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Author HSING-CHUN CHUNG
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Abstract
Atherosclerosis in western countries and Taiwan is the major cardiovascular diseases. The migration of vascular smooth muscle cells (VSMCs) from the media into the neointima is a key event in the development of atherosclerotic lesion and in restenosis. An important hallmark of vascular injury is the increased proliferation and migration of vascular smooth muscle cells (VSMCs) associated with the transformation from contractile to synthetic phenotype. Antrodia camphorate (AC) is a well-known traditional Chinese medicine. It has been shown to inhibit the proliferation and migration of cancer cells. This study examined whether AC could inhibit rat aortic smooth muscle cell (RASMC：A7r5) proliferation and migration and evaluated its effect on neointima formation in mouse carotid artery after injury. MTT and BrdU proliferation assay were used to evaluate AC effect on SMC proliferation. Both assays showed AC inhibits PDGF-induced SMC proliferation. In Transwell migration assay, RASMCs were treated with AC or saline. PDGF (10 ng/ml) was added to the lower compartment. Migrated cells on the lower side were fixed and counted. In wound scratch assay, RASMCs were pretreated with AC or saline and were seeded into confluent cell monolayers. They were wounded with pipette tips and stimulated with 10 ng/ml PDGF for 24 hrs. Both assays also showed AC could significantly inhibit PDGF-induced SMC migration. The left common carotid arteries of C57BL/6 mices were ligated near the carotid bifurcation. The mice were fed with water or AC for 4 wks. The severity of neointima was expressed as neointima/media (N/M) ratio. The AC-treated mice had less neointima formation at 4 wks after carotid ligation (N/M ratio, water, n=13 vs 250 mg/kg AC, n=12 vs 1250 mg/kg AC, n=12；1.33±0.87 vs 0.83±0.45 vs 0.63±0.32, $P < 0.05$). The data indicate that AC is an effective inhibitor of PDGF-induced RASMC proliferation and migration. AC treatment reduced neointima formation in mouse carotid ligation model.
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