Commentary

Nowadays, poisoning with an abundant variety of substances is not unknown to the clinical practitioner– regardless to the local setting. The patient' status is imminent for further treatment; priority lies in stabilization, airway assessment and (maybe) drug sample drawing, as well as taking a myriad of (often) useless blood tests.

Nevertheless, most alcohol poisonings present in the same manner: An almost or already comatose patient with maybe a history of substance abuse in a fast deteriorating condition. It is imminent to assess his or her neurological status (check for seizures, ataxia, tetany, etc.) and to exclude neurological diseases which may need urgent treatment, for example, diffuse cerebral swelling, meningitis, or brain hemorrhage; just to name a few. A cranial computed tomography (CT) is indispensable at that moment.

After excluding these life-threatening diseases, turn your attention to the blood tests you have taken: At least there should be arterial blood gases (ABG), glucose level, lactate level, creatinine, urea, creatine kinase, white blood cell count (WBC), and ethanol level. Necessary is also a urine analysis for drugs and hematuria.

In the case of "simple" ethanol intoxication, treatment possibilities lie somewhere between a "wait and see" strategy and a more aggressive therapy, like hemodialysis.

If you are uncertain, which treatment to choose, check the blood ethanol level, the stage of metabolic acidosis, and the osmolal gap.

Ethylene glycol (EG) intoxication (e.g., contained in antifreeze) can be suspected if the patients' setting or the history are evidentiary, the metabolic acidosis exceeds an arterial pH <7.3 or serum hyperosmolality (osmolal gap >20 mOsm/kg) occurs; if the serum EG level cannot be measured.[1] A higher osmolal gap can now be suggestive for initiating hemodialysis, which for example reduces the blood ethanol level up to 10 times faster than conventional treatment with fluid boluses.[2]

Specific treatment for toxic alcohol poisoning is more delicate: As ethanol and fomepizole (4-methylpyrazole) are also catalyzed by the liver enzyme alcohol dehydrogenase (ADH), similar to EG and methanol, treatment of the toxic alcohol poisoning would be to supply one of these to the patient.

However, ethanol treatment is a risky business: It is like playing with fire, as the ethanol blood level has to be continuously monitored and it has some disadvantageous effects on its own. Fomepizole, on the other hand, is expensive and not available in all clinical settings, especially not in the more rural hospitals.

Single therapy with fomepizole alone is not advisable, if the patient presents with acute renal failure and a severe metabolic acidosis; in that case, hemodialysis is the most important treatment with clearing EG, methanol, and its metabolites effectively.[3] In the majority of cases, it is recommended with EG or methanol concentrations above 50 mg/dl.

Santana-Cabrera and colleagues describe in this issue of "Journal of Neurosciences in Rural Practice" the toxic effects of EG on the cerebral white matter (caudate and putamen nuclei) with an initially unremarkable CT scan in a severe intoxication.[4] The treatment chosen was based on the clinical and laboratory findings mentioned earlier; however, the patient did not recover and maintained comatose. The EG concentration was about 285 mg/dl and compelled starting hemodialysis due to an accompanying acute renal failure.

So, what is the truth within? Always consider toxic alcohol intoxication in a comatose patient and concomitant metabolic acidosis or even hyperosmolality. Do not forget to order an initial CT scan, even if it may return nonspecific. Targeted treatment consists of fomepizole, ethanol, and even hemodialysis in severe cases.

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