Proteinuria in Ethylene Glycol Induced Acute Renal Failure

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From days 4-18 after intoxication, we repeatedly measured the urinary concentration of immunoglobulin G, albumin, \( \alpha \)-microglobulin and endogenous creatinine clearance (fig. 1). Ethylene glycol itself is probably not nephrotoxic, but in the metabolism of ethylene glycol, glycolate and oxalate are generated (fig. 2). Glycolate causes a metabolic acidosis, while oxalate can lead to crystalluria and renal failure.

Dear Sir,

Acute renal failure is one of the major complications in ethylene glycol intoxication [1]. To describe the functional relevance of histologically proven glomerular and tubular damage in ethylene glycol intoxication, we serially measured glomerular and tubular marker proteins in urine by a highly sensitive immunoluminometric assay [2] in a patient with ethylene glycol intoxication.

A 22-year-old man drank about 500 ml of rye (40 vol% ethanol), and afterwards ingested 100-200 ml of ethylene glycol. Five hours after ingestion, vomiting was induced by syrup of ipecac in order to achieve primary elimination of ethylene glycol. Afterwards, forced diuresis was started (about 400 ml/h) and ethanol was administered continuously. A metabolic acidosis was corrected by infusion of 250 mmol sodium bicarbonate. After 36 h serum creatinine rose from 72 to 259 µmol/l and crystalluria was observed. The patient was transferred to our hospital, where hemodialysis was started immediately (bicarbonate dialysis, on first day two times 2.5 h, afterwards daily 3-4 h, blood flow 200 ml/h, PAN dialysator, 1.4 m²). As symptoms of intoxication besides acute renal failure, perimyocarditis and transient palsy of the oculomotor nerves were seen. The acute renal failure was nonoliguric, serum creatinine rose to a peak concentration of 542 µmol/l 7 days after intoxication but returned to 95 µmol/l 22 days after ethylene glycol ingestion. A microscopic erythrocytu-ria was detectable during the first week after intoxication, urinary pH was 6-7. Ultrasonography showed swollen kidneys with increased cortical echogenicity, a typical picture in ethylene glycol poisoning [3].

Days after intoxication

Fig. 1. Immunoglobulin G (□), albumin (○) and \( \alpha \)-microglobulin (α-MG, •) concentration in urine related to urinary creatinine concentration (mg protein/g creatinine). There is no abnormal IgG excretion, albumin and αi-MG excretion initially exceeded the normal range by 5.5 and 250, respectively, and normalised during the observation period. With recovery of tubular concentrating capacity creatinine clearance (■) improves.

70-1
acidosis with an elevated anion gap [4], and oxalate leads to the generation of calcium oxalate crystals. Ethylene glycol is eliminated by the kidneys with a half-life of about 18 h [5] but the kidneys only exhibit a low clearance of its metabolites which can be removed effectively by hemodialysis [4].

In the treatment of ethylene glycol intoxication, the goal is to prevent the generation of toxic metabolites. Ethylene glycol is converted to glycolaldehyde by alcohol dehydrogenase, which can be inhibited by ethanol [6] or 4-methylpyrazole [7]. The renal clearance of ethylene glycol can be increased by forced diuresis [8]. The development of a metabolic acidosis indicates that ethylene glycol was already metabolised and hemodialysis is the treatment of choice.

Oxalate is held responsible for most of the organ damage in ethylene glycol intoxication: calcium oxalate crystals cause endothelial damage and induce a vasculitis [9]. Deposition of the crystals probably induces neurologic symptoms and perimyocarditis [10, 11]. In renal biopsies after ethylene glycol intoxication crystals are found in large quantities in the glomerular interloop spaces as well as in the tubules and interstitium [5, 11]. The functional relevance of these different sides of injury is not clear. In our measurement of glomerular and tubular marker proteins no glomerular protein leakage was detectable, but a massive, reversible tubular proteinuria was. Since proteinuria was of tubular origin and quickly reversible, we did not perform a renal biopsy.

Our case indicates that renal impairment in ethylene glycol intoxication is not primarily caused by a glomerular damage but by acute tubular necrosis as a consequence of tubular or interstitial deposits of calcium oxalate crystals. Glomerular proteinuria, which could be expected in renal vasculitis, was not seen.

References


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