≈ ORAL PRESENTATIONS ≈

OP 019

Identification of Acute Kidney Injury and Associated Renal Biomarker Panels Due To an Emerging Epidemic of Oxalic Acid Self Poisoning

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Abstract

Objective: Oxalic acid poisoning is causing an emerging epidemic of fatal human self poisoning in the rural communities of southern Sri Lanka as a component of locally produced laundry detergents. Ingestion of oxalic acid commonly causes renal toxicity and has a case fatality of around 25%. The current study aims to study further the acute kidney injury (AKI) using conventional and novel renal biomarker panels.

Methods: Clinical and demographic data were collected from 26 (Male/Female:11/15) oxalic acid intoxicated patients. Serial urine and blood samples were collected at 8, 16, 24 and 48 hours post-ingestion and 1 and 3 months after discharge. Serum creatinine levels were measured using the jaffe reaction. Serum cystatin C and urinary biomarkers (kidney injury molecule-1 (KIM-1), clusterin, albumin, beta-2-microglobulin, cystatin C, neutrophil gelatinase-associated lipocalin (NGAL), osteopontin and trefoil factor 3 (TFF 3)) were quantified using enzyme linked immunosorbent assays (ELISA). AKI grade (I-3) was diagnosed based on Acute Kidney Injury Network (AKIN) criteria. The time point where each biomarker reached its maximum (Tmax) was recorded. Peak biomarker concentrations within 24-hours were used to create receiver-operating characteristic (ROC) curves and correlations.

Results: 20 patients developed AKI and the majority had severe AKI (AKIN1 (4), AKIN2 (3), AKIN3 (13)). Baseline characteristics between patients with and without AKI were similar. Clusterin, albumin and urinary cystatin C had a Tmax at 8 hours post-ingestion. Serum creatinine, serum cystatin C and NGAL had a Tmax at 16 hours after intoxication. Area under ROC curves (AUC-ROCs) for serum cystatin C, clusterin, albumin, urinary

cystatin C and NGAL were 0.93 (95% CI: 0.80 – 1.0), 0.74 (95% CI: 0.55 - 0.94), 0.76 (95% CI: 0.57 – 0.95) 0.81 (95% CI: 0.61 – 1.0), and 0.82 (95% CI: 0.64 - 0.99) respectively. Correlation coefficient for serum creatinine and serum cystatin C was 0.82 (p= 0.0001).

Conclusions: Intoxication with oxalic acid led to severe AKI. Serum cystatin C, clusterin, albumin, urinary cystatin C and NGAL were the most promising novel renal biomarkers for AKI following self-poisoning with oxalic acid. Peak biomarker concentrations of serum creatinine and serum cystatin C within 24 hours were strongly correlated. Further studies should be conducted with larger patient numbers to confirm if these urinary markers can reliably predict AKI earlier than serum creatinine.