Role of stem cell factor and monocyte chemoattractant protein-1 in the interaction between fibroblasts and mast cells in fibrosis

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Mast cell infiltration and accumulation is known to occur in tissue fibrosis. Increased numbers of mast cells are detected in scleroderma or hypertrophic scar skin, however, neither the role of mast cells nor the interaction of fibroblasts and mast cells in fibrosis are fully understood. A growing body of evidence indicate that mast cells are rich source of cytokines, growth factors or chemokines, which are suggested to play an important role in the induction of fibrosis. Recent in vivo and in vitro studies suggest the involvement of monocyte chemoattractant protein-1 (MCP-1), a member of the C-C chemokine family, in fibrosis. Here, we examined the effect of stem cell factor (SCF), a mast cell growth factor, on MCP-1 gene expression in a human mast cell line, HMC-1, and as well as the effect of MCP-1 on alpha1(I) collagen gene expression in human skin fibroblasts. HMC-1 cells spontaneously expressed MCP-1 mRNA transcripts, which was detectable by in situ hybridization and Northern blot analysis. Stimulation with SCF further upregulated MCP-1 mRNA expression in a time- and dose-dependent manner, and stimulation with 100 ng/ml SCF for 24 h induced a 3-fold increase of MCP-1 mRNA expression in HMC-1 cells as compared with unstimulated cells. The concentration of MCP-1 protein in the culture supernatants of 50 ng/ml SCF-stimulated HMC-1 cells (3816+/-70 pg/ml) was significantly elevated compared to unstimulated cells (2588+/-130 pg/ml) ($P < 0.01$), as assessed by ELISA. Adversely, MCP-1 induced alpha1(I) collagen mRNA expression in normal skin fibroblasts dose-dependently. Finally, comparative study revealed that the concentration of SCF in the culture supernatants of scleroderma fibroblasts at primary passages was significantly increased (344.6+/-182.4 pg/ml), as compared with normal skin fibroblasts (72.4+/-20.2 pg/ml) ($P<0.05$). These results suggest that fibroblast-derived SCF upregulates MCP-1 expression and synthesis in mast cells, which acts on fibroblasts to enhance alpha1(I) collagen mRNA expression. Our data may indicate an important interaction of fibroblasts and mast cells, via SCF and MCP-1, in the induction of fibrosis.

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