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Control of glomerular hypertension limits glomerular injury in rats with reduced renal mass.

S Anderson, T W Meyer, H G Rennke, and B M Brenner

Abstract

Micropuncture and morphologic studies were performed in four groups of male Munich-Wistar rats after removal of the right kidney and segmental infarction of two-thirds of the left kidney. Groups 1 and 3 received no specific therapy. Groups 2 and 4 were treated with the angiotensin I converting enzyme inhibitor, enalapril, 50 mg/liter of which was put in their drinking water. All rats were fed standard chow. Groups 1 and 2 underwent micropuncture study 4 wk after renal ablation. Untreated group 1 rats exhibited systemic hypertension and elevation of the single nephron glomerular filtration rate (SNGFR) due to high average values for the mean glomerular transcapillary hydraulic pressure difference and glomerular plasma flow rate. In group 2 rats, treatment with enalapril prevented systemic hypertension and maintained the mean glomerular transcapillary hydraulic pressure gradient at near-normal levels without significantly compromising SNGFR and the glomerular capillary plasma flow rate, as compared with untreated group 1 rats. Groups 3 and 4 were studied 8 wk after renal ablation. Untreated group 3 rats demonstrated persistent systemic hypertension, progressive proteinuria, and glomerular structural lesions, including mesangial expansion and segmental sclerosis. In group 4 rats, treatment with enalapril maintained systemic blood pressure at normal levels over the 8-wk period and significantly limited the development of proteinuria and glomerular lesions. These studies suggest that control of glomerular hypertension effectively limits glomerular injury in rats with renal ablation, and further support the view that glomerular hemodynamic changes mediate progressive renal injury when nephron number is reduced.

Full text

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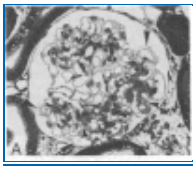

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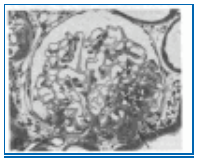

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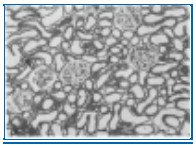

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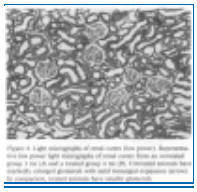

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