Incidence, severity and outcome of acute kidney injury according to KDIGO criteria between intensive care unit patients

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Objective: Aim of this study was to evaluate the incidence, severity and outcome of AKI in intensive care unit (ICU) patients according to KDIGO criteria.

Materials & Methods: This retrospective cohort study, involved adult patients (≥18 years) admitted in two ICUs: The cardio-surgical and polyvalent ICU of a tertiary care hospital centre, between January 2007 and December 2007. Patients who stayed more than 24 hours were included. Transplanted and chronic dialysis patients before admission to the ICU were excluded. AKI was classified according to the KDIGO criteria using both serum creatinine and urine output (UO) criterion during their hospital stay. Demographic, severity scores of illness (SOFA score) on admission and outcome data were collected.

Results: 382 ICU patients were included for the study. According to the KDIGO criteria 176 (46.1%) patients met criteria for AKI during the study period and were classified as non AKI 53.9%, stage I 12.8%, and stage III 8.6%. AKI patients were aged, 60.5±12 years vs. non AKI patients 52.2±16 years; p<0.001 and had higher SOFA score: AKI 6.4±3.5 vs. non AKI 3±2, p<0.001. Male AKI patients were significantly more than non AKI 67% vs. 54.4% p=0.012. Recovery of renal function worsened with the progression of KDIGO stage: stage I 71.3%, stage II 38.8% and stage III 9.1%; p<0.001. Renal replacement therapy (hemodialysis) was used in 1.6% patients. Overall in-hospital mortality was 14.4%. Mortality according to the groups was: non AKI patients 4.4%; stage I 12.8%, stage II 18.4%, stage III 75.8%; p<0.001. Kaplan-Meier curve for ICU survival by KDIGO stage with Cox regression analysis was statistically significant, p<0.001 (Figure 1).

Conclusion: High incidence of AKI according to KDIGO criteria was found between ICU patients. Even small changes of renal function are associated with significant worse outcome.

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Short-chain fatty acids inhibit oxidative stress and inflammation in mesangial cells induced by high glucose and lipopolysaccharide

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Recently, a connection between Short-chain fatty acids (SCFAs) produced by intestinal microbiota and kidney has been revealed. The aim of this study was to explore whether SCFAs or their specific G protein-coupled receptors 43 (GPR43) agonist inhibit oxidative stress and inflammatory response in glomerular mesangial cells (GMCs) induced by high glucose and lipopolysaccharide (LPS). Our research showed that treatment with SCFAs, especially acetate and butyrate, or GPR43 agonist significantly inhibited GMCs proliferation induced by high glucose and LPS, and then reversed the production of reactive oxygen species (ROS) and malondialdehyde (MDA), but increased the levels of antioxidant enzyme superoxide dismutase (SOD). Furthermore, SCFAs or GPR43 agonist obviously increased the protein expression of GPR43 induced by high glucose and LPS, but diminished the expression of adhesion molecule intercellular adhesion molecule-1 (ICAM-1), and then decreased the proinflammatory cytokine monocyte chemoattractant protein (MCP-1) and interleukin-1β (IL-1β) release from GMCs stimulated by the high glucose and LPS. These combined results support the hypothesis that SCFAs or GPR43 agonist can inhibit oxidative stress and inflammation of GMCs induced by high glucose and LPS, suggesting that SCFAs induced signaling pathway and may act as new therapeutic targets of diabetic nephropathy (DN).

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