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Role of angiopoietin 1 on the mechanism of canstatin inhibiting tumor growth and lymphangiogenesis in colon carcinoma CT-26 animal model.

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대장암 CT-26 동물 모델에서 tumor growth와 lymphangiogenesis를 억제하는 canstatin의 mechanism에서의 angiopoietin 1의 역할

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Objectives

Canstatin, a noncollagenous domain of collagen type IV α-chains, is potent inhibitor of angiogenesis with a distinct antitumor activity. And Tumor lymphangiogenesis is an attractive target for therapeutics designed to restrict the metastatic spread of cancer. So, we investigated the inhibitory effect of recombinant canstatin on tumor growth and lymphangiogenesis in a colon carcinoma CT-26 animal (BALB/c) model.

Materials and Methods

○ Materials

Mouse colon carcinoma CT-26 cell, Human Lymphatic endothelial cell(LEC), recombinant canstratin, recombinant angiopoietin 1

○ Methods

Cell culture, MTT assay, immunohistochemistry, western blot, RT-PCR, proliferation assay, migration assay, tube formation assay

Results

In colon carcinoma CT-26 animal model for in vivo analysis, the final volume and weight of tumors in groups treated with purified canstatin were reduced compared to a control group treated with PBS. Lymphatic vessel density of tumors was significantly decreased in canstatin-treated tumors. Expression of angiopoietin 1 at hypoxia condition was down-regulated in canstatin-treated CT-26 cells. The proliferation, migration and tube formation of angiopoietin 1-treated lymphatic endothelial cells (LEC) were reduced by recombinant canstatin. Taken together, our results indicate that angiopoietin 1 plays an important role on the mechanism of canstatin inhibiting tumor growth and lymphangiogenesis in colon carcinoma CT-26 animal model.

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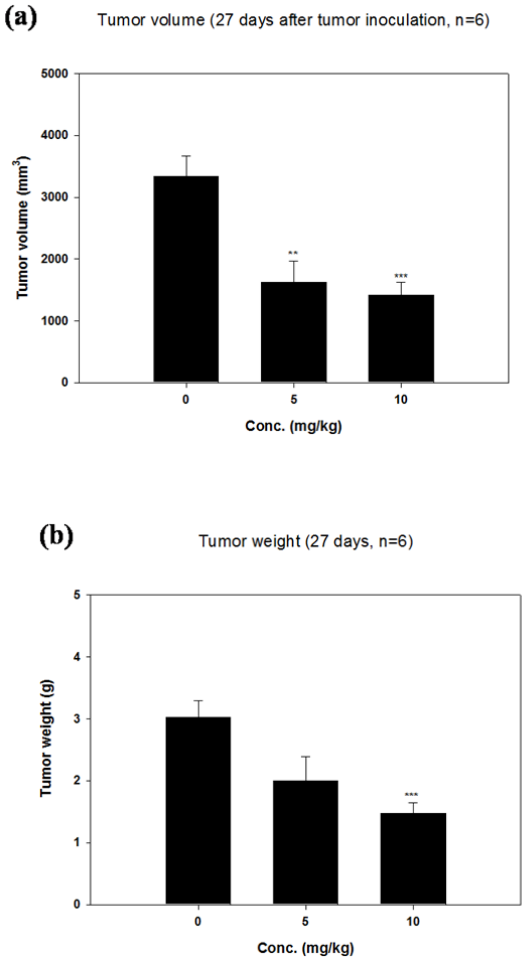

Fig. 1. Recombinant canstatin inhibited tumor growth in a colon carcinoma CT-26 animal model. CT-26 cells (5×10^5 cells in 50 μl PBS) were injected into the right flank of BABL/C mice. Six days after tumor inoculation, mice were treated daily with a intraperitumor injection of recombinant canstatin (5 mg/kg·day and 10 mg/kg·day in PBS) and PBS (control) for 13 days. All mice were sacrificed 27 days after CT-26 injection, and tumor volume (a) and tumor weight (b) were measured. Data are presented as mean ± S.E. Statistically significant differences compared with the control group were determined using Student's t test (* $p<0.05$).