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Acute renal injury following methanol poisoning: analysis of a case series.

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Abstract

The objective of this paper is to document the prevalence of indicators of acute renal injury in a series of methanol-poisoned patients treated in an intensive care unit and to discuss the possible mechanisms. This is a retrospective analysis of the medical records of 25 consecutive patients admitted to the intensive care unit after severe intentional methanol poisoning. Acute renal impairment was defined as a serum creatinine concentration higher than 177 micro mol/L and/or a urinary output on admission and for the first 24 h below 0.5 ml/kg/h. Clinical pathological signs of acute renal injury were found in 15 patients. In comparison with the 10 other patients taken as control group, the patients who developed renal injury had a lower blood pH value on admission, a higher serum osmolality, and a higher peak formate concentration. Two factors contributing to renal injury could be identified: hemolysis and myoglobinuria. The role of osmotic changes (osmotic nephrosis) or of a direct cytotoxic effect of formic acid remains speculative. Analysis of proteinuria suggests that proximal tubular cells may be preferentially affected. Results of histopathological evaluation of the kidney on a limited sample size (n = 5) were inconclusive but suggestive of possible hydropic changes in the proximal tubule secondary to methanol toxicity. Acute renal injury may be associated with other signs of severity in methanol poisoning, but it is almost always reversible in survivors. Indicators of acute renal injury were identified. The pathophysiology of this acute renal injury is multifactorial and far more complex than shock-related tubular necrosis.

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