Impact of chronic inflammatory processes on kidney fibrosis

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Kidney fibrosis is an important pathway to progressive kidney diseases independent of etiologies, resulting in end-stage kidney diseases. Cellular mechanisms have been proposed in the pathogenesis of fibrotic processes in the kidney as well as other organs. In these clinical and pathophysiological settings, cell sources underlying the generation of matrix-producing cells in diseased kidneys have been categorized as activated resident stromal cells, infiltrating bone-marrow-derived cells, and cells derived from epithelial-mesenchymal transition/endothelial-mesenchymal transition.

Based on recent studies, accumulating evidence has shed light on the involvement of bone-marrow-derived cells, including monocytes/macrophages, and a circulating mesenchymal progenitor cell in the progression of fibrosis in kidney diseases. Bone-marrow-derived cells positive for CD45 and type 1 (pro)collagen dependent on the chemokine and renin-angiotensin systems migrate into diseased kidneys and enhance cascades of matrix protein production, expression of cytokines/chemokines and profibrotic growth factors, which may promote and escalate chronic inflammatory processes and possible interaction with resident stromal cells, thereby perpetuating kidney fibrosis. In contrast, little is known on anti-inflammatory and anti-fibrotic mechanisms during the progression of kidney fibrosis. Myeloid derived suppressor cells and adipose-derived stem cells may be involved in anti-inflammatory processes, leading to the reduction in kidney fibrosis. Further studies on the interactions between fibrotic and anti-fibrotic mechanisms involved will be required for the better understanding of kidney fibrosis.

Key words: kidney, fibrosis, inflammation