Renal ischemia/reperfusion injury inhibits differentiation of dendritic cells derived from bone marrow monocytes in rats.

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Abstract

Dendritic cells (DCs) are impacted by surgical injury, exercise, and other physiological stressors. This study aims to determine whether renal I/R injury affects 1) the differentiation of myeloid DCs from bone marrow monocytes (BMMos) and the maturation and activation state of these DCs and 2) DC infiltration of kidney. Sprague-Dawley rats were subjected to I/R injury or sham-operated. Creatinine clearance was monitored daily during the 14 d of reperfusion that followed the ischemic insult. At 2 and 14 d of reperfusion, the following were assessed 1) properties of BMMo-derived DCs (i.e., the amount of generated DCs, differentiation state markers [CD11c, CD80, CD86, and Ia], and functional state [MLR and amount of IL-12 produced]), and 2) the presence of DCs in the kidney. Numbers of BMMo-derived DCs were significantly decreased in the I/R injured group (compared with the sham-operated group) at 2 d but not 14 d. A comparison of the their functionality found mixed lymphocyte response [MLR] and IL-12 production were similar in the two groups at both time points. Also, immunohistochemistry showed infiltrating DCs in the outer medulla of the I/R injured kidney at 2 d but not 14 d of reperfusion. Thus, I/R stress reduces the number of DCs differentiated from BMMos but not the functional activity of these DCs. This decrease may reflect a stress-induced downshift in the capacity of BMMos to differentiate into DCs and a parallel upshift in the capacity of DCs to infiltrate the kidney.