

Renal Injury with Isopropyl Alcohol?

A 30-year-old female with a history of heavy alcohol use presented to the ED after ingestion of isopropyl alcohol (rubbing alcohol). On presentation, she had mild tachycardia, was clearly intoxicated, and had some interesting laboratory abnormalities. She had an osmolal gap of 48 (normal -2 to 10), anion gap of 8 (normal 3 to 10), a blood urea nitrogen (BUN) of 4 mg/dL (normal 7 to 20), and a serum creatinine (SCr) of 2.02 mg/dL (normal 0.6 to 1.2).

Isopropyl alcohol is reported to be approximately twice as inebriating as ethanol and can cause significant CNS depression (*Clin Toxicol* 2014;52(5):470-478). The onset of action is about 30 minutes, which is similar to ethanol. Common clinical effects include inebriation, CNS depression, and vomiting. Some reports describe coma, hypothermia, and shock. There is cross-tolerance with ethanol and people who regularly drink ethanol will have a higher tolerance to isopropyl alcohol. Initial assessment includes ABCs along with a basic metabolic panel (BMP) and a measured osmolality.

Isopropyl alcohol also causes pseudo-renal failure with a false elevation of the SCr (*Southern Med J* 2009;102(8):867-869; *Am J Ther* 2011;18(4):e113-e116). In many clinical/hospital laboratory basic metabolic panels (BMP), the serum creatinine is measured via the Jaffe reaction (*PLoS ONE* 2015;10(11):e0143205-e0143221). Creatinine in the plasma interacts with picric acid resulting in a change in the color of the plasma that is directly proportional to the concentration of the creatinine. Experiments have shown that many products with a carbonyl group (including creatinine) will react with picric acid to produce a color change (*Clin Chem* 1987;33(7):1129-1132). The metabolic byproduct of isopropyl alcohol is acetone, which has a carbonyl group and will interact with the creatinine assay. It is estimated that about 100 mg/dL of acetone will raise the SCr by 1 mg/dL SCr (*Southern Med J* 2009;102(8):867-869; *Am J Ther* 2011;18(4):e113-e116). Acetone has a long but variable half-life of 7.7-27 hours (*Clin Toxicol* 2014;52(5):470-478). This means that patients may have an elevated serum creatinine for a prolonged period (2+ days).

One of the main alternative methods for measuring serum creatinine is enzymatic. This is utilized in most point of care creatinine tests and some other central laboratory assays. While the point of care test may not be as accurate as the central laboratory assay under normal circumstances, there is no interference from acetone, so in cases of large isopropyl alcohol ingestions, the point of care test will likely be more accurate than the central laboratory.

While it is possible that a patient with significant poisoning from isopropyl alcohol may have prolonged coma, hypotension, and rhabdomyolysis resulting in an acute kidney injury, this usually does not result in an isolated increase in SCr. In a true acute kidney injury, an increase in creatinine phosphokinase (CPK) and BUN are also expected, along with likely a decrease in urinary output.

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Did you know?

An elevated serum creatinine is a marker of isopropyl alcohol ingestion but not disease.

In a patient with elevated serum creatinine and no change in BUN or urine output after ingestion of isopropyl alcohol, an elevated serum creatinine is a marker of ingestion, and not necessarily associated with acute kidney injury. This is probably a false elevation due to laboratory interference. Confirmation can be performed through an alternative assessment of the creatinine (e.g. an enzymatic assay) and identification of a difference between the two results.



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