Modulation of monocyte-derived dendritic cell differentiation is associated with Ischemic acute renal failure.

許準榕

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Abstract

BACKGROUND: Dendritic cells (DCs) play a central role in both stimulating and suppressing immune responses and are impacted by surgical injury, exercise, and other physiological stressors. This study aims to determine whether renal ischemia/reperfusion (I/R) injury alters the differentiation, maturation, and activation of DCs from peripheral blood monocytes (PBMo). MATERIALS AND METHODS: Sprague-Dawley (SD) rats were subjected to I/R injury or sham-operated. Creatinine clearance (CCr) was monitored daily during the 14 days of reperfusion that followed the ischemic insult. At 2 and 14 days of reperfusion, the following properties of PBMo derived-DCs were assessed: the amount of generated DCs, surface markers [CD11c, CD80, CD86, and MHC-II (IA)], and functional status including magnitude of mixed lymphocyte reaction (MLR), production of IL-12 p70 by DCs, and production of IFN-gamma and IL-4 by DC-stimulated T cells. RESULTS: CCr was greatly reduced in the injured rats 0 to 4 days after ischemia. Two days after I/R injury to kidney, the numbers of DCs differentiated from PBMo, IL-12 production by DCs, expression of MHC-II (IA), and IFN-gamma production by DC-stimulated T cells were significantly increased in the I/R injured group (compared to the sham-operated group). After 14 days of reperfusion, there was no between-group differences in the numbers of DCs derived from PBMo, MLR, expression of CD80, CD86, and MHC-II (IA), and production of IL-12, IFN-gamma, and IL-4. CONCLUSIONS: The increases seen at 2 days of reperfusion may reflect a preparatory step in the renal I/R injury pathway. The relationship between up-regulation of DC differentiation and ischemic acute renal failure (ARF) remains to be elucidated.