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Microvascular Cell Death In Spontaneously Hypertensive Rats During Experimental Inflammation

Dong San Kidney Institute Keimyung University School of Medicine,
Department of Bioengineering University of California[1]

Eun-Joo Chang, Kyo-Cheol Mun, Geert W. Schmid-Schönbein[1],
Seung-Yeup Han Eun-Ah Hwang, Sung-Bae Park, Hyun-Chu Kim

Objective: Hypertension is one of the most common complications in patients with end-stage renal disease. Hypertensives have an increased risk for tissue injury that may be mediated in part by endothelium and inflammatory cells. To clarify a possible underlying mechanisms, we examined leukocyte migration in the microcirculation and concomitant parenchymal cell death.

Methods: The rat mesentery of spontaneously hypertensive rats (SHRs) and their normotensive controls, the Wistar Kyoto (WKY) rat, was examined with digital fluorescence microscopy after topical stimulation with an inflammatory mediator (f-met-leu-phe, 10-8M). The migratory pathways of individual leukocytes were traced while at the same time cell death was detected by use of a life-death indicator (propidium iodide) over a period of 3 hours.

Results: Both WKY and SHR had a progressively increasing number of leukocytes migrating across the endothelium in postcapillary venules into the tissue parenchyma. But parenchymal cell death was detected in a random pattern in the mesentery tissue, without correlation to the migratory positions of the leukocytes. While younger SHR (about 17weeks) exhibited the same level of cell death as age matched WKY rats, older WKY rats (about 30 weeks) had significantly lower levels of cell death while the SHR maintained the same number of parenchymal cell death as younger animals.

Conclusions: These results suggest that in the presence of an inflammatory mediator, the SHR may exhibit a stronger response to an inflammatory mediator than normotensive WKY rats, in a fashion that is age but not blood pressure dependent. Parenchymal cell death does not correlate with migration of activated leukocytes at the microvascular level.