Complicated diagnosis of ethylene glycol poisoning: A case report

Ruta Leksiene, Laima Peleckaite, Inga Skarupskiene, Asta Stankuviene, Jonas Surkus, Vytautas Kuzminskis, Inga Arune Bumblyte, Edita Ziginskiene

Nephrology Clinic, Lithuanian University of Health Sciences, Kaunas, Lithuania

Acute ethylene glycol poisoning usually results in typical clinical and laboratory findings in early hospital presentation[1]. The diagnostic of such poisoning in early presentation is based upon the case history about intentional or unintentional ethylene glycol ingestion, clinical view of metabolic acidosis, CNS depression signs, abdominal irritation and subsequent renal injury. Laboratory findings in early presentation are typical and evident also: severe metabolic acidosis, with elevated anion and osmolar gap, possible elevated lactates in blood and typical in shape calcium oxalates crystals in urine.

Sometimes the diagnostic may be complicated if patients are admitted late and renal insufficiency is already apparent. In such cases a lot of causes of renal injury must be differentiated.

Renal failure in ethylene glycol poisoning is one of typical crystal-induced acute kidney injury (AKI). Crystal-induced AKI is caused by the intratubular precipitation of crystals, which results in obstruction[2]. In the case of ethylene glycol poisoning the calcium oxalates crystals precipitates in the intratubular lumen and induce intratubular renal obstruction with subsequent clinical symptoms of renal obstruction and AKI. Another etiology of crystal-induced nephropathy could be considered and differentiated because multiply drugs and toxins can induce it. Most common agents: uric acid, methotrexate, acyclovir, megadose of vitamin C, sulfonamide antibiotics, protease inhibitors[2].

Crystal-induced nephropathy's clinical symptoms depend upon the initiating agent. Most of drug related crystal-induced nephropathies can be asymptomatic and only elevated serum creatinine can be the diagnostic value[2]. In some acute cases, such as ethylene glycol poisoning, patient can present within one to seven days after poisoning with the symptoms of renal colic: acute flank or abdominal pain, nausea or vomiting.

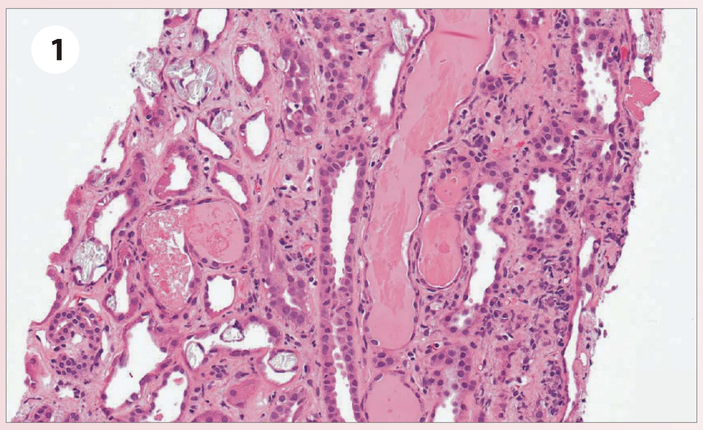
Most common findings in urine are hematuria, pyuria, crystaluria. Proteinuria usualy isn't common and can be if patient have underlying proteinuric kidney disease. The appearance of typical crystals in urine can let to establish true diagnosis, but not in all cases such crystals can be estimated in urine, especially in anuric or oliguric renal failure[2].

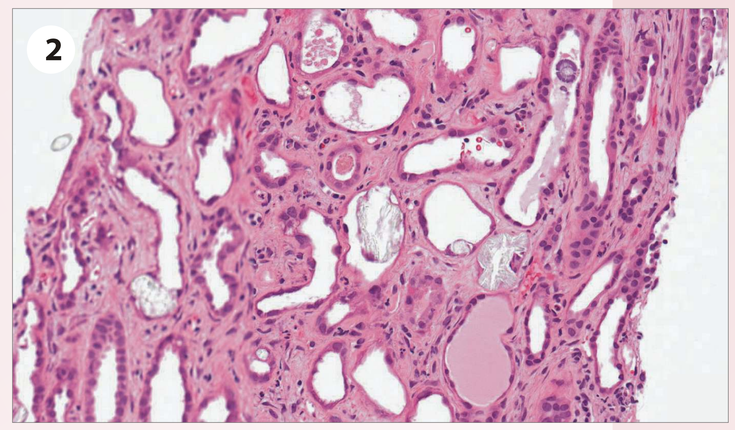
Definitive diagnosis of crystal-induced nephropathy could be made by histological examination obtained by renal biopsy. In most AKI cases renal biopsy isn't indicated if the AKI etiology agent is known or suspected unless atypical features are present[2].

