Myocardial Infarction Enhances Renal Tubular Damage in Rats with Chronic Renal Failure

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Background: Cardiac events are the main cause of death among patients with end-stage renal failure (RF). To date even a mild renal disease is considered a major risk factor for cardiovascular complications after myocardial infarction. To improve our understanding of the potential additional renal damage introduced by subsequent cardiac events and to propose measures for early diagnostics of this grave condition, we have established a model for myocardial infarction in chronic RF (CRF)-inflicted animals.
Methods: We have utilized a rat model in which myocardial infarction was induced four weeks post establishment of subtotal nephrectomy. Changes in renal performance were then assayed using two platforms: histological and biochemical.
Results: The data demonstrate that even though creatinine (Cr)-clearance and sera BUN levels are not further deteriorated compared to CRF alone, the combined disease- cardiorenal syndrome enhances pathological fibrosis in the kidney's medulla. We further show that acute MI enhances moderate renal injury as determined by Ngal levels which remain stably elevated upon CRS.
Conclusions: Acute cardiac injury in the setting of chronic renal failure is associated with biochemical and histological changes in the renal tubular sections. The data suggest that elevated urine Ngal can serve as a non-invasive biomarker for an LV dysfunction-related renal injury, even in the setting of stable renal function.