Searching for the Lost Volatile Alcohol

Authors: Aleksandros Mihilli, Sandus Khan, Jifon Wang, Urvesh Patel

Anion gap metabolic acidosis is a commonly encountered condition in the hospital, with a wide array of possible etiologies. It is important to differentiate between the causes, and work through the differential, as the less common etiologies can be difficult to identify leading to delay in treatment.

Our patient is a 43-year-old male with past medical history of asthma and ADHD who presented to the ED with shortness of breath, difficulty with ambulation and altered mental status. The patient's symptoms had begun early that morning, primarily with progressively worsening shortness of breath. On arrival, he was tripoding; initial vitals remarkable for respiratory rate of 40 and oxygen saturation of 87% on room air, which improved to 94% after albuterol and solumedrol administration. He presented with creatinine of 2.61mg/dL, elevated liver enzymes, troponin and pro-BNP elevation, and on ABG his pH was 6.952, pCO2 11.5mmHg, and bicarbonate 2.4mmol/L. Lactic Acid was also noted to be elevated to 21mmol/L and he was negative for ketones. Initial workup ruled out ethanol, salicylate, and acetaminophen poisoning. Drug screen positive for prescribed Adderall. Urinalysis showed calcium oxalate crystals. No obvious source of infection was found; pan-cultures remained negative throughout the hospitalization.

At this point, possible accidental methanol or ethylene glycol ingestion was considered to be the cause after excluding other causes of anion-gap metabolic acidosis. A serum osmolal gap was obtained, which was found to be elevated at 34. The poison control toxicologist was contacted and he agreed that there was extremely high suspicion for possible toxic alcohol ingestion. We then gave fomepizole per their direction, as well as N-Acetylcysteine. Unfortunately, patient's sample for volatile alcohol panel and heavy metal panel was obtained 24 hours after symptom onset and were both negative.

He had to be dialyzed twice for solute clearance; lab values had started to normalize at the time of discharge. His liver enzymes continued to uptrend before downtrending and his encephalopathy persisted for several days. Over time, the lab values and clinical picture improved on supportive treatment and patient was discharged home.

Overall, despite being negative for volatile alcohols, the suspicion remains elevated due to presenting >12h after symptom onset and getting tested for volatile alcohols 24 hours after that. The half-life of ethylene glycol is 3-8 hours which could be enough for him to test negative if he was tested >24h after ingestion. Very few conditions can cause such profound metabolic acidosis (bicarbonate <8mmol/L), and he did not present with any of them. His elevated osmolal gap can be seen in conditions other than volatile alcohol poisoning but he tested negative for all of them. Lactate rapidly decreased within 24h of admission. In many cases lactate concentration may be dramatically high due to inability of instruments to differentiate between lactate and ethylene glycol metabolites. This case provides a textbook case of the workup for anion gap metabolic acidosis, while also giving an example of using diagnostic reasoning skills, when testing may have been delayed and impact the results.