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Prior Iscehmia Results the Resistance to Second Ischemic Insult

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A mouse model was established in which prior exposure to ischemia protected against a second ischemic insult imposed 8 or 15 days later (Park KM et al., 2001) The mechanisms of protection against ischemic insult may suggest the other approach of therapeutic development in ischemic acute renal failure. The aims of this study are to establish the model in Sprague Dawley rat with prior renal ischemia resistant to secondary ischemic insult, and to evaluate the degree of apoptosis in functional protection. We divided male Sprague Dawley rats into six groups. We induced ischemia by bilateral renal pedicle clamping for 45 minutes in Group II, III, IV, and in Group V, VI, first ischemia and second ischemia after 7 days by same method. Kidneys were harvested on day 1(II), 2(III), and 7(IV) after first operation, day 1(V), 2(VI) after second operation, and after sham operation as control(I). As renal function parameters, we measured BUN, serum creatinine, creatinine clearance, and fractional excretion of sodium, and analysed the degree of apoptosis by TUNEL method and DNA laddering. Serum creatinine was 0.52±0.46 mg/dL in Group I, 2.26±0.45 in II, 3.4±0.52 in III, 0.38±0.20 in IV, and significantly not increased in V(0.84±0.53) and VI(0.45±0.18). The internucleosomal DNA cleavage decreased in Group V, VI comparing to Group II, III. TUNEL-positive nuclei(X 400, 6~11HPF), restricted mainly to the outer medulla, were also significantly decreased in Group V, VI (0.25±0.45 in Group I, 21.94±11.43 in II, 11.13±5.33 in III, 8.08±4.70 in IV, 3.5±2.99 in V, and 4±3.09 in VI). Prior ischemia in rat was also resistant to secondary ischemic insult, and significantly decreased apoptosis to insult.