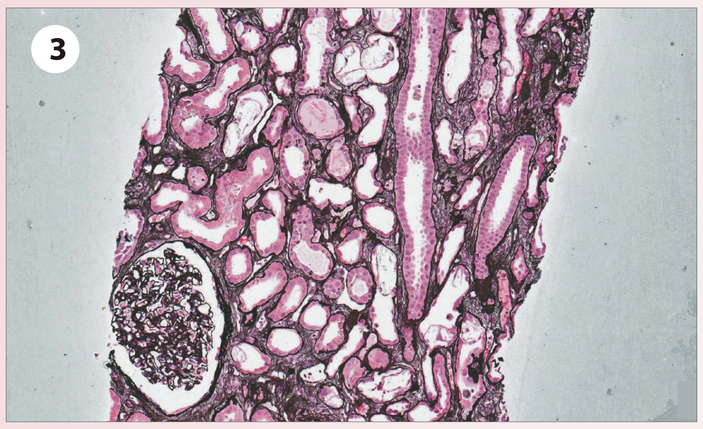
We need to differentiate crystal-induced AKI from any cause AKI[2]. In cases when patients present with hematuria and proteinuria the differentiation must be done to rule out the diagnosis of glomerulonephritis.

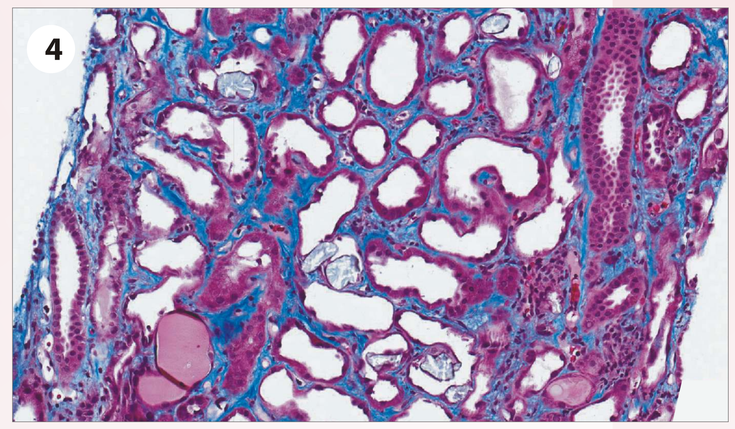
Crystal-induced AKI is generally reversed, although temporary dialysis may be necessary in some cases[2]. Chronic kidney disease may be a long-term consequence of crystal-induced AKI[2] also.

Case report: A 77-year-old male presented at the regional hospital after 3 days of illness with headache, nausea, vomiting, left-sided back pain and decreased diuresis. He had become unwell after ingestion of a soft drink. On physical examination he was cardiovasculary stable with symptoms of dehydration, mild hypoxemia, mild painful left-side abdominal palpation and superficial trophic ulcer on the left shin. Laboratory testing reveled elevated inflammatory markers and leucocyturia and uremia. Hepatic and pancreatic markers were normal; blood gases analysis was also normal (pH -7,4). Urinary infection was suspected and antibacterial treatment was started. Despite intensive antibiotic and rehydration treatment lasting 3 days the patient's condition worsened and renal failure progressed. He was transferred to the University hospital to start hemodialysis and to differentiate causes of renal insufficiency. Ultrasound examination showed normal kidney size with thickened renal parenchyma. It was differentiated from acute post-infection glomerulonephritis, sepsis, anti-neutrofil cytoplasmic antibodies (ANCA) associated vasculitis. A renal biopsy revealed unexpected calcium oxalate crystals in the renal cannaliculi with diffusive necrosis (figure 1, 2, 3, 4). The causes of renal oxalosis were discussed and the case history was revised. The patient and a friend had drunk antifreeze 3 days before admission to the regional hospital. His friend survived without sequel because he had also consumed ethanol.









https://www.vpc.lt/

Conclusions: Late presentation to the hospital after ethylene glycol poisoning can complicate and delay diagnosis. Prolonged vomiting with hypochloremic alkalosis can mask metabolic acidosis, a typical sign of such poisoning.

References:

1. Megarbane B, Borron SW, Baud FJ Ethylene glycol. In: Shannon: Haddad and Winchester's Clinical Management of Poisoning and Drug Overdose, 4th ed. by Saunders, an imprint of Elsevier Inc., 2007: 611-622.
2. Perazella M A Crystal-induced acute kidney injury (acute renal failure) Available at: http://www.uptodate.com
3. Fraser AD. Clinical toxicologic implications of ethylene glycol and glycolic acid poisoning. Ther Drug Monit 2002; 24:232.