LABORATORY STUDY

Renal Ischemic Injury Affects Renal Hemodynamics and Excretory Functions in Sprague Dawley Rats: Involvement of Renal Sympathetic Tone

Ibrahim M. Salman and Munavvar A. Sattar
Department of Cardiovascular and Renal Physiology and Pharmacology, School of Pharmaceutical Sciences, Universiti Sains Malaysia, Minden, Penang, Malaysia

Nor A. Abdullah
Department of Pharmacology, Faculty of Medicine, Universiti Malaya, Kuala Lumpur, Malaysia

Omar Z. Ameer and Muin F. Yam
Department of Cardiovascular and Renal Physiology and Pharmacology, School of Pharmaceutical Sciences, Universiti Sains Malaysia, Minden, Penang, Malaysia

Gurjeet Kaur
Department of Integrative Medicine, Advanced Medical and Dental Institute, Universiti Sains Malaysia, Kepala Batas, Penang, Malaysia

Md. Abdul Hye Khan
Tulane Hypertension and Renal Center of Excellence, Tulane University Health Science Center, New Orleans, Louisiana, USA

Edward J. Johns
Department of Physiology, Aras Winte, University College Cork, College Road, Cork, Ireland

The role of renal sympathetic nerves in the pathogenesis of ischemic acute renal failure (ARF) and the immediate changes in the renal excretory functions following renal ischemia were investigated. Two groups of male Sprague Dawley (SD) rats were anesthetized (pentobarbital sodium, 60 mg kg⁻¹ i.p.) and subjected to unilateral renal ischemia by clamping the left renal artery for 30 min followed by reperfusion. In group 1, the renal nerves were electrically stimulated and the responses in the renal blood flow (RBF) and renal vascular resistance (RVR) were recorded, while group 2 was used to study the early changes in the renal functions following renal ischemia. In post-ischemic animals, basal RBF and the renal vasoconstrictor response to renal nerve stimulation (RNS) were significantly lower (all p < 0.05 vs. control). Mean arterial pressure (MAP), basal RVR, urine flow rate (URF), absolute and fractional excretions of sodium (U₁₅ V and FE₀₂), and potassium (U₁₅ K and FE₁₅) were higher in ARF rats (all p < 0.05 vs. control). Post-ischemic animals showed markedly lower glomerular filtration rate (GFR) (p < 0.05 vs. control). No appreciable differences were observed in urinary sodium to potassium ratio (U₁₅ K/Fe₁₅) during the early reperfusion phase of renal ischemia (p > 0.05 vs. control). The data suggest an immediate involvement of renal sympathetic nerve action in the pathogenesis of ischemic ARF primarily through altered renal hemodynamics. Diuretic, natriuretic, and kaliuretic effects were used to improve renal tubular functions. Novel responses to renal ischemia are of comparable magnitudes.

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Address correspondence to Ibrahim M. Salman, Department of Cardiovascular and Renal Physiology and Pharmacology, School of Pharmaceutical Sciences, Universiti Sains Malaysia, 11800 Minden, Penang, Malaysia, Tel.: +601 64611514; E-mail: ibrahim_msalman@yahoo.com

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