs. 7.1%, $p$<0.01). Arrhythmic event was frequent in non-LAD culprit group (21.3% vs. 7.0%, $p$<0.01). On initial laboratory finding, B-type natriuretic peptide level was elevated in LAD culprit group (173±364 pg/mL vs. 234±565 pg/mL, $p$=0.03). LV ejection fraction was lower in LAD culprit group (50.6% vs. 47.7%, $p$<0.01). On angiographic finding, use of glycoprotein IIb/IIIa inhibitor (4.7% vs. 2.5%, $p$=0.04) and thrombectomy (27.1% vs. 19.7%, $p$<0.01) was frequent in non-LAD culprit group. During median 690 days of follow-up, there was a trend for increased incidence of MACE in LAD culprit group (13.5% vs. 17.0%, $p$=0.08). **Conclusion:** In patients with acute MI, LAD culprit lesion is associated with pump failure and poorer long term outcome but non-LAD culprit lesion with higher incidence of arrhythmia and larger thrombus burden. Tailored treatment strategy should be considered according to the culprit lesion.



# Combining microRNA-449a/b with a HDAC inhibitor has a synergistic effect on growth arrest in lung cancer

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Histone deacetylases (HDACs) play a crucial role in tumorigenesis. Over-expression of HDACs has been reported in lung cancer. The mechanism of highly expressed HDAC1 in lung cancer has yet not been determined. In the present study, we showed that miR-449a/b regulates HDAC1 by directly binding with the 3′ untranslated region of the HDAC1. The expression of miR-449a/b was down-regulated and the expression of HDAC1 was up-regulated in primary lung cancer. The down expression of miR-449a/b might be one mechanism for over-expression of HDAC1 in lung cancer. miR-449a/b inhibited cell growth and anchorage-independent growth. Furthermore, co-treatment with miR-449a and HDAC inhibitors had a significant growth reduction compared with HDAC inhibitors mono-treatment. These results suggest that miR-449a/b may has a tumor suppressor function and might be a potential therapeutic candidate in patients with primary lung cancer